

BEFORE THE STATE WATER RESOURCES BOARD

AT CHARLESTON

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WATER RESOURCES BOARD

Transcript of the public hearing had at
Conference Room C, Conference Center, State Capitol Complex,
Charleston, West Virginia on the 20th day of June, 1991,
commencing at 7:00 p.m..

BEFORE: DR. CHARLES R. JENKINS, Acting Chairman

MS. SARAH LEE NEAL, Member

DR. EDWARD M. SNYDER, Member

DR. DONALD C. TARTER, Member

STAFF: JAN R. TAYLOR, Ph.D., Technical Advisor;

FRANCES E. HUNTER, Executive Secretary.

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I N D E X O F S P E A K E R S

	<u>Page</u>
Tennant, Peter	6
Reger, R. B.	9
Sherman, Richard	16
Calwell, Stuart	22
Flannery, David	28
Schatzow, Steven	36
Keenan, Russell	43
Brown, Steven	54
Packard, Vince	61
Gillespie, Donnie	62
Hagenboch, Brian	63
Bennett, Matthew	74
Lewis, Conni Gratop	76
Chmiel, Christopher	78
Taylor, Keith	80
Ragette, Bill	84
Woolverton, Missy	93
Bady, Rick	96
McDermitt, C. T.	101

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ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

I N D E X O F S P E A K E R S (cont'd)

	<u>Page</u>
McDaniel, Perry	104
Rank, Cindy	108
Hamilton, Beth	111
Steenstra, Norm	113
Reporter's Certificate	118

DR. JENKINS: Good evening. I'm used to speaking before a group of students, and so I'll make the same points I usually do there. I'll invite all of you people in the back of the room to move up closer here if you'd like, so you can participate a little more easily.

This hearing is-- I should, first of all, take care of some introductions. I'm sorry. And I'd like to start out by introducing fellow members of the Board. Sarah Lee Neal over here (indicating), and Ed Snyder (indicating) and Donald Tarter (indicating). And I'm Bob Jenkins, and I'll be standing in tonight for Dave Samuel, our normal chairman, who is out of the country. I'd also like to introduce our staff people. Jan Taylor here (indicating). And I saw Fran Hunter out in the hall. I'm not sure where Fran is.

MS. NEAL: She's over here (indicating).

MS. HUNTER: Here.

DR. JENKINS: There she is. I'm sorry. She moved on me. And our court reporter tonight is Deborah Dickens.

This hearing is convened pursuant to Chapter 29A of the West Virginia Code of Regulations under the Federal Clean Water Act for the purpose of hearing comments on proposed revision to the legislative rules. The proposed

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rule and the notice of public comment period was filed with the Secretary of State on May 17, 1991. Public notice was also published in the Charleston Gazette and Daily Mail on May 17, 1991 as well as mailed to over a hundred individuals on the Board's mailing list.

Corrections of typographical and clerical errors were filed with the Secretary of State as an errata sheet and sent to the individuals on the mailing list on May 19th.

We are hearing comments tonight only on Title 46, Series I, requirements governing water quality standards. I want to give the people here an opportunity to present their main concerns tonight, but I ask you to limit your oral comments to a summary of your position and to no more than five to ten minutes so that everyone who wants to speak will have a chance to speak.

We encourage you to submit detailed written comments tonight and through the close of business on June 28, 1991. All comments, both written and oral, will be carefully considered by the Board in preparing the final position to be submitted to the Legislature. The Board will meet several times in the next month to study the comments and to make final decisions.

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The Board will also prepare a rationale document which will respond to all comments received during the public comment period and explain the Board's decisions. Both the final proposal and rationale document will be filed with the Secretary of State and the Legislative rulemaking Review Committee in mid-August. The revised rule will become effective following approval by the full Legislature and the Governor in late spring 1992.

I have a group of cards here, and I'll be calling your names from those cards. And when I do, please come to the podium and present your statement. Again, let me remind you to limit your presentation to five to ten minutes. I believe that there's also an attendance record, and if there is anyone here who did not get their name on that attendance record, why, please see Fran over here. I think she's probably the person who has that, Fran Hunter.

First of all, I'd like to invite Peter Tennant.

MR. TENNANT: Thank you. I have a written statement with me. I'll leave some copies with you and in the interest of time just summarize the written comments.

I'm Peter Tennant. I'm the Technical Programs Manager of the Ohio River Valley Water Sanitation

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Commission, or ORSANCO. As many people know, ORSANCO is an eight-state interstate water pollution control agency for the Ohio River Valley.

West Virginia is a member state as represented by three members of our commission, the Commissioner of the Bureau of Public Health, William T. Wallace, Edgar Henry, and Ron Potesta.

One of the major objectives of ORSANCO is to try to maintain compatible water quality standards for the Ohio River since nine hundred and forty-one of the nine hundred and eighty-one mile length deals with the interstate boundary, and so water quality standards adopted by one state can affect conditions in adjoining states and downstream states.

Our commission has taken a tact of adopting its own pollution control standards which set the basis for discharge requirements. The stream criteria contained in those standards represent the commission's opinion as to what should be the water quality standards of the Ohio River, and those numbers are developed by a committee of representatives from the state agencies who are involved with their own state's water quality programs.

Our standards were recently revised in October

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of this past year, and among the significant changes were the adoption by reference of U.S. EPA's recommended criteria promulgated under Section 304A for the protection of human health, and a cancer risk level of one in one million was selected as the appropriate level for the Ohio River.

We were, therefore, pleased to see the revisions proposed by the Board were totally in line with that, and a review of the standards as they would appear in the revised fashion indicates that in all cases they are consistent with the numbers we have adopted. There are, in fact, several parameters for which the Board has adopted specific criteria which do not appear in our standards at this time, and we would encourage the Board in our continuing endeavors and our next group meeting, perhaps, to present these, several of which we know to be present in the Ohio River and feel that the Board is taking a fine action in developing specific criteria for those parameters which are of concern. I mention specifically in the statement dichlorobenzenes, which have been found in this stretch of the river.

There are some minor omissions that I noted, missing power plants, and these are noted in the written statement, but overall we feel that the proposed revisions

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are a step forward and we support them.

Thank you.

DR. JENKINS: Thank you, Mr. Tennant.

Is Doctor R. B. Reger here? Doctor Reger.

DR. REGER: Thank you, Mr. Chairman, Members of the Board. The name is Bob Reger. I'm Research Associate Professor of Occupational Medicine, University Medical Center. I'm happy to be here.

The subject on which I will speak is dioxin. The major purpose of this testimony that I'm going to give is not to put forth some magic number which will provide man adequate protection from dioxin, but it's rather to put forth some concepts, merely concepts, to be considered in the development of a sensible standard.

So, in general, today what do we know about dioxin? In humans, chloracne can result. These lesions can last a long time, and in the healing process they can produce significant scarring. Liver function may also be impaired. While central and peripheral nervous system effects have been reports, that's fatigue, drowsiness, sleep disturbance, et cetera, chloracne remains the definite documented condition in man which is resulted from dioxin exposure.

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Of major concern, however, for the setting of water and other standards is the carcinogenic potential of substances such as dioxin which exist at very low levels. The issue of carcinogenesis relating to dioxin, when viewed in its infancy, could clearly not be settled using the experience of man or by using the experience of human epidemiology. Thus, the synthesis of other information is necessary.

Cancer of the liver and other sites have been induced in animal species, rats, guinea pigs and mice. There have been vast inner-species differences and responses, and high doses of dioxin have been administered to evoke the carcinogenic response. From such data, specific models for disease outcome have thus developed which are, or were, based largely on our dose response experience with other substances.

For example, a popular basic model involves the thesis that cancer is caused by the mutation of cellular DNA. Continued decreasing doses of the material, therefore, in animals and short-term bioassays become related to observable induced mutations. In short, this hypothesis involves no threshold of response whatsoever. Thus it is suggested that any exposure, however small, is associated

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with a finite positive likelihood of a deleterious effect.

Now from this type of information, how do we obtain estimates of carcinogenic risk for man from exceedingly low level exposures to some chemical compounds such as dioxin? Results from high-dose animal experimentation are personally extrapolated to untested and unproven low-dose exposure levels within the same species.

Secondly, extrapolation is then made across species to homo sapiens. The validity of the results from this double extrapolation procedure is directly correlated with the validity of at least two basic assumptions, generally whether or not an implied linear and non-threshold model for disease exists and whether or not man is like a mouse.

Clearly both of these assumptions are now and have been open to a lot of question. However, in the absence of other information, if we are to form opinions and base regulations on the only information available, this might seem very, very reasonable. To be sure, the framework of toxic substance regulation rests on the fragile premise that it is possible to identify which chemicals are hazardous and should be regulated and which are safe and can be ignored.

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This premise, in turn, rests on the assumption that there is a close correlation between carcinogenic effects in animals and those in man. Moreover, it may also rest on the even greater assumption that a similar correlation exists between results from short-term assays and long-term tests in animals such as skin painting.

Unfortunately, while regulatory agencies are being pressed to make rapid decisions about the safety of chemicals, the validity of the assumptions have been and continue to be a focus of sharp contention. There is enough doubt lingering around each assumption to cloud the issue when a proposed regulatory action is pending.

In short, with scientific rationale for regulation, while it is often questionable, regulations emanating from it is certain to be highly controversial. Dioxin is clearly a case in point. Reference to dioxin, which is carcinogenic to laboratory animals at high doses, is not a critical evaluation of the significance of this information for man.

Excess mortality from cancer amongst exposed homo sapiens, if it exists, is, without question, the least controversial basis for regulation when data exists as human epidemiology. While studying large numbers of individuals

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who have been exposed to specific environmental factors, it is often possible to show the amount of association between exposure and the onset of cancer or some other disease.

Nonetheless, from a public health perspective and when dealing with the issue of cancer, one cannot wait to count the bodies before we act. And we must make use of other and all information for the overall protection of the population.

Well, science marches onward, and what other information has more recently emerged on the scene as it relates to dioxin? The short-term bioassays for mutagenicity and bacterial systems are essentially negative. Moreover, there are at least three recent epidemiological efforts, albeit these are fraught with a lot of shortcomings. They're uniformly negative. Now from the negative mutagenic response from bioassays and other information, one might call into serious question whether or not the correct model for dioxin and carcinogenesis involves no threshold.

In fact, a body of scientific evidence now suggests that there, indeed, may be a dose threshold below which no response is likely. This is a critical departure from the use of untested assumptions now considered by some.

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In short, if the dioxin-reduced carcinogenesis from high-dose exposure in animals, if receptor mediated or involves toxicity-induced cell proliferation rather than the concept of a model based on a thesis that cancer is caused by the mutation of cellular DNA, then clearly the extrapolation of information to man by a linear non-threshold model for carcinogenic action is inappropriate. It would not adequately describe the cancer causing potential for man.

In addition to considering the adequacy of a non-threshold model, it is likewise implicit that dioxins would likely act as a promoter of cancer instead of acting as a direct carcinogen.

These are clearly the most critical bits of new information to be considered by those making policy for protecting the general health of the population. In addition to the most up-to-date scientific information relating to basic mechanisms of disease being considered by regulators, it is, indeed, necessary to also consider the site-specific variance associated with other important elements which are involved in risk assessment.

For example, for dioxin, within defined geopolitical limits, water intake as well as fish

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consumption by the population with all the minute details associated with that, such as percent fatty tissue by fish variety and so forth, these must all be considered in the development of a sensible standard.

While the absolute truth and correct answer relating to the carcinogenic risk to man from dioxin exposure will likely remain a mystery for some time to come, our estimates for human protection will have to suffice. However, it is now clear that our estimates now need to consider all available relevant information, scientific and otherwise, and we need not totally rely on information which was developed during the infancy of the issue on dioxin.

Rather than develop an entire series of magic numbers based on various and sundry models and criteria, all of which then become subject to administrative and political debate, it seems a more reasonable procedure to satisfy ourselves regarding the scientific underpinning involved in risk assessment, selecting those models and factors which are most nearly scientifically valid, and then proceeding with our derivations of risk.

As mentioned earlier, perhaps the most critical factor to now consider relates to what is the appropriate model. Regarding dioxin, the use of a threshold

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model of disease, cancer, seems to be the prevailing scientific trend, which, incidentally, supports a standard roughly a thousand or more times higher than the EPA recommendation of .013 ppq. We must, thus, move forward using the best data available to provide adequate protection for the population at risk.

Thank you very much.

DR. JENKINS: Thank you, Doctor Reger.

Is Richard Sherman here?

MR. SHERMAN: Good evening. My name is Dick Sherman. I'm here tonight on behalf of the West Virginia Chamber of Commerce and the West Virginia Manufacturers Association.

The West Virginia Chamber of Commerce represents more than a thousand businesses, trade and professional associations within the state. The West Virginia Manufacturers Association represents more than two hundred and fifty companies, including chemical, metal and glass manufacturers and manufacturers of various wholesale and consumer products.

We urge the Board to seriously consider the comments prepared by these organizations which we believe, if adopted will protect the state's water and help the state's economy by allowing existing industries to continue

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to operate and attract new industry to the state.

I'll comment on several specific amendments.

Measuring the bivalent forms of metals. The joint commentators urge the Board to recognize that current water quality criteria as expressed as total recoverable metals is unnecessarily stringent. Instead of continuing to establish criteria based on total recoverable metals, we urge the Board to established criteria based on acid soluble measurements and to approve a method for analyzing effluent that measures the bioavailable quantity of metals.

Failing that, the Board should allow dischargers to filter effluent samples or develop a site specific method dischargers may use to measure dissolved metals.

Control by appropriate methods for calculating bioavailable metals. The Board should give strong consideration to adopting one or more of these.

Analytical variability. The Board should recognize the existence of analytical variability when it sets criteria and should allow dischargers to demonstrate compliance with water quality standards by reporting results as below the practical quantification level, that is, pql, when the pql is larger than the water quality standards.

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Test results which establish that an animal life is not present at the pql would constitute compliance with permit limits for the parameter.

We further propose that the pql, for the purpose of determining permit compliance, be no less than ten times the mean of the method detection limit value applied by dischargers within the industry. Since method detection limits vary and are not necessarily reproducible over time in a given laboratory, even when the same analytical procedures and sampling methods are used, dischargers should be allowed to establish method detection limits that are specific to their individual discharges in laboratories.

Instream biomonitoring. We believe that the Board and Water Resources Section should not rely heavily on chemical specific data or all technical bioassay results in determining this attainment. Bioassay results can misleadingly suggest that quantified uses are not being obtained when population monitoring shows the use is being attained.

By evaluating the status of aquatic life and water quality, the Division can more accurately assess aquatic life use and determine whether effluent treatment is

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necessary. We suggest the addition of a new section that would provide that instream biological survey data can be used to assess the aquatic life use for the water body to determine appropriate concentration levels of constituents to support a base for consistent use.

Prohibition of acute toxicity, mixing zones. We oppose the prohibition of acute toxicity to aquatic organisms. The Board's regulations contain no guidance as to what constitutes acute toxicity or what organisms are to be considered in making that determination. Some definition of acute toxicity should be provided by the Board and the Board should clarify the mere exceedance of water quality based permit limit does not necessarily result in acute toxicity in the mixing zone.

The Board should also clarify that acute toxicity should be determined using only native species likely to inhabit the mixing zone and using exposure times which approximate the actual time of those already in a mixing zone rather than an arbitrary forty-eight or ninety-six hours.

In light of these deficiencies, we urge the Board to reconsider it's prohibition of acute toxicity, mixing zone in favor of a policy which defines acute

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toxicity and confines it to organisms likely to be present in the mixing zones.

Designated uses. The Board proposes, quote, when a discharge permit is to be issued, all uses shall be assumed present unless the applicant demonstrates that the designated uses do not apply to the stream segment in question, end quote.

This provision places guilt on every NPDES permit applicant on, for example, the Kanawha River, to prove that it is not discharging into, say, a trout stream. Such an approach is excessively burdensome and inconsistent with the preceding sentence which designates most waters of the state for fishable or swimmable uses. It is also inconsistent with Appendices A through D which specifically identify water bodies which are fit for certain uses.

These categories are unnecessary if each of the uses is presumed present for every state stream. Furthermore, there is no reason why a permit holder must disprove the existence of other uses when that is not required of non-point sources of pollution to which the same water quality standards should apply.

Finally, the Board's proposal raises the possibility that a discharger which obtains a site-specific

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variance for one year will be required to justify the variance once again when a permit is issued. For these reasons, we would urge the Board not to adopt the presumption that all uses apply to each West Virginia water body.

Time extensions. Many of the existing proposed water quality criteria are near or below the analytical detection level. Waste water treatment technology is not always available to consistently and efficiently remove pollutants to the low levels required to meet the criteria. A provision is necessary to allow for an extension of time to meet water quality based effluent limits when waste water treatment technology is not available to achieve those plans.

Water use categories, special criteria. Category B of the water use categories of Section 6.3 should be revised to include a category for other warm weather streams consisting of very small creeks and streams which typically flow only a portion of the year. Because low water in such streams is intermittent, stream use by fish is incidental but normally limited to these waters and the like.

We believe that a new use category such as

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intermittent streams should be created and the criteria in Series I should be revised to include new criteria for the protection of the streams.

Thank you for the opportunity to speak for the Chamber of Commerce and the West Virginia Manufacturers Association.

DR. JENKINS: Thank you, Mr. Sherman.

Is Stuart Calwell here?

MR. CALWELL: Before I address the Board with my comments, I feel constrained to mention, as a lawyer, that I should point out that this is a legal holiday and there may be some question about the notice under West Virginia Code 22-1 in which the public has a right to believe that this meeting would take place tomorrow rather than today. So you're here at the wrong time. And I would note my objection on the record in the event that I may need that at some future time.

As I stated, I am Stuart Calwell of the law firm of Calwell, McCormick, and Peyton, in Charleston, West Virginia. I am appearing on behalf of the Tri-State Building and Construction Trades Council, which is an association of construction trade unions affiliated with the AFL-CIO. The association is made up of local trade unions

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in West Virginia, Ohio, and Kentucky. The Tri-State Building and Construction Trades Council is also a member of the West Virginia Building and Construction Trades Council.

The members of these local unions live in hundreds of communities in Ohio, West Virginia, and Kentucky, and I am appearing on behalf of them as well. Collectively these unions represent over forty-four thousand tradesmen. Their families and the people in the communities where they live are worried about your decision to increase the amount of cancer-causing chemicals that can lawfully be dumped into the rivers and streams of West Virginia and the rivers that border our surrounding states.

Our recent studies, done on behalf of the unions that we represent, of certain rivers in West Virginia show that if your proposed rule goes into effect, we can expect in the rivers studied an increase of five and a half to almost eight times the amounts of carcinogens presently dumped in those rivers. Because of this increase, we can expect an increase in the risk of cancer to many of the citizens and residents of West Virginia, Ohio, and Kentucky.

Proposed rule 8.2.C, appearing on page nineteen of the new proposed rules, mandates that, and I quote, critical design flow for carcinogens shall be the

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harmonic mean flows when a permit applicant requests it. However, your notice of this proposed rule change does not explain to the public that with this rule you are advocating increasing the risk of cancer to human beings.

As you know, proposed rule 8.2.C has to do with how the dilution of carcinogens is calculated once they are discharged to a river. This calculation is important in determining the risk of cancer posed by a discharge. The more water there is in a river to dilute the carcinogen, the lower the risk of cancer. Low water in a river means there's not as much water to dilute the carcinogen.

Therefore, if the low water mark of a river is used when deciding how much of a carcinogen discharge to allow, the amount of carcinogen permitted will be small because when the river's at its low mark, there's not as much water available for dilution. It follows, therefore, that if the low water mark is used to fix the maximum amount of carcinogens discharged, then when the river is up, the carcinogen is diluted even more. Thus, as river flow increases, the level of cancer-causing chemicals becomes more and more diluted and the risk of cancer goes down.

During periods of drought and low water, because the initial determination of levels of carcinogens

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was based on the low water mark, no increase of cancer occurs.

The present rule, the one you are trying to change, has this built-in safeguard. It requires dischargers to limit carcinogens to the amount that can be discharged when a river is at the low water mark. The new rule, the one you're trying to implement, does not use the low water mark as the starting point to calculate how much river water there is to dilute carcinogens. Instead, the so-called, quote, harmonic mean flow, end quote, is recommended as the starting point. The harmonic mean flow of a river is always at a higher water mark than the low water mark.

Therefore, from the very beginning there will be an increase in the amount of carcinogens that can be dumped into a river. The risk of cancer, therefore, is increased from the beginning. In times of low water, because there is less water to dilute the already increased amount of carcinogens allowed by the new rule, the risk of cancer goes even higher.

Our studies show that the new rule will allow dischargers on the Ohio River to dump seven point four times more cancer-causing substances into the river than the

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present rule allows. On the Kanawha River our study shows five point four times more cancer-causing substances will be allowed. And on the Greenbrier River, if you pass this rule, seven point eight times more cancer-causing chemicals will be allowed.

Clearly, the new rule will put West Virginia on the cutting edge of increasing the cancer risk to humans. What you are proposing with this new rule is to take the state backwards in the fight against the risk of cancer. Instead of working to eliminate the risk of cancer, you are actually working to increase the risk of cancer.

Dischargers that are presently meeting the limits imposed by the old low water mark rule will be allowed to begin dumping, in some instances, almost eight times more cancer-causing agents into our rivers. No one knows for sure, in other instances it could be much higher than eight times. No one knows for sure because you haven't evaluated the rivers and streams of West Virginia to find out. New companies will be allowed to locate in our state and begin dumping more carcinogens than ever allowed since the beginning of regulatory time in West Virginia.

Our information is that you have not adequately investigated the impact the proposed rule change

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will have on the environment and human health. EPA Technical Support Document for Water Quality Based Toxics Control, March 1991 recommends that the harmonic mean flow be calculated directly from the historical daily flow records of affected rivers and streams. No such investigation has been undertaken by the Water Resources Board.

We recommend the Board undertake this investigation and calculate the increased risk of cancer implementation that the new rule will occasion. If after undertaking such an investigation you still insist on recommending this change, at least make a plain statement to the people of West Virginia that what you are proposing is to increase the risk of cancer in the state. Call it what it is, a rule to increase the risk of cancer. Don't try to sneak it through as a rule to implement harmonic mean design flow.

We will be submitting a written comment embodying the results of our studies with the aid of Carpenter Environmental Associates of Ramsey, New Jersey on or before June 28, 1991.

Thank you for the opportunity to speak.

DR. JENKINS: Thank you, Mr. Calwell.

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Most of us here on the Board are university types, and some of our leadership, like President Buckalew, has us work today. So we don't really know when these holidays are occurring.

Is David Flannery here, please?

MR. FLANNERY: Thank you, Mr. Chairman. Ladies and gentlemen of the Board, my name is David Flannery. I'm an attorney with the Charleston law firm of Robinson and McElwee. I appear here this evening on behalf of the West Virginia Chamber of Commerce and the West Virginia Manufacturers Association but will limit my comments to the dioxin standard instead of covering any other ground which might have been covered by Mr. Sherman in his remarks.

This, of course, is the first time which this Board has considered the adoption of a dioxin standard. It is an issue of first discussion but one we're pleased to be here tonight to comment on.

We join the Board in its obvious attempt to find a water quality standard for dioxin which is protective of human health and the environment. We're concerned, however, that the value which has been proposed by the Board is more restrictive than can be justified based upon an objective examination of the scientific data that's

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available.

Fortunately, we believe there is precedential value in the fact that a number of other states, including our neighboring states of Maryland and Virginia, have already been down this road and have adopted dioxin standards which have already been through the rigorous process of approval by EPA as scientifically defensible values protective of human health and the environment.

Realistically, we must recognize that West Virginia is in a competitive position with respect to a number of other states and that we cannot enjoy the luxury of constricting our opportunities to compete and to attract development by adopting regulatory requirements that are more restrictive than they need to be to protect the quality of our environment.

This very issue has been addressed by our Legislature in stating the following policy that I believe apparently guides you in your actions in adopting a water quality standard for dioxin, and let me quote this:

It is declared to be the public policy of the State of West Virginia to maintain reasonable standards of purity and quality of the water of the State consistent with (1) public health and public enjoyment

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thereof; (2) the propagation and protection of animal, bird, fish, aquatic and plant life; and (3) the expansion of employment opportunities, maintenance and expansion of agriculture and the provision of a permanent foundation for healthy industrial development.

This policy statement has been at the heart of West Virginia's water quality program for years. It was reaffirmed by our Legislature earlier this year with the adoption of the state's Groundwater Protection Act. It has stood the test of time. I submit it constitutes this Board's mandate, and simply stated, I believe it fairly says to this Board that our laws require you to set a standard which is consistent both with economic protection and with-- I'm sorry. With environmental protection and with economic development.

Now prior to discussing the scientific and policy issues that are involved in establishing a water quality standard for dioxin, it's appropriate to discuss the role of EPA and EPA's water quality criteria for dioxin.

It is well established that individual states such as West Virginia, rather than EPA, have the primary responsibility for the adoption and implementation of water quality standards. Consistent with that role and consistent

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with EPA's obligation to provide support for the states in discharging that role, EPA has published a number of criteria documents for various pollutants including dioxin.

Apparently EPA has criteria which this Board has relied upon for its proposal, but EPA's criterion document for dioxin represents a non-binding, non-regulatory guidance from which other states and EPA itself has deviated on many occasions.

Indeed, in a graphic which I've prepared and which is attached also to my statement, I have indicated the states in which EPA has approved dioxin limits. And on the one I've shown here, the ones in red are dioxin limits of 1.0 and greater in comparison with the .013 ppq standard which the Board has proposed. The ones in blue are states which have standards adopted at the 1.2 parts per quadrillion level.

I might mention that the New York standard of 1.0 has been approved by EPA and was approved as far back as 1985. I might also mention that the Maryland standard of 1.2 was approved in September of 1990 by EPA and that the Virginia standard was approved in February of this year. Both of those states are in Region III, the same region that will approve any standard adopted by West Virginia.

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I might also mention that the Georgia standard has been approved by EPA Region IV as recently as the 3rd of June of this year, just a few weeks back. And you can see the proximity of West Virginia to those states located as they are with standards at the level of 1.0 and 1.2 in comparison with the proposal which this Board is currently considering on, perhaps, two orders of magnitude less.

In March of this year, as Mr. Calwell indicated, EPA issued a technical support document that offers procedures for use by boards such as you in establishing water quality based controls for toxic pollutants. EPA acknowledged in that document that, and I quote, safety does not require the absence of all risk, end of quote.

The goal of this Board, I believe, fairly interpreted, is to arrive at a reasonable and acceptable level of risk utilizing scientifically defensible factors. EPA described the process this way, and I quote:

Any human criterion for a carcinogen is based on at least three interrelated considerations: potency, exposure, and risk characterization. States may make their own judgments on each of these factors within reasonable scientific bounds, but documentation to

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support their judgments must be clear and in the public record.

While EPA has it's own criterion for dioxin, it does not-- I'm sorry. I end that quote there. While EPA has it's own criterion for dioxin, it does not insist upon the adoption of that criterion by the states or by West Virginia. Rather, EPA recognizes the wide latitude which West Virginia has to adopt dioxin standards that are protective of public health and the environment. And, clearly, a level of 1.2 parts per quadrillion has met that test in both Virginia, Maryland, and elsewhere.

One of the most significant factors that will contribute to a determination of the value which the Board must come up with for dioxin is the actual human exposure to dioxin from the consumption of contaminated fish. But I submit to you that the real issue for the Board is to determine how much of the average fish consumption by West Virginians actually comes from West Virginia waters which might be impacted by the standards you would set.

Since West Virginia does not have estuarine waters and does not produce significant amounts of shell fish, it is reasonable to assign a significantly lower number to average fish consumption than the value that was

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assumed by EPA of 6.5 grams per day.

An additional factor that bears upon the appropriateness of a dioxin water quality standard is the risk level which is determined in order to make that computation. The Maryland, Virginia, and Georgia criterias of 1.2, in each of those cases criterias approved by EPA, were all based on the ten to the minus five risk factor. A ten to the minus five risk factor is well within the risk levels the EPA has stated that it will approved and has approved when it has reviewed state dioxin standards. That value is also consistent with risk levels used by many other states, and I won't read the list of those, they're in my written statement, as they have developed their water quality standards for dioxin.

It's common for the public to erroneously assume that all occupational environmental regulations should be based on a one in a million or ten to the minus six risk factor and that any risk above that level poses a serious threat to public health. But, in fact, risks at the level of ten to the minus six are negligible.

The current background for cancer in the United States is generally believed to be no less than one in four, meaning that in a population of one million people,

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two hundred and fifty thousand of those people can be expected to contract cancer in their lifetime. And that relates, I submit to you, to a statistical probability that indicates that that kind of risk is truly negligible.

To illustrate that, let me show you, again, a blowup of an exhibit which I have attached to my testimony showing on the top line the background risk of twenty-five percent for the cancer risk that the population normally sees. And on the second line I've added to that a ten to the minus six risk factor and on the third line a ten to the minus five risk factor. And I submit to you that it is immeasurable. One cannot tell the difference by any device which, other than by a simple arithmetic calculation, there is no way to perceive the difference in population.

I realize that the Board has historically utilized a risk factor of ten to the minus six. However, given the fact of the Kociba study, which EPA relied upon for its risk factor, has been extensively discredited, given the fact that many scientists question the assumptions, the assumption that dioxin causes cancer, together with EPA's widespread acceptance of ten to the minus six, makes it time for the Board to consider that.

Let me close by leaving the Board with three

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guidelines that I would offer for your consideration maybe to continue these deliberations.

One, is that EPA's criteria is not binding on the Board. Secondly, that in a range of values, 1.2 is a perfectly acceptable value for a dioxin standard. And third, given the fact that our neighbors have dioxin standards of 1.2 and we're forced to compete with them in a very competitive market, I believe our statute requires the Board to consider 1.2 as a standard.

I thank you for your time, sir.

DR. JENKINS: Thank you, Mr. Flannery.

Steven Schatzow.

MR. SCHATZOW: Good evening. I am testifying on behalf of the Apple Grove Pulp and Paper Company. I'm an environmental lawyer in private practice in Washington D.C. with the law firm of Morton, Lewis, and Botkis.

I spent ten years at the United States Environmental Protection Agency from 1976 through 1986. I was legal counsel to the EPA's water program from '76 to 1980. And I followed up as director of EPA's Office of Water Regulations from 1980 through 1984. As director of the Office of Water Regulations and Standards, I was responsible for EPA's water quality standard program and, in

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fact, for the 1983 EPA rulemaking which developed the federal water quality standard regulations which are still applicable today.

I want to make four major points. One is to support the development of an appropriate dioxin water quality standard which provides for protection of human health and the environment and yet which also allows for necessary economic and industrial development; second of all, to emphasize that under the federal statute and under the EPA regulations the development of state water quality standards is a program in which the states have primacy which calls for state decisions and for limited EPA review; and, third of all, to note that a dioxin standard of 1.2 ppq has been adopted by the neighboring states of Virginia and Maryland and has been approved by EPA; and, fourthly, to emphasize that the policy decision of setting an appropriate risk level for protection for a carcinogen is one where states have and should utilize great flexibility and where EPA itself has exercised very significant flexibility when EPA is making regulatory and enforcement decisions.

I am, as I said earlier, a lawyer and not a scientist, but it must be clear to all of us that there is no one right or wrong number for dioxin. The risk

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assessment process utilized by EPA and by other government agencies both here and abroad has led to estimates of dioxin risk which differ from agency to agency by three to four orders of magnitude.

All of these agencies are looking at the same animal tests, in this case a very limited number of animal tests, and they are attempting to extrapolate from these two-year rodent studies in which the rodents were fed very high doses of chemicals such as dioxin to determine what cancer risks may exist in human beings who are exposed to very small levels of dioxin over a seventy year lifetime.

This kind of extrapolation is not based on science. It is based on regulatory policy, and that regulatory policy varies dramatically from government agency to government agency, and that's why we have dioxin risk estimates that go orders of magnitude apart from each other.

I would note, however, that the EPA methods for analyzing chemical risks are perhaps the most conservative of those used by any government agency and result in the most stringent numbers.

I should also note that the EPA Administrator, Bill Reilly, has recently announced that the agency is reconsidering entirely its dioxin risk assessment based upon

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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some significant new data, significant new analysis, much of which suggests that dioxin may not be of as much concern as EPA previously estimated when people are exposed to very low levels of the chemical.

I mentioned the issue of state primacy. I think that's particularly important in the context of dioxin. I think it's already been stated that in its statute and in its regulations EPA makes clear that the states have the primary authority to determine appropriate water quality standards. Now EPA does publish, quote, recommended criteria but leaves it up to the states to consider those criteria, accept them in part or in whole, reject them and come up with their own criteria.

Of the thirty-eight states which have now developed dioxin criteria, and EPA, I think, at this point has approved approximately thirty of those criteria, of those thirty-eight states only sixteen states have adopted the dioxin criteria recommended by EPA. A total of twenty-two states have adopted criteria different than those recommended by EPA. And, again, I think that provides a sense of the controversies surrounding this chemical and a sense of the uncertainty surrounding the EPA risk assessment.

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And I should note that it's very unusual to have that many states going off on their own and adopting their own criteria rather than following the EPA criteria. If you look at most constituents that are regulated, most toxic constituents regulated by the states in the state water quality standards, the states overwhelmingly have accepted the EPA criteria recommendations.

It's more difficult, no doubt about it, for a state to adopt a standard that is different than the EPA recommendation, because it means the state has to make its own independent consideration of the science. And, yet, it seems to me clear that in the case of dioxin the majority of states which have looked at this issue have decided to take on the issue as independently assessed science.

The recent public discussions about dioxin suggest to me that EPA has an absolute commitment to a ten to the minus six risk level as the only acceptable risk level for a carcinogen. Nothing could be further from the truth. In fact, EPA's historical position regarding water quality criteria has been that the only absolutely risk free water quality criteria for any carcinogen is zero.

Since EPA policy, and, again, this is policy, not science, EPA policy assumes that some risk exists for

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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any exposure to a carcinogen, in first developing its recommended water quality criteria for carcinogens, including dioxin, back in 1980, EPA both recommended a zero level and then provided numerical values corresponding to risks of ten to the minus five, ten to the minus six and ten to the minus seven.

When EPA published this recommendation, it did not provide any preference for any particular level of carcinogenic risk. Most recently, of course, in its guidance to the states in these criteria EPA has suggested that a ten to the minus six level of protection is appropriate. It is clear, however, when one looks at EPA's own decision making for carcinogens, when one steps out of the arena of looking at what does EPA recommend that the states should do, and when EPA gets in the area of itself regulating carcinogens, then EPA, under a variety of statutes, most often regulates carcinogens at a level less stringent than ten to the minus six.

It seems clear that when EPA must consider the pragmatic implications of its decisions, as it must when it develops enforceable regulations and when it puts out specific regulations that will control a specific chemical as opposed to putting out guidance, the EPA does consider

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ALUM CREEK, WEST VIRGINIA 25003-9601

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and balance the economic implications of its decisions with the scientific information available.

We will be submitting written testimony before the end of the public comment period, and in that testimony we will give examples to demonstrate in terms of EPA decision making under the Safe Drinking Water Act where EPA promulgates regulations called maximum contaminant levels for a variety of carcinogens, that EPA has most often set those risk levels at ten to the minus four or ten to the minus five; that similarly when EPA is regulating carcinogens under the federal Insecticide, Fungicide and Rodenticide Act that the risk levels that EPA most often chooses are levels of ten to the minus four or ten to the minus five; that when EPA is regulating hazardous air pollutants under Section 112 of the Clean Air Act, such as benzene, radionuclides, arsenic and vinylchloride, EPA, once again, is selecting risk levels of ten to the minus four to ten to the minus five; and, finally, that when EPA is making judgments in the context of CERCLA or Superfund as to appropriate cleanup levels, that those cleanup levels are most often set at the range of ten to the minus four and ten to the minus five; that it is only in the context when EPA provides guidance that EPA is suggesting that ten to the

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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minus six level is the appropriate level.

And we would urge the state in this matter to look at both the science and to look at its own options in terms of these policy issues and to realize that most of the issues here are science and policy issues on which the state has that kind of flexibility, the state is encouraged by EPA to utilize that flexibility, and where EPA has clearly approved standards for states that have utilized that flexibility.

Thank you.

DR. JENKINS: Thank you, Mr. Schatzow.

I would-- If your points that you're wishing to make have been pretty well covered by someone else, I'd like to encourage you not to dwell on them too long. I just made a quick calculation. We're shooting at midnight here, so.

Russell Keenan.

MR. KEENAN: Mr. Chairman, I'll try to keep that in mind. Also, my voice is giving out. I came down with a cold. And I'll try to be brief here.

But, for the record, my name is Doctor Russell Keenan. I am Principal Toxicologist and Regional Manager of the ChemRisk Division of the McLaren/Hart Environmental

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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Science engineering firm. Tonight I'm here representing the Apple Grove Pulp and Paper Company, and I'd like to briefly address the three key issues that you, as a Board, must look at in terms of coming up with a scientifically valid water quality standard.

And those three variables are, of course, the acceptable daily intake for this chemical, the bioaccumulation potential of dioxin on aquatic organisms, and also the selection of an appropriate rate of fresh water fish consumption. And a number of speakers have already touched upon these.

I would first like to just address some of the recent scientific developments which have led to the conclusion that dioxin is much less toxic to humans than originally believed and that the low levels found in the environment are not harmful to humans.

And these developments come in two areas. They fall into two areas, the recent pathology review of the one bioassay that's been used to establish standards regarding dioxin throughout the world and new data on dioxin standards of action.

First of all, we can look at a decade ago and find that there was very little conclusive data regarding

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

the human health effects of dioxin. And for that reason, we had to rely upon animal bioassays to predict safe levels of exposure to this chemical.

Agencies relied on the long-term rat bioassay that Richard Kociba performed in 1978 and was reviewed in 1980 by U.S. EPA's consultant, Doctor Robert Squire of Johns Hopkins University. And over the last ten years we have various levels of acceptable intakes which have been established on the basis of that study. And I'll just pass this down to the Board so that you can refer to it.

Other speakers have already mentioned that there's over a one thousandfold range between the numbers which the governments of western Europe and Canada have been using and what EPA's water quality criteria developed in 1984 was based on. So I won't dwell any more on that.

I'd like to talk to you a little bit about the pathology review, and the original results of the Kociba study were based on criteria for evaluating rat liver lesions which were established in 1975. And since that time, a great deal has been learned about the biology of rat liver lesions and how they developed and how cancer might develop in laboratory animals.

As we have come to a greater understanding of

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

cancer biology in rodents, a more valid and scientifically appropriate classification system has been developed by the National Toxicology Program, in 1985, and it is this system which all regulatory agencies in the U.S. federal government are now using for evaluating rat lesions.

Now it was back in 1989 that based upon my recommendations to the chairman of the main science advisory panel that he requested that this Kociba study be re-evaluated according to now accepted and standard criteria of the NTP. The chairman basically wished to know whether the new classification system would change the cancer potency factor for dioxin, would it change the acceptable level of intake, and so that in 1990 Doctor Robert Squire, the same individual who did the EPA's evaluation in 1980, and other well-respected pathologists which are known as the Pathology Working Group, or PWG, re-evaluated these liver lesions. And the members of this group, following NTP practice, represented government, academia, and industry.

The group concluded, in brief, that there were approximately two-thirds fewer tumors in the animals than had been reported in 1978. And at the medium and low doses, in fact, there were no malignant tumors. In addition, the tumors were only seen at levels of extreme toxicity and not

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

at the low-dose levels where tumors would be expected if dioxin really were a potent carcinogen in rats.

It was the PWG's conclusion, in fact, that dioxin had only a weak carcinogenic effect in the livers of female rats. As you remember, male rats showed no tumors. The PWG's findings were in contrast to the EPA's earlier position that dioxin is a strong or potent carcinogen. In fact, Doctor Squire concluded, and I'll quote: Based upon all the biological evidence, I do not believe that dioxin poses a cancer risk to humans at any anticipated levels of exposure, end quote.

Now what Doctor Squire, of course, was talking about there were environmental levels of exposure. And recent developments in the field of molecular biology help to point as to why these statements are true and why this is so.

Recently in the scientific community we've come to a consensus, or a growing consensus, that all observed toxic effects associated with dioxin exposure are immediate to the induction of the Ah receptor, and induction of Cytochrome P450 is a marker of that type of induction or activation.

In other words, for dioxin to have an effect,

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

it must first reach a specific site, bind to a receptor, move into the nucleus and then bind to the genetic material. For dioxin to elicit any toxic response, you have to have a sufficient number of receptors which are occupied. And this level has to be exceeded before you have any even markers of exposure. And it's the induction of Cytochrome P450 which becomes the most sensitive marker of exposure.

Though not a toxic effect in and of itself, it lets us know whether there's been sufficient dioxin at the cell level to activate the Ah receptor system. So, therefore, at doses which are too low to cause induction of Cytochrome P450, no effects can occur.

Now based on this mechanism and the results of the numerous bioassays which have been done in studies of human populations, a safe dose level would lie reasonably between one and ten picograms per kilogram per day. And current environmental exposures are below this level, and it is based on this information and the pathology review that I talked about earlier as to why EPA is now undergoing the re-evaluation of dioxin potency and looking at the risk assessment all over again.

In the meantime, there are various methods to do a dioxin risk assessment. I have mentioned that the

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

computer model or linear model which EPA had used in the past and continues to use is scientifically invalid because it's not a non-threshold response here.

The scientific community is in general agreement with this and is developing a biologically-based model to replace that. But what can we do in the meantime? Well, the nations of western Europe and Canada, of course, have used the classic and toxicological safety factor approach. They take the known data from the Kociba study and apply a safety factor of one hundred and arrive at an allowable daily intake of ten picograms per kilogram per day.

Interestingly, we can look at what the World Health Organization has done recently. They have recently concluded that ten picograms per kilogram per day also represents a safe level of exposure for all potential health effects of dioxin including cancer, reproductive toxicity and any immune system effects.

Recently the Washington Department of Health recently proposed a range of allowable daily intakes of twenty to eighty picograms per kilogram per day using the receptor mediated model and pharmacokinetic considerations and binding, receptor binding in rats from the Kociba study

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

to come up with that daily guide.

Also recently the Florida Department of Health and Rehabilitative Services also concluded that dioxin has a threshold mechanism, and they calculated a safe dose of 12.4 picograms per kilogram per day.

So, in summary, the weight of evidence clearly supports the use of a threshold model for evaluating dioxin toxicity and for evaluating the human health hazard. To be conservative and health protective, an allowable daily intake of one picogram per kilogram per day would be consistent with a range of safe doses which has been developed by countries of western Europe and Canada and these agencies such as the Washington Department of Health, the Florida DHS, in addition to the World Health Organization.

In fact, the estimate of one picogram per kilogram per day would be at the conservative end of this range which these other bodies have found to be scientifically defensible and protective of human health for all toxic responses.

I just wanted to touch briefly upon the two exposure factors, the issue of how do you project what levels of dioxin you'll find in fish based upon

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

bioaccumulation throughout the aquatic environment and also a fish consumption rate.

Now, with bioaccumulation we find a range of multipliers which have been reported in the literature. And the reason for this variance is because the experimental methodologies have not been standardized, and also the definition of bioaccumulation has not been standardized. We find people reporting on various lipid contents. We find, we find also the same type of confusion in the literature in terms of what levels in fish, what levels in the aquatic environment are they referring to. Are they looking at water column concentrations, dissolved concentrations, suspended concentrations and all of that?

Using a consistent methodology to express bioaccumulation, we find that the world's literature results in a very narrow range of bioaccumulation values, all which hover around five thousand.

So, based upon this approach of standardizing the weight of the reporting mechanism, we would support the EPA's parameter in this case of five thousand as the most scientifically based multiplier for bioaccumulation.

On fish consumption, this is the last factor that needs to be considered, and the proposed West Virginia

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

standard uses the EPA fish consumption rate of six and a half grams per day, which, I believe, is health protective. You can look at the national survey which was done by Rupp (spelled phonetically) and his associates which found that about eighty-five percent of the U.S. population does not eat any fresh water fish on a regular basis.

The study also shows that the average resident of this region of the country eats only 1.1 grams per day, as low as that might sound.

Now, it's true that there are very little or very few comprehensive studies on fresh water fish consumption which have actually been conducted. There is a recent study which was conducted in the State of Maine and was commented upon by the New England Fisheries and Wildlife, which is our state department which views these issues, which said it was the best study which is around.

And in this particular case, the Maine study, we found a median consumption rate of only one gram per day for successful anglers on rivers and streams. So it shows that fish consumption rates for fresh water fish are very low and that the six and a half gram per day estimate of EPA is conservative and health protective.

Now, I'll just summarize, then, for the

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

purpose of developing the water quality standard for West Virginia, the allowable dose of one picogram per kilogram per day is conservative and protective of the public health. Based on this allowable dose of one picogram, a regulatory bioaccumulation multiplier of five thousand, and a fish consumption rate of six and a half grams per day, one could calculate a water quality standard of 2.1 parts per quadrillion.

If you were to say that West Virginia is similar to Maine and that one gram per day of fish consumption is health protective, then you could come up with a water quality standard as high as fourteen picograms, or fourteen parts per quadrillion.

However, I'd like to note again that EPA Region III has approved the neighboring state standards of Maryland and Virginia of 1.2 parts per quadrillion. Therefore, because of this precedence, it might be best well served by selecting the number of 1.2 parts per quadrillion as your standard.

I'd like to thank you for this opportunity.

DR. JENKINS: Thank you, Mr. Keenan.

I think there's folks around here that need a biological break. So let's take a ten minute break.

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(WHEREUPON, a break
was taken.)

DR. JENKINS: Steven Brown, please. Is Doctor Brown
here?

DR. BROWN: Mr. Chairman, Members of the Board, previous
speakers have stolen all my best lines, so I hope maybe to
make this a little shorter than I had first intended. I had
intended to make it pretty short to begin with anyway. I am
submitting written comments here for the record.

My name is Steven Brown. I am a principal
with Environ Corporation, which is a firm that consults in
environmental health risk assessment and associated issues.
I am here today speaking on behalf of Westvaco Corporation,
which operates bleached paper mills in three of the
adjoining states, namely, Maryland, Virginia, and Kentucky.

Because of my extensive written comments and
because of what's gone before, I'm going to try to just hit
on a few issues that I hope will explain my position to you,
and if I could just pass out these tables which summarize a
concluding table in my written remarks, I think it would
help take us through it.

Now, your job as a state water resources board

is to set a standard for, among other pollutants, dioxin, as several of our speakers have already addressed. And the key thing, I think, in doing that for you is to start with what model of carcinogenesis that you are going to adopt to lead you to that number. And I say carcinogenesis deliberately. Most of the speakers here have talked principally about cancer because that's been the principal issue that most people have talked about with respect to dioxins.

However, we have looked at other potential toxic effects of dioxin, reproductive effects, developmental effects, effects on the immune system, and effects in aquatic organisms, which hasn't been mentioned, I think, tonight.

In every case, as long as you are reasonably protective against cancer, you're going to be protective against those other effects as well. So I wanted to get that on the table before going back, like my colleagues before me, to the cancer issue.

But the key factor, when you're looking at cancer development from dioxin, is to decide whether you're going to use a threshold model of carcinogenesis or a non-threshold model. Are we going to assume that any level of dioxin, no matter how small, raises the risk of cancer to

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

some degree or whether there is some level below which the risk of cancer is essentially zero, no risk at all, and that's the threshold model.

Now, EPA, back in 1984, which is the lefthand column on this chart, used the non-threshold model and, I think, all the best science that was available in 1984 to come up with a water quality criterion among two others that was at the level of .013 parts per quadrillion. They were not, I think, in any way mistreating the science back then. But there have been seven years since they visited that issue. And I think things have changed substantially.

This table, and I'm sorry that I don't have this to project for the audience. I'm assuming that you can get this from the record later if you want it. This table shows the various factors that go into computing a quantitative water quality standard for dioxin. And in addition to some rather agreed upon values like body weight of an average person, the amount of water an average person needs per day, there are such factors as fish consumption of an individual; a number that I call the fish to water ratio, which is what some other speakers have referred to as bioaccumulation or bioconcentration; the slope factor, which is the number that shows how much extra cancer in the non-

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

threshold model goes with a given dose of dioxin, a given daily intake of dioxin; and then the risk criterion, which, I believe, Mr. Schatzow talked to you in great detail.

Those two can be combined to give a criterion dose, which is the daily dose below which risk is inconsequential or zero. And in the case of the threshold model, it is zero below the criterion dose. Below the criterion dose for the non-threshold model, there is supposedly some additional risk, but it becomes inconsequential in a regulatory context. And you can put all those numbers together and come up with a standard in parts per quadrillion.

Now, one thing that wasn't said was how those standards of 1.2 in the neighboring states of Maryland and Virginia were derived. Those standards begun development at least two years ago, and at that time the validity of the threshold model was still somewhat in question. And so they adopted a linear no threshold model that says risk increases with dose even at low levels. And they used all of the standard EPA values with the exception of two. One was the slope factor, and one was the risk criterion.

For the slope factor, they adopted a number from the Food and Drug Administration that was developed

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

from the same bioassay by Richard Kociba as the EPA used, and the principal difference between that number and the EPA number was the question of how one extrapolates from rat to human. And they used a body weight extrapolation factor instead of a surface area extrapolation factor. I'm not going to go into detail on that now. The difference amounts to a factor of 5.4. Some other factors were ruled in. And the FDA concluded that the slope factor was approximately nine times less than the EPA concluded.

And, so, instead of a slope factor of a hundred and fifty-six thousand, the FDA used a slope factor of seventeen thousand five hundred. The other number that changed was the risk criterion. EPA was recommending one in a million, and the states of Virginia and Maryland used a factor of one in a hundred thousand. That's the ten to the minus fifth that Mr. Schatzow was talking about. I might mention the neighboring state of Ohio apparently also uses one in a hundred thousand as its risk factor.

And to support what Mr. Schatzow said, the Environmental Protection Agency, in its maximum contaminant level proposed for dioxin about a year ago came up with a risk level that was approximately two and a half in ten thousand, two point four times ten to the minus fourth,

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

which resulted in a drinking water quality standard of fifty parts per quadrillion.

So, now, what I'm showing you here are what I think is the best science now, 1991, not the best science in 1984. The first column is the non-threshold model. I'm not going to go into details you've already heard. I use a fish consumption rate of one, a fish to water ratio or bioaccumulation factor of five thousand. My slope factor is now fourteen thousand four hundred, which is close to that FDA value. I arrive at this in a way that is explained in my written comments. The principal difference is the adjustment from the Pathology Working Group re-assessment of the Kociba data that, I believe, Russ Keenan alluded to.

If you put all those factors in with a one in a hundred thousand risk factor, the standard that you come up with is seven parts per quadrillion. However, I would like to emphasize the threshold model. I truly believe that the best science available now says the threshold model has been approved to be the correct one. We can't look into a crystal ball, but I think so.

Linda Burnbaum (spelled phonetically), one of the EPA scientists who was at the Dioxin Consensus Conference in Banbury, recommended to the administrators

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

that the threshold model was the best available science. And she concurred in the number that Doctor Keenan quoted to you from one to ten picograms per kilogram per day dose as fully protective of human health. And those numbers translate, with all the other assumptions that we've talked about, to ten to one hundred parts per quadrillion in water.

Now, the decision you have to make is all these numbers and all these choices. And one thing I want to point out to you is the one really vexing question, which is a policy question and not a scientific one, is the choice of the risk criterion, one in a hundred thousand versus one in a million, because that is an emotion issue. There's no scientific right way to do it. It's a policy choice.

Once you see the threshold model is probably the one that's going to prevail, you don't have to deal with that. You've got a number there that says below a certain level of exposure the risk of dioxin is essentially zero. And so you can go with that without going around and around on what the proper risk level is.

So I'm going to stop with that, and thank you very much for your attention.

DR. JENKINS: Thank you, Doctor Brown.

Is Vince Rackard here?

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

MR. PACKARD: Packard? P-a-c-k-a-r-d?

DR. JENKINS: Maybe so. I have a little trouble once in a while.

MR. PACKARD: I just-- I'm a concerned citizen, and I just wanted to say that I think we've compromised enough on our river systems. I think it's deplorable that I can't fish as much as I want in the Ohio River. I like to supplement my diet with fish or wild food, especially with food prices going up as high as they are, and because of the PCB levels and chlordane levels, ORSANCO has warned us against eating fish out of the Ohio River. In fact, certain times of the year you can't even dip your feet in the Ohio River. That's mostly the bacteria levels.

From what I understand about dioxin, dioxin has been known as one of the three most toxic chemicals known to humans. And the EPA says that it's one million times more toxic than arsenic and cadmium.

We've lost a lot of life in the past out of the Ohio River, including the river sturgeon, blue pike, hairlipped sucker, alligator gar and many others. And a lot of the other life is threatened in the Ohio River, including the paddle fish and many of the mussels in the Ohio River. The Ohio River used to be known for its conglomerate of

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

mussels, and now you find mostly weed species.

In case you haven't noticed, we're witnessing an ecological problem on our planet, and this is not the time to be allowing more pollutants in a river that's, that's famous for being polluted. You know, people think of the Ohio River, and they think of an industrial sewage system. They don't think of complete ecological systems. The river is a part of our basics of life along with air and land. And I just don't think we should be comprising it.

Thank you.

DR. JENKINS: Thank you, Mr. Packard.

Donnie Gillespie. Mr. Gillespie.

MR. GILLESPIE: Ladies and gentleman of the Board, my name is Donnie Gillespie, and I'm from Apple Grove. And what I have here is two newspapers that I want to give the Board members to go over. One is from the Alabama Journal. It's entitled, "Alabama's Rivers, An Endangered Resource." And the other one is from Tallahassee, Florida, the Tallahassee Democrat, and it's got an article in here about Fenholloway River in, near Tallahassee, Florida. I'd like for you people to read these papers over and look at the rivers in these states. And I would personally urge the Board to adopt the lower standards because I don't want to

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

see the rivers of our state looking like these rivers here. And if you need additional copies of these papers, get in touch with me, and I'll try to get more to you. But I think they should be looked at real close before you make a decision.

DR. JENKINS: Thank you.

Brian, is it Hagenbuch? Hagenboch?

MR. HAGENBOCH: Congratulations. You're a lot closer than a lot of folks.

Thank you. My name is Brian Hagenbock, Project Director of the Ohio Valley Environmental Coalition. I'd like to take the opportunity to comment on these standards tonight.

I guess what I'd like to do is-- I have a scientific background. I got my start in science playing with aquatic organisms when I was a little kid. And so rather than read to you a script, I think I'm going to give you a little demonstration on how OVEC feels, our position on these standards.

Now, what I've done is I brought-- Now, there was a lot of industry assumptions tonight, okay. So we're going to make some assumptions here. We're going to assume that the goldfish is a channel catfish, okay, which is a

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

popular fish that is consumed by fisherman in the state. Okay. Now we're also going to assume that this beautiful, clear water up here is Ohio River water. Well, that's a big assumption, but we'll make that assumption, too. Okay. I got one more in here, one more goldfish. They're kind of mad at me. I told them when I bought them that I'd put them on TV. They seem to have left.

But, so I've got three samples up here. Ohio River water and, we're going to call them, channel catfish. Now channel catfish are bottom feeders, okay. Now in West Virginia there are currently seven rivers which public health advisories have been placed on the fish, okay. Now in the Kanawha River, the Pocatalico River, Armour Creek, and thanks to Westvaco, the Potomac and the North Branch of the Potomac, the public health advisories are due to dioxin levels.

In the Ohio River we have public health advisories due to chlordane and due to PCBs. Now in dioxin testing that was done on the fish in the Ohio River, they found levels in Huntington at 9.2 parts per trillion. The EPA level for public health advisories for women, infants, and children is twenty-five parts per trillion.

Now we're going to do a little study here

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

because I like to play biologist every now and then. We're going to assume that this here, fat jar here (indicating) is OVEC's proposal. OVEC is proposing zero discharge on dioxins. So I'm going to leave this fish alone.

Now in the middle one here (indicating), I'm going to take the EPA standard, 0.013 parts per quadrillion, okay, and we're going to say this is the EPA proposal.

And over here (indicating), we're going to take industry's hundredfold increase, okay, on their proposed dioxin standard. Now day one comes along. Industry has their proposal. And we're going to assume here that one drop of this dye, which I'm getting all over me, is .013 parts per quadrillion. So what I'm going to do is I'm just going to put one drop in here. So now we've got one part per quadrillion in the Ohio River. It gets a little blue. It will settle out.

But in this one here (indicating), now industry wants a hundredfold increase, and I'm not going to count a hundred dots, or a hundred ink things in here, but, oh, let's just go with that (indicating). Okay. So that's time period one. Oops, what happened to our catfish? Okay. Now I could keep going here. OVEC (indicating), EPA (indicating), industry (indicating). Huh.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

A VOICE: That's only twenty, Brian.

MR. HAGENBOCH: Twenty? well, I don't have all day. I want to let some of my friends come up here and talk. I think what you see here is, you get the point. If I lived in Huntington, and I do, or I lived in Apple Grove, or I lived in Charleston, where dioxins, with public health advisories in the rivers, I'd be very concerned about these proposed standards tonight.

What I'll do is abbreviate some of what else that I brought. I have extensive comments, which I have given. I hope that you will take them under serious consideration because I argue a lot of what you've already heard tonight.

I want to talk about health effects. There is a considerable debate over this. Dioxins are the most potent animal carcinogen ever tested. They're a potent teratogen, too. They're a potent chemical producing toxic effects on the human immune and reproductive systems. Some studies which have just come out reveal a connection between dioxin exposure and non-cancer health effects, heart attack, cirrhosis of the liver, gallbladder, biliary tract diseases, others.

Some studies have shown dioxins are more toxic

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

and have a higher bioaccumulative potential than DDT or PCBs. You remember those. Recent studies of German-American workers found increases in cancer rates. In a study of five thousand chemical workers, Fingerhut found nine times the normal amount on soft tissue sarcomas. They also found forty-two percent more cancers of the respiratory tract. The death rate for workers was two to the thousandth, which was well above the EPA acceptable levels.

Now the authors do admit that this was a long-term study and they were chemical plant workers and the potential is there that other chemicals could have maybe synergistically worked on the development of these cancers. But it was quoted by one person in the New England Journal of Medicine that the evidence must be taken seriously as a flag of possible human risk.

Now, scientists haven't determined whether dioxins are initiators and promoters. Remember, dioxins are already found in the channel catfish, in the sediments, in the sediment core samples that we've been taking. Okay. You have to remember, too, everybody in this room has dioxins in their system. Seventy-five percent of the people in this room have TCDD, the worst dioxin, in their tissue system right now.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Okay. So when we start to talk about promoters or initiators, we've already got a buildup of dioxins in our system, okay, and the question is, where is that level? Dioxins have also been found to work synergistically with some chemicals like PCBs. A low level, low risk PCB combined with this dioxin has increased the carcinogeneses of the dioxins. And I have referenced everything that I've put in my comments. I'm not speaking off the cuff up here.

People are already exposed to multiple initiator and promotor chemicals in the environment. And when we're looking at initiators or promoters, what you really want to look at in an initiator is the highest single day consumption of fish, because that's going to initiate or elicit the response. And there's been some comments about the amount of fish people consume. And I think in the Chamber of Commerce comments that I've read they looked at a study from 1973. Well, I took a look at the 1985 fishing and hunting catalog that got put out, and it said that over four hundred thousand people fished in West Virginia in 1985. Over three hundred and seventy thousand were residents. Eighty percent of those fished in the rivers and streams of the state. Ninety percent of those were fishing

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

for channel catfish.

Now the most probable way of-- Well, let's hold off on the dioxin levels of fish. There's been some argument. Industry has said that previous studies found no increase in cancer rates among chemical workers. The Monsanto study, a study over in Germany. These studies have been found faulty and possibly fraudulent.

Let's go on. The new information that we've been talking about from that 1978 rat experiment which formed the basis for EPA's current estimate, when they went in and redid the evaluation on the tumors, they took-- What ChemRisk did and several other industries did was they took many other things besides that one under consideration. They assumed too much. Anytime you make an assumption in a scientific study, you have to be able to validate it. ChemRisk could not validate many of the assumptions that placed upon it.

If you looked at, if you recounted the number of tumors, okay, that were recounted and changed from the original study to 1990, you only see that the estimates on cancer were increased two to three times. Not ten, not a hundred times, not a thousand, only two to three times. Industry has admitted as much. Again, I reference that.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

This is an insignificant change given the uncertainty of risk assessment.

Somebody mentioned the Banbury Conference at Cold Spring Harbor, how it was such a great dioxin consensus conference. Well, that's bull. The keynote speaker of the conference said by, in no means was it a consensus conference with respect to regulatory conclusions related to the risk assessment of dioxins. So that conference should not be used as developing a consensus on a dioxin standard.

Let's talk about fish, the EPA and the FDA level. Well, the EPA has said that the FDA level is not an appropriate basis for a water quality criterion. And I talked about fishing in West Virginia, four hundred thousand individuals. It's a hundred and twenty-eight million dollar industry. Current EPA water quality standards for dioxins in fish bases it on a consumption rate of six and a half grams per day and a bioaccumulation factor of five thousand.

New data, new studies have found that sport fishermen can consume about thirty grams per day. That's about a half a pound a week. And subsistence fishermen consume an average of about a hundred and forty grams per day, five to twenty-five times higher than the EPA levels. Further studies on the bioconcentration factor have found

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

coalition.

We're very concerned about the toxins in and around the Ohio River. We are also extremely concerned about the dioxin discharges that will follow from the pulp mill that's being proposed. And we're very opposed to raising the dioxin discharge standards to levels a hundred times higher than the EPA standards such as has been requested by the Alabama River Pulp Company.

Considering the pollution already in the Ohio River, zero discharge makes perfect sense. In any case, it would be blatantly irresponsible to raise the level of dioxin discharge standards any higher than the .013 parts per quadrillion level set by the EPA.

Dioxin is one of the most toxic substances known. As was mentioned before, the EPA says dioxin in food is a million times more toxic than cadmium or arsenic, and that's not even counting the cancer hazard from dioxins.

Some people have been cited in studies that play down the risk of dioxin. It's pretty easy to see through that. As mentioned also, there was a Monsanto study in which an EPA scientist said Monsanto clearly falsified the data. There was a BASF study. West German scientists reported that BASF disguised the cancer cases in the study.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Raising the dioxin standard is clearly not warranted, and it could lead to lawsuits such as have been filed in Virginia and other states, or Maryland, excuse me, and other states.

The Ohio River is already so polluted that it's not safe to swim or fish in the river. And it's clear that we must reverse trend of pollution and not increase it. So we at the Student Environmental Action Coalition urge you to not cave in and raise these levels but to let the minimum hold to the EPA suggested level. This is a matter of life and death.

Thank you.

DR. JENKINS: Thank you, Mr. Bennett.

Connie Lewis, please.

MS. LEWIS: My name is Connie Grato Lewis. I represent the League of Women Voters of West Virginia. We wish to comment on the proposed standard for 2,3,7,8-TCDD.

There are various points of view concerning the appropriate standard for TCDD. The League believes that in the absence of conclusive data to prove otherwise EPA's criterion is the most defensible. The threshold model is not conclusive. I wish to remind you again that several participants in the Banbury Conference did not agree that

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

dioxin was less dangerous than many of the scientists claim.

The League also believes that any company which discharges dioxin should be required to monitor, in addition to the discharge waters, the receiving sediments and suspended particulates and conduct biomonitoring. In order to insure quality assurance and quality control, the laboratories used to analyze the samples should be EPA certified for low level dioxin testing, and the program should require frequent testing.

Because dioxins are persistent in the environment and have already been found in a tributary of the Kanawha River, we believe they should be strictly controlled.

And I would like to remind you that the water is not the only way we're exposed to dioxin. Bioaccumulation of dioxins are already a problem in the Kanawha itself. Dioxins are also present in sludge from coal bleaching and from waste water treatment and pulp mills. By landfilling or spreading them on the land, they could then pose long-term threats to the environment.

It would be more provident to prevent future problems than to try to remediate them. By setting EPA's standard now, a standard in use in the neighboring states of

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Kentucky and Pennsylvania, a company can plan its facility with processes appropriate for meeting the standard rather than installing expensive equipment later if the standard should be made tighter.

Pulp mills are currently using manufacturing processes that meeting the EPA standard. We believe that pulp mills in West Virginia should use contemporary processes to meet the environmental concerns and protect the public health.

Thank you.

DR. JENKINS: Thank you, Ms. Lewis.

Christopher, is it Chmiel?

MR. CHMIEL: My name is Christopher Chmiel. I go to Ohio University, and I'm a member of SEAC also. I was wondering if you've ever seen the TV show where they try to bribe people till they do things like swallow goldfish or put pantyhose on their head or whatever.

Well, it's like I'm beginning to hear these professionals come up here tonight, and I'm wondering how much it cost, you know, to get you people to discredit your professional so much and to go back on these goals of protecting human life.

Dioxin, from what I understand, is one of

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

the-- You've heard it all. Someone just sent me something today about paper and about the process of making paper and dioxin being the most potent animal carcinogen known. I'm an animal.

But what I want to ask, if you ever thought about the loss of trees and if the loss of trees will affect the ability for nature to purify the water and stuff. You're the Water Board. I'm just wondering if you've thought about that. And water being a very basic need, when I hear talk about reasonable standards and acceptable levels of risk, I hear short-term gratification over long-term sustainability.

And being a young person, twenty-six percent of the population of the United States is under the age of eighteen. If you have children and stuff, you might be able to empathize with this. Sustainability is the ability for society to supply its basic needs and health without jeopardizing the future generations.

And how long are we going to wait for people trashing the place? I mean, it's piling up on the younger generations. You're university people, and you work with young people. And you talk about-- I think a lot of it's jobs. No one wants to buy paper made at a virgin pulp mill

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

anymore.

Ohio University has got a recycling program, and we're switching over to buying all recycled paper. We want minimum impact paper. We want paper that doesn't have bleach using dioxins. We know there are alternatives.

And I think enough people came up here and told you a good truth of it all. And so I just hope that you guys-- You have a choice.

Thank you.

DR. JENKINS: Thank you, Mr. Chmiel.

Keith Taylor, please.

MR. TAYLOR: Good evening. I won't take much of your time. I only got two pages, but I think I got a lot to say. I'm Keith Taylor. I'm State President of the Isaak Walton League in West Virginia. And since 1922 the Isaak Walton League of America has fought for clean water. In fact, the League made the nation's first water pollution survey back in 1927 for President Coolidge.

Now when the League was founded by fifty-four sportsmen and conservationists in 1922 in Chicago, they chose the name to name it, Isaak Walton, who history notes to be one of the first conservationists in England in the fifteen-hundreds.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Now it seems ironic that Isaak Walton of jolly old England is here to take a strong positive position with Parsons and Whittemore of jolly old England. They're the owners of the Alabama Pulp and Paper Company.

Now Alabama Pulp and Paper proposes to build a plant on thirteen hundred acres in Mason County about halfway between Point Pleasant and Huntington. If built, it would be one of the largest paper mills in the nation.

Now we all know that we need industry in West Virginia, and we want to welcome each and every one provided our environmental laws set down by our state and federal governments are adhered to. We seek and want good citizens to do business in our state, those that will protect our environment.

One of the League's main concerns here is the dioxin discharge that will be made into the Ohio River which may cause cancer in human beings. Another concern is the odorous smell from the exhaust stacks of paper plants that will unquestionably be released by this operation. We oppose anything that fowls fresh air.

Now, back in June 1989 Luigi Terizotta, that's T-e-r-i-z-o-t-t-a, the general manager of the Alabama Pulp and Paper Company, which is owned by Parsons and Whittemore,

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

says that this mill will use the latest technology from Scandinavia and the water discharged will not emit dioxin detectible at one part per quadrillion. That's a millionth of a billionth.

He stated this mill will operate, and I quote, with no detectible discharge of dioxin if built in Mason County. Now this is the plant manager here speaking of the Alabama Pump and Paper Company, Alabama.

Now, as far as the odor goes, Mr. Terizotta, I think says, and I quote, we don't say no odor, we say little odor, unquote. Old mills use a sulphur compound to cook wood. This new mill will not use that process or have that smell, so states Mr. Terizotta.

Now, in May 1991, about two years later, at a Charleston meeting, Alabama Pulp officials proposed raising the standard for dioxin emission by a factor of a hundred, or the above-suggested federal guidelines, if you will.

Now, really, ladies and gentlemen, if we are to believe the plant manager, Mr. Terizotta, that dioxin will be virtually non-detectible, why would Alabama Pulp officials try to get West Virginia to raise the dioxin emission standard by a factor of a hundred? Now who's kidding who here?

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25903-9601

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If standards are lower, could more profits be made? I took a look at these people here tonight who are paid a high price to come here and try to get these standards lowered. There's profit involved.

Now does West Virginia look like a soft touch by a new industry? And lower standards are sought as a prerequisite to obtaining this facility. Something is amiss, too, when on June the 7th, 1991 the Water Resources Section of our Department of Natural Resources was asked to submit the application it filed for a federal permit necessary to discharge plant water into the Ohio River.

Was this done to bring more pressure on our Department of Natural Resources, or has Alabama Pulp and Paper decided to be a good citizen, as Mr. Luigi Terizotta said they could be regarding dioxin discharge?

The Isaak Walton League will support safe and environmentally sound industries to come to West Virginia. We don't need or want any industry that operates through loopholes. We will support an industry that is honest and abides by both the state and federal environmental laws. The time has come in the United States of America that no industry in any state will operate outside the laws laid down by the state and federal government.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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Ladies and gentlemen, that is what we ask in this paper mill case, change no regulations and ask that they abide by all set regulations of water and air discharge presently enforced.

Thank you.

DR. JENKINS: Thank you, Mr. Taylor.

Bill Ragette.

MR. RAGETTE: My name is Bill Ragette. I'm here as a represent-- As Vice-President of Homeplace, which is a citizens' group in the western counties of Lincoln, Mason, Putnam County who are concerned to protect the quality of their homes and their lives.

I'm speaking mostly on the dioxin issue, but I'm also concerned about the measuring of the harmonic mean flow that was mentioned. That's really scary to me. But I think Stuart Calwell took care of the question. But I'm also concerned about that, that you do not lower that standard and we stay with the minimum flow, the ten year minimum flow. I sort of thought we were all along going to go with that.

Most of the stuff I was meaning to say has been covered, so I'll just try to give you some new stuff. There's been talk about whether dioxin is a carcinogen or

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

whether it's a promoter of a carcinogen, and there was talk about the Banbury Conference at Cold Spring Harbor Laboratories.

My understanding of the situation was that there was a general consensus that dioxin was appearing not to be a carcinogen as such but to be a promoter. And then industry-- This conference was supported by the EPA and by the chlorine industry, the Chlorine Institute. Most of the participants in this conference did not even know that the Chlorine Institute was sponsoring it.

After its conclusion, a spokesman for the Chlorine Institute said the scientists had agreed on a model for the toxicity of dioxin that allows for the presence of the substance in the environment with no risk experienced below a certain level of exposure. This is the threshold and non-threshold idea.

I think they're confusing the idea of being a promotor of cancer or being a mediator or being a carcinogen itself, and I think those necessarily go together. And a lot of people that attended the conference were upset with this conclusion because they said because, even though dioxin may not be a carcinogen as such, it may only be a promotor, that that's not saying that there is a level below

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

which dioxins will not cause cancer, will not promote cancer.

Bioaccumulation. I have a paragraph on that where the Environmental Research Laboratory in Duluth, Minnesota this year, and you have a submission-- Everything I'm talking about tonight is in your six-inch stack of material that you all got. And I read through the whole thing. But this is coming out. This is a 1991 release. And it said that bioaccumulation has been found from fifty thousand to a hundred and forty thousand.

So this factor alone would make us want to have the level of dioxin in our waters one tenth of what the EPA has requested, you know, be ten times lower.

Reading through this whole thing, you've got about five or six inches of what industry has submitted, and about, maybe half an inch of what environmentalists have submitted. And I get totally disgusted. What industry does over and over again is it tries to attack from every single angle how much, you know, to raise this level so they can make more money.

And what I did was I did two things, water consumption and fish consumption, and analyzed what they were trying to do. Fish consumption is a good place to give

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

you an idea of industry's shameless attitude. In trying to justify an increased level of dioxin in our nation's water, the reports submitted by the West Virginia Manufacturers and various other industrial lobbies attack from every angle the idea that people get much dioxin from fish. Most people assume that the main manner by which we absorb dioxin is through fish consumption, although a Canadian study, this is the Birmingham 1990 study, found that only about one-half of our dioxin in our bodies comes from fish.

And also, somebody also mentioned that, that the only place you're not going to get dioxin from is not from the water. It also comes the from the air and other places. So allowing the maximum amount of dioxin in the water is not a good idea because you're getting it from other substances, other places, too.

The EPA says that the average citizen eats 6.5 grams of fish a day, or seven ounces a month. When you consider that a hamburger that you buy at a fast food joint is a quarter pound, if you eat two or three of those, you've already got, from one meal, your protein is three-quarters of pound. And they're saying people eat, what is that, one fifth, or one-fourth of a gram per day as an average.

This does not take care of the people, I think

Brian mentioned, that eat many times that. Somebody mentioned the study that was done in Maine on the Maine anglers and how they eat one or two grams a day, but they gave a chart, and it showed the French-Canadians and the Indian native population were eating three to six ounces a day. So this is like a thirtyfold or fortyfold increase.

In order to protect certain subpopulations, I think we can't go the six point. To protect me, because I don't eat any fish at all, but some people eat a lot of fish. You have to protect them, too. Okay. I talked about the report.

This is what got me mad. Industry says things like this. This is from the reports they submitted to you. Only twenty-five percent of the six and a half grams of average daily consumption of fish is from dioxin laden streams. They say you don't get it often. You get it from some streams that don't have dioxin.

Suppose someone's fish all comes from the same stream or that finally all streams are degraded to the industrial level. They say that sediments are a sink for dioxin, therefore, lowering the concentrations in fish, but that's not considering the concentration of the bottom feeders. And they say that if fish eaters don't any of the

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

fat of the fish-- I'm surprised industry would bring this up because like they spent thirty or forty pages in the reports they submitted to you on this one issue, because dioxin is, of course, concentrated in the fat tissue of the fish. And they say then dioxin concentrations can increase a hundredfold more if you take the fat away, but that no longer allows me to fry my fish up and, you know, to soak up the fat.

They say I can't eat-- They're just like telling me where I can get my fish, and how I can eat my fish, how I can cook my fish. So, therefore, they can have a thousandfold increase in their dioxin.

All this juggling by industry so they can pollute and make a biggest profit cuts down on the options we have on how much fish we eat, where we can fish, and how we can cook our fish.

I feel a great revulsion at all the fact manipulation and verbal footwork these corporations and their hired guns are doing, none of which is sound science, to further degrade our waters.

I'll try to be brief. They also do this with water consumption. The army and the EPA say that we drink two liters of water a day. I know on hot days in the summer

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

I'll drink at least a gallon or four liters. One consultant for industry spent a page and a half, a typed page, single-spaced, trying to put forth the argument that a drinking water level should be at 1.2 liters.

So because of their greed and disregard for the environment, many people will have to cut back on their water consumption or buy some product like bottled spring water or soda from them.

They mentioned something about aquatic life. EPA said there's insufficient data to determine the safe level for aquatic life. Until we know, we should err on the safe side. The wild critters are already under heavy assault by us humans.

I'll read this next one real quick. It hasn't been mentioned tonight. Mother's milk and the effects on fetal development. The EPA has found dioxins in the bodies of everyone tested. They found that mother's milk has eleven thousand times the acceptable level. Babies nursing off mothers whose principal protein is fresh water fish could potentially be getting thousands of times this level.

Ellen Silbergeld, a staff scientist with the Environmental Defense Fund, states that dioxin alters hormone levels which then disrupt development of the immune

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

system and interferes with fetal and child development. Studies with rhesus monkeys exposed to dioxin prenatally and through mother's milk later had behavioral and mental difficulties.

Considering its bioaccumulation potential, its persistence in the environment and in the body, and for the sake of the children, we should set a zero discharge level for dioxin.

There are over a hundred dioxins and furans, although none may be as carcinogenic as TCDD, they are all very poisonous and have other health effect. We should also set standards for all these and consider their synergistic effects. Suppose TCDD is only a mediator, but in combination with some furan that is an initiator it could have very powerful effects.

Like I say, you can't consider this one dioxin alone. The pulp mill everybody is talking about will be spewing out all the different dioxins, or a lot of different dioxins.

Subpopulations consisting of breast-fed babies, native Americans and others that depend on fish for survival, developing children and fetuses, chemically sensitive people need special protection, and the level of

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

dioxin allowed in the water needs to be low enough so that these people are not endangered.

West Virginia, being a rural state, has many people who fish in our waters and eat the fish on a regular basis. The EPA, in a memorandum of January 5, 1990, that you all have, has allowed the states to set more stringent levels if certain people in the state eat more than seven ounces of fish a month. I suggest that the Water Resources Board does this.

You can read the rest of it. The last thing that got me upset was this idea of acceptable risk. And I'm not a mathematician, but I just don't want to be judged on figures on the graph we saw before where we took the acceptable risk of one in ten thousand, one in a hundred thousand, one in a million, and one in ten million and they compare it against the backdrop cancer rate. You saw the boxes. They weren't any size at all. But to me, that's manipulation of numbers.

It's very simple to see that if you're counting a cancer rate of one in a hundred thousand, one in a million and one in ten million, by setting it at one in a hundred thousand rather than one in a million or one in ten million, as some people suggested, you're increasing the

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALLEN CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

number of cancers in the population by ten or a hundred, in fact, by a hundredfold.

And it's not exactly the same. They're just increasing by a number like it. I still think somebody is manipulating numbers to make it look like that's not what happening. And I think that we should go with the one in a million or one in ten million risk factor.

That's all I have. I'd submit this
(indicating).

DR. JENKINS: Thank you, Mr. Ragette.

Missy Woolverton, please.

MS. WOOLVERTON: My name is Missy Woolverton, and I work with the West Virginia Citizens Action Group, and I'm here to urge you, the Water Resources Board, to adopt a dioxin standard at least as stringent as .013 ppq that you're currently considering.

I'll skip a lot of this, as many of the things I was going to say about dioxin have already been said. One thing I'd like to say is for those of us who work and lobby on behalf of the environment in Charleston, we know that everything is political, and you, the Board, you just can't operate in a vacuum.

While there are those of us tonight who

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

registered health concerns about dioxin, there is another contingent who, for economic reasons, wants to see the proposed dioxin standard relaxed. Apple Grove Pulp Company is one of them. This company wants to build one of the largest pulp and paper mills in North America at Apple Grove, Mason County, West Virginia.

They have asked the Division of Water Resources to initiate a dioxin standard, and you've heard it many times tonight, of 1.2 ppq, a standard a hundred times weaker than the .013 that you're currently considering and that the EPA institute it if the state doesn't set its own standard.

Should Apple Grove come to West Virginia, they'll be releasing dioxin from their effluent into the already heavily polluted Ohio River. This dioxin is a by-product of the chlorine bleaching used in the pulp and paper making process.

It's important to note that it is possible, it is possible to produce paper without using chlorine. If Apple Grove is coming to our state, they should be required to be chlorine-free.

The weaker 1.2 ppq dioxin standard is also being supported by such groups as the West Virginia

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Manufacturers Association, the Farm Bureau, the Chamber of Commerce and the Oil and Gas Association.

As I said earlier, you are, we are not operating in a vacuum. As Peter Defer (spelled phonetically) of the Environmental Defense Fund assured me, the setting of this dioxin criterion is the focus of national and international attention. While our neighboring states of Virginia and Maryland have adopted the weaker 1.2 ppq, as has Alabama, the home state of Apple Grove Pulp's parent company, every state that's passed the 1.2 ppq standard has faced litigation from major environmental groups.

It's my understanding that there are twenty-two other states that have proposed the .013 standard that our Board is proposing, that you're proposing, or standards even more stringent.

In conclusion, I urge the Water Board to hang tough. Let those companies that wish to do business in West Virginia adhere to a strict standard. We're for economic development, but not at the expense of the health and safety of our residents and the environment.

Thank you very much.

DR. JENKINS: Thank you, Ms. Woolverton.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

Rick Bady, please.

MR. BADY: My name is Doctor Richard Bady, and I'm in the Department of Physics and Physical Science at Marshall University, and I'm testifying on behalf of OVEC, the Ohio Valley Environmental Coalition, an environmental group started in the West Virginia, Kentucky and Ohio tri-state area, as we call it.

These comments are relevant to the discussion of setting a standard for dioxins, but are intended to apply to the regulation of water pollutants in general. I am professionally interested in issues of science and public policy, that is, how policy makers such as the Water Resources Board can use science to make policy decisions, and, more importantly, how policy makers can deal with situations where the available science is inadequate.

Even when adequate science is available, public policy must involve more than scientific arguments, but the situation is especially tricky in the case of pollutants like dioxins where a scientific consensus does not exist and is not likely to exist in the near future.

While I won't attempt to define science here, let me point out three requirements in any discussion needed for it to qualify as scientific.

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

First, there are no preordained conclusions. Second, if the data is inadequate, there is no need to come to a conclusion, but rather it is appropriate to wait for further data. Third, the parties involved must be honest in their use of the information that does exist.

A policy making group like the Board is inevitably confronted with situations where all three of these requirements are violated, yet you are required to make a decision anyway. It is perfectly possible for an industry to decide how much of a pollutant their planned activity will release and then try to come up with a rationale for a standard that will fit their needs. Not only it is possible, it's become standard practice.

Thus, my first requirement for a scientific argument is routinely violated as industries hire scientists and lawyers to concoct scientific-sounding arguments to defend what they want to do. Note I'm not saying this is dishonest necessarily, just that it is not science, because the result was chosen ahead of time.

This approach is believable only when the data is inadequate, making possible a wide variety of possible solutions to the question. This violation of my second requirement shows that the Board is in a difficult position

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

of having to make a decision that ideally would be based on science alone, but, in fact, it cannot be. The science just isn't there.

Interested parties can and have come up with plausible arguments for higher or lower standards, all based on the little evidence that exists. I will suggest below that in some cases, for example this one, it is possible to invoke more general principles using what is known to make a decision.

Let me first mention that, unfortunately, my third requirement, that of honesty by all discussants, is also a problem, and the classic example tonight is the now infamous Banbury Conference, and I have included an addendum here with a little article from Science describing the charade that followed that.

So it seems that the Water Resources Board will need to do more than hear scientific arguments to come to a decision in cases like that of dioxin. So, of course, what the Board must do is go back to basics and recall what makes the idea of setting standards sensible in the first place. Why is it good policy to set standards? Are there cases where zero is the only sensible standard?

In other words, are there cases where the only

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

sensible policy is to require a more acceptable technology rather than trying to determine an acceptable level of a pollutant? Are there cases where preventing the pollution in the first place is the only responsible policy? I will argue that this is such a case.

First, consider a classic water pollutant for which a standard can be set, not just because adequate scientific knowledge exists, but because of the nature of the pollutant itself, namely, BOD. It makes sense to set a particular water standard for BOD because, one, it is always present at levels that make it a part of the natural system and non-toxic, two, it quickly biodegrades, and, three, it is not bioaccumulative.

Because of these three characteristics, it is possible to reach a scientific consensus on a standard for BOD that will protect the waters of the state. Notice that dioxin fails on all three of these criteria. It does not naturally occur. It is highly toxic. It's slow to degrade and instead bioaccumulates.

You recall that one of the fundamental goals of the Clean Water Act itself is that the waters of the nation should be fishable. Because of dioxin accumulating in fish tissue, there are already fish advisories, and in

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

some cases the source of the dioxin is not even known. Allowing any dioxin at all to be dumped into any river would only make this worse, and there is no excuse for allowing it.

I would like to ask the Water Resources Board to recognize that in cases of chemicals like dioxin, setting an allowable standard above zero does not constitute sensible public policy. Several objections will arise such as the problem of detection. The whole point of my argument is to try to prevent the pollution in the first place rather than focusing on what we can detect once it is too late.

The focus of public policy must be not just on how much pollution we should accept, but rather on how to prevent it in the first place. A classic example in air pollution is of all the various well-known air pollutants lead is the only one that has, since the Clean Air Act, significantly gone down. The only reason that's significantly gone down is because we banned the use of lead in gasoline. With the other air pollutants where we simply set acceptable standards, we still have problems. We still have non-attainment areas all over the country, including West Virginia.

That example shows that there are cases where

JANET T. SURFACE

COUNT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

the only sensible public policy is to prevent pollution in the first place. In the case of chemicals like dioxin, the sensible thing to try to do is to prevent the use of technologies that produces it. The industries that produce dioxins will simply have to change. It is perfectly possible to produce paper products without using chlorine chemicals, pick a random example, thus eliminating the major source of the dioxins.

We need to establish zero standards in some cases-- The need to establish zero standards in some cases should be recognized by the Board and implemented in the case of chemicals like dioxins.

Thank you.

DR. JENKINS: Thank you, Mr. Bady.

C. T. McDermitt, please.

MR. McDERMITT: First of all, I get red hot when I hear about the Ohio River being polluted, especially by a British company. I hunt and fish along the Ohio River, and now I can't even fish out of the river. Yet, the state requires me to pay twenty-five dollars and fifty cents for a hunting and fishing license. Now that makes a lot of sense to me.

And the fish in the river is paid by the tax payers. They spend millions of dollars a year turning

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

millions of fish in the river loose each year. Now what are they going to do? Are they going to poison the fish more than they are already?

Now you talk about hot. I am hot, especially at the British coming in. My people thought they was suckers, what, two hundred years ago. And they can do it again.

Okay. It just don't make sense to me. I see the Governor get on television. We're going to clean the state up. Now what do I hear here tonight? We're going to lower the regulations. That just don't make sense to me.

And as far as the water pollution, the state is not doing their job on that. We've had trouble with the garbage dumps. ERA, it has finally been shut down, but there have been two streams already polluted and one lake. I mean, it's too late. You can't wait till the pollution is there and done its damage and then say, well, we're going-- Just like the horse getting out of the barn. We're going to shut the door. It's too late. It's gone.

So what's the state going to do? Are they going to turn the Ohio into a sewer? I mean, it's already a sewer. Commercial fishing has been stopped. That's what we've got. And the flow of the river, it's almost like a

JANET T. SURFACE

COUNTY REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

pond. You've got so many lakes, I mean so many dams, there's no flow. And I fish in the Racine Pool. I don't know if you know where that's at or not, but you can go up there and catch a string of fish that long (indicating). You can't eat these fish. There you've spent all day fishing just for the fun of it. You'd like to take them home and eat them, but, no, they're polluted.

So how much more pollution can this river stand? The state has to make up their mind, like I said, whether they're going to let industry in and turn it into a sewer or they're going to clean it up. And right now the river is clean compared to what it was twenty years ago. You can't smell the river, especially the oil smell. That is gone.

But I just can't see letting a paper mill in to do this kind of damage. And I know they do damage because I visited one this spring up at Lucan, Maryland, Westvaco. You talk about Maryland. What gives them the right to poison our rivers? I just can't tolerate that.

So if this comes to Mason County, there's going to be a fight. No doubt about it. That's all I've got to say.

DR. JENKINS: Thank you, Mr. McDermitt. It's good to

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

hear there have been some improvements.

And I've got a request for another break over here, a very urgent request. So let's take another ten minutes.

(WHEREUPON, a break
was taken.)

DR. JENKINS: Let's get started. We just have a few more. For the Board, I'd like to thank all of you whom I know had a prepared statement to make but have been considerate and have shortened things. We certainly appreciate your cooperation. This has been a fine group of people that have come to present their views to the Board tonight. I want to commend you on the way things have gone.

The next person is Dan Jordan.

A VOICE: He left.

DR. JENKINS: Well, if Dan comes back, we'll give him another change.

Perry McDaniel.

MR. McDANIEL: Ladies and gentlemen of the Board, my name is Perry McDaniel, and I'm the President of the West Virginia Environmental Council. On behalf of the Council, I urge the Board to adopt a dioxin standard no less

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALLEN CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

stringent than the EPA recommended criteria of .013 parts per quadrillion with a risk factor of ten to the minus six, and I also encourage the Board to consider adopting a standard more stringent.

You've heard testimony tonight that sixteen states have adopted the EPA criteria and that twenty-two have diverted from that and adopted another criteria. Of interest here is that of those twenty-two, many adopted a more stringent standard.

I'm an attorney here in Charleston, and a good part of my practice deals with environment law and occupational safety and health. My clients don't come from the Chamber of Commerce or from the Manufacturers Association, at least not directly. My clients are those individuals and the families of those individuals who have been impacted by risks and hazards around their homes or in their place of work.

And on behalf of those individuals, I encourage the Board to adopt a standard that will protect the citizens of West Virginia and surrounding states as well as the workers in pulp plants and other industries that emit dioxins.

But it's not my present profession that makes

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

that me familiar with the discussion that's going on here tonight. It's my prior profession and education as a forester. I'm a 1981 graduate of the Division of Forestry at WVU.

In the late seventies, as you may know, the big controversy with regard to forestry and chemicals was with herbicides, especially 2,4,5-T, which you may know by its military name of Agent Orange. And back then the scream was to get rid of 2,4,5-T and not use it. First they restricted its use, very limited in forest lands that were unoccupied, until they finally banned it.

What was the problem with 2,4,5-T? A by-product in the processing of 2,4,5-T was dioxin. Now its proponents said, well, 2,4,5-T doesn't create dioxin. It's just if you make it with too much heat, too much pressure you create dioxin. But still it was unavoidable. Dioxin was created with 2,4,5-T.

And what happened when they finally banned 2,4,5-T? Well, despite even a cry from the forestry industry, it pains me so, they said that we can't live without it. They come up with other chemicals to deal with problems in the forest the 2,4,5-T program had.

So what I'm asking this Board to consider is

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

that, the comments you've heard earlier tonight, is that we do not need to create dioxins in the pulp and paper industry. It can be done without chlorine. And I'm asking the Board not to follow the lead of Maryland and Virginia. What was not said here tonight is that Maryland and Virginia have existing pulp and paper plants, and especially Maryland has an aging plant near Lucan.

Those states, I'm suggesting to the Board, had to consider protection of the existing industries. If we get a pulp and paper plant in West Virginia, it will be a pulp and paper plant built in the nineteen-nineties. It should not be held to the standards of those that were built in the thirties, forties, and fifties when possibly the knowledge of dioxin wasn't as great as it is now.

Dioxin is a carcinogen. It's a very dangerous chemical. And I'm asking the Board to adopt the EPA criteria or more stringent and send a signal that, yes, pulp and paper may come into West Virginia, but it's not going to come in with weak standards that our neighboring states unfortunately have adopted.

And, finally, on behalf of many of the individuals in West Virginia who couldn't be here tonight for various reasons, there are a lot of comments you've

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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heard tonight from industry, and we do ask the Board to consider the technical data on dioxin and its hazards. It has been interpreted one way, mainly by most of the speakers who have been, of course, paid by the industry to be here.

We do not need to weaken the standard that the Board has proposed, and I ask you to stick to your guns and adopt the standard that EPA has suggested, or even to strongly consider a more stringent standard.

Thank you.

DR. JENKINS: Thank you, Mr. McDaniel.

Cindy Rank.

MS. RANK: Good evening. My name is Cindy Rank. Right now I'm President of the West Virginia Highlands Conservancy. I've spoken before this Board, previous members of this Board on behalf of Friends of the Little Kanawha several times, and I'd like to speak on behalf of both of those groups tonight.

I do have written comments that I will submit to you all. I'd like to change gears just for a couple minutes tonight and not talk about dioxin for a moment or two. I would like to register some very strong concerns about subtle changes that have happened in the regulations from the 1984 regs to the 1989 regs that are of concern,

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great concern to some of us who live near very small headwaters areas and very little streams that are sometimes called intermittent.

There were suggestions here that we have another category of stream use tonight called intermittent. Some of the changes that we've had in the anti-degradation policy and in the use in the standards have been very subtle, but for those of us who depend upon high quality streams in those areas, I think that there have been, have the possibility of having substantial changes in our area. In that regard, my comments you will receive. I know that's not something you're considering at this particular moment for this go-around, but I'd like to talk further about that.

Secondly, the second of my three comments tonight was that the Highlands Conservancy would like to go on record certainly in supporting the dioxin standard that you are recommending in the standards for the many reasons that the Environmental Council and several other people have mentioned tonight. I would much prefer a zero discharge level, certainly. If we're going to go with one standard, I would suggest that the EPA standard is one that will drive us to a better technology.

In an ideal world, I think we would all

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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consider everyone in this room as capable of sitting down together and doing what's best for all of us and for the future, but I think in a practical world we do need standards such as the one that you're trying to set now that will drive the technology to improve their impacts on society. And I would strongly recommend that you hang tight and promulgate, at least as far as the Legislature, the standard that you are now proposing.

We would also go on record as supporting and applauding your expansion of your clarification of the existing uses in the definitions. That's been a problem for some of us, your designated used. We've had that problem on some of the small streams, and the definition of existing uses in the clarification and the anti-degradation policy is a step forward as far as I'm concerned.

Also, I applaud you for getting into the wetlands controversy. In Hampshire County, where I'm from, it's very difficult for farmers to understand why they can't fill in some of their wetlands and grow corn when the federal government has destroyed thousands of acres of wetlands, and I think the state has to really face up to dealing with the problem and the preservation of a disappearing resource as well as interfering with some of

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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the people's, what they consider their rights on their own property.

My final point that I'd like to make tonight is just a suggestion that there is an updated version of the high quality streams list put out by the West Virginia Department of Natural Resources. It's the Fifth Edition, and it's dated 1986. We still refer in our water quality standards to the 1979 Fourth Edition, and there's no significant change in the language in here.

There are some additional, perhaps, three hundred additional streams that are listed, and it just probably would be appropriate if we updated our water quality standards to refer to the most recent list.

That's it. And I thank you very much. I'll give you my comments.

DR. JENKINS: Thank you, Ms. Rank.

Beth Hamilton.

MS. HAMILTON: Hi. I'm speaking on behalf of myself and of other people my age, but also I work at Wangunger (spelled phonetically) at Kyle, West Virginia until tomorrow. I have been working there for the past four years putting myself through college. And I've had a bunch of callers recently that are concerned about this pulp mill

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

going in at Apple Grove. And at first I was kind of shocked, but then I was glad, I was real glad that they got me as the operator because most of the operators there, which they number twenty-seven right now, wouldn't really take the time to talk with somebody about a concern like that.

And I had three callers today, and they knew that it was West Virginia day, and they got me. And they expressed their concern, and they wanted me to represent them. And they're from New Jersey, Connecticut and another one from Connecticut. And they wanted to express their concern to the Board that they won't be visiting West Virginia anymore if the pulp mill goes in because they're aware of what pulp mills do.

And it seems unreasonable to me that you would lower the standards, or lower the standards to what they already are. It makes no sense. It should be zero, and it's not. But, anyway, these people wanted me to express their concern, and I told them I would.

And I'd like to express my concern. I hope that you would use good judgment and not be pushed over by industry and money, because it doesn't really matter in the long run.

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

That's all I have to say.

DR. JENKINS: Thank you, Ms. Hamilton.

Norm Steenstra.

MR. STEENSTRA: It's been a long night. Besides, it's our state's birthday. And I think it's a cause for celebration tonight even though you may have wanted to get done earlier, we did too, because there's a lot of interest. This is the third type of meeting this week I've gone to on three different subjects. I go to lots of these things. I've never seen a room with so many ties and so many shirts. I mean, they're all here. And I also know that people drove literally hundreds of miles today at their own expense with no organized campaign to express their concern.

The first ten speakers or so you heard tonight fulfilled every one of the stereotypes that we hold for them, and probably the last ten speakers that you heard tonight fulfilled every stereotype that they or maybe you hold for us.

The point of it is this is the big one. I believe this is probably the most well attended triennial, I think that's what we're talking about, review of water standards in the state. I know it's my first. I think it's because every one of us are looking more and more at what

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ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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we're doing to this world.

And as we look at it more and more, they have to hire more and more consultants and lawyers to balance us. And so I think we're good for economic development, but, anyway, this isn't about the pulp mill and it is about the pulp mill. You've heard it officially. This is about the dioxin standard. But, in reality, we're looking at a company that before they break ground are coming in and asking you, people who don't live here, people who will not live downstream from it, lawyers who came down here at several hundred dollars an hour on airplanes to tell us to reduce our standards.

And if Robinson and McElwee would just let me use their little map a second, I'll just put it there (indicating). It's their map, but it's our country. If you notice those highlighted states, there's a couple things in common there that I think we have to look at.

The first one is they're very heavily dominated by the timber and/or pulp industries. And basically all those little colored states mean is the suits were able to convince a board like you that you need to lower standards. I think, in perspective, that's what we're here about tonight. You've got all these different ideas

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

out in front of you, different points of view, different lifestyles arguing, but, in reality, we should have brought a map that showed the opposite colors.

The point is, you are going to make that decision. I think we've demonstrated, and I promise a continued and sustained demonstration, that the people of this state care about water quality.

The thing that bothers me is that we're leaving out a whole class of people when we do the risk assessment, when we figure out who's eating the fish, and that is a socio-economic and racial class with minority groups that eat a whole lot more of this fish than, say, myself or a high priced lawyer. Or if we try to average it out across the whole entire state population or national population, we are missing an entire segment of society who is dependent to a large extent. It's like if you go into rural West Virginia and you try to get the average venison consumption across this country versus what it might be in Webster County.

The other thing that I think we have to look at, and I hope you'll look at, and I don't have a background in science, but our accumulation in our bodies and in the fish and in the wildlife in this state of dioxin doesn't

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COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

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only come from water. Please remember that. I understand from some of my friends if I took this thing and put it my mouth (indicating) and ate it because it was bleached paper, I, in fact, am digesting some kind of a dioxin.

Dioxins come from burning. They come from pulp mills. But we're concerned with water here today. So if we're looking at risk assessment, please take into consideration there's more than one way we're going to get the dioxin.

I know you've got, in an imperfect world you've got to make quick and hasty decisions. I can promise you that many of these people in this room from both sides will be following the entire regulatory process on this. Your decisions on this, particularly on dioxin, will be reviewed, argued and continued up until probably late March or early April of 1992, an election year. I don't envy your job. I don't envy it.

But I do want to close with the concept that plug into your thinking that we do care. We're not, at least this guy here, is not opposing a pulp mill. What we are looking at is a holistic approach to the environment in West Virginia. Yes, we can have industry. Yes, we can have industry if they demonstrate to us they'll come down and

JANET T. SURFACE

COURT REPORTER

ROUTE 2, BOX 9

ALUM CREEK, WEST VIRGINIA 25003-9601

PHONES: (304) 756-3302 OR 756-3611

play by West Virginia's rules, not by rules they suggest we set for them when they come in.

Thank you very much. And, again, I do certainly respect the decision you have to make. And I'm sure we'll be seeing each other again. Thank you.

DR. JENKINS: Thank you, Mr. Steenstra.

I think I saw somewhere that the total production of dioxin at a paper plant is forty percent in the paper or pulp and thirty percent in the sludge and thirty percent in the effluent. That may not be correct.

This was the last card that I hand in my hand, but I would like to invite anyone in the audience who would like to speak at this time to rise and come forward.

(No response.)

DR. JENKINS: There being none, Jan, should we adjourn this operation?

MS. TAYLOR: I think so.

MS. NEAL: I move that we adjourn.

DR. JENKINS: We are adjourned. Thank you very much for attending.

STATE OF WEST VIRGINIA

COUNTY OF KANAWHA

I, the undersigned, Deborah A. Dickens,
Stenomask Reporter, do hereby certify that the foregoing is,
to the best of my skill and ability, a true and accurate
transcript of the proceedings had in the above-styled public
hearing on the 20th day of June, 1991.

Given under my hand this 3rd day of July,
1991.

Deborah A. Dickens
Reporter

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**Statement of Peter A. Tennant, Ohio River Valley Water Sanitation Commission
Public Hearing before the West Virginia State Water Resources Board
as Proposed Amendments and Revisions to Series 1 of the Legislative Rules
"Requirements Governing Water Quality Standards"
Charleston, West Virginia
June 20, 1991**

The Ohio River Valley Water Sanitation Commission is an interstate agency established in 1948 to carry out a Compact signed by eight states and approved by the United States Congress. The Compact pledges the signatory states to faithful cooperation in the control of future pollution and the abatement of existing pollution from the rivers, streams and water in the Ohio River basin. The guiding principle of the Compact is that pollution originating in one state shall not injuriously affect the uses of the waters of another state.

The Commission, also known as ORSANCO, is made up of three representatives of each state appointed by the respective Governors and three representatives of the federal government who are appointed by the President. Commissioners currently representing West Virginia are William T. Wallace, M.D. (ex officio as Commissioner of the Bureau of Public Health), Edgar N. Henry and Ronald R. Potesta.

Water quality standards have long been a matter of particular concern to the Commission. Since the Ohio River serves as a boundary between states for 941 miles of its 981 mile length, coordination among the states in establishing applicable water quality standards is necessary. West Virginia shares its portion of the river with the State of Ohio; Ohio, in turn, shares its stretch with West Virginia and Kentucky, and Kentucky shares its portion with Ohio, Indiana and Illinois. Thus the decisions made by one state can affect waters shared by several other states.

ORSANCO has adopted Pollution Control Standards for discharges to the Ohio River (copy attached) pursuant to authorities established under Article VI of the Compact. These standards include stream criteria which must be maintained in order to support the designated uses of the river. While those criteria are not considered water quality standards per se, they represent the Commission's recommendations to the states for Ohio River standards. The Commission's criteria are developed by a work group of representatives from the states water quality standards programs. Both the Compact and the Commission standards recognize the powers of the states to adopt more stringent requirements.

The ORSANCO Pollution Control Standards were most recently revised in October 1990. With regard to the stream criteria, the most significant changes were the adoption of Escherichia Coli as an alternative bacterial indicator for protection of contact recreation, establishment of a one in one million cancer risk level as the basis for stream criteria for carcinogens, and use of the harmonic mean as the design stream flow for waste water discharge requirements for carcinogens.

Because each of the six states along the Ohio River operate on their own three year cycles of water quality standards review, uniformity in states' standards for the Ohio River is probably not an attainable goal. As states develop and adopt standards which go beyond the Commission's current regulations, they are encouraged to bring them forward for consideration by the other member states.

A review of the proposed revisions to the West Virginia Water Quality Standards indicates that in all instances they are at least as stringent as the stream criteria in the ORSANCO Pollution Control Standards. In addition to the specific criteria included in the Commission's criteria, the West Virginia standards include limits for aluminum, antimony, beryllium, iron, manganese, nickel, thallium, and total residual chlorine. Specific limits are also included for 28 organic chemicals, whereas the ORSANCO criteria adopt U.S. EPA recommendations by reference. If the Board feels that specific limits for any of these chemicals should be included in the ORSANCO standards and in the other states' water quality standards for the Ohio River, I would strongly urge you to submit appropriate comments to the Commission. I note that among the organics for which specific limits are being proposed at this time are 1,2-, 1,3-, and 1,4-dichlorobenzene. All three of these chemicals were found in the Ohio River during an intensive survey for toxics in the Wheeling to Parkersburg segment. That survey, by the way, was a joint undertaking by ORSANCO, the West Virginia Division of Water Resources, and the Ohio Environmental Protection Agency.

There are several provisions in the proposed revisions which I find especially commendable. In Section 3, the coverage would be expanded to incorporate wetlands and to address aquatic ecosystems. By the proposed change to Section 7.2, the provisions of Section 3 would apply without exception.

The mixing zone criteria are concise and readily understandable. The proposed revision would specifically prohibit acute toxicity to aquatic organisms within a mixing zone. This is an area where I believe ORSANCO could profit by considering West Virginia's approach.

I have some concern with the proposed Section 8.2.C. The second sentence seems overly broad. It seems to me that use of a specific time period would offer an improvement, i.e. annual compliance with carcinogenic standards shall be determined by computing the arithmetic mean of all samples analyzed within one year for the carcinogen.

In reviewing the proposed revisions, I noted several minor points to bring to your attention. In Appendix C, the hydroelectric power plant at Hannibal (Wetzel County) on the Ohio River is not listed. On page 2 of Appendix E, a cadmium criterion of 10 micrograms per liter is listed for public water supply protection in the Ohio River. That criterion is no longer included in the ORSANCO standards due to the adoption of the more stringent criterion for aquatic life protection. (Since the entire Ohio River has been designated for all uses, the Commission criteria only include the most stringent limit for a given parameter.) Finally, the units for body burden concentrations on page 7 of Appendix E are listed as " $\mu\text{g/L}$ "; they should be micrograms per gram ($\mu\text{g/g}$).

In summary, the proposed revisions add much to the West Virginia Water Quality Standards and help to maintain West Virginia's place at the forefront of the states in protecting water quality. For the record, I would like to note that ORSANCO, in adopting many of the specific criteria which are included in the West Virginia water quality standards, recognized that their attainment would require control of nonpoint sources. I am sure that this is also the case on West Virginia's intrastate waters. The revised standards you are considering represent a challenging job ahead in their attainment. On behalf of ORSANCO, I look forward to working with the Board and the Water Resources Section to achieve our common goal of clean water.

Ohio River Valley Water Sanitation Commission

Pollution Control Standards

for discharges to the Ohio River

1990 Revision

NOTICE OF REQUIREMENTS

You are hereby notified that, having considered all the evidence presented at public hearings, the Ohio River Valley Water Sanitation Commission, at its regularly held meeting on October 18, 1990, acting in accordance with and pursuant to the authority contained in Article VI of the Ohio River Valley Water Sanitation Compact, adopted and promulgated, subject to revision as changing conditions require, Pollution Control Standards 1990 Revision for the modification or treatment of all sewage from municipalities or other political subdivisions, public or private institutions, corporations, or watercraft, and for the modification or treatment of all industrial wastes discharged or permitted to flow into the Ohio River from the point of confluence of the Allegheny and Monongahela Rivers at Pittsburgh, Pennsylvania, designated as Ohio River mile point 0.0, to Cairo Point, Illinois, located at the confluence of the Ohio and Mississippi Rivers, and being 981.0 miles downstream from Pittsburgh, Pennsylvania.

Under terms and provisions of the Ohio River Valley Water Sanitation Compact all sewage from municipalities or other political subdivisions, public or private institutions, corporations or watercraft and all industrial wastes discharged or permitted to flow into the Ohio River will be required to be modified or treated to the extent specified in the standards established as above set forth.

To the extent that Pollution Control Standards (September 10, 1987 Revision), which were established by Commission action September 10, 1987, have been amended or restated by virtue of Pollution Control Standards 1990 Revision, the Pollution Control Standards 1987 Revision, including any definitions and application procedures appended to or incorporated therein, are rescinded.

Alan H. Vicory, Jr.

Executive Director and Chief Engineer

Water as herein defined, resulting from any process or operation including storage and transportation, manufacturing, commercial, agricultural, and government operations.

E. "*Mixing Zone*" means that portion of the water body receiving a discharge where effluent and receiving waters are not totally mixed and uniform with the result that the zone is not representative of the receiving waters and may not meet all ambient water quality standards or other requirements of any signatory state applicable to the particular receiving waters.

F. "*Net Discharge*" is determined by excluding the amount of pollutant in an intake water when determining the quality of a discharge if both the intake and discharge are from and to the same body of water.

G. "*96 hour LC50*" as used in these regulations means the concentration of a substance that kills 50 percent of the test organisms within 96 hours. The test organisms shall be representative important species indigenous to the Ohio River or standard test organisms.

H. The "*Ohio River*," as used in these regulations, extends from the point of confluence of the Allegheny and Monongahela Rivers at Pittsburgh, Pennsylvania, designated as Ohio River mile-point 0.0 to Cairo Point, Illinois, located at the confluence of the Ohio and Mississippi Rivers and being 981.0 miles downstream from Pittsburgh.

I. "*Ohio River Valley Water Sanitation Commission*" (Commission) means a body corporate created by authority of the Compact and is the operating agency established to implement the Compact. It consists of three (3) representatives of each signatory state and three (3) representatives of the Federal government.

J. "*Other Wastes*" means any waste other than Sewage, Cooling Water, Residues from

Potable Water Treatment Plants, Industrial Wastes or Toxic Wastes, which if discharged to the Ohio River could cause or contribute to any violations of these regulations, or of any water quality standards of any signatory state or which may be deleterious to the designated uses.

K. "*Persistent Substances*" means those substances which have a half-life for degradation under natural environmental conditions of more than 4 days. All other substances are non-persistent.

L. "*Representative Important Species*" means those species of aquatic life whose protection and propagation will assure the sustained presence of a balanced indigenous community. Such species are representative in the sense that maintenance of suitable water quality conditions will assure the overall protection and sustained propagation of the balanced indigenous community.

M. "*Residues from Potable Water Treatment Plants*" means those wastes emanating from processes used in water purification. Such processes may include sedimentation, chemical coagulation, filtration, iron and manganese removal, softening and disinfection.

N. "*Sewage*" means water-carried human or animal wastes from such sources as residences; industrial, commercial or governmental establishments; public or private institutions; or other places. The admixture of Sewage with Industrial Wastes, Toxic Wastes or Other Wastes, in amounts detrimental to the quality of the combined effluent shall also be regarded as Sewage.

O. "*Substantially Complete Removal*" means removal to the lowest practicable level attainable with current technology.

P. "*Toxic Wastes*" means wastes containing substances or combinations of substances in

concentrations which might reasonably be expected to cause death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions, including malfunctions in reproduction, or physical defor-

mations in fish, other aquatic life, wildlife, livestock, or humans.

Q. "Wastewater" means sewage and/or industrial wastes as herein defined.

III. DESIGNATED USES

The Ohio River, as hereinbefore defined, has been designated by the Compact as available for safe and satisfactory use as public and industrial water supplies after reasonable

treatment, suitable for recreational usage capable of maintaining fish and other aquatic life and adaptable to such other uses as may be legitimate.

IV. STREAM CRITERIA

A. The minimum conditions which the wastewater discharge requirements (Section V) are intended to achieve in the receiving waters outside the Mixing Zone are as follows:

- 1. Freedom from anything that will settle to form objectionable sludge deposits which interfere with designated water uses.
- 2. Freedom from floating debris, scum, oil and other floating material in amount sufficient to be unsightly or deleterious.
- 3. Freedom from materials producing color or odors in such a degree as to create unaesthetic conditions or a nuisance.
- 4. Freedom from substances in concentrations which are toxic or harmful to humans, animals, or fish and other aquatic life; which would in any manner adversely

affect the flavor, color, odor, or edibility of fish and other aquatic life, wildlife, or livestock; or which are otherwise detrimental to the designated uses specified in Section III.

B. To assure that the foregoing conditions will be attained, the following criteria shall be met outside the Mixing Zone:

- 1. DISSOLVED OXYGEN: Concentrations shall average at least 5.0 mg/L per calendar day and shall not be less than 4.0 mg/L at any time provided that a minimum of 5.0 mg/L at any time is maintained during the April 15-June 15 spawning season.

2. TEMPERATURE: Allowable stream temperatures are:

<u>Month/Date</u>	<u>Period Average</u>		<u>Instantaneous Maximum</u>	
January 1-31	45 °F	7.2 °C	50 °F	10.0 °C
February 1-29	45	7.2	50	10.0
March 1-15	51	10.6	56	13.3
March 16-31	54	12.2	59	15.0
April 1-15	58	14.4	64	17.8
April 16-30	64	17.8	69	20.6
May 1-15	68	20.0	73	22.8
May 16-31	75	23.9	80	26.7
June 1-15	80	26.7	85	29.4
June 16-30	83	28.3	87	30.6
July 1-31	84	28.9	89	31.7
August 1-31	84	28.9	89	31.7
September 1-15	84	28.9	87	30.6
September 16-30	82	27.8	86	30.0
October 1-15	77	25.0	82	27.8
October 16-31	72	22.2	77	25.0
November 1-30	67	19.4	72	22.2
December 1-31	52	11.1	57	13.9

3. pH: No value below 6.0 nor above 9.0.

4. BACTERIA:

- a. Maximum allowable level of fecal coliform bacteria for use as a source of public water supply -- For the months of November through April, content shall not exceed 2,000/100 ml as a monthly geometric mean based on not less than five samples per month.
- b. Maximum allowable level of fecal coliform bacteria for Contact Recreation -- For the months of May through October, content shall not exceed 200/100 ml as a monthly geometric mean based on not less than five samples per month; nor exceed 400/100 ml in

more than ten percent of all samples taken during the month.

- c. Maximum allowable level of Escherichia coli bacteria for Contact Recreation -- For the months of May through October, measurements of Escherichia coli bacteria may be substituted for fecal coliform. Content shall not exceed 130/100 ml as a monthly geometric mean, based on not less than five samples per month, nor exceed 240/100 ml in any sample.

5. DISSOLVED SOLIDS: Not to exceed 500 mg/L as a monthly average value, nor exceed 750 mg/L at any time. (Equivalent 25°C specific conductance values are 800 and 1,200 micromhos/cm, respectively.)

6. AMMONIA: The concentration of un-ionized ammonia (as NH_3) shall not exceed 0.05 mg/L; un-ionized ammonia shall be determined from values for total ammonia-N, pH and temperature, by means of the following equation:

$$Y = \frac{1.2 (\text{Total ammonia-N})}{[1 + 10^{(\text{pK}_a - \text{pH})}]}$$

$$\text{pK}_a = 0.092 + \frac{2730}{(273.2 + T_c)}$$

Where:

T_c = Temperature, degrees Centigrade

Y = Un-ionized ammonia, milligrams per liter

Combinations of values for total ammonia-N, pH, and temperature which yield an un-ionized ammonia concentration of 0.05 mg/L are shown in Appendix A.

7. CHEMICAL CONSTITUENTS

Not to exceed the following concentrations:

Constituents	Concentration mg/L
Arsenic	.05
Barium	1.0
Chloride	250
Fluoride	1.0
Nitrite + Nitrate Nitrogen	10.0
Nitrite Nitrogen	1.0
Phenolics	.005
Selenium	.01
Silver	.05
Sulfate	250

Constituent ²	Chronic Criteria Concentration ug/L	Acute Criteria Concentration ug/L
Cadmium	$e^{(.7852[\ln \text{Hard.}] - 3.490)}$	$e^{(1.128[\ln \text{Hard.}] - 3.828)}$
Chromium (hexavalent)	11	16
Copper	$e^{(.8545[\ln \text{Hard.}] - 1.465)}$	$e^{(.9422[\ln \text{Hard.}] - 1.464)}$
Cyanide (free)	5	22
Lead	$e^{(1.273[\ln \text{Hard.}] - 4.705)}$	$e^{(1.273[\ln \text{Hard.}] - 1.460)}$
Mercury	.012	2.4
Zinc	$e^{(.8473[\ln \text{Hard.}] + .7614)}$	$e^{(.8473[\ln \text{Hard.}] + .8604)}$

(Note: Concentrations for metals are total recoverable except hexavalent chromium which is dissolved.)

²Wastewater discharge requirements for these constituents shall be calculated based on the chronic criteria concentrations, the in-stream concentration above the point of discharge, and the minimum 7-day, 10-year stream flow as contained in Appendix B. The acute criteria concentrations shall not be exceeded in the stream at any time. Criteria for cadmium, copper, lead, and zinc at specific hardness values are listed in Appendix C.

8. RADIONUCLIDES:

Gross total alpha activity (including radium-226 but excluding radon and uranium) shall not exceed 15 picocurie per liter (pCi/L) and combined radium-226 and radium-228 shall not exceed 4 pCi/L. Concentration of total gross beta particle activity shall not exceed 50 pCi/L; the concentration of tritium shall not exceed 20,000 pCi/L; the concentration of total Strontium-90 shall not exceed 8 pCi/L.

9. OTHER TOXIC SUBSTANCES:

Stream criteria for substances not otherwise specified in this section shall be derived based on the following:

a. For the protection of aquatic life:

i. Non-Persistent Substances - not to exceed an average of one-twentieth (0.05), nor at any time exceed one-tenth (0.1) of the 96 hour LC₅₀ of representative important species indigenous to the Ohio River or standard test organisms.

ii. Persistent Substances - not to exceed an average of one one-hundredth (0.01), nor at any time exceed one-twentieth (0.05) of the 96 hour LC₅₀ of representative important species indigenous to the Ohio River or standard test organisms.

b. For the protection of human health, criteria published by the United States Environmental Protection Agency pursuant to Section 304(a) of the Federal Clean Water Act shall be used.

i. For substances identified as human carcinogens, wastewater discharge requirements shall be developed based on the instream concentration above the point of discharge, and calculated so as to prevent one additional cancer per one million population at the harmonic mean stream flow (see Appendix B).

ii. For substances not identified as human carcinogens, wastewater discharge requirements shall be developed based on the instream concentration above the point of discharge and calculated to meet the stream criteria at the minimum seven-day, ten year flow (see Appendix B).

c. Limiting concentrations other than those derived from paragraphs a. and b. above may be used for the protection of human health or aquatic life when justified on the basis of scientifically defensible evidence.

V. WASTEWATER DISCHARGE REQUIREMENTS

A. General

1. No discharge of any Sewage, Industrial Wastes, Toxic Wastes, Other Wastes, Cooling Water or Residues from Potable Water Treatment Plants shall cause or contribute to a violation of these Wastewater Discharge Requirements, shall preclude the attainment of any designated use of the mainstem waters of the Ohio River or shall interfere with the attainment of the stream criteria set forth in Section IV.

2. All discharges of Sewage, Industrial Wastes, Toxic Wastes, Other Wastes, Cooling Water or Residues from Potable Water Treatment Plants shall be treated or otherwise modified so as to provide:

a. Substantially Complete Removal of settleable solids, which may form sludge deposits;

b. Substantially Complete Removal of oil, debris, scum and other floating material;

c. Reduction of total suspended solids and other materials to such a degree that the discharge will not produce a substantial negative visible contrast to natural conditions in turbidity, color or odor of the river, or impart taste to the potable water supplies or cause tainting of fish flesh;

d. Reduction of all substances in amounts which, when concentrated or combined in the receiving stream, would result in conditions toxic or harmful to humans, animals, or fish and other aquatic life; which would in any manner adversely affect the flavor, color, odor, or edibility of fish and other aquatic life, wildlife, or livestock; or which are otherwise detrimental to the designated water uses specified in Section III.

B. Sewage

1. MINIMUM LEVEL OF TREATMENT

Sewage shall be treated, prior to discharge, to meet the following effluent limitations in addition to contained in the requirements of Section V.A.

a. Biochemical Oxygen Demand

i. Five day Biochemical Oxygen Demand (BOD₅) - the arithmetic mean of the values for effluent samples collected during a month shall not exceed 30 mg/L, and the arithmetic mean of the values for effluent samples collected during a week shall not exceed 45 mg/L.

ii. Five-day Carbonaceous Biochemical Oxygen Demand (CBOD₅) may be substituted for Five-Day Biochemical Oxygen Demand provided that the arithmetic mean of the values for effluent samples collected during a month shall not exceed 25 mg/L, and the arithmetic mean for the values of effluent samples collected during a week shall not exceed 40 mg/L.

b. Suspended Solids

The arithmetic mean of the values for effluent samples collected during a month shall not exceed 30 mg/L, and the arithmetic mean of the values for effluent samples collected during a week shall not exceed 45 mg/L.

c. pH

The effluent values for pH shall be maintained within the limits of 6.0 to 9.0.

d. Bacteria

i. During the months of November through April, the geometric mean of the fecal coliform bacteria content of effluent samples collected during a month shall not exceed 2000/100 ml.

ii. During the months of May through October, the geometric mean of the fecal coliform bacteria content of effluent samples collected during a month shall not exceed 200/100 ml, and no more than 10 percent of the values shall exceed 400/100 ml.

iii. During the months of May through October, *Escherichia coli* may be substituted for fecal coliform provided that the geometric mean of the values for effluent samples collected during a month shall not exceed 130/100 ml, and no more than 10 percent of the values shall exceed 240/100 ml.

2. ALTERNATIVE TREATMENT

Such facilities as waste stabilization ponds and trickling filters shall be deemed to provide effective treatment of sewage provided that the requirements of Sections V.A., V.B.1.(c) and (d) are met, that the effluent does not cause any violations of applicable states' water quality standards or Sections III and IV of these regulations, and that the following requirements are met.

a. Biochemical Oxygen Demand

i. Five-day Biochemical Oxygen Demand (BOD₅) -- the arithmetic mean of the values for effluent samples collected during a month shall not exceed 45 mg/L; the arithmetic mean of the values for effluent samples collected during a week shall not exceed 65 mg/L; and the average percent removal for any month shall not be less than 65 percent.

ii. Five-day Carbonaceous Biochemical Oxygen Demand (CBOD₅) may be sub-

stituted for BOD₅, provided that the levels are not less stringent than the following: the arithmetic mean of the values for effluent samples collected during a month shall not exceed 40 mg/L; the arithmetic mean of the values for effluent samples collected during a week shall not exceed 60 mg/L; and the average percent removal for any month shall not be less than 65 percent.

b. Suspended Solids

The arithmetic mean of the values for effluent samples collected during a month shall not exceed 45 mg/L; the arithmetic mean of the values for effluent samples collected during a week shall not exceed 65 mg/L; and the average percent removal for any month shall not be less than 65 percent.

C. Industrial Wastes, Including Toxic Wastes

1. The minimum level of treatment for Industrial Wastes including Toxic Wastes, prior to discharge shall be in accordance with national effluent limitations and guidelines adopted by the Administrator of the United States Environmental Protection Agency pursuant to Sections 301 and 302 of the Federal Clean Water Act, national standards of performance for new sources adopted pursuant to Section 306 of the Federal Clean Water Act, and national toxic and pretreatment effluent limitations, adopted pursuant to Section 307 of the Federal Clean Water Act.

2. In addition, the net discharge of the following toxic pollutants is prohibited:

a. Aldrin(1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1, 4-endo-5, 8-exo-dimethanonaphthalene)

b. Dieldrin (1,2,3,4,10,10-hexachloro-6, 7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1, 4-endo-5, 8-exo-dimethanonaphthalene)

c. DDT, including DDD and DDE

i. DDT means 1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethane and some o,p'-isomers

ii. DDD (TDE) means 1,1-dichloro-2, 2-bis(p-chlorophenyl) ethane and some o,p'-isomers

iii. DDE means 1,1-dichloro-2, 2-bis(p-chlorophenyl) ethylene

d. Endrin (1,2,3,4,10,10-hexachloro-6, 7-epoxy-1, 4,4a,5,6,7,8,8a-octahydro-1, 4-endo-5, 8-endo-dimethanonaphthalene)

e. Toxaphene - a material consisting of technical grade chlorinated camphene having the approximate formula of $C_{10}H_{10}Cl_8$ and normally containing 67-69 percent chlorine by weight

f. Benzidine - the compound benzidine and its salts as identified by the chemical name 4,4-diaminobiphenyl

g. Polychlorinated Biphenyls (PCB) - a mixture of compounds composed of the biphenyl molecule which has been chlorinated to varying degrees

D. Residues from Potable Water Treatment Plants

The use of controlled discharge for Residues from Potable Water Treatment Plant processes of sedimentation, coagulation and filtration may be authorized provided that as a minimum the discharge meets all the requirements of Section IV.A. and V.A.

VI. MIXING ZONE DESIGNATION

A. A Mixing Zone shall be deemed to exist for each discharge. When required, the specific numerical limits for any Mixing Zone shall be determined on a case by case basis, and shall include considerations for existing uses, linear distance (i.e., length and width) from the point of discharge, surface area involved, and volume of receiving water within the defined zone.

B. Conditions within the Mixing Zone shall not be injurious to human health, in the event of a temporary exposure.

C. Conditions within the Mixing Zone shall not be lethal to aquatic life or wildlife that may enter the zone.

D. The Mixing Zone shall be free from substances attributable to Sewage, Industrial Wastes, Toxic Wastes, Other Wastes, Cooling Water, or Residues from Potable Water Treatment Plants in quantities which:

1. Settle to form sludge deposits;
2. Float as debris, scum, or oil;
3. Contaminate natural sediments so as to cause or contribute to a violation of:
 - a. appropriate stream criteria outside the Mixing Zone, or
 - b. any condition of the designated uses of the water.
4. Impart a disagreeable flavor or odor to flesh of fish or other aquatic life, wildlife or livestock which are consumed by man and

which acquire such a flavor because of passage through or ingestion of the waters from the Mixing Zone.

E. The Mixing Zone shall be located so as not to interfere significantly with migratory movements and passage of fish, other aquatic life, and wildlife. No waste discharge related to the Mixing Zone shall, outside the limits of the Mixing Zone interfere with potable water supply intakes, bathing areas, reproduction of fish, other aquatic life and wildlife; or adversely affect fish or aquatic life normally inhabiting waters prior to addition of waste discharged; or result in any other violations of appropriate stream criteria relating to the designated use at or above the appropriate critical river flow as shown in Appendix B.

VII. LIMITATION

Nothing contained in these regulations shall be construed to limit the powers of any state signatory to the Compact to promulgate

more stringent criteria, conditions and restrictions to further lessen or prevent the pollution of waters within its jurisdiction.

VIII. VARIANCE

A. The Commission may grant a variance from the provisions of Section V of these regulations, provided that the uses set forth in Section III are maintained. The applicant for a variance shall adhere to the following:

1. The specific reasons for the variance shall be clearly stated in writing;
2. The burden of proof is upon the applicant to assure that the uses set forth in Section III are maintained;
3. Prior concurrence of the state where the applicant's discharge is located and those

state(s) which may be affected must be obtained;

4. Such additional information shall be provided to the Commission as it may request.

B. A variance may be granted for a period not to exceed the life of the applicable discharge permit; the applicant may apply for a variance renewal prior to the expiration of the permit.

IX. ANALYTICAL METHODS

Tests or analytical determinations to determine compliance or non-compliance with the Wastewater Discharge Requirements and stream criteria established herein shall be made in accordance with accepted procedures such as those contained in the (a) latest edition of Standard Methods for the Examination of Water and Wastewater prepared and published jointly by the American Public Health Association (APHA), American Water Works Association (AWWA), and Water Pollution Control

Federation (WPCF); (b) Annual Book of ASTM Standards, Part 31 - Water published by the American Society for Testing and Materials; (c) Guidelines Establishing Test Procedures for the Analysis of Pollutants (40 CFR 136) by the U.S. Environmental Protection Agency; or (d) by such other methods as are approved by the Commission equal to or superior to or not available within methods in documents listed above, provided such other test methods are available to the public.

X. SEVERABILITY CLAUSE

Should any one or more of the Pollution Control Standards hereby established or should any one or more provisions of the regulations herein contained be held or

determined to be invalid, illegal or unenforceable, for any reason whatsoever, all other Standards and other provisions shall remain effective.

APPENDIX A

Combinations of Values for Total Ammonia-N,
Temperature, and pH Which Yield an
Un-ionized Ammonia Concentration of .05 mg/L

pH	T _F	T _C	41	45	50	55	59	64	68	70	72	75	77	80	84	86	89
			5	7.2	10	12.8	15	17.8	20	21.1	22.2	23.9	25	26.7	28.9	30	31.7
6.5	106	89	71	56	46	38	31	27	23	20	18	16	15	13	11	10	9.2
6.6	84	71	56	46	36	30	25	21	17	16	14	13	12	10	9.0	8.2	7.3
6.7	67	56	46	36	29	24	20	17	13	12	11	10	9.4	8.2	7.2	6.5	5.8
6.8	53	45	36	28	23	19	16	13	11	9.8	9.0	8.0	7.5	6.5	5.7	5.2	4.6
6.9	42	36	28	22	18	15	12	11	8.4	7.8	7.2	6.3	5.9	5.3	4.5	4.1	3.7
7.0	33	28	22	18	14	12	9.7	7.8	6.7	6.2	5.7	5.1	4.7	4.1	3.6	3.3	2.9
7.1	27	22	18	14	12	9.2	7.7	6.2	5.3	4.9	4.5	4.0	3.8	3.3	2.9	2.6	2.3
7.2	21	18	14	11	9.2	7.3	6.1	4.9	4.2	3.9	3.6	3.2	3.0	2.6	2.3	2.1	1.9
7.3	17	14	11	8.9	7.1	5.8	4.9	3.9	3.4	3.1	2.9	2.6	2.4	2.1	1.8	1.7	1.5
7.4	13	11	8.9	7.1	5.7	4.6	3.9	3.1	2.7	2.5	2.3	2.0	1.9	1.7	1.5	1.3	1.2
7.5	11	8.9	7.1	5.7	4.6	3.7	3.1	2.5	2.1	2.0	1.8	1.6	1.5	1.3	1.2	1.1	.95
7.6	8.4	7.1	5.7	4.6	3.7	3.1	2.5	2.0	1.7	1.6	1.5	1.3	1.2	1.1	.94	.85	.77
7.7	6.7	5.7	4.6	3.7	3.1	2.5	2.0	1.6	1.4	1.3	1.2	1.0	.97	.85	.76	.69	.62
7.8	5.3	4.5	3.6	2.9	2.3	2.0	1.6	1.3	1.1	1.0	.94	.84	.79	.69	.61	.55	.50
7.9	4.2	3.6	2.9	2.3	1.9	1.5	1.3	1.0	.88	.82	.76	.67	.63	.55	.49	.45	.40
8.0	3.4	2.9	2.3	1.8	1.5	1.2	1.0	.82	.71	.66	.61	.54	.51	.45	.40	.37	.33
8.1	2.7	2.3	1.8	1.5	1.2	.94	.81	.66	.57	.53	.49	.44	.41	.37	.33	.30	.27
8.2	2.1	1.8	1.5	1.2	.93	.76	.65	.53	.46	.43	.40	.36	.34	.30	.27	.25	.22
8.3	1.7	1.5	1.2	.93	.75	.62	.52	.43	.38	.35	.33	.29	.28	.25	.22	.20	.19
8.4	1.4	1.2	.93	.75	.60	.50	.43	.35	.31	.29	.27	.24	.23	.20	.18	.17	.16
8.5	1.1	.93	.75	.60	.49	.40	.35	.29	.25	.24	.22	.20	.19	.17	.16	.14	.13
8.6	.88	.75	.60	.49	.40	.33	.28	.24	.21	.20	.18	.17	.16	.14	.13	.12	.11
8.7	.71	.60	.49	.40	.33	.27	.23	.20	.17	.16	.16	.14	.14	.12	.11	.11	.10
8.8	.57	.49	.40	.32	.27	.22	.19	.17	.15	.14	.13	.12	.12	.11	.10	.09	.09
8.9	.46	.40	.32	.27	.22	.19	.17	.15	.13	.12	.11	.10	.10	.09	.09	.09	.09
9.0	.38	.32	.27	.22	.19	.17	.15	.13	.11	.10	.09	.08	.07	.06	.05	.04	.03

T_F : Temperature in degrees Fahrenheit
T_C : Temperature in degrees Centigrade

APPENDIX B
CRITICAL FLOW VALUES

River Reach			
From	To	Min. 7-day, 10 yr. Low Flow in cfs*	Harmonic Mean Flow in cfs**
Pittsburgh	Montgomery Dam (MP 32.4)	4,800	20,600
Montgomery	Willow Island Dam (MP 161.8)	5,800	24,900
Willow Island	Gallipolis Dam (MP 279.2)	6,800	29,200
Gallipolis	Greenup Dam (MP 341.0)	8,500	36,500
Greenup	Meldahl Dam (MP 436.2)	9,800	42,100
Meldahl	McAlpine Dam (MP 605.8)	11,000	47,300
McAlpine	Uniontown Dam (MP 846.0)	13,000	55,900
Uniontown	Smithland Dam (MP 918.5)	18,800	80,800
Smithland	Cairo Point (MP 981.0)	46,300	199,000

* Minimum 7-day, 10-year low flow (in cubic feet per second) based on calculations by the U.S. Corps of Engineers.

** Harmonic mean flow (in cubic feet per second) based on analysis of stream flow data from U.S. Geological Survey.

APPENDIX C
NUMERICAL VALUES OF CADMIUM, COPPER, LEAD, AND ZINC CRITERIA
AT SPECIFIED HARDNESS LEVELS

	CADMIUM		COPPER		LEAD		ZINC	
Hardness mg/L	Chronic Criterion ug/L	Acute Criterion ug/L	Chronic Criterion ug/L	Acute Criterion ug/L	Chronic Criterion ug/L	Acute Criterion ug/L	Chronic Criterion ug/L	Acute Criterion ug/L
50	0.7	1.8	7	9	1.3	34	59	65
100	1.1	3.9	12	18	3.2	82	106	117
150	1.6	6.2	17	26	5.3	137	149	165
200	2.0	8.6	21	34	7.7	197	191	211
250	2.3	11.0	26	42	10	262	230	254
300	2.7	13.5	30	50	13	331	269	297

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COMMENTS OF DAVID M. FLANNERY ON BEHALF OF
THE WEST VIRGINIA CHAMBER OF COMMERCE
AND WEST VIRGINIA MANUFACTURERS ASSOCIATION
REGARDING THE PROPOSED WATER QUALITY STANDARD FOR DIOXIN

JUNE 20, 1991

1. Introduction.

My name is David M. Flannery, I am an attorney with the Charleston law firm of Robinson & McElwee. I appear as the Chairman of the Environment and Land Use Committee of the West Virginia Chamber of Commerce and as a member of the Environmental Committee of the West Virginia Manufacturers Association. My comments will address the proposal of the Board to establish, for the first time, a West Virginia water quality standard for dioxin.

While we join the Board in its objective of establishing a dioxin standard that is protective of human health and the environment, we are concerned that the proposal by the Board is significantly more restrictive than can be properly justified. Fortunately, we have as a precedent the fact that a number of other states, including our neighbors Maryland and Virginia, have adopted water quality standards for dioxin which have been approved by the United States Environmental Protection Agency (EPA) as reasonable and scientifically defensible to protect human health and the environment.

2. Economic Development.

Realistically, we must recognize that West Virginia is in direct economic competition with other states. We must not constrict our opportunities to compete and attract development by

adopting regulatory requirements that are more restrictive than they need to be to protect the quality of our environment.

This very issue has been addressed by our Legislature in stating the following public policy to guide your actions in establishing water quality standards:

It is declared to be the public policy of the State of West Virginia to maintain reasonable standards of purity and quality of the water of the State consistent with (1) public health and public enjoyment thereof; (2) the propagation and protection of animal, bird, fish, aquatic and plant life; and (3) the expansion of employment opportunities, maintenance and expansion of agriculture and the provision of a permanent foundation for healthy industrial development. W.Va. Code §20-5A-1.

This policy statement has been at the heart of our water quality program for many years and was reaffirmed by the Legislature earlier this year when it enacted the West Virginia Groundwater Protection Act. W.Va. Code §20-5M-2. This policy statement has thus stood the test of time and establishes a clear mandate for action by this Board. Simply stated, our laws require you to set a standard for dioxin which is consistent with both environmental protection and economic development.

3. EPA's Role.

Prior to discussing the scientific and policy issues involved in establishing a water quality standard for dioxin, it is appropriate to discuss the proper role of EPA and its water quality criteria in this rulemaking.

It is well established that the individual states, rather than EPA, have primary responsibility for the adoption and

implementation of water quality standards. Consistent with its role under the federal Clean Water Act to provide support and assistance to the states, EPA has published a number of water quality criteria covering a variety of compounds, including dioxin. It is EPA's dioxin criteria that has formed the apparent basis for the Board's proposal. EPA's dioxin criteria, however, represents non-regulatory, non-binding guidance from which other states and EPA itself has deviated on many occasions.

EPA's regulations promulgated pursuant to Section 304(1) of the Clean Water Act have expressly recognized that:

[S]tates may derive values different from the values in the EPA's water quality criteria documents to reflect site-specific factors, including predicted local human consumption of aquatic foods, as well as the state's determination of an appropriate risk level, and any more recent scientific data that may not be included in EPA's criteria documents.¹

Indeed, this principal is most clearly illustrated by Exhibit 1, which shows that there are a total of seven states that have approved dioxin water quality standards of 1.0 ppq or 1.2 ppq. The states which have approved dioxin water quality standards at 1.0 ppq are New Hampshire, New York, and Tennessee. Alabama, Georgia and our neighboring states of Virginia and Maryland have approved a 1.2 ppq standard. The Maryland standard of 1.2 ppq was approved by EPA Region III on September 12, 1990 while an identical standard for Virginia was approved on February 25, 1991. The 1.2 ppq standard for Georgia was approved by EPA Region IV on June 3,

¹ 54 Fed. Reg. 23868, 23876 (June 2, 1989)

1991. The 1.0 ppq standard for New York was approved by EPA Region II on September 30, 1985.

To understand why EPA so freely allows such deviation from its dioxin criteria, the Board need only consider that EPA's criteria were developed in the late 1970s and early 1980s based upon very limited data and information. The data were all from a single study on rats, commonly known as the Kociba study. Since then, a significant body of new information and data has been developed which shows that the EPA criteria overestimate the risk to human health from dioxin. That new information is described in the other documents we have submitted for the record and will, therefore, not be addressed in detail in these comments.

Suffice it to say that EPA recognizes (as it did in approving 1.2 ppq standards for Maryland, Virginia and Georgia) that its estimate of upper limit cancer risk is a higher number than that of other federal agencies. It is nine times greater than the Food and Drug Administration (FDA) and four times higher Center for Disease Control (CDC). These differences occur even though each agency based its risk estimates on the Kociba study. In 1988, EPA convened a Workgroup to reexamine the scientific basis and methods used by EPA for estimating the cancer risk for dioxin. The Workgroup reviewed the cancer risk factors used by EPA, FDA and CDC and observed that there was no obvious scientific basis for excluding any of these values from the acceptable range.²

² A Cancer Risk-Specific Dose Estimate for 2,3,7,8-TCDD, June 1988, p. 49.

In March of this year, EPA's Office of Water published a Technical Support Document for Water Quality Based Toxics Control to provide guidance to states and EPA regional offices on procedures for use in the water quality-based control of toxic pollutants. EPA acknowledged in that document that "... safety does not require the absence of all risk...." Since safety does not equate to an absence of all risk, the goal of this Board's efforts must be to arrive at a reasonable, acceptable level of risk utilizing scientifically defensible factors. EPA describes the process this way:

Any human criterion for a carcinogen is based on at least three interrelated considerations: potency, exposure, and risk characterization. States may make their own judgments on each of these factors within reasonable scientific bounds, but documentation to support their judgments must be clear and in the public record.³

Thus, while EPA has its own criteria for dioxin, it does not insist on the adoption of that criteria by the states. Rather, EPA recognizes the wide latitude of the states to adopt dioxin standards that are protective of human health and the environment. Certainly, standards at the level of 1.2 ppq have met this test.

4. Fish Consumption.

One of the most significant factors bearing upon the establishment of a dioxin water quality standard is the potential of actual human exposure to dioxin from the consumption of contaminated fish. Unlike shellfish, the fish which predominate

³ Technical Support Document for Water Quality-based Toxics Control, (TSD) EPA, March 1991, at page 40.

West Virginia streams are subject to cleaning and removal of internal organs which removes tissue that could accumulate contaminants.

Obviously then, any scientific analysis done on the basis of whole body amalgamation of raw test species can, and should, be adjusted downward significantly to accommodate the reality that most people in this state eat only the lean edible tissue of fish and usually only after it has been well cooked.

EPA's current figure of 6.5 grams per day represents its estimate of average consumption of fish and shellfish from estuarine and freshwater by the entire U.S. population. By far, most states have defaulted to the EPA's 6.5 grams per day consumption value. However, EPA recommends use of consumption values that reflect the most current, relevant and/or site-specific information available.

Thus, the real issue for this Board is to determine how much of the average fish consumption by West Virginians actually comes from West Virginia waters which might be impacted by the standards being set. If, as a result of this investigation there is a particular water body or segment that produces unusually high amounts of fish for consumption, the Board can establish individual water quality standards for those individuals segments. Since West Virginia does not have estuarine waters and does not produce significant amounts of shellfish, it is reasonable to assign a significantly lower number to the average fish consumption than the EPA figure of 6.5 grams per day.

5. Risk Level.

An additional significant factor that bears upon the appropriateness of any dioxin water quality standard adopted by the Board is the risk level which is determined to be most appropriate. The Maryland, Virginia and Georgia criteria of 1.2 ppq were based on a 10^{-5} risk level. This risk level is frequently used in state and federal regulatory actions, and is considered by EPA to be within an adequately protective range. A 10^{-5} risk level is within the range of risk levels that EPA has stated it will approve, and has approved, when reviewing state dioxin standards.⁴ It is also consistent with risk levels used by other states such as Michigan, Minnesota, Maryland, Ohio, Iowa, Indiana, Alabama, South Carolina, Nebraska, New Hampshire, Virginia, Wisconsin and Georgia in the development of their water quality standards for dioxin.

It is common for the public to erroneously assume that all occupational and environmental regulations are based on a maximum cancer risk of 1×10^{-6} (1 in 1,000,000) and that any risk above this level poses a serious threat to public health. In fact, risks at and below 1×10^{-6} are so small as to be negligible, and environmental regulatory decisions are frequently based on risks of 1×10^{-5} and higher.

The current background for cancer in the United States is generally believed to be no less than one in four, meaning that in a population of one million people, 250,000 will develop some form

⁴ Memorandum to Water Management Division Directors from LaJuana S. Wilcher, EPA Assistant Administrator (EPA, Jan. 5, 1990).

of cancer in their lifetimes. This translates to a statistical calculation of each person's risk of contracting cancer as is illustrated below and in Exhibit 2.

Background	0.250000	(25.0000 percent risk)
1×10^{-6}	0.250001	(25.0001 percent risk)
1×10^{-5}	0.250010	(25.0010 percent risk)

In the final analysis, selection of a risk level is a policy decision. We recognize that the Board has historically utilized the risk factor of 10^{-6} in setting water quality standards. However, given the fact that the Kociba study has been extensively discredited and that many scientists are questioning the assumption that dioxin causes cancer, together with the widespread use and acceptance of 10^{-5} by EPA, other states, and other federal agencies, the Board would be clearly justified in using 10^{-5} in setting its dioxin standard.

This is particularly so in view of the extremely conservative assumptions built into the potency factor.⁵ As EPA stated in its March, 1991 Technical Support Document:

It is important to note that cancer potency factors may overestimate actual risk. Such potency estimates are subject to great uncertainty due to two primary factors: (1) adequacy of the cancer data base (i.e., human versus animal data) and (2) limited information regarding the mechanism of cancer causation. The actual risk may be much lower, perhaps as low as zero, particularly for those chemicals for which human carcinogenicity information is lacking. Risk levels of 10^{-5} , 10^{-6} ,

⁵ For an extensive discussion supporting the use of a 1×10^{-5} risk level, see Comments in Support of a Dioxin Water Quality Standard for South Carolina. (Anderson, ENSR, Aug. 7, 1990).

and 10^{-7} are often used by states as minimal risk levels in interpreting their standards.⁶

On behalf of the West Virginia Chamber of Commerce and West Virginia Manufacturers Association, Dr. Mark Reasor, Professor of Pharmacology and Toxicology at West Virginia University will be filing written comments with the Board. In his comments, Dr. Reasor will address the significance of risk assessment calculations and point out that the modeling involved is not an exact science. He will also point out that the process will produce estimates based on multiple assumptions which may or may not have relevance to circumstances of interest. Dr. Reasor's comments will counsel the Board to avoid getting caught up in the emotion of this issue and to apply scientific facts and principals to address this matter. I commend Dr. Reasor's written comments to your careful consideration.

6. Conclusion.

As you deliberate towards your final decision about the adoption of a water quality standard for dioxin, there are three principles that should guide you:

1. EPA's dioxin criteria are not binding on Board and EPA encourages the Board to adopt alternative limits where the Board believes that justification exists;
2. In the range of values that would be appropriate to protect public health and the environment, EPA and various states, including our neighbors Maryland and

⁶ TSD 40

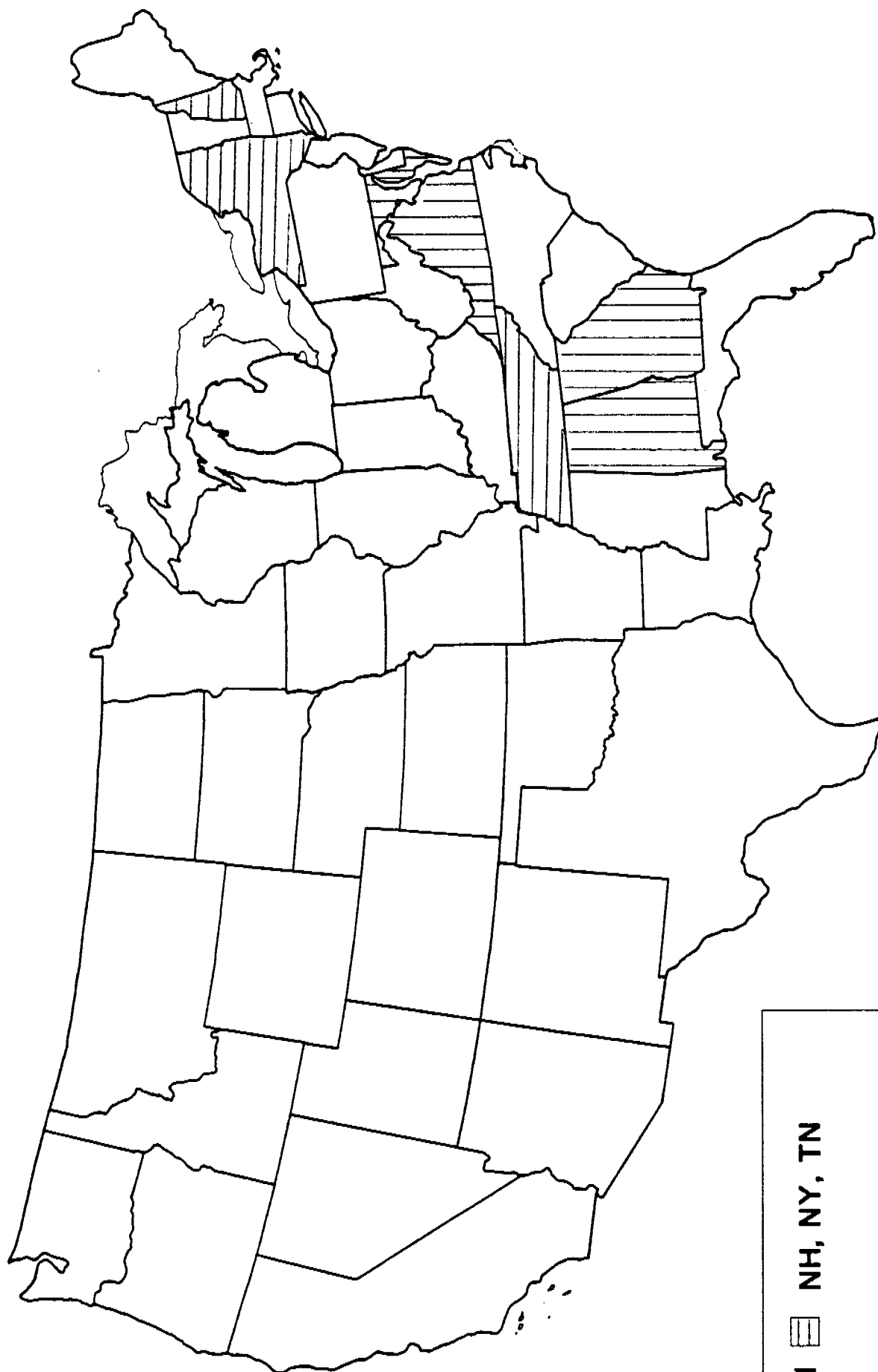
Virginia, have determined that 1.2 ppq is a perfectly acceptable water quality standard for dioxin; and

3. Given West Virginia's competitive position relative to states such as Maryland, Virginia, Georgia and Alabama, which have adopted dioxin water quality standards of 1.2 ppq, the public policy statement of our Water Pollution Control Act requires that no more stringent standards be adopted for West Virginia. Any more stringent standards would be unnecessary to protect the water quality of our streams and would not be consistent with the statutory objective of "the expansion of employment opportunities, maintenance and expansion of agriculture and provision of a permanent foundation for healthy industrial development."

I thank you for the opportunity to bring these comments to your attention on behalf of these organizations and I would be pleased to address any questions you may have.

DIOXIN CRITERIA

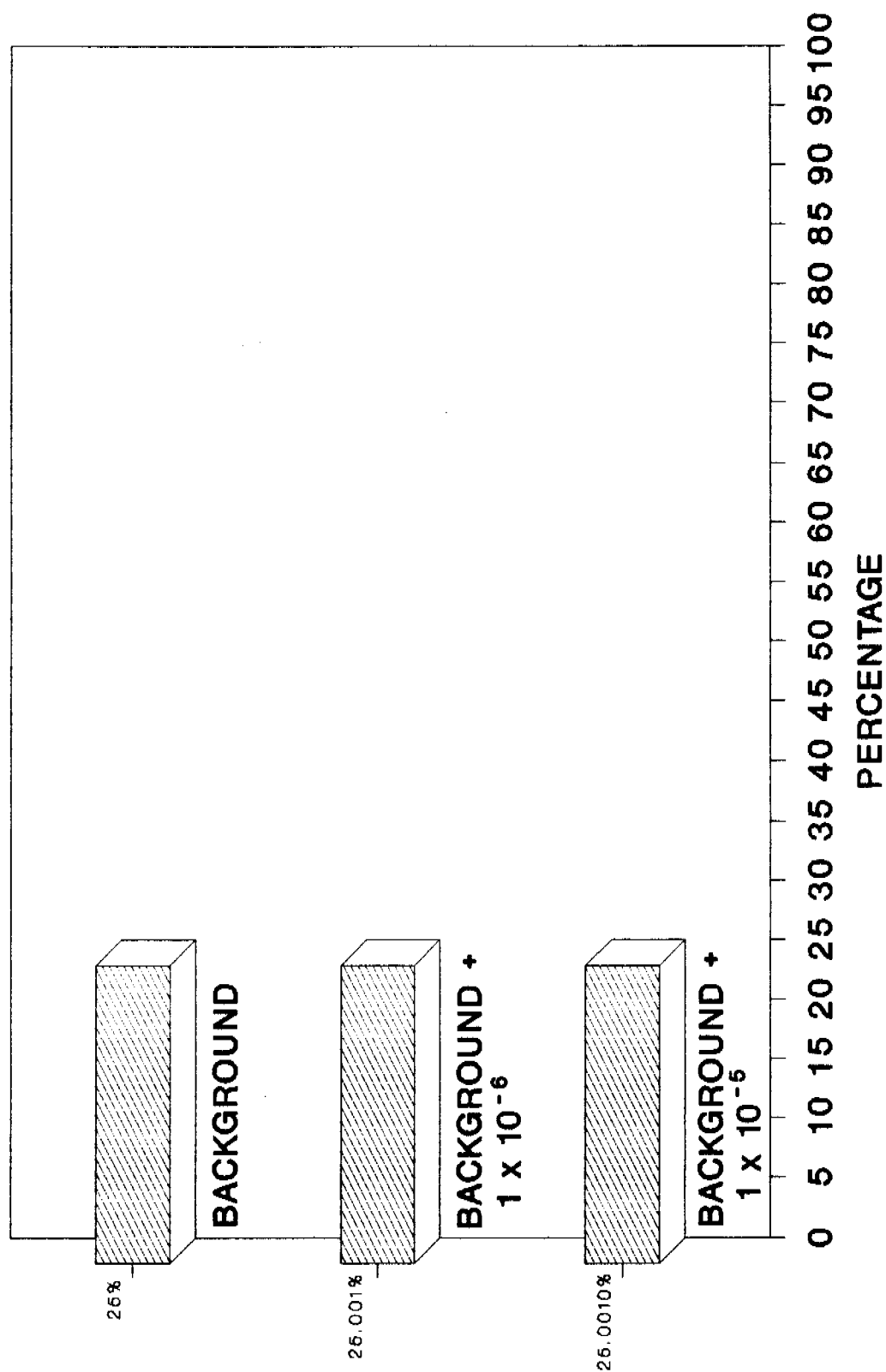
EXHIBIT 1



1 NH, NY, TN

1.2 AL, GA, MD, VA

CANCER RISK LEVELS



**A WATER QUALITY STANDARD
FOR DIOXIN IN THE
STATE OF WEST VIRGINIA**

Prepared for

Westvaco Corporation
New York, NY

Prepared by

ENVIRON Corporation
Arlington, VA

June 20, 1991

CONTENTS

	<u>Page</u>
I. INTRODUCTION	1
II. DERIVATION OF AN AMBIENT WATER QUALITY STANDARD	3
III. CONSUMPTION FACTORS AND BODY WEIGHT	7
IV. BIOCONCENTRATION AND BIOACCUMULATION	10
V. THRESHOLD MODEL OF CARCINOGENESIS	12
VI. NON-THRESHOLD MODEL OF CARCINOGENESIS	14
VII. DISCUSSION OF ALTERNATIVE STANDARDS	19
VIII. REFERENCES	20

I. INTRODUCTION

To meet requirements under the 1987 amendments to the federal Clean Water Act administered by the U.S. Environmental Protection Agency (EPA), the State of West Virginia must establish ambient water quality standards for surface waters of the State. These standards will be used as the basis for limiting discharges to surface water by point sources under the National Pollutant Discharge Elimination System (NPDES), taking into account any site-specific conditions that affect the relationship between discharges and ambient water quality.

Among the substances for which the State must issue such standards is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, also known as TCDD or, more simply, as dioxin. Although this substance can produce a variety of adverse biological effects at relatively low levels of exposure, the State's principal concern is with its potential to cause cancer. Dioxin has caused cancer in experiments with laboratory animals at doses at or above 0.01 μg per kg of body weight per day. Toxicologists generally assume, in the absence of evidence to the contrary, that substances which cause cancer in animals can also cause cancer in humans at sufficient doses. Various procedures have been used to estimate how much risk of cancer in humans is associated with various levels of exposure, starting with the data from the animal experiments. The quantitative description of that association is called the dose-response relationship.

Humans can be exposed to dioxin entering surface waters by using those waters as a source of drinking water or by consuming fish caught in those waters. Fish concentrate or "bioaccumulate" dioxin in their fatty tissues to concentrations substantially higher than those in the water in which they live. By making assumptions about the average quantities of water and fish consumed by a person over a lifetime and the extent of bioaccumulation in fish, the State can estimate the average daily dose that person would receive for any concentration of dioxin in the surface water body in question. If the estimated doses are combined with the dose-response relationship, the risk of human cancer can be estimated as a function of concentration of dioxin in surface water. Finally, the State can choose a level of protection from risk that it considers adequate, given the uncertainty whether dioxin can cause any human cancer at very low doses.

Based on an assessment completed in 1984, the EPA has recommended criteria for standards. In terms of concentrations in ambient water, the criteria are 13 and 14 parts per quintillion (ppqt) for consumption of fish only or consumption of both fish and water, respectively. The criteria are based on a series of assumptions that seemed reasonable to EPA at the time they were first developed, but which are now subject to considerable debate. The EPA criteria concentrations are well below current limits of detection for dioxin in water, but by using assumptions about dilution of effluent by normal stream flow downstream of a discharger, the State can translate the ambient water quality standard to a discharge concentration limit. Depending on the amount of dilution, the limiting concentration in discharges may or may not be detectible.

The Westvaco Corporation, which operates paper mills in neighboring states of Kentucky, Maryland, and Virginia, has requested ENVIRON Corporation to assist in the preparation of comments on the contemplated standards.

II. DERIVATION OF AN AMBIENT WATER QUALITY STANDARD

The intent of a water quality standard is to limit human and environmental exposures to levels that are unlikely to have a significant adverse effect on human health or environmental resources such as aquatic life. In the case of dioxin, limiting risks to human health appears to limit effects on aquatic life as well, and our comments will focus principally on human health.

The first step in setting a water quality standard is to determine what levels of exposure will be adequately protective of human health. Although chloracne, a skin condition, is associated with exposure to dioxin and other chlorinated organic compounds, no other human health effects have been unambiguously associated with such exposure, and human data alone are clearly inadequate for developing a numerical estimate for protective exposure levels. In laboratory animals, exposure to dioxin has been associated with a "wasting syndrome" traceable to a generalized chronic toxicity, to effects on the immune system, to problems in reproduction and development, to cancers of the liver and perhaps other organs, and possibly to toxicity of other organs and the nervous system. Some or even all of these effects could occur in humans at sufficient levels of exposure, and the information obtained in experiments with the laboratory animals can potentially be used to estimate what levels of exposure are protective for health effects in humans.

Toxicologists generally agree that all of the effects mentioned above, with the possible exception of cancer, will not occur unless some "threshold" level of exposure is exceeded. For such effects, the exposures that do and do not cause toxicity in animals can be used as a starting point for estimating the exposures at which human health effects might begin to become significant, through application of standardized "safety" or "uncertainty" factors. The resulting exposure levels, expressed as daily doses, are called "Reference doses (RfDs)" by EPA or more generally "Acceptable Daily Intakes (ADIs)." Some of the toxicological information on dioxin is summarized in Table 1. From these data, toxicologists have speculated that ADIs in humans might range from near 1 picogram per kilogram of body weight per day (pg/kg bw/day)--from the immunotoxicity and reproductive toxicity studies--to hundreds or thousands of pg/kg bw/day. More recently, a consensus has begun to emerge that most or even all of the toxic effects of dioxin are mediated by the same biological

mechanisms and that interspecies differences in toxicity can be understood by examining how these mechanisms work in the different species (Roberts 1991). This line of reasoning results in estimates of an ADI in the range 1 to 10 pg/kg bw/day for non-cancer effects (Roberts 1991; WHO 1990).

TABLE 1 Summary of Non-cancer Effect Levels for Dioxin		
Non-cancer Effect	Lowest-Observed- Effect Level pg/kg/ bw/day	Reference
Lethality	10,000	Allen et al. 1977
Immunotoxicity	120	Hong et al. 1987
Liver Toxicity	1,000	Kociba et al. 1979, NTP 1982
Developmental toxicity/teratogenicity	125,000	Sparschu et al. 1971a,b
Reproductive toxicity/fetal toxicity	120	Bowman et al. 1987
Dermal toxicity (chloracne)	10,000	Allen et al. 1977
Neurotoxicity	*	
Kidney toxicity	*	
* No numerical data for this endpoint were found in the literature reviewed.		

If dioxin-related cancers are also mediated by the same mechanisms, as many toxicologists now believe, then limiting exposure to the same range of doses will be protective against cancer as well. EPA policy, however, directs the Agency to assume that any level of exposure to a carcinogen, no matter how small, carries some risk of cancer, unless specific evidence to the contrary is available. This non-threshold model for carcinogens requires regulators to take a different approach to establishing exposures that are without significant health consequence. The usual assumption is that risk, expressed as a lifetime probability of cancer from lifetime exposure to the carcinogen, increases in proportion to the daily dose; the constant of proportionality is called the "slope factor" or

potency of the carcinogen. To establish a limiting dose, the regulator must select a level of risk ("Risk Criterion") that is in some sense acceptable or below regulatory concern. This risk criterion can then be divided by the slope factor to derive a "Risk-Specific Dose" (RSD):

$$\text{RSD} = \text{risk criterion} / \text{slope factor} \quad (1)$$

In its recommendations to the states regarding a water quality standard for dioxin, EPA has used a risk criterion of one in a million (10^{-6}) and a slope factor of 156,000 (1.56×10^5 kg-day/mg). Applying Equation (1), EPA derives an RSD of 0.006 pg/kg bw/day, which is about 160 to 1600 times lower than the range of ADIs derived from the mechanistic explanation for the carcinogenic action of dioxin.

Whether the criterion dose used is an ADI or an RSD, however, and no matter whether the lowest criterion dose is for cancer or non-cancer effects, the remainder of the standard setting process is the same: calculate a concentration in water that will keep human doses below the criterion dose. The daily human dose caused by a concentration CW in water depends on the amount of water consumed daily, WC, the amount of fish consumed daily, FC, the concentrations in water and fish, and the body weight, BW, of the exposed human. If the ratio, R, between the concentration in fish and the concentration in water is known, then the dose can be expressed as follows:

$$\text{Dose} = \text{CW} \times (\text{WC} + \text{R} \times \text{FC}) / \text{BW} \quad (2)$$

In this equation, we have deliberately avoided using the term "bioconcentration factor" (BCF) that usually appears in the position where we have placed R, for reasons that will be discussed later in this document.

The ambient water quality standard, expressed as a concentration WQS, can be derived by setting the calculated dose from Eq. (2) to be just equal to the criterion dose:

$$\text{WQS} = \text{criterion dose} \times \text{BW} / (\text{WC} + \text{R} \times \text{FC}) \quad (3)$$

The numerical value of the standard therefore depends on the choice of values for WC, FC, and R as well as on whether a threshold or non-threshold model is chosen for carcinogenicity. If a threshold model is chosen, a value for the ADI must be selected; if a non-threshold

model is chosen, then values for both the risk criterion and slope factor must be selected. The following sections discuss each of these choices.

III. CONSUMPTION FACTORS AND BODY WEIGHT

Water consumption, fish consumption, and body weight are all factors that are characteristics of individuals and are known to differ among individuals and with age in the same individual. To some extent, these variations cancel out in the computation of a water quality standard for dioxin because large people are more likely to consume large amounts of water and fish than are small people. Furthermore, many of dioxin's potential effects on human health would not occur unless exposure continued over a sufficiently long period of time for the total dose to build to a critical level. For example, the risk of cancer is assumed to depend on the cumulative dose over an entire lifetime of exposure, and is expressed in terms of a daily dose only for the convenience of using daily doses in animals to estimate slope factors for humans. Consequently, the fact that children may have a different ratio of consumption to body weight than do adults has relatively little effect on lifetime cancer risk.

A. Body Weight

The International Commission on Radiological Protection (ICRP 1984) is the source of the 70-kg value for the body weight of "standard man," which is widely used in risk assessments. EPA (1989) has recently suggested that adult U.S. males average closer to 78 kg, but that children and adult females weigh less. Fish and water consumption also differ by age and sex, of course. Although it could probably be demonstrated that the lifetime average weight for the whole U.S. population is slightly lower than 70 kg, the standard is not very sensitive to such small differences, and the State would be justified in using the traditional value of 70 kg.

B. Water Consumption

The consumption of drinking water is usually assumed in risk assessments to average 2 liters per day. Recent guidelines (EPA 1989) have suggested that consumption of tap water averages 1.4 liters per day, and that 2 liters per day is a reasonable maximum consumption rate. In any case, because the estimated contribution to total dose from drinking water is much lower than that from fish consumption, the resulting standard is not sensitive to this assumption.

C. Fish Consumption

Any assumption regarding fish consumption is very difficult to defend as a generality. Fish consumption can differ markedly from person to person, with many people eating essentially no fish and others being nearly "subsistence fishermen." The difference is exaggerated when one examines the available data for freshwater fish, the commodity which the dioxin standard would affect. Moreover, consumption habits can vary greatly over a person's lifetime. The statistic used by EPA in developing its dioxin recommendations (6.5 g/day) is the national average consumption of freshwater and estuarine fish and shellfish, as determined from a survey conducted by the National Marine Fisheries Service in the mid 1970s (SRI 1980), and excludes marine fish.

In West Virginia, no estuarine regions are found. Because the EPA figure of 6.5 g/day includes both estuarine fish and shellfish, which would not be affected by the standard, a substantially lower figure seems more appropriate. ENSR (1989) estimated average freshwater fish consumption in the Mid and South Atlantic regions to be near 1 g/day, which is about 30% lower than the national average of 1.38 g/day (see Table 2). This value seems appropriate for West Virginia, given that the fish constituting these statistics are caught in a variety of waters, only a fraction of which are likely to be influenced by sources of dioxin in West Virginia.

TABLE 2 Regional Freshwater Fish Consumption 1980s Per Capita Average, All Ages (g/day)	
United States	1.38
New England	0.26
Mid Atlantic	0.87
S. Atlantic	<u>1.05</u>
E.S. Central	2.24
E.N. Central	1.79
W.N. Central	2.05
W.S. Central	2.69
Mountain	1.47
Pacific	1.05
Adapted from ENSR (1989)	

IV. BIOCONCENTRATION AND BIOACCUMULATION

Dioxin is more soluble in fatty substances than it is in water, i.e., it is lipophilic. Consequently, dioxin in water tends to move preferentially into the fatty tissues of fish if taken in through the gills or, more importantly, through the mouth while feeding. The concentrations of dioxin in fish are therefore likely to be considerably higher than are the concentrations in the water in which they live. In deriving a water quality standard using Eq. (3), it is important to use a value for the ratio R that represents the true relationship between dioxin measurements in water and the concentrations of dioxin that can be expected in edible fish tissue after equilibrium between dioxin discharges and concentrations in water, sediments, and fish has been reached. This point has often been overlooked in recent calls for using a higher ratio for R than the 5,000 used by EPA in developing its water quality criteria.

EPA originally referred to a bioconcentration factor (BCF) in deriving its criteria. Technically, the BCF is the ratio of dioxin in fish tissue to the concentration of dioxin dissolved in water free of suspended sediment or other materials that would compete with the fish for the dissolved dioxin. While measurable in the laboratory (although often with difficulty), the BCF does not represent the real world, where most of the dioxin in fish tissues arises through ingestion of food or suspended solids to which dioxin is attached. In a sense, the bioaccumulation factor (BAF), which relates dioxin concentrations in fish to the concentrations in their food, would be useful for describing dioxin uptake. However, the relationships between dioxin discharges, suspended solids, and feeding habits is too complicated to model well from laboratory data. Finally, it is important to recognize that compliance with discharge permits is monitored by measuring the total dioxin content in discharges, including the vast majority of dioxin on suspended solids and not just the dissolved fraction, so that the ratio R in Eq. (3) must be chosen to conform with this measurement procedure.

William Sherman (1991) of the S.D. Warren Company has taken all these factors into account and has reanalyzed laboratory and field data on dioxin in fish normalized to a lipid content of 2.5% in edible fish tissues. He concluded that all of the BCFs and BAFs reported are consistent with a "Regulatory BAF" of 5,000 or lower. ENSR (1989) has suggested that

site-specific standards could be set using site-specific ratios derived by measuring dioxin concentrations in representative fish in the affected stream and comparing them with water concentrations measured, or more likely calculated, in the affected stream. Following this suggestion, ENVIRON compared observed concentrations in fillets of fish caught downstream of a paper mill in Virginia with ambient water concentrations estimated from mill discharge data and stream dilution factors. The calculated average ratio was approximately 4,000. The same calculation has been performed using the data in the 104-Mill Study of dioxin in paper mill effluents and the data on fish concentrations from the National Bioaccumulation Study (); this comparison also yielded an average R value of about 5,000.

In West Virginia, an even lower number could be justified, because the lipid content of local fish is often lower than 1% (West Virginia Department of Natural Resources survey, as cited in the comments submitted by the West Virginia Chamber of Commerce et al.). With dioxin content approximately proportional to lipid content, a Regulatory BAF or R value near 2,000 may be appropriate in West Virginia. Furthermore, the R value could be adjusted to account for losses of dioxin from fish in the course of cooking. Dioxin accumulates preferentially in fat, and cooking losses of at least 25% have been measured (ENSR 1989). In summary, the value of R to be used in Eq. (3) need be no higher than 5,000 for protection of public health and could be lower because of low lipid content of West Virginia fish and cooking losses.

V. THRESHOLD MODEL OF CARCINOGENESIS

In 1984, relatively little was known about the ways in which dioxin might have influenced the development of tumors in laboratory animals, and it was reasonable to assume that it might increase cancer risk at any level of exposure above zero. The balance of scientific evidence now suggests strongly that dioxin acts as a promoter of cancer rather than as an agent that acts directly on genetic material, i.e., an initiator. Dioxin's carcinogenic potential is almost surely mediated by receptor binding and enzyme induction, which implies that a non-zero threshold dose is required to create any risk of cancer whatsoever. This position has been articulated more and more clearly over the past several years, for example in two reports of the National Council of the Paper Industry for Air and Stream Improvement (NCASI 1987, 1989), in a report by ENSR (1989), in an update by ENVIRON (1989), and in a report of the Banbury Conference on dioxin (Roberts 1991). Whether the mechanism of dioxin toxicity is via the aryl hydrocarbon (Ah) receptor as concluded at the Banbury Conference or through enzymes involved in the synthesis of glucose (Rozman 1989), many receptor sites need to be occupied before cancer risk begins to rise and a threshold dose is required.

The threshold hypothesis is also consistent with the original experiments that implicated dioxin as a carcinogen. At the request of the Maine Scientific Advisory Panel, a pathology working group convened by PATHCO (Sauer 1990) reexamined the slides from the cancer bioassay of dioxin conducted by Kociba et al. (1978) and found that the observed tumors were seen only in animals that showed liver toxicity, suggesting that toxicity-induced cell proliferation was necessary for tumor formation. Toxicity to the liver would require a threshold concentration of dioxin to be present and implies that dioxin carcinogenicity would also exhibit a threshold. Quantitatively, the Kociba data show no excess tumors at the lowest dose, again consistent with a threshold behavior.

Values for the threshold of dioxin toxicity in the range 1 to 10 pg/kg bw/day are proposed in the documents cited above. In the earlier documents, the proposals were based on the no-observed-effect level of 0.001 $\mu\text{g/kg/day}$ and a hundred-fold safety factor for projecting from animal experiments to human risk. In the later reports, quantitative predictions of dioxin uptake and metabolism in humans are used to derive the estimates.

EPA has recently established a working group to revise its model for dioxin carcinogenicity; it seems likely that the group will develop a threshold model with a threshold in this same range.

In other countries, dioxin is regulated on the basis of such a threshold assumption. A recent World Health Organization conference recommended the use of 10 pg/kg bw/day as a regulatory guideline (WHO 1990).

In summary, West Virginia would be using the best and most current science by adopting a threshold model for dioxin and using a criterion dose in the range 1 to 10 pg/kg bw/day.

VI. NON-THRESHOLD MODEL OF CARCINOGENESIS

If the State insists upon using a non-threshold model for dioxin carcinogenesis, then it should use the best available models and data for a non-threshold approach. The State must make two significant decisions in doing so: choice of the value for the slope factor and choice of a risk criterion. We discuss each of these choices below.

A. Slope Factor

Although other data sets exist that could be used to estimate a slope factor for dioxin, the Kociba et al. (1978) data on tumors in female rats are commonly used as a starting point for projecting tumor rates seen in animal experiments to risks in humans, because they yield one of the highest potencies among the data available; that is, they are conservative. Because the Kociba slides show liver toxicity, some toxicologists would argue that the Maximum Tolerated Dose was exceeded and the study was invalid. Other aspects of the test conformed to the scientific standards then in force and are comparable to those in other experiments with dioxin.

Slope factors for dioxin have been derived at the National level by the EPA, the Food and Drug Administration (FDA), and the Centers for Disease Control (CDC). The slope factor used by EPA is 156,000 kg bw-day/mg and is based on the assumption that rats and humans are equally susceptible to dioxin if measured by dose per unit of surface area ("surface area scaling rule"). The FDA uses a lower slope factor, 17,500 kg bw-day/mg, based on the assumption that rats and humans are equally susceptible to dioxin if measured by dose per unit body weight ("body weight scaling rule"). The difference in scaling accounts for a factor of 5.4 between these two estimates. The balance (factor of about 1.7) is attributable mainly to a correction made by EPA to account for early mortality in the Kociba rats. The CDC estimate is derived differently, but in effect uses the surface area scaling rule without an early mortality correction, and so is about two times lower than EPA's estimate.

Comparison of potencies for substances shown to be carcinogenic in both humans and animals has shown that the body weight scaling rule is a less biased predictor than the surface area scaling rule and that the latter tends to overestimate human risk substantially (Allen et al. 1987). This empirical argument is bolstered by a theoretical one: although carcinogens that

act on systems dominated by cell membranes or that are cleared by metabolic deactivation may follow a surface-area rule, dioxin appears to act on cytosolic receptors (Roberts 1991), which would be distributed over a volume, not a surface.

The reanalysis of the Kociba slides by PATHCO (Sauer 1990) used new rules, developed in 1986 by the National Toxicology Program, for classifying lesions as tumors. With the new rules, fewer tumors were reported in the rats at all dose levels, including the controls. When the linearized multistage model used by EPA is applied to the revised tumor counts (benign and malignant combined), the slope factor drops by a factor of at least 2, depending on the details of modeling. If only malignant tumors are counted, the decrease is even greater. This reduction applies equally to the potencies calculated by the EPA and FDA methods.

If the State decides it must use a linear, non-threshold model for the carcinogenicity of dioxin in its standard setting for dioxin, it should use the best currently available values for the parameters of the model. To the 1984 EPA slope factor of 156,000 kg bw-day/mg, a factor of 5.4 should be applied to restore the scaling factor to a body weight basis and a factor of 2 should be applied to adjust for the new pathology of the Kociba data. Dividing 156,000 by 10.8 yields an estimate of 14,400 kg bw-day/mg.

B. Risk Criterion

The choice of a risk criterion is a policy decision, not a scientific issue. The risk level of one in a million (10^{-6}) used by EPA in its recommendation for the water quality standard, was originally developed with a specific application in mind, namely the practical definition of "no risk" for food additives under the Delaney clause of the Food, Drug, and Cosmetic Act (Rodricks et al. 1987). The assumption was that essentially all people in the United States would be exposed to the same level of risk, and if FDA limited that risk to one in a million over a lifetime exposure, no more than 3 people out of the entire U.S. population would be affected in any one year. Applying that risk criterion to the much smaller population potentially affected by dioxin in surface water adds substantial conservatism with respect to public health, and most regulatory agencies have shown a tendency to use less stringent risk criteria for small populations at risk, because the likelihood of any person being injured even under a non-threshold assumption becomes small.

Many precedents can be cited for appropriate values of the risk criterion. EPA (1989) suggests, but does not have the authority to mandate, that the range 10^{-7} to 10^{-5} is appropriate for setting water quality standards. The State of California uses one in one hundred thousand

(10^{-5}) as the risk criterion for limiting discharges of pollutants to surface and ground water under its Safe Drinking Water and Toxic Enforcement Act (Proposition 65)(CHWA 1987). Travis et al. (1987) have shown that many federal decisions on risk have allowed residual individual risks of 10^{-4} or even more.

These precedents are supported by a more fundamental argument. The current rate of cancer mortality in the United States approaches 20%. Even after removing the most obvious lifestyle contributions to the cancer rate (tobacco and alcohol), the rate would be in the vicinity of 10% (10^{-1}). A reasonable public health goal would be to limit the contribution of industrial sources to 1% of that value (10^{-3}), and a reasonable rule for a single hazard would be to limit it to 1% of that value, or 10^{-5} . This argument takes into account the fact that any one person is very unlikely to be simultaneously exposed to a vast array of cancer hazards.

From the above precedents and principles, the State could reasonably adopt a risk criterion of 10^{-5} instead of its usual criteria of 10^{-6} , especially in the case of dioxin where current science suggests that the non-threshold model is substantially conservative at low dose level. A risk criterion of 10^{-5} is near the lower limit of risks that attempt a balance between protection of the individual and unreasonable overregulation. A value of 10^{-5} is more consistent with the trend of regulatory decisions regarding water pollutants and is used in the neighboring states of Maryland, Ohio, and Virginia, and may be adopted in Kentucky following the legislative review scheduled for dioxin in 1992. In suggesting this approach, we emphasize that nearly all of the assumptions used in calculating a proposed dioxin water quality standard with a non-threshold model are conservative and that true risk, which will likely never be determined, could easily be precisely zero for a tumor promoter such as dioxin.

The degree of health protection afforded by a lifetime risk level of one in 100,000 can be adequately judged only by referring to the assumptions upon which the risk assessment rests. To learn what the figure means, first assume that a standard of 1.2 ppq leads to a computed extra lifetime risk of one in 100,000, and that this determination is scientifically accurate. If this assumption is accepted, and if 100,000 people were to consume fish at the assumed rate daily over their full lifetimes, and if all the fish they consume were to contain dioxin at the maximum level associated with a given standard, then it would be expected that (at most) one of these people might develop dioxin-induced cancer over a 70-year period. If every person living in West Virginia were to consume dioxin-containing fish at the same rate, then the one in 100,000 lifetime risk level would translate to (at most) 20 extra cancer cases over a 70-year period (for a total population of approximately 2,000,000) under this worst-

case set of assumptions. During a 70-year period, there would be approximately 1,800,000 actual cancer cases from all causes other than dioxin exposure reported in West Virginia, assuming current cancer rates remain relatively constant in the future.

This worst case scenario could actually occur only if all of the following are all true:

- (1) Dioxin causes cancer in humans.
- (2) Every person has the same susceptibility to dioxin-induced cancers as the species of rats used as the basis for the risk assessment (the most susceptible of all species and strains that have been studied).
- (3) All excess tumors observed in rats, both benign and malignant, translate to malignancies in humans.
- (4) The biological mechanisms by which dioxin-induced rat tumors are produced are identical to those that occur in people.
- (5) There is no threshold in the dose-response relationship for dioxin-induced cancers - every exposure, no matter how small, increases cancer risk to some degree.
- (6) The risk increases in direct proportion to the dose -- the dose response relationship is linear (a straight line) all the way down to zero dose and zero risk.
- (7) People and rats are at identical risk when exposed at the same dose of dioxin expressed in mg/kg body weight (the FDA-preferred scaling rule).
- (8) All 2,000,000 West Virginia residents consume fish from waters containing dioxin at the standard, from birth to death. All waters contain dioxin at the maximum allowable level, indefinitely into the future.
- (9) Each person's lifetime is 70 years, and all 70 years are spent in West Virginia.
- (10) All fish consumed contain the maximum amount of dioxin that could be accumulated from waters containing dioxin at the water quality standard.

- (11) No dioxin is lost from the fish during cooking, even though studies indicate that at least 25% loss occurs.

From the earlier comments on each of these issues, it should be obvious that few, if any, of these assumptions have a high probability of being correct, and many are highly improbable. Moreover, almost every alternative and more probable assumption that could be listed would show the extra lifetime risk at the standard will be less than one in 100,000, by a non-quantifiable amount. The cumulative effect of incorporating all of these improbable assumptions into the risk assessment can not be completely quantified, but it could be quite large. If, for example, only 10% of West Virginians consume fish from affected waters, and all other assumptions remained the same, then the worst-case estimate of (at most) 20 extra cases would fall to (at most) 2 extra cases over a 70-year lifetime (vs. 1,800,000 in total). If humans were as susceptible to dioxin as the most susceptible strain of mice instead of the most susceptible strain of rats, the cancer potency factor should be smaller by a factor of two or more (UAREP 1988). If the standard is enforced at or near the few points of dioxin discharge, then most waters will contain much lower levels of dioxin (because of dilution) so that both assumptions (8) and (10) will be incorrect, by a wide (but non-quantifiable) margin. As discussed in Chapter V, the weight of scientific evidence supports a threshold model for dioxin-induced carcinogenicity, contrary to assumption (5), and suggests a non-linear dose-response relationship, contrary to assumption (6). If a threshold model were correct, risk would be near zero, and if a non-linear model were correct, risk would be substantially lower than is predicted by the linear model.

The fact that the cumulative effect on risk of all of these improbable assumptions can not be quantified does not mean that they should be ignored in a decision on whether the one in 100,000 risk level is sufficient to protect the public health. Indeed, in this case, it seems clear that the public health will not be threatened in any significant way.¹

¹ This statement should not be construed to imply that the one in 100,000 risk level will be protective for every chemical and exposure scenario. For some chemicals, the assumptions behind the risk assessments are highly probable of being correct, so that the resulting risk estimates are more likely to be correct than in the present case. Each situation should be evaluated on its own merits.

VII. DISCUSSION OF ALTERNATIVE STANDARDS

Based on the above discussions, it is clear that the State has scientifically more justifiable options than to accept the EPA recommendation on the water quality standard for dioxin. Table 3 shows the effect of incorporating better information into either a non-threshold or threshold model for dioxin.

TABLE 3 Alternative Water Quality Standards for Dioxin			
Variable or Parameter	EPA Value Proposed	Alternative Values	
		Non-threshold	Threshold
Body Weight (BW)	70 kg	70 kg	70 kg
Water Consumption (WC)	2 l/day	2 l/day	2 l/day
Fish Consumption (FC)	6.5 g/day	1 g/day	1 g/day
Fish/Water Ratio (R)	5,000 l/kg	5,000 l/kg	5,000 l/kg
Slope Factor	156,000 kg-day/mg	14,400 kg-day/mg	--
Risk Criterion	10^{-6}	10^{-5}	--
Criterion Dose	0.0064 pg/kg/day	0.7 pg/kg/day	1-10 pg/kg/day
Standard	0.013 ppq	7 ppq	10-100 ppq

Table 3 clearly suggests that the EPA recommendation is substantially more conservative than is supported by current understanding of the scientific information about dioxin. Whether or not the State decides to use the threshold model or a 10^{-5} risk criterion in a non-threshold model, a standard in the vicinity of 1.2 ppq (used in the neighboring states of Maryland and Virginia) should be protective of human health.

VII. REFERENCES

- Allen, B.M., A.M. Shipp, K.S. Crump, B. Killian, M.L. Hogg, J. Tudor, and B. Keller. 1987. Investigation of cancer risk assessment methods: Summary. Prepared by Clement Associates, Inc. for the U.S. Environmental Protection Agency. EPA/600/6-87/007a. Washington, DC: EPA.
- Allen, J.R., D.A. Barsotti, J.P. Van Miller, L.J. Abrahamson, and J.J. Lalich. 1977. Morphological changes in Monkeys consuming a diet containing low levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Fd. Cosmet. Toxicol.* 15:401-410.
- Agency of Toxic Substances and Disease Registry (ATSDR). 1988. Toxicological profile for 2,3,7,8-tetrachlorodibenzo-p-dioxin.
- Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross, and D.A. Barsotti. 1987. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 and 25 parts per trillion in the monkey: TCDD kinetics and dose-effect estimate of reproductive toxicity. Presented at Dioxin '87: Seventh International Symposium on Chlorinated Dioxins and Related Compounds, October 4-9, Las Vegas. (Abstract).
- California Health and Welfare Agency (CHWA). 1987. No significant risk levels. Article 7 of Safe Drinking Water and Toxic Enforcement Act of 1986, Chapter 3. 22 Calif. Code of Regulations Division 2. Sacramento, CA: Health and Welfare Agency.
- DeCaprio, A.P., D.M. McMartin, P.W. O'Keefe, R. Rej, J.B. Silkworth, and L.S. Kaminsky. 1986. Subchronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the guinea pig: Comparisons with a PCB-containing transformer fluid pyrolysate. *Fund. Appl. Toxicol.* 6:454-463.
- ENSR Consulting and Engineering (ENSR). 1989. Technical support document for a proposed Alabama water quality standard for dioxin. Boston, MA: ENSR.
- ENVIRON Corporation. 1989. An update on the literature on dioxin. Washington, DC: ENVIRON Corporation.
- Environmental Protection Agency (EPA). 1984. Ambient water quality criteria for 2,3,7,8-tetrachlorodibenzodioxin. EPA-440/5-84-007. Washington, DC: EPA.
- Environmental Protection Agency (EPA). 1989. Exposure factors handbook. EPA/600/8-89/043. Washington, DC: EPA.

Hong, R., K. Taylor, and R. Abonour. 1987. Immune abnormalities associated with chronic TCDD exposure in rhesus monkeys. Presented at Dioxin '87: seventh International Symposium on Chlorinated Dioxins and Related Compounds, October 4-9, Las Vegas. (Abstract).

International Commission on Radiological Protection (ICRP). 1984. Report of the task group on Reference Man. ICRP Publication No. 23. New York: Pergamon Press.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreson, C.E. Wade, D.A. Dittenber, P.O. Kalmins, L.F. Franson, P.N. Park, S.D. Barnard, P.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-TCDD in rats. *Toxicol. Appl. Pharmacol.* 46:279-303.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, and P.J. Gehring. 1979. Long-term toxicologic studies of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in laboratory animals. *Ann. NY Acad. Sci.* 320:397-404.

National Council of the Paper Industry for Air and Stream Improvement (NCASI). 1987. Dioxin: A critical review of its distribution, mechanism of action, impacts on human health, and the setting of acceptable exposure limits. Technical Bulletin No. 524. New York, NY: NCASI.

National Council of the Paper Industry for Air and Stream Improvement (NCASI). 1989. Biological risk assessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin. Technical Bulletin No. 567. New York, NY: NCASI.

National Toxicology Program (NTP). 1982. Bioassay of 2,3,7,8-tetrachlorodibenzo-p-dioxin for possible carcinogenicity (gavage study). DHHS Publ. No. (NIH) 82-1765.

Rodricks, J.V., S.M. Brett, and G.C. Wrenn. 1987. Significant risk decisions in federal regulatory agencies. *Reg. Toxicol. Pharmacol.* 7:307-320. SRI International (SRI). 1980. Seafood Consumption Data Analysis. Report prepared by SRI International for the U.S. Environmental Protection Agency. Menlo Park, CA: SRI.

Roberts, Leslie. 1991. Dioxin risks revisited. *Science* 251:624-626. February 8.

Rozman, K. 1989. A critical view of the mechanism(s) of toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Implications for human safety assessment. *Dermatosen in Beruf und Umwelt* 37:81-92.

Sauer, Robert M. 1990. Pathology Working Group: 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Sprague-Dawley rats. Report prepared by PATHCO, Inc. for the Maine Scientific Advisory Panel. March 13.

Sherman, William R. 1991. Letter to Philip M. Cook, US Environmental Protection Agency dated April 22. S.D. Warren Company.

SRI International (SRI). 1980. Seafood Consumption Data Analysis. Report prepared by SRI International for the U.S. Environmental Protection Agency. Menlo Park, CA: SRI.

Sparschu, G.L., Jr., F.L. Dunn Jr., and V.K. Rowe Jr. 1971a. Study of the teratogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the rat. *Fd. Cosmet. Toxicol.* 9:405-412.

Sparschu, G.L., F.L. Dun, R.W. Lisowe, and V.K. Rowe. 1971b. Effects of high levels of 2,4,5-trichlorophenoxyacetic acid on fetal development in the rat. *Fd. Cosmet. Toxicol.* 9:527-530.

Squire, R.A. 1990. Letter from Robert A. Squire to Robert A. Michaels, RAM TRAC Corporation, dated January 8.

Travis, C.C., S.A. Richter, E.A.C. Crouch, R. Wilson, and E.D. Klema. 1987. Cancer risk management: A review of 132 federal regulatory decisions. *Environ. Sci. Technol.* 21:415-420.

Universities Associated for Research and Education in Pathology, Inc. (UAREP). 1988. Human health aspects of environmental exposure to polychlorinated dibenzo-p-dioxins and polychlorinated dibenzo furans. Bethesda, MD: UAREP.

World Health Organization (WHO). 1990. Working Group on Dioxin. Press Release. December 7.

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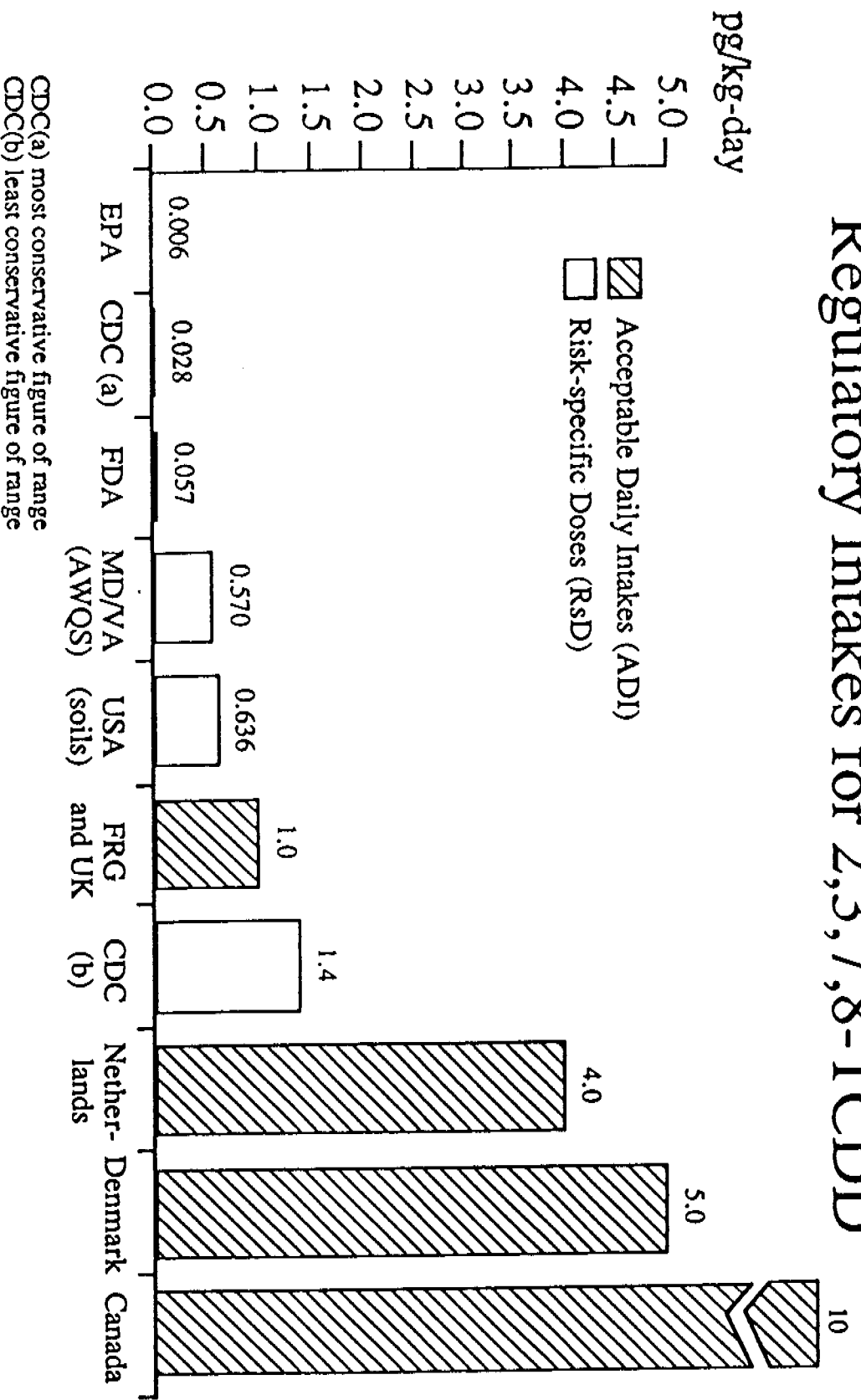
ALTERNATIVE WATER QUALITY STANDARDS FOR DIOXIN

	Non-		
	<u>EPA</u>	<u>Threshold</u>	<u>Threshold</u>
<i>Fish Consumption (g/day)</i>	6.5	1	1
<i>Fish/Water Ratio (l/kg)</i>	5000	5000	5000
<i>Slope Factor (kg-day/mg)</i>	156000	14400	--
<i>Risk Criterion</i>	10^{-6}	10^{-5}	--
<i>Criterion Dose (pg/kg/day)</i>	0.0064	0.7	1-10
<i>Standard (ppq)</i>	0.013	7	10-100

BW = 70 kg; WC = 2 l/day for all cases

ENVIRON

Regulatory Intakes for 2,3,7,8-TCDD



TESTIMONY BEFORE THE
WV WATER RESOURCES BOARD
ON THE SETTING OF WATER QUALITY STANDARDS

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on behalf of

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These comments are relevant to the discussion of setting a standard for dioxins, but are intended to apply to regulation of water pollutants in general. I am professionally interested in issues of science and public policy, that is, how policy makers such as the Water Resources Board can use science to make policy decisions, and, more importantly, how policy makers can deal with situations where the available science is inadequate. Even when adequate science is available, public policy must involve more than scientific arguments, but the situation is especially tricky in the case of pollutants like dioxins where a scientific consensus does not exist and is not likely to exist in the near future.

While I won't attempt to define science here, let me point out three requirements of any discussion needed for it to qualify as scientific.

First, there are no pre-ordained conclusions.

Second, if the data is inadequate, there is no need to come to a conclusion, but rather it is appropriate to wait for further data.

Third, the parties involved must be honest in their use of the information that does exist.

A policy making group like the WRB is inevitably confronted with situations in which all three requirements are violated, yet you are required to make decisions anyway. It is perfectly possible

for an industry to first decide how much of a pollutant they
planned activity will release, and then try to come up with a
rational activity that will fit their needs. Not only is it
possible, it has become standard practice. Thus my first
requirement for a scientific argument is routinely violated, as
industries hire scientists and lawyers to concoct scientific
sounding arguments to defend what they want to do. Note that I am
not saying this is dishonest, just that it is not science, because
the result was chosen ahead of time.

This approach is only believable when the data is inadequate,
making a wide range of possible solutions. This violation of my
second requirement shows that the Board is in the difficult position
of having to make a decision that ideally would be based on science
alone, but, in fact, cannot be. The science just isn't there.
Interested parties can, and have, come up with plausible arguments
for much higher or lower standards, all based on the little evidence
that exists. I will suggest below that in some cases, for example
this one, it is possible to invoke more general principles using
what is known to make a decision.
But let me mention that, unfortunately, my third requirement,
of honest by the discussants, is also a problem. Some of those
for a weak standard have attempted to use the now infamous
conference to give the illusion of a scientific consensus on
ter. I'm sure you are aware of the dishonest attempts to
portray that conference in a press release written by vest
(without the knowledge of the participants (see
)). Sadly, complex and ambiguous scientific
for unethical but smiling consultant
seems that the WRB will need to
arguments to come to a decision
course what the
all what

responsible policy? I will argue that that this is such a case.

First, consider a classic water pollutant for which a standard can be set, not just because adequate scientific knowledge exists, but because of the nature of the pollutant itself, namely BOD. It makes sense to set a particular water standard for BOD because:

1. It is always present at levels that make it part of the natural system, and non-toxic.
2. It quickly bio-degrades.
3. It is not bio-accumulative.

Because of these three characteristics it is possible to reach a scientific consensus on a standard for BOD that will protect the waters of the state. Note that dioxin fails on all three of these criteria: it does not naturally occur and is highly toxic, it is slow to degrade, and instead bio-accumulates.

Recall one of the fundamental goals of the clean water act itself: the water of the nation should be fishable. Because of dioxin accumulating in fish tissue, there are advisories in several tributaries to the Ohio River already, in some cases the source of the dioxins is not even known. Allowing any dioxin at all to be dumped into any river would only make this worse and there is no excuse for allowing it.

I would like to ask the WRB to recognize that in cases of chemicals like dioxin, setting an allowable standard above zero does not constitute sensible public policy.

Several objections will arise such as the problem of detection. But the whole point of my argument is to try to prevent the pollution in the first place rather than focus on what we can detect once it is too late. The focus of public policy must not just be on how much pollution we should accept, but rather on how to prevent it in the first place.

Let me illustrate with the history of the six criterion air pollutants and how public policy has worked (and not worked) with them. The clean air act took the same standard setting approach, focusing on what level of these pollutants is acceptable, with one exception: lead. Lead is different from the other criterion air pollutants in the same way that dioxin is different from BOD. So as

public policy we treated lead differently; we tried to prevent its entry into the environment in the first place by banning its use in gasoline, the main source. The industries using lead claimed that it couldn't be done. But of course it could, and it was.

So what has happened to levels of the six criterion air pollutants? Levels of lead have dropped dramatically, while levels of the others have remained at pretty much the same levels as in the past. Only particulates have decreased significantly, this largely due to the death of the steel industry.

This example shows that there are cases where the only sensible public policy is to prevent pollution in the first place. In the case of chemicals like dioxins the sensible thing to do is to try to prevent the use of technologies that produce it in the first place. The industries that produce dioxins will simply have to change. It is perfectly possible to produce paper products without using chlorine chemicals, thus eliminating the major source of the dioxins. The need to establish zero standards in some cases should be recognized by the Board and implemented in the case of chemicals like dioxins.

Flap Erupts Over Dioxin Meeting

Everything about dioxin is so politically charged, even the science, that the organizers of a Banbury Center dioxin conference last fall expected their meeting would generate some controversy. But no one anticipated the furor that erupted in early February when participants learned that a public relations firm hired by the Chlorine Institute, which helped pay for the conference, was circulating a "consensus" summary of the meeting. The document, not surprisingly, supports the industry line on dioxin.

"I'm outraged," says Ellen Silbergeld, a toxicologist at the University of Maryland, who insists that the press materials—which none of the participants approved—misrepresent her views. "I agreed to participate based on my previously held high regard for Banbury and Cold Spring Harbor," she wrote in a letter to Banbury Center director Jan Witkowski. "I did not expect to be manipulated by industry or government spokespeople." Silbergeld sent copies of her letter to all the participants and to the press.

"It was basically mishandled," sighs Witkowski, who admits to a degree of naïveté in dealing with such a "highly politicized" issue as dioxin. He has just written a letter to all the meeting participants explaining that the Banbury Center in no way authorized the press materials that were sent out. "I am very sorry that what was intended to help promote the goals of the meeting has gone so awry," he wrote.

The intensity of Silbergeld's reaction may say more about the extreme sensitivities over dioxin than about the culpability of any of the parties involved, all of whom have taken some heat. The flap nevertheless illustrates what can go wrong when the roles and agendas of sponsors and participants aren't clearly spelled out.

What Silbergeld, who was formerly with the Environmental Defense Fund, finds most disturbing about the events is the "violation of process." And she has found an unexpected ally in Vernon Houk, director of the Center for Environmental Health and Injury Control at the Centers for Disease Control. Houk is in almost total disagreement with Silbergeld on the dangers of dioxin—he believes that they have been vastly overrated. Yet Houk told *Science* that he, too, is "disturbed" by the publicity campaign. "I don't think it is fair to

represent consensus when none was really sought," says Houk. Silbergeld's view is also supported by a number of the other participants, albeit with somewhat less emotion, who say they feel manipulated and misused.

When the Chlorine Institute, an industry trade group, approached Banbury director Jan Witkowski early last year about holding a dioxin meeting, he had no inkling it would turn out differently from the 15 or so other meetings the center runs each year, some of which are also sponsored by industry. And to the Chlorine Institute, the time was ripe for another look at dioxin's risks, given shifting sentiments among at least some scientists that dioxin may be less dangerous than previously believed. Chlorine Institute officials thus believed that a scientific meeting could be "beneficial to our interests, particularly our interest in the paper industry," explains Joe Walker, the institute's head of communications. Walker is referring to the enormous pressure the paper industry is now under to reduce the amount of dioxin that arises as a by-product in the bleaching process.

The Chlorine Institute lined up Robert Scheuplein of the Food and Drug Administration to run the meeting. He then asked Michael Gallo, a toxicologist at the Robert Wood Johnson Medical School in New Jersey, and Cornelius A. van der Heijden, a regulatory official in the Netherlands, to chair the meeting with him. From then on, the Chlorine Institute studiously kept itself out of the picture, say both Witkowski and the meeting organizers.

The three organizers picked all participants, with one exception: George Carlo, who was invited as an observer for the institute at its request. Carlo, an epidemiologist and lawyer who heads the Health and Environmental Sciences Group in Washington, D.C., is a regular consultant to the institute.

The meeting itself went surprisingly well; indeed, some agreement was reached by people from opposite sides of the dioxin debate on a number of issues. For example, there was nearly unanimous agreement that, in light of a new understanding of dioxin's molecular actions, the Environmental Protection Agency should take another look at how it assesses the risk of this chemical. Several people, most notably the meeting organizers, speculated

**"I am very sorry
that what was
intended to
help promote
the goals of the
meeting has
gone so awry."**

—JAN WITKOWSKI

that such a reexamination would reveal that EPA overestimates how risky dioxin is—though others, like Silbergeld, rejected that idea (*Science*, 8 February, p. 624).

But the harmony was to prove short-lived. In retrospect, most participants with whom *Science* spoke say they failed to realize that the meeting was sponsored by industry, along with the EPA, even though the invitation clearly said so. And none of the participants *Science* spoke with, nor even meeting co-organizer Gallo, knew Carlo was there as the institute's observer. Silbergeld, Houk, and others say they have no problem with industry sponsorship of scientific meetings—as long as everyone's role is clear.

At the meeting Carlo heard the message the Chlorine Institute hoped he would. The institute's intention, from the outset, was that "if the conference outcome was favorable we would take advantage of it and bring it to the attention of key people in the media," says Walker. They hired Edelman Medical Communications to do just that.

Witkowski saw no problem with the institute publicizing the results of the meeting—after all, they paid for half of it—as long as it was well done. But what he thought would be a straightforward press release turned into a fiasco of crossed signals and miscommunication. For example, Gallo, Scheuplein, and van der Heijden were delighted to cooperate—and indeed, each wrote his own one-page summary of the meeting for the public relations firm. But Gallo and Scheuplein now complain that they had assumed that Edelman was representing the Banbury Center, not the Chlorine Institute.

In addition, Witkowski's only condition in agreeing to the press release was that he have final approval. As agreed, the Edelman staff sent him the cover letter to the press, which he edited to remove any references to the Cold Spring Harbor Laboratory. But in December, Edelman sent out a press packet that included, along with the statements from the three chairmen alluded to in the letter, a background paper purportedly written by Carlo that none of the organizers or participants had seen. The paper asserted, among other things, that the Banbury meeting "reinforced the notion that dioxin is much less toxic to humans than originally believed."

The cover letter also differed from the version Witkowski had approved in two important respects. First, it referred to the meeting



No consensus. Ellen Silbergeld's objection to the press release won support from other participants.

as a "consensus conference." Says Witkowski: "Just those two words set the whole framework. It was not a consensus conference. That is what is causing all the trouble." What's more, the draft he approved carried a disclaimer, which was missing from the final version, saying that the three statements were the views of the chairmen and not the conference as a whole. Meeting organizers Gallo and Scheuplein also object to the letter—essentially to the two offending words—and insist that they never saw it.

When Silbergeld got hold of the press packet from a reporter at the end of January, she exploded, writing a letter to Witkowski that took all of the parties to task. Events then took a bizarre twist when Carlo, who received a copy of Silbergeld's letter, called Witkowski to say he had not written the paper after all and had no idea how his name ended up on it.

Carlo has since launched a massive telephone campaign—calling the participants and this reporter repeatedly—to clear his name. Carlo concedes that he did work with Edelman and the Chlorine Institute in developing the paper but asserts, nonetheless, that, "No one has the right to put someone's name on a document."

Nancy Turett, senior vice president at Edelman, admits to putting Carlo's name on the paper—she says because he was so extensively involved in drafting it. "The end product is very much a reflection of what Dr. Carlo thought should be in it," says Turett, who says she assumed that the Chlorine Institute had cleared the final document with Carlo. Institute officials say, meanwhile, that they thought Edelman had gotten Carlo's permission.

Turett also takes the blame for the revised letter, calling it a simple editorial mistake. "I thought there was a consensus. There is no big agenda. It is just a cover letter." She called *Science* back to add, however, that Edelman never distributes a single word without the client's approval.

All of which leaves Witkowski at the Banbury Center shaking his head in disbelief. He realizes, much to his dismay, that this flap threatens to "poison" the entire meeting and undermine the fragile—and unexpected—agreement the group forged on the molecular action of dioxin. "That is the kind of thing you would hope would happen at a scientific meeting," says one participant who asked to remain unnamed. "It was like a little flower just starting to bloom, then along came this mess. I feel very sad." ■ LESLIE ROBERTS

Happy Birthday WV June

Report to WV Water Resources Board on
Water Quality Standards for Dioxin

Certainly the question of a safe level for dioxin in our waters is complicated by its very nature and because of politics and economics. The Water Resources Board had a 5" stack of zeroxed material on it when I reviewed the submissions two weeks ago. Certainly I can't cover the whole subject tonight, but I'd like to make a few comments on several topics. All my references to various studies are from the stack of material in the Water Resources Board's file.

Toxicity
mediator vs initiator

While it appears that scientists are beginning to come to some agreement that dioxin acts more like a mediator rather than a initiator of cancer, there is by no means agreement that the threshold model for a lower limit is valid. The Chlorine Institute and the EPA hosted a conference on dioxin at Cold Spring Harbor Laboratories. After its conclusion a spokesman for the Chlorine Institute said that the scientists had agreed on a model for the toxicity of dioxin that allows for the presence of the substance in the environment, with no risk experienced below a certain level of exposure. This statement is extremely misleading if not an outright lie. Many of the conferees were extremely upset by this false conclusion. Yet this statement appears over and over again in the material Industry supplied to the WRB.

NO threshold nature of dioxin does NOT imply

Bioaccumulation

The EPA has based their allowable level of dioxin in rivers on a bioconcentration factor of 5000. That is the Dioxin levels inside fish are 5000 times greater then in the water around them. But recent experiments by the Environmental Research Laboratory in Duluth Minnesota have found concentration levels of 50,000 to 140,000. In the great lakes some fish are so contaminated that eating one fish from the lakes would put you over your yearly dosage, despite the fact that the level of dioxin cannot be measured in the lake waters.

1/4 *
w?
Fish consumption is a good place ^{you} ~~to give~~ to give an idea of industry's shameless attitude. In trying to justify an increased level of dioxin in our nations waters, reports submitted by the WV manufacturers and various other industrial lobbies, attack from every angle the idea that people get much dioxin from fish. Most people assume that the main manner by which we absorb dioxin is through fish consumption, although a Canadian study (Birmingham 1990) found that only about one half of our dioxin in our bodies comes from fish. The EPA says that the average citizen eats 6.5 grams of fish a day (7 ounces a month). A report of a survey of anglers in Maine found that some sub populations like Native Americans and other ethnic groups average 5 times as much and that some individuals eat 20 to 30 times as much as this. These people are, by this factor alone, that many times

more likely to get cancer from dioxin ~~cancer~~. So industry says things like this; only 25% of the 6.5 grams of average daily consumption of fish is from dioxin laden streams (suppose someone's fish all comes from the same stream or that finally all streams are degraded to the industrial level), that sediments are a sink for dioxin therefore lowering concentrations in fish (but how about bottom feeders); that if fish eaters don't eat any of the fat of the fish, then dioxin concentrations can increase a hundredfold more (but suppose you fry up the fish and use the grease as part of the meal).

All this juggling by industry, so they can pollute and make a bigger profit, cuts down on the options we have on how much fish we eat, where we can fish, even how we cook our fish. I feel a great revulsion at all the fact manipulation and verbal footwork that these corporations and their hired guns are doing, none of which is sound science, to further degrade our waters.

Water consumption

Although drinking water is a minor pathway for dioxin to enter our bodies, the way industry handled this factor is another eye opener. The Army and the EPA give out water consumption as 2 liters a day and the EPA bases a small part of their water ambient levels for dioxin on this amount. Granted many people do not drink this much water, but some people do. I know I drink twice this much working outside on hot summer days. A consultant for Industry spent a page trying to put forth an argument that the drinking water level should be 1.2 liters. So because of their greed and disregard for the environment, many people will have to cut back on their water consumption or buy some product like bottled spring water or soda from them.

Aquatic life The EPA says there is insufficient data to determine a safe level for aquatic life. Until we know we should err on the safe side, the wild critters are already under heavy assault by us humans.

Mothers milk and effects on fetal development

The EPA has found dioxins in the bodies of everyone they tested. They found that mother's milk has 11,000 times the acceptable level. Babies nursing off mother's who principal protein is fresh water fish could potentially be getting thousands of times this level. Ellen Silbergeld, a staff scientist with the EDF, states that dioxin alters hormone levels which then disrupt development of the immune system and interferes with fetal and child development. Studies with rhesus monkeys exposed to dioxin prenatally and through mother's milk later had behavioral and mental difficulties. Considering its bioaccumulation potential, its persistence in the environment and the body, and for the sake of the children we should set a zero discharge level for dioxin.

other dioxins, accumulative effects

There are over a hundred dioxins and furans, although none may be as carcinogenic as TCDD, they are all very poisonous and have other health effects. We should also set standards for all these

and consider their synergistic effects. Suppose TCDD is only a mediator, but in combination with some furan that is an initiator it could have very powerful effects.

Subpopulations consisting of breast fed babies, native Americans and others that depend on fish for survival, developing children and fetus' ~~X~~chemically sensitive people need special protection and the level of dioxin allowed in our waters needs to be low enough so that these people are not endangered. West Virginia being a rural state has many people who fish in our waters and eat the fish on a regular basis. The EPA in the memorandum of JAN 5 1990 has allowed the states to set more stringent levels if certain people in the state eat more the 7 ounces a fish a month. I suggest that the WRB does this.

Acceptable risk

The EPA lets states set a risk factor of between one new cancer death in 100,000 to one new death in 10,000,000. Industry is asking for the more deadly level, so they can pollute ten times as much with "only" 9 more deaths per million. One Industry submission on this particular figure uses garbage computer runs to show how even the one in 10,000 new deaths is below a "background" cancer death rate fluctuation, and therefore was acceptable, because it was not noticeable in the computer simulation. As a mathematician I find this abuse of numbers extremely disturbing. Increasing the risk assessment by ten or hundredfold means exactly that, no matter how you try to manipulate the figures. I hope we in West Virginia can provide the highest protection via the 1 in 10 million risk factor, although this would mandate a level for dioxin ten times as stringent as the EPA's present standard. In no case should we settle for the 1 in 100,000 risk factor.

EPA future study. A memorandum in the WRB's files by EPA director William Reilly called for a year long reassessment of TCDD toxicity, and that at this time it was impossible to say if it was more or less toxic then previously stated. Until this study is completed it would be a grave error to set lower standards for this tremendously toxic material.

Submitted by
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Dioxin levels not measurable in Rivers
But they are already in the water, detected in sediments &
Rivers - so how can we measure or tell if new sources
of dioxin will push the level ~~of~~ of contamination
above acceptable levels

Harmonic Mean Flow

LEAGUE OF WOMEN VOTERS
OF
CHARLESTON AREA, WEST VIRGINIA

June 25, 1991

This is the written version of the League's
comments on the 1991 Water Quality
revisions. I forgot to leave them with the
board, so here they are now.

Conni Grady Lewis
LWVWV



LEAGUE OF WOMEN VOTERS OF WEST VIRGINIA

June 20, 1991

STATEMENT TO THE WEST VIRGINIA WATER RESOURCES BOARD ON THE PROPOSED REVISIONS TO WEST VIRGINIA WATER QUALITY STANDARDS

The League of Women Voters of West Virginia wishes to comment on the proposed standard for 2,3,4,8 - TCDD (Dioxin).

There are various points of view concerning the appropriate standard for TCDD. The League believes that in the absence of conclusive data to prove otherwise, EPA's standard is the most defensible.

The League also believes that any company which discharges dioxins should be required to monitor in addition to the discharge waters, the receiving sediments and suspended particulates and conduct bio-monitoring. In order to insure Quality Assurance/Quality Control, laboratories used to analyze the samples should be EPA certified for low level dioxin tests. The program should require frequent testing.

Because dioxins are persistent in the environment and have already been found in a tributary of the Kanawha River, we believe that they should be strictly controlled. Bio-accumulation of dioxins are already a problem in the Kanawha River. Dioxins are also present in sludge from pulp bleaching and from wastewater treatment at pulp mills. By landfilling or spreading them on the land they can pose long-term threats to the environment. It would be more provident to prevent future problems than to try to remediate them.

By setting EPA's standard now, a company can plan its facility with processes appropriate for meeting the standard rather than installing expensive equipment later if standards should be made tighter. Pulp mills are using manufacturing processes that can meet EPA's standards. We believe that pulp mills in West Virginia should use state of the art processes to meet environmental concerns. The League also believes that the public will gradually accept many paper products that do not require bleaching, a process which causes many of our environmental problems.

Karen Lukens, President, League of Women Voters of West Virginia
1127 Montrose Dr., South Charleston, WV 25303

Helen Gibbins, Natural Resources Chair, LWV of West Virginia
6128 Gideon Rd., Huntington, WV 25705

Conni Lewis, Natural Resources Committee, LWV of West Virginia
4406 Venable Ave., SE, Charleston, WV 25304

ALABAMA PULP & PAPER MILL HEARING, CHARLESTON, W.V.

JUNE 20, 1991

I am Keith Taylor state president of the Izaak Walton League in West Virginia. Since 1922 the Izaak Walton League of America has fought for clean water. In fact, the league made the nations first water pollution survey back in 1927 for President Calvin Coolidge.

When the league was founded by 54 Sportsmen and Conservationists in 1922 in Chicago, they chose to name it Izaak Walton who history notes to be one of the first conservationists in England in the 1500's.

It seems ironic that Izaak Walton of Jolly Ol' England is here to take a strong positive position with Parson and Whittemore of Jolly Ol' England.

Alabama Pulp and Paper Co., proposes to build a plant on 1300 acres in Mason County about half way between Pt. Pleasant and Huntington on the Ohio River. If built it would be the largest paper mill in the nation.

Now we all know that we need industry in West Virginia, and we want to welcome each and everyone, providing our environmental laws set down by our state and federal governments are adhered to. We seek and want good citizens to do business in our state, that will protect our environment.

One of the leagues main concerns here is the dioxin discharge that will be made into the Ohio River, which may cause cancer in human beings. Another concern is the odorous smell from the exhaust stacks of paper plants that will unquestionably be released by this operation. We oppose anything that fowls fresh air.

Now back in June 1989 Luigi Terziotta the General Manager of the Alabama Pulp and Paper Co., which is owned by Parson and Whittemore, says this mill will use the latest technology from Scandinavia, and the water discharge will not emit dioxins detectable at one part per quadrillion (a millionth of a billionth). He stated this mill will operate and I quote "with no detectable discharge of dioxin" if built in Mason County.

Page 2

As far as the odor goes Mr. Terziotta said and I quote "we don't say no odor, we say low odor," unquote. Old mills use sulphur compounds to cook wood. This new mill would not use that process or have that smell so states Mr. Terziotta.

Now in May 1991, at a Charleston meeting Alabama Pulp officials proposed raising the standard for dioxin emission by a factor of 100, on the above suggested Federal Guide Lines if you will.

Now really ladies and gentlemen if we are to believe the plant manager Mr. Terziotta that dioxin would be virtually non detectable, why would Alabama Pulp officials try to get West Virginia to raise the dioxin emission standard by a factor of 100? Who is kidding who here? If standards are lowered could more profits be made? Does West Virginia look like a soft touch by a new industry and lower standards are sought as a prerequisite to obtain this facility.

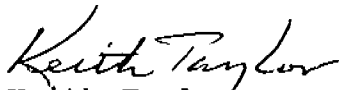
Something is amiss too when on June 7, 1991, the Water Resource Section of our Department of Natural Resources was asked to suspend the application it filed for a federal permit necessary to discharge plant water into the Ohio River. Was this done to bring more pressure on our Department of Natural Resources or has Alabama Pulp & Paper decided to be a good citizen as Mr. Luigi Terziotta said they could be, regarding dioxin discharge.

The Izaak Walton League will support safe and environmentally sound industries to come into West Virginia. We don't need or want an industry that operates through loop holes. We will support an industry that is honest and abides by both the state and federal environmental laws.

The time has come in the United States of America, that no industry in any state will operate outside laws laid down by the state and federal government.

Ladies and gentlemen that is what we ask in this paper mill case. Change no regulations, and ask that they abide by all set regulations of water and air discharge presently in force.

Thank you,



Keith Taylor
West Virginia Division President

O.V.E.C.

Ohio Valley Environmental Coalition

P.O. Box 970
Proctorville, OH 45669

June 20, 1991

Dr. Jan Taylor
West Virginia Water Resources Board
1260 Greenbrier Street
Charleston, West Virginia 25301

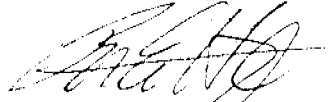
Dear Dr. Taylor,

Enclosed are comments prepared by the Ohio Valley Environmental Coalition (OVEC) with regards to the proposed 1991 West Virginia Water Quality Standards.

OVEC is a non-profit, tax-exempt environmental organization based in the West Virginia, Ohio, Kentucky Tri-State region. OVEC's primary activities focus on toxics and air and water pollution in the Ohio Valley bioregion.

OVEC would like to thank the Water Resources Board for the opportunity to comment on the proposed regulations. Should you need further information or have questions regarding OVEC's comments, please contact me at (304) 525-4803.

Sincerely,



Brian E. Hagenbuch
OVEC Project Director

encl.

COMMENTS REGARDING THE 1991 PROPOSED WEST VIRGINIA
WATER QUALITY STANDARDS

WEST VIRGINIA WATER RESOURCES BOARD
PUBLIC HEARING
JUNE 20, 1991

COMMENTS PREPARED AND SUBMITTED BY:
OHIO VALLEY ENVIRONMENTAL COALITION, INC.

P.O. Box 970
Proctorville, Ohio 45669

For more information, contact:
Brian E. Hagenbuch, Project Director (304) 525-4503

COMMENTS REGARDING THE 1991 PROPOSED WEST VIRGINIA

WATER QUALITY STANDARDS

Respected members of the Water Resources Board (WRB), the Ohio Valley Environmental Coalition (OVEC) would like to thank you for the opportunity to comment on the proposed 1991 West Virginia water quality standards (46 CSR 1 Title 46 Legislative Rule Series 1). OVEC is a non-profit, tax-exempt environmental organization based in the West Virginia, Ohio, Kentucky Tri-State region. OVEC's primary activities focus on toxics and air and water pollution in the Ohio Valley bioregion.

BACKGROUND

OVEC supports the WRB use of the EPA estimated risk level of one additional cancer death per million persons (10^{-6}). This is in accordance with Section 46-1-8.2.a and 46-1-8.2.b. It is OVEC's opinion that 10^{-5} risk levels should be the least stringent applicable in setting standards, and where shown to be scientifically defensible, more stringent standards should be applied.

While several groups such as the Dioxin Working Group have argued for using a risk level of 10^{-5} , lowering the dioxin standard with this criteria would be strictly for policy purposes and not based on scientific credibility.¹ Chemrisk, an industry consultant group, uses 10^{-5} as a basis of policy to weaken a standard and is not based on science.^{2,3} As it relates to dioxin, only eight states use the 10^{-5} standard of 1.2 ppq and in two, Maryland and Virginia, the EPA is being sued for permitting the relaxation of the standard. Fifteen states use a standard between 0.01 and 0.1 ppq and five states have standards more stringent than 0.01 ppq.

I. Status of water quality in West Virginia

Public health advisories have been placed on the consumption of fish in the following seven rivers (with mileage); Ohio (277), North Branch of the Potomac (51), Kanawha (46), Potomac (38), Shenandoah (19), Pocatalico (2) and Armour Creek (2).⁴ In each of the rivers except the Ohio and Shenandoah (139 total miles), the cause for the advisory has been dioxin contamination in fish tissues. In the North Fork of the Potomac and Potomac rivers (89 total miles), the dioxin was attributed to the Westvaco pulp mill in Luke, Maryland. Advisories were placed in the Ohio river because of chlordane and PCB levels, and in the Shenandoah for PCB's. Many of these rivers are popular for recreational activities such as fishing and boating, yet do not meet their designated uses.

According to the Section 305(B) relative assessment of causes for waterbodies not fully supporting uses, priority organics had a major impact on 149.4 stream miles and moderate/minor impact on 331.5 stream miles.⁵ Chlorine had a major impact on 91.1 stream miles and moderate/minor impact on 459.6 miles.

Table 1 represents 2,3,7,8-tetrachlorodibenzo-p-dioxin (herein referred to as TCDD or dioxin) levels in fish fillets.⁶ TCDD levels in channel catfish, bottom feeders, ranged from 2 ppt (parts per

trillion) near St. Albans to 45 ppt at Armour Creek. For game species, values ranged from 1 ppt at Institute to 18 ppt at the Pocatalico river. FDA guidelines for TCDD levels in fish fillets is 25 ppt for women and children.

In sediment core samples from two Kanawha river tributaries, TCDD levels ranged from <1 ppt to over 3044 ppt (Table 2).⁷ In sediments from the Kanawha river, TCDD levels ranged from <1 ppt to 100 ppt (Table 3).⁸

In 1990, TCDD levels were analyzed in channel catfish and bass in the Ohio river at several locations (Table 4).⁹ Channel catfish had TCDD levels ranging from 2.8 ppt at Willow Island to 9.2 ppt in Huntington.

II. Regulatory authority on the Ohio river

West Virginia is a signatory state in the Ohio River Valley Water Sanitation Compact, hereby referred to as ORSANCO. ORSANCO is an interstate compact water pollution control commission established in 1948 and comprised of representatives from all states contiguous to the Ohio river. ORSANCO has the vested authority to promulgate water quality standards for the treatment of sewage and industrial wastes into the Ohio to assure multi-purpose uses of the river for all affected states. The principle guiding development of these standards is that pollution "originating within a signatory state shall not injuriously affect the various uses of the interstate waters..."¹⁰

West Virginia is responsible for regulating water discharge rates into the Ohio river based on standards promulgated, not by the State of West Virginia, but by ORSANCO. Article V (Wastewater Discharge Requirements) of ORSANCO's 1990 pollution control standards for discharges into the Ohio River, states that:¹¹

No discharge of any Sewage, Industrial Wastes, Toxic Wastes, Other Wastes . . . shall cause or contribute to a violation of these (ORSANCO'S) Wastewater Discharge Requirements . . ."

Furthermore, Article V, Section C (Industrial Wastes, Including Toxic Wastes) states that:

The minimum level of treatment for Industrial Wastes including Toxic Wastes, prior to discharge shall be in accordance with national effluent limitations and guidelines adopted by the Administrator of the United States Environmental Protection Agency pursuant to Sections 301 and 302 of the Federal Clean Water Act, national standards of performance for new sources adopted pursuant to Section 306 of the Federal Clean Water Act, and national toxic and pretreatment effluent limitations, adopted pursuant to Section 307 of the Federal Clean Water Act."

While variances are permitted under ORSANCO regulations, no blanket variances exist. It is not the state's responsibility to petition for the variance but that of the toxic waste discharge facility to provide a scientifically-defensible argument that the discharge standard should be made less stringent than EPA standards.

While ORSANCO does not permit standards less stringent than EPA Clean Water Act standards, nothing precludes a state agency from setting a more stringent standard for discharges.

III. Dioxin

A. Health Effects--Human Considerable arguments rage between industry, government, academicians and environmentalists over the toxicity and hazards of dioxin. Despite the debate, dioxin is by far the most potent animal carcinogen ever tested, most potent teratogen (embryonic defects) known, and may be the most potent chemical in producing toxic effects on the human immune and reproductive systems. Some studies reveal a connection between dioxin exposure and non-cancer health effects including heart attacks, digestive diseases, cirrhosis of the liver, gall bladder and biliary tract diseases and genitourinary diseases.¹²

Some studies have indicated that dioxins are longer lived, more toxic and have higher bioaccumulative potential than DDT or PCB's.¹³ Dioxin is toxic to a wide variety of animals, from rats to rhesus monkeys. Dioxin's close relatives, PCB's and dibenzofuran, may cause birth defects in monkeys and learning/behavioral changes in children of exposed women.¹⁴

Recent studies of German and American workers exposed to dioxins found statistically significant increases in cancer rates.¹⁵ In a study of 5172 chemical workers, Fingerhut found nine times the normal amount of soft tissue sarcoma--a malignant cancer of soft connective tissues.¹⁶ The same group had 42% more cancers of the respiratory tract. The death rate for workers was 2 individuals per 1000, well above the EPA "acceptable" level. While the authors admit that other chemicals may be involved, the evidence must be taken seriously as a flag of probable human risk.¹⁷

While scientists have not conclusively determined whether dioxins are initiators or promoters, people are exposed to multiple initiator and promotor chemicals in the environment. If dioxin is a potent cancer initiator, the relevant statistic would be the highest single-day consumption of fish, not the average consumption. Scientists have not been able to determine the latency period between dioxin exposure and onset of cancer.^{18,19} Dioxins may also interact with other environmental pollutants in a synergistic pattern. Studies have shown that certain PCB's may enhance the toxicity of TCDD.²⁰

Despite the deadly nature of dioxins, their presence is ubiquitous. Detectable dioxins in the human body have been found in 100% of the U.S. population with 75% of the population having detectable levels of TCDD.²¹ The EPA estimates the average American ingests 150 to 500 times the EPA acceptable dose on a daily basis.²² EPA also estimates that nursing babies can be exposed to dioxins 11,000 times above the acceptable daily amount. This data further affirms the potential health risks regardless of whether dioxin is an initiator or promotor.

B. Health effects--previous studies Primarily sponsored by industry trade groups, previous studies found no increase in cancer rates among chemical workers exposed to dioxins. These studies, however, have been found to be faulty and possibly fraudulent.²³ Dr. Cate Jenkins, an EPA chemist, determined that Monsanto falsified data used to support the claim that dioxin does not cause

cancer in humans.²⁴ EPA, itself, relied on Monsanto's fraudulent data in setting health standards for dioxin.

Industry trade groups assert that "new" scientific evidence shows that dioxin is far less dangerous than previously imagined.^{25,26} Despite these assertions, the new information is actually a reinterpretation of the 1978 rat experiment that forms the basis for the EPA's current estimate of dioxin's ability to cause cancer.

A proposed acceptable daily dose of TCDD is claimed to be based on new science regarding the reclassification of tumors. If all other assumptions remain the same, recounting tumors according to the revised rules would result in an "acceptable" daily dioxin dose that is only 2-3 times larger than current estimates.²⁷ Industry groups have admitted as much.^{28,29} This is an insignificant change given the uncertainty in risk assessment. The much larger change was proposed by Chemrisk whose evaluation altered a number of other assumptions without proper justification.

Industry also points to the recently held Banbury Conference at Cold Spring Harbor as evidence that a safe dose which is "fully protective" of human health was reached by consensus.³⁰ No consensus was reached, however, with respect to regulatory conclusions related to risk assessment of dioxins. According to one participant, the contents of press material released after the conference "do not accurately reflect views of all participants, or even a consensus of those views."³¹ Developing a regulatory risk assessment was not a focus of the conference.

C. Role of EPA As a result of new evidence on the potency of dioxin, the EPA will probably upgrade the qualitative standing of TCDD to Class B1--probable human carcinogen.³² Because the debate still rages on most dioxin issues including potency, mode of action, bioaccumulative effects and risk level, EPA Chief William Reilly has requested an in-depth EPA evaluation on dioxin with a final report due April, 1992. At that time, EPA may make the standard more or less restrictive or retain the present value.³³

While the EPA has permitted state's to develop regulations on dioxin discharges which are different, this does not represent a license to relax standards. For any deviance from EPA standards, the state's decision must be scientifically-defensible and protective of designated uses.

According to EPA Assistant Administrator Lajuana Wilcher, "Any human health criterion for a carcinogen is based on at least three interrelated considerations--potency, exposure and risk. States may make their own judgements on each of these factors within reasonable scientific bounds, but documentation to support their judgements must be clear and in public record."³⁴ Overwhelming animal evidence has led EPA to conclude that in establishing criteria, animal studies should be considered until more reliable human data available.

D. Dioxin levels in fish The EPA and Food and Drug Administration (FDA) have agreed that some of the assumptions made in deriving FDA action and tolerance levels for fish and shellfish are inappropriate for use in regulating water quality.³⁵ The EPA has determined that the FDA level is not an appropriate basis for water quality criterion.

Fishing in West Virginia is a huge and vitally important industry. In 1985, over 401,800 individuals over the age of 16

attempted to hook a fish in West Virginia, including over 371,000 residents.³⁶ Over 88% of these fishermen fished in state rivers and streams. Almost 9 million fishing days were recorded that year. Over \$128 million were spent on fishing-related activities. Catfish was a goal of 90% of the fishermen.

The current EPA water quality standard for TCDD assumes a fish consumption rate of 6.5 grams per day and a bioaccumulation factor of 5,000.³⁷ New data, however, indicate that sport fishermen can consume 30 grams per day (1/2 pound per week) while subsistence fishermen consume an average of 140 grams per day, 5-24 times higher than EPA levels.³⁸ Studies on bioconcentration have found values between 39,000 and 140,000.³⁹

The National Marine Fisheries Service survey in 1973-1974 should be discounted based on the EPA and U.S. Fish and Wildlife reports, age of the study and lack of current consideration for the number of subsistence fishermen in West Virginia.⁴⁰ Channel catfish, a popular food fish, is a bottom feeder and more likely to accumulate dioxins in the lipid tissues than surface feeders. A study to indicate this may result in strengthening the EPA standard for West Virginia.

RECOMMENDATIONS

The Ohio Valley Environmental Coalition makes the following recommendations to the West Virginia Water Resources Board concerning the proposed 1991 West Virginia Water Quality Standards:

I. NO NEW DISCHARGES OF 2,3,7,8--TCDD SHOULD BE PERMITTED.

OVEC recommends adoption of a ZERO DISCHARGE standard regarding the release of 2,4,7,8-TCDD, 2,4,7,8-TCDF and the 75 associated chemical compounds in the dioxin family, including 135 furans with respect to NEW sources. Dioxin is an unintentional industry by-product with no use nor benefit. Hence, the goal should be zero discharge. This is consistent with the International Joint Commission's goal of zero discharge of persistent toxic chemicals.⁴¹

A. Dioxin statute with regards to Legislative rules governing water quality standards. Dioxin is known to bioaccumulate at levels between 5,000 to 50,000 times. According to WRB definitions, dioxin is cumulative (46-1-2.2) and persistent (46-1-2.9). Conditions not allowable in state waters (46-1-3) include materials in concentrations which are harmful, hazardous or toxic to man, animal or aquatic life (46-1-3.2.e). No significant adverse impact to the chemical, physical, hydrologic, or biologic components of aquatic ecosystems will be allowed (46-1-3.2.i). Because levels of dioxins accumulate in fish tissues and clearly reach harmful concentrations, dioxin discharges should be banned.

Any state regulations which proclaim an anti-degradation policy (46-1-4) in which existing instream water uses and the level of water quality necessary to protect the existing uses shall be maintained and protected (46-1-4.1) would be violated based on the bioaccumulative nature of the toxin. For this reasoning, dioxin discharges should be banned. Waste assimilation and transport are not recognized as designated uses.

B. Risk assessment. Risk assessment, which allows an acceptable discharge level, is not a scientific concept. It is an estimation of the probability of a hazard causing harm. It is a policy decision. Is risk acceptable with regards to dioxins? OVEC is of the opinion that no level of safety exists in risk assessment. It is a government mechanism for permitting industries to release chemicals into the environment which are known to cause adverse effects to public health--it lets industry off the hook with acceptable levels of pollution. Risk assessment is not a scientific decision, but a political one. In the words of Dr. Barry Commoner, "Politics should not hide behind the skirt of bad science."⁴²

II. DIOXIN RELEASES SHOULD BE PHASED OUT

The proposed water quality standard of 0.013 ppq should be implemented as a least stringent standard with a complete phaseout of dioxin discharges over the next five years. Interim standards during dioxin's phaseout should be based on dioxin concentrations in fish tissues. The most likely vehicle for human exposure to unsafe levels of dioxin is through consuming contaminated fish. A standard of dioxin concentrations in fish should be adopted which would protect individuals who regularly fish downstream from a dioxin source.

The EPA and FDA standards for dioxin in fish are based on the national average fish consumption of seven ounces per month.⁴³ A health-based standard should reflect the fact that dioxin-contaminated fish will not be consumed by the national average. Instead, levels should reflect that many individuals who rely on fish as a staple part of their diet may eat 2-5 pounds of contaminated fish at a setting then not eat fish again for several weeks or months. This would enhance cancer initiation, which is related to maximum single dose.

A. Industrial discharges of dioxin TCDD is not used in any industrial processes and is not intentionally produced at any facilities. TCDD does not occur naturally. It is an unwanted by-product of many industrial processes. Practical industrial alternatives exist for those industries such as pulp mills which generate dioxin.⁴⁴

III. EXISTING SOURCES OF DIOXIN SHOULD NOT BE ALLOWED ANY INCREASES IN PERMIT DISCHARGES.

Consistent with banning dioxin discharges for new sources and phasing out discharges at current facilities, no permit increases should be granted for dioxin releases.

IV. OTHER PROPOSED DISCHARGE STANDARDS

As mentioned in the beginning, OVEC supports use of the 10^{-6} estimated risk level as the least stringent standard for water quality criteria. As long as WRB proposed standards meet or are more stringent than EPA 10^{-6} guidelines, OVEC will support these standards.

V. PROPOSED STUDIES

A. Dioxin monitoring During the dioxin phaseout period, the WV DNR should continue studies to determine the extent of dioxin contamination in tissues of both game-fish and non-game fish, sediments and sediment cores. Studies should be initiated on the impact and accumulation of dioxins in benthic invertebrates who serve as a base in the aquatic food web.

As a requirement for any facility that discharges dioxins, studies should be initiated to determine impact of discharges on aquatic habitats at various distances downstream. An upstream indicator site should also be selected as a control. These studies should be run under quality-control guidelines.

B. Total organic halogens The WRB should initiate studies to determine the total organic halogen (TOX) and adsorbable organic halogen (AOX) load being discharged into West Virginia rivers and the impact of TOX and AOX on aquatic life. Because dioxins comprise a family of 220 related chemicals, these studies can be used to develop TOX and AOX standards. These studies would be much more indicative of true organohalogen discharges than those for individual chemicals.

C. Fish consumption Based on data previously given on fishing activities in the state, it is the opinion of OVEC that new studies should focus on fish consumption by both sport and subsistence fishermen. Data from the National Marine Fisheries Service is woefully out-of-date and does not consider the recent explosion in tourism activity in the state.

Since health-based standards based on fish consumption are advised, short-term and long-range epidemiological studies should begin on initiation or promotion of dioxin-related cancers from eating contaminated fish and impact of large consumptions at one sitting versus small consumptions over time. Studies should concentrate on large rivers, such as the Ohio and Potomac, which do not meet their designated uses because of industrial discharges. The results could be used in setting applicable standards not only in West Virginia, but other states as well.

CONCLUSIONS

2,3,7,8-TCDD is the most potent animal carcinogen and teratogen ever tested. Even though a few highly publicized studies claiming that dioxin acts with a carcinogenicity threshold, these studies have not yet demonstrated what the threshold is. Available data indicate that even if these still controversial interpretations of dioxin's carcinogenicity mechanism are accepted, estimates of its potency will be reduced by a factor of only 2, not 10, 100 or 1000, as certain non peer-reviewed consultant reports recommend.

Alternative industrial processes exist for those industries that generate dioxin as a by-product. In light of dioxin's unquestioned extreme carcinogenic potency, its persistence in sediments, bioaccumulation in wildlife, and its completely unnecessary production, OVEC firmly believes that the long range goal of eliminating the discharge of dioxin should be adopted as the strong pro-active policy of the State of West Virginia to protect the health and welfare of its residents, visitors and environment.

REFERENCES

- 1 Webster, Tom. 1991. Downgrading dioxin's cancer risk: Where's the Science? J. Pest. Ref. 11:11-14.
- 2 Webster. Ibid.
- 3 West Virginia Chamber of Commerce, et. al. 1991. Supplemental proposals for revisions to West Virginia water quality standards. Charleston, WV. March 22.
- 4 West Virginia Dept. Natural Resources. 1989. WV water quality status assessment 305(b) report: 1987-1989. Charleston, WV. 131 pp.
- 5 Ibid.
- 6 Ibid.
- 7 Ibid.
- 8 Ibid.
- 9 WV-DNR. Water Resources Division. Pers. Comm.
- 10 Ohio River Valley Water Sanitation Commission. 1989. Annual report of activities during 1989. Cincinnati, OH 22 pp.
- 11 ORSANCO. 1990. Pollution Control Standards for discharges to the Ohio River, 1990 Revision, Notice of Requirements. Cincinnati, OH 13pp.
- 12 Environmental Research Foundation. 1990. New evidence that dioxin causes human cancers and other diseases. Rachel's hazardous waste news #175. April 4.
- 13 Thorton, Joe. 1991. The dioxin deception. Greenpeace May/June 16-21pp.
- 14 Webster. Ibid.
- 15 Webster. Ibid.
- 16 Fingerhut, Marilyn. et. al. 1991. Cancer mortality in workers exposed to 2,3,7,8-TCDD. New England J. Med. 324:212-218.
- 17 Bailar, John. 1991. How dangerous is dioxin? New England J. Med. 324:260-262.
- 18 Ezzell, C. 1990. Clarifying dioxin's cellular invasion. Sci. News 139:308.
- 19 Fingerhut. Ibid.
- 20 Project Environment Foundation. 1991. Position on the need for a dioxin standard. Minneapolis, MN. 4pp.
- 21 Ezzell, Ibid.

- 22 Thorton. Ibid.
- 23 Webster. Ibid.
- 24 Jenkins, in Environmental Research Foundation. 1990. Dioxins and cancer: Fraudulent studies. Rachel's hazardous waste news #171. Princeton, NJ. March 7.
- 25 WV Chamber of Commerce. Ibid.
- 26 Webster. Ibid.
- 27 Ibid.
- 28 WV Chamber of Commerce. Ibid.
- 29 Keenan, in Webster. Ibid.
- 30 WV Chamber of Commerce. Ibid.
- 31 Silbergeld, Ellen. 1991. Correspondence to Dr. Jan Witkowski. January 29.
- 32 Anonymous. 1991. EPA likely to upgrade dioxin cancer classification to reflect new data. Inside EPA. Feb. 1. p.4.
- 33 Ezzell. Ibid.
- 34 Wilcher, LaJuana. 1990. Memorandum on State policies, water quality standards, and permit limitations related to 2,3,7,8--TCDD in surface water. U.S. EPA January 5.
- 35 Wilcher. Ibid.
- 36 U.S. Fish and Wildlife Service. 1989. 1985 National survey of fishing, hunting and wildlife-associated recreation: West Virginia. U.S. Dept. of Interior. 81pp.
- 37 Wilcher. Ibid.
- 38 U.S. Environmental Protection Agency. 1990. EPA's position on Georgia's adoption of a dioxin water quality standard. Region IV. March.
- 39 Mehrle and Cook, in Webster. Ibid.
- 40 WV Chamber of Commerce. Ibid.
- 41 Project Environment Foundation. Ibid.
- 42 Thorton. Ibid.
- 43 Wilcher. Ibid.
- 44 U.S. Office of Technology Assessment. 1989. Technologies for reducing dioxin in the manufacture of bleached wood pulp. OTA-BP-G-54. Washington, DC. 74pp.

TABLE I.

TCDD levels in fish fillets collected from the Kanawha River.

River Kilometer	Sampling Site	TCDD Levels [*] (ppt)	
		Channel Catfish	Game ^{**} Species
79	Institute	5	2 1
74	St. Albans	5 2	4 3
68	Nitro	21 17	12 3
66	Armour Creek	45 36	3 2
63	Pocatalico River	9 5	18 6
58	Plymouth	35 10	4 4
50	Winfield Locks & Dam	26	
49	Winfield Dam Tailwater	15 8	16 5 8

* Determined for 4-6 fillets; more than one number denotes replicate samples.

** Spotted Bass, Largemouth Bass, or Smallmouth Bass.

TABLE II.

TCDD levels in sediment core samples collected from Kanawha River tributaries.

River/Creek Kilometer	Depth in Sediments	TCDD Level* (ppt)	
		Left Bank	Right Bank
Pocatalico River			
0.3	0 - 10 cm	-	< 1
	10 - 20 cm	-	< 1
0.5	0 - 10 cm	3044	-
	10 - 20 cm	2516	-
2.6	0 - 10 cm	< 1	-
	10 - 20 cm	80/92	-
2.7	0 - 10 cm	< 1	< 1
	10 - 20 cm	< 1	< 1
2.8	0 - 10 cm	-	< 1
	10 - 20 cm	-	< 1
Armour Creek			
0.2	0 - 10 cm	91	-
	10 - 20 cm	1889	-
0.3	0 - 10 cm	< 1	260
	10 - 20 cm	-	136
0.5	0 - 10 cm	47	-
0.6	0 - 10 cm	< 1	-
1.0	0 - 10 cm	-	< 1
	10 - 20 cm	-	81

*Two numbers listed indicate laboratory duplicate analyses; a dash indicates that a site was not sampled.

TABLE III.

TCDD levels in sediments collected from the Kanawha River.

River Kilometer	Sampling Site	TCDD Level [*] (ppt)	
		Left Bank	Right Bank
96.2	Charleston	< 1	-
95.0	Charleston	< 1	-
93.0	Elk River Mouth	-	< 1
90.3	Blaine's Island	< 1	< 1
85.1	South Charleston	< 1	< 1
77.2	St. Albans	< 1	< 1/< 1
73.2	Coal River Mouth	-	43
71.3	Nitro	< 1	< 1
66.9	I-64 Bridge	36/40	100
65.0	Below Armour Ck Mouth	17	26
61.8	Raymond	< 1	< 1/< 1
54.6	Little Guano Creek	14	-
52.0	Winfield Bridge	< 1	21/37
51.7	Winfield Bridge	-	20
50.7	Winfield Locks & Dam	< 1	-
7.1	Point Pleasant	28	26

*Two numbers listed indicate laboratory duplicate analyses; a dash indicates that a site was not sampled.

Table 4. Ohio River Dioxin Fact Sheet

Sample Site: Ohio River at Willow Island Tailwaters
Sample Date(s): July 3, 1990

Species	# of Fish	Sample Type	Length (cm)	Weight (gr)	% Lipid	Total TCDD (PPT)	Total TCDF (PPT)
Channel Catfish	5	whole body	50-55	1198-1746	16.45	2.8	3.3

Sample Site: Ohio River at Racine Tailwaters
Sample Date(s): July 31, 1990

Species	# of Fish	Sample Type	Length (cm)	Weight (gr)	% Lipid	Total TCDD (PPT)	Total TCDF (PPT)
Channel Catfish	5	fillet	40-57	482-2041	8.24	4.4	1.7

Sample Site: Ohio River at Gallipolis Tailwaters
Sample Date(s): July 5, 1990

Species	# of Fish	Sample Type	Length (cm)	Weight (gr)	% Lipid	Total TCDD (PPT)	Total TCDF (PPT)
Channel Catfish	5	fillet	42-60	703-2282	11.96	3.8	1.1
Hybrid Striped Bass	4	fillet	33-37	347-659	1.77	2.7	1.3

Sample Site: Ohio River at Huntinton
Sample Date(s): July 30, 1990

Species	# of Fish	Sample Type	Length (cm)	Weight (gr)	% Lipid	Total TCDD (PPT)	Total TCDF (PPT)
Channel Catfish	3	fillet	42-61	610-1949	4.01	9.2	0.52

NOTES: One pound approximately = 454 grams.
One inch approximately = 2.5 centimeters.
Analyses based on a wet tissue basis.

ALABAMA RIVER PULP COMPANY INC.



P. O. Box 100
Claiborne Mill
Perdue Hill, Alabama 36470
Phone (205) 575-2000

June 27, 1991

West Virginia State Water Resources Board
1260 Greenbriar Street
Charleston, West Virginia 25311

Dear Sir,

Our sister company, Apple Grove Pulp and Paper Company, Inc. submits the following documents for your consideration on the establishment of a water quality standard for 2,3,7,8 - Tetrachloro-Dibenzo-p-Dioxin:

- (1) Support Document for the Establishment of a Water Standard for Dioxin for the State of West Virginia

by: ChemRisk
A Division of McLaren/Hart
Stroudwater Crossing
1685 Congress Street
Portland, Maine 04102

- (2) West Virginia Ambient Water Quality Standard for 2,3,7,8-TETRACHLORO-DIBENZO-p-DIOXIN

by: Joseph V. Rodricks, Ph.D.,
Senior Vice President of
EVIRON Corporation
Arlington, Virginia

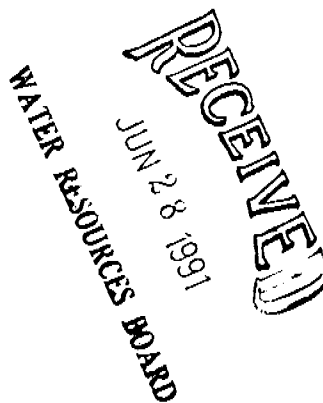
The two consulting firms conclude that a dioxin standard of 1.2 ppq is conservative and protective of human health. Apple Grove Pulp and Paper Company, Inc. respectfully requests that the West Virginia State Water Resource Board adopt 1.2 ppq as their dioxin standard.

Sincerely,

Donald B. Morris
Manager of Environmental Affairs

DBM:ab

Hand Deliver 7/28/91





MINI SUMMARY

Support Document for the Establishment of a Water Quality
Standard for Dioxin for the State of West Virginia

By:

ChemRisk
A Division of McLaren/Hart
Stroudwater Crossing
1685 Congress Street
Portland, Maine 04102

ChemRisk states: "For the purpose of developing a dioxin water quality standard for the State of West Virginia, an allowable dose level of 1.0 pg/kg-day is conservative and protective of public health. Based on this allowable dose of 1.0 pg/kg-day, a regulatory bioaccumulation multiplier of 5,000 and a fish consumption rate of 6.5 g/day, a water quality standard of 2.1 ppq can be derived for the State of West Virginia, based on human health considerations. If the aforementioned freshwater fish consumption rate of 1.0g/day based on the Maine study were to be used instead of the 6.5 g/day, a water quality standard of 14 ppq would be derived. Similarly, if the fish consumption rates of 2.1 to 5.8 g/day based on the Ohio River data were used instead of this 6.5 g/day, a water quality standard of 6.6 to 2.4 ppq would be derived. Incidentally, EPA Region III approved the neighboring states of Maryland's and Virginia's water quality standards of 1.2 ppq. Because of this precedence, it may be most appropriate for the state of West Virginia to adopt a 1.2 ppq standard as well. In addition, the scientific evidence at present can document that toxic effects in aquatic organisms have not occurred at water concentrations of 3.5 ppq and below. Thus the proposed water quality standard of 1.2 ppq is protective of both human health and aquatic life."

Basis of calculation used:

Daily dose 2,3,7,8 TCDD - 1.0 pg/kg-day
Bioaccumulation factor - 5,000
Fish consumption - 1 - 6.5 g/day

Donald B. Morris
Manager of Environmental Affairs

**SUPPORT DOCUMENT
FOR THE ESTABLISHMENT OF A
WATER QUALITY STANDARD FOR DIOXIN
FOR THE STATE OF WEST VIRGINIA**

ChemRisk™
A Division of McLaren/Hart
Stroudwater Crossing
1685 Congress Street
Portland, Maine 04102
(207) 774-0012

June 25, 1991



ChemRisk
A Division of McLaren/Hart

Support Document for the Establishment of a
Water Quality Standard for Dioxin in the State of West Virginia

Table of Contents

Executive Summary	i
1.0 INTRODUCTION	1-1
2.0 ACCEPTABLE LIMITS OF HUMAN EXPOSURE	2-1
2.1 Human Health Effects	2-2
2.1.1 Noncarcinogenic Health Effects	2-3
2.1.2 Human Carcinogenicity	2-5
2.2 Mechanism of Action	2-13
2.3 Carcinogenic Dose Response	2-15
2.3.1 Nonthreshold Models	2-17
2.3.2 Threshold Models	2-19
2.3.3 Summary: Carcinogenic Dose Response	2-20
2.4 Noncarcinogenic Dose Response	2-21
2.4.1 Reproductive / Teratogenic / Fetotoxic Effects	2-21
2.4.2 Immunotoxicity	2-24
2.4.3 Summary: Noncarcinogenic Dose Response	2-24
2.5 Recommended Acceptable Daily Intake	2-25
3.0 EFFECTS OF DIOXIN ON AQUATIC BIOTA	3-1
3.1 Effects of Acute Exposure to TCDD in Water	3-2
3.2 Effects of Chronic Exposure to TCDD in Water	3-4
3.3 Other Toxicity Studies	3-6
3.4 Conclusion	3-9
4.0 BIOCONCENTRATION/BIOACCUMULATION OF TCDD IN FISH ..	4-1
4.1 Measuring Dioxin Accumulation in Fish	4-1
4.2 Factors Affecting Accumulation in Fish	4-3
4.3 Establishing A Regulatory Bioaccumulation Multiplier (RBM)	4-6
4.4 Conclusions	4-10

Support Document for the Establishment of a
Water Quality Standard for Dioxin in the State of West Virginia

Table of Contents (cont'd)

5.0	FISH CONSUMPTION	5-1
5.1	General Population	5-1
5.2	Recreationa Anglers	5-2
5.2	Summary	5-4
6.0	PERSPECTIVE ON RISK	6-1
6.1	Regulatory Positions on Risk	6-2
6.1.1	Federal Agencies	6-3
6.1.2	State Agencies	6-5
6.1.3	Summary	6-6
6.2	Comparison of Risks	6-6
6.2.1	Commonplace Risks	6-7
6.2.2	Occupational Risks	6-8
6.2.3	Dietary Risks	6-9
6.3	The Cost of Compliance with a 10^{-6} Risk Limit	6-10
6.4	Conclusions	6-13
7.0	RECOMMENDATION FOR A SCIENTIFICALLY SUPPORTABLE WATER QUALITY STANDARD	7-1
8.0	REFERENCES	8-1
APPENDIX A		A-1

Support Document for the Establishment of a
Water Quality Standard for Dioxin in the State of West Virginia

List of Figures

Figure 2-1.	Mechanism of Dioxin Action on Target Cells	2-15a
Figure 3-1.	Summary of Effect Levels from Acute Exposure Studies of Aquatic Organisms to TCDD in Water	3-1c
Figure 3-2.	Summary of Effect Levels from Chronic Exposure Studies of Aquatic Organisms to TCDD in Water	3-1f
Figure 7-1.	States Proposing Independently Derived Dioxin Standards	7-1a

Support Document for the Establishment of a
Water Quality Standard for Dioxin in the State of West Virginia

List of Tables

Table 3-1.	Effects of Acute Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water	3-1a
Table 3-2.	Effects of Chronic Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water	3-1d
Table 3-3.	Effects of Exposure of Fish and Other Aquatic Organisms to TCDD via Several Exposure Routes	3-2a
Table 5-1.	Results of Fish Consumption Survey of Recreational Anglers in Maine . . .	5-3a
Table 5-2.	Distribution of Species and Average Weight of Species Harvested	5-4a
Table 6-1.	Selected MCLs and Associated Risk Levels	6-4a
Table 6-2.	Commonplace Risks	6-7a
Table 6-3.	Occupational Risks of Death	6-8a
Table 6-4.	Risk Estimates of Various Food Catagories Containing Carcinogenic Substances	6-9a
Table 6-5.	Naturally Occuring Carcinogens in Food	6-9b



ChemRisk
A Division of McLaren/Hart

**Support Document for the Establishment of a
Water Quality Standard for Dioxin in the State of West Virginia**

EXECUTIVE SUMMARY

The federal government has mandated that, by 1992, all states establish ambient water quality standards for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as one of the 126 "priority pollutants" listed under Section 307(a) of the Clean Water Act (CWA). There are two basic options available to the states. One option available to states is to adopt EPA Ambient Water Quality Criteria (AWQC) as enforceable water quality standards. A number of state agencies have taken this approach and have adopted the default values proposed in the EPA's 1984 document, *Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin*.

A second option, as referenced by Section 304(a)(1) of the CWA, is the development of a water quality standard based on the latest scientific knowledge regarding effects on human health and the environment. A number of states, including New Hampshire, Virginia, New York, Tennessee, South Carolina, Georgia, Alabama, Arkansas, Texas, and Maryland have chosen to use this approach in developing state-specific standards. New Hampshire and New York have adopted water quality standards of 1 part per quadrillion (ppq). EPA Region III has recently approved the 1.2 ppq standards for TCDD adopted by Maryland and Virginia. EPA Region IV has also approved a 1.2 ppq standard for TCDD in Georgia. These state-specific standards are substantially different from the criteria proposed in the 1984 EPA document.

This second option is preferable because it reflects, among other things, the states' use of site-specific or regional-specific exposure factors and more recent scientific data on TCDD. Although EPA is now reviewing its own criteria through a formal process, it has not withdrawn the previously proposed criteria as a recommended standard for states.

A scientifically sound and health-protective water quality standard for dioxin can be developed through a critical analysis of the following key factors:

- the selection of a health-protective acceptable daily intake (ADI) for TCDD;

- the potential for bioaccumulation of TCDD in fish and other aquatic organisms; and,
- the selection of an appropriate rate of freshwater fish consumption.

In addition, acceptable limits of exposure for aquatic organisms are examined. This technical support document presents the most recent literature on each of the key factors outlined above and develops a basis for a scientifically defensible and health-protective water quality standard for TCDD in the State of West Virginia.

Acceptable Limits of Human Exposure

Different threshold approaches (i.e., threshold models, pharmacokinetic modeling, or toxicological safety factors) have been used to estimate consistent ADIs for TCDD. A number of European and North American governments have used a safety factor approach to estimate ADIs ranging from 1 to 10 picogram TCDD per kilogram body weight per day (pg/kg-day) (Ontario, 1985; van der Heijden et al., 1982; NCASI, 1987; U.K., 1989; Tollefson, 1991). Most of these countries have based their estimates primarily on the 1,000 pg/kg-day No-Observed-Adverse-Effect-Level (NOAEL) reported in the Kociba et al. (1978) cancer bioassay of rats.

The most sensitive study of noncarcinogenic effects (Bowman et al., 1989) was used to derive an ADI of 13 pg/kg-day. A daily intake for TCDD of 10 pg/kg-day was recently recommended by a working group of the World Health Organization (WHO, 1990) based on the application of safety factors to data reflecting reproductive effects, immunotoxicity, and carcinogenicity in the various laboratory animal species. An ADI of 20 to 80 pg/kg-day recently proposed by the Washington Department of Health (WDH, 1990), and developed using a pharmacokinetic model, is consistent with the ADIs developed by these other groups.

The numerical consistency evident among these estimated allowable exposure levels is not surprising in view of the fact that a common receptor mechanism is believed to mediate all toxic responses to TCDD. The weight of evidence clearly supports consideration of the receptor-mediated or threshold mechanism of toxicity when evaluating the human health hazards associated with exposure to dioxin. Based on the most current scientific evidence, an ADI of 1 to 10 pg/kg-day is protective of human health for all toxic responses. This range of ADIs is orders of magnitude greater than the EPA's recommended intake level of 0.006 pg/kg-day based on a linear,

nonthreshold model and a risk level of 10^{-6} (EPA, 1984). The selection of an ADI of 1.0 pg/kg-day to be used in the development of a water quality standard for West Virginia is in fact, at the conservative end of this range of allowable exposure levels estimated via a threshold approach.

Effects of Dioxin on Aquatic Biota

Although the available data are still somewhat limited, it appears that some species of fish are sensitive to the toxic effects of TCDD. The age and physiological state of the fish, concentration of TCDD in the water, and duration of exposure have a significant impact on the toxic effects observed. For short-term exposures to different life stages of fish, the Lowest-Observed-Effect-Concentrations (LOEC; the lowest concentrations at which effects have been observed) range from 100 to 107,000 ppq whereas the No-Observed-Effect-Concentrations (NOEC; the concentrations at which no effects have been observed) range from 10 to 1,050 ppq. For long-term exposures to different life stages of fish, published LOECs range from 38 to approximately 3,000 ppq. Recent experimental stream studies conducted on pulp and paper mill effluent discharges indicate that NOECs exceed 5.8 ppq for warm-water species and 3.5 ppq for cold-water species. Other published NOECs range as high as 3,000 ppq. Based on a review of the available data regarding aquatic toxicity, the weight of evidence supports the finding that ambient water concentrations of 3.5 to 5.8 ppq are protective of freshwater aquatic organisms.

Bioconcentration/Bioaccumulation of TCDD in Fish

Bioaccumulation multipliers for dioxin reported in the literature vary considerably. This variance can be attributed to differences in the experimental methodologies used in laboratory and field studies conducted over the past ten years to derive the multipliers.

Using a consistent methodology to calculate a bioaccumulation multiplier results in a narrow range of bioaccumulation multipliers below 5,000. This approach is based upon a nominal dioxin water concentration which is calculated from the total amount of dioxin added to the system and fish fillet concentrations determined from the edible portion of the fish and can be defined as a "regulatory bioaccumulation multiplier" (RBM). Regulatory agencies use a similar approach to establish discharge limits which are based on total dioxin entering the system. Based on the RBM analysis, the value of 5,000 constitutes the most scientifically based multiplier for regulatory purposes.

Fish Consumption

When setting a water standard for dioxin, another key factor to consider is the fish consumption rate. The EPA has used the value of 6.5 g/day as an estimate of fish consumption in developing its Ambient Water Quality Criteria (AWQC). According to a national survey by Rupp and Associates, about 85% of the U.S. population does not eat any freshwater fish on a regular basis. This study also shows that the average resident of the South Atlantic U.S. eats much less than 6.5 g/day of freshwater fish. According to Rupp and associates, the per capita rate of freshwater fish consumption was estimated to be 1.1 g/day.

A recreational use survey of the Ohio River provides an estimate of angler freshwater fish consumption for this part of West Virginia. It is estimated that the average angler consumes between 2.1 and 5.8 g/day of fish obtained from the Ohio River. Another recent study on the consumption of freshwater fish by Maine anglers resulted in a median consumption rate of 1 g/day.

In addition to the aforementioned surveys, it is important to remember that a consumption rate assumes that a person eats fish at this rate on a daily basis for 70 years, and that all of the fish comes from an impacted stream. In conclusion, using the EPA fish consumption rate of 6.5 g/day is certainly conservative in lieu of existing surveys in West Virginia and in other states.

Recommendation for a Scientifically Supportable Water Quality Standard

Although the EPA AWQC are non-regulatory guidelines, when formally adopted by a state as part of its water quality standards, AWQC may become enforceable maximum acceptable levels of a substance in ambient waters. While states may adopt a water quality standard that is identical to the EPA AWQC, they have been given the option of considering local environmental conditions and human exposure patterns in order to develop site-specific or region-specific numerical limits that are based on the latest scientific knowledge regarding effects on health and the environment.

The EPA AWQC for dioxin for bodies of water from which fish are consumed were developed in 1984 and calculated using the following equation:

$$WQS = (ADI \times BW) / (BCF \times FCR)$$

Where:

- WQS = Water quality standard (pg/L)
- ADI = Allowable daily intake (pg/kg-day)
- BW = Body weight (kg)
- BCF = Bioconcentration factor (L water/kg fish)
- FCR = Fish consumption rate (kg/day)

When deriving their AWQC for the ingestion of fish, the EPA assumed that the maximum allowable dose for humans was 0.006 pg/kg-day, based on a non-threshold approach and a 10⁻⁶ risk level. It was assumed that individuals ingested 6.5 grams of fish per day from a single source. Using a BCF of 5,000 for fish and a human body weight of 70 kg, an AWQC of 0.014 pg/L (ppq) was derived.

Since that time, however, considerably more has been learned about the behavior of dioxin. Our scientific understanding of the behavior of TCDD in the environment, in fish, and in humans has evolved over time to the extent that the EPA (1984) criteria are seriously outdated. Careful consideration of each factor in the AWQC equation, and an evaluation of the weight of scientific evidence supporting their selection, will result in a scientifically based standard for dioxin that is protective of human health.

For the purpose of developing a dioxin water quality standard for the State of West Virginia, an allowable dose level of 1.0 pg/kg-day is conservative and protective of public health. Based on this allowable dose of 1.0 pg/kg-day, a regulatory bioaccumulation multiplier of 5,000 and a fish consumption rate of 6.5 g/day, a water quality standard of 2.1 ppq can be derived for the State of West Virginia, based on human health considerations. If the aforementioned freshwater fish consumption rate of 1.0 g/day based on the Maine study were to be used instead of the 6.5 g/day, a water quality standard of 14 ppq would be derived. Similarly, if the fish consumption rates of 2.1 to 5.8 g/day based on the Ohio River data were used instead of this 6.5 g/day, a water quality standard of 6.6 to 2.4 ppq would be derived. Incidentally, EPA Region III approved the neighboring states of Maryland's and Virginia's water quality standards of 1.2 ppq. Because of this precedence, it may be most appropriate for the state of West Virginia to adopt a 1.2 ppq standard as well. In addition, the scientific evidence at present can document that toxic effects in

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Page vi

aquatic organisms have not occurred at water concentrations of 3.5 ppq and below. Thus the proposed water quality standard of 1.2 ppq is protective of both human health and aquatic life.

Support Document for the Establishment of a Water Quality Standard for Dioxin in the State of West Virginia

1.0 INTRODUCTION

The federal government has directed all states to establish ambient water quality standards for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as one of 126 "priority pollutants" listed under Section 307(a) of the Clean Water Act (CWA). Although the EPA Ambient Water Quality Criteria (AWQC) are non-regulatory guidelines, they become enforceable maximum acceptable levels in ambient waters if they are adopted as part of a state's water quality standards. While states may adopt a water quality standard that is identical to the EPA AWQC, they have been given the option of considering local environmental conditions and human exposure patterns in order to develop a site-specific or region-specific numerical limit. In developing such a limit for West Virginia's ambient waters, it is imperative that relevant local and regional information be considered along with the most current scientific evidence concerning the human and environmental impacts of TCDD. The purpose of this document is to provide technical support for the development of a scientifically valid water quality standard for TCDD in West Virginia.

A critical analysis of a number of key factors is essential to the development of a scientifically defensible and health-protective water quality standard for TCDD. These factors include:

- the selection of a health-protective acceptable daily intake (ADI) for TCDD;
- the potential for bioaccumulation of TCDD in fish; and
- the selection of an appropriate rate of freshwater fish consumption.

Each of these critical factors is discussed in this technical support document. Chapter 2 presents a detailed discussion of the most current and relevant scientific studies on the carcinogenic, reproductive, teratogenic, fetotoxic, and immunotoxic effects associated with exposure to TCDD. Both threshold and non-threshold models for carcinogenic dose response are presented along with a discussion of the most current thought on TCDD's mechanism of action.

The effects of dioxin on fish and other aquatic organisms are discussed in Chapter 3. Although the development of an ambient water quality standard does not consider non-human health effects, a discussion of levels of TCDD that are safe for aquatic organisms is included to ensure that a standard based on human health will also be protective of aquatic organisms.

The importance of establishing a scientifically defensible bioaccumulation factor for TCDD in fish is presented in Chapter 4. Based on a review of all significant studies of bioaccumulation and bioconcentration reported in the scientific literature, a "regulatory" BAF is developed that constitutes the most scientifically based multiplier to be used for regulatory purposes.

In Chapter 5, a freshwater fish consumption rate that is health protective is presented. A recent comprehensive study on freshwater fish consumption is discussed along with the EPA fish consumption rate of 6.5 g/day.

Although current scientific thought indicates that TCDD acts via a threshold mechanism (Chapter 2), regulatory policy has previously been based on the belief that TCDD had no threshold. Thus, the EPA developed a risk-specific dose (RsD) based on a cancer potency estimate for TCDD and a one in a million level of risk. Chapter 6 presents a perspective on risk and discusses regulatory precedence in the selection of appropriate levels of risk.

A short summary of the major factors to be considered in developing a water quality standard for West Virginia is included in Chapter 7. Based on the analysis of key factors presented in each of the chapters, a scientifically supportable water quality standard that is fully protective of human health and aquatic organisms is derived.

2.0 ACCEPTABLE LIMITS OF HUMAN EXPOSURE

A central tenet of the science of toxicology, first articulated by Paracelsus in the 1500s, states that the toxic and therapeutic properties of a given chemical are differentiated by the dose received. In the words of Paracelsus:

"All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy."

It is this determination of an acceptable dose level, one at which there is reasonable certainty that toxic effects will not occur, which is the focus of this chapter.

Acceptable doses of human exposure to agents in the environment can be expressed in several ways, depending on the mechanism of action of the particular chemicals. An acceptable daily intake (ADI) is broadly defined as the threshold level of exposure to a chemical below which no adverse health effects would be expected to occur. More specifically, the toxic manifestations of a threshold-acting agent only occur when a specific level of exposure has resulted in the saturation of biological receptor sites. For example, compounds suspected of being carcinogens by promoting, rather than initiating carcinogenesis do not act directly upon the genetic material. Rather, mediation via a receptor or metabolic event is required before the promoter compound can cause a biologic response. Most of the phenobarbital type compounds such as DDT, PCBs, TCDD, and other chlorinated hydrocarbons are believed to be carcinogenic through a promoting mechanism on preexisting abnormal cells (Klaassen et al., 1986), thus a threshold is thought to exist for these types of chemicals.

In contrast, another type of exposure limit, the risk-specific dose (RsD), represents a dose of a substance which is associated with a specified cancer risk level (i.e., one in one hundred thousand) (EPA, 1988). Derivation and use of an RsD implies that any dose of the nonthreshold agent, regardless of quantity, would be associated with some level of risk. Direct acting genotoxic agents, such as 1,2,3,4-butadiene epoxide, dimethyl sulfate, and bis(chloromethyl)ether, are highly electrophilic compounds which directly interact with nucleophilic molecules such as DNA (Klaassen et al., (1986). For these types of compounds, mediation through an intermediary is not necessary for a toxic effect to occur, therefore, no threshold is thought to exist. Determining a

"safe" level of exposure to these types of compounds involves the determination of an acceptable level of risk as well as the RsD that would be associated with that risk.

Critical to the evaluation of the human risks associated with exposure to TCDD is the understanding of the mechanism by which TCDD elicits a toxic response. The following sections contain a detailed review of the noncarcinogenic and carcinogenic health effects of TCDD in humans and animals, the mechanisms of action of TCDD, threshold and nonthreshold models for evaluating the carcinogenic response in humans, and dose-response models for noncarcinogenic extrapolations. ADIs are recommended in the final section (2.5), based on a consideration of the total weight of scientific evidence pertaining to TCDD's mechanism of action and toxicity in humans and animals.

2.1 Human Health Effects

Numerous epidemiologic studies have examined the potential association between TCDD exposure and a number of disease or mortality endpoints in humans (AMA, 1984; EPA, 1985; Fishbein, 1987; NCASI, 1987; UAREP, 1988; Bond et al., 1989; Tollefson, 1991). Available data on human exposure is found primarily in studies involving industrial workers, herbicide sprayers, the exposed population in the Seveso, Italy accident, residents of Times Beach, Missouri, and U.S. Air Force personnel involved in the use of Agent Orange in Operation Ranch Hand. In these studies, the concentrations of TCDD to which people were exposed are generally considered to be much greater than would typically be encountered in the environment. In addition, the populations evaluated in many of these studies were exposed to multiple chemicals, thereby complicating the assessment of disease endpoints potentially attributable to TCDD exposure (Tollefson, 1991). Furthermore, the cohort sizes for many of the studies have been too small to allow statistical identification of small increases in cancer risk (Tollefson, 1991).

Epidemiological studies are generally observational rather than experimental and may, therefore, include a number of known or unknown biases. Though there are methodologies that can be used to minimize and control for many confounding factors inherent to epidemiological studies, there are usually several obstacles that cannot be adequately treated. These discrepancies must be considered when establishing a causal link between exposure and disease. For example, it is usually misleading to infer causality from a single epidemiological study because confounding

factors or errors in characterizing exposure or disease within the study may lead to biases which affect the determination of relative risk (incidence rate of the exposed population divided by the incidence rate of the unexposed population). Therefore, epidemiologists look for consistency of findings among multiple studies before causal inference is made. The strength of the association is also considered in this characterization. In addition, the presence of a dose-response relationship adds credence to the studies since a positive correlation between the level of exposure and the occurrence of disease is generally expected to occur. Finally, biological plausibility is considered. The existence of supporting laboratory bioassay data substantiates similar findings in epidemiological studies.

2.1.1 Noncarcinogenic Health Effects

While epidemiologic studies have shown a definitive relationship between exposure to TCDD and chloracne, associations between exposure to high concentrations of chemicals containing TCDD and other non-cancerous effects have not been definitively established. Studies of the noncarcinogenic effects of TCDD in humans are briefly discussed below.

Chloracne is one long-term adverse health effect that can be definitively associated with human exposure to TCDD (Suskind, 1985; UAREP, 1988). This characteristic persistent dermatosis has been observed in cases of both acute and chronic exposure to relatively high concentrations of TCDD and can be induced following systemic uptake or dermal exposure (Kociba and Schwetz, 1982; Suskind, 1985; Kimbrough and Houk, 1987). The persistence of chloracne for several years following high-level occupational exposures is consistent with the relatively long (7 years) half-life of TCDD in humans. While chloracne is associated with exposure to a number of other chlorinated aromatic hydrocarbons (Kimbrough et al., 1984), Suskind (1985) has identified TCDD as the most potent chloracnegen.

Porphyria cutanea tarda (PCT) has been reported among workers accidentally exposed to complex chemical mixtures containing TCDD (UAREP, 1988). This condition is manifested by discoloration and increased fragility of the skin and hirsutism reportedly accompanies the altered urinary porphyrin excretion pattern in certain cases. Jirasek and coworkers (1973, 1974 as reported in UAREP, 1988) reported PCT in 11 of 78 workers from a plant in Czechoslovakia where the workers were engaged in the manufacture of phenoxy herbicides and

hexachlorobenzene. Bleiberg et al. (1964) found PCT in 11 of 29 workers examined from a plant in New Jersey. Suskind (1983) observed PCT in workers manufacturing phenoxy herbicides at a plant in Nitro, West Virginia. In each of these populations, reexamination of workers at a later date indicated that the skin lesions associated with PCT had disappeared and only a few abnormalities in urinary porphyrin excretion patterns remained (Poland et al., 1971; Pazderova-Vejlupkova et al., 1981; Suskind and Hertzburg, 1984). Hobson (1984) concluded that the PCT seen in these populations was most likely attributable to exposure to hexachlorobenzene. Furthermore, PCT has not been a finding in studies of other human populations that were exposed to TCDD in the absence of hexachlorobenzene and trichlorophenol (UAREP, 1988).

In 1976 an industrial explosion at a 2,4,5-T plant in Seveso, Italy contaminated the surrounding area with several chemicals that contained concentrations of TCDD. Within days of the explosion, 187 cases of chloracne were reported (Crow, 1981). Kimbrough and Houk (1987), in their review of health effects of dioxins, noted that although liver effects, enlarged livers, and some abnormal results of certain liver function tests were reported, evaluation of the overall results of the health studies on the exposed population indicated no severe systemic health effects (Reggiani, 1978; 1980). In addition, Bruzzi (1983) compared rates of congenital anomalies (genital and neural tube defects) in the Seveso region with those in the unexposed Lombardi region. Incidence rates were found to be higher in the Seveso region than in the Lombardi area. When looking only at the Seveso region, however, there was little correlation between incidence rates and the extent of potential exposure within the region. In fact, fewer genital anomalies were observed in the high exposure area than in the unexposed sections of Seveso. Therefore, no positive conclusions could be drawn from the study. In a more recent report concerning birth defects, Mastroiacovo et al. (1988) did not demonstrate an increased risk of birth defects associated with exposure to TCDD (UAREP, 1988).

In the Times Beach, Missouri area, residents were potentially exposed to TCDD when wastes contaminated with TCDD were mixed with salvage oil and sprayed on various sites for dust control (Kimbrough and Houk, 1987). Several acute effects were reported in individuals exposed to contaminated soil in riding arenas (Kimbrough and Houk, 1987). A pilot study conducted on a small group of people from another contaminated area found no significant differences in test results between unexposed and possibly exposed groups (CDC, 1984). In a related study, Hoffman et al. (1986) reported results of a study intended to assess immunologic response of

mobile home park residents potentially exposed to areas in which TCDD-contaminated waste oil had been sprayed. These authors reported a significantly increased frequency of anergy and relative anergy compared to controls. Hoffman et al. (1986) interpreted these findings as evidence that TCDD exposure was associated with depressed cell-mediated immunity. However, because the results may have been biased due to shortcomings in experimental design and because researchers eliminated a significant portion of the data, reviewers of this study have questioned its value for identifying an immunologic hazard (Dean and Kimbrough, 1986). Results of a follow-up study (Evans et al., 1987) involving participants who were initially anergic or relatively anergic indicated that none of the participants were anergic, and only one exposed subject and one unexposed subject were relatively anergic in the repeat test. In view of the experimental design and data interpretation problems and in light of the failure of Evans et al. (1987) to corroborate the initial results, an immunologic hazard has not been demonstrated by Hoffman et al. (1986).

2.1.2 Human Carcinogenicity

A number of studies have examined the association of increased cancer rates with exposure to dioxin and dioxin contaminated materials (Armstrong, 1983; Bond et al., 1983, 1989; Cook et al., 1986; Eriksson et al., 1984; Filipini et al., 1981; Fingerhut et al., 1991a,b; Kimbrough, 1990; Lipson, 1983; Mastroiacovo et al., 1988; Minister of Veteran's Affairs, 1983; Moses et al., 1984; Nelson et al., 1979; Pocchiari et al., 1979; Reggiani, 1978, 1980; Smith et al., 1982; Suskind and Hertzberg, 1984; Zack and Gaffery, 1983). Certain studies have suggested a positive association between exposure to chemicals containing TCDD and soft tissue sarcoma and malignant lymphoma (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981). Fingerhut et al. (1991) have recently reported a possible relationship between high-level occupational exposures and soft tissue sarcoma and respiratory cancers. However, the positive associations identified in these studies are equivocal and are not supported by the results of other epidemiologic studies (Smith et al., 1982a,b, 1983; Pearce et al., 1986; Smith and Pearce, 1986; Wiklund and Holm, 1986).

Case-control studies of workers in Sweden exposed to phenoxy acid herbicides or chlorophenols reported a statistically significant increased risk of soft-tissue sarcomas and malignant lymphomas (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981). These Swedish studies have been interpreted as showing evidence for an association between soft-tissue sarcoma and exposure to TCDD (Fingerhut, 1991a). However, the major limitations of the studies concern

the amount of exposure to TCDD. Exposure history was assessed retrospectively by questionnaire at a time when herbicide use was receiving widespread media attention; 2,4,5-T was banned in Sweden in 1977 (Tollefson, 1991). This publicity could have influenced the soft-tissue sarcoma cases to overestimate their exposure or may have contributed to recall bias (Tollefson, 1991).

The results of other epidemiologic studies involving herbicide exposure (Smith et al., 1982a; 1982b; 1983; Pearce et al., 1986; Smith and Pearce, 1986; Wiklund and Holm, 1986) have not supported the findings of the Hardell studies (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981). For example, a cohort study of Swedish agricultural and forestry workers, a cohort similar to the earlier Hardell studies, linked occupational data from the census with mortality data. Using this approach no increased relative risk of soft-tissue sarcoma was observed when the cohort was compared to Swedish men employed in other industries, even though the exposure of agricultural and forestry workers to phenoxy acids was estimated to be greater than that of other occupational groups (Wiklund and Holm, 1986).

Interestingly, the Wiklund and Holm (1986) census linkage study showed no increased risk for all agriculture and forestry workers, even at the upper 95% confidence limit of the relative risk estimate which was 1.0. These findings raise questions concerning the results of the Hardell case-control studies (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981), which have not been satisfactorily resolved (UAREP, 1988). Limitations associated with any case-control study, however, includes the absence of an objective means to qualify or validate human exposure levels, recall bias, observation bias, and misdiagnosis of tumors (UAREP, 1988). In addition, the high relative risks reported in the Hardell studies (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981) were associated with exposure to "TCDD-contaminated herbicides" and therefore included exposure to a myriad of chemicals that may produce adverse health effects (Tollefson, 1991). Furthermore, corroborative evidence has not been found from studies in other countries.

Case-control studies in New Zealand have found no evidence of a relationship between occupational exposure to phenoxy herbicides and soft-tissue sarcoma (Smith et al., 1982b, 1983; Smith and Pearce, 1986). Pearce et al. (1985), however, reported that agricultural workers were at an increased risk of developing non-Hodgkin's lymphoma. The subsequent interview phase of this study, however, did not suggest that exposure to phenoxy herbicides was the explanation for

the increased risk estimate; no significant difference regarding potential exposures to phenoxy herbicides or chlorophenol was observed between cases and controls (Pearce et al., 1986). In addition, a study of professional 2,4,5-T herbicide sprayers in New Zealand also found no significant increase in the risk of miscarriages or birth defects (Smith et al., 1982a).

Hoar et al. (1986), in a National Cancer Institute case-control study of workers involved in the agricultural use of herbicides in Kansas, demonstrated an association between the use of phenoxyacetic acid herbicides, specifically 2,4-dichlorophenoxyacetic acid (2,4-D), and non-Hodgkin's lymphoma. An association was not found between exposure and either soft-tissue sarcoma or Hodgkin's disease. Hoar et al. (1986), however, reported that 2,4-D does not contain TCDD, nor would one expect it to be formed in the synthesis of this herbicide.

Axelson et al. (1980) reported statistically ($p < 0.05$) increased mortality associated with stomach cancer ($RR = 7.7$) in Swedish railroad workers exposed to phenoxy acids possibly contaminated with TCDD. Interpretation of these results are limited by inadequately controlled confounding factors (NCASI, 1987) and exposure to a mixture of chemicals including amitrole which has been demonstrated to be carcinogenic in both mice and rats (IARC, 1987).

Numerous epidemiological studies have been conducted on Vietnam veterans exposed to Agent Orange and other defoliants used extensively in Vietnam. Agent Orange contains two phenoxyherbicides, 2,4,5-T and 2,4-D, in addition to low levels of TCDD, a known contaminant of 2,4,5-T. In a recently released report, the Center for Disease Control (CDC) (1990) examined the risks related to a number of cancers (non-Hodgkin's lymphoma, soft tissue and other sarcomas, Hodgkin's disease, and nasal, nasopharyngeal, and primary liver cancers) in veterans who had served in Vietnam during the time period when Agent Orange was used. The focus of the study was to determine whether the incidence of these cancers was higher in Vietnam veterans than in men who did not serve in Vietnam. This study reported an increased risk of non-Hodgkin's lymphoma among Vietnam veterans relative to men who did not serve in Vietnam, but no increased risk for the other five cancers. However, the study concluded that there was no evidence to support the contention that an increased risk of contracting non-Hodgkin's lymphoma was related to Agent Orange. In fact, the pattern of risk among subgroups of Vietnam veterans appeared to be opposite of the pattern of Agent Orange use in Vietnam. Those personnel involved in areas with heavy Agent Orange use were at somewhat lower risk than those land-based

personnel in areas of lower Agent Orange use, and Navy veterans who served on ocean-going ships tended to be at higher risk of non-Hodgkin's lymphoma than Vietnam veterans who served on land (CDC, 1990).

Lathrop et al. (1987) reported that there is not sufficient plausible or consistent scientific evidence at this time to implicate a causal relationship between herbicide exposure and the adverse health effects observed in the Ranch Hand group. In a study conducted in New York State, no statistically significant positive association was found between soft-tissue sarcoma and history of Vietnam service, history of exposure to Agent Orange, TCDD, or 2,4,5-T, history of herbicide or pesticide exposure, or history of any military service (Greenwald et al., 1984). Kang et al. (1986) found no association between soft-tissue sarcoma and previous military service in Vietnam in a case-control study of Vietnam veterans diagnosed with soft-tissue sarcoma and treated at Veterans Administration hospitals.

Zack and Suskind (1980) conducted a mortality study of 121 white male chemical workers exposed to TCDD contaminated chemicals at a Monsanto chemical plant in Nitro, West Virginia. The primary exposure occurred when a trichlorophenol (TCP) reactor malfunctioned, releasing TCDD contaminated TCP into the environment. The workers in this study were assigned to contain and cleanup following the TCP release. Following a latency period of 29 years, no statistically significant increased mortality was found to be associated with any health effect. A total of nine malignant neoplasms were reported, five of which were respiratory cancers. According to Zack and Suskind (1980), four of the five respiratory cancer cases were smokers.

In a subsequent study, Zack and Gaffey (1983) evaluated the health effects associated with long-term exposures at the same plant, including exposures during the runaway reactor accident. The cohort was comprised of 884 white male workers of which 163 were deceased. The only statistically significant finding was increased mortality associated with bladder cancers. When the authors split the cohort into two groups, those exposed to 2,4,5-T and those not exposed to 2,4,5-T, they found that the unexposed group showed a statistically significant increased mortality associated with bladder cancers while the exposed group did not.

Suskind and Hertzberg (1984) noted that the Monsanto workers were exposed to many different chemicals including p-aminobiphenyl, a known bladder carcinogen in humans (IARC, 1987;

DHHS, 1989). Bladder tumors and bladder cancers associated mortality were evaluated separately for workers with either no 2,4,5-T or p-aminobiphenyl exposure, only 2,4,5-T exposure, only p-amino biphenyl exposure, or with both 2,4,5-T and p-aminobiphenyl exposure. All bladder tumors and cancers occurred in either the p-aminobiphenyl only exposure group or the group exposed to both p-aminobiphenyl and 2,4,5-T (Suskind and Hertzberg, 1984). Consequently, the International Agency for Research on Cancer (IARC) has used the Zack and Gaffey (1983) study in support of p-aminobiphenyl as a Group 1 carcinogen (sufficient evidence for carcinogenicity in humans) based on increased mortality associated with bladder cancers (IARC, 1987).

Thiess et al. (1982) reported a statistically significant ($p < 0.05$) increase in mortality associated with stomach cancer (3 cases) in a cohort of 76 workers potentially exposed to TCDD following a trichlorophenol reactor accident in 1953 at a BASF plant in Ludwigshafen, Federal Republic of Germany. However, the choice of comparison groups and the small number of stomach cancer deaths observed raise important questions (Kimbrough and Houk, 1987). Follow-up data for the workers in the Thiess et al. (1982) study indicated that no additional stomach cancer deaths had occurred, and although deaths due to stomach cancer were reported to be greater than expected, the differences were not statistically significant (Lehnert and Szadkowski, 1986). Moreover, the positive finding of the Thiess et al. (1982) study associating stomach cancer with TCDD exposure has not been duplicated in other epidemiologic studies (Tollefson, 1991).

Zober et al. (1990) recently reported results of a 34-year follow-up study of chemical workers previously studied by Thiess et al. (1982) and Lehnert and Szadkowski (1986). The Zober et al. (1990) study evaluated mortality of a cohort of 247 BASF employees who were divided into four different cohorts based on exposure information and were then compared to the national population. Cohort C1 ($n=69$) consisted of the most heavily exposed workers, i.e. those exposed in the TCP reactor accident. Cohort C2 ($n=84$) was identified as members of cohort C1 plus individuals who were primarily involved in cleanup and medical activities related to the reactor accident, whereas cohort C3 included all of cohorts C1 and C2 in addition to all other potentially exposed individuals through December 31, 1987. Finally, these investigators evaluated mortality for those employees who had been diagnosed with chloracne.

No statistically significant increased mortality associated with any malignant neoplasm was observed in the C1 and C3 cohorts. However, in the C2 cohort, Zober et al. (1990) reported a

statistically significant increase in mortality associated with cancer of the larynx (1 case observed, 0.03 cases expected). Because the increased mortality associated with this cancer was due to a single case, the statistical significance of that increase may not be biologically compelling. Furthermore, the authors noted that the power of the study to detect levels of risks associated with specific tumor sites was weak. In the same cohort, however, a statistically significant increase in mortality associated with all malignant neoplasms and "other and unspecified" malignant neoplasms was also reported. According to Zober et al. (1990), the "other and unspecified" malignant neoplasms include a case of pleural mesothelioma in a worker with known asbestos exposure. These investigators further noted that workers were also exposed to other chemicals at the plant including aromatic amines. Finally, in the chloracne cohort with over 20 years of exposure, presumably those with the greatest exposure to TCDD, a statistically significant increase in mortality associated with all malignant neoplasms was observed (SMR=201; 14 cases observed and 6.96 cases expected).

Except for the single larynx cancer case in the C2 cohort, there were no statistically significant increases associated with specific tumor sites. It appears that the statistical significance of the combined tumor mortalities is primarily due to cancer cases reported as "other and unspecified" malignant neoplasms. According to Zober et al. (1990), this classification included a biliary tract carcinoma, gastrointestinal tract carcinoma, and a pleural mesothelioma. If mortality associated with malignant cancers at individual tumor sites had been evaluated separately, it is likely that the SMRs would not be significantly elevated. Zober et al. (1990) concluded that a substantial excess of cancer should have been observed in this study, if in fact TCDD was a highly potent carcinogen. However, the authors also noted that no strong conclusions could be drawn from the results of this study. The possible misclassification of tumors, appropriateness of the reference group, initial definition of the cohort, and most importantly, inadequately controlled confounding factors, particularly those of other chemical exposures, suggest that there are a number of shortcomings associated with this mortality study which may also be common to other mortality studies of TCDD.

Most recently, Fingerhut et al. (1991a,b) reported the results of an epidemiologic study conducted by NIOSH involving male chemical workers at 12 chemical plants in the U.S., one of which was the Monsanto chemical plant discussed earlier in this review. A cohort consisting of 5,172 individuals who had been assigned to the production of substances that were evidently

contaminated with TCDD was studied. Mortality associated with combined cancers was slightly but significantly increased for the overall cohort. When separated into low- and high-exposure subcohorts, however, the low-exposure group showed no increased mortality associated with any cancers (Fingerhut, 1991a,b). In the high-exposure subcohort of 1,520 workers who had exposure periods of at least one year and latency periods of 20 years or more, mortalities for soft tissue sarcomas and respiratory cancers were significantly increased.

A number of confounding factors, including smoking and exposure to other chemicals, have bearing upon the interpretation of the results of this study. The workers in this cohort were exposed to high concentrations of a number of chemicals, including 2,4,5-trichlorophenol, monochloroacetic acid, 1,2,4,5-tetrachlorobenzene, hexachlorophene, and phenoxy herbicides. According to Suskind and Hertzberg (1984), workers at the Nitro, West Virginia plant were also exposed to p-aminobiphenyl, diphenylguanidine, the benzothiazolesulfonamides, m-cresol derivatives, p-phenylenediamine derivatives, 4,4'-dithiodimorpholin, ethyl parathion, methyl parathion, o-dichlorobenzene, p-dichlorobenzene, p-nitrophenol, and toluene and mercaptobenzothiazole derivatives of tetramethylinramdisulfide and trimethylquinoline components. The impact on mortality associated with these additional and combined chemical exposures was not evaluated by Fingerhut et al. (1991a,b). Although TCDD was a contaminant of some of these chemicals, it was only present at approximately the part per million level, therefore, any conclusions suggesting that TCDD was the causative agent in this study would be speculative.

Other limitations associated with the Fingerhut et al. (1991a,b) study may affect its interpretation. First, mortality in this study was measured using Standard Mortality Ratios (SMRs), which are calculated by comparing the observed deaths in the cohorts with the expected number of deaths for the general population. For rare cancers, such as soft-tissue sarcoma, it is necessary to compare cohorts with a sufficiently large population in order to generate a statistically meaningful number of expected deaths. This approach assumes that the expected number of deaths in the general population for a given disease endpoint is comparable to the expected number of deaths in the study population. However, national mortality rates do not always agree with local rates, and when local or county mortality rates are higher than national rates, comparison to national mortality rates may result in artificially elevated SMRs (Mausner and Kramer, 1985). In addition, though the Fingerhut et al. (1991a,b) cohort consisted only of male chemical manufacturing workers,

comparisons were made to national mortality rates. A more appropriate reference group would have been male chemical workers not involved with production of TCDD contaminated chemicals.

Furthermore, Fingerhut et al. (1991a,b) noted that a lack of diagnostic consistency among clinicians and pathologists resulted in the misclassification of tumor types and cause of death for cases both in the cohort and in the comparison group. The authors concluded that "the interpretation of the increased mortality from soft-tissue sarcoma in our study is limited by the small number of cases and the fact that the cause of death was sometimes misclassified on the death certificates of the workers and in the U.S. population." Finally, an increased incidence in the types of tumors that one might expect to see based on studies of highly exposed laboratory animals, namely hepatocarcinomas or adenomas, was not observed in the NIOSH study.

Fingerhut et al. (1991a,b) also measured serum lipid levels in a sample of 253 workers from two of the twelve plants studied. In the most highly exposed individuals, post-latency serum lipid TCDD levels were found to be as much as 500 times higher than normal background levels of approximately 7 ppt (Patterson et al., 1987). By extrapolating measured serum levels back to the date of last exposure, Fingerhut et al. (1991b) estimated that pre-latency serum levels were as much as 4,500 times background levels, indicating that exposures were considerably higher than those incurred today by the general population from environmental sources.

Because Fingerhut et al. (1991a,b) demonstrated a significant trend in increasing TCDD serum levels with increasing chemical exposure, it is very likely that the same type of trend for the other chemical exposures would have been observed had these data been gathered. No causative relationship was established for human carcinogenicity and TCDD exposure from this analysis. In fact, even though the authors observed very high serum lipid TCDD levels in some of the exposed population, they (Fingerhut et al., 1991a) concluded that:

"This study of mortality among workers with occupational exposure to TCDD does not confirm the high relative risks reported for many cancers in previous studies."

Although certain studies have suggested a positive association between exposure to chemicals containing TCDD as a contaminant and certain types of cancers (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981; Fingerhut et al., 1991a,b), other epidemiologic studies

have not confirmed these positive associations (Smith et al., 1982a,b, 1983; Pearce et al., 1986; Smith and Pearce, 1986; Wiklund and Holm, 1986). In recent reviews of the major human studies, Kimbrough (1991) and Tollefson (1991) have concluded that the evidence for an association of TCDD with human cancer is equivocal. The majority of the available epidemiologic studies on the association of cancer to TCDD exposure provide little evidence that dioxin is a potent carcinogen in humans (Tollefson, 1991). While the converse cannot be ruled out on the basis of currently available data (Tollefson, 1991), it is unlikely that TCDD is a human carcinogen at low doses (Bond et al., 1989). The results of the very recent Fingerhut et al. (1991a,b) studies are consistent with this conclusion.

2.2 Mechanism of Action

The mechanism by which TCDD elicits a toxic response in humans and laboratory animals is critical to understanding the significance of low level environmental exposures. Equivocal epidemiologic data suggesting increased health risks from relatively high level occupational and catastrophic exposures and no evidence of increased cancer risk from low level environmental exposures, suggests that there exists a threshold level of exposure at which no toxic effects occur (Fingerhut et al., 1991a,b; Kimbrough, 1990). In combination with human and animal data, information elucidated from mechanistic studies can be used to quantitatively evaluate whether there is a biological threshold which must be exceeded before a toxic response is observed.

The underlying biological characteristics describing TCDD's threshold have been widely studied (Greig and Dematties, 1973; Poland et al., 1976; Nebert and Jenson, 1979; Poland and Knutson, 1982; Safe, 1986; Shu et al., 1987; Goldstein, 1989; Silbergeld and Gasiewicz, 1989; Greenlee, 1990; Leung et al., 1990; Nessel et al., 1990; Poland et al., 1990). In general, a biologic threshold for hepatotoxic effects was evident from studies of mice in which toxic effects to the liver were only observed when liver to adipose TCDD ratios exceeded 1.0 (Allen et al., 1975; Olson et al., 1980; Gasiewicz et al., 1983; Leung et al., 1990). Control animals in these studies had liver to adipose TCDD ratios of less than one, similar to the ratios observed in environmentally exposed human populations (Ryan et al., 1985; Faccetti et al., 1981; Leung et al., 1990). These studies suggest that not only have doses administered to experimental animals exceeded the doses that are biologically relevant to human environmental exposures, but that a definitive level of exposure can

be correlated with a physiologic response, a level that must be exceeded to result in a biologic response.

At the sub-cellular level, the receptor-mediated mechanism proposed by Poland et al. (1990), Greenlee et al. (1990), and Nessel et al. (1990) is viewed as the most scientifically valid hypothesis for explaining TCDD's mechanism of action (WDH, 1991; Roberts, 1991; Holloway, 1990). Poland and co-workers demonstrated that TCDD reversibly binds to a soluble cytosolic receptor protein (Ah receptor) to initiate a coordinated gene expression similar to the action of steroid hormones (Poland et al., 1976; Poland et al., 1985). Results of recent research indicate that the cytosolic receptor-TCDD complex (Ah/TCDD) translocates into the nucleus of the cell and binds to XRE, a xenobiotic response element (Gallo, personal communication, 1991), as well as induces arylhydrocarbon (AHH) activity (Greig and Dematties, 1973; Nebert and Jensen, 1979). The binding of the TCDD/Ah receptor with XRE is reversible and results in the induction of the cytochrome P450 enzymes, arylhydrocarbon hydralase P4501A1 and P4501A2 binding proteins, as well as synthesis of other biologically active proteins and hepatic microsomal binding proteins (Gallo, personal communication, 1991).

Activation of the Ah receptor and induction of AHH and the cytochrome P450 system are clearly biological markers of xenobiotic exposure, but not necessarily markers of TCDD exposure and toxicity. In fact, P450 induction is associated with both the positive and negative regulation of many naturally occurring enzyme systems in the body. Results of a recent bioassay conducted by Nessel et al. (1990) support previous in vitro mechanistic studies which had suggested that doses below a certain threshold should not activate the Ah receptor or induce enzymatic activity. Nessel et al. (1990) intratracheally instilled varying concentrations of TCDD into rats in a study designed to test the hypothesis that an enzymatic threshold for TCDD action could be demonstrated. This hypothesis was based on the premise that an exposure level could be defined that did not produce a positive Ah receptor response or induction of the cytochrome P450 system. These investigators observed that at TCDD dosages below 0.55 µg/kg no enzymatic reaction was elicited, whereas dosages above 0.55 µg/kg induced both AHH and cytochrome P450.

Consistent with the recent work of Poland et al. (1990), Greenlee et al. (1990), and Nessel et al. (1990), many researchers in the scientific community believe that all observed toxic effects

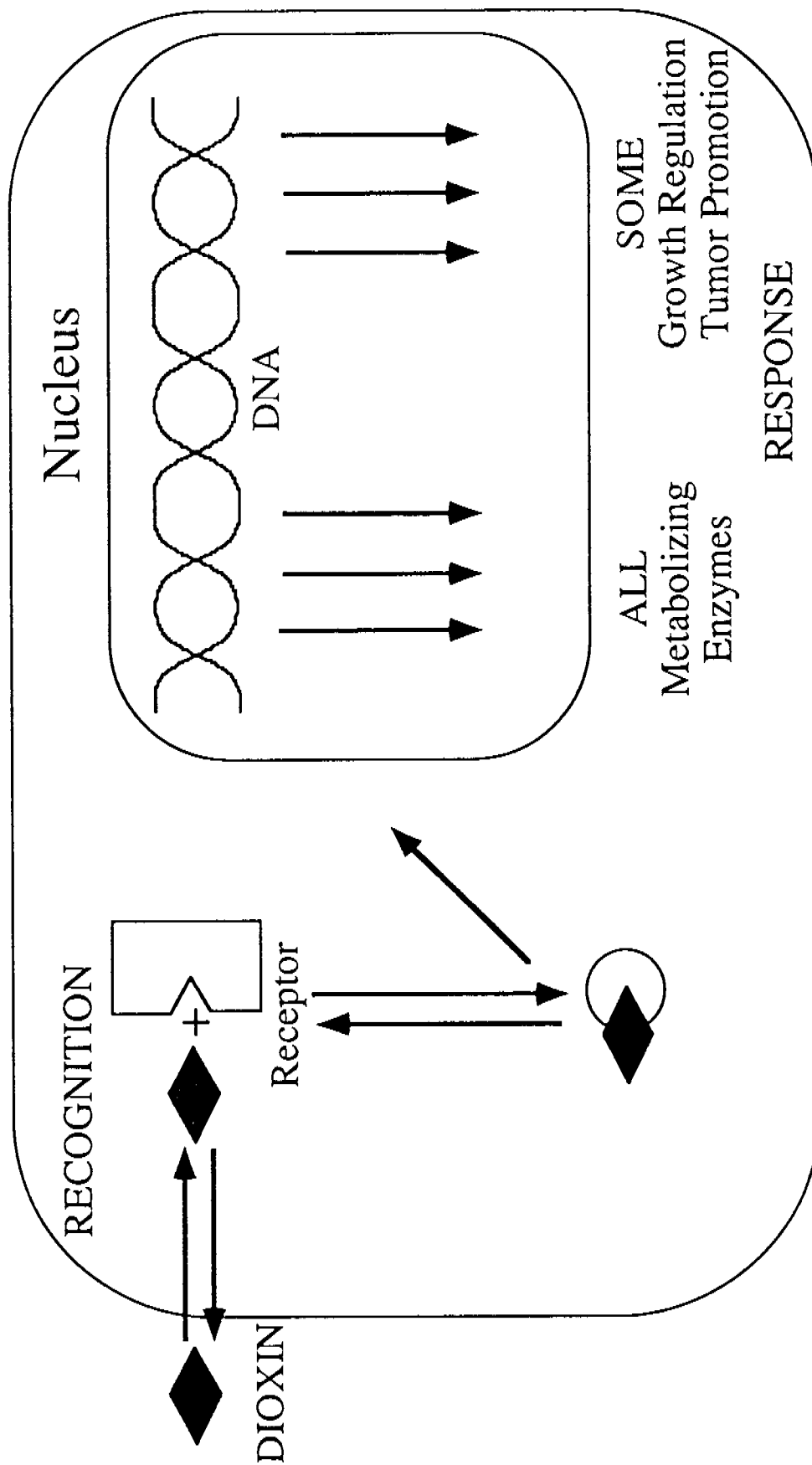
associated with exposure to dioxin, including immunotoxicity, reproductive effects, and cancer, appear to be mediated through the Ah receptor (Birnbaum, 1990; Gallo, 1990; Scheuplein, 1990a; van der Heijden, 1990). According to Gallo (1990), for dioxin to have an effect, it must reach a specific site, bind to a receptor, move into the nucleus, and bind to the genetic material (Figure 2-1). Gallo (1990) concluded that several thousand receptors must be occupied before any biological or toxic effect is seen. At doses which are too low to cause induction of cytochrome P450, no effects can occur (Birnbaum, 1990; Gallo, 1990; Scheuplein, 1990a; van der Heijden 1990). Therefore, a "safe" dose for dioxin can be established (Birnbaum, 1990; Gallo, 1990; Scheuplein, 1990a; van der Heijden, 1990).

2.3 Carcinogenic Dose Response

The epidemiologic evidence to date indicates that dioxin is less toxic to humans than once believed, and that the low levels of TCDD presently in the environment are not harmful to humans (Roberts, 1991; Scheuplein, 1990a; van der Heijden, 1990; Gallo, 1990; Birnbaum, 1990; Kimbrough, 1990). Even at the much higher levels of TCDD exposure seen in certain chemical workers and other highly exposed populations (Fingerhut et al., 1991a,b; Kimbrough, 1990), data are limited and often equivocal (Tollefson, 1991). Without adequate dose-response data from epidemiologic studies, studies of laboratory animals have been used in an attempt to characterize the relationship between chronic exposure to TCDD and chronic health effects. In particular, carcinogenic effects and reproductive effects, the most sensitive endpoints observed in laboratory animals, are the basis for developing ADIs and RsDs for TCDD.

In the absence of adequate dose-response data from epidemiologic studies, extrapolations from animal toxicity bioassays have been made in an attempt to characterize the relationship between TCDD uptake and possible carcinogenic response in humans. The two-year chronic rat study conducted by Kociba et al. (1978) is typically cited as the primary evidence supporting the carcinogenicity of TCDD in laboratory animals. Based on the Kociba et al. (1978) bioassay and on the National Toxicology Program's (NTP) 1982 bioassay, the EPA (1985) classified dioxin as a probable (B2) human carcinogen because, in the opinion of EPA, the evidence in support of TCDD as a carcinogen in animals was sufficient while the evidence supporting TCDD's carcinogenicity in humans was inadequate. Consequently, the Kociba et al. (1978) rat bioassay has been used by the U.S. Environmental Protection Agency, the Centers for Disease Control, the U.S. Food and Drug

Figure 2-1. Mechanism of Dioxin Action on Target Cells



Administration, and many government agencies in North America and Europe for estimating the human cancer risk to TCDD.

In the Kociba et al. (1978) study, 50 Sprague-Dawley (Spartan substrain) rats of each sex were maintained for up to 24 months on diets which provided 1,000, 10,000 or 100,000 pg/kg-day of TCDD with an additional 86 animals of each sex being maintained as study controls. At termination of the experiment, an extensive gross and histopathological examination was conducted on each animal. This examination revealed a dose-related toxic response to the liver characterized by severe hepatic toxicity and the presence of hepatocellular lesions (Kociba et al., 1978). Early mortality occurred throughout the study but was statistically significant only among female rats in the high TCDD dose group.

Because the incidence of hepatocellular lesions was the most sensitive adverse effect in the Kociba et al. (1978) bioassay, an accurate histopathological classification of these lesions is critical. In 1990, at the request of members of the Maine Scientific Advisory Panel (SAP), the histopathology slides of the liver lesions from the Kociba et al. (1978) study were reevaluated by an independent Pathology Working Group (PWG) using the current and widely accepted NTP classification system for proliferative lesions in the rat liver (Maronpot et al., 1986; McConnell et al., 1988). This protocol or criterion is the one now accepted by U.S. federal agencies, including the EPA. It is considerably different from the classification scheme of Squire and Leavitt (1975) used by Kociba and coworkers in 1978 and Squire in 1980 when they classified certain lesions as pre-malignant "neoplastic nodules" or "hyperplastic nodules". The current NTP guidelines distinguish between hyperplasia, a nonneoplastic response to degenerative changes in the liver, and an adenoma, a benign condition involving clear differentiation of cells from the surrounding tissue (Maronpot et al., 1986).

The independent panel was organized under the auspices of PATHCO, Inc. and included pathologists from the National Toxicology Program, Mallory Institute of Pathology, the Chemical Industry Institute of Toxicology (CIIT), as well as the participation of scientists from the EPA, the FDA, and the National Cancer Institute (NCI) (PWG, 1990a). The reevaluation of the liver pathology slides from the Kociba et al. (1978) study was conducted using the consensus diagnosis format endorsed by the NTP. A consensus was reached when 4 out of the 7 pathologists agreed on the classification of a lesion. Unlike previous histopathology reviews of the slides, the slides

were read by PWG pathologists without their prior knowledge of the dose group from which the animals were drawn (PWG, 1990a).

The PWG (1990a) concluded that there were substantially fewer cancerous tumors (about 2/3 fewer) observed in the study than previously reported. The lesions previously referred to as "hyperplastic nodules" or "neoplastic nodules" were predominantly benign, hepatocellular adenomas and were usually associated with lesions of hepatic toxicity. While the original results indicated 11 hepatocellular carcinomas in the high dose group, the PWG (1990a) diagnosed only four malignant tumors. Furthermore, there was a distinct correlation between the presence of overt hepatotoxicity and the development of hepatic lesions (PWG, 1990a, 1990b), suggesting that the maximum tolerated dose had been exceeded in animals exposed at the higher doses (PWG, 1990b).

2.3.1 Nonthreshold Models

In the United States federal agencies have used nonthreshold dose-response models to extrapolate human dose response relationships from the Kociba et al. (1978) rat bioassay data. Differing cancer potency estimates for TCDD have been derived by the EPA (1985), the Centers for Disease Control (CDC) (Kimbrough et al., 1984), and the Food and Drug Administration (FDA) (1983) using these models. The use of these models was based on the assumption that there is no threshold for carcinogenesis. For example, the linearized multistage (LMS) model used by the EPA (1985) to estimate TCDD's carcinogenic potency forces the dose response curve through zero; i.e., it assumes that any dose results in a carcinogenic response and a certain level of risk.

Recent scientific information indicates that the use of linear dose-response models to estimate human risks from exposure to TCDD was not valid. TCDD is a considerably less potent carcinogen than previously thought by the EPA and by many well-respected scientists and, as described in Section 2.2.3, the weight of evidence to date clearly supports a receptor mediated response for all toxic responses to TCDD. Based on the acceptance of an exposure level of dioxin below which no effects will occur, the linear models traditionally used for dioxin should no longer be used to evaluate dioxin risk (van der Heijden, 1990).

If, however, one were to use the LMS model, then only the most recent and scientifically defensible tumor incidence data reported by PWG (1990a) should be modelled. Survival-adjusted tumor incidence rates among treated and untreated animals have usually been considered the appropriate basis for extrapolating risks from laboratory animals to humans when a significant number of unscheduled deaths occur in a bioassay (Gart et al., 1986; McConnell et al., 1986; Haseman, 1990). For example, when evaluating the Kociba et al. (1978) study, the USEPA (1985) corrected for the high mortality observed in the high and mid-dose groups by censoring all animals that died prior to the appearance of the first "neoplastic nodule" (Portier et al., 1984).

In evaluating the PWG (1990a) histopathology results, the same early mortality correction procedure used by the USEPA to evaluate the Kociba et al. (1978) study in 1985 was applied. The following pathology information on each rat in the bioassay was available from the PWG (1990a): 1) the exact date of death or sacrifice; 2) the presence or absence of hepatocellular carcinoma or adenoma; 3) the pathological diagnosis of cause of death; and, 4) the age of the animal at death or sacrifice. The adjusted tumor incidences were then subjected to a Fischer Exact Test to determine whether significant differences in tumor incidences existed between dose groups.

In this analysis, a toxicologically equivalent human dose was estimated by scaling the PWG (1990a) results based on body weight. Currently, it is USEPA policy to extrapolate from rats to humans on the basis of relative surface area (estimated as body weight to the $2/3$'s power) for all carcinogens (USEPA, 1986a). This was considered a more protective scaling factor since: 1) it tends to give higher risk estimates per unit of dose (USEPA, 1986a); and, 2) if the parent compound is the active carcinogen, its activity is related to its time in the body, which in turn is related to clearance time and dose / surface area allometry (Bayard, 1988). In contrast, both the FDA and the CDC concur that surface area scaling is not as appropriate as body weight scaling when the active carcinogen is thought to be the administered compound itself and does not have to be metabolized to be carcinogenic (FDA, 1986; Bayard, 1988).

Although it remains unclear which scaling factor is most appropriate for 2,3,7,8-TCDD, recent reviews of interspecies scaling factors indicate that all measures of dose, except dose rate per unit of body weight, tend to overestimate human risk (Mordenti, 1986; Brown et al., 1988; Crump et al., 1989). Since 2,3,7,8-TCDD appears to exert its toxicological effects through a receptor-mediated mechanism (Greenlee, 1988; Greenlee et al., 1990; Harris et al., 1990) analogous to that

demonstrated by steroid hormones (Yamamoto, 1985; Gallo, 1988; Umbreit and Gallo, 1988), extrapolation of the bioassay dose-response data to humans can be conducted using a body weight scaling factor (Meistrich, 1988).

Using the LMS model, ChemRisk (Keenan et al., 1990a; 1991a,b) calculated a cancer potency factor for TCDD of $9,700 \text{ (mg/kg-day)}^{-1}$ by (1) adjusting the PWG (1990b) tumor incidence data to account for early mortality in the Kociba et al. (1978) bioassay; (2) considering the combined incidence of hepatocellular carcinomas and adenomas; and (3) extrapolating from rats to humans using a body weight correction factor in accordance with the policies and scientific judgment of the U.S. FDA (1983) and the CDC (Kimbrough et al., 1984). This cancer potency factor is approximately 16-fold lower than the EPA's cancer potency factor of $156,000 \text{ (mg/kg-day)}^{-1}$ (EPA, 1985), and equates to a risk-specific dose, at a 10^{-5} risk level, of 1.0 pg/kg-day .

2.3.2 Threshold Models

A number of countries including Canada, the Netherlands, West Germany, and the United Kingdom have recognized that TCDD acts via a threshold mechanism. These countries have historically used a safety factor approach to estimate ADIs for TCDD (Ontario, 1985; van der Heijden et al., 1982; NCASI, 1987; U.K., 1989; Tollefson, 1991). Nearly all ADIs for TCDD estimated by North American and Western European countries are based on the $1,000 \text{ pg/kg-day}$ NOAEL reported in the Kociba study (Kociba et al., 1978). Following a review of the available data on exposure and uptake of TCDD, an ADI for TCDD was recently recommended by a working group of the World Health Organization (WHO, 1990). It was agreed that for general toxicological effects including reproductive effects, immunotoxicity, and carcinogenicity in the various laboratory animal species, a tolerable daily intake (TDI) of 10 pg/kg-day could be determined using the safety factor approach.

Recently, the Washington Department of Health (WDH) developed an ADI for 2,3,7,8-TCDD of 20 pg/kg-day . This ADI was derived using the results of pharmacokinetic modelling for TCDD (Leung et al., 1990). WDH (1991) estimated the TCDD dose that correlated with five percent occupancy of the cytosolic (Ah) receptor, a level of occupancy considered by the WDH to be a valid and conservative approach which would be supported by most scientists. Based on the model predictions of Leung et al. (1990), this level of occupancy falls between the Kociba et al.

(1978) LOAEL of 10,000 pg/kg-day and the NOAEL of 1,000 pg/kg-day. Assuming a linear relationship for receptor occupancy between these two dose levels, WDH (1991) estimated a dose of 2,000 pg/kg-day. Thus, a NOAEL of 2,000 pg/kg-day was derived for 5% receptor occupancy, a level at which no biologic response would be expected. WDH (1991) applied a 100-fold safety factor to this NOAEL resulting in an ADI of 20 pg/kg-day.

The classical safety factor approach addresses the problem associated with species to species extrapolations (laboratory animals to man) and the variability among humans. Most often a safety factor of ten is used to account for differences between species and another factor of ten is used to account for individual variability (Klaassen et al., 1986). In the case where a no observable adverse effect level (NOAEL) has been determined in a chronic bioassay, the ADI would be estimated by dividing the NOAEL by a safety factor of 100. The resulting ADI represents a daily dose of TCDD below which no adverse health effects would occur.

The cancer potency factor of 809 (mg/kg-day)⁻¹, recently derived by the Florida HRS is based on a log-normal model which considers a biological threshold in the calculation of the CPF. The log-normal model that HRS (1991) used does not force the dose extrapolation through zero, rather, it interprets the linear slope as it passes through the threshold level. At a 10⁻⁶ level of risk, this cancer potency factor correlates to a RsD of 1.2 pg/kg-day, an estimate that is on the conservative end of other risk estimates which consider TCDD's threshold mechanism.

2.3.3 Summary: Carcinogenic Dose Response

Threshold models are the most appropriate method for estimating the dose-response relationship for TCDD in humans. The current understanding of the mechanism of action for TCDD, as discussed in Section 2.1, clearly indicates that a threshold exists for all toxic responses to TCDD. The requirement of Ah receptor mediation before the most sensitive marker of exposure is observed, induction of cytochrome P450, provides strong support for the growing scientific opinion that there is a dose at which no toxic effects occur. Consistent with other national and local governments who have developed acceptable daily intakes ranging to 20, or even 80 pg/kg-day, an ADI for carcinogenic effects of 10 pg/kg-day, based on the application of a safety factor of 100 to the NOAEL of 1,000 pg/kg-day observed in the 1978 Kociba et al. rat bioassay, is certainly fully protective of human health.

2.4 Noncarcinogenic Dose Response

Associations between exposure to TCDD and non-cancerous effects in humans have not been established. However, the noncarcinogenic toxicity of TCDD in animals has been studied in a number of acute, subchronic, and chronic studies (Kimbrough et al., 1984). These studies can be used to establish noncarcinogenic dose-response relationships in animals, and such information can be extrapolated to arrive at health-protective ADIs for humans.

TCDD has been shown to be extremely toxic to certain rodent species. The acute LD₅₀ (the dose which is lethal to 50% of the animals tested) for guinea pigs is reported to be 0.6 µg/kg body weight. The sensitivity to TCDD toxicity is extremely variable among laboratory animal species. Kociba and Cabey (1985) found the LD₅₀ for hamsters to be as high as 5,051 µg/kg; i.e., the hamster is over 8,400 times less sensitive to TCDD than is the guinea pig. Clinical signs of acute toxicity in laboratory animals are severe weight loss, hepatotoxicity, chloracne, thymic atrophy, and death.

Furthermore, chronic high-level TCDD exposure has been shown to result in severe hepatotoxic effects in rats and mice (Kociba, 1984). Although numerous tumor types were observed in the Kociba et al. (1978) bioassay, the liver was identified as the primary and most important target tissue for overt toxicity (Kociba et al., 1978). Furthermore, carcinogenicity observed in the liver was evident only at doses that elicited a severe noncarcinogenic toxic response in the study animals (Squire, 1990). This was evidenced by the Kociba et al. (1978) observations of severe liver toxicity, diminished weight gain, and increased mortality.

2.4.1 Reproductive / Teratogenic / Fetotoxic Effects

Reproductive, immunotoxic, fetotoxic, and teratogenic effects in animals are considered to be the critical endpoints for assessing the chronic noncarcinogenic risk from exposure to TCDD, since human studies are limited and therefore insufficient for establishing a reference dose (RfD). Chronic exposure to TCDD has been shown to induce adverse reproductive, immunotoxic, fetotoxic, and teratogenic effects in several species of laboratory animals (EPA, 1985).

Exposure to TCDD has induced teratogenic effects, predominantly cleft palate and kidney anomalies, in several strains of mice (Smith et al., 1976). The no-observed-adverse-effect-level (NOAEL) for a teratogenic response in the mouse is 0.1 µg/kg-day (100,000 pg/kg-day) (Smith et al., 1976). Teratogenic effects have also been reported for several strains of rats (EPA, 1985). Review of the scientific literature clearly indicates that the rat is more susceptible to these types of noncarcinogenic effects than is the mouse. Because of the number of different studies conducted on laboratory animals, only those studies which demonstrate the lowest levels at which effects were observed are discussed below.

Murray et al. (1979) conducted a three-generation study of Sprague-Dawley rats fed a diet containing TCDD at dose levels of 0, 0.001, 0.01, and 0.1 µg/kg-day. At the highest dose group (0.1 µg/kg-day), fertility and neonatal survival were significantly reduced in the f₀ litters, and neonatal survival was reduced in the f₁ generation. At 0.01 µg/kg-day, fertility was significantly reduced in f₁ and f₂ generations but not in the f₀ generation, and statistically significant decreases in litter size, fetal and neonatal survival, and growth were observed. No effects on fertility, litter size, or postnatal body weight were observed in the 0.001 µg/kg-day dose group, nor was any consistent effect on neonatal survival observed. A significant decrease in postnatal survival was observed in an f₁ litter of the 0.001 µg/kg-day dose group but not in subsequent generations. Murray et al. (1979) concluded that the reproductive capacity of rats ingesting 0.001 µg/kg-day (1,000 pg/kg-day) TCDD through three generations was not affected; consequently, 1,000 pg/kg-day represents a NOAEL for reproductive effects in rodents.

Nisbet and Paxton (1982) have argued that a more appropriate statistical analysis of the Murray et al. (1979) data indicates that there were decrements in reproductive performance at the 1,000 pg/kg-day dose level. They therefore maintain that the 1,000 pg/kg-day dose level was actually a LOAEL and not a true NOAEL. Scientists from the U.S. Centers for Disease Control (CDC) and the U.S. Department of Agriculture (USDA) have reviewed the Nisbet and Paxton (1982) reanalysis and have not agreed with their findings (Kimbrough et al., 1984). Furthermore, the FIFRA Scientific Advisory Panel concluded that although the data suggested an embryotoxic effect at 1,000 pg/kg-day, they concluded that 1,000 pg/kg-day represented a NOAEL (EPA, 1985).

A series of studies conducted on rhesus monkeys have evaluated the effects of chronic exposure to TCDD on body burden levels, reproductive success, maternal-to-infant transfer, and behavioral

patterns (Schantz et al., 1986; Bowman et al., 1989a; 1989b). In these studies, adult female rhesus monkeys were chronically exposed (prior to and during gestation over 3.5 to 4 years) to 0, 5, or 25 ppt of TCDD in the diet. Reproductive toxicity was determined according to an Index of Overall Reproductive Success (IORS) which scored several reproductive events including numbers of conceptions, abortions, stillbirths, live births, and survival to weaning. The authors reported consistent evidence of reproductive impairments in monkeys fed 25 ppt TCDD in the diet, whereas there was no indication of reproductive deficit in the 5 ppt group (Bowman et al., 1989a). The daily dose of TCDD that each group of monkeys received can be estimated by considering the average adult monkeys' body weight (7.5 kg), the average daily consumption rate of food (190 g), and the concentration of TCDD in the food. Using the average body weight and food consumption rate one can estimate that the 5 ppt dose group received a daily TCDD dose of 130 pg/kg-day. This dose represents a NOAEL for reproductive toxicity in female rhesus monkeys.

Other chronic studies on monkeys have reported decreases in reproductive capacity following exposure to 50 and 500 ppt TCDD in the diet (1,300 and 13,000 pg/kg-day) for at least 7 months prior to breeding (Barsotti et al., 1979; Allen et al., 1979; Schantz et al., 1979). These studies substantiate the finding that 130 pg/kg-day is the most sensitive NOAEL reported for reproductive toxicity in primates. It should be noted that this NOAEL is lower than the NOAEL of 1,000 pg/kg-day reported by Murray et al. (1979) for reproductive success in rats.

In a subchronic fetotoxicity study, McNulty (1984) administered 2,3,7,8-TCDD to rhesus monkeys at cumulative doses of 0.2, 1.0, and 5.0 $\mu\text{g/kg}$ between days 20 to 40 of gestation. The doses in this study were not chosen to establish a statistically rigorous dose-response relationship, but rather to find a dose that was reliably fetotoxic or teratogenic. Monkeys treated with 1.0 or 5.0 $\mu\text{g/kg}$ showed signs of maternal toxicity and fetotoxicity; two of two monkeys in the high dose group aborted, while abortions occurred in 13 of the 16 pregnant monkeys in the 1.0 $\mu\text{g/kg}$ dose group. None of the four mothers given 0.2 $\mu\text{g/kg}$ in divided doses showed signs of toxicity and the three surviving offspring in the 0.2 $\mu\text{g/kg}$ dose group showed no gross, radiologic, or histological abnormalities. However, there was one spontaneous abortion in this dose group. The abortion rate in the low dose group was similar to that in the controls (3/12), yet the authors concluded that the number of animals in the 0.2 $\mu\text{g/kg}$ was far too small for statistical evaluation or to warrant a conclusion of no observable effect (McNulty, 1984).

Review of the scientific literature clearly indicates that primates are the most sensitive species to adverse reproductive effects, teratogenicity, and fetotoxicity following exposure to TCDD. Cleft palates and other anomalies of the soft palate have been reported in rhesus macaques administered either 1.0 µg/kg or 0.2 µg/kg TCDD in corn oil by gavage. The total dose was administered in nine doses from days 20 to 40 following insemination (Zingesser, 1979). Maternal toxicity, effects on fertility, and fetotoxicity (reduced conception, fetal resorption, abortion) were reported in monkeys exposed to 500 ppt TCDD (approximately 10 µg/kg-day) in the diet for 6 months prior to and during pregnancy (Barsotti et al., 1979). As previously described, Bowman et al. (1989) reported reduced reproductive capacity in monkeys exposed to 25 ppt TCDD (0.63 µg/kg-day) in the diet. This effect level is lower than the level of 10 µg/kg-day reported by Murray et al. (1979) to affect fertility in rats.

2.4.2 Immunotoxicity

The effects of TCDD on the immune response of animals vary widely and are highly dependent on species and age (U.K., 1989). The guinea pig has been identified as a species which is highly susceptible to TCDD's immunotoxicity (U.K., 1989). A LOAEL of 0.04 µg/kg-week was identified for immunotoxic effects in adult guinea pigs fed a diet containing TCDD weekly for 8 weeks. For adult animals, a "minimal effect level" of 0.04 µg/kg-week (40 ng/kg-week or 6,000 pg/kg-day) was determined (U.K., 1989).

2.4.3 Summary: Noncarcinogenic Dose Response

Studies of noncarcinogenic effects among various laboratory animals exposed to TCDD clearly indicate that primates are one of the most sensitive species. In fact, the scientific evidence to date suggests that nonhuman primates are more sensitive to the effects of TCDD than are humans (Kimbrough, 1990). Furthermore, humans appear to be less sensitive to the toxic effect of TCDD than most animals (Kimbrough, 1991). Animals and humans demonstrate different levels of sensitivity to toxicological responses to dioxin due in part to toxicokinetics and Ah-receptor differences. As a result of toxicokinetic differences, the highest levels of dioxin in humans are found in fatty tissues, however, the reverse is true for animals. Most animals accumulate high concentrations of dioxin in vital organs (Kimbrough, 1991). In addition, human cells have fewer

cytosolic Ah-receptors and a lower binding affinity to TCDD than animal cells (Kimbrough, 1990). As a result, animals would be expected to demonstrate a greater sensitivity to dioxin than humans.

When deriving acceptable daily intakes for humans based on chronic animal studies, a safety or uncertainty factor of 100 is typically applied to laboratory-derived NOAELs. This safety factor includes a factor of 10 for species differences (usually between rodents and humans) and a factor of 10 for variations in sensitivity within the human population. It is important to note that sub-human primates, in comparison to more resistant species, are very susceptible to the adverse effects of TCDD (Kimbrough, 1990). Therefore, the classical use of a 10-fold safety factor for species to species extrapolation may not be warranted in this particular case and a single safety factor of 10 to account for individual variability within the human population should provide an adequate margin of safety. Based on a NOAEL of 130 pg/kg-day for reproductive effects in rhesus monkeys (Bowman et al., 1989a), an ADI of 13 pg/kg-day can be determined by application of a 10-fold safety factor.

2.5 Recommended Acceptable Daily Intake

The weight of evidence clearly supports consideration of the receptor mediated mechanism for toxicity when evaluating the human health risks from exposure to TCDD. Incorporation of this concept has resulted, even when different threshold approaches (i.e., mathematical threshold models, pharmacokinetic modelling, or the classical safety factor approach) have been used, in consistent ADI estimates. A number of European and North American governments have considered TCDD's receptor mediated mechanism of action when establishing allowable daily intakes. These countries have historically used a safety factor approach to estimate ADIs for TCDD of between 1 and 10 pg/kg-day (Ontario, 1985; van der Heijden et al., 1982; NCASI, 1987; U.K., 1989; Tollefson, 1991). They have based their estimates primarily on the 1,000 pg/kg-day NOAEL reported in the Kociba et al. (1978) study and the application of safety factors of between 100 and 1,000. A TDI for TCDD of 10 pg/kg-day was recently recommended by a working group of the World Health Organization (WHO, 1990) based on the application of safety factors to data reflecting reproductive effects, immunotoxicity, and carcinogenicity in the various laboratory animal species. The ADI of 20 to 80 pg/kg-day proposed and developed using a pharmacokinetic model by the Washington Department of Health (WDH, 1991), is also consistent with the ADIs developed by these other groups.

In addition, ADIs estimated from the Kociba et al. (1978) and Bowman et al. (1989) NOAELs are very similar. Whereas the Bowman et al. (1989) study supports an ADI of 13 pg/kg-day for chronic noncarcinogenic effects, the Kociba et al. (1978) study supports an ADI of 10 pg/kg-day for carcinogenic effects. This consistency is not surprising in view of the mechanism by which the Ah receptor is believed to mediate all toxic responses (Birnbaum, 1990; Poland et al., 1990; Greenlee et al., 1990; Nessel et al., 1990). When one correctly accounts for physiologic differences among species, regardless of the toxic endpoint, extrapolation from laboratory animals to humans at the cellular level should arrive at similar results.

As has been discussed in many of the above sections, the scientific weight of evidence indicates that the linear model should not be used to assess the risks of dioxin (van der Heijden, 1990; Kimbrough, 1991; Squire, 1991). However, if one is compelled to use the linear model in conjunction with the Kociba et al. (1978) rat data reported by PWG (1990a), a cancer potency factor for TCDD of $9,700 \text{ (mg/kg-day)}^{-1}$ can be calculated. This cancer potency factor equates to a risk-specific dose, at the 10^{-5} risk level, of 1.0 pg/kg-day (Keenan et al., 1990a; 1991a,b). Clearly, the more appropriate methods for extrapolating to humans are models which incorporate the threshold mechanism and the safety factor approach and which recognizes the existence of a biological threshold. Based on the most current scientific evidence, an ADI in the range of 1 to 10 pg/kg-day is protective of human health for all toxic responses.

3.0 EFFECTS OF DIOXIN ON AQUATIC BIOTA

In deriving the 1984 Ambient Water Quality Criteria (AWQC) guidelines, the EPA concluded that the available data pertaining to the aquatic toxicity of TCDD were insufficient to support the development of national criteria based on aquatic health (EPA, 1984). While the available data are still somewhat limited they do provide a basis for understanding the potential impacts of ambient water levels of dioxin on aquatic organisms. This section summarizes the available scientific literature regarding the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) to fish and other aquatic organisms.

The available data indicate that fish are sensitive to the toxic effects of 2,3,7,8-TCDD. This sensitivity, however, varies considerably among species. Additionally, age, concentration, physiological state, and duration of exposure also have a significant impact (Cooper, 1989). It appears that smaller fish are more sensitive to TCDD than larger fish (Norris and Miller, 1974). Studies indicate that the manifestation of toxic effects are almost always delayed and occur after exposure has ended (Norris and Miller, 1974; Helder, 1980, 1981; Adams et al., 1986; Cooper, 1989).

The most commonly observed signs of TCDD toxicity in fish include edema, cutaneous hemorrhages, fin necrosis, decreased food intake, reduced growth rate, and delayed mortality (Miller et al., 1973; Norris and Miller, 1974; Hawkes and Norris, 1977; Yockim et al., 1978; Helder 1980, 1981; Branson et al., 1985; Kleeman et al., 1988; Mehrle et al., 1988; Spitsbergen et al., 1988a, 1988b; Wisk and Cooper, 1990). Immune responses appear to be unaltered except at very high, almost lethal concentrations (Spitsbergen et al., 1987). The histopathology of adult fish exposed to TCDD includes epithelial, lymphomyeloid, and cardiac lesions (Cook et al., 1991). However, no studies have been undertaken to determine if TCDD produces a carcinogenic response in fish (Cooper, 1989). Although an Ah receptor has now been identified in fish (personal communication Dr. K. Cooper, Rutgers University, 1990), additional research needs to be undertaken to define the mechanism of dioxin's toxicity.

Results of studies of acute and chronic exposure of fish and other aquatic organisms to TCDD in water are summarized in Table 3-1, Figure 3-1, Table 3-2, and Figure 3-2, respectively. Table 3-1 reports nominal concentrations (total amount of TCDD added to the exposure system divided by

Table 3-1. Effects of Acute Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water

Species	Life Stage Exposed	Exposure Duration (hours)	Test Duration (days)	Carrier Solvent	TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration ^a (ppq)	Effects ^a	Reference
Fathead minnow <i>Pimephales promelas</i> (1 - 2 g)	juvenile	24 - 96	150	acetone	0; 120; 720 7,100; 82,000	720	7,100	Mortality ^b	Adams et al., 1986
Guppy <i>Poecilia reticulata</i>	9 - 40 mm	120	37	chloroform, acetone	0; 100,000; 1,000,000; 10,000,000	ND	100,000	Reduced survival ^c , reduced food consumption ^b , fin necrosis ^b	Miller et al., 1973; Norris and Miller, 1974
Japanese medaka <i>Oryzias latipes</i>	embryo	3 (exposed on day of fertilization)	3	acetone	0; 400; 1,700; 5,900; 13,200; 31,700; 50,800	ND	400	Decrease in hatching incidence (EC50 14,000 ppq) ^d , lesions (EC50 3,500 - 14,000 ppq) ^d , mortality (3-day LC50 9,000 ppq) ^d	Wisk and Cooper, 1990
Japanese medaka <i>Oryzias latipes</i>	embryo	11 (exposed on day of fertilization)	11	acetone	0; 500; 2,400; 7,000; 12,000; 33,500; 57,900	NR	NR	Lesions (EC50 2,200 ppq) ^d	Wisk and Cooper, 1990
Pike <i>Esox lucius</i>	eggs	96	≥23	DMSO, acetone	0; 100; 1,000; 10,000	ND	100	Retarded embryonic development ^b , decreased growth of fry ^c , increased fry mortality ^b , edema ^b , pathological changes in liver ^b	Helder, 1980
Rainbow trout <i>Salmo gairdneri</i>	eggs	96	168	DMSO, acetone	0; 100; 1,000; 10,000; 100,000	ND	100	Growth retardation ^c , edemas ^b , mortality ^c , teratologic defects ^b	Helder, 1981

Table 3-1. Effects of Acute Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water (Continued)

Species	Life Stage Exposed	Exposure Duration (hours)	Test Duration (days)	Carrier Solvent	TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration (ppq)	Effects ^a	Reference
Rainbow trout <i>Salmo gairdneri</i>	yolk sac fry	96	168	DMSO, acetone	0; 1,000	ND	1,000	Growth retardation ^c , edemas ^b , mortality ^c , teratologic defects ^b	Helder, 1981
Rainbow trout <i>Salmo gairdneri</i>	juvenile (0.85 g)	(16 hr/day, 4 days)	70	DMSO, acetone	0; 10,000; 100,000	ND	10,000	Growth retardation ^c , mortality ^b , edemas ^b	Helder, 1981
Rainbow trout <i>Salmo gairdneri</i>	35 g	6	139	acetone	0; 107,000	ND	107,000	Increased relative liver weight ^c , decreased body weight gain ^b , fin rot ^b , mortality ^b	Branson et al., 1985
Waterflea <i>Daphnia magna</i>	young, adults	48	9	acetone	0; 200 - 1,030,000	1,030,000	ND	No effects reported at any concentration ^b	Adams, et al., 1986

a. Lowest-Observed-Effect-Concentration (LOEC) given in table relates to the most sensitive effect reported by the authors. The effects listed occurred either at the LOEC or at higher TCDD concentrations used in the study.

b. Statistical analysis was not reported.

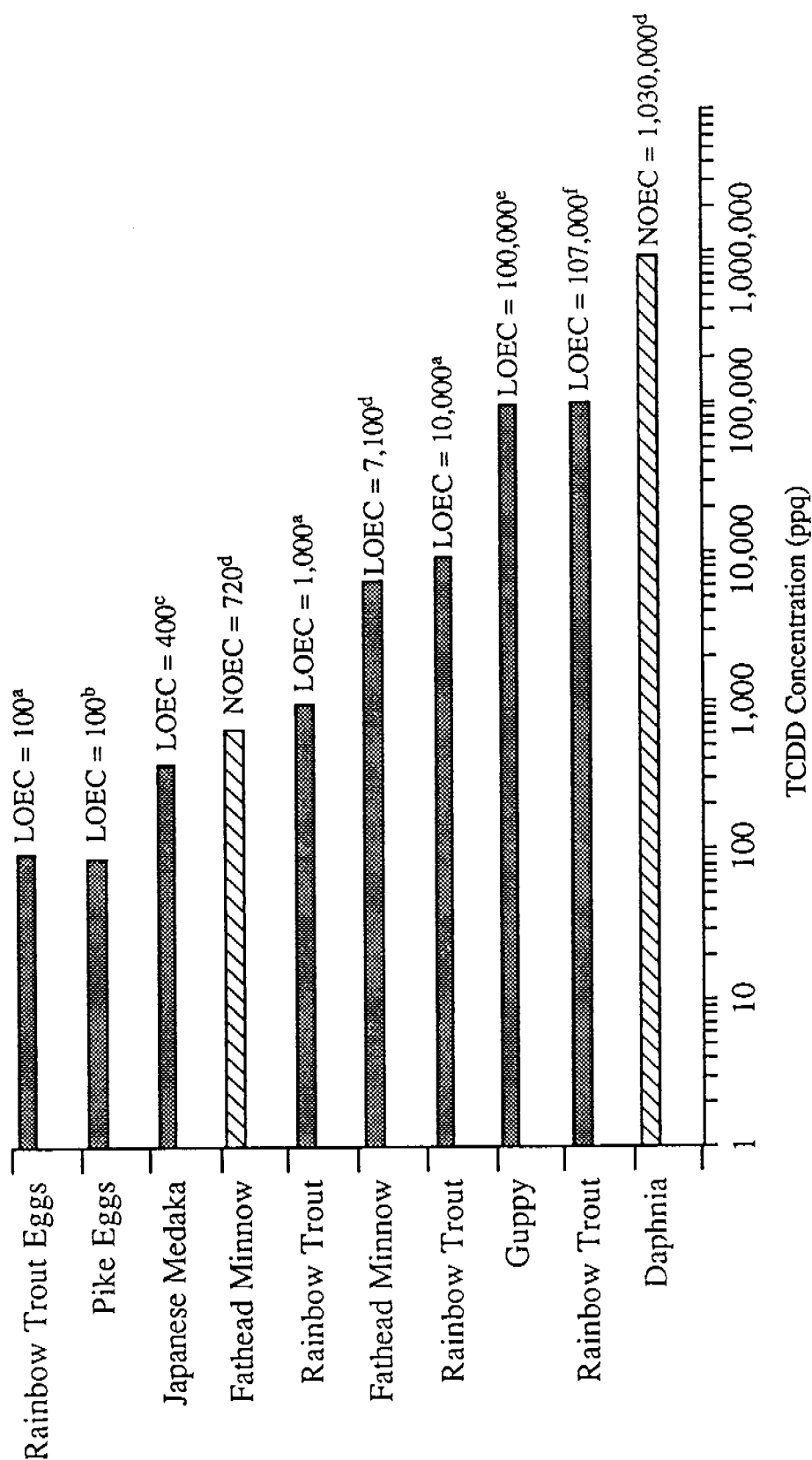
c. Statistically significant.

d. LC50 or EC50 was statistically derived; however statistical analysis was not reported for comparison of treatment groups to controls.

ND = Not determined

NR = Not reported

Figure 3-1. Summary of Effect Levels from Acute Exposure Studies of Aquatic Organisms to TCDD in Water



References:
 a. Helder, 1981
 b. Helder, 1980
 c. Wisk and Cooper, 1990
 d. Adams et al., 1986
 e. Miller et al., 1973; Norris and Miller, 1974
 f. Bronson et al., 1985

Table 3-2. Effects of Chronic Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water

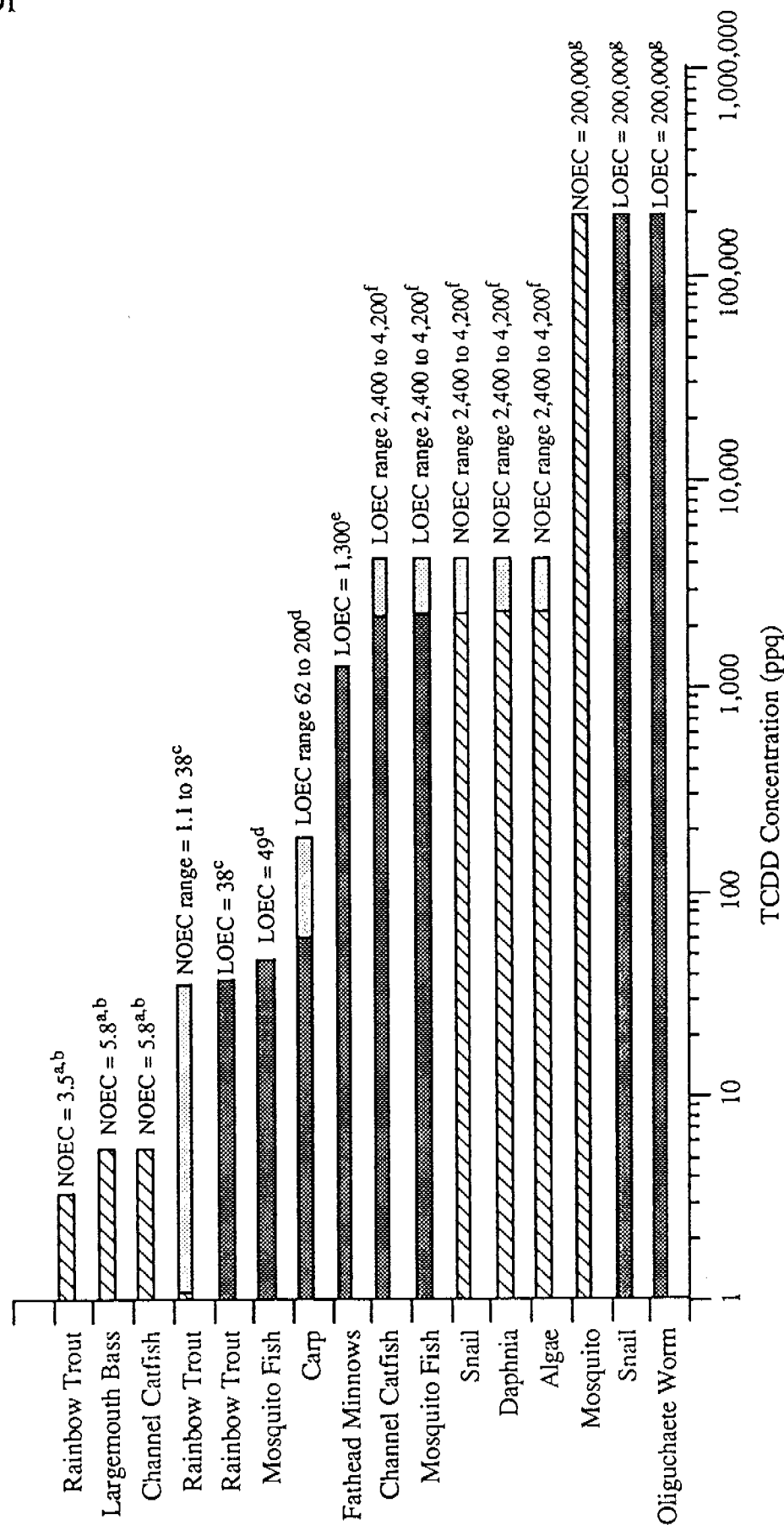
Species	Life Stage Exposed	Exposure Duration (days)	Test Duration (days)	Carrier Solvent	Nominal (Measured) TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration (ppq)	Effects ^a	Reference
Carp <i>Cyprinus carpio</i>	1-yr old (15 g)	71	132	dimethyl-formamide	0; 200 (62)	ND	62 - 200	Mortality ^b , signs of overt toxicity ^b , extensive pathology ^b	Cook et al., 1991
Channel catfish <i>Ictalurus punctatus</i>	fingerling	20	20	benzene	(2,400 - 4,200)	ND	2,400 - 4,200	Mortality ^b , erratic swimming ^b , fin necrosis ^b , anal and lower jaw hemorrhaging ^b	Yockim et al., 1978
Fathead minnow <i>Pimephales promelas</i>	juvenile (0.5 - 1.0 g)	28	48	acetone	(0; 1,700; 6,700; 63,000)	ND	1,700	Mortality ^b	Adams et al., 1986
Fathead minnow <i>Pimephales promelas</i>	1 g	71	132	dimethyl-formamide	0; 200 (49 or 67)	ND	49 - 200	"Variety of toxic signs" ^b	Cook et al., 1991
Mosquito fish <i>Gambusia affinis</i>	NR	15	15	benzene	(2,400 - 4,200)	ND	2,400 - 4,200	Mortality ^b , nasal hemorrhaging ^b , listless swimming ^b	Yockim et al., 1978
Rainbow trout <i>Salmo gairdneri</i>	fry (0.38 g)	28	56	acetone	0 (1.1); 115 (38); 231 (79); 463 (176); 925 (382); 1,850 (789) to authors	Between 1.1 and 38, according to authors	38	Reduced growth and survival ^c , altered behavioral responses ^b	Mehrle et al., 1988
Algae <i>Oedogonium cardiacum</i>	NR	32	32	benzene	(2,400 - 4,200)	2,400 - 4,200	ND	No apparent adverse effects ^b	Yockim et al., 1978
Mosquito <i>Aedes aegypti</i>	larvae	17	30	acetone	0; 200,000	200,000	ND	No effects on pupation ^b	Miller et al., 1973

Table 3-2. Effects of Chronic Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water (Continued)

Species	Life Stage Exposed	Exposure Duration (days)	Test Duration (days)	Carrier Solvent	Nominal (Measured) TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration ^a (ppq)	Effects ^a	Reference
Oligochaete worm <i>Paranais sp.</i>	adult	55	55	acetone	0; 200,000	ND	200,000	Reduced number of worms ^c	Miller et al., 1973
Snail <i>Helosoma sp.</i>	NR	32	32	benzene	(2,400 - 4,000)	2,400 - 4,200	ND	No apparent adverse effects ^b	Yockim et al., 1978
Snail <i>Physa sp.</i>	adult	36	48	acetone	0; 200,000	ND	200,000	Reduced snail hatch ^d	Miller et al., 1973
Water flea <i>Daphnia magna</i>	NR	32	32	benzene	(2,400 - 4,200)	2,400 - 4,200	ND	No apparent adverse effects ^b	Yockim et al., 1978

- a. Lowest-Observed-Effect-Concentration (LOEC) given in table relates to the most sensitive effect reported by the authors. The effects listed occurred either at the LOEC or at higher TCDD concentrations used in the study.
 b. Statistical analysis was not reported.
 c. Statistically significant.
 d. Effect was reported, but was not statistically significant.
 ND = Not determined
 NR = Not reported

Figure 3-2. Summary of Effect Levels from Chronic Exposure Studies of Aquatic Organisms to TCDD in Water



a. Highest concentrations tested, actual NOEC may be higher.

References:

- b. NCASI, 1991
c. Mehrle et al., 1988

- d. Cook et al., 1991
e. Adams et al., 1986

- f. Yockim et al., 1978
g. Miller et al., 1973

total volume of water in the system), as well as measured concentrations. Lowest-Observed-Effect-Concentrations (LOECs) and No-Observed-Effect-Concentrations (NOECs) were determined from these studies and are included in the summary tables. Table 3-3 summarizes the studies in which exposure was through routes other than the ambient water or in which LOECs and NOECs are expressed on a body burden basis. The toxicity studies are discussed in the following sections.

3.1 Effects of Acute Exposure to TCDD in Water

Concentrations of 7,100 ppq TCDD and greater caused significant mortality to fathead minnows exposed for 1 to 4 days in a static renewal system (Table 3-1) (Adams et al., 1986). Concentrations of 120 and 720 ppq TCDD did not result in increased mortality during the 60-day observation period.

In an additional experiment, Adams et al. (1986) exposed fathead minnows to TCDD concentrations of 1,700 ppq to 630,000 ppq for 28 days. The authors report a LC_{50} of 1,700 ppq, however no statistical analysis was performed.

Guppies exposed to 100,000 ppq TCDD or greater for 120 hours exhibited increased mortality (Table 3-1) (Miller et al., 1973; Norris and Miller, 1974). All treated fish died within 37 days following the start of the exposure period, compared to no deaths in the control group. Survival time was reported to be significantly and positively correlated with body length, suggesting that smaller fish are considerably more sensitive to TCDD than larger fish. The authors reported a declining interest in swimming and feeding approximately 1 week after initial exposure. Fish that survived greater than 10 days exhibited fin necrosis.

Miller et al. (1973) fed young rainbow trout a diet containing 0, 0.0063 ng, 6.3 ng, and 6,300 ng TCDD per week for four weeks. Mortality and growth parameters were measured. There was no significant mortality in any of the exposure groups. Significant differences in growth were only observed in the high dose group (6,300 ng TCDD per week).

Wisk and Cooper (1990) exposed embryos of the Japanese medaka to TCDD in a static renewal system (Table 3-1). A concentration-related increased incidence of embryos with lesions was observed at water concentrations of 400 ppq (the lowest concentration tested) and greater. The

Table 3-3. Effects of Exposure of Fish and Other Aquatic Organisms to TCDD via Several Exposure Routes

Species	Life Stage Exposed	Exposure Duration	Test Duration	Exposure Route	Dose Regimen	No-Observed-Effect-Concentration or Level ^a	Lowest-Observed-Effect-Concentration or Level ^a	LD50	Effects ^a	Reference
Bluegill <i>Lepomis macrochirus</i>	30 g	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	1 µg/kg	5 µg/kg	16 µg/kg	Mortality ^b , decreased body weight gain ^c , fin necrosis ^d , cutaneous hemorrhage ^d	Kleeman et al., 1988
Bullhead <i>Ictalurus melas</i>	6 g	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	1 µg/kg	5 µg/kg	5 µg/kg	Mortality ^b , decreased body weight gain ^c , fin necrosis ^d	Kleeman et al., 1988
Carp <i>Cyprinus carpio</i>	20 g	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	1 µg/kg ^f	5 µg/kg ^f	3 µg/kg	Mortality ^b , decreased body weight gain ^e , fin necrosis ^d , cutaneous hemorrhage ^d , cutaneous hyperpigmentation ^d	Kleeman et al., 1988
Coho Salmon <i>Oncorhynchus kisuich</i>	young	24, 48, 96 hrs	114 days	water	0; 0.054; 0.54; 5.4; 54.0 ng/g wet wt. fish	0.54-5.4 ng/g wet wt. fish	0.054 ng/g wet wt. fish	ND	Feeding rate, survival ^e and reduced growth ^c	Miller et al., 1979
Guppy <i>Poecilia reticulatus</i>	8-12 mm	24 hrs	69 days	water	0; 0.08; 0.8; 8.0; 80.0 ng/g wet wt. fish	0.08 ng/g wet wt. fish	0.8 ng/g wet wt. fish	ND	Incidence of fin necrosis ^c	Miller et al., 1979

Table 3-3. Effects of Exposure of Fish and Other Aquatic Organisms to TCDD via Several Exposure Routes (Continued)

Species	Life Stage Exposed	Exposure Duration	Test Duration	Exposure Route	Dose Regimen	No-Observed-Effect-Concentration or Level ^a	Lowest-Observed-Effect-Concentration or Level ^a	LD50	Effects ^a	Reference
Lake trout <i>Salvelinus namaycush</i>	eggs	48 hrs	NR	water	0; 34; 55; 121; 226; 302 pg/g egg weight	34 pg/g	55 pg/g	65 pg/kg	Sac fry mortality ^d , hatchability ^d , edema ^d , hemorrhages ^d	Cook et al., 1991
Largemouth bass <i>Micropterus salmoides</i>	7 g	single dose	80 days	ip injection	0, 1, 5, 25, 125 µg/kg	5 µg/kg	25 µg/kg	11 µg/kg	Mortality ^b , decreased body weight gain ^c , fin necrosis ^d , cutaneous hyperpigmentation ^d	Kleeman et al., 1988
Rainbow trout <i>Salmo gairdneri</i>	fingering (3 - 7 g)	13 weeks	26 weeks	feeding	0; 494 pg/g diet	494 pg/g diet	ND	ND	No signs of TCDD toxicity, reduced growth rate, or increase in relative lethality ^d	Kleeman et al., 1986a
Rainbow trout <i>Salmo gairdneri</i>	35 g	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	1 µg/kg	5 µg/kg	10 µg/kg	Mortality ^b , decreased body weight gain ^c , fin necrosis ^d	Kleeman et al., 1988
Rainbow trout <i>Salmo gairdneri</i>	juvenile (7.8 cm)	105 days	105 days	feeding	0; 2.3; 2,300; 2,300,000 pg/g diet (avg. intake of 0, 3.2 x 10 ⁻⁸ , 3.6 x 10 ⁻⁵ , 2.1 x 10 ⁻² µg/g fish)	2,300 pg/g diet	2,300,000 pg/g diet	ND	Reduced food consumption ^d , reduced body weight ^c , fin erosion ^d , reduced survival ^d , liver pathology ^d	Hawkes and Norris, 1977

Table 3-3. Effects of Exposure of Fish and Other Aquatic Organisms to TCDD via Several Exposure Routes (Continued)

Species	Life Stage Exposed	Exposure Duration	Test Duration	Exposure Route	Dose Regimen	No-Observed-Effect-Concentration or Level ^a	Lowest-Observed-Effect-Concentration or Level ^a	LD50	Effects ^a	Reference
Rainbow trout <i>Salmo gairdneri</i>	juvenile (6 - 10g, 25 - 45 g, 100 - 250 g)	single dose	up to 80 days	ip injection	0; 0.1; 1; 5; 25; 125 µg/kg	ND	1 µg/kg	10 µg/kg	Leukopenia and thrombocytopenia ^c , mortality ^b , reduced body weight gain ^c , fin necrosis ^d , lymphomyeloid lesions ^d , epithelial lesions ^d	Spitsbergen et al., 1988a
Rainbow trout <i>Salmo gairdneri</i>	1.6 g	once a week	4 weeks	feeding	0; 0.0063; 6.3; 6,300 ng	6.3 ng	6,300 ng	ND	Mortality and growth ^c	Miller et al., 1973
Yellow perch <i>Perca flavescens</i>	fingerling (3 - 6 g)	13 weeks	26 weeks	feeding	0; 494 pg/g diet	494 pg/g diet	ND	ND	No effects on growth rate or lethality, or signs of TCDD toxicity ^d	Kleeman et al., 1986b
Yellow perch <i>Perca flavescens</i>	40 g	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	1 µg/kg	5 µg/kg	3 µg/kg	Mortality ^b , decreased body weight gain ^c , fin necrosis ^d , cutaneous hemorrhaged	Kleeman et al., 1988

Table 3-3. Effects of Exposure of Fish and Other Aquatic Organisms to TCDD via Several Exposure Routes (Continued)

Species	Life Stage Exposed	Exposure Duration	Test Duration	Exposure Route	Dose Regimen	No-Observed-Effect-Concentration or Level	Lowest-Observed-Effect-Concentration or Level ^a	LD50	Effects ^a	Reference
Yellow perch <i>Perca flavescens</i>	juvenile (20 g, 40 g)	single dose	80 days	ip injection	0; 1; 5; 25; 125 µg/kg	ND	1 µg/kg	3 µg/kg	Mortality ^b , reduced body weight gain ^c , fin necrosis ^d , hemorrhages ^d , ascites ^d , lymphomyeloid lesions ^d , cardiac lesions ^d , epithelial lesions including hepatocyte, lipidosis ^d	Spitsbergen et al., 1988b
Frog <i>Rana catesbiana</i>	tadpoles	single dose	50 days	ip injection	0; 25; 50; 100; 200; 1,000 µg/kg	1,000 µg/kg	ND	ND	No effects on survival ^d , metamorphosis ^d , or histology ^d	Beatty et al., 1976
Frog <i>Rana catesbiana</i>	adult (150 - 250 g)	single dose	35 days	ip injection	0; 50; 100; 250; 500 µg/kg	250 µg/kg	500 µg/kg	ND	Temporary reduced food consumption ^d , no effects on survival or histology ^d	Beatty et al., 1976

a. Lowest-Observed-Effect-Concentration (LOEC) given in table relates to the most sensitive effect reported by the authors. The effects listed occurred either at the LOEC or at higher doses.

b. LD50s were statistically derived; statistical analysis was not reported for comparison of treatment groups to controls.

c. Statistically significant.

d. Statistical analysis was not reported.

e. Effect was reported by authors but was not statistically significant.

f. Although cutaneous hyperpigmentation was observed at 1 µg/kg, it is not clear whether this effect was significant.

ND = Not determined

NR = Not reported

EC50s for embryos with lesions and embryos with severe lesions were reported to be 3,500 and 14,000 ppq, respectively. The authors reported a concentration-dependent decrease in hatching incidence and a concentration-dependent increase in embryos that were dead by 3-day posthatch. The EC50 to prevent hatching was 14,000 ppq TCDD, and the LC50 for survival to 3-day posthatch was 9,000 ppq TCDD. In a separate experiment, the EC50 for embryos with lesions was reported to be 2,200 ppq TCDD. Based on a calculated ED50 for lesions of 0.24 pg TCDD/mg of dechorionated embryo (0.24 ppb), the authors noted that the Japanese medaka embryo is one of the most sensitive animals to TCDD. From experiments in which embryos were exposed to TCDD beginning on different days of embryonic development, Wisk and Cooper (1990) observed that the sensitive period for toxicity was during liver formation on days 4 and 5 of development.

Pike eggs exposed to TCDD at concentrations of 100 ppq and greater for 96 hours were reported to exhibit retarded egg development (Table 3-1) (Helder, 1980). Growth of fry was significantly reduced for several weeks following exposure to TCDD at all the concentrations tested. While egg mortality was not influenced by TCDD treatment, dose-related mortality of yolk sac and swimming fry was reported. Generalized edemas and pathological changes in the liver were also observed.

Helder (1981) found that exposure of rainbow trout eggs to 100 ppq TCDD (the lowest water concentration tested) for 96 hours resulted in significant growth retardation after 72 days (Table 3-1). Where eggs were exposed to 1,000 or 10,000 ppq TCDD, yolk sac fry developed edemas, and mortality was significantly greater than controls. In the 1,000 and 10,000 ppq TCDD treatment groups, teratologic defects were observed 12 weeks after fertilization. Yolk sac fry exposed to 1,000 ppq TCDD after hatching showed similar effects to those of fry exposed to TCDD at the egg stage (Helder, 1981). Juvenile rainbow trout exposed to 10,000 or 100,000 ppq TCDD for 96 hours exhibited growth retardation and showed slight edematous changes (Helder, 1981).

Branson et al. (1985) reported that rainbow trout exposed to 107,000 ppq TCDD for 6 hours exhibited an increase in relative liver weights by day 42 and a decrease in body weight gain (Table 3-1).

Cook et al. (1991) exposed fertilized eggs from Lake Superior lake trout to TCDD in water for 48 hours (Table 3-3). Rather than report the water concentrations used, the authors reported the

concentrations of TCDD accumulated in the eggs by the end of the exposure period; these ranged from 34 to 302 pg/g for the treatment groups. After the 48-hour exposure, the eggs were removed to TCDD-free water. The authors reported that embryo development and mortality was not affected by TCDD until about one week before the onset of hatching, at which time embryos containing the highest TCDD concentration exhibited toxicity. Hatchability of eggs was reported to be decreased at whole egg TCDD concentrations greater or equal to 226 pg/g. Cook et al. found that the sac fry stage exhibited the greatest mortality in their study. The No-Observable-Adverse-Effect-Level (NOAEL) for sac fry mortality was 34 pg/g in the egg, while the Lowest-Observable-Adverse-Effect-Level (LOAEL) was 55 pg/g. An LD50 of 65 pg/g was calculated based on sac fry mortality. Sac fry that died were reported to develop subcutaneous yolk sac edema and hemorrhages that morphologically resemble blue sac disease.

Daphnia magna of three different age groups (less than or equal 1 day, 7 days, 21 days) exposed to TCDD concentrations ranging from 200 to 1,030,000 ppq were not adversely affected following 48-hour exposures and a 1-week observation period (Table 3-1) (Adams et al., 1986).

3.2 Effects of Chronic Exposure to TCDD in Water

Yockim et al. (1978) exposed a number of aquatic organisms to TCDD in a recirculating static model ecosystem in which soil was treated with TCDD and flooded with water (Table 3-2). *Daphnia*, snails, and algae were placed in one chamber and mosquito fish in another chamber one day after flooding the soils. Additional mosquito fish were added on day 15 following the death of the first group. All remaining organisms were harvested after 32 days. Channel catfish fingerlings were added on day 32. Water concentrations of TCDD in the model ecosystem tanks ranged from 2,400 to 4,200 ppq TCDD with an average of approximately 3,000 ppq. The authors reported that nasal hemorrhaging and listless swimming accompanied death in exposed mosquito fish; all mosquito fish died within 15 days of exposure. All exposed fingerling channel catfish died after 15 to 20 days of exposure compared to no deaths in the control fish. Erratic swimming, fin necrosis, and anal and lower jaw hemorrhaging were reported to accompany death in the catfish. However, algae, *Daphnia*, and snails exposed for 32 days were reported to have no apparent adverse effects, as measured by feeding, growth, and reproductive activity (Yockim et al., 1978).

Juvenile fathead minnows exposed to 1,700 to 63,000 ppq TCDD for 28 days in a static renewal system exhibited increased mortality at all concentrations (Table 3-2) (Adams et al., 1986). The authors reported a 28-day LC50 of 1,700 ppq. The authors noted that the TCDD whole-body residues observed in dead fish ranged from 16.7 µg/kg to 2,042 µg/kg.

Exposure of mosquito larvae to TCDD at an initial water concentration of 200,000 ppq for 17 days produced no effects on pupation (Table 3-2) (Miller et al., 1973). A 36-day exposure of snails to water initially containing 200,000 ppq TCDD resulted in a reduced snail hatch (Miller et al., 1973). No significant differences were observed in the survival of adult or juvenile snails in this study. Miller et al. (1973) exposed adult Oligochaete worms for 55 days at an initial concentration of 200,000 ppq TCDD. The exposure resulted in a significant decrease in number of worms at the end of the exposure period.

In a study of rainbow trout fry exposed to TCDD for 28 days using an intermittent-flow proportional diluter, Mehrle et al. (1988) found that adverse effects were observed at all dose levels (Table 3-2). Significant mortality was observed at concentrations of 176, 382, and 789 ppq TCDD within the 28-day exposure period. Mortality was also significantly increased at lower concentrations of 38 and 79 ppq TCDD during the 28-day depuration phase. The NOEC for this effect was determined to be less than the lowest exposure concentration of 38 ppq TCDD. Mehrle et al. (1988) determined that the control solution contained 1.1 ppq TCDD (as detected by radiometric analysis). Based on the 5% mortality observed in the control group during the exposure phase and much of the depuration phase, the authors suggested that the NOEC for mortality was between 1.1 and 38 ppq TCDD. The authors calculated a 56-day LC50 of 46 ppq based on the combined mortality data for the exposure and depuration phases. Growth was significantly decreased at all TCDD concentrations after 28 days of exposure, resulting in a NOEC less than 38 ppq TCDD. Mehrle et al. (1988) reported behavioral changes in all treatment groups by the end of the 28-day exposure, compared to normal behavior of the control fish. The whole-body residue of TCDD in fish exposed to 38 pg/L for 28 days was approximately 980 pg/g.

NCASI (1991) investigated the toxicity of TCDD to rainbow trout using an experimental stream in which exposures from the water column, sediments, and food organisms were considered. The results of this study indicate that the NOECs for cold-water species (including rainbow trout) were on the order of 3.5 ppq TCDD. For warm-water species, NOECs were estimated to be 1.6 ppq

TCDD based on long-term survival of juvenile bass (NCASI, 1991). More recent work by NCASI, based on actual effluent levels, resulted in a NOEC of 5.8 ppq for long-term growth and survival of adult largemouth bass and channel catfish (NCASI, 1991). The actual NOECs are probably larger than these levels as NCASI has indicated that these were the highest concentrations tested. The specific methodologies of these studies are not available as NCASI is in the process of preparing their report.

Cook et al. (1991) reported in a manuscript (preprint) that carp and fathead minnows exposed to TCDD for 71 days and observed for an additional 61 days exhibited toxicity (Table 3-2). A dimethylformamide solution of TCDD was continuously fed into the aquaria during the exposure period. During the exposure period, the nominal TCDD water concentration of 200 pg/L in the aquaria decreased to 62 pg/L for exposed carp and 49 or 67 pg/L for exposed fathead minnows. One of the three treatment groups of carp was previously exposed to 1,2,3,4-TCDD, 1,3,6,8-TCDD, and 1,3,7,9-TCDD. The authors reported that both carp and fathead minnows exhibited toxic effects following TCDD exposure. Carp were reported to be more severely affected than fathead minnows even though they showed lower whole-body TCDD concentrations. However, the specific incidences of effects in the treatment groups and the control groups were not provided in the manuscript. In addition to mortality, carp reportedly showed signs of overt toxicity including fin darkening, fin erosion, caudal fin deformation, hemorrhages in lateral body wall, lateral line lesions, cranial deformation, edema, difficulty swimming, body wall ulcers, and exophthalmia. Extensive pathology was reported in histological analyses of liver, spleen, gill, fins, and gastrointestinal tract, however, the specific findings were not reported in this manuscript. Carp were reported to have a whole-body concentration of approximately 2,200 pg/g at the end of the exposure period. The nature of the toxic effects observed in fathead minnows in this study was not provided in the manuscript.

3.3 Other Toxicity Studies

Juvenile rainbow trout were exposed to TCDD in their diets at concentrations of 2.3, 2,300, and 2,300,000 pg/g (ppt) of diet for 6 days per week for a total of 105 days resulting in an average intake of 3.2×10^{-8} , 3.6×10^{-5} , and 2.1×10^{-2} µg/g fish, respectively (Table 3-3) (Hawkes and Norris, 1977). No effects on mortality, food consumption, growth, or fin erosion were observed in trout at 2.3 or 2,300 pg/g diet. The highest dose level of 2,300,000 pg/g TCDD in the diet was

reported to cause increased mortality, reduced feeding activity and growth, and increased fin erosion and liver pathology. The authors noted that the "no-effect" level for survival, growth, feeding activity, and fin erosion in rainbow trout treated with TCDD orally is between the levels of 2,300 and 2,300,000 pg/g (ppt).

In two separate studies Kleeman and coworkers (1986b, 1988b) examined metabolism and disposition of TCDD in rainbow trout and yellow perch. In both experiments, fingerling fish were fed a diet containing 494 ppt [³H]TCDD for 13 weeks followed by 13 weeks of the same diet without TCDD. Neither rainbow trout or yellow perch showed any evidence of overt toxicity as measured by fin necrosis, reduced growth, cutaneous hemorrhage, or an increase in lethality.

Kleeman et al. (1988) determined LD50s at 80 days post-treatment for a number of fish species: rainbow trout - 10 µg/kg, yellow perch - 3 µg/kg, carp - 3 µg/kg, bluegill - 16 µg/kg, largemouth bass - 11 µg/kg, and bullhead - 5 µg/kg (Table 3-3). At the highest dose of 125 µg/kg TCDD, all species showed a latency period of 1 to 4 weeks prior to death. Latency periods were longer at lower lethal doses. All fish species exhibited fin necrosis. Decreases in body weight gain were found to be dependent on the species and the dose. Cutaneous hemorrhage was found only in perch, carp, and bluegill treated with TCDD, while cutaneous hyperpigmentation was observed only in carp and largemouth bass treated with TCDD.

Spitsbergen et al. (1988a) exposed juvenile rainbow trout to TCDD by intraperitoneal injection at doses ranging from 0.1 to 125 µg/kg. A significant depression in the rate of body weight gain was observed at doses of 5 µg/kg and greater. Mortality was increased to 20% at 5 µg/kg but was not affected at 1 µg/kg. The authors reported the 80-day LD50 of TCDD to be 10 µg/kg. Rainbow trout treated with 10 µg/kg exhibited gross and microscopic lesions while trout treated with 1 or 0.1 µg/kg did not exhibit lesions. Morphological lesions included those of the epithelial and lymphomyeloid tissues. Fish treated with 10 µg/kg showed marked leukopenia and thrombocytopenia; fish treated with 1 µg/kg showed a moderate depression of these cell numbers. This finding led the authors to conclude that hematologic parameters may be one of the most sensitive indicators of exposure to TCDD in rainbow trout.

Yellow perch were exposed to TCDD at doses of 1 to 125 µg/kg intraperitoneally (Table 3-3) (Spitsbergen et al., 1988b). A dose-related increase in lethality was observed. At 5 µg/kg,

mortality was approximately 80% at 80 days after injection, while fish that received 1 µg/kg did not exhibit mortality. The authors calculated the 80-day LD50 of TCDD to be 3 µg/kg. Significant loss of body weight was observed in perch treated with 5 µg/kg, but was not observed in perch treated with 1 µg/kg. Perch administered 5 µg/kg TCDD or greater exhibited fin necrosis, petechial cutaneous hemorrhage, and ascites. Lymphomyeloid and epithelial tissues were identified as the primary targets for TCDD-induced lesions in yellow perch. The lowest dose which was associated with histopathologic lesions (hepatocyte lipidosis) was 1 µg/kg.

Beatty et al. (1976) exposed tadpoles to 25 to 1,000 µg/kg TCDD intraperitoneally (Table 3-3). In this study, no effects on mortality, metamorphosis, or histology were observed at any dose level. Adult bullfrogs treated with TCDD were reported to show no effects on mortality and no significant lesions. Though somewhat lessened food intake was observed in frogs receiving 500 µg/kg during the initial part of the study, food consumption in this treatment group was not different from that of controls by the end of the experiment.

Young coho salmon were exposed to TCDD water concentrations of 0.054 to 54.0 ng/g wet weight fish for 24, 48, or 96 hours (Miller et al., 1979). Food consumption, weight gain, and survival were measured during a 114-day post exposure period. Coho salmon exposed to 54.0 ng/g wet weight fish for 96 hours showed a significant difference in feeding 7 days following exposure. Those fish exposed to 54.0 ng/g wet weight fish for 48 hours showed a significant response after 15 days and similarly, the group exposed for 24 hours at 54.0 ng/g wet weight fish showed a significant response 20 days after exposure.

Growth and survival were not significantly influenced by exposure duration, and weight gain decreased with increasing TCDD concentration. Coho salmon exposed to 5.4 ng/g wet weight fish showed significant differences 56 days following exposure. Survival was measured at 60 days and 144 days following exposure. Mortality reached 100% for the high dose group (54.0 ng/wet weight fish) after 60 days and was as high as 85% for the 5.4 ng/wet weight fish dose group. No significant mortality was evident for doses at or below 0.54 ng/g wet weight fish at 60 or 114 days. The threshold response level for growth and survival in young coho salmon for exposure periods up to 96 hours is between 0.54 and 5.4 ng/g wet weight fish (Miller et al., 1979). A conversion to a water level was not possible based on the information provided.

Miller et al. (1979) also exposed guppies to concentrations ranging from 0.08 to 80 ng/g wet weight fish for 24 hours. Incidence of fish necrosis was measured for 69 days following exposure. A significant incidence of fin necrosis was observed in fish exposed to 0.8 ng/g wet weight fish or higher 42 days following exposure. Similar to Miller and coworkers' (1979) coho salmon study, TCDD water concentrations were not reported.

3.4 Conclusion

Based on the acute toxicity studies described above, the Lowest-Observed-Effect-Concentrations (LOECs) for short-term exposures to different life stages of fish have been reported to range from 100 to 107,000 ppq (Miller et al., 1973, 1979; Norris and Miller 1974; Helder, 1980, 1981; Branson et al., 1983, 1985; Adams et al., 1986; Cooper et al., 1986; Wisk and Cooper, 1990). The No-Observed-Effect-Concentrations (NOECs) for short-term exposures of fish have been reported to range from 10 to 1,050 ppq TCDD (Miller et al., 1973, 1979; Adams et al., 1986).

For long-term exposures to fish, LOECs have ranged from 38 to approximately 3,000 ppq, while NOECs have ranged from 1.1 to approximately 3,000 ppq (Helder, 1980, 1981; Yockim et al., 1978; Mehrle et al., 1988; Adams et al., 1986). The experimental findings of Mehrle et al. (1988) indicate no adverse effects in any of the control rainbow trout which were exposed to a measured TCDD level of 1.1 ppq. The authors concluded that the NOEC was between 1.1 and 38 ppq. A NOEC in this range is supported by recent experimental stream studies conducted by NCASI (1991), which indicate a NOEC of 3.5 ppq for cold-water species and a NOEC of 1.6 ppq for warm-water species. More recent data reported by NCASI (1991) provide a NOEC of 5.8 ppq for warm-water species.

Examination of several of the toxicity studies conducted on TCDD provides information regarding the whole-body concentrations in fish at which toxicity is observed. Carp containing approximately 2,200 pg/g (ppt) of TCDD by the end of a 71-day exposure exhibited increased mortality, signs of overt toxicity, and pathological changes (Cook et al., 1991). Carp chronically exposed to TCDD and containing a whole-body TCDD concentration in excess of 1,000 pg/g (ppt) showed ascites, subcutaneous hemorrhage, and fin necrosis (Cook et al., 1986). Adverse effects on growth, survival, and behavior were observed in rainbow trout swim-up fry that had accumulated 980 pg/g (ppt) at the end of a 28-day exposure (Mehrle et al., 1988). The most

sensitive indicators of toxicity in juvenile rainbow trout were leukopenia and thrombocytopenia that occurred following a single dose of 1 µg/kg (1,000 pg/g) (Spitsbergen et al., 1988a).

The available data suggest fish are more sensitive to TCDD than other aquatic biota. Studies conducted by Helder (1980, 1981) show fish eggs to be the most sensitive life stage with a reported acute NOEC of 100 ppq for rainbow trout and pike eggs. Cook et al. (1991) reported a LOAEL of 55 pg/g egg concentration for lake trout. The acute NOEC was *Daphnia* of 1,030,000 ppq TCDD (Adams et al., 1986). Long-term exposure to approximately 3,000 ppq TCDD resulted in no adverse effects in algae, snails, and *Daphnia* (Yockim et al., 1978).

The review of currently available data regarding toxicity suggests that ambient water concentrations of 3.5 to 5.8 ppq TCDD would likely be protective of freshwater aquatic organisms. This conclusion is based on the weight of evidence provided by the NCASI (1991) experimental stream studies which reported a NOEC of 3.5 ppq for cold-water species and 5.8 ppq for warm-water species based upon a nominal water concentration. AWQS compliance is determined by a calculated nominal water concentration, (i.e., effluent concentration diluted by receiving water body) rather than on an actual measured ambient water concentrations. Laboratory experiments have demonstrated that measured water concentrations are consistently lower than nominal concentrations. For example, in the study conducted by Mehrle et al. (1988), a nominal concentration of 115 ppq resulted in a measured concentration of only 38 ppq. Based on this approach, TCDD concentrations higher than 5.8 ppq would likely be protective of aquatic life.

4.0 BIOCONCENTRATION/BIOACCUMULATION OF TCDD IN FISH

In aquatic environments, the primary route of potential human exposure to TCDD is through the ingestion of fish tissue (EPA, 1984). Due to the importance of this pathway, an ambient water quality standard based upon fish ingestion will be more restrictive than those based upon other considerations. When EPA developed its ambient water quality criteria for TCDD (EPA, 1984), it used a bioconcentration factor (BCF) approach to predict how much dioxin a fish will accumulate from its surroundings. The predicted concentration of TCDD in fish, used in conjunction with an estimated rate of fish consumption, provided an estimate of potential human exposure to dioxin via this pathway. The BCF approach, however, addresses only the uptake of dissolved compounds across the membranous gill surfaces (EPA, 1989b). BCFs are calculated by dividing the concentration of a chemical in fish tissue by its dissolved concentration in water (EPA, 1989c). Recently, much scientific discussion has focused on the apparent inappropriateness of using the BCF approach in the ambient water quality equation (Keenan et al., 1990b; Rifkin and LaKind, 1991; LaKind and Rifkin, 1990). Criticism of the BCF is related to the observation that hydrophobic substances like TCDD have extremely low dissolved concentrations in water because of their very strong tendency to sorb to organic matter.

In order to accurately predict how much dioxin a fish will accumulate from its surroundings, it is essential that an appropriate "accumulation factor" be used in the equation for a water quality standard (Rifkin and LaKind, 1991). Understanding what constitutes a suitable factor is fundamental to deriving scientifically based water quality standards. The ramifications of choosing incorrect factors are significant and will be discussed in the following sections of this chapter.

4.1 Measuring Dioxin Accumulation in Fish

Historically, scientists have used several approaches to predict the uptake and accumulation of chemicals in fish. Two major approaches have been used to estimate the tendency of an animal to accumulate environmental contaminants: bioconcentration and bioaccumulation. Methods for estimating bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) include the use of direct measurement *in vivo*, or the prediction of chemical behavior in a biological system based on physicochemical constants.

Until recently, the development of a BCF has been the most common approach used by scientists to predict the concentrations of environmental contaminants in fish tissues. As discussed earlier, the BCF model addresses only the uptake of a compound by fish via the transfer of a dissolved compound across the membranous gill surfaces (EPA, 1989b). BCFs can be calculated by dividing the concentration of a chemical in the fish tissue by its dissolved concentration in water (EPA, 1989c). Studies conducted in the past few years, however, indicate that this model does not adequately characterize the behavior of lipophilic chemicals like dioxins, PCBs, and other organochlorine compounds. The hydrophobic nature of dioxin, combined with its great affinity for organic carbon, means that the amount of dioxin sorbed to organic matter far exceeds that dissolved in the aqueous environment (Rifkin and LaKind, 1991). A number of considerations, including the cross-sectional size of sorbed dioxin, the molecular weight, and solubility are important in limiting the ability of TCDD to penetrate the gills of aquatic organisms (McKim et al., 1985; Gobas et al., 1987; Rifkin and LaKind, 1991). Therefore, in the natural environment, where an insignificant fraction of dioxin is dissolved, bioconcentration is not the primary route of uptake of strongly hydrophobic chemicals, and BCFs are not good predictors of fish tissue levels.

Scientific evidence in recent years indicates that the body burden of dioxin in fish comes primarily from ingestion of food and sediment (Kenaga and Goring, 1980; Spacie and Hamelik, 1982; Rand and Petrocelli, 1985; Gobas et al., 1987; Kuehl et al., 1987; Cook, 1990). For example, results reported by Cook et al. (1990) demonstrate that sediments and various food sources represent the most significant sources of dioxin found in lake trout from Lake Ontario. This is not surprising given that the majority of dioxin introduced into an aquatic system binds to sediment and is not usually detectable in the water column (Lodge and Cook, 1989). In the EPA's (1984) water quality criteria formula, however, the ingestion route of uptake is not considered. A factor other than the BCF should be incorporated into the equation for deriving a scientifically based water quality standard. This factor should, ideally, serve two functions:

- (1) realistically predict or estimate the accumulation of sorbed dioxin by aquatic organisms; and,
- (2) practically serve as an accumulation multiplier which can be implemented in a regulatory context for the purpose of establishing effluent permit limits.

One useful approach for developing an appropriate "accumulation factor" for dioxin and other hydrophobic compounds is the Bioavailability Index (BI). Coined by Kuehl et al. (1987) and further applied by Goeden and Smith (1989), the BI is defined as the ratio of the concentration of the contaminant in the lipid portion of the fish to the concentration in the organic carbon portion of the sediment (Kuehl et al., 1987; Goeden and Smith, 1989). The use of the BI in the water quality criteria equation would appear to be suitable for hydrophobic chemicals, like dioxin, where the uptake of the dissolved fraction of the chemical is insignificant. However, the implementation of the BI approach for regulatory purposes will require the development and use of a model to calculate the distribution of dioxin on a site-specific basis (Rifkin and LaKind, 1991). This requirement may prove to be impractical at the present time and, thereby, encourages the development of alternative "accumulation factors" that satisfy both prerequisites mentioned above.

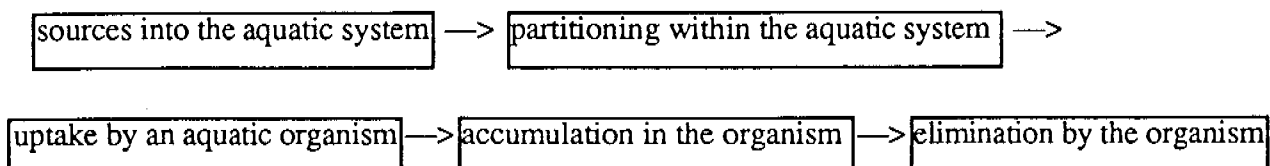
Although field research is still in progress, the bioaccumulation factor (BAF) approach and a variant of the BAF, which we have defined as the Regulatory Bioaccumulation Multiplier (RBM) (Sherman and Keenan, 1991), is the best available method for predicting dioxin uptake by fish while allowing regulatory agencies to directly calculate point source limits. It incorporates oral uptake from sediment, from food-chain sources, and from particulate-bound dioxin suspended in the water column. The bioaccumulation potential of dioxin can be adequately characterized by a BAF because uptake via ingestion and gill transfer, elimination, and changes resulting from fish growth can be incorporated into the model (Gobas et al., 1987, 1988, 1989; Spacie and Hamelik, 1982; Sijm and Opperhuizen, 1988).

4.2 Factors Affecting Accumulation in Fish

There are a number of interdependent factors which influence the potential for various chemicals to accumulate in the tissues of fish. Among these are the physicochemical characteristics of the chemical of concern, species differences, health status, age, sex, size, tissue lipid content, and rate of food intake (Spacie and Hamelik, 1982; Spigarelli et al., 1982; Rand and Petrocelli, 1985; Gobas et al., 1987). One of the most critical factors in evaluating the bioaccumulation potential of dioxin in fish is the lipid content of the species of concern. Consequently, there are several adjustments which must be considered before using laboratory-derived data in environmental modeling. The application of an intraspecies correction factor may be necessary if age, sex, and

health data indicate differences in lipid content within the same species of laboratory-raised and naturally-occurring fish. An interspecies correction factor may be needed when extrapolating from one species to another. A correction factor should also be used to adjust for the low lipid content of the fillet and the unequal partitioning of TCDD between edible and non-edible tissues.

In general, chemical accumulation in fish tissues and other aquatic organisms is a net balance between the rate of uptake and the rate of elimination/depuration. The rate of chemical uptake is primarily a function of the exposure concentration and the bioavailability of the compound in the environment. The elimination/depuration rate is primarily a time-dependent biological function. The pathways whereby fish or other aquatic organisms accumulate dioxin can be described in the following box model:



Fish can assimilate dioxin from three compartments of the aquatic system: through the water column, through incidental ingestion of sediments, and through ingestion of food material containing dioxin (Cook et al., 1990). The importance of each contributing component is, in part, determined by the physical and chemical properties of TCDD.

Once absorbed by the fish, dioxin will partition to various organs or be eliminated through the feces (Kleeman et al., 1986a,b). Due to its lipophilic nature, TCDD readily partitions to those organs with the highest fat or lipid content. The majority of TCDD will concentrate in the visceral fat and cranial fat (Kuehl et al., 1987; Kleeman et al., 1986a, 1986b). Skeletal muscle (fillet tissue) contains a low percentage of lipid and, therefore, accumulates the least amount of TCDD (Kuehl et al., 1987; Kleeman et al., 1986a, 1986b). Since the lipid content of a fish can vary with climate and seasonal water temperatures, cold-water fish generally will have a higher percentage of lipid in their tissues than warm-water fish. Seasonal temperature changes can lead to increased metabolism and reduction of lipid stores. When the fat and its associated dioxin are mobilized, dioxin will re-enter the blood stream and eventually may be eliminated through the excretory system.

According to Cook et al. (1990) uptake and depuration of TCDD can be described by first order kinetics equation:

$$\frac{dC_f}{dt} = k_1 C_w - k_2 C_f \quad (1)$$

where the change in the concentration of the chemical in fish over time is a function of the first order rate constant for bioaccumulation (k_1), first order rate constant for depuration (k_2), chemical exposure concentration (C_w), and the fish tissue concentration (C_f).

Equation (1) can be redefined as:

$$C_f = k_1/k_2 \times C_w \times (1 - e^{-k_2 t})$$

The primary parameter that influences dioxin accumulation (C_f) is the initial exposure concentration (C_w). It is evident from this equation that a decrease in the amount of dioxin which is discharged into the aquatic system will decrease the amount of dioxin available to the fish (C_w). As a result, the balance between uptake and depuration will be shifted and fish tissue dioxin levels will be expected to decrease with time.

The period of time that is necessary to detect a measurable reduction in the concentration of dioxin in fish tissues as a result of a reduction in effluent discharges from a suspected source to a water body is dependent upon several factors. Two factors, the biological half-life of dioxin in fish and the amount of dioxin stored in the various compartments of the aquatic system, are most significant. For example, half-life estimates reported by Kleeman et al. (1986a) ranged from 8 weeks in heart tissue to 15 weeks in skeletal muscle in rainbow trout. When whole-body elimination is considered, the half-life of TCDD has been shown to vary from 8 weeks in rainbow trout (Niimi and Oliver, 1986) to as much as 18 weeks in yellow perch (Kleeman et al. 1986b).

Empirical data on the behavior of chemicals in the environment indicate that a measurable reduction in the levels of dioxin in sediments may take several months after the levels of dioxin in industrial discharges to surface waters have been reduced. Over time, the sediment reservoir will be depleted through physicochemical exchange to the water column or through stochastic events such as spring

or storm scouring of the sediments. Natural deposition of new cleaner sediments over older deposits will also reduce the bioavailability of TCDD. Bottom dwelling fish are likely to show the slowest rate of TCDD reduction in their tissues due to their relatively high exposure to sediments.

4.3 Establishing a Regulatory Bioaccumulation Multiplier (RBM)

Bioaccumulation multipliers are derived from a ratio of the concentration of TCDD in the fish to the environmental concentration of TCDD. Oftentimes, when regulatory agencies have used BCFs or BAFs, there is confusion regarding exactly what fish concentration and what environmental concentrations have actually been measured. For example, with respect to the fish concentration; i.e., the numerator in the BAF ratio, one study reported the concentration in whole juvenile fish weighing less than half a gram each; there was no "edible" portion and lipid content was not reported (Adams et al., 1986). In another study the fish concentration referred to whole fish with 19% lipid (Cook et al., 1986, 1991). Other studies have reported the dioxin content of edible fish as containing 1% lipid. To compare BAF values from different studies, a consistent basis for expressing fish concentrations must be defined.

With respect to the environmental concentration; i.e., the denominator in the BAF ratio, a similar problem is apparent. Many studies use a measured concentration in water. In some cases the measurement is on a "whole" water sample; others are filtered or centrifuged. Sometimes a theoretical calculated "dissolved" concentration is used. Still others have used values based upon the amount of suspended particulate and the concentration of dioxin adsorbed on the particulate. A nominal concentration determined by the total amount of TCDD added to the system is also used. Therefore, to compare BAF values, a consistent basis for expressing the environmental or exposure concentration must also be defined.

The amount of dioxin in the effluent receiving water can be calculated according to the following equation:

$$C_w = C_e \times V_e / V_r \quad (1)$$

where:

C_w = Concentration of TCDD in the ambient water

C_e = Concentration of dioxin in the effluent

V_e = Volume of effluent discharged

V_r = Volume of river flow

Because the maximum acceptable level of dioxin in the receiving water is limited by the water quality criteria (WQC), the following equation is derived:

$$WQC = C_e \times V_e / V_r \quad (2)$$

where the WQC is a function of the total quantity of dioxin in an effluent (dissolved and absorbed) per unit volume of effluent (C_E), the effluent flow (V_E), and river flow (V_R).

A site-specific BAF can be derived using the following ratio:

$$BAF = C_f / C_E \quad (3)$$

where the C_f represents the concentration in fish tissue and C_E is the environmental concentration of TCDD.

Solving for the environmental concentration:

$$C_E = C_f / BAF \quad (4)$$

The environmental concentration is limited by the WQC. Thus,

$$WQC = C_f / BAF \quad (5)$$

This methodology has typically been used to define an acceptable human health-based environmental concentration or a water quality criteria.

By combining equations (2) and (5),

$$C_f / \text{BAF} = (C_e \times V_e) / V_r \quad (6)$$

Solving in terms of BAF:

$$\text{BAF} = \frac{C_f}{C_e \times V_e / V_r} \quad (7)$$

The BAF, when used for regulatory purposes, is defined as the dioxin concentration in the portion of fish consumed by humans, (i.e., fish fillet), divided by the total quantity of dioxin (dissolved and adsorbed) added to the water body per unit volume of water containing the fish. We have renamed this regulatory BAF with a new term, the Regulatory Bioaccumulation Multiplier, or RBM (Sherman and Keenan, 1991).

If the total quantity of dioxin added (dissolved and adsorbed) per unit volume of water is defined as the "nominal" concentration (C_N), then:

$$\begin{array}{l} \text{Regulatory} \\ \text{Bioaccumulation} \\ \text{Multiplier} \end{array} = \text{RBM} = \frac{C_f}{(C_e \times V_e / V_r)} = \frac{C_f}{C_N}$$

The derivation of an RBM is not as simplistic as dividing the fish concentration by the nominal river concentration. To properly use this equation to derive a site-specific RBM, the following criteria must be met.

1. The system must be in equilibrium or quasi-equilibrium.

TCDD concentration in fish are proportional to the environmental concentration (see discussion in Section 4.2), provided a sufficient length of time has elapsed since the last alteration in the environmental concentration; i.e., equilibrium is reached. A linear regression analysis is inappropriate when the aquatic system being investigated is not in equilibrium. This can occur after individual discharges of TCDD have been reduced and fish levels have not reached an equilibrium state with the new environmental concentration.

2. A demonstrated correlation must exist between fish concentration and nominal river concentration.

A correlation is required in order for the slope of the regression line to have any meaning. Often a correlation does not exist when nominal river concentrations are low; i.e., less than 0.5 ppq, or when anadromous fish species are sampled.

A positive intercept suggests that the background concentration of dioxin is not attributable to pulp and paper mill discharges. This is consistent with the detection of dioxin in fish from rivers with no industrial discharges (Mower, 1990).

If all conditions are met, then the RBM will be equal to the slope of the regression line. This indicates the increase in fish concentration related to the increase in the nominal river concentration.

The use of an RBM based upon the fish fillet and the nominal water concentrations allows regulatory agencies to directly calculate point source limits. This also obviates the need to apportion dioxin into different parts of the ecosystem; e.g., food, suspended sediment and "dissolved" in water.

A wide range of BCFs and BAFs have been reported in the literature. A detailed discussion of these studies is included in Appendix A. These widely varying BCFs and BAFs reported for dioxin in the scientific literature actually fall within a rather narrow range if expressed and compared on a consistent basis as defined by the RBM. Employing these consistent definitions, the RBMs generally fall below 5,000. For example, RBMs can be calculated from the "pure water" studies of Cook et al., (1990; 1991), Mehrle et al., (1988), Adams et al., (1986) and Branson et al., (1985). The average RBM normalizing to a fillet lipid content of 2.5% is 3,600 with a range from 600 to 6,440.

Reanalyzing the BCFs of 66,000 reported for carp (Cook et al., 1991), 128,000 for fathead minnows (Cook et al., 1991) and 1,357 for lake trout (Cook et al., 1990), RBMs of 5,680, 4,880, and 600, respectively, can be derived. Similarly, using the reported BCFs of 39,000 and 9,270 for rainbow trout (Mehrle et al., 1988; Branson et al., 1985) and 7,900 for fathead minnows

(Adams et al., 1986), RBMs of 6,400, 970 and 2,820, respectively, can be derived. An average of these estimated RBMs is 3,600.

All of these "pure water" laboratory studies were conducted under 'unnatural' conditions where no sediment was present and only low levels of suspended solids and organic carbon were available in the dilution water. Natural systems include sediment, suspended solids and increased amounts of organic carbon. These are likely to decrease the concentration of dissolved dioxin in the water, and thus, decrease the uptake of dioxin across gill surfaces while the presence of contaminated food, sediment, and suspended particulates may increase the uptake of dioxin via ingestion. In the Cook et al. (1990) simulated field studies, these factors were considered. In two specific experiments, Cook and coworkers exposed lake trout to contaminated food, water, and sediment. In the first experiment, TCDD levels approximated those found in Lake Ontario, and in the second experiment TCDD concentrations were approximately ten times greater than in Lake Ontario. Results of these studies indicated a BAF of between 7,700 and 11,000, respectively. However, when expressed as a Regulatory Bioaccumulation Multiplier (RBM) with a 2.5% lipid content and nominal water concentrations based on the total added dioxin, the BAFs may be as low as 450 and 310. When only suspended and dissolved dioxin are included in the nominal concentration, the RBMs become 4,580 and 3,210.

4.4 Conclusions

A wide variety of BCFs and BAFs for dioxin have been reported in the literature (Adams et al., 1986; Mehrle et al., 1988; Branson et al., 1985; Cook et al. 1990, 1991). Differences between reported BCFs and BAFs are not surprising as they are due, in part, to a number of environmental factors that affect the fate of dioxin in an aquatic ecosystem and within a fish. These factors include the amount of organic material in the water column and in the sediment, water flow rates, fish lipid content, as well as the age, size, sex, and rate of food intake by each species of concern (Spacie and Hamelik, 1982; Spigarelli et al., 1982; Rand and Petrocelli, 1985; Gobas et al., 1987). Differences in the experimental methodologies used in laboratory or field studies conducted over the past ten years to derive BCFs or BAFs, also have added to the wide range of values reported in the literature. Before a definitive accumulation factor can be assigned for dioxin, it is essential that the terminology be defined and standardized for use in a regulatory context.

Although a wide range of accumulation factors have been reported, when these values are re-analyzed using a common methodology to permit comparisons, the reported BCFs and BAFs fall within a narrow range. The Regulatory Bioaccumulation Multiplier (RBM) uses consistent criteria to define exposure concentrations of TCDD, normalizes to a common fish lipid content (in this case, 2.5%), and measures TCDD fish concentrations in the edible portion or fillet tissue. Re-analyzing existing literature values, the calculated RBMs generally fall below the value of 5,000 and do not support a value of 50,000. Therefore, the value of 5,000 constitutes the most scientifically based multiplier for regulatory purposes. Coincidentally, the RBM methodology produces an accumulation factor of equal magnitude to that of the EPA's (1984) recommended ambient water quality criteria value of 5,000, albeit for different reasons and based upon a different rationale. EPA Region III, in its approval of Maryland's and Virginia's water quality standards, has affirmed the selection of a value of 5,000 for regulatory purposes.

5.0 FISH CONSUMPTION

The primary route of human exposure to many pollutants occurs through the ingestion of fish obtained from waterbodies containing those compounds (Rifkin and LaKind, 1991). Because of this, the estimation of a representative rate of fish consumption from potentially impacted waterways is important to the derivation of a scientifically-based and health protective water quality standard. Most of the estimates of fish consumption rates that are found in the scientific literature are based either on national surveys or are specific to a particular region of the United States (Puffer et al., 1981; Humphrey, 1978; Javitz, 1980; Rupp et al., 1980). Many of these surveys have either not adequately characterized the types of fish consumed (EPA, 1989c), or no distinction has been made between the consumption of commercially-harvested and recreationally-harvested fish (Javitz, 1980; EPA, 1989c). These factors are important to define in a risk assessment approach to deriving a water quality standard, as there may be interspecies differences in potential to bioaccumulate TCDD (Spacie and Hamelik, 1982; Spigarelli et al., 1982; Rand and Petrocelli, 1985; Gobas et al., 1987). In addition, regional variations in consumption of preferred species, availability of these species, access to productive fisheries, length of fishing season, and cultural heritage can greatly influence fish ingestion habits.

5.1 General Population

The EPA has used the value of 6.5 g/day as an estimate of fish consumption in developing its Ambient Water Quality Criteria (AWQC) (EPA, 1984). This value is based on the estimated national per capita rate of fish consumption and includes all commercially-harvested and recreationally-caught freshwater and estuarine fish and shellfish (EPA, 1989a). Although the EPA's estimate may be appropriate for estimating an average consumption rate for the U.S. population as a whole, its ability to predict regional consumption or consumption by recreational anglers or other subpopulations is often limited.

An analysis conducted by Rupp et al. (1980) revealed that in addition to regional variations in fish consumption, there are often substantial variations in fish consumption patterns among individuals living in the South Atlantic region of the U.S. The authors reported that only 12.4 percent of the fish consumers surveyed in the South Atlantic region consumed freshwater fish, whereas 89.2 percent of those individuals consumed saltwater fish (Rupp et al., 1980). These results clearly

suggest that most people do not eat freshwater fish. Rupp et al. (1980) estimated that the average rate of freshwater fish consumption by adults in the South Atlantic region, which includes the state of West Virginia, was 1.1 g/day.

Ideally, it is most accurate to use state- or region-specific data to account for regional and individual differences in fish consumption (EPA, 1989a). However, specific estimates of the consumption of freshwater fish by the general population of West Virginia have not been made and published in the literature.

5.2 Recreational Anglers

If dioxin is present in the tissue of freshwater fish species, then recreational anglers may represent an important receptor population if their rate of intake of freshwater fish is significantly elevated above the norm. Consequently, it is important that the rate of consumption of recreationally-caught fish from potentially impacted waterbodies be evaluated when developing a water quality standard.

Rates of freshwater fish consumption by anglers who fish rivers in other parts of the country have been investigated. Honstead et al. (1971) conducted a diet recall survey of 10,900 individuals from households in which there was at least one angler who fished the Columbia River in the Tri-City area of Washington. The average size of a fish meal was estimated to be approximately 200 grams and it was reported that individuals ate an average of 14 such meals per year. Thus, the annual average rate of consumption was 2.8 kilograms per year, or 7.7 g/day.

In a creel survey of recreational anglers who fished the same area of the Columbia River, Soldat (1970) observed that 15 percent of the anglers surveyed caught 90 percent of the fish. The distribution of species creeled and consumed was similar to that reported in the Honstead (1971) diet recall survey. From the data generated from the Soldat (1970) creel survey, a considerably lower average consumption rate of 1.8 g/day could be estimated. Because it was based on actual landings data rather than recall, it is likely that Soldat's (1970) estimate of 1.8 g/day is more accurate for anglers in the upper reaches of the Columbia River.

Similar rates of fish consumption among recreational anglers were observed in a recently completed survey of recreational anglers in Maine (ChemRisk, 1991). In that study, anglers were

asked to recall the number, size and species of all of the fish they had caught and consumed from Maine's lakes, ponds, rivers and streams. The survey period included ice fishing season as well as open water season. In addition to the fish they had caught themselves, anglers were asked to report any fish they had consumed that were caught by other anglers either within or outside of their households. Anglers were also asked to report how many individuals in their household shared in the catch.

In this survey, the weight of each fish reported by a survey respondent was estimated from the reported length using Maine-specific weight/length regressions for each species. Fish weights were then adjusted for edible portion and summed to derive a total fish weight per angler. This total fish weight was then divided over the number of individuals reported to share in the catch and annualized over the year to derive a daily fish consumption rate per individual. Finally, these individual rates were combined to provide a distribution of fish consumption rates among the angling population surveyed.

According to the results of the survey, the distribution was positively skewed. The median rate of consumption for anglers who consumed fish from Maine rivers and streams was 0.99 g/day (Table 5-1). For all freshwater bodies, the median rate of consumption for consuming anglers was 2.0 g/day (Table 5-1). The arithmetic mean rates of consumption were 3.6 g/day for consumers of river fish and 6.4 g/day for consumers of fish from all freshwater sources. These represented the 81st and 77th percentiles of the distributions, respectively (Table 5-1).

It is well documented in the literature that the number of anglers that eat small amounts of fish greatly exceeds the number of anglers that eat large amounts of fish (Soldat, 1970; Landolt et al., 1985, 1987; West et al., 1989; Meunz and Peterson, 1990; ChemRisk, 1991). Soldat (1970) observed that 15 percent of the anglers surveyed caught 90 percent of the fish. In a survey of anglers in Michigan (West et al., 1989), 59.3 percent of respondents reported eating no fish meals, while only 1.6 percent reported eating more than four meals in a seven-day period. Results of the Maine survey indicated that when considering all anglers on all freshwater fisheries, 10 percent of the anglers ate 90 percent of the total freshwater fish consumed. For rivers and streams, the distribution of consumption was more exaggerated; 7 percent of the anglers ate 93 percent of the fish consumed (ChemRisk, 1991).

Table 5-1. Results of Fish Consumption Survey of
Recreational Anglers in Maine

	Rivers and Streams ^a	All Waters ^a
Median (50th Percentile)	0.99	2.0
75th Percentile	2.5	5.8
Arithmetic Mean	3.6	6.4
Percentile at Mean	81	77
90th Percentile	6.1	13

a. All rates are in g/person-day. Based on freshwater fish consumption rates for consuming anglers as presented in ChemRisk, 1991. Percentiles were calculated by rank without any assumption of statistical distribution.

Miles (1991) reported a 1983 recreational use survey of the Ohio River near the proposed Ashton Pulp Plant conducted by Pierce et al. In the results of that survey, it was reported that 8,362 fish were harvested during 14,534 angler trips (Miles, 1991) for an average of 0.575 fish per trip. According to the U.S. Fish and Wildlife Service (1989) the average resident angler in West Virginia spends 20 days fishing in West Virginia each year. Using this information, it can be estimated that the average angler harvests 11.5 fish per year. Based on the reported species distribution of harvest (Table 5-2) and average weights of individual species obtained from the Savannah River (Table 5-2) (Turcotte, 1983; Schmitt and Hornsby, 1985) a weighted average weight for fish harvested was estimated to be 0.61 kg/fish. This results in a harvested weight per angler of 7.0 kg/year.

Not all fish harvested is edible. The EPA estimates that approximately 30 percent of whole fish weight is edible (EPA, 1989a). Assuming this is true, the average angler in West Virginia harvests approximately 2.1 kg of edible fish each year from the Ohio River. In general, an angler is likely to share his/her catch with other individuals within his/her household. According to the 1980 census, average household size in West Virginia was 2.79 individuals. Thus, the average angler harvests 0.75 kg of edible fish per household member. On an annual basis, this equates to 2.1 g/individual-day. If the angler is assumed to consume all of the harvested fish, the consumption rate would be 5.8 g/day. Thus, it can be estimated that the average angler in West Virginia consumes between 2.1 and 5.8 g/day of fish obtained from the Ohio River.

5.3 Summary

Fish consumption is an important component of the dioxin ambient water quality standard equation since ingestion of fish is the most likely route of human exposure to TCDD found in aquatic environments (EPA, 1984; Keenan et al., 1990; Parsons et al., 1991; Rifkin and LaKind, 1991). For this reason, it is important to characterize the freshwater fish consumption habits of all potentially exposed populations.

Using the Rupp et al. (1980) estimates of consumption, it can be estimated that average consumption by freshwater fish consumers in the general population is approximately 1.1 g/day. This estimate reflects consumption of freshwater fish obtained from all sources. A review of available data for angler use on the Ohio River in West Virginia indicates that the average resident

Table 5-2. Distribution of Species and Average Weight of Species Harvested

	Percent of Harvest ^a	Average Weight (kg)
Freshwater drum	58	0.68 ^b
Channel catfish	18	0.32 ^c
Crappie	3.5	0.21 ^b
Sauger	3.5	0.55 ^d
Walleye	3.5	0.55 ^d
White bass	3.5	1.68 ^e
Flathead catfish	3.5	0.55 ^d
Carp	3.5	0.55 ^d

a. Reported by Miles (1991).

b. Based on average weight of red drum in study of Savannah River (Turcotte, 1983).

c. Based on average weight reported for Savannah River (Schmitt and Hornsby, 1985).

d. Based on average weight of "Others" category reported for Savannah River (Schmitt and Hornsby, 1985).

e. Based on average weight of striped x white bass reported for Savannah River (Schmitt and Hornsby, 1985).

angler is likely to consume between 2.1 and 5.8 g/day of freshwater fish from the Ohio River. This estimate is very conservative as it assumes that all fish harvested during the year came from the Ohio River. Thus, use of the EPA's proposed value of 6.5 g/day is a reasonable and conservative estimate of freshwater fish consumption by the general population and recreational anglers in the state of West Virginia.

6.0 PERSPECTIVE ON RISK

Risk can be defined as an estimate of the probability that a given exposure to an agent in the environment will result in an adverse health effect. Adverse health effects may include mortality, morbidity, or injury to individuals or a population as a whole (Derby and Keeney, 1981;Graham, 1990). Risk is a function of both hazard and exposure. The innate toxicity of a given compound contributes to its hazard, while the frequency, duration, and intensity of contact with the compound defines potential exposure. Risk assessment evaluates both components and characterizes the probability that adverse health effects in humans, domestic animals, wildlife or ecological systems will result from exposures to environmental hazards.

Cancer risks may be expressed as either individual or population risks. An individual cancer risk value is an estimate of the probability that an individual member of a population will develop cancer as a result of a lifetime of exposure to a cancer-causing agent. Given that the background incidence of cancer in the U.S. population is about 30%, or 30 cases of cancer in 100 people (American Cancer Society, 1989), exposure to a chemical resulting in a risk level of 1 in 1,000,000 (10^{-6}) is equivalent to ensuring that the lifetime cancer risk for any person exposed to this level of contamination is not greater than 300,001 in 1,000,000 (30.0001%), rather than 300,000 in 1,000,000. Population risk is expressed as the product of the individual risk and the size of the exposed population. It is measured as an upper-limit estimate of the number of additional incidences of cancer in the exposed population (Travis et al., 1987).

Health risk assessments have become so widely adopted in the United States that their conclusions are now major factors in many environmental decisions. Risk assessment is often used as the basis for deriving ambient standards and target concentrations or cleanup goals which ensure that the estimated plausible risk to humans will be below a specific, established level. The science of risk assessment provides a bridge between scientific research on the health effects of chemicals and the various risk management options that may be considered by regulatory agencies. The risk assessment process has helped reduce unwarranted concern over trivial hazards by helping the public put into proper perspective the magnitude of the risks posed by both naturally occurring toxicants in food, and manmade chemicals (Young, 1987). Risk assessment provides the means whereby those chemical hazards which are significant health risks may be identified and appropriate action may be taken to limit exposure.

Putting risks into perspective is an important facet of risk management. Virtually every aspect of human life exposes individuals to health risks (Klaassen et al., 1986). Each day, people take risks or make decisions to avoid risks. Risks associated with common activities may be readily acceptable to individuals. In establishing a regulatory acceptable risk level for the State of West Virginia, it is important that one have a perspective of the environmental health risks relative to those voluntary and involuntary risks to which individuals are exposed every day. Furthermore, it is important that state regulators consider previous risk decisions made in their state or region, as well as pertinent existing state and federal regulations, such as those for drinking water. Risk management decisions should address exposure assessments and the question of acceptable risk on a case-by-case basis.

This chapter presents a perspective on the risk levels that have prompted regulatory action and discusses the selection of an acceptable risk level for the State of West Virginia. For the purpose of comparison, various risks that are incurred daily as a result of voluntary and involuntary activities are discussed.

6.1 Regulatory Positions on Risk

Various federal and state regulatory agencies are responsible for establishing regulations and standards which protect the public from exposure to carcinogens and environmental toxins (Travis et al., 1987). An underpinning of regulatory and risk management decisions is the level of cancer risk which is considered to be acceptable or *de minimis*. The term *de minimis* risk is used by risk assessors and regulators to define insignificant risk levels, or those risks that are below regulatory concern (Travis et al., 1987). A common misconception within risk assessment is that all occupational and environmental regulations adopt a theoretical maximum cancer risk of 10^{-6} . When this level of risk is exceeded, the public and the media quite often view the situation as a serious threat to public health. In 1987, the former commissioner of the U.S. Food and Drug Administration (FDA), Dr. Frank Young, discussed this misunderstanding (Young, 1987).

"The risk level of one in one million is often misunderstood by the public and the media. It is not an actual risk; i.e., we do not expect one out of every million people to get cancer if they drink decaffeinated coffee. Rather, it is a mathematical

risk based on scientific assumptions used in risk assessment. FDA uses a conservative estimate to ensure that the risk is not understated. We interpret animal test results conservatively and we are extremely careful when we extrapolate risks to humans. When FDA uses the risk level of one in one million, it is confident that the risk to humans is virtually nonexistent."

Cancer risk levels, which are often perceived to represent trigger levels for regulatory action, frequently represent levels of risk that are so small that they are of negligible concern.

The *de minimis* cancer risk levels adopted by risk managers and regulators are based typically on a range of acceptable risk levels which depend upon a given situation and the size of the population potentially at risk. In addition, the development of an acceptable level of risk often involves consideration of the technological and economic feasibility of risk reduction strategies, as well as the societal costs and benefits of alternative risk levels.

6.1.1 Federal Agencies

Recent reviews indicate that the theoretical cancer risks associated with currently enforced environmental regulations are in the vicinity of 1 in 100,000, not 1 in 1,000,000 (Travis et al., 1987; Travis and Hattemer-Frey, 1987). In a retrospective review of the use of cancer risk estimates in 132 federal decisions, Travis et al. (1987) examined the level of cancer risk that triggered regulatory action. The authors considered three measures of risk: individual risk, the size of the population exposed, and the population risk. The results of the review showed that for exposures resulting in a small-population risk, the level of risk above which agencies almost always acted to reduce risk was approximately 4×10^{-3} . For large-population risks (the entire U.S. population) agencies typically acted on risks of about 3×10^{-4} . For effects on small populations, regulatory action was never taken for individual risk levels below 1×10^{-4} . For large-population effects, the *de minimis* risk level dropped to 1×10^{-6} . Based on the findings of Travis (1987; Travis and Hattemer-Frey, 1987) and upon further examination of the database, Graham (1990) has suggested using a range of acceptable lifetime cancer risk levels in risk management decisions (1×10^{-2} to 1×10^{-4} for maximally exposed individuals and 1×10^{-4} to 1×10^{-6} for the average exposed individuals).

Rodricks et al. (1987) also evaluated regulatory decisions and reached similar conclusions. In decisions relating to promulgation of National Emission Standards for Hazardous Air Pollutants (NESHAPS), the EPA found that the maximum individual risks and total population risks from a number of radionuclide and benzene sources were too low to be judged significant. Maximum individual risks were in the range of 3.6×10^{-5} to 1.0×10^{-3} . In view of the risks deemed insignificant by EPA, Rodricks et al. (1987) noted that 1×10^{-5} appears to be in the range of what EPA might consider an insignificant average lifetime risk, at least where annual aggregate population risk is concerned.

As mandated by the 1986 Safe Drinking Water Act (SDWA), the EPA has established Maximum Contaminant Level Goals (MCLGs) for chemical substances listed under the Act. These non-enforceable goals have been established at levels considered protective of human health (40 CFR 141, July 25, 1990). The EPA has established MCLGs of zero for known or suspected carcinogens that are believed to have no threshold. Recognizing that MCLGs of zero cannot be measured, the SDWA directed that Maximum Contaminant Levels (MCLs) be established as close to the MCLGs as feasible.

"Based on the statutory directive for setting the MCLs, EPA derives the MCLs based on an evaluation of (1) the availability and performance of various technologies for removing the contaminant, and (2) the costs of applying those technologies. Other factors which are considered in determining the MCL include the ability of laboratories to measure accurately and consistently the level of the contaminant with available analytical methods. For carcinogens, EPA also evaluates the health risks that are associated with various levels of the contaminant with the goal of ensuring that the risks at the MCL fall within the 10^{-4} to 10^{-6} risk range that the Agency considers protective of public health and therefore achieves the overall purpose of the SDWA" (40 CFR 141, July 25, 1990).

A number of the known or suspected carcinogens for which MCLs have been promulgated are listed in Table 6-1. If the estimated lifetime average daily doses, based on 2 l/day drinking water consumption by a 70 kg adult averaged over a lifetime, are multiplied by the cancer potency factor for each contaminant (EPA, 1989d), the incremental risks associated with the EPA MCLs range

Table 6-1. Selected MCLs and Associated Risk Levels

Chemical	USEPA Maximum Contaminant Level (mg/l)	USEPA Carcinogen Classification ^d	Cancer Potency Factor (mg/kg-day) ^{-1d}	Incremental Cancer Risk Associated with Lifetime Exposure to Chemical in Drinking Water ^e
Benzene	0.005 ^c	A	2.9×10^{-2}	4.1×10^{-6}
Carbon Tetrachloride	0.005 ^c	B2	1.3×10^{-1}	1.9×10^{-5}
Chlordane	0.002 ^a	B2	1.3×10^0	7.4×10^{-5}
para - Dichlorobenzene	0.075 ^c	B2	2.4×10^{-2}	5.1×10^{-5}
1,2- Dichloroethane	0.005 ^c	B2	9.1×10^{-2}	1.3×10^{-5}
1,1-Dichloroethylene	0.007 ^c	C	6.0×10^{-1}	1.2×10^{-4}
1,2-Dichloropropane	0.005 ^a	B2	6.8×10^{-2}	9.7×10^{-6}
Ethylene dibromide (EDB)	0.00005 ^a	B2	8.5×10^{-1}	1.2×10^{-4}
Heptachlor	0.0004 ^a	B2	4.5×10^0	5.1×10^{-5}
Hexachlorobenzene	0.001 ^b	B2	1.7×10^0	4.9×10^{-5}
Polychlorinated biphenyls	0.0005 ^a	B2	7.7×10^0	1.1×10^{-4}
2,3,7,8 - TCDD	5×10^{-8b}	B2	1.56×10^{-5}	2.2×10^{-4}
Tetrachloroethylene	0.005 ^a	B2	5.1×10^{-2}	7.3×10^{-6}
Toxaphene	0.005 ^c	B2	1.1×10^0	1.6×10^{-4}
1,1,2 - Trichloroethane	0.005 ^b	C	5.7×10^{-2}	8.1×10^{-6}
Trichloroethylene	0.005 ^a	B2	1.1×10^{-2}	1.6×10^{-6}
Total Trihalomethanes	0.10 ^c			
Bromodichloromethane ^f		B2	1.3×10^{-1}	3.7×10^{-4}
Dibromochloromethane ^f		C	8.4×10^{-2}	2.4×10^{-4}
Bromoform ^f		B2	7.9×10^{-3}	2.3×10^{-5}
Chloroform ^f		B2	6.1×10^{-3}	1.7×10^{-5}
Vinyl Chloride	0.002 ^c	A	2.3×10^0	1.3×10^{-4}

a. EPA 40 CFR 141. May 22, 1989.

b. EPA 40 CFR 141 July 25, 1990.

c. EPA Fac. Sheet, Office of Drinking Water, Feb 12, 1988.

d. EPA, 1989g. Health Effects Assessment Summary Tables. Fourth Quarter. OERR 9200.6 - 303 - (89-4).

e. Calculations based on ingestion of 2 liters per day, 365 days per year, for a 70 year lifetime, and a body weight of 70 kg.

f. Assuming single chemical contaminant is responsible for MCL of 0.10 mg/L.

A - Human Carcinogen (Sufficient evidence of carcinogenicity in humans).

B2 - Probable human carcinogen (sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans).

C - Possible human carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data).

from 10^{-6} to 10^{-4} . This range is also mentioned in the recently promulgated Hazardous Waste Management System Toxicity Characteristics Revisions (55 FR 11798-11863):

"For drinking water contaminants, EPA sets a reference risk range for carcinogens at 10^{-6} to 10^{-4} excess individual cancer risk from lifetime exposure. Most regulatory actions in a variety of EPA programs have generally targeted this range using conservative models which are not likely to underestimate the risk."

Final revisions to the National Contingency Plan (NCP) (EPA, 1990c) set the acceptable risk range between 10^{-6} and 10^{-4} at hazardous waste sites regulated under CERCLA. Previously, the range of risks was 10^{-7} to 10^{-4} . Since the NCP revisions, the EPA has selected and promulgated a single risk level of 10^{-5} (1 in 100,000) in the Hazardous Waste Management System Toxicity Characteristics (TC) Revisions (55 FR 11798-11863). In their justification, the EPA cited the following rationale:

"The chosen risk level of 10^{-5} is at the midpoint of the reference risk range for carcinogens (10^{-6} to 10^{-4}) targeted in setting MCLs. This risk level also lies within the reference risk range (10^{-6} to 10^{-4}) generally used to evaluate CERCLA actions. Furthermore, by setting the risk level at 10^{-5} for TC carcinogens, EPA believes that this is the highest risk level that is likely to be experienced, and most if not all risks will be below this level due to the generally conservative nature of the exposure scenario and the underlying health criteria. For these reasons, the Agency regards a 10^{-5} risk level for Group A, B, and C carcinogens as adequate to delineate, under the Toxicity Characteristics, wastes that clearly pose a hazard when mismanaged."

6.1.2 State Agencies

The states of Alabama, Georgia, South Carolina, Virginia, Maryland, Michigan, Minnesota, Ohio, Maine, and Wisconsin have used or propose to use a 10^{-5} risk level in their risk management decisions. Similarly, a lifetime incremental cancer risk of 10^{-5} is used by the Commonwealth of Massachusetts as a cancer risk limit for exposures to substances in more than one medium at hazardous waste disposal sites (Mass DEQE, 1988). This risk limit represents the total cancer risk at the site associated with exposure to multiple chemicals in all contaminated media. The State of

California has also established a risk criterion of 10^{-5} for use in determining levels of chemicals and exposures that pose "no significant risk" of cancer under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) (CCR Section 12703) (California, 1987).

The EPA has recently affirmed a risk level of 10^{-5} in its approval of the Maryland water quality standard. In its comments, EPA stated:

"The Maryland criterion is based on a 10^{-5} risk level. This risk level is frequently used in state and federal regulatory actions, and is considered by EPA to be within an adequately protective range" (EPA, 1990b).

In the case of EPA's approval of Virginia's water quality standard of 1.2 ppq, the EPA (1991) noted that:

"... EPA set forth a range of criteria values corresponding to risk levels of 10^{-5} to 10^{-7} in its 1984 criteria document. The Virginia criterion is based on a 10^{-5} risk level. This risk level is frequently used in state and federal regulatory actions, and is considered by EPA to be within an adequately protective range."

6.1.3 Summary

This review of policies on acceptable risk indicates that the application of a universal acceptable risk level of 10^{-6} would be inconsistent with current policies and may in some cases be overly conservative. As previously discussed, a range of risk levels from 10^{-4} to 10^{-6} is currently being used in other states and at the federal level. In summary, selection of a universal 10^{-6} risk level for all chemicals in all media may not reflect site-specific data and may be overly conservative.

6.2 Comparison of Risks

Quantitative cancer risk estimates can be more readily understood when placed in perspective with health risks associated with familiar activities, occupations, or commodities. Comparisons are made between cancer risk estimates and everyday risks. Most of the risk values cited in this section are annual risk estimates, or reflect mortality rates during any given year. The examples

used in this section are summarized for the purpose of providing a conceptual yardstick for measuring the magnitude of a risk.

6.2.1 Commonplace Risks

Many common human activities entail risks greatly in excess of one in one million (Table 6-2). For example, although risks are inherent in various modes of transportation, people generally accept those risks. Driving a car not only subjects the driver and passengers to the risk of an accident, but also poses a risk to pedestrians. The total annual risk of dying in a car accident is about 2 in 10,000 (2.4×10^{-4}). The risk to a pedestrian dying in a motor vehicle accident is approximately 4 in 100,000 (4.2×10^{-5}). This equates to a lifetime risk of dying in a motor vehicle accident of approximately 7 in 1,000. An airline passenger that flies an average of four hours per week has an annual risk of 1 in 100,000 (1.0×10^{-5}) (Wilson and Crouch, 1987). An average bicyclist also has an annual risk of dying of 1 in 100,000 (Hutt, 1978; as cited in Rodricks and Taylor, 1983).

In general, accidents occurring in the home pose an annual risk of about 1 in 10,000 (1.1×10^{-4}). This overall risk is comprised of risks from falls, drowning, fires, inhalation and ingestion of objects, firearms, accidental poisoning and electrocution (Crouch and Wilson, 1982). Living in an average stone or brick building for about 20 months increases an individual's risk of cancer caused by natural radioactivity by 1 in 100,000 (1.0×10^{-5}) (Allman, 1985).

Exposures to radiation, either natural background radioactivity or radiation for medical purposes, increases the chances of cancer. Average diagnostic medical chest x-rays have been reported to increase the risk of cancer by 2 in 100,000 (2.0×10^{-5}) annually. Exposure to natural radiation at sea level (excluding radon) poses a similar level of risk (2.0×10^{-5}). Exposure to air pollution in the eastern United States poses an even greater risk of 2 in 10,000 (2.0×10^{-4}) annually (Wilson and Crouch, 1987). Furthermore, Gough (1990) estimates that between 2 and 3 percent of all cancers are associated with environmental pollution, while between 3 and 6 percent of all cancers may be attributed to total radiation sources.

Similarly, numerous popular sporting activities pose an increased potential risk of dying due to accidents. Boating, hunting, swimming and football pose an annual risk level of between 3 and 5

Table 6- 2. Commonplace Risks^a

Action	Annual Risk
Travel	
Motor vehicle accidents	2.4×10^{-4}
Collisions with pedestrians	4.2×10^{-5}
Frequent airline passenger (4 hrs per week) ^b	1.0×10^{-5}
Bicycling	1.0×10^{-5}
Housing	
Home accidents (all causes)	1.1×10^{-4}
Natural background radiation (sea level)	2.0×10^{-5}
Living in a natural stone or brick building	1.0×10^{-5}
Average diagnostic medical x-rays in U.S.	2.0×10^{-5}
Air pollution	2.4×10^{-4}
Sports	
Boating	5.0×10^{-5}
Hunting	3.0×10^{-5}
Swimming	3.0×10^{-5}
Football ^b	4.0×10^{-5}
Smoking	
Smoking, all effects (1 pack per day)	3.6×10^{-3}
Person sharing room with smoker	1.0×10^{-5}

a. Wilson and Crouch, 1987.

b. Hutt, 1978 as cited in Rodericks and Taylor, 1983.

in 100,000 (3.0 to 5.0×10^{-5}) (Crouch and Wilson, 1982). Other sports that were not included in Table 6-2 have even higher levels of risk such as flying amateur aircraft (3 in 1,000), mountaineering (6 in 10,000) and scuba diving (4 in 10,000) (Crouch and Wilson, 1982).

Cigarette smoking has been suggested to increase one's annual average risk of cancer and other health effects by more than 1 in 1,000. In addition, chronic exposure to secondary tobacco smoke has been estimated to increase individual's risks of health problems associated with tobacco smoke by 1 in 100,000 or 1.0×10^{-5} (Crouch and Wilson, 1982).

6.2.2 Occupational Risks

Many common occupations pose risks to individuals on the order of 10^{-6} or greater. For example, working in manufacturing or farming industries may increase an individual's risk of death during any year by 8×10^{-5} or 4×10^{-4} , respectively (Crouch and Wilson, 1982). Workers at gas stations are exposed to benzene vapors from volatilizing fuel levels ranging up to $32 \mu\text{g}/\text{m}^3$ (API, 1986). Assuming that a worker is exposed to this maximum concentration during one-half of his occupational lifetime (20 hours/week, 240 days/year, 45 years), an associated lifetime cancer risk of about 3×10^{-5} can be calculated. That risk is expected to be much higher if the attendant is also exposed to additional vapors when gasoline is used to clean automobile parts in the workshop.

In the dry cleaning industry, workers are exposed daily to levels of tetrachloroethylene ranging from 10,000 to 20,000 $\mu\text{g}/\text{m}^3$ (Wallace, 1990). The risk associated with exposure to a midrange level of 15,000 $\mu\text{g}/\text{m}^3$ for a worker's occupational lifetime can be calculated to be approximately 3×10^{-3} . Occupational exposure limits established by OSHA may also be associated with relatively high risk values. Several OSHA time-weighted-average threshold limit values (OSHA, 1989) for carcinogens are associated with 10^{-3} risks (acrylamide and epichlorohydrin) and 10^{-2} risks (carbon tetrachloride, chlordane, and chloroform) to workers, assuming that a 70 kg worker inhales 10 m^3/day for 240 days/year over a 45-year occupational duration. Additional occupations that pose 10^{-5} and 10^{-4} risks to individuals are listed in Table 6-3.

Table 6-3. Occupational Risks of Death

Occupational or Industry	Annual Risk Per Person at Risk
Manufacturing	8.2×10^{-5}
Trade	5.3×10^{-5}
Service and government	1.0×10^{-4}
Transport and public utilities	3.7×10^{-4}
Agriculture (Includes transportation-related accidents)	6.0×10^{-4}
Construction	6.1×10^{-4}
Mining and quarrying	9.5×10^{-4}
Farming	3.6×10^{-4}
Tractor fatalities per tractor	8.8×10^{-5}
Metal mining and milling	9.4×10^{-4}
Nonmetal mining and milling	7.1×10^{-4}
Policeman	2.2×10^{-4}
Railroad employees	2.4×10^{-4}
Fire fighter	8.0×10^{-4}

Adapted from Crouch and Wilson, 1982.

6.2.3 Dietary Risks

Dietary factors, such as alcohol consumption and a high fat diet, have been implicated in many studies as a major contributor to cancer deaths (Ames et al., 1987; Scheuplein, 1990b). In addition, people are exposed to risks every day by ingesting natural carcinogens in food (Scheuplein, 1990b; Ames et al., 1987). Dr. Robert Scheuplein, Director of the Office of Toxicological Sciences at the FDA, estimated the risk of dying from cancer from dietary exposure to natural and man-made carcinogens is about 7 in 100 (7.7×10^{-2}). Of this, about 98% of the cancer risk in the diet may be attributable to natural carcinogens in food (Scheuplein, 1990b). Diet contributes approximately 35% of the total human cancer risk, and smoking, the other major risk factor for cancer, accounts for another 30% (Doll and Peto, 1981).

Ames (1983) estimated that the daily intake of natural carcinogens in traditional food may exceed several grams. Scheuplein (1990b) more conservatively assumed that of 1,000 grams of solid food consumed per day, approximately 0.1% or one gram of food per day consists of carcinogens. Table 6-4 lists intake and risk estimates for various food categories containing carcinogenic substances. This analysis clearly indicates that the risks from natural carcinogens are much greater than those posed by traces of pesticide residues or contaminants (Scheuplein, 1990b). Methods of storing, preparing and consuming food, as well as dietary patterns (protein to fat ratios), also contribute to the high cancer risks.

Table 6-5 lists some of the naturally-occurring carcinogens in food. Natural carcinogens in food and those produced during food preparation have been shown to occur in amounts that exceed environmental exposure levels. For example, certain molds in foods synthesize toxins that are mutagenic or carcinogenic; e.g., aflatoxin, is found in nuts (peanut butter), wheat, and corn, and through food chain exposure in cow's milk, (Ames et al., 1987). Furthermore, consumption of as little as 40 tablespoons of peanut butter has been reported to increase the chance of liver cancer caused by aflatoxin by 1 in a million (Covello, 1989).

Similarly, large amounts of hydrazine, a known carcinogen and mutagen, have been found in most edible mushrooms. The most common commercial mushroom (*Agaricus bisporus*) contains about 300 mg of a hydrazine derivative per 100 grams of mushrooms. In addition, formaldehyde is a

Table 6-4. Risk Estimates of Various Food Categories Containing Carcinogenic Substances

Food Category	Amount of Food Consumed	(Estimated) Amount of Carcinogen Consumed	Risk
Traditional food	1,000 g	1,000 mg	7.61×10^{-2}
Spices and flavors	1.0 g	10 mg	7.61×10^{-4}
Indirects	20 mg	2 mg	1.52×10^{-4}
Pesticides and contaminants	200 μ g	0.1 mg	7.61×10^{-6}
Animal drugs	1.0 mg	0.1 mg	7.61×10^{-6}
Food preparation (charred protein only)	1.0 g	0.1 mg	7.61×10^{-6}
Mycotoxins	10 μ g	0.001 mg	7.61×10^{-8}
Total Risk =			7.7×10^{-2}

Adapted from Scheuplein, 1990.

Table 6-5. Naturally Occurring Carcinogens in Food^a

Food	Carcinogen
Apples	Patulin
Mushrooms	Hydrazines
Parsnips, celery ^b	Psoralens
Cereals, corn, seeds, nuts	Aflatoxins
Plants (herbal teas)	Pyrrolizidine alkaloids
Spinach, beets, lettuce, radishes	Nitrates --> nitrosamines
Phytoplankton (fish and shellfish)	Polyaromatic hydrocarbons
Garlic ^b	Alkyl isothiocyanate
Chilies ^b	Capsaisin
Oranges ^b	d-limonene
Spices: ^b	
Mustard	Allylisothiocyanate
Pepper, nutmeg	Safrole
Cinnamon	Cinnamaldehyde
Smoke	Nitrosamines
Marjoram	Carvacrol
Tarragon	Estragole

a. Compiled from Scheuplein, 1990; Cheeke and Shull, 1985.

b. Foodstuffs or spices with known or suspected carcinogenic activity.

natural carcinogen that is ubiquitous in foods. Daily consumption of shrimp, bread, cola, and beer in various combinations could result in significant exposure to formaldehyde (Ames et al., 1987).

Cooking of food generates a variety of carcinogenic substances. Carcinogens formed during food processing include: ethyl carbamate (fermentation), nitrosamines (curing, frying, salting, pickling), polynuclear aromatics (broiling meat, smoking), and heterocyclic amines and nitropyrenes (grilling and charring of fish or meat) (Scheuplein, 1990b). Intake of nitropyrenes from grilled chicken has been estimated to be much higher than that from air pollution (Sugimura et al., 1986; Kinouchi et al., 1986). In fact, according to Ames (1983), the amount of burnt material eaten in a typical day is at least several hundred times more than that inhaled from severe air pollution.

Currently, safety information on direct food additives is more available than for most dietary categories because of consumer concern. Direct food additives are not approved by the FDA unless they are either non-carcinogenic or *de minimis* in animal tests; the *de minimis* risk in this context is 10^{-6} . However, saccharin is an exception to this rule because of a Congressional moratorium of an FDA regulation banning its use (Scheuplein, 1990b). According to another study (Wilson, 1979), drinking a 12-ounce can of diet soda containing saccharin every day of the year results in an annual cancer risk of 1 in 100,000 (1.0×10^{-5}).

In summary, potential cancer risks are associated with intake of foods containing carcinogenic substances that are naturally occurring, or are formed during cooking and preparation. Comparison of these risk values for common, everyday exposures with proposed acceptable levels of risk, such as 10^{-5} and 10^{-6} , is an effective means of placing risks in perspective.

6.3 The Cost of Compliance with a 10^{-6} Risk Limit

A range of risk levels has been used in U.S. federal and state regulatory decisions. Although an incremental risk level of 10^{-5} , rather than 10^{-6} , has often been used by the EPA to protect public health (EPA, 1990d; Rodricks et al., 1987; 55FR 11798-11863), the 10^{-6} risk level is frequently proposed as a universal acceptable level of risk which is protective of human health. Under certain circumstances, the application of an overly conservative risk level may necessitate the implementation of new, expensive technologies and substantially increase the cost of compliance.

When selecting an acceptable level of risk, regulatory officials must take into account the number of people exposed to the risk in question. This principle is a fundamental component of public health evaluation. For instance, the currently popular 1×10^{-6} risk standard was originally intended to be protective of the entire U.S. population from exposure to cancer causing contaminants in the food supply. Yet, when localized populations are exclusively exposed to a risk, as is the case with dioxin-contaminated fish, a less stringent numerical standard is adequate to protect the public health. When choosing appropriate risk levels, regulators should also weigh the economic costs and benefits that may be associated with risk reduction. Although some environmental laws try to restrict economic considerations, common sense and studies of regulatory behavior indicate that economic factors play a critical role in environmental decision making. The economic consequences of regulatory decisions must be heeded so that public health is not adversely affected. Public health professionals have recognized for decades that reducing family income impairs public health. The costs of environmental regulation may reduce real family income by increasing the prices of goods and services that all of us purchase, which ultimately causes a reduction in real family incomes. Subsequently, when families have less income, they have less money available for everything from preventive checkups to smoke detectors. If regulatory costs are excessive, the regulator may inadvertently cause more harm to the health status of families than will be prevented.

As a case in point, consider the chlorination of public drinking water supply by municipalities. As a direct result of chlorination, a number of potentially harmful compounds known as trihalomethanes are produced. Most notable of these is chloroform. Based on exposure calculations which assume that the average 70 kg adult ingests 2 liters of water per day (EPA, 1989d), the dose of individual trihalomethanes received in water is associated with risk levels ranging from 1.7×10^{-5} to 3.7×10^{-4} (Table 6-1). In order to comply with a 10^{-6} risk level, treatment systems would have to be modified in order to reduce the levels of these compounds in finished water. Implementation of an activated charcoal filter system is one method which would reduce trihalomethane levels sufficiently to bring the risk associated with these compounds in drinking water into compliance. The life of a charcoal bed and, thus, the cost of system maintenance, is highly dependent upon the level of organic matter entering the treatment system. In a worst-case estimation in which the bed life is only 30 days, use of the charcoal filter water treatment system would raise the cost of municipal drinking water by \$1.562 per 1,000 gallons (Adams and Clark, 1989). For a 1,000,000 gallon per day facility supplying water to 3,000 to

4,000 homes, this would result in an added water treatment plant operating cost of \$1,562 daily or \$570,130 yearly. Conversely, in an optimal situation in which the water entering the system is low in organic material, a bed life of 730 days would result in a daily system cost of \$417 (\$152,205 annually) for the same size plant (Adams and Clark, 1989). These costs, as well as costs in the range of \$100,000 for the initial installation of the system, would have to be met by municipalities and passed on to the consumers. If an acceptable risk level of 10^{-6} were applied to this industry, municipalities would be faced with a dilemma. Since shutting down the public water treatment facility is not an option, each town and city would need to make costly process changes or apply to the state for an exemption from this regulation.

As a second example of the significant costs of implementing an across-the-board limit of risk, consider the changes that will be required at every gasoline station in the state. In order to reduce occupational exposures to benzene fumes from gasoline to the proposed 10^{-6} risk level, retrofitting of all pump nozzles and underground storage tanks would be required to recover vapors. According to a study conducted by the American Petroleum Institute in 1988 (personal communication, H. Thompson, API, 1990), the cost of retrofitting the average 6 nozzle service station with a vapor collection system ranges from \$2,582 to \$2,795 per nozzle. These costs would be passed on to the consumer in the form of increased gasoline prices. Likewise, similar types of costly vapor recovery systems would most likely be required at dry cleaning facilities in order to comply with a 10^{-6} risk level.

The food industry provides a final example of the costs of compliance. Based on the discussion presented on risks associated with common activities and consumption of natural carcinogens in food and beverages, many areas could be affected by a universal acceptable risk level of 10^{-6} . The risks associated with exposure to naturally-occurring carcinogenic substances in a number of foods and spices exceed the 10^{-6} level (Table 6-4). The costs of bringing these products into compliance with a 10^{-6} risk level would be high. For example, the aflatoxin mold is a carcinogenic substance which is associated with high moisture crops like peanuts and corn. When crops are subjected to the warm, moist, dark environments which are created during storage and transport, the spore is released and the mold grows. Reduction of aflatoxin levels for the purpose of compliance with a 10^{-6} risk level would require major changes in the technologies involved in storage and transport of fresh crops. Crops would need to be quickly and carefully dried and stored in cool environments in order to prevent release of the spore. Methods of transport to other parts of the country would

need to be modified in order to reduce the amount of time the crops are subjected to a moist, warm environment. Without major changes in the way the crops are handled, high percentages of these crops would not be permitted to be sold to consumers, and would need to be discarded. The cost to the consumer would be decreased availability and higher prices. The cost to the farmer or transporter would be significant financial loss.

Based on the approaches taken by the federal and state governments toward regulating chemicals in the environment, the adoption of a single acceptable risk level of 10^{-6} for all substances in all media is excessively conservative and may be technologically, economically and practically infeasible under certain circumstances. Acceptable risk levels need to be based on social, economic, and policy issues which are likely to vary by site and by substance.

6.4 Conclusions

The EPA has stated that it is most appropriate to model exposure to the most exposed population (MEP) rather than the most exposed individual (Inside EPA, 1991). As a result, it is population risk, as well as individual risk, that needs to be considered when establishing regulatory standards and cleanup goals. The population risk is the product of the individual risk and the size of the impacted population. When the size of the impacted population is small, the use of a less stringent risk level results in the same net population risk as that derived using a more stringent risk level and a larger population size. To arbitrarily use a 10^{-6} risk level for a small impacted population is overly conservative and not in keeping with regulatory precedents.

The use of the 10^{-6} level of risk has not been a consistent practice at either the federal or state level in risk management decision making. In fact, as has been clearly shown in the preceding discussion, risk management decisions made by the EPA, as well as numerous state agencies, have generally used considerably lower risk levels. It is reasonable that the West Virginia risk assessment policies be consistent with and reflect currently accepted practices.

Major business enterprises may be impacted by the establishment of a stringent acceptable risk level, should this risk level be applied to their industry. These industries could then find it necessary to either close their doors or apply for an exemption to the rule. Likewise, municipalities may be required to seek alternate methods to maintain a potable water supply for the general public

as an alternative to chlorination which would result in an unacceptable increase in cancer risk. Limits for the acceptable concentrations of hazardous contaminants in groundwater used for human consumption frequently exceed the 10^{-6} risk level.

Imposed unilaterally as a management guideline, a 10^{-6} risk level would be imprudent. The more appropriate approach to risk management decision making should address the issue of acceptable risk on a case-by-case basis.

7.0 RECOMMENDATION FOR A SCIENTIFICALLY SUPPORTABLE WATER QUALITY STANDARD

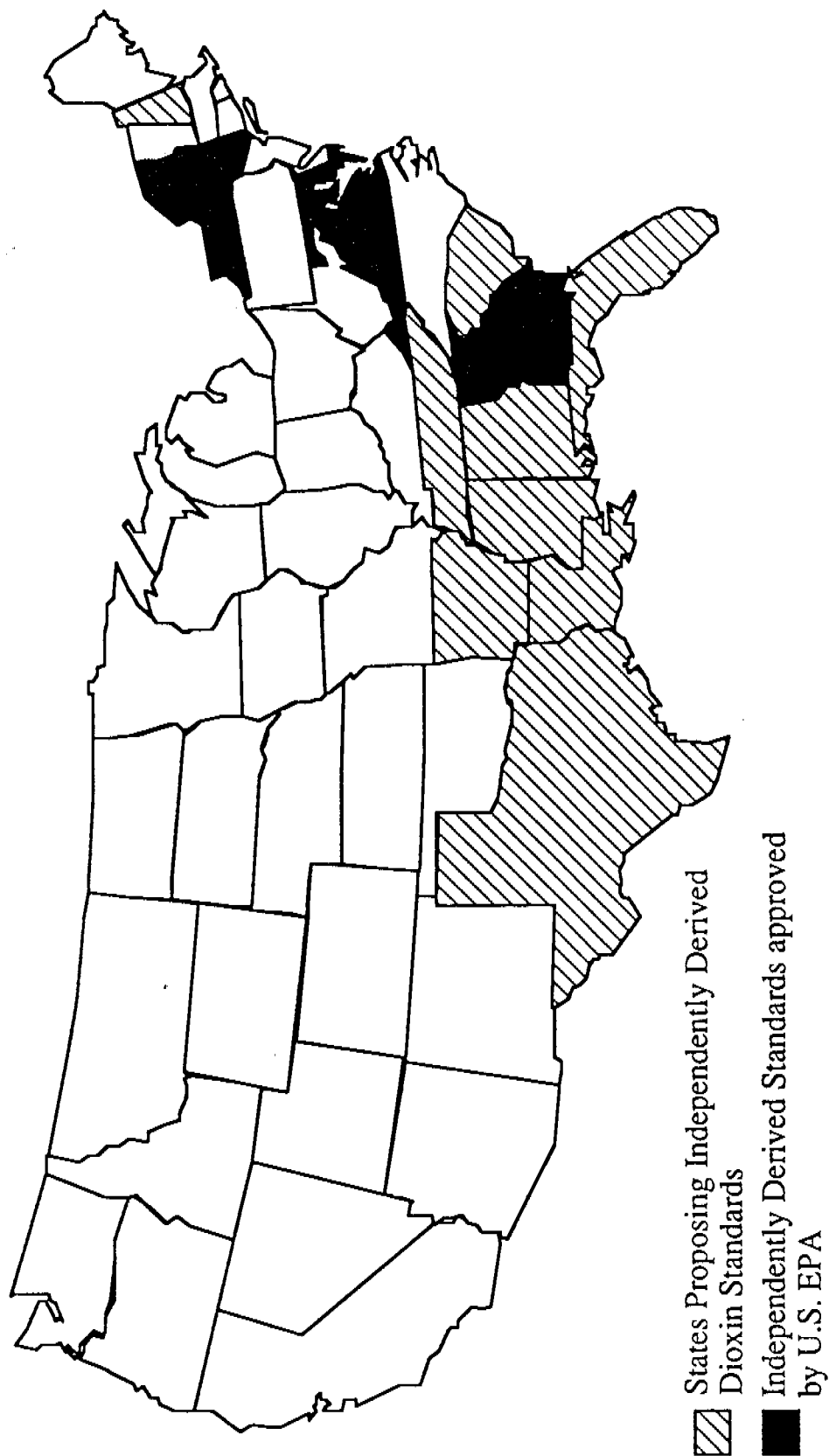
The federal government has mandated that by 1992, all states establish ambient water quality standards for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as one of the 126 "priority pollutants" listed under Section 307(a) of the Clean Water Act (CWA). One option available to states is to adopt the EPA Ambient Water Quality Criteria (AWQC) as enforceable water quality standards. A number of state agencies have taken this generic approach and have adopted the default values proposed in the EPA's 1984 document, *Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin*.

A second option, as referenced by Section 304(a)(1) of the CWA, is the development of a water quality standard based on the latest scientific knowledge regarding the effects of TCDD on human health and the environment. This option is particularly relevant in light of the recent announcement by EPA Administrator William Reilly that the EPA will conduct a year-long study to reevaluate the risks associated with dioxin exposure (EPA, 1991). In his announcement, Administrator Reilly stated, "our efforts to reduce risk must be based on the best available scientific information" (EPA, 1991).

A number of states including New Hampshire, Virginia, New York, Tennessee, South Carolina, Georgia, Alabama, Arkansas, Texas, and Maryland have chosen to use this approach in developing state-specific standards (Figure 7-1). New Hampshire and New York have adopted water quality standards of 1 ppq. EPA has recently approved the 1.2 ppq standards for TCDD adopted by Maryland, Virginia, and Georgia. These state-specific standards are substantially different from the criteria proposed in EPA's "Gold Book".

The EPA AWQC for dioxin (EPA, 1984) for bodies of water from which fish are consumed were calculated using the following equation:

Figure 7-1. States Proposing Independently Derived Dioxin Standards



$$WQS = (ADI \times BW) / (BCF \times FCR)$$

Where:

WQS	=	Water quality standard (pg/L)
ADI	=	Acceptable daily intake (pg/kg-day)
BW	=	Body weight (kg)
BCF	=	Bioconcentration factor (L water/kg fish)
FCR	=	Fish consumption rate (kg/day)

When deriving their AWQC for the ingestion of fish, the EPA (1984) assumed that the maximum allowable dose for humans was 0.006 pg/kg-day based on a linear, non-threshold model. They also assumed a fish consumption rate of 6.5 g/day, a BCF of 5,000, and a human body weight of 70 kg. Using these assumptions, an AWQC of 0.014 pg/L (ppq) was derived.

Since that time, however, considerably more has been learned about the behavior of dioxin. Our scientific understanding of the behavior of TCDD in the environment, in fish, and in humans has evolved to the degree that it would be imprudent for regulators and risk assessors to ignore these factors when establishing health-based regulatory standards. Only through a critical evaluation of these key variables can one derive a reasonable human health-based ambient water quality standard for dioxin.

There have been considerable advances in understanding the mechanism of dioxin's toxicity that have changed the way scientists view its potential effects on human populations. Recent scientific thought indicates that dioxin acts via a threshold mechanism and that its carcinogenic hazard has been overstated. Thus, acceptable dose levels of between 1 and 80 pg/kg-day have been proposed by various state agencies, and by the governments of Canada and several western European nations (HRS, 1991; WDH, 1991; Ontario, 1985; van der Heijden et al., 1982; NCASI, 1987; UK, 1989; Tollefson, 1991). These dose levels are orders of magnitude greater than the EPA's earlier estimate of 0.006 pg/kg-day.

Clearly, the more appropriate methods for extrapolating to humans are models which incorporate the threshold mechanism and the safety factor approach which recognizes the existence of a biological threshold. Based on the most current scientific evidence, an ADI in the range of 1 to 10 pg/kg-day is protective of human health for all toxic responses. A value of 1.0 pg/kg-day was chosen because it falls at the conservative end of the range of ADI values.

The rate of fish consumption is an important consideration in developing a water quality standard because the primary route of human exposure to TCDD in aquatic environments is through the ingestion of fish tissue (EPA, 1984). Although it is true that there are few comprehensive studies on freshwater fish consumption, a recreational use survey of the Ohio River provides an estimate of average fish consumption of between 2.1 and 5.8 g/day for anglers in this part of West Virginia. Using a conservative 6.5 g/day for the derivation of a standard for West Virginia is certainly conservative in lieu of this survey and surveys from other states.

When EPA (1984) developed its AWQC for TCDD, it used a BCF of 5,000 as a representative multiplier to predict how much dioxin aquatic organisms will accumulate from their surroundings. The BCF approach, however, addresses only the uptake of dissolved compounds across the membranous gill surfaces (EPA, 1989b). Recently, much scientific discussion has focused on the apparent inappropriateness of using the BCF approach in the AWQC equation. Attention has been placed on defining a factor, other than the BCF, that should be used in its place. Ideally, this "accumulation factor" should serve two functions:

- (1) realistically predict or estimate the accumulation of sorbed dioxin by aquatic organisms; and,
- (2) practically serve as an accumulation multiplier which can be implemented in a regulatory context for the purpose of establishing effluent permit limits.

An empirical approach to develop an accumulation factor that meets these two requirements has resulted in the definition of a Regulatory Bioaccumulation Multiplier or RBM (Sherman and Keenan, 1991). The RBM uses consistent criteria to define exposure concentrations of TCDD, normalizes to a common fish lipid content, and measures TCDD fish concentrations in the edible portion or fillet tissue. The use of the RBM allows regulatory agencies to directly calculate point source limits and obviates the need to apportion dioxin into different parts of the ecosystem (e.g., food, suspended sediment, and dissolved in water) via elaborate modelling techniques. Furthermore, the wide range of reported values for accumulation factors in the literature generally fall below a value of 5,000 when reanalyzed and reported as RBMs. Therefore, an RBM value of

5,000 constitutes the most scientifically based estimate of bioaccumulation for regulatory purposes in the AWQC equation.

Upon consideration of each of these factors, and an evaluation of the weight of scientific evidence supporting their selection, a scientifically based and health-protective standard for dioxin can be proposed. Based on a conservative and health-protective exposure level of 1.0 pg/kg-day, a body weight of 70 kg, an RBM of 5,000, and a fish consumption rate of 6.5 g/day for recreational anglers, the scientific evidence is supportive of a water quality standard of 2.1 ppq for the State of West Virginia using the following equation:

$$WQS = \frac{ADI \times BW}{(RBM \times FCR)}$$

Where:

WQS	=	Water quality standard (ppq)
ADI	=	Allowable daily intake (pg/kg-day)
BW	=	Body weight (kg)
RBM	=	Regulatory Bioaccumulation Multiplier (L water/kg fish)
FC	=	Fish consumption (kg/day)

However, since EPA Region III has already approved a water quality standard of 1.2 ppq for the states of Maryland and Virginia, it seems appropriate for West Virginia to adopt a 1.2 ppq standard as well. As it has been shown that NOECs for freshwater fish and other aquatic organisms range from 3.5 to 5.8 ppq, this proposed water quality standard will also be protective of those species.

8.0 REFERENCES

- Adams, W.J., G.M. DeGraeve, T.D. Sabourin, J.D. Cooney and G.M. Mosher. 1986. Toxicity and bioconcentration of 2,3,7,8-TCDD to fathead minnows (*Pimephales Promelas*). *Chemosphere* 15(9-12): 1503-1511.
- Adams, J.Q. and R.M. Clark. 1989. Cost estimates for GAC treatment systems. *J Amer. Water Works Assoc.* 81(1):35-42.
- Allen, J.R., J.P. Van Miller and D.H. Norback. 1975. Tissue distribution, accumulation and biological effects of [C14] tetrachlorodibenzo-p-dioxin in rats. *J. Food. Cosmet. Toxicol.* 13: 501-505.
- Allen, J.R., D.A. Barsotti, L.K. Lambrecht and J.P. van Miller. 1979. Reproductive effects of halogenated aromatic hydrocarbons on nonhuman primates. *Ann. NY Acad. Sci.* 320: 419-425.
- Allman, W.F. 1985. Staying alive in the 20th century: We have nothing to fear. *Science* 6(8):31-41.
- AMA. 1984. *The Health Effects of "Agent Orange" and Polychlorinated Dioxin Contaminants: An Update.* American Medical Association, Council on Scientific Affairs Advisory Panel on Toxic Substances, Chicago, IL. Updated 05-29-85.
- American Cancer Society. 1989. Cancer Facts and Figures.
- Ames, B.N. 1983. Dietary carcinogens and anticarcinogens. *Science* 221:1256-1264.
- Ames, B.N., R. Magaw and L.S. Gold. 1987. Ranking possible carcinogenic hazard. *Science* 236: 271-279.
- API. 1986. Benzene in Florida Groundwater: An Assessment of the Significance to Human Health. Florida Petroleum Council: A Division of the American Petroleum Institute. October.
- Armstrong, B. 1983. Australians report no link between service in Vietnam and birth defects among offspring. *Epidemiol. Monit.* 3(4):1.
- Atkinson, C. 1990. Personal communication regarding state of Connecticut hazardous waste site cleanup goals and regulations.
- Axelsson, O., L. Sundell, K. Anderson, C. Edling, C. Hogstedt and M. Kling. 1980. Herbicide exposure and tumor mortality. *Scand. J. Work Environ. Health* 6: 73-79 (cited in NCASI, 1987).
- Barsotti, D.A., L.J. Abrahamson and J.R. Allen. 1979. Hormonal alterations in female Rhesus monkeys fed a diet containing 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Bull. Environ. Toxicol.* 21: 463-469.
- Bayard, S.P. 1988. Quantitative implications of the use of different extrapolation procedures for low-dose cancer risk estimates from exposure to 2,3,7,8-TCDD. In: *A Cancer Risk-Specific Dose Estimate for 2,3,7,8-TCDD. Review Draft. Appendix A.* EPA/600/6-88/007Aa and Ab. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C.
- Beatty, P.W., M. A. Holscher and R.A. Neal. 1976. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in larval and adult forms of *Rana catesbeiana*. *Bull. Env. Cont. Toxicol.* 16(5): 578-581.
- Birnbaum, Linda. 1990. Consensus at Banbury DIOXIN Conference. United States Environmental Protection Agency. Health Effects Research Laboratory Research Triangle Park, North Carolina.

- Bleiberg, J., M. Wallen, R. Brodtkin and I.L. Applebaum. 1964. Industrially acquired porphyria. *Arch. Dermatology* 89: 793-797.
- Bond, G.G., M.G. Ott, F.E. Brenner and R.R. Cook. 1983. Medical and morbidity surveillance findings among employees potentially exposed to TCDD. *Brit. J. Ind. Med.* 40: 318-324.
- Bond, G.G., K.M. Bodner and R.R. Cook. 1989. Phenoxy herbicides and cancer: Insufficient epidemiologic evidence for a causal relationship. *Fund. Appl. Toxicol.* 12: 172-188.
- Borton, D. 1991. Memo to W. Gillespie, National Council of the Paper Industry for Air and Stream Improvement (NCASI), regarding: Evaluation of data used by EPA to support a bioconcentration factor (BCF) for 50,000 for 2,3,7,8-TCDD. February 5, 1991.
- Bowman, R.E., S.L. Schantz, M.L. Gross and S.A. Ferguson. 1989a. Behavioral effects in monkeys exposed to 2,3,7,8-TCDD transmitted maternally during gestation and for four months of nursing. *Chemosphere* 18(1-6): 235-242.
- Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross and D.A. Barsotti. 1989b. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 or 25 parts per trillion in the monkey: TCDD kinetics and dose-effects estimate of reproductive toxicity. *Chemosphere* 18(1-6): 235-242.
- Branson, D.R. 1983. Letter to C.E. Stephan. Dow Chemical U.S.A., Midland, MI. September 20. (cited in EPA, 1984).
- Branson, D.R., I.T. Takahashi, W.M. Parker and G.E. Blau. 1985. Bioconcentration kinetics of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. *Environ. Toxicol. Chem.* 47: 79-788.
- Brown, S.L., Brett, S.M., Gough, M., Rodericks, J.V., Tardiff, R.G., and Turnbull, D. 1988. Review of interspecies risk comparisons. *Regul. Toxicol. Pharmacol.* 8, 191-206.
- Bruzzi, P. 1983. Birth defects in the TCDD polluted area of Seveso: Results of a four-year follow-up. In: *Accidental Exposure to Dioxins, Human Health Aspects.* F. Coulston and F. Pochiari(eds.) Academic Press. New York. 271-280.
- California. 1987. Notice of Public Interest: Health and Welfare Agency the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65). State of California, Health and Welfare Agency, Office of the Secretary.
- Cantor, K.P., R. Hoover, P. Hartge et al. 1987. Bladder cancer, drinking water source, and tap water consumption: A case-control study. *National Cancer Institute* 79(6): 1269-1279. In: EPA, 1989d.
- CDC. 1984. Centers for Disease Control. *Morbidity and Mortality Weekly Report* 33: 54-61 (cited in Kimbrough and Houk, 1987).
- CDC. 1990. *The Association of Selected Cancers with Service in the U.S. Military in Vietnam. I. Non-Hodgkin's Lymphoma.* Selected Cancers Cooperative Study Group. March 29, 1990.
- Cheeke, P. R. and L. R. Shull. *Natural Toxicants in Feeds and Poisonous Plants.* AVI Publishing Company, Inc. Westport, Connecticut. 1985.

- ChemRisk. 1991. Consumption of freshwater fish by Maine anglers. ChemRisk™, A Division of McLaren/Hart. March 26, 1991.
- CMA. 1988. Chemicals in the Community: Methods to Evaluate Airborne Chemical Levels. Chemical Manufacturers Association, Washington, D.C. May.
- Cook, R.R., G.G. Bond, R.A. Olson, M.G. Ott and M.R. Gondek. 1986. Evaluation of the mortality experience of workers exposed to the chlorinated dioxins. *Chemosphere* 15(9-12): 1769-1776.
- Cook, P.M., A.R. Batterman, B.C. Butterworth, K.B. Lodge, and S.W. Kohlbry. 1990. *Laboratory Study of TCDD Bioaccumulation by Lake Trout from Lake Ontario Sediments, Food Chain and Water*. Ch. 6. United States Environmental Protection Agency, Environmental Research Laboratory, Duluth, MN.
- Cook, P.M., M.K. Walker, D.W. Kuehl and R.E. Peterson. 1991. Bioaccumulation and toxicity of 2,3,4,8-tetrachlorodibenzo-p-dioxin and related compounds in aquatic ecosystems. Preprint.
- Cooper, K.R., J. Schell, P. Kahn and M. Gallo. 1986. The effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on the development and survival of the Japanese Medaka (*Oryzias latipes*). Abstract No. 108. In: *The Toxicologist* Abstracts of the 25th Anniversary Meeting of the Society of Toxicology. Vol. 6 (1), March.
- Cooper, K.R. 1989. Effects of Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans on Aquatic Organisms. *Reviews in Aquatic Sciences* 1(2):227-242.
- Covello, V.T. 1989. Communicating right-to-know information on chemical risks. *Environ. Sci. Tech.* 23(12): 1444-1449.
- Crow, K.D. 1981. Chloracne and its potential clinical implications. *Exper. Dermatol.* 6243-257.
- Crouch, E.A.C. and R. Wilson. 1982. Risk/Benefit Analysis. Ballinger Publishing Company. Cambridge, MA.
- Crump, K., Allen, B., and Shipp, A. 1989. Choice of dose measure for extrapolating carcinogenic risk from animals to humans: An empirical investigation of 23 chemicals. *Health Physics* 57, 387-393.
- Dean, J. and R. Kimbrough. 1986. *Immunotoxicity of the Chlorinated Dibenzodioxins and Dibenzofurans*. In *Report of the Dioxin Update Committee*. Convened by EPA Office of Pesticides and Toxic Substances. Dioxin Update Conference, July 1-2.
- Derby, S.L. and R.L. Keeney. 1981. Risk analysis: Understanding 'How safe is safe enough?' *Risk Anal.* 1(3): 217-224.
- DHHS. 1989. *Fifth Annual Report on Carcinogens Summary 1989*. U.S. Department of Health and Human Services, Public Health Service. Washington, DC.
- Doll, R and R. Peto. 1981 *J. Natl. Cancer Inst.* 66:1193-1308 (as cited in Gough, 1989).
- Endicott, D.D., W.L. Richardson and D.M. Di Toro. 1988. Lake Ontario TCDD modeling report. U.S. Environmental Protection Agency, Washington, DC.
- EPA. 1983. Dow Chemical Company - Midland Plant Wastewater Characterization Study. Preliminary summary of results. U.S. EPA Region V, Environmental Sciences Division, Eastern District Office. March 28. (as cited in EPA, 1984).

EPA. 1984. *Ambient Water Quality Criteria for 2,3,7,8-tetrachloro-dibenzo-p-dioxin*. EPA 440/5-84-007. Office of Water Regulations and Standards, U.S. Environmental Protection Agency. Washington, D.C.

EPA. 1985. *Health Assessment Document for Polychlorinated Dibenzo-p-dioxins*. Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Washington, DC. EPA/600/8-84/014F. September.

EPA. 1986. *Development of Risk Assessment Methodology for Land Application and Distribution and Marketing of Municipal Sludge*. Prepared by Environmental Protection Agency. EPA/600/8-86/018A. June.

EPA. 1988. *A Cancer Risk-Specific Dose Estimate for 2,3,7,8-TCDD*. U.S. EPA, Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-88/007A. June.

EPA. 1989a. *Assessing human health risks from chemically contaminated fish and shellfish - A guidance manual*. U.S. Environmental Protection Agency, Office of Water Regulations and Standards. EPA 503/8-89-002. U.S. Environmental Protection Agency. September.

EPA. 1989b. *Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDDs and CDFs) a 1989 Update*. Risk Assessment Forum. U.S. Environmental Protection Agency, Washington, DC. EPA/625/3-89/016.

EPA. 1989c. *Exposure Factors Handbook*. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-89/043. July.

EPA. 1989d. *Risk Assessment Guidance for Superfund. Volume II: Environmental Evaluation Manual*. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA 540/1-89/001. March.

EPA. 1989e. *Health Effects Assessment Summary Tables*. Fourth Quarter. U.S. Environmental Protection Agency.

EPA. 1990a. *Risk Assessment for 2,3,7,8-TCDD and 2,3,7,8-TCDF Contaminated Receiving Waters From U.S. Chlorine-Bleaching Pulp and Paper Mills*. U.S. Environmental Protection Agency. August 1990.

EPA. 1990b. Technical support document for EPA's September 12, 1990 Approval/Disapproval of Maryland's Water Quality Standards revision. U.S. Environmental Protection Agency.

EPA. 1990c. Red Border Review of Proposed Revisions to National Contingency Plan. U.S. Environmental Protection Agency.

EPA. 1991. Technical support document for EPA's February 25, 1991 Approval of Virginia's Water Quality Standards revisions. U.S. Environmental Protection Agency.

Eriksson, J., L. Hardell, N. Berg and et. al. 1981. Soft-tissue sarcomas and exposure to chemical substances; a case-referent study. *Br. J. Indus. Med.* 38: 27-33.

Eriksson, J., J. Mulinare, P. McClain, et al. 1984. Vietnam veterans' risks for fathering babies with birth defects. *J. Am. Med. Assoc.* 252(7): 903-912.

- Evans, R.G., K.B. Webb, A.P. Knutsen, S.T. Roodman, D. Roberts, J. Bagby, W.A. Garrett and J.S. Andrews. 1987. A medical follow-up of the health effects of long-term exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. Seventh International Symposium of Chlorinated Dioxins and Related Compounds. Las Vegas, NV.
- Faccetti, S., A. Fomari, M. Montagna. 1981. Distribution of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the tissues of a person exposed to the toxic cloud of Seveso. *Forens. Environ. Appl.* 1: 1405-1414. (as cited in WDH, 1991).
- FDA. 1983. Statement by Sanford A. Miller, Ph.D., Director, Bureau of Foods, Food and Drug Administration, before the subcommittee on Natural Resources, Agriculture, Research and Environment, Committee on Science and Technology, U.S. House of Representatives. June 30.
- FDA. 1986. Biological bases for interspecies extrapolation of carcinogenicity data. Food and Drug Administration. July.
- Filippini, G., B. Bordo, P. Crenna, N. Massetto, M. Musicco, R. Boeri. 1981. Relationship between clinical and electrophysiological findings and indicators of heavy exposure to 2,3,7,8-tetrachlorodibenzo-dioxin. *Scand. J. Work. Environ. Health* 7: 257-262.
- Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland and A.J. Suruda. 1991a. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *N.Eng. J. Med.* 324(4): 212-218.
- Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honchar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland and A.J. Suruda. 1991b. *Mortality among U.S. workers employed in the production of chemicals contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)*. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health. January.
- Fishbein, L. 1987. Health-risk estimates for 2,3,7,8-tetrachlorodibenzodioxin: An overview. *Tox. and Ind. Health* 3(1): 91-134.
- Gallo, M. 1990. Statement on the outcome of The Banbury conference. "Biological Basis for Risk Assessment of Dioxins and Related Compounds".
- Gammage, R.B. 1990. Exposure to formaldehyde in indoor air. *Risk Anal.* 10(1): 77-83.
- Gart, J.J., Krewski, D., Lee, P.N., Tarone, R.E., and Wahrendorf, J. 1986. *Statistical Methods in Cancer Research. III. The Design and Analysis of Long-Term Animal Experiments*. International Agency for Research on Cancer, Lyon, France.
- Gasiewicz, T.A., J.R. Olson, L.E. Geiger and R.A. Neal. 1983. Absorption, distribution and metabolism of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in experimental animals. In: *Human and Environmental Risks of Chlorinated Dioxins and Related Compounds*. R.E. Tucker, A.L. Young and A.P. Gray(eds.) Plenum Press. New York. 495-525.
- Gillies, M.E. and H.V. Paulin, 1983. Variability of mineral intakes from drinking water: A possible explanation for the controversy over the relationship of water quality to cardiovascular disease. *International Journal of Epidemiology* 12(1): 45-50. In: EPA, 1989c.
- Gobas, F.A.P.C., W.Y. Shu and D. Mackay. 1987. *Factors Determining Partitioning of Hydrophobic Organic Chemicals in Aquatic Organisms*. D. Reidel Publishing Company. New York.

Gobas, F.A.P.C., Derek C.G. Muir and Donald Mackay. 1988. Dynamics of dietary bioaccumulation and faecal elimination of hydrophobic organic chemicals in fish. *Chemosphere* 17(5): 943-962.

Gobas, Frank A.P.C., Kathryn E. Clark, Wan Ying Shu and Donald Mackay. 1989. Bioconcentration of polybrominated benzenes and biphenyls and related superhydrophobic chemicals in fish: Role of bioavailability and elimination into the feces. *Environ. Tox. Chem.* 8: 231-245.

Goeden, H.M. and A.H. Smith. 1989. Estimation of human exposure from fish contaminated with dioxins and furans emitted by a resource-recovery facility. *Risk Analysis* 9(3): 377-383.

Goldstein, B.D. and N. Kim. 1989. Letter to Dr. Raymond C. Loehr, Chairman, Executive Committee of the Science Advisory Board. November 28. U.S. EPA, Washington, D.C.

Gough, M. 1990. How much cancer can EPA regulate away? *Risk Analysis*. 10(1): 1-6.

Gough, M. 1989. Estimating Cancer Mortality. *Environ. Sci. Technol.* 23(8): 925-930.

Graham, J.D. 1990. Letter to Dean C. Marriott. Commissioner, Department of Environmental Protection for the State of Maine. October.

Greenlee, W.F. 1988. Dioxin carcinogenesis - A perspective. *CIIT Activities* 8, 1-6.

Greenlee W.F., T.R. Skopek, K. Gaido and C. Walker. 1990. Comparative genetic mechanisms of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)-induced tumors. In: Mouse Liver Carcinogenesis: Mechanisms and Species Comparison D.E. Stevenson, J.A. Pop, J.M. Ward, R.M. McClain, T.J. Slaga, H.C. Pitot (eds). Alan R. Liss, Inc. NY, NY.

Greenwald, P., B. Kovaszny, D. Collins and G. Therriault. 1984. Sarcomas of soft tissue after Vietnam service. *JNCI* 73: 1107-1109.

Greig, J.B. and F. Dematties. 1973. Effects of TCDD on drug metabolism and hepatic microsomes in rats and mice. *Environ. Health Perspect.* 5: 211-219. (as cited in WDH, 1991).

Hardell, L., M. Eriksson, P. Lenner and E. Lundgren. 1981. Malignant Lymphoma And Exposure To Chemicals, Especially Organic Solvents, Chlorophenols And Phenoxy Acids: A Case-Control Study. *Br. J. Cancer* 43: 169-176.

Hardell, L. and A. Sandstrom. 1979. Case-control study: Soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. *Br. J. Cancer* 39: 711-717.

Harris, M., Zacharewski, T., Piskorska-Pliszczyńska, J., Rosengren, R., and Safe, S. (1990). Structure-dependent induction of aryl hydrocarbon hydroxylase activity in C57BL/6 mice by 2,3,7,8-tetrachlorodibenzo-p-dioxin and related congeners: Mechanistic studies. *Toxicol. Appl. Pharmacol.* 105, 243-253.

Haseman, J.K. (1990). Use of statistical decision rules for evaluating laboratory animal carcinogenicity studies. *Fundam. Appl. Toxicol.* 14, 637-648.

Hawkes, C.L. and L.A. Norris. 1977. Chronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) to rainbow trout. *Trans. Am. Fish. Soc.* 106(6): 641-645.

- Helder, T. 1980. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on early life stages of pike (*Esox lucius* L.). *Sci. Total Environ.* 14(3): 255-265.
- Helder, T. 1981. Effects of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) on early life stages of rainbow trout (*Salmo gairdneri* Richardson). *Toxicol.* 19: 101-112.
- Hoar, S., A. Blair, F. Holmes, C. Boysen, R. Robel, R. Hoover and J. Fraumeni. 1986. Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *JAMA* 256(9): 1141-1147.
- Hobson, L.B. 1984. Human effect of TCDD exposure. *Bull. Environ. Contam. Toxicol.* 33:696-701.
- Hoffman, R.E., P.A. Stehr-Green, K.B. Webb, R.G. Evans, A.P. Knutsen, W.F. Schramm, J.L. Staake, B.B. Gibson and K.R. Steinberg. 1986. Health effects of long-term exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *JAMA* 255: 2031-2038.
- Holloway, M. 1990. A Great Poison. Science and the Citizen. *Sci. Am.* November, 1990.
- Honstead, J.F., T.M. Beetle and J.K. Soldat. 1971. A Statistical Study of the Habits of Local Fishermen and Its Application to Evaluation of Environmental Dose, A Report to the Environmental Protection Agency by Battelle Pacific Northwest Laboratories, Richland, WA 99352. (cited in TetraTech, 1988).
- HRS. 1990. Scientific Justification for the Department of Health and Rehabilitative Service Recommendation for Human Consumption of Fish Containing TCDD. Florida Department of Health and Rehabilitative Services, December.
- Humphrey, H.E.B. 1978. Personal communication. (as cited in Rupp, 1980).
- Hutt, et al. 1978. (as cited in Rodericks and Taylor, 1983).
- IARC. 1987. Evaluation of Carcinogenic Risks to Humans. In: supplement 6. Volumes 1 to 42. International Agency for Research on Cancer. Lyon, France.
- ICRP. 1974. Report of the Task Group on Reference Man. International Commission on Radiological Protection.
- Inside EPA. 1991. New, Less Conservative EPA Risk Model Could Spawn Less Stringent Results. Inside EPA Weekly Report. Volume 12(13): 1. March 29.
- Javitz, H. 1980. Seafood consumption data analysis. SRI International. Final report prepared for EPA Office of Water Regulations and Standards. EPA Contract 68-01-3887.
- Jirasek, L., J. Kalensky and K. Kubec. 1973. Acne chlorina and porphyria cutanea tarda during the manufacture of herbicides. *Cesk. Dermatol.* 48: 306-317. (as cited in UAREP, 1988).
- Jirasek, L. J. Kalensky, K. Kubec, J. Pazderove and E. Lukes. 1974. Acne chlorina, porphyria cutanea tarde and other manifestations of general poisoning during the manufacture of herbicides. II. *Cesk. Dermatol.* 49: 145-157. (as cited in UAREP, 1988).
- Kang, H.K., L. Weatherbee, P. Breslin, Y. Lee and B.M. Shepard. 1986. Soft-tissue sarcomas and military service in Vietnam: A case comparison group analysis of hospital patients. *J. Occupational Med.* 28: 1215-1218.

Keenan, R.E., R.J. Wenning, A.H. Parsons and D.J. Paustenbach. 1990a. A re-evaluation of the tumor histopathology of Kociba et al. (1978) using 1990 criteria: Implications for the risk assessment of 2,3,7,8-TCDD using the linearized multi-stage model. Proceedings Dioxin '90. Tenth International Conference on Organohalogen Compounds, Volume 1: EPRI Seminar Toxicology-Environment, Food, Exposure-Risk, ed by O. Hutzinger and H. Fiedler, pp. 549-554. Ecoinforma Press, Bayreuth, FRG.

Keenan, R.E., A.H. Parsons, E.S. Ebert, R.J. Wenning, and D.J. Paustenbach. 1990b. Setting rational health-based water quality standards for dioxin risk assessment for the Columbia River. Proceedings, 1990 TAPPI Conference.

Keenan, R.E., D.J. Paustenbach, R.J. Wenning and A.H. Parsons. 1991a. A re-evaluation of the tumor histopathology of Kociba et al. (1978) using 1990 criteria: Implications for the risk assessment of 2,3,7,8-TCDD using the linearized multi-stage model. *Chemosphere* (Submitted).

Keenan, R.E., D.J. Paustenbach, R.J. Wenning and A.H. Parsons. 1991b. A pathology re-evaluation of the Kociba et al. (1978) bioassay of 2,3,7,8-TCDD. Implications for risk assessment. *J. Tox. Env. Health* (In Press).

Kenaga, E.E. 1980. Correlation of bioconcentration factors of chemicals in aquatic and terrestrial organisms with their physical and chemical properties. *Amer. Chem. Society* 14(5): 553-556.

Kenaga, E.E. and C.A.I. Goring. 1980. Relationship between water solubility, soil sorption, octanol-water partitioning, and concentration of chemicals in biota. In: *Aquatic Toxicology* J.C. Eaton, P.R. Parrish and A.C. Hendricks (eds). American Society for Testing and Materials. p. 78-115.

Kimbrough, R., H. Falk, P. Stehr and G. Fries. 1984. Health implications of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) contamination of residential soil. *J. Toxicol. Environ. Health* 14: 47-93.

Kimbrough, R.D. 1990. How toxic is 2,3,7,8-tetrachlorodibenzodioxin to humans? *J. Tox. Environ. Health* 30: 261-271.

Kimbrough, R.D. 1991. Examining and communicating risk from dioxin exposure. Conference Proceedings from 1991 Rifkin Conference, "Dioxin: National Conference on Establishing Multi-Media Controls", May 9-10, Washington D.C.

Kimbrough, R.D. and V.N. Houk. 1987. Effects of Chlorinated Dibenzodioxins. In: *Solving Hazardous Waste Problems*. American Chemical Society(eds.) Washington, DC. 68-79.

Kinouchi, T., H. Tsutsui, Y. Obnishi. 1986. *Mutat. Res.* 171: 105. (as cited in Ames et al., 1987).

Klaassen, C.D., M.O. Amdur and J. Doull. 1986. Casarett and Doull's Toxicology. MacMillan Publishing Company, New York.

Kleeman, J.M., J.R. Olson, S.M. Chen and R.E. Peterson. 1986a. Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. *Toxicol. & Appl. Pharm.* 83: 391-401.

Kleeman, J.M., J.R. Olson, S.M. Chen and R.E. Peterson. 1986b. 2,3,7,8-Tetrachlorodibenzo-p-dioxin metabolism and disposition in Yellow Perch. *Toxicol. & Appl. Pharm.* 83: 402-411.

Kleeman, J.M., J.R. Olson and R.E. Peterson. 1988. Species differences in 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity and biotransformation in fish. *Fund. Appl. Tox.* 10: 206-213.

Kociba, R. and B. Schwetz. 1982. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). *Drug Metab. Rev.* 13(3): 387-406.

Kociba, R.J. 1984. Summary and Critique of Rodent Carcinogenicity Studies of Chlorinated Dibenzo-p-dioxins. In: *Public Health Risks of the Dioxins*. W.W. Lowrance (ed.) William Kaufmann, Los Altos. 77-98.

Kociba, R. and O. Cabey. 1985. Comparative toxicity and biologic activity of chlorinated dibenzo-p-dioxins and furans relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). *Chemosphere* 14(6/7): 649-660.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46: 279-303.

Kuehl, D W., P. M. Cook, A. R. Batterman, D Lothenbach and B. C. Butterworth. 1987. Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp. *Chemosphere* 16(4): 667-678.

Lakind, J. and E. Rifkin. 1990. Current method for setting dioxin limits in water requires reexamination. *Environ. Sci. Technol.* 24(7): 963-965.

Landolt, M.L., F.R. Hafer, A. Nevissi, G. van Belle, K. Van Ness, and C. Rockwell. 1985. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound. NOAA Technical Memorandum NOS OMA 23. National Oceanic and Atmospheric Administration, Rockville, MD.

Landolt, M.L., D.L. Kalman, A. Nevissi, G. van Belle, K. Van Ness, and F.R. Hafer. 1987. Potential toxicant exposure among consumers of recreationally caught fish from urban embayments of Puget Sound. NOAA Technical Memorandum NOS OMA 33. National Oceanic and Atmospheric Administration, Rockville, MD.

Lathrop, G.D., W.H. Wolfe, S.G. Machado, J.E. Michalek, T.G. Karrison, J.C. Miner, W.D. Grubbs, M.R. Peterson and W.F. Thomas. 1987. *Air Force Health Study: An Epidemiologic Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicides*. Science Applications International Corporation, VA and Epidemiology Division, United States Air Force. October.

LeBlanc, G.A. 1984. Interspecies relationships in acute toxicity of chemicals to aquatic organisms. *Environ. Toxicol. Chem.* 3:47-60.

Lehnert, V.G. and D. Szadkowski. 1986. The Carcinogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in humans: Evaluation of liability. *Arbeitsmed. Sozialmed. Praventivmed.* 21: 153-175 (cited in UAREP, 1988).

Leung, H.W., D.J. Paustenbach, F.J. Murray, M.E. Anderson. 1990. A physiological pharmacokinetic description of the tissue distribution and enzyme induction properties of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the rat. *Toxicol. Appl. Pharmacol.* 103: 399-410.

Lipson, A., W. Gaffery and F. LaVecchio. 1983. Letter to the Editor regarding Agent Orange and birth defects. *N.Engl. J. Med.* 309(8): 491-492.

Lodge, K.P. and P.M. Cook. 1989. Partitioning studies between the sediment and water: The measurement of Koc for Lake Ontario sediment. *Chemosphere* 19(1-6): 439-444.

Maine-DHS. 1988. Policy for Identifying and Assessing the Health Risks of Toxic Substances. Maine Department of Human Services, Environmental Toxicology Program, Division of Disease Control, Bureau of Health. 1: 310.1012. February.

- Maronpot, R.R., C.A. Montgomery Jr., G.A. Boorman and E.E. McConnell. 1986. National Toxicology Program nomenclature for hepatoproliferative lesions of rats. *Toxicol. Path.* 14(2): 263-273.
- Mass DEQE. 1988. Massachusetts Contingency Plan Fact Sheet Waiver of Department Approvals for Non-Priority Disposal Sites (310 CMR 40.533). Commonwealth of Massachusetts, Executive Office of Environmental Affairs, Department of Environmental Quality Engineering, Division of Hazardous Waste. April.
- Mastroiacovo, P., A. Spagnolo, E. Marni, L. Meazza, R. Bertollini and G. Segni. 1988. Birth defects in the Seveso area after TCDD contamination. *JAMA* 259(11): 1668-1672.
- Mausner, J.S. and S. Kramer. 1985. *Epidemiology*. W.B. Saunders Company. Philadelphia, PA. 361 p.
- McConnell, E.E., H.A. Solleveld, J.A. Swenberg and G.A. Boorman. 1988. Guidelines for combining neoplasms for evaluation of rodent carcinogenesis studies. In: *Carcinogenicity: The Design, Analysis and Interpretation of Longterm Animal Studies*. ILSI Monographs. H.C. Grice and J.L. Ciminieri(eds.) Springer-Verlag. New York, NY. 183-196.
- McKim, J., P. Schmeider and G. Veith. 1985. Absorption dynamics of organic chemical transport across trout gills as related to octanol-water partition coefficient. *Tox. Appl. Pharm.* 77: 1-10.
- McNulty, W.P. 1984. Fetotoxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) Rhesus macaques (*Macaca mulatta*). *Am. J. Primatol.* 6: 41-47.
- Mehrle, P.M., D.R. Buckler, E.W. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. De Graeve, J.J. Coyle and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-Tetrachlorodibenzofuran in rainbow trout. *Environ. Tox. Chem.* 7: 47-62.
- Meistrich, M.L. 1988. Estimation of human reproductive risk from animal studies: Determination of interspecies extrapolation factors for steroid hormone effects on the male. *Risk Anal.* 8, 27-33.
- Meunz, L.R. and B. Peterson. 1990. Final report on the analysis of fish consumption data. Technical Assessment Systems, INC. Prepared for U.S. Environmental Protection Agency. August 3, 1990.
- Miles, B. 1991. Fish consumption near the proposed Ashton Pulp Plant. Memorandum from B. Miles, Dept. of Commerce, Labor and Environmental Resources, State of West Virginia, to J.E. Hamrick III, Director. June 10.
- Miller, R.A., L.A. Norris and C.L. Hawkes. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. *Environ. Health Perspect.* 5: 177-186.
- Miller, R.A., L.A. Norris and B.R. Loper. 1979. The response of coho salmon and guppies to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in water. *Trans. Am. Fish. Soc.* 105: 401-407.
- Minister of Veterans' Affairs. 1983. Case-control study of congenital anomalies and Vietnam service. Australian Government Publishing Service (as cited in AMA, 1984).
- Moore, L.W., H. Matheny, T. Tyree, D. Sabatini and S.J. Klaine. 1988. Agricultural runoff modeling in a small west Tennessee watershed. *J. WPCF* 60(2): 242-249.
- Mordenti, J. 1986. Man versus beast: Pharmacokinetic scaling in mammals. *J. Pharmacol. Sci.* 75, 1,028-1,040.

Moses, M. R. Lilis, K.D. Crow, J. Thornton, A. Fischbein, H.A. Anderson, I. J. Selikoff. 1984. Health status of workers with past exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in the manufacture of 2,4,5-tetra-chlorophenoxy-acetic acid: Comparison of findings with and without chloracne. *Am. J. Ind. Med.* 5: 161-182.

Mower, B. 1990. Maine's Dioxin monitoring program. 1988-1990. December.

Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba and B.A. Schwetz. 1979. Three-generation reproductive study of rats given 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the diet. *Toxicol. Appl. Pharmacol.* 50: 241-252.

Murphy, M. 1990. Personal communication regarding state of Massachusetts regulations.

NAS. 1983. *Risk Assessment in the Federal Government: Managing the Process*. National Academy Press. Washington, DC.

Nash, D.A. 1971. A survey of fish purchases of socio-economic characteristics. Data Report No. 62. National Marine Fisheries Service, Seattle, WA. 162pp. (cited in Tetra Tech, 1988).

NCASI. 1987. *Dioxin: A Critical Review of its Distribution, Mechanism of Action, Impacts on Human Health, and the Setting of Acceptable Exposure Limits*. National Council of Paper Industry for Air and Stream Improvement, Inc., New York. NCASI Technical Bulletin No. 524.

NCASI. 1991. Attached submittal to EPA of information derived from the NCASI experimental streams program on estimated 'No Effect' concentrations of TCDD/TCDF on fish. Memorandum from W.J. Gillespie to Corporate Dioxin Contacts Operating Committee. National Council of the Paper Industry for Air and Stream Improvement, Inc. January 4.

Nebert, D.W. and N.M. Jensen. 1979. The Ah locus; genetic regulation of the metabolism of carcinogens, drugs, and other environmental chemicals by cytochrome P-450-mediated monooxygenases. *CRC Crit. Rev. Biochem.* 6:401-437. (as cited in WDH, 1991).

Neely, W. B. 1979. Estimating rate constants for the uptake and clearance of chemicals by fish. *Environ. Sci. Technol.* 13(12): 1506-1510.

Neely, W.B. 1983. Letter to C.E. Stephan. Dow Chemical U.S.A., Midland, MI. June 10. (as cited in EPA, 1984).

Nelson, C.J., J.F. Holson, H.G. Green and D.W. Gaylor. 1979. Retrospective study of the relationship between agricultural use of 2,4,5-T and cleft palate occurrence in Arkansas. *Teratology* 19: 377-384.

Nessel, C.S., M.A. Amoruso, T.H. Umbreit and M.A. Gallo. 1990. Hepatic Aryl hydrocarbon hydroxylase and cytochrome P450 induction following the transpulmonary absorption of TCDD from intratracheally instilled particles. *Fund. Appl. Toxicol.* 15(3): 500-509.

Niimi, A.J. and B.G. Oliver. 1986. Biological half-lives of chlorinated dibenzo-p-dioxins and dibenzofurans in rainbow trout. *Environ. Toxicol. Chem.* 5: 49-53.

Nisbet, I.C.T. and M.B. Paxton. 1982. Statistical aspects of three-generation studies of the reproductive toxicity of TCDD and 2,4,5-T. *Am. Stat.* 36(3): 290-298.

NMFS. 1989. *Fisheries of the United States, 1988*. National Oceanic and Atmospheric Administration National Marine Fisheries Service. Current Fishery Statistics No. 8800. U.S. Department of Commerce, Washington, D.C. May.

Norris, L.A. and R.A. Miller. 1973. The toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in guppies (*poecilia reticulatus* peters). *Bull. Environ. Contamin. Tech.* 12(1): 76-80.

Nygren, M., C. Rappe, G. Lindstrom, M. Hansson, P-A. Bergqvist, S. Marklund, L. Domellof, L. Hardell and M. Olsson (1986). Identification of 2,3,7,8-substituted polychlorinated dioxins and dibenzofurans in environmental and human samples. In: Chlorinated Dioxins and Dibenzofurans in Perspective. C. Rappe, G. Choudhary, L.H. Keith, eds. Lewis Publishers, Inc., Chelsea, Michigan.

Olson, J.R., T.A. Gasiewicz and R.A. Neal. 1980. Tissue distribution, excretion, and metabolism of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) in the Golden Syrian hamster. *Toxicol. Appl. Pharmacol.* 56: 78-85.

Ontario Ministry of the Environment. 1985. *Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs)*. Intergovernmental Relations and Hazardous Contaminants Coordination Branch. Scientific Criteria Document for Standard Development No. 4-84. September.

OSHA. 1989. OSHA-final rule air contaminants-permissible exposure limits (Title 29 Code of Federal Regulations Part 1910.1000). Am. Ind. Hyg. Assoc.

Parsons, A.H., S.L. Huntley, E.S. Ebert, E.R. Algeo and R.E. Keenan. 1991. Risk Assessment for dioxin in Columbia River Fish. *Chemosphere* (submitted).

Patterson, D.G. Jr., L. Hampton, C.R. Lapeza Jr., W.T. Belser, V. Green, L. Alexander and L.L. Needham. High-resolution gas chromatographic/high-resolution mass spectrometric analysis of human serum on a whole-weight and lipid basis for 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Anal. Chem.* 59: 2000-2004.

Pazderova-Vejlupkova, J., M. Nemcova, J. Pickova, L. Jirasek and E. Lukas. 1981. The development and prognosis of chronic intoxication by tetrachlorodibenzo-p-dioxins in men. *Arch. Environ. Health* 36(1): 5-11.

Pearce, N.E., A.H. Smith and D.O. Fisher. 1985. Malignant lymphoma and multiple myeloma linked with agricultural occupations in a New Zealand Cancer Registry-based study. *Amer. J. Epidemiology* 121: 225-237.

Pearce, N.E., A.H. Smith, J.R. Howard, R.A. Sheppard, H.J. Giles and C.A. Teague. 1986. Non-Hodgkin's lymphoma, and exposure to phenoxy herbicides, chlorophenols, fencing work and meat works employment: A case control study. *Br. J. Ind. Med.* 43: 75-83.

Pennington, J.A.T. 1983. Revision of the total diet study food list and diets. *J. Amer. Dietetic Assoc.* 82(2): 166-173.

Pierce, D., D. Noviello and S.H. Rogers. 1981. *Commencement Bay seafood consumption study*. Preliminary report. Tacoma-Pierce County Health Dept., Tacoma Washington. December.

Pocchiari, F., V. Silano, and A. Zampieri. 1979. Human health effects from accidental release of tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. *Ann. NY Acad Sci.* 320: 311-320.

Poland, A.P., E. Glover, A.S. Kende, M. DeCamp and C.M. Giandomenico. 1976. 3,4,3',4'-tetrachloro azoxybenzene and azobenzene: Potent inducers of aryl hydrocarbon hydroxylase. *Science* 194: 627-630.

- Poland, A.P. and J.C. Knutson. 1982. 2,3,7,8-Tetrachlorodibenzodioxin; rates of volatilization and photolysis in the environment. *Environ. Sci. & Tech.* 20: 490-492.
- Poland, A.P., D. Smith, G. Metter and P. Possick. 1971. A health survey of workers in a 2,4-D and 2,4,5-T plant with special attention to chloracne, porphyria cutanea tarda, and psychological parameters. *Arch. Environ. Health* 22: 316-327.
- Poland A.P., J. Knutson, and E. Glover. 1985. Studies on the mechanism of action of halogenated aromatic hydrocarbons. *Clin. Physiol. Chem.* 3: 157-174.
- Poland, A. P., P. Greiger and E. Glover. 1990. Factors affecting the elimination rate of [I125I] -2-iodo-3,7,8-trichlorodibenzo-p-dioxin in mice as determined by whole body counting. {Manuscript in prep.}
- Portier, C.J., Hoel, D.G., and Ryzin, J.V. 1984. Statistical analysis of the carcinogenesis bioassay data relating to the risks from exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. In *Public Health Risks of the Dioxins*. (Lowrance, W.W., Ed.) William Kaufmann, Los Altos, CA. pp. 99-120.
- Puffer, H.W., S.P. Azen, M.J. Duda and D.R. Young. 1981. Consumption rates of potentially hazardous marine fish caught in the metropolitan Los Angeles area. University of Southern California School of Medicine for Environmental Research Laboratory, U.S. Environmental Protection Agency. Grant No. R. 807 120010.
- PWG. 1990a. *Pathology Working Group Report on 2,3,7,8-Tetrachlorodibenzo-p-dioxin Chronic Toxicity/Carcinogenicity Study in Sprague-Dawley Rats*. Prepared by R.M. Sauer, PWG Chairperson, PATCHO, INC. and submitted to R.A. Michaels, Chairperson, Maine Scientific Advisory Panel. March 13.
- PWG. 1990b. *Hepatotoxicity in Female Sprague-Dawley Rats Treated with 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)*. Prepared by R.M. Sauer, PWG Chairperson, and D.G. Goodman, PATHCO, INC. and submitted to R.A. Michaels, Chairperson, Maine Scientific Advisory Panel. April 27.
- Rand, G.M. and S.R. Petrocelli. 1985. *Fundamentals of Aquatic Toxicology*. Hemisphere Publishing Corp., New York, NY.
- Reggiani, G. 1978. Medical problems raised by the TCDD contamination in Seveso, Italy. *Arch. Toxicol.* 40: 161-188.
- Reggiani, G. 1980. Acute human exposure to TCDD in Seveso, Italy. *J. Toxicol. Environ. Health* 6(10):27-43.
- Rifkin, E. and J. Lakind, 1991 Dioxin Bioaccumulation: Key to a sound risk assessment methodology. *J. Toxicol. Env. Health.* (submitted).
- Roberts, L. 1991. Flap erupts over dioxin meeting. *Science* 251: 866-867.
- Rodricks, J.V. and M.R. Taylor. 1983. Role of risk assessment to food safety decision making. *Reg. Tox. Pharm.* 3:275-284, 302-307.
- Rodricks, J.V., S.M. Brett and G.C. Wrenn. 1987. Significant risk decisions in federal regulatory agencies. *Reg. Tox. Pharm.* 7:307-320.
- Rodricks, J.V. and M.R. Taylor. 1989. Comparison of risk management in U.S. regulatory agencies. *J. Hazmat* 21:239-253.

- Rupp, Elizabeth M. 1980. Age Dependent Values of Dietary Intake for Assessing Human Exposures to Environmental Pollutants. *Health Physics* 39:151-163.
- Rupp, E.M., F.L. Miller and I.C.F. Baes. 1980. Some results of recent surveys of fish and shellfish consumption by age and region of U.S. residents. *Health Physics* 39:165-175.
- Ryan, J.J., A. Schecter, R. Lizotte, W. Sun and L. Miller. 1985. Tissue distribution of dioxins and furans in humans from the general population. *Chemosphere* 14(6/7):929-932.
- Safe, S.H. 1986. Comparative toxicology and mechanism of action of polychlorinated dibenzo-p-dioxins and dibenzofurans. *Annu. Rev. Pharmacol. Toxicol.* 26:371-399. (as cited in WDH, 1991).
- Schantz, S.L., D.A. Barsotti and J.R. Allen. 1979. Toxicological effects produced in nonhuman primates chronically exposed to 50 part per trillion 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Abstract # 360, In: *Abstracts of the Eighteenth Annual Meeting, Society of Toxicology Applied Pharmacology*.
- Schantz, S.L., N.K. Laughlin, H.C. Van Valkenberg and R.E. Bowman. 1986a. Maternal care by Rhesus monkeys of infant monkeys exposed to either lead or 2,3,7,8-tetrachlorodibenzo-p-dioxin. *NeuroToxicology* 7(2):637-650.
- Schantz, S.L., R.E. Bowman, S.A. Ferguson, M.C. VanValkenberg and E.M. Boehm. 1986b. Discrimination-reversal learning in offspring of rhesus monkeys exposed to low levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Soc. for Neurosci. Abstracts* 12(November):
- Scheuplein, R.J. 1990a. Statement on the outcome of The Banbury Conference "Biological Basis for Risk Assessment of Dioxins and Related Compounds".
- Scheuplein, R.J. 1990b. Perspectives on Toxicological Risk: An Example: Food-Borne Carcinogenic Risk. Food and Drug Administration.
- Schmitt, D.N. and J.H. Hornsby. 1985. A fisheries of the Savannah River. Georgia Department of Natural Resources, Game and Fish Division. September.
- Sherman, W. 1990. Personal communication. Maine Science Advisory Panel.
- Sherman, W.R. and R.E. Keenan. 1991. A comparison of pure water bioconcentration factors, field simulated bioaccumulation factors and field estimated bioaccumulation factors for dioxin and the relationship between these values. (To be submitted).
- Shu, H., D. Paustenbach and F.J. Murray. 1987. A critical evaluation of the use of mutagenesis, carcinogenesis, and tumor promotion data in a cancer risk assessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Reg. Toxicol. Pharm.* 7:57-88.
- Sijm, D.T.H.M. and A. Opperhuizen. 1988. Biotransformation, Bioaccumulation and Lethality of 2,8-Dichlorodibenzo-p-dioxin: A Proposal To Explain the Biotic Fate and Toxicity of PCDD's and PCDF's. *Chemosphere* 17(1): 83-99.
- Silbergeld, E.K. and T.A. Gasiewicz. 1989. Dioxins and the Ah receptor. *Amer. J. Indust. Med.* 16:455-474.
- Smith, A.B., J. Schloemer, L.K. Lowry, A.W. Smallwood, R.N. Ligo, S. Tanaka, W. Stringer, M. Jones, R. Hervin and C.J. Glueck. 1982. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. *Brit. J. Ind. Med.* 39:361-369.

Smith, A.H., D.O. Fisher, N. Pearce and C. Chapman. 1982a. Congenital defects and miscarriages among New Zealand 2,4,5-T sprayers. *Arch. Environ. Health* 37(4):197-200.

Smith, A.H., D.O. Fisher, N. Pearce and C.A. Teague. 1982b. Do agricultural chemicals cause soft tissue sarcoma? Initial findings of a case-control study in New Zealand. *Community Health Studies* 6(2):114-119.

Smith, A.H., D.O. Fisher, H.J. Giles and N. Pearce. 1983. The New Zealand soft tissue sarcoma case-control study. Interview findings concerning phenoxyacetic acid exposure. *Chemosphere* 12(4/5):565-571.

Smith, A.H. and N.E. Pearce. 1986. Update on soft tissue sarcoma and phenoxyherbicides in New Zealand. *Chemosphere* 15(9-12):1795-1798.

Smith, F.A., B.A. Schwetz and K.D. Nitschke. 1976. Teratogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in CF-1 mice. *Toxicol. Appl. Pharmacol.* 38:517-532.

Smith, L.M., T.R. Schwartz and K. Feltz. 1990. Determination and occurrence of AHH-active polychlorinated biphenyls, 2,3,7,8-tetrachloro-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran in Lake Michigan sediment and biota. The question of their relative toxicological significance. *Chemosphere* 21(9): 1063-1085.

Soldat, J.K. 1970. A Statistical Study of the Habits of Fishermen Utilizing the Columbia River Below Hanford. Chapter 25. In: *Environmental Surveillance In the Vicinity of Nuclear Facilities: Proceedings of a Symposium Sponsored by the Health Physics Society*. January 24-26, 1968. William C. Reinig (ed.) Springfield, IL, Charles C. Thomas. 302-308.

Spacie, A. and J.L. Hamelik. 1982. Alternative Models for Describing the bioconcentration of organics in fish. *Environ. Toxicol. Chem.* 1:309-320.

Spigarelli, S.A., M.M. Thommes and A.L. Jensen. 1982. Prediction of Chemical Accumulation by Fish. Ecological Sciences Section Radiological and Environmental Research Division, Argonne National Laboratory. PB824-156918. January 1982.

Spitsbergen, J.M., K.A. Schat, J.M. Kleeman, and R.E. Peterson. 1987. Interactions of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) with immune response of rainbow trout. *Vet. Immunol. Immunopathol.*, 12:263. In: Cooper, 1989.

Spitsbergen, J.M., J.M. Kleeman and R.E. Peterson. 1988a. Morphologic lesions and acute toxicity in rainbow trout (*Salmo gairdneri*) treated with 2,3,7,8-tetrachlorodibenzo-p-dioxin. *J. Tox. Env. Health* 23: 333-358.

Spitsbergen, J.M., J.M. Kleeman and R.E. Peterson. 1988b. 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity in yellow perch (*Perca flavescens*). *J. Tox. Env. Health* 23: 359-383.

Squire, R.A. and M.H. Levitt. 1975. Report of a workshop on classification of specific hepatocellular lesions in rats. *Cancer Research* 35:3214-3223.

Squire, R.A. 1990. Letter to LaJuana S. Wilcher, Assistant Administrator, Environmental Protection Agency. May 24.

Squire, R.A. 1991. *An Assessment of Potential Carcinogenic Risk from 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)*. May.

Sugimura T. et al. 1986. In: *Genetic Toxicology of the Diet*. I. Knudsen, Ed. Liss, New York. pp. 85-107 (as cited in Ames et al., 1987).

- Suskind, R.R. 1983. Long-term health effects of exposure to 2,4,5-T and/or its contaminants. *Chemosphere* 12:79.
- Suskind, R.R. and V.S. Hertzberg. 1984. Human health effects of 2,4,5-T and its toxic contaminants. *JAMA* 251(18):2372-2380.
- Suskind, R.R. 1985. Chloracne, the hallmark of dioxin intoxication. *Scand. J. Work Environ. Health*. 11:165-171.
- Thiess, A.M., R. Frentzel-Beyme and R. Link. 1982. Mortality study of persons exposed to dioxin in a trichlorophenol process accident that occurred in the BASF AG on November 17, 1953. *Am. J. Ind. Med.* 31:79-189 (cited in NCASI, 1987).
- Thomas, N.A. 1983. Memorandum to C.E. Stepan, U.S. EPA Duluth, MN. July 22. (as cited in EPA, 1984).
- Tollefson, L. 1991. Use of epidemiology data to assess the cancer risk of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Reg. Tox. & Pharm.* 13:150-169.
- Travis, C.C., S.A. Richter, E.A. Crouch, R. Wilson and E. Wilson. 1987. Cancer Risk Management. A review of 132 federal regulatory decisions. *Environ. Sci. Technol.* 21(5):415-420.
- Travis, C.C. and H. A. Hattemer-Frey. 1987. Human exposure to 2,3,7,8-TCDD. *Chemosphere* 16(10-12): 2331-2342.
- Turcotte, M-D.S. 1983. Georgia Fishery Study: Implications for dose-calculations. DuPont de Nemours & Co., Arkey, SC. Sponsored by Dept. of Energy, Washington, DC. August 5, 1983. Report # DE86-008041.
- UAREP. 1988. *Human Health Aspects of Environmental Exposure to Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans*. Universities Associated for Research and Education in Pathology, Inc. June.
- UK. 1989. *Dioxins in the Environment*. United Kingdom. Pollution Paper No. 27.
- Umbreit, T.H. and Gallo, M.J. 1988. Physiological implications of estrogen receptor modulation by 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol. Lett.* 42, 5-14.
- USDA. 1983. Food Consumption: Households in the United States, Seasons and Year 1977-1978. U.S. Department of Agriculture. Report # H-6. June.
- van der Heijden, C.A., A. Knapp, P. Kramers, M. Van Logten. 1982. Evaluation of the carcinogenicity and mutagenicity of 2,3,4,8-tetrachlorodibenzo-1,4-dioxin (TCDD) classification and no-effect level. State Institute of National Health. Bilthoven, The Netherlands.
- van der Heijden, C.A. 1990. Statement on the outcome of the Banbury Conference "Biological Basis for Risk Assessment of Dioxins and Related Compounds".
- Veith, G.D. and P. Kosian. 1983. Estimating bioconcentration potential from octanol/water partition coefficients. In: *PCBs in the Great Lakes*. D. Mackay, R. Patterson, S. Eisenreich and M. Simmons (eds) Ann Arbor Science Publishers. (as cited in EPA, 1984).
- Veith, G.D., K.J. Macek, S.R. Petrocelli and J. Carroll. 1980. An evaluation of using partition coefficients and water solubility to estimate bioconcentration factors for organic chemicals in fish. *Aquatic Toxicol.* 1:116-129. (as cited in EPA, 1984)

West, P., J.M. Fly, R. Marans and F. Larkin. 1989. Michigan sport anglers fish consumption survey. Supplement I - Non-response bias and consumption suppression effect adjustments, A report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #2. September.

Wallace, L. 1990. Major sources of exposure to benzene and other volatile organic chemicals. *Risk Anal.* 10(1): 59-64.

WDH. 1991. Health Implications of PCDD and PCDF Concentrations Reported from Lake Roosevelt Sport Fish. Washington State Department of Health. April.

WHO. 1990. Press release: Consultation on tolerable daily intake from food of PCDDs and PCDFs.

Wiklund, K. and L-E. Holm. 1986. Soft tissue sarcoma risk in Swedish agricultural and forestry workers. *JNCI* 76(2):229-234.

Wilson, R. and E.A.C. Crouch. 1987. Risk assessment and comparisons: An introduction. *Science* 236: 267-270.

Wilson, R. 1979. Analyzing the daily risks of life. *Tech. Rev.* 81(4): 41-46.

Wisk, J.D. and K.R. Cooper. 1990. The stage specific toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in embryos of the Japanese medaka (*Oryzias latipes*). *Env. Tox. Chem.* 9: 1159-1169.

Yamamoto, K.R. 1985. Steroid receptor-regulated transcription of specific genes and gene networks. *Annu. Rev. Genet.* 19, 209-252.

Yockim, R.S., A.R. Isensee and G.E. Jones. 1978. Distribution and toxicity of TCDD and 2,4,5-T in an aquatic model ecosystem. *Chemosphere* 3: 215-220.

Young, F.E. 1987. Risk assessment: The convergence of science and the law. *Reg. Tox. Pharm.* 7:179-184.

Zack, J.A. and W.R. Gaffey. 1983. A mortality study of workers employed at the Monsanto Company plant in Nitro, West Virginia. *Environ. Sci. Res.* 26: 575-591.

Zack, J.A. and R.R. Suskind. 1980. The mortality experience of workers exposed to tetrachlorodibenzodioxin in a trichlorophenol process accident. *J. Occup. Med.* 22(1): 11-15.

Zingeser, M.R. 1979. Anomalous development of the soft palate in rhesus macaques (*Macaca mulatta*) prenatally exposed to 3,4,7,8-tetrachlorodibenzo-p-dioxin. Oregon Regional Primate Research Center, Beaverton, OR.

Zober, A., P. Messerer and P. Huber. 1990. Thirty-four-year mortality follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident. *Int. Arch. Occup. Environ. Health* 62: 139-157.

APPENDIX A

Review of Reported Fish Bioaccumulation/Bioconcentration Factors

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Review of Reported Fish Bioaccumulation/Bioconcentration Factors

The bioconcentration of TCDD has been evaluated in a number of studies, based on both predictive and measured data. Several studies have developed equations for predicting the BCF for an organic chemical based on its octanol-water partition coefficient, K_{ow} (Kenaga and Goring, 1980; Veith et al., 1980; Veith and Kosian, 1983; cited in EPA, 1984). Several K_{ow} values may be found in the literature for a given chemical, since factors such as pH, temperature, purity of the chemical, purity of the solvents, time of phase separation, and time of mixing may influence the results (Kenaga and Goring, 1980). For many chemicals, K_{ow} values are not available, and must therefore be predicted. For TCDD, octanol-water partition coefficients have been measured (Neely, 1979, 1983; Kenaga, 1980; Branson, 1983; cited in EPA, 1984). Using the measured partition coefficient, 6.15, in various equations (Kenaga and Goring, 1980; Veith et al., 1980; Veith and Kosian, 1983), the predicted BCFs range from 3,000 to 68,000.

In its AWQC document for dioxin (EPA, 1984), the EPA recommended a BCF of 5,000 on the basis of several studies. In one field study, a whole-body BCF of 2,000 was reported for channel catfish kept in a cage in a discharge plume of a river for 28 days (EPA, 1983; Thomas, 1983; cited in EPA, 1984). In a laboratory study the steady-state whole-body BCF in rainbow trout was projected to be 5,450 if growth dilution was not taken into account, and 9,270 if a correction is made for growth dilution (Branson et al., 1983; cited in EPA, 1984).

Cook et al. (1990) conducted laboratory experiments using contaminated food, sediment, and water to simulate exposure to lake trout from the Lake Ontario. These researchers verified TCDD concentrations and carefully accounted for losses through sorption processes, water outflow and fish uptake. The precision of their analyses, exemplified by comparing duplicate tanks results in which all but one duplicate produced TCDD concentrations within 7.5% of the mean, adds to the reliability of the data. The results of Cook et al. (1990) showed a whole-body BAF range of 1,860 - 22,700 (median = 6,660) with seven of nine exposure groups resulting in BAF values less than the mean of 7,230. If the 0.5 correction factor suggested by EPA (1990a) to compensate for the unequal partitioning of contaminants between the edible and inedible fish tissues is used, then the mean BAF for TCDD would be 3,615 (range: 930 - 11,350).

Additional field sampling also was conducted to verify the data collected in the laboratory. Because TCDD cannot be detected in the water of aquatic ecosystems, even when biota are highly contaminated (Cook et al., 1990, 1991), modeling was required to estimate the dissolved water concentration of TCDD in Lake Ontario. Based on this, Cook et al. (1990) reported a BAF of 140,000. It is likely, however, that this modeled BAF considerably overestimated the actual BAF as the model used (Endicott et al., 1988) predicted that 80% of the TCDD was dissolved in the water column. This is an unreasonably high percentage due to the superhydrophobic characteristic of TCDD. Cook and coworkers (1990) have attributed the differences between the laboratory values and the field values to two factors: 1) the technical inability to accurately measure dissolved TCDD concentrations in Lake Ontario, and 2) TCDD adsorption to suspended solids in laboratory experiments.

William Sherman, a member of the Maine Scientific Advisory Panel, recently reevaluated the model (Endicott et al., 1988) used by Cook et al. (1990) to develop a BAF of 140,000 for Lake

Ontario. The Lake Ontario model (Endicott et al., 1988) predicts a TCDD input of 1,000 g/yr into Lake Ontario (Borton, 1991). The measured flow rate of Lake Ontario is 7,100 m³/sec. Using this information a nominal TCDD concentration of 4.5 ppq can be calculated. Sherman (1990) derived a BAF based on the nominal concentration, a lipid correction from 18% to 2.5%, and a correction factor of 0.5 to convert from whole body to fillet concentrations. In his reanalysis, Sherman (1990) derived a BAF of approximately 5,000.

It has been suggested that the BCF should be increased to 50,000 based on a previously unpublished BCF study of fathead minnows and carp conducted in 1986 by Cook et al. (1991). Cook et al. (1991) cited that study, as well as the study by Mehrle et al. (1988), as supporting an increase in the BCF (normalized to 7 % lipid) to 51,000. However, it is critical to note that, in these studies, the concentrations of dioxin used produced toxicity in the test animals. Mehrle et al. (1988) noted in their report that BCF determinations should only be estimated in fish at exposure levels that do not induce toxic responses.

There are several additional points of concern which may influence application of the Cook et al. (1991) data for regulatory purposes. First, carrier solvents were used to enhance the solubility of TCDD in both the Mehrle et al. (1988) and Cook et al. (1991) studies. Cook et al. (1991) recommends that future investigations avoid using this technique with superhydrophilic compounds such as TCDD.

The BCF values estimated in the 1986 study cited by Cook et al. (1991) were based on two exposure treatments for fathead minnows and three exposure treatments for young carp. One of the carp exposure groups had been previously exposed for 105 days to a mixture of 1,2,3,4-, 1,3,6,8- and 1,3,7,9-TCDD. Despite the large number of fish in each exposure group, only a single aquarium (experimental unit) was used for each treatment level in both studies. Such a small number of exposure groups precludes one's ability to apply any parametric statistical test of significance. Furthermore, due to mortality associated TCDD toxicity, the experiment was terminated earlier than originally scheduled. As a result, only two samples were collected during the depuration phase of the experiment. The combination of toxic effects, which likely altered depuration rates, and the small number of sample points used to calculate the BCF limited the conclusions that can be drawn from these two studies and therefore limit its usefulness in a regulatory context.

Lower BCF values have been reported in the scientific literature. Adams et al. (1986) report a whole-body BCF of 7,900 for fathead minnows. In several rainbow trout studies, whole-body BCF values were reported to range from 9,270 to 39,000 (Mehrle et al., 1988; Branson et al., 1985).

A reanalysis of the data reported by Mehrle et al. (1988) indicates that the steady-state correlation in fathead minnows may have occurred at the lowest exposure level (38 pg/L) between 14 and 21 days when the calculations are based on the TCDD concentrations measured in the water rather than on average values. Using the actual data, the BCF was 22,647 at 14 days, 24,146 at 21 days, and 22,273 at 28 days. If steady-state occurred at 21 days in the lowest exposure group, then the steady-state BCF would be approximately 24,000.

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June 25, 1991

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JUN 28 1991

West Virginia State Water Resources Board
1260 Greenbriar Street
Charleston, West Virginia 25311

WATER RESOURCES BOARD

Re: Proposed Amendments to Water Quality Standards

Dear Sirs:

I am submitting these comments regarding the proposed water quality standard for dioxin on behalf of Apple Grove Pulp & Paper Company. I am an environmental lawyer in private practice in Washington, DC. I spent ten years at the United States Environmental Protection Agency from 1976 to 1986. I was legal counsel to EPA's water program from 1976 through 1980. From 1980 through 1984 I was the Director of EPA's Office of Water Regulations and Standards ("OWRS"). As Director of OWRS, I was responsible for EPA's water quality standards program and for the 1983 EPA rulemaking which developed the federal water quality standard regulations which are still applicable today.

My comments are presented for the following reasons:

- A. To support the development of an appropriate dioxin water quality standard which provides for protection for human health and the environment and yet which also allows for necessary industrial development;
- B. to emphasize that under the federal statute and regulations the states have the primary authority to determine the appropriate water quality standards and criteria to protect the public health and the environment;
- C. to note that a dioxin standard of 1.2 ppq has been adopted by the neighboring states of Virginia and Maryland and has been approved by EPA; and
- D. to emphasize that the policy decision of setting an appropriate risk level of protection for a carcinogen is one where states have, and should utilize, great

West Virginia State Water
Resources Board
Page 2

flexibility, and where EPA itself has exercised significant flexibility.

THE APPROPRIATE STANDARD

I am not a scientist, but it must be clear that there is no one right or wrong number for dioxin. The risk assessment process utilized by EPA and by other government agencies here and abroad has led to estimates of dioxin risk which differ from agency to agency by three to four orders of magnitude. All of these agencies are looking at the same animal tests; attempting to extrapolate from a two-year rat study in which rats were fed very high doses of dioxin to determine what cancer risks might exist for human beings who may be exposed to very small levels of dioxin over a lifetime. Such an extrapolation is based not on science, but on regulatory policy - and such regulatory policy varies dramatically from government agency to government agency. I should note, however, that the EPA methods for analyzing chemical risks are the most conservative of those used by any government agency and result in the most stringent numbers. I should also note that the EPA Administrator, Bill Reilly, has recently announced that the Agency is reconsidering entirely its dioxin risk assessment based upon some significant new data, much of which suggests that dioxin may not be of as much concern as EPA previously estimated when people are exposed only to very low levels of the chemical.

STATE PRIMACY

The federal Clean Water Act clearly provides that states have the primary responsibility to develop appropriate water quality standards in each state. EPA's role is limited to publishing recommended criteria for specific constituents and to reviewing state rulemaking decisions regarding state water quality standards. In its regulations and in its guidance documents, EPA has constantly emphasized that the states are not required to follow the EPA recommended criteria, but instead are free to develop criteria appropriate in each state, utilizing their own scientific judgment and policy judgment. In fact, a total of 38 states have now developed dioxin criteria and EPA has approved 27 of the 38 state actions. Of the 38 states which have adopted numerical criteria for dioxin, only 16 states have adopted the criteria recommended by EPA while 22 states have adopted criteria different than those recommended by EPA.

West Virginia State Water
Resources Board
Page 3

The extent to which states have developed independent criteria for dioxin rather than relying upon the EPA criteria is unusual. For most constituents, states have historically tended to adopt EPA criteria. It is certainly much easier to adopt an EPA criteria, rather than to independently develop a state criteria. In fact, a number of states have adopted the EPA criteria wholesale for all pollutants by reference to the federal documents. That the majority of states which have adopted numerical dioxin standards have developed criteria different than those recommended by EPA, and have undertaken to scientifically and judicially defend their independent criteria, is clearly a demonstration that significant scientific controversy surrounds the dioxin issue and the EPA criteria recommendation. EPA's acceptance of other criteria is a repeated demonstration that a broad range of state criteria are within EPA's own standards for protection of the environment and public health.

The two neighboring states of Virginia and Maryland, with whom West Virginia shares common waterways, have adopted a dioxin standard of 1.2 ppq. Since this standard of 1.2 ppq has been approved by EPA for both Virginia and Maryland, it is clear that EPA would approve a similar standard for West Virginia, if such a standard is supported by a scientific rationale similar to that developed in those two states.

THE APPROPRIATE RISK LEVEL

Recent public discussion regarding dioxin, EPA risk analysis for carcinogens, and the water quality criteria may incorrectly lead some to believe that EPA has an absolute commitment to a 1×10^{-6} risk as the only acceptable risk level for a carcinogen. Nothing could be further from the truth. In fact, EPA's historical position regarding water quality criteria has been that the only absolutely risk-free water quality criteria for any carcinogen is zero, since EPA policy assumes that some risk exists for any exposure to a carcinogen.

In first developing its recommended water quality criteria for carcinogens, EPA both recommended a zero level and then provided numerical values corresponding to risks of 10^{-5} , 10^{-6} , and 10^{-7} . EPA, when publishing such recommendations, did not provide any preference for any particular level of carcinogen risk. Most recently, of course, EPA has suggested that a 10^{-6} level of protection is appropriate. It is clear, however, that although

West Virginia State Water
Resources Board
Page 4

EPA suggests such a level of protection can be set by states, when EPA actually develops regulations for carcinogens under a variety of statutes, it most often provides for a risk level less stringent than 10^{-6} , often a 10^{-5} or even 10^{-4} risk level. It seems clear that when EPA must consider the pragmatic implications of its decisions, as it must when it develops enforceable regulations, as opposed to guidance and suggestions for states, EPA does consider and balance the economic implications of its decisions with the scientific information available. For example, under the Safe Drinking Water Act, where EPA regulates carcinogens in drinking water, EPA has recently promulgated Maximum Contaminant Levels ("MCLs") for a variety of carcinogens at a risk range between 10^{-4} and 10^{-6} . In fact, most of the MCLs for carcinogens are set at either the 10^{-4} or 10^{-5} risk level. When regulating pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act and the Food, Drug, and Cosmetic Act, EPA has allowed for the continued registration of a number of pesticides where the cancer risk level was estimated to be either 10^{-4} or 10^{-5} . Where it is regulating hazardous air pollutants under Section 112 of the Clean Air Act, EPA has regulated carcinogens such as radionuclides, vinyl chloride, benzene, and arsenic most often at a level of 10^{-4} . Finally, under the Comprehensive Environmental Response, Compensation, and Liability Act, the so-called Superfund, EPA has stated through its National Contingency Plan that for known or expected carcinogens clean-up levels should be set at a level that poses a risk between 10^{-4} and 10^{-6} .

Thus, the 10^{-6} risk level, which EPA suggests to states in their setting water quality standards, is significantly more stringent than the risk levels which EPA itself adopts as a regulation when it is itself forced to regulate and control a particular substance or a particular industry. EPA's own decision-making provides very adequate justification for a state adoption of a risk level less stringent than 10^{-6} .

In conclusion, the State Water Resources Board clearly has the general flexibility to set an appropriate water quality standard for dioxin; in addition, the demonstrated range of scientific conclusions and policy assumptions regarding dioxin provide a significant basis upon which to exercise such flexibility. By proposing a criteria significantly more stringent than those developed by other federal agencies and by regulatory authorities in other countries, and yet at the same

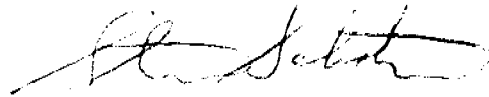
MORGAN, LEWIS & BOCKIUS

West Virginia State Water
Resources Board
Page 5

time by accepting state decisions which result in significantly less stringent criteria for dioxin, EPA itself has clearly signaled to all states that the regulation of dioxin is an area in which significant flexibility can and should be exercised.

I appreciate the opportunity to participate in this rulemaking exercise.

Sincerely yours,

A handwritten signature in dark ink, appearing to read "Steven Schatzow", with a stylized, flowing script.

Steven Schatzow

SS:tr

ALABAMA RIVER PULP COMPANY INC.



P. O. Box 100
Claiborne Mill
Perdue Hill, Alabama 36470
Phone (205) 575-2000

June 27, 1991

West Virginia State Water Resources Board
1260 Greenbriar Street
Charleston, West Virginia 25311

Dear Sir,

Our sister company, Apple Grove Pulp and Paper Company, Inc. submits the following documents for your consideration on the establishment of a water quality standard for 2,3,7,8 - Tetrachloro-Dibenzo-p-Dioxin:

- (1) Support Document for the Establishment of a Water Standard for Dioxin for the State of West Virginia

by: ChemRisk
A Division of McLaren/Hart
Stroudwater Crossing
1685 Congress Street
Portland, Maine 04102

- (2) West Virginia Ambient Water Quality Standard for 2,3,7,8-TETRACHLORO-DIBENZO-p-DIOXIN

by: Joseph V. Rodricks, Ph.D.,
Senior Vice President of
EVIRON Corporation
Arlington, Virginia

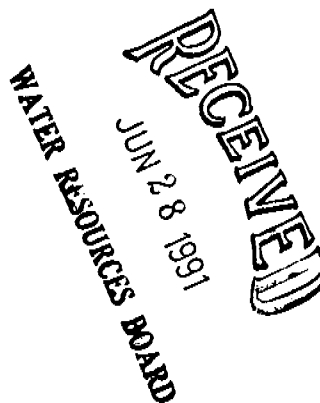
The two consulting firms conclude that a dioxin standard of 1.2 ppq is conservative and protective of human health. Apple Grove Pulp and Paper Company, Inc. respectfully requests that the West Virginia State Water Resource Board adopt 1.2 ppq as their dioxin standard.

Sincerely,

Donald B. Morris
Manager of Environmental Affairs

DBM:ab

Hand Deliver 7/28/91



TEXT OF COMMENTS MADE TO WEST VIRGINIA STATE WATER RESOURCES BOARD,
JUNE 20, 1991.

I'm Stuart Calwell of the law firm of Calwell, McCormick and Peyton, Charleston, West Virginia. I am appearing on behalf of the Tri-State Building and Construction Trades Council, which is an association of Construction Trades Unions affiliated with the AFL-CIO. The association is made up of local trade unions in West Virginia, Ohio, and Kentucky. The Tri-State Building and Construction Trades Council is also a member of the West Virginia Building and Construction Trades Council. The members of these local unions live in hundreds of communities in Ohio, West Virginia and Kentucky, and I am appearing on behalf of them as well. Collectively, these unions represent over 44,000 tradesmen. Their families and the people in the communities where they live are worried about your decision to increase the amount of cancer causing chemicals that can lawfully be dumped into the rivers and streams of West Virginia, and the rivers that border our surrounding states. Our recent studies of certain rivers in West Virginia show that if your proposed rule goes into effect, we can expect, in the rivers studied, an increase of 5.5 to almost 8 times the amounts of carcinogens presently dumped in those rivers. Because of this increase, we can expect an increase in the risk of cancer to many of the citizens and residents of West Virginia, Ohio, and Kentucky.

Proposed Rule 8.2.c., appearing on page 19 of the

new proposed rules, mandates that "critical design flow for carcinogens shall be the harmonic mean flows" when a permit applicant requests it. However, your notice of this proposed rule change does not explain to the public that, with this rule, you are advocating increasing the risk of cancer to human beings.

As you know, Proposed Rule 8.2.c. has to do with how the dilution of carcinogens is calculated once they are discharged to a river. This calculation is important in determining the risk of cancer posed by the discharge. The more water there is in a river to dilute the carcinogen, the lower the risk of cancer. Low water in a river means there's not as much water to dilute a carcinogen. Therefore, if the low water mark of a river is used when deciding how much of a carcinogen discharge to allow, the amount of carcinogen permitted will be small, because when the river's at its low mark, there's not as much water available for dilution. It follows, therefore, that if the low water mark is used to fix the maximum amount of carcinogens discharged, then, when the river is up, the carcinogen is diluted even more. Thus, as river flow increases, the levels of cancer causing chemicals become more and more diluted, and the risk of cancer goes down. During periods of draught and low water, because the initial determination of levels of carcinogens was based on the low water mark, no increased risk of cancer occurs.

The present rule, the one you are trying to change, has this built-in safeguard. It requires dischargers to limit carcinogens to the amount that can be discharged when a river is

at the low water mark. The new rule, the one you are trying to implement, does not use the low water mark as the starting point to calculate how much river water there is to dilute carcinogens. Instead, the so-called "harmonic mean flow" is recommended as the starting point. The "harmonic mean flow" of a river is always at a higher water mark than the low water mark. Therefore, from the very beginning, there will be an increase in the amount of carcinogens that can be dumped into a river. The risk of cancer, therefore, is increased from the beginning. In times of low water, because there is less water to dilute the already increased amount of carcinogens allowed by the new rule, the risk of cancer goes even higher. Our studies show that the new rule will allow dischargers on the Ohio River to dump 7.4 times more cancer causing substances into the river than the present rule allows. On the Kanawha River, 5.4 times more cancer causing substances will be allowed, and on the Greenbriar River 7.8 times more cancer causing chemicals will be allowed.

Clearly, the new rule will put West Virginia on the cutting edge of increasing the risk of cancer to humans. What you are proposing, with this new rule, is to take the state backwards in the fight against the risk of cancer. Instead of working to eliminate the risk of cancer, you are actually working to increase the risk of cancer. Dischargers that are presently meeting the limits imposed by the old low water mark rule, will be allowed to begin dumping, in some instances, almost 8 times more cancer causing agents into our rivers. In other instances, it could be

much higher than 8 times. No one knows for sure because you haven't evaluated the rivers and streams of West Virginia to find out. New companies will be allowed to locate in our state and begin dumping more carcinogens than ever allowed since the beginning of regulatory time in West Virginia.

Our information is that you have not adequately investigated the impact the proposed rule change will have on the environment and human health. EPA Technical Support Document For Water Quality-based Toxics Control, March, 1991, recommends that the "harmonic mean flow" be calculated directly from the historical daily flow records of effected rivers and streams. No such investigation has been undertaken by the Water Resources Board. We recommend the board undertake this investigation and calculate the increased risk of cancer implementation of the new rule will occasion. If, after undertaking such an investigation you still insist on recommending this change, at least make a plain statement to the people of West Virginia that what you are proposing is to increase the risk of cancer in this state. Call it what it is: rule to increase the risk of cancer. Don't try to sneak it through as a "rule to implement harmonic mean design flow."

We will be submitting a written comment embodying the results of our studies with the aid of Carpenter Environmental Associates of Ramsey, New Jersey, on or before June 28, 1991.

Health Sciences Center

West Virginia University

School of Medicine
Institute of Occupational Health and Safety
2264 Health Sciences Center South
Morgantown, WV 26506
304 293-3693

Mario C. Battigelli, M.D.—Director
Robert B. Reger, Ph.D.—Research Associate Professor

24 June 1991

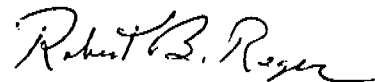
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Ref: Public Hearing, 20 June 1991, "Water Quality Standards",
Charleston, WV

Sir:

Thank you for the opportunity of providing testimony at the referenced public hearing. A typescript of my testimony is enclosed.

Sincerely,

A handwritten signature in cursive script, reading "Robert B. Reger".

Robert B. Reger

PUBLIC HEARING STATEMENT

before the

State Water Resource Board
Charleston, WV
20 June 1991

Mr. Chairman & Members of the Board:

My name is Robert B. Reger. I am a Research Associate Professor, Institute of Occupational Health and Safety, WV University School of Medicine, Morgantown, WV. I am here this evening representing myself and Westvaco. The subject on which I will speak is dioxin.

The major purpose of this testimony is not to put forth a magic number which will provide homo sapiens with ample and adequate protection from dioxin -- but rather to put forth some concepts to be considered in the development of a sensible standard.

In general, what do we know about the health effects from dioxin?

In humans, chloracne can result -- these skin lesions can last a long time and when healing occurs, significant scarring can develop. Liver function may also be impaired.

While central and peripheral nervous system effects have been reported (fatigue, drowsiness, depression, sleep disturbance, etc.), chloracne remains the definite documented condition in man which has resulted from dioxin exposure.

Of major concern, however, for the setting of water (and other) standards, is the carcinogenic potential of substances (such as dioxin) which exist at very low levels.

The issue of carcinogenesis (relating to dioxin) when viewed in its infancy, could clearly not be settled using the experience of man -- or with human epidemiological studies.

Thus, the synthesis of other information was necessary. Cancer of the liver and other sites have been induced in animal species -- rats, mice, guinea pigs. Vast inter species differences exist and high doses of dioxin have been administered to evoke carcinogenic responses. From such data, specific models for disease outcome have thus developed which are (or were) based largely on our dose response experience with other substances. For example, a popular basic model involves the thesis that cancer is caused by the mutation of cellular DNA. Continued decreasing doses of a material, therefore, (in animals and short term bioassays) become related to observable induced mutations. In short, this hypothesized model involves no threshold of

response. It is thus suggested that any exposure (however small) is still associated with a finite positive likelihood of deleterious effects.

From this type of information, how do we obtain estimates of carcinogenic risk for man from exceedingly low level exposures to some chemical compound; e.g. dioxin? Results from the high dose animal experimentation are firstly extrapolated to untested and unproven low dose exposure levels within the same species. Secondly, extrapolation is then made across species to homo sapiens. The validity of the results from this double extrapolation procedure is directly correlated with the validity of at least two basic assumptions; generally whether or not an implied linear and non threshold model for disease exists and whether or not man is like a mouse. Clearly, both of these assumptions are now and have been open to serious question. However, in the absense of other information, forming opinions and basing regulations on the only information available might be regarded as sensible.

To be sure, the framework of toxic substance regulation rests on the fragile premise that it is possible to identify which chemicals are hazardous and should be regulated and which are safe and can be ignored. This premise, in turn, rests on the assumption that there is a close correlation between carcinogenic effects in animals and those in man. Moreover, it may also rest on the even greater assumption that a similar correlation exists between results from short term assays and long term tests in animals (e.g. skin painting). Unfortunately, while regulatory agencies are being pressed to make rapid decisions about the safety of chemicals, the validity of the assumptions have been (and continue to be) the focus of sharp contention. There is enough doubt lingering around each assumption to cloud the issue when a proposed regulatory action is pending. In short, while the scientific rationale for regulation is often questionable, regulation emanating from it is certain to be highly controversial.

Dioxin is clearly a case in point. Reference to dioxin which is carcinogenic to laboratory animals at high doses is not a critical evaluation of the significance of this information for man; excess mortality from cancer amongst exposed homo sapiens - if it exists - is. Without question, the least controversial basis for regulation when data exists, is human epidemiology. By studying large numbers of individuals who have been exposed to specific environmental factors, it is (often) possible to show the amount of association between that exposure and the onset of cancer or some other disease.

Nonetheless, from a public health perspective and when dealing with the issue of cancer, one cannot wait to count the bodies before acting and we must make use of other (and all) information for the overall protection of man.

Science marches onward and what other information on the subject of dioxin and cancer has emerged in the near past?

Short term bioassays for mutagenicity in bacterial systems are essentially negative. Moreover, recent epidemiological evaluations (albiet fraught with shortcomings) are uniformly negative.

From the negative mutagenic response from bioassays and other information, one might thus call into serious question whether or not the correct model (for dioxin and carcinogenesis) involves no threshold. In fact, a body of scientific evidence now suggests that there indeed may be a dose threshold below which no response is likely. This is a critical departure from the use of untested assumptions now considered by some. In short, if the dioxin induced carcinogenesis (from high dose exposure in animals) is receptor mediated or involves toxicity induced cell proliferation rather than the concept of a model based on the thesis that cancer is caused by the mutation of cellular DNA, then clearly the extrapolation of information to man via a linear non threshold model for carcinogenic action is inappropriate and would not adequately describe the cancer causing potential for man. In addition to considering the adequacy of a non threshold model, it is likewise implicit that dioxin would likely act as a promoter of cancer instead of acting as a direct carcinogen; i.e. an initiator of cancer. These are clearly the most critical bits of new information to be considered by those making policy for protecting the general health of the population.

In addition to the most up-to-date scientific information relating to basic mechanisms of disease being considered by regulators, it is indeed necessary to also consider site specific variances associated with other important elements which are involved in risk assessment. For example (for dioxin), within defined geopolitical limits, water intake as well as fish consumption by the population -- with all of the minute details associated with both -- such as percent fatty tissue by fish variety, etc. must be considered in the development of a sensible standard.

While the absolute "true" and "correct" answer relating to the carcinogenic risk to man from dioxin exposure will likely remain a mystery for some time to come, our estimates for human protection will have to suffice. However, it is now clear that our estimates now need to consider all available relevant information -- scientific and otherwise -- and we need not totally rely only on information which was developed during the infancy of the issue on dioxin.

Rather than develop an entire series of magic numbers based on various and sundry models and criteria, all of which then become subject to administrative and political debate, it seems a more reasonable procedure to satisfy ourselves regarding the

scientific under-pinning involved in risk assessment -- selecting the model and factors which are most nearly scientifically valid -- and then proceeding with our derivations of risk.

As mentioned earlier, perhaps the most critical factor to now consider relates to "what is the appropriate model"? Regarding dioxin, the use of a threshold model for disease (cancer) is clearly the prevailing scientific trend -- which supports a water standard for dioxin which is orders of magnitude higher than the EPA recommendation of 0.013 ppq. We must thus move forward using the "best" data available to provide adequate protection to the population.



VIRGINIA POWER

June 13, 1991

Dr. David Samuels
Chairman
West Virginia State Water Resources Board
1206 Greenbrier Street
Charleston, West Virginia 25311

Dear Dr. Samuels:

Virginia Power supported the proposals for Revision of Title 46 of the Code of State Regulations as submitted by the West Virginia Chamber of Commerce, February 22, 1991. However, we have noted that several issues were not addressed by the Board during its May 17, 1991, revision of the West Virginia Water Quality Standards. Virginia Power operates the Mount Storm Power Station in Grant County under the terms and conditions of an NPDES Permit and we are therefore concerned about these issues. Specifically, we feel that the Board still needs to address the following:

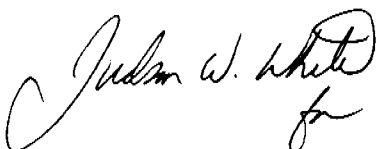
1. Separate mixing zones for thermal and non-thermal pollutants.
2. No-net addition exemption to provide relief for discharges to which no pollutants have been added.
3. Specific exemptions from those numeric standards which are below approved analytical detection limits.
4. Consideration of analytical variability through use of practical quantitation limits (PQLs).
5. Zones of initial dilution.
6. Total recoverable versus dissolved method of measuring metals in water.
7. Instream biomonitoring as a method to verify lack of environmental impact. Ohio uses the results of such studies in-lieu of whole effluent toxicity tests.
8. Separate selenium criteria for lake and stream systems.

Dr. Samuels
June 13, 1991
Page 2

We commend the Board for the improvements they have proposed for the Water Quality Standards but we also want to urge the Board to reconsider the remaining issues raised by the West Virginia Chamber of Commerce. These issues are important to West Virginia industry and we would hope that appropriate changes could be made to the standards before the revisions become final.

We would be happy to answer any questions you may have or provide additional information on the above issues.

Sincerely,

A handwritten signature in cursive script, appearing to read "B. M. Marshall".

B. M. Marshall
Manager
Water Quality

TWL/jmh

Mark J. Reasor, Ph.D.

1153 Cambridge Avenue
Morgantown, WV 26505

(304) 293-2418 (office)
(304) 598-0227 (home)
(304) 293-6854 (FAX)

June 20, 1991

Mrs. Frances E. Hunter
Executive Secretary
West Virginia Water Resources Board
1260 Greenbrier St.
Charleston, WV 25311

Dear Mrs. Hunter:

Enclosed please find comments that I am submitting to the
West Virginia Water Resources Board for their consideration
in setting a water quality standard for dioxin.

Sincerely,



Mark J. Reasor, Ph.D.

Mark J. Reasor, Ph.D.

1153 Cambridge Avenue
Morgantown, WV 26505

(304) 293-2418 (office)
(304) 598-0227 (home)
(304) 293-6854 (FAX)

HOW DOES THE CANCER RISK ASSESSMENT PROCESS WORK?

I am Professor of Pharmacology and Toxicology at the West Virginia University School of Medicine. I am submitting this statement at the request of the West Virginia Chamber of Commerce and the West Virginia Manufacturers Association to discuss the process by which cancer risk is estimated and water quality standards are set. I would like to state that my comments are made as a consultant to the organizations I mentioned and not as a representative of West Virginia University.

While my training and experience is in the field of toxicology, I have taught and written on the qualitative aspects of risk assessment and have a working understanding of the principles involved. I will not address specifically the quantitative issues associated with establishing a specific cancer risk value or an allowable water level for dioxin (2,3,7,8-TCDD). Rather, I would like to put into context the process as it relates to basic scientific principles.

Risk, in general terms, is the probability or expected frequency of an adverse occurrence. For many situations, the risk is easy to estimate because data exist to allow the assessments to be made. For example, available data allow us to state that the risk of death in an automobile accident is 1 in 4,000, from drowning, is 1 in 30,000, and from being

Page 2

struck by lightening is 1 in 2 million. In each of these examples, the endpoint is clear and the cause is obvious.

For the assessment of cancer risk, the determination of a risk factor is scientifically much more difficult and is usually fraught with numerous uncertainties. By definition cancer risk is the probability, or expected frequency, of the development of excess cancers arising from lifetime exposure to a chemical. Unfortunately, reliable data involving human exposure to chemicals at the low levels encountered in the environment are very rarely available. In the absence of such data, regulators are required to estimate levels of exposure or concentrations of chemicals in air or water that, in their judgement, are protective of human health. To do this, mathematical models are employed, frequently utilizing data from animal studies where exposure levels were much higher than would occur in the human situation. Given that animal data are used, a number of assumptions and generalizations, specific for the chemical and exposure circumstances involved, are then made that incorporate the best estimates available into a working model. Included in the model is extrapolation of high-dose exposures of animals to low-dose exposures of humans. From this mathematical exercise, cancer risk factors, and subsequently, reference ambient concentrations are developed. Many different extrapolation models exist resulting in confusion and uncertainty as to which is the most appropriate.

To illustrate the problem associated with cancer risk assessment, I would now like address the major factors used in the calculations of a water quality standard (or reference ambient concentration [RAC]) for dioxin. As you will see, values for these factors involve one or more assumptions that increase the uncertainty associated with their determination.

a) Cancer potency factor: A cancer potency factor is an indication of a chemical's potential to cause cancer and is derived using animal studies or human epidemiological data when available. It corresponds to the upper 95% confidence limit of the slope of the dose-response curve derived from extrapolating high dose experimental data to more relevant low dose human exposure conditions and extrapolation of animal doses to human doses. The higher the value the more potent the chemical is estimated to be in inducing cancer.

Cancer potency factors are subject to great uncertainty due to first, the adequacy or inadequacy of the cancer data base (human versus animal) and second, to the limited information regarding the mechanism of cancer causation. Using the same animal study, but different assumptions, three federal agencies have estimated three different cancer potencies for dioxin as a result of using three different models.

All of the models used to estimate cancer potency factors make the implicit assumption that animal data are

valid and are quantitative predictors of human risk. For several reasons, there is a lack of agreement in the scientific community as to whether such assumptions are justified (Gori, 1982). For example, animals and humans usually metabolize chemicals differently, and this can have a bearing on the formation and elimination of carcinogenic metabolites. Additionally, the dose of the chemical employed and time frames of exposure are often very different from those experienced by humans.

The cancer potency factor for dioxin was developed based upon studies reported by Kociba et al. (1978) using the rat. The carcinogenic response to chemicals varies among species (i.e. is species-dependent). There is no a priori reason for believing that the rat serves as the best model for humans. This adds a considerable degree of uncertainty as to whether these data are valid for cancer risk assessment for humans.

Criteria used for the presence of cancer have changed since the Kociba study was published. A reevaluation of the histopathology from the Kociba study by the Pathology Working Group using more contemporary criteria is compatible with the determination that dioxin is a less potent rat carcinogen than previously thought. The use of a model incorporating these revised data would result in a lower estimate of cancer potency of dioxin.

An additional consideration in developing a cancer

Page 5

potency factor is the understanding of the mechanism by which dioxin may cause cancer. The EPA has historically utilized the concept that there is no threshold for the induction of cancer, i.e. any exposure, no matter how small, results in an increase risk of cancer. Since EPA set its dioxin standard, recent scientific information on the mechanism by which dioxin acts in the cell is suggestive of a minimum threshold level below which dioxin would not cause cancer. The conclusions of a recent conference of leading scientists in the field (Roberts, 1991) suggest that the conservative model used by EPA may be inappropriate. Therefore, the use of a model incorporating a threshold concept may result in a more realistic estimate of the cancer potency of dioxin.

In evaluating the cancer potency of chemicals, data relevant to human studies should be considered. Such data are obtained through epidemiological studies. The results of such studies are difficult to assess because exposure to the chemical is usually poorly documented. Additionally, exposure to other potentially carcinogenic chemicals throughout the lifetime is difficult to assess and may contribute to an increased cancer rate. A recent review of the epidemiological studies on dioxin carcinogenicity in humans (Bond et al., 1990) report that there is insufficient evidence to conclude that dioxin poses a carcinogenic hazard to humans.

A recent paper by Fingerhut et al. (1991) in the New England Journal of Medicine on cancer mortality in workers exposed to levels of dioxin higher than are present in the ambient environment has been cited as evidence that dioxin is a human carcinogen. While there were small, but statistically significant increases in cancer mortality reported, the authors stated that they could not exclude the possible contribution of factors such as smoking and occupational exposure to other chemicals. Unfortunately this study, which probably defines the upper limit of human exposure, failed to answer the question of whether or not dioxin is a human carcinogen.

- b) Risk level: Realizing the limitations of cancer risk estimates, and in the spirit of conservatism associated with the no-threshold concept of carcinogenesis, the EPA has suggested to the states, that for a number of chemicals, a cancer risk of 10^{-6} is appropriate as protective of human health. This number infers that exposure to a chemical has the statistical risk of resulting in one excess cancer per million people exposed for a lifetime. For other chemicals, the EPA has used a cancer risk of 10^{-5} or 10^{-4} as protective of human health. These figures means that exposure to a chemical has the statistical risk of resulting in one excess cancer per one hundred thousand people or one excess

cancer ten thousand people, respectively, exposed for a lifetime. Given that approximately 25% of all deaths are due to cancer, this risk estimate predicts that exposure to a chemical with a cancer risk of 10^{-5} would increase the death rate by .001 percent. Importantly, this value does not mean that one additional cancer will develop in one hundred thousand people, rather that there is a mathematical probability that this will occur. Such values are considered the upper level of risk and mean that the true risk, which cannot be accurately defined, is not likely to be higher than this and may, in fact, be lower.

- c) Bioaccumulation/bioconcentration factors: These factors represent the extent to which a chemical accumulates in fish tissues resulting in a concentration greater than in the surrounding water. The EPA recommends a bioconcentration factor of 5,000 for dioxin. This is an example of one of the generalizations that impacts upon cancer risk assessment. Different fish will concentrate dioxin to different levels depending upon the level of fat in their tissues. The value chosen should be as site-specific as possible, that is, representative of the fish population of concern. Additionally, the fat content of the portions of the fish eaten by people and the loss of dioxin associated with cooking should also be considered in deriving this figure.

- d) Fish consumption rate: The per capita consumption rate of fish will influence the exposure of a population to dioxin. The EPA uses 6.5 grams per day as its national default value for estimating average U.S. per capita consumption of fish and shellfish from estuarine and fresh waters. Again, this is a generalization which attempts to estimate a specific. Because West Virginia has no estuarine waters and produces few if any shellfish, the figure is probably inappropriately high for per capita consumption of freshwater fish for West Virginia. The value should be adjusted to more closely approximate consumption rates of fish harvested from West Virginia waters, since that is the real issue.
- e) Water intake rate: If the impacted body of water is used as a source of drinking water, exposure to dioxin from this route must be considered. The EPA uses 2 liters of water as the average human water intake across the general population. While this assumption would appear to have less uncertainty associated with it than other factors, an attempt should be made to use a value that is as representative of the population of concern as possible.

The equation that incorporates these factors in the calculation of a reference ambient concentration is presented at the end of this statement.

It should be obvious that cancer risk assessment is not an

Page 9

exact science, but in most situations is used as a substitute for the presence of specific and reliable scientific data. Accordingly, cancer risk values and reference ambient concentrations are usually estimates based upon multiple assumptions which may or may not have relevance to the circumstance of interest. In spite of these uncertainties, cancer risk assessment is a process that is carried out by regulatory agencies with the public's best interests in mind, and is often the only way to address the question of how carcinogenic a chemical may be. We have to live with the process, therefore, it is incumbent upon those involved to be aware of what it entails.

There is nothing concrete about a cancer risk value; EPA has approved changes if scientifically defensible. Generally, such changes involve incorporation of new scientific information that was not available when EPA's criterion was initially established.

In the final analysis, given the scientific uncertainties associated with developing cancer risk estimates, these issues before this Board come down to policy decisions. My suggestion is that you examine the growing body of scientific studies questioning EPA's original assumptions, and base your decisions on reason and not emotion.

Thank you for your attention.

A handwritten signature in cursive script, appearing to read "Hank Pearson".

Equation for the calculation of a reference ambient concentration (RAC) (US EPA 1991):

$$\text{RAC (mg/l)} = \frac{(\text{RL} \times \text{WT})}{\text{ql}^* [\text{WI} + \text{FC} \times \text{L} \times (\text{FM} \times \text{BCF})]}$$

RAC = reference ambient concentration (mg/l)
RL = risk level (10^{-x})
WT = Weight of an average human adult (70 kg)
ql* = carcinogenic potency factor (mg/kg/day)⁻¹
WI = average human adult water intake (2 liters/day)
FC = daily fish consumption (kg fish/day)
L = ratio of lipid fraction of fish tissue consumed to 3 percent
FM = food chain multiplier
BCF = bioconcentration factor (mg toxicant/kg fish divided by mg toxicant/l water) for fish with 3 percent lipid.

References

- Bond, GG, Bodner, KM, and Cook, RR. (1989), Phenoxo Herbicides and Cancer: Insufficient Epidemiologic Evidence for a Causal Relationship. Fundamental and Applied Toxicology 12: 172-188.
- Fingerhut, MA, Halperin, WE, et al. (1991), Cancer Mortality in Workers Exposed to 2,3,7,8,-tetrachlorodibenzo-p-dioxin. New England Journal of Medicine 324: 212-218.
- Gori, GB. (1982), Regulation of Cancer-causing Substances: Utopia or Reality. Chemical and Engineering News Sept. 6, pp. 25-32.
- Kociba, RJ, Keyes, DG, et al. (1978), Results of a Two-Year Chronic Toxicity and Oncogenicity Study of 2,3,7,8-TCDD in Rats. Toxicology and Applied Pharmacology 46: 279-303.
- Roberts, L. (1991), Dioxin Risks Revisited (editorial), Science 251: 624-626.
- US EPA. (1991), Technical Support Document for Water Quality-based Toxics Control. EPA/505/2-90-001, PB91-127415, March.



WEST VIRGINIA COAL ASSOCIATION

June 21, 1991

Dr. David Samuel, Chairman
Water Resources Board
1201 Greenbrier Street
Charleston, West Virginia 25311

Re: Water Quality Standards

Dear Dr. Samuel and Members of the Board:

The West Virginia Coal Association hereby submits the following comments in response to the Board's new draft water quality standards and EPA's proposal that the Board adopt a new definition of "existing uses" in the state anti-degradation policy.

As required by EPA's regulations (40 C.F.R. § 131.12), the regulations promulgated by the Water Resources Board contain an anti-degradation policy which provides that "existing in-stream water uses and the level of water quality necessary to protect the existing uses shall be maintained and protected." 46 C.S.R. 1 § 4.1.A. Prior to 1983, "existing uses" was not defined in the federal or state regulations. EPA subsequently promulgated the following definition:

existing uses are those uses actually attained in the water body on or after November 28, 1975, whether or not they are included in the water quality standards.

40 C.F.R. § 131.3(e) (1984). EPA now, apparently for the first time, seeks to require the Board to include the same definition in the state regulations. "Existing uses" is not currently defined in the state regulations.

Adoption of EPA's definition to define "existing" uses as those which existed 16 years ago, however, defies common logic and retroactively alters the rules that dischargers have been playing under, with EPA's approval, for over a decade. In

WEST VIRGINIA COAL ASSOCIATION

Dr. David Samuel, Chairman

June 21, 1991

Page 2

many cases, uses existing in 1975 may no longer exist. The sudden protection of uses which existed in the past, but which no longer exist, will penalize facilities which have constructed treatment facilities in reliance upon NPDES permits issued by DNR that protected actual existing uses but which may not have protected "extinct" uses that existed in 1975. A new requirement to protect former uses will in some cases render as obsolete treatment facilities that were constructed in reliance upon an NPDES permit that protected actual existing uses.

At the very least, trying in 1991 to define "existing" uses by reference to those uses which existed in 1975 will undoubtedly provide fertile ground for permit appeals to the Board by those claiming that permit limitations do not protect a use that formerly existed in the receiving stream. Such cases are doomed to degenerate into swearing matches between aging area residents, with varying motivations and recollections as to the former uses of a stream, rather than being susceptible to scientific analyses through stream surveys and biological monitoring.

Furthermore, we do not believe that the proposed definition is mandated by the Clean Water Act. The statute itself does not contain an anti-degradation policy and does not expressly protect existing uses. It does, however, set forth the goal of attainment or maintenance of water quality standards which assure the protection of public water supplies and the protection and propagation of a balanced, indigenous population of shellfish, fish and wildlife, and allow recreational activities in and on the water. 33 U.S.C. § 1311(h)(2).

In its efforts to achieve this goal, EPA promulgated an antidegradation policy on November 28, 1975 and required the states to include a consistent policy in their programs. 40 Fed. Reg. 55336 (Nov. 28, 1975). However, while those regulations provided for the protection of existing uses, there was no discussion in the regulations or the preamble regarding a reference date for such uses. As previously indicated, existing uses was not defined by EPA until 1983. Even then, there was no discussion or rationale for this definition offered in the proposed or final rules. See 47 Fed. Reg. 49234 (Oct. 29, 1982); 48 Fed. Reg. 51400 (Dec. 8, 1983). It is our position that the goals of the anti-degradation policy, i.e.,

WEST VIRGINIA COAL ASSOCIATION

Dr. David Samuel, Chairman
June 21, 1991
Page 3

maintaining water quality standards, do not require adoption of this definition, particularly when equitable and practical considerations suggest a more reasonable approach.

In light of the concerns expressed above, we believe that the anti-degradation policy should be applied to protect those uses that are known to exist at the time a permit application is submitted. This will protect truly "existing" uses, but will not penalize facilities which have acted in accordance with past NPDES permits. Such a policy will also limit the scope of disputes over what uses are to be protected to the scientific arena. Alternatively, should a benchmark date be utilized for defining existing uses, we believe that the only appropriate date is 1991, which at least gives the regulated community fair warning of the rules. EPA has approved a flexible approach in allowing limited degradation, based on necessary and justifiable economic and social development, in waters which exceed the levels necessary to achieve the goals of the Clean Water Act. 40 C.F.R. § 131.126(a)(2).

We appreciate this opportunity to comment on your proposed regulations and trust that the Board, as it has in the past, will adopt workable and realistic standards that protect our waters without creating needless grounds for many more permit appeals to the Board.

Sincerely,



GARY G. WHITE
President, West Virginia
Coal Association

GGW:dd



STATE OF WEST VIRGINIA
DEPARTMENT OF COMMERCE, LABOR AND ENVIRONMENTAL RESOURCES
DIVISION OF NATURAL RESOURCES
WATER RESOURCES SECTION

1201 Greenbrier Street
Charleston, West Virginia 25311
Telephone (304)348-2107

GASTON CAPERTON
Governor

J. EDWARD HAMRICK III
Director

June 19, 1991

Dr. David E. Samuel, Ph.D.
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Re: Proposed Water Quality
Standards Revisions

Dear Dr. Samuel:

On behalf of Permits, Water Resources Section, we have reviewed the proposed water quality standards revisions and would like to make a few brief comments regarding them. Our comments are submitted as an enclosure with this letter. We request these comments be made a part of the official record.

Sincerely,

A handwritten signature in cursive script, reading "Jerry L. Ray".

Jerry L. Ray
Assistant Chief, Permits

JLR/gs

Enclosure

Comments on Proposed Revisions
to Title 46 Legislative Rules of the
State Water Resources Board, Series I

Section 5.2.a.4.

The addition of the phrase "nor exhibit acute toxicity to aquatic organisms" is redundant in that 5.2.a.6, states that mixing zones shall not cause or contribute to any of the conditions prohibited in Section 3. Section 3.2.e. states that a condition not allowable in State waters is the presence of wastes contributing to materials in concentrations which are harmful, hazardous or toxic to man, animal or aquatic life.

Therefore, the proposed additional language should be deleted, as it is unnecessary.

Section 6.3.d.

With the proposed language that "stream criteria may not be appropriate for application to wetlands", it is unclear what the Board would intend be considered in the permitting process if an applicant proposes to use a wetland either for construction of wastewater facilities or a discharge.

Such occasion is rare and we have no specific recommendations as to how to consider this. Some guidance from the Board is believed essential.

Section 7.2.a.2.

The references to "Division of Water Resources" and "Department of Natural Resources" should be changed appropriately.

Section 7.2.c.

For clarity, add to the reference in parenthesis to change 7.2. to 7.2.d.

Section 8.2.c.

This subsection introduces a new concept to the water quality standards. Therefore, we suggest this new concept, harmonic mean flow, be defined in Section 2.

We believe that critical design flow should more properly be critical stream flow.

Section 9.4.

The reference to EPA-6001 4-78-013 should properly be EPA-600/4-78-013.

APPENDIX B

We have had at least one water treatment plant permit applicant claim other waters used for public water supplies, but we have not needed to verify it. If the Board has not done so, the latest list from the Health Department should be obtained and used.

APPENDIX E

8.1.

We have no specific recommendation here for an aluminum criteria for warmwater fisheries. However, we did want to bring to the Board's attention that a review of existing Storet water quality data over the last ten years reveals that many streams, including warmwater fisheries, have aluminum values far exceeding the proposed criteria of 748 ug/l, up to 100% of the time. We bring this to your attention so that when we issue certain water quality-based permits (due to aluminum) prohibiting discharges of aluminum in some instances, that you can most likely expect some appeals and water quality revision requests. We have the referenced data for your review, if you would care to see it.

8.31.2

Add units to the "x" axis.



*Natural Resources
Defense Council*

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202 783-7800
Fax 202 783-5917

Dr. Jan Taylor
West Virginia State Water Resources Board
1260 Greenbrier St.
Charleston, WV 25311

June 19, 1991

Dear Dr. Taylor,

The Natural Resources Defense Council (NRDC) has a longstanding interest in the adoption of sound, protective State water quality standards that are solidly based on accurate, up-to-date scientific data. We thus are submitting the enclosed comments for the record on the proposed West Virginia 1.2 parts per quadrillion water quality standard for 2,3,7,8-TCDD (dioxin). The enclosed comments were written for the then-proposed Maryland dioxin water quality standard; since the number being proposed for West Virginia for dioxin is identical, our comments on the scientific and other aspects of the Maryland number will be relevant in the decisionmaking process in West Virginia.

NRDC is opposed to the 1.2 parts per quadrillion water quality standard for dioxin because it is far less protective than EPA's recommended number (0.013 ppq), which is itself far less protective than it should be, given current scientific findings about bioaccumulation, reproductive and other non-cancer health effects, and fish consumption rates. On all three of these aspects, both EPA's recommended dioxin standard, and the paper industry's recommended level of 1.2 ppq, fail to incorporate recent scientific data into the risk assessment calculations. We discuss these and other issues in greater detail in the enclosed comments.

The State of West Virginia, in proposing to adopt a non-recommended dioxin water quality standard, is assuming a heavy burden of proof that it has thoroughly researched and up-dated each and every component in the dioxin risk assessment equation. Under 40 CFR 131.11 (b)(1), States are to establish numerical water quality criteria values based on:

- (i) 304(a) guidance; or
- (ii) 304(a) guidance modified to reflect site-specific conditions; or
- (iii) other scientifically defensible methods.

NRDC to Dr. Jan Taylor
June 19, 1991

pg. 2

These numeric values must protect designated uses, all of which must be consistent with "protection and propagation of fish, shellfish and wildlife and for recreation in and on the water..." (often referred to as the fishable and swimmable waters requirement).

Thus, the West Virginia State Water Resources Board has a duty to ensure that its dioxin water quality standard will be fully based on scientifically-defensible methods, and that it will protect the fisheries of West Virginia rivers, streams, and lakes. The enclosed comments on the 1.2 ppq standard for dioxin, show that it will not protect fish or the people who eat them from potentially harmful effects of dioxin exposure.

Thank you for considering our comments on the proposed West Virginia dioxin water quality standard.

Sincerely,

A handwritten signature in cursive script that reads "Diane M. Cameron".

Diane M. Cameron
Environmental Engineer
Water and Coastal Program

ENVIRONMENTAL DEFENSE FUND

Plantation House
1108 East Main Street
Richmond, VA 23219
(804) 780-1297

June 19, 1991

Dr. Jan Taylor
Technical Advisor
West Virginia State Water Resources Board
1260 Greenbrier Street
Charleston, West Virginia 25311

Dear Dr. Taylor:

I am writing in regard to the West Virginia proposed water quality standard for dioxin. I understand that West Virginia has proposed a numeric standard of 1.2 parts per quadrillion for the protection of human health due to cancer risk from TCDD (dioxin). The Environmental Defense Fund (EDF) is opposed adoption of a water quality standard of 1.2 ppq as not protective. EDF submits the enclosed materials into the official record for consideration by the Board and staff.

EDF has actively sought to protect the nation's waters, living resources and human population from contamination and adverse effects of dioxin (and related compounds). We are opposing water quality standards for dioxin of 1.2 ppq in the states of Maryland and Virginia, and are collaborating with other interested parties to accomplish the same goal in other states, particularly southeastern states.

The enclosed materials were developed to present the scientific, technical and legal arguments against adoption of a 1.2 ppq standard and show why the EPA criterion of 0.013 ppq must be adopted. These issues of stream flow, fish consumption, bioaccumulation factor, risk level, and cancer potency are applicable to the West Virginia case. State-wide fish consumption is the only factor that could be expected to differ, because of a smaller fishery in West Virginia. I must point out, however, that subsistence fishing in the Ohio River MUST be taken into account. Local subsistence fishing results in a significantly higher fish consumption for that sub-population.

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Dr. Jan Taylor
June 19, 1991
Page 2

There are two critically important policy issues presented to the Water Resources Board in considering the water quality standard for TCDD in West Virginia. The first of these is the willingness of the state to literally sell the natural resource base of the state, in the form of aquatic life and all that is dependent thereon, in exchange for possible short term gain. The great temptation to bring jobs and taxes into the state is an empty hope. Economists have demonstrated that it is not cost effective to forego long term benefits from a natural resource in exchange for short term gain- even if there is only a small chance that the long term benefit will be lost.

A case in point was decided in Louisiana a year ago. Dredging of shells in Lake Pontchartrain was halted to prevent low level build-up of sediment in the water that was harming the natural resources of the state. The cost of losing natural resources was greater than the benefit from jobs, royalties and taxes from the dredging.

The other issue in the case of dioxin is the use and misuse of scientific information, especially evident in news reports in recent months. News reports, at least in Virginia and nationally, have carried stories that dioxin may not be as harmful as was once believed, giving scientific papers as the basis. The scientific papers, however, did not address the point regarding the danger of dioxin- they addressed the question of how dioxin acts on living cells. The paper in the New England Journal of Medicine that did report on cancer incidence from dioxin exposure in the work place reported higher, not lower incidence.

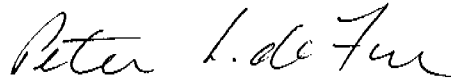
The "science" of dioxin will continue to change over the next decades as experimental results become available. What is known now will be viewed in light of new information, but the decisions will be made this year, using present information. Today we know that TCDD acts on individual cells, probably through a mechanism involving a protein receptor in each cell. This receptor is activated by individual molecules of TCDD and acts to modify cell function, acting through gene expression.

EDF urges the West Virginia Water Resources Board to reject a water quality standard for dioxin of 1.2 ppq and instead enact a numeric standard no greater than 0.013 ppq. The 0.013 ppq standard is supported by EPA documentation and validated, reviewed data. There is substantial data available now that any change in the EPA criterion would result in a lowering of the value to less than 0.013 ppq, based on a ten fold increase in bioaccumulation and a four fold increase in fish consumption.

Dr. Jan Taylor
June 19, 1991
Page 3

EDF appreciates your consideration of these comments on the water quality standard for dioxin in West Virginia. I would be pleased to discuss with you or staff the points raised in my letter or in the documents.

Respectfully submitted,

A handwritten signature in cursive script that reads "Peter L. deFur".

Peter L. deFur, Ph.D.
Staff Scientist
Environmental Defense Fund

enclosures

cc without encl.: Dr. Eli McCoy
E. Erickson, EPA Region III
V. Binetti, EPA Region III

ENVIRONMENTAL DEFENSE FUND

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BEFORE

THE UNITED STATES HOUSE OF REPRESENTATIVES

COMMITTEE ON

PUBLIC WORKS

SUBCOMMITTEE ON WATER RESOURCES

RE:

WATER QUALITY STANDARD FOR DIOXIN

TESTIMONY BY:

Peter L. deFur
Environmental Defense Fund
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Before the United States
House of Representatives
Committee on Public Works

Testimony of
Peter L. deFur, Ph.D.
Scientist
Environmental Defense Fund

Re: Water Quality Standards for Dioxin -HR 2084

Mr. Chairman and members of the Subcommittee, thank you for the opportunity to testify on behalf of the Environmental Defense Fund (EDF) concerning water quality standards for the chemical dioxin (2,3,7,8- tetrachlorodibenzo-p-dioxin) and House Bill number 2084. The Environmental Defense Fund is a national, non-profit environmental organization with over 250,000 members nationally dedicated to the preservation and protection of our natural resources. EDF has worked to resolve environmental problems since 1967 when EDF scientists and lawyers first became involved with the damage caused by the pesticide DDT. My name is Peter L. deFur; I am a scientist in the Virginia office of EDF.

I come before the Subcommittee as a biologist with more than fifteen years of research and teaching experience concerning the effects of toxic substances and environmental physiology of animals that live our nation's rivers, streams, estuaries and coastal waters. I have authored research papers, review papers, textbook chapters; I have reviewed books, scientific papers and grant proposals for state and federal programs and I have spoken before numerous scientific societies. I am presently involved in reviewing and providing scientific and technical comments on water quality standards for several states including Virginia, Maryland and West Virginia.

As an environmental scientist and a member of the scientific community, I offer comments on the scientific factors used to determine water quality standards for dioxin, on the policies regarding the use of science by regulatory agencies dealing with dioxin, and on the perceptions regarding "scientific uncertainty" surrounding dioxin. The science of dioxin has been in the public press for a number of years and has recently resurfaced since the discovery that dioxin occurs in the waste effluent of pulp and paper mills (1). Today, this issue, the regulation of dioxin in our nations waters, is affecting every segment of both the environmental community and the broader society, from laboratory scientists to weekend fisherpersons. I will tell you today that there is more than substantial scientific evidence to regulate dioxin as stringently as possible, with the ultimate goal of removing it entirely from our environment.

The present situation is that there is a 1000 fold variation

in water quality standards for dioxin among the states (California has the most stringent, Virginia and Maryland the most lenient). This situation has come about because EPA has failed to ensure that state agencies set standards that are shown to be protective of stream uses, aquatic life and human health. EPA's deference to the states has resulted in failure by the states to document the standards with basic facts, and failure to obtain new factual information in the entire process. The present bill offers an important step forward in requiring EPA to address this serious national health threat at the national level. The facts before the committee clearly show that the existing system cannot be allowed to continue.

Water Quality Standards for Dioxin

Briefly, the scientific and technical issues in question are those used in the regulatory process and discussed in greater detail elsewhere (2,3). These are derived from the method used to set a numerical water quality standard and how that number is used by staff of state (or federal) regulatory agencies. The formula for calculating a water quality standard (WQS) for dioxin (or any chemical that results in cancer) uses the following:

- the seafood or fish consumption rate
- the extent to which dioxin accumulates in animals
- body weight
- the cancer-causing strength of dioxin
- the risk, in additional cancer deaths/population size

The water drinking rate is also a part of the equation, and in the case of dioxin it is small and not at all controversial.

The first two factors, fish consumption and bioaccumulation factor (BAF) combine to yield the "dose". Both of these affect the predicted "risk" or adverse effect directly. When either seafood consumption or BAF increases, the WQS must decrease in order to maintain risk or adverse effect at a low and constant level.

Body weight is the weight used to determine how much living tissue in one person will be "dosed" with the dioxin. The standard value used here is 70 kilograms (about 158 pounds), the weight of an average adult male. As weight decreases, the WQS must decrease to keep the dose per kilogram constant.

The cancer causing strength (cancer potency) is a number used to predict incidence of cancer in humans based on laboratory studies. At higher values (greater potency) the risk or effect increases and therefore the WQS must decrease to keep the risk or adverse effect constant.

The "risk" in this case specifically, refers to the number

of additional cancer deaths that can be expected from the dioxin dose given under the other conditions. When used to calculate a WQS, "risk" is the number of additional cancer deaths (in deaths / unit population) that must NOT be exceeded. This value is usually 1/1,000,000 and applies to the lifetime risk for the entire population in a state.

Once the WQS number is determined, it is used to set limits on the amount of dioxin that may be discharged by a facility such as a pulp or paper mill. In using the number, the concentration of dioxin on the stream that must not be exceeded, the regulatory staff must determine what is coming out of the effluent pipe and what is already in the stream. The two important numbers are the water flow and the dioxin concentration in the effluent and the stream. The maximum dioxin allowed in the effluent is calculated from the stream flow, background dioxin concentration and effluent flow. If water flow in the river decreases, and the discharge flow remains constant, then either the dioxin concentration in the effluent must decrease, or the amount of dioxin in the river will increase. Obviously, the value (or values) used to determine the river flow will greatly affect the amount of dioxin that can be discharged.

Present Facts

Each of the specific factors used to calculate the WQS has been the subject of discussion and controversy in the WQS setting process in a number of states, particularly in the southeastern states (4). Research by EDF staff, other environmental groups, and state and federal resource agencies has revealed a number of facts regarding the values of numbers used to set the standard. These are not, so far as EDF staff know, questioned or doubted by any of the parties involved in the process.

Seafood consumption in the United States has more than tripled in the past ten years as people have become more health conscious (5). Populations on the coasts and states with numerous lakes and rivers have a much higher fish/seafood consumption (6).

The accumulation of dioxin is many times higher than originally believed (5,000) when EPA first published recommendations for WQS's for dioxin (7). The bioaccumulation factor presently reported by scientists is at least 40,000, and is as high as 150,000 for some fish (8,9,10).

The use of a body size of 70 kg is a bias against persons of lesser body weight, notably children and most women. Both of these groups are given special notice to avoid consuming dioxin contaminated fish, when such fish advisories are issued (11).

Cancer potency has been one of the most controversial factors because EPA, CDC, and FDA each have calculated different cancer potency values, at one time or another. All three numbers are based on the same results from the same investigations. But, using different methods of analysis and criteria, have determined numbers that differ by nearly ten fold (an order of magnitude) (12).

Risk from dioxin has been based on cancer alone and the states have been allowed to use "discretion" in setting appropriate lifetime cancer risks for dioxin. The common options are 1/1,000,000 and 1/100,000, altering the final WQS by tenfold. None of the states has yet conducted a complete "risk assessment" for the state population, especially for sub-populations (with the possible exception of the northwest, where the result was a lower WQS) not even for dioxin alone.

Water flow is not constant in any river over time, and some states have used mean annual river flow as an indicator of long term trends, rather than use measured values obtained each day. But, the arithmetic average flow is not correct to use unless low and high flow occur in equal proportion. EPA has recently recommended using harmonic mean (about 30% of arithmetic), but many states have resisted this guidance.

The "model" to explain the mechanism by which dioxin acts within individual cells has as a central element the involvement of a "receptor" molecule, acting in a way that enables single molecules of dioxin to exert adverse effects.

Data recently published indicate that dioxin is causally related to elevated incidence of several types of cancer in workers exposed to dioxin.

Obviously, there are several technical factors (e.g. bioaccumulation), and policy questions (risk level), as well as basic science issues (the mechanism and mode of action of dioxin) that state and federal agency staff and water resource boards (the decision making bodies) must consider. It is common for staff and board members to claim that these are highly technical and complicated matters, requiring substantial expertise in each area in order to make an informed decision. I disagree.

Every piece of current information offers evidence that greater protection of public health is needed in regulating dioxin (and its related compounds). The actual numbers indicate only how much more stringent the regulations must be in order to

achieve the protection that the population deserves and expects from the regulatory agencies. The vast majority of new data (if not all) indicates that dioxin is of greater concern than previously believed- industry has been successful in using the press to create a different impression.

Scientific Debate

The greatest "debates" regarding the science of dioxin have been over 1) the "scientific" issue of demonstrating adverse effects on the human population, 2) the mechanism of action of dioxin, and 3) how that mechanism translates into regulatory decisions. Until quite recently, opponents of strict dioxin controls made the claim that no data existed to show that dioxin caused serious adverse health effects in humans. With the publication by Fingerhut et al (13) of the results of the NIOSH study, showing increased cancer incidence and deaths in workers exposed to dioxin, this issue should be put to rest. There will undoubtedly be challenges to the Fingerhut et al paper, but the conclusion remains that dioxin is a cancer causing agent in humans.

The mechanism of action of dioxin (14) has been examined for a number of years in laboratory situations because the basic system is common to other biological systems and chemicals. The dioxin molecule enters the cell, and is then believed to bind to a protein, called a receptor, within the cell. In the past few weeks, an article in Science magazine (15) reports that another protein must then bind to this receptor-dioxin combination. The entire association then moves into the nucleus of the cell and changes the normal process of the DNA. This explanation is, of course, greatly simplified, but includes the basic steps- enter the cells, bind to a receptor, adding another protein, move to the nucleus, act on the chromosomes. Nothing in that process dictates or defines the regulatory strategy or approach.

The use of the "receptor binding" mechanism in making regulatory decisions was brought into the public press and into the policy arena when the results of an October 1990 meeting were released. This conference, the Banbury Conference, was held to exchange information among scientists working on the mechanism of toxicity of dioxin. The press carried stories that the conferees had achieved a consensus and agreed that dioxin was less dangerous to people. Subsequent reports indicated that the conference did not accomplish those outcomes, nor even attempt to do so.

There are now two consequences of the Banbury Conference that are causing real problems in the scientific community and in setting policy on dioxin. The erroneous reports about the Banbury Conference cast the entire proceedings in a suspicious light and tend to weaken the credibility of the conference. The

conference was organized and conducted by a highly respected organization that was free from political processes. Previous results from Banbury have always been above reproach- the individual scientists presented and defended their own work in an open exchange free from outside influence. Now the scientific community must face the possibility that sponsors (or sponsor) of this conference may have functioned merely in their own self interest, exploiting the attending scientists.

The other problem is that the "receptor model" was promoted in the press and by others as offering a new approach to regulating dioxin. Furthermore, this new approach supposedly included, by virtue of the receptor system, the use of a "threshold" for dioxin. A threshold would mean that there is some "safe" level, below which no adverse effect would be seen. The receptor system does not imply or demand a threshold. The "receptor model" certainly has no direct bearing on the regulatory process.

Measurement

One of the most public debates over dioxin has probably been the measurement of very low concentrations in the receiving waters. When a WQS is set at 0.013 parts per quadrillion (ppq), how can it possibly be measured for enforcement purposes? Existing methodologies claim that routine measurements are limited to values in the range of greater than 2 ppq. How can a standard be set at levels less than what can be measured? The question itself ignores the real issue: protection of living resources and human health. The standard is not to be set for the convenience of measurement, but for the protection of wildlife, living aquatic resources and human health. The Clean Water Act recognizes that standards must drive technology and not the reverse.

There are, in fact several means of solving this logistic difficulty. First, dioxin can be measured in the effluent, before mixing with river water, where the concentration is much higher. Second, there are at least three alternative means of making such measurements, and none have been refuted; each has been used, in some cases by the paper industry.

The three methods are:

- measure dioxin in the pulping process, where the concentration is much higher than in the river or stream;
- measure total chlorinated organics, of which dioxin is but one, thus increasing the total amount of material being measured;
- use biological measures, such as fish tissue concentrations.

Risk Level

A policy issue that is causing incredible problems in the current process of setting WQS's for dioxin is the discretion that EPA has given to the states in determining what "risk" level is used (12). The position EPA has taken, that states make the "risk" choice as a matter of policy not subject to EPA review, has been one source of variability in health standards (the other has been the "choice" of cancer potency). Those states that are setting lenient standards for dioxin are not conducting any "risk assessment", but are simply using the "choice" as a means of accommodating the wishes of an industry or segments thereof.

A proper "risk assessment" is an evaluation and identification of the potential and actual health hazards and the estimate of the health consequences to the population exposed to those hazards. In the past ten years, our society has begun to use a specialized meaning of that definition: a quantitative estimation of the relationship between toxic substance exposure and cancer induction in a population. Such assessments are not simple and require a substantial database, neither of which is apparent in the WQS setting process used by states setting lenient dioxin standards.

Specific Comments on HR 2084

The Subcommittee is to be commended for considering this bill designed to eliminate the present problems with dioxin regulation. These problems are not limited to dioxin, although this example is by far the most onerous. The general issue is that more than 20 states have yet to comply with the 1987 amendments to the Clean Water Act and set numeric standards for the 126 priority pollutants, of which dioxin is simply the most contentious and the one that acts at the lowest concentrations.

The overall goal of the bill is laudatory, but must not be used as a means to continue discharging dioxin when it can be eliminated. When capital expenditures are great and process changes required, as may often be the case, then use of chlorine bleaching should be eliminated as the only means of eliminating dioxin. This feature is especially effective because there are now alternatives to chlorine bleaching. These alternatives include other chemical bleaching and production and use of unbleached paper products.

There are several major issues that need to be addressed in the bill.

The language concerning "high exposure group" would seem to mean that persons who consume fish from waters receiving dioxin in discharges must receive equal (not equivalent) protection from cancer. The language needs to be clear that no person in the United States may be

exposed to a risk greater than 1/1,000,000. A greater risk level is simply not permissible for isolated populations. In many cases, this would amount to environmental discrimination. Subsistence fishers in the Pacific northwest, Louisiana, native American populations on the East Coast are all examples of sub-populations at greater risk.

Similar chemical compounds need to be addressed by the legislation. Dioxin, 2,3,7,8 TCDD is but one of many related chemicals, including the furans and the dioxins. Most processes that will remove TCDD will also remove all others, but the possibility must not be excluded that this will change and other dioxins and furans will need attention. HR 2084 properly focuses attention on TCDD; it would be an error to bring similar attention to all more than 100 such compounds.

Cancer is not the only adverse effect from dioxin exposure, nor is there but a single cancer type resulting from dioxin exposure. Reproductive effects are demonstrable now in primates, as are adverse effects on immune systems, growth, development, liver function and other body systems in numerous species. The protection needs to include all adverse effects and on more than humans.

Wildlife surveys are an important and valuable component of the bill and EDF has two recommendations for this provision. First, offshore species, such as marine mammals and lobster, need to be included, perhaps specifically. Second, EDF recommends that the bill include provisions to require impact monitoring of the dischargers (both air and water).

The present situation with regard to dioxin waters quality standards has resulted in a 1000 fold range in numeric standards among the states. California and Virginia have standards that differ by approximately 1000 fold. This variation has come about because EPA has permitted the state agencies deference in setting standards for their particular conditions. It is inconceivable that such variability provides equivalent protection for the human populations in each state. In fact, states that have adopted the most lenient dioxin standards have stated that the cancer risk will be 1/100,000, as opposed to the 1/1,000,000 that is used by EPA. When combined with the lower cancer potency factor, low bioaccumulation, and low seafood consumption, the result is a risk that may be less than 1/10,000.

There is no evidence to support a range in health protection of three orders of magnitude among the states. State resource agencies either have not the political will or have not the technical expertise to support and defend protective dioxin water quality standards from the attacks by industry. EPA has not been rigorous in approving dioxin water quality standards that are consistent among the states. EPA has deferred to state actions instead of meeting the duties imposed by 33 USC § 1313 to ensure compliance with the requirements of the Clean Water Act. This bill provides the legislative means to remedy the present inequity among the states.

REFERENCES

1. U.S. Environmental Protection Agency. 1987. The National Dioxin Study. EPA 440/4-87-003. Feb. 1987. Office of Water Regulations and Standards. 104 mill study
2. deFur, P.L. "Water Quality Standards for Dioxin are not Protective of Human Health or the Environment." Paper delivered at the CRC Conference on the Status of the Chesapeake Bay, Baltimore, MD, December 1990.
3. U.S. Environmental Protection Agency. EPA Guidance Document for Dioxin
4. Virginia, Maryland, Tennessee, West Virginia, South Carolina, North Carolina, Alabama, Florida
5. Report of the Virginia Seafood Council on 1990 seafood consumption patterns across the U.S.
6. Seafood Marketing Guide. 1985. Published by Seafood Business Report, Cambridge, Mass.
7. Bioaccumulation is simplistically the concentration in the fish divided by the concentration in the water: Fish/water.
8. Mehrle, P.M., et al. 1988. Toxicity and bioconcentration of 2,3,7,8- tetrachlorodibenzo-p-dioxin and 2,3,7,8- tetrachlorodibenzo-p- furan in rainbow trout. Environ. Toxic. and Chem. 7: 47-62.
9. Muir, D.C.G. et al. 1986. Bioconcentration and disposition of 1,3,6,8 tetrachlorodibenzo-p-dioxin and octachlorodibenzo-p-dioxin by fathead minnows. Environ. Toxicol. and Chem. 5:261-272.
10. Cook, P.M., et al. 1991. Bioaccumulation and toxicity of TCDD and related compounds in aquatic ecosystems. Proceedings of Conference held in Banbury.
11. U.S. Public Health Service. 1988. Toxicological Profile for 2,3,7,8- Tetrachlorodibenzo-p-dioxin. Feb. 22, 1988. Dept Health Human Serv., Atlanta, GA
12. Memorandum from LaJuana Wilcher, January 5, 1990, Regarding State Policies, WQS, and Permit Limitations for 2,3,7,8-TCDD in Surface Waters.
13. Fingerhut, M.A. et al. 1991. Cancer mortality in workers exposed to 2,3,7,8- tetrachlorodibenzo-p-dioxin. New England Journal of Medicine 324:212-218.

WATER QUALITY STANDARDS FOR DIOXIN THREATEN AQUATIC LIFE AND HUMAN HEALTH

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SUMMARY

The 1987 amendments to the Clean Water Act require all states to set numeric standards for priority pollutants established by EPA and approved by Congress. One of these chemicals is dioxin, considered by some to be the most toxic substances ever studied. The water quality standard for dioxin is based on an equation developed by EPA and uses data for exposure, toxicity, bioaccumulation, cancer potency and health risk. Exposure is based on consumption of contaminated seafood, toxicity is derived from laboratory studies, health risk is a policy decision, and cancer potency is a technical decision based on laboratory data. The standard is used at ambient conditions that are measured in the field. The values used for each of these factors will directly affect the final value of the water quality standard, and the ultimate . In both Maryland and Virginia, the values chosen yield a water quality standard that is nearly 100 times higher than that recommended by EPA in their criteria documents. This has come about because the states are relying on the lowest cancer potency value, used by the Centers for Disease Control (CDC), are protecting against a health risk of 1/100,000 deaths not 1/1,000,000, and are not considering other toxic end points or effects on wildlife.

The present standards in Virginia, approved by the State Water Control Board (SWCB), set limits on the maximum concentration of dioxin allowed in ambient water. These standards are supposed to protect human health and aquatic

life. The standard of 1.2 parts per quadrillion (ppq), set by the SWCB, and nearly 100 times higher than the EPA value of 0.013 ppq, does not offer protection. At that concentration, fish will accumulate dioxin to levels of parts per trillion in their tissues, and fish egg and larvae do not develop normally. In laboratory experiments, primates exposed to that level of dioxin experience spontaneous abortions. Furthermore, the standards are determined on the basis of mean annual flow in the receiving stream, although actual flows in this region of the country vary by more than an order of magnitude in the course of the year. The result of that factor alone will raise dioxin concentrations in the waters by the same amount. Clearly, the best scientific data are not being used wisely to make public policy decisions that affect human health and protection of the environment.

INTRODUCTION

The controversy over the toxicity of dioxin has its origins in the accidental exposure of isolated groups of people in their work or homes. Agent Orange use in Vietnam and toxic chemical disposal in Love Canal brought this substance into the news and into the regulatory arena. In the mid 1980's, however, the discovery of dioxin in the effluent of pulp and paper mills brought dioxin much closer to daily lives of the population. By that time, laboratory experiments into the toxicity and mode of action of dioxin had already progressed to the point where dioxin was recognized as having a broad range of cellular level actions at concentrations lower than previously known in other chemicals (Silbergeld and Gasiewicz, 1989). The mode of action of dioxin was investigated at the cellular and molecular level because the results offered insight into the regulation of fundamental cellular processes. Now, the mechanism of action, based on a cellular receptor, offers an explanation why the toxicity of dioxin is so great and the effects so diverse.

Dioxin is a general term that refers to an entire group of compounds, the dioxins, that are formed from two benzene rings (Swanson, 1988). The specific compound that has received so much attention is one with four chlorine atoms substituted. The complete chemical name is 2,3,7,8 tetrachlorodibenzo-para-dioxin, also known as TCDD and commonly called dioxin. The process that forms TCDD requires high temperatures and the organic precursors, specifically the benzene ring structures. The same process forms other chlorine substitutions, such as penta-, hexa-, octa- forms of dioxin, and other benzene structures, especially furans such as tetrachlorodibenzo-p-furan or TCDF. In cases where these compounds are formed as by-products or contaminants, numerous chlorine substitution products may result, and more than a hundred TCDD's and TCDF's can contaminate the final product.

The scientific issues over water quality standards for TCDD center on the confidence in the numeric values for cancer potency, toxicity and bioaccumulation (or bioconcentration) in the water, and subsequently in humans. Much of the data has been obtained in the laboratory using experimental animals, especially rodents. The applicability of laboratory data to human exposure has been questioned recently, but no alternative to animal testing can presently provide the comparable data. Humans have not and will not be used for such determinations, thus some

extrapolation is required. The cancer potency values used in determining water quality standards for carcinogens (or suspected carcinogens) are supposed to provide that extrapolation. In the case of dioxin, cancer potency values have been determined by three federal agencies: Food and Drug Administration (FDA), CDC, and EPA. There is a 9.5 fold difference in the values, primarily because of the method of classifying the results of carcinogenicity tests.

Data on toxicity and bioaccumulation are also based on laboratory work on animals, but the data are applied to human and non-human animal species, particularly aquatic species. There are also some recent results of field tests and surveys on the extent of tissue contamination in fish and shellfish. Human tissue samples have also provided information on exposure and uptake. In the field (non laboratory) work, the basic limitation is the lack of quantitative information on exposure. The data to derive a standard are, for the most part, from non-human species.

THE WATER QUALITY STANDARD

The equation used for determining the water quality standard to protect human health (not aquatic animals or other wildlife) is:

$$\text{WQS} = \frac{(\text{Risk level}) (\text{Body Weight})}{(\text{Cancer potency})[\text{water intake} + (\text{Fish intake} \times \text{BAF})]}$$

where BAF = bioaccumulation factor, used to indicate uptake and concentration of dioxin in tissues. The critical factors are the risk level, cancer potency, fish consumption and bioaccumulation factor. The other two elements in the equation, body weight and water intake rate, though determinants of the final value, are not controversial from a technical position. The policy aspect of both body weight and water consumption are topics for another discussion. Each one of the four critical factors is discussed individually below. In addition, several factors not in the equation are critical in determining the effect of dioxin on human health and wildlife, and thus need to be addressed. The additional factors are:

- the choice of cancer as the only toxic endpoint used
- lack of consideration of the effects on wildlife
- application of the WQS at mean annual (stream) flow

RISK FACTOR

The level of health risk for dioxin exposure chosen by a state is a matter of policy set by the agency (Wilcher, 1990), in this case the State Water Control Board (SWCB). Each state is responsible for setting the health risk for the citizens. The decision is not a technical one, but is a critical social choice made by the state, in Virginia, the SWCB. The risk level will apply to all citizens throughout the

state, not simply a small or "target" population. The resulting standard will apply to all waters under circumstances the SWCB may not even foresee. And the risk is not one taken on voluntarily, rather it is an involuntary one that is accidental.

The Virginia Health Department has the technical expertise to consider such matters, and has done so for specific cases where there is a health risk. In the case of the water quality standard for dioxin, the Health Department has also reviewed the facts in both specific rivers and for the state. At the state level, the Health Department used a risk of one per million (Buttery, 1989). This risk level is the one used by EPA for carcinogens, and is the one used by other states in setting dioxin standards; Maryland is one of the states using the more lenient risk level of 1/100,000.

CANCER POTENCY

The cancer potency factor in the equation is used to give some numerical weight to the potent carcinogens as well as to build in some protective margins. The three factors used by CDC, FDA and EPA vary because of the method of interpreting the results from the same experiment. The original work by Kociba *et al.* (1976) used rats to determine carcinogenicity. The authors classified the tissues from the experimental animals based on examining slides, and reported a range of abnormalities. The differences in the cancer potencies are based on interpretations of results, arising from correcting for mortality of the rats or not, using body mass or surface area to extrapolate to humans, and use of all abnormalities or only clear cancers (EPA, 1984).

As a result of the different interpretations, there is a 9.5 fold variation in the values; the higher the value, the more potent the carcinogen and the smaller the resulting WQS. CDC uses the lowest value, EPA the highest and FDA has an intermediate one. The technical issues of the advantages and disadvantages of the methods to obtain a result are academic; the consequences are profound. The result must protect living tissues at the cellular level against the known effects.

Silbergeld and Gasiewicz (1989) reviewed the mechanisms of action of TCDD (dioxin) at the cellular level. TCDD first enters the cell, where it binds in the intracellular space with a protein receptor. The receptor, denoted the Ah receptor, has a very high affinity for TCDD, on the order of 10^{-9} molar, undergoes a transformation after binding, and enters the nucleus. In the nucleus, the TCDD-Ah receptor complex acts at the level of DNA transcription, altering the function of both regulatory and structural genes. The results are: 1) an increase cytochrome P-450 and cellular metabolism, 2) alteration of membrane growth factors and thus differentiation, and 3) an increased incidence of mutagenesis.

Immunological effects are observed in several species, and do not act exclusively through the Ah receptor mechanism. Holsapple *et al.* (1986) identified a more direct mechanism of action of TCDD on antibody responses in mice. The non- Ah receptor mechanism was questioned for several years, but has now been

accepted (Safe, 1990). The consequences for regulation are two-fold. First, loss of immune system function has debilitating effects on human populations that may not be manifested immediately. Second, there is another quite distinct toxic end-point that is being totally ignored by using carcinogenicity in setting WQS's.

FISH CONSUMPTION

The mechanism for estimating dose of dioxin is to assume that seafood is the major route, and then estimate seafood consumption (EPA, 1984). Recent analyses (Taylor and Rudo, 1990) confirm that the major route for exposure of humans to dioxin is via seafood, including fish and shellfish from both fresh and salt waters. Earlier estimates of seafood consumption, used in the 1984 calculations of a dioxin WQS, were based on a nationwide survey of eating habits. The survey was actually conducted in 1980 (Seafood Business Report, 1985). Since that study, there have been limited data, and both EPA and the states have relied on the resulting value of 6.5 gm/day. That value translates into an annual consumption of approximately 2.37 kg/year or 0.2 kg/month, slightly less than 0.5 lbs per month.

The most recent data from national and state surveys of seafood consumption (Virginia Marine Products, 1990) indicates that seafood consumption in the nation has increased about three -fold in the past ten years. Previous data (Seafood Business Report, 1985) reported that the southeast coastal region consumed about 33% more than the national average. Neither of these sets of data are factored in to a 6.5 gm/day consumption rate. Clearly, the WQS for dioxin, as for other substances, needs to use the most recent data.

BIOACCUMULATION

EPA (1984) originally used a bioaccumulation factor to quantify both the rate of uptake from the water, and the retention and concentration in the tissues of animals like fish. The value used in the 1984 document was 5,000, based primarily on the ratio of ambient to tissue concentration. Since that time, work at independent as well as EPA labs has revealed two important errors in the original approach. First, the magnitude of the value is in error by as much as several orders of magnitude. Second, the reliance on the ratio between ambient concentration and tissue concentration oversimplifies the real nature of the uptake and concentration process.

Mehrle *et al.* (1988) and Muir and Yarechewski (1988) each reported bioaccumulation factors 5 to 10 times higher than the one used by EPA. The lowest, in fact, was 26,000 for rainbow trout under timed experiments. Mehrle *et al.* (1988) continued the exposure for 28 days, and no steady-state was reached, indicating even greater rates than the 39,000 measured. Similarly, Pruel *et al.* (1990) measured uptake of dioxin from sediments for more than 120 days in several invertebrates. Recent data from the EPA research lab in Duluth (Cook, 1990) indicates that not only is uptake greater, but a major route is from sediments into

bottom feeding and dwelling fish. Clearly, the available data agree that the level of bioaccumulation is substantially greater than previously believed.

TOXIC END POINT

The use of cancer as the end point for the human health effects of dioxin makes the assumption that carcinogenicity is the most sensitive (EPA, 1984). In some cases, the data may support such an assumption, but not in the case of dioxin. EPA (1984) even summarized the data on reproductive toxicity in the form of fetal abnormalities and spontaneous abortions. The research includes not only rodent work, but also work on primates. When corrected for dose and body mass, spontaneous abortions were induced at levels just greater than 3 ppq (by mass). That work was more comparable than others, because monkeys were given dioxin at low doses in the food for short periods during gestation.

Carcinogenicity has been used as the toxic end-point by EPA because that point has proven to be the most sensitive, especially over the long term-compared to short term exposure (EPA, 1984). That assumption must be assessed for each application, and in this case the assumption is false. If, as is the case here, other effects actually are noted at lower concentrations during the short term, then that end point needs to be added in to the risk assessment.

WILDLIFE EFFECTS

The WQS for dioxin in both Bay states is only for human health; no attempt was made to protect fish or other species. There is an extensive body of information on the toxicity of dioxin to fish, shellfish, and avian species. Rainbow trout, yellow perch, crabs clams and terns both accumulate dioxin and suffer mortality when exposed. Uptake occurs directly from the water, via the sediments, and from eating contaminated animals. At levels of ppb, dioxin is acutely toxic in short periods, and at ppt over somewhat longer, >4-5 days. But, at levels as low as ppq, mortalities are significant and occur for days (Mehrlle *et al.*, 1988).

The research on uptake and accumulation indicates that the long term effects have been greatly underestimated. Uptake of dioxin from sediments is the primary route (Taylor and Rudo, 1990) and from dioxin that has persisted in sediments for many years (Cristini, 1988; Belton, 1985). These results indicate that long term low level bioaccumulation of dioxin in aquatic animals such as fish, clams and crabs can be delayed for months, or even perhaps years, until dioxin concentrations increase to the point of causing tissue damage, molting and growth abnormalities or reproductive failures.

STREAM FLOW

The numeric WQS is the upper limit for the in-stream concentration after the discharge has mixed in the stream. When the WQS is set, the flow conditions are described for calculating the final concentration; in both Bay states, the regulations

use mean annual flow. Data from the U.S. Geological Survey (USGS, 1990) indicate that flow in the mid-Atlantic states is dependent on rain fall and thus varies seasonally by an order of magnitude or more. In fact, frequency distributions of monthly and daily flows indicate a skewed distribution to the data, with >60 % of the flow values falling below mean annual flow.

The result of this distribution of flows is that the concentration of dioxin will increase in direct proportion to the reduction in flow. In some rivers, the increase will be an order of magnitude. This increase takes place in the summer and early fall, July - September, but could be in January in some of the inland rivers. The higher concentration in the warm season will not only increase the uptake rate, but also increase the effect on living resources.

CONCLUSIONS

There are some factors in the setting of the standard that require policy decisions and cannot be analyzed scientifically; these are not treated here. The scientific and technical issues indicate that extreme caution is, indeed, justified because of what is known about the mechanism of action. Dioxin acts as do many hormones, and individual dioxin molecules can exert an influence over cell processes. The level of action (at the DNA), and the high affinity of the molecules provides sufficient evidence for this concern. The most recent data on toxicity and effects on human and non-human species support that level of caution. Unfortunately, the regulatory agencies do not appreciate the molecular level of action, and have not applied a sufficient level of conservatism. The regulatory agencies are conducting an experiment on dioxin exposure of living resources using the Chesapeake Bay ecosystem as the laboratory.

LITERATURE CITED

- Belton, T.J., R. Hazen, B.E. Ruppel, K. Lockwood, R. Mueller, E. Stevenson and J.J. Post. 1985. A study of dioxin contamination in select finfish, crustaceans and sediments of New Jersey waterways. NJDEP, Off. Sci. Res., Trenton, N.J. 102 pp.
- Buttery, C.M.G. 1989. Letter from Virginia Department of Health to State Water Control Board director, R.N. Burton regarding dioxin water quality standard, Dec. 5, 1989.
- Cook, P.M. 1990. Alternatives for predicting TCDD bioaccumulation: the Lake Ontario experience. Paper presented at National Conference on Dioxins and PCB's, May 10-11, 1990, Embassy Row Hotel, Washington, DC.
- Cristini, A. 1988. the distribution of dioxins in the tissues of juvenile blue crabs. Toxicol. abs.

- EPA. 1984. Ambient Water Quality Criteria for 2,3,7,8- Tetrachlorodibenzo-p-dioxin.
Office of Water, Regulations and Standards, EPA 440/5-84-007.
- Holsapple, M.P., R.K. Dooley, P.J. McNerny and J.A. McCay. 1986. Direct suppression of antibody responses by chlorinated dibenzodioxins in cultured spleen cells from (C57BL/6 x C3H)F₁ and DBA/2 mice. *Immunopharmacol.* 12: 175- 186.
- Kociba, R.J., P.A. Keeler, C.N. Park, and P.J. Gehring. 1976. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD): results of a 13 week oral toxicity study in rats. *Toxicol. Appl. Pharmacol.* 35: 553- 574.
- Mehrle, P.M., D.M. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. DeGraeve, J.J. Coyle and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-Tetrachlorodibenzodioxin and 2,3,7,8-Tetrachlorodibenzofuran in rainbow trout. *Environ. Tox. Chem.* 7: 47-62.
- Muir, D. C.G. and A.L. Yarechewski. 1988. Dietary accumulation of four chlorinated dioxin congeners by rainbow trout and fathead minnows. *Environ. Tox. Chem.* 7: 227-236.
- Pruell, R.J., N.I. Rubenstein, B.K. Taplin, J.A. Livolsi and C.B. Norwood. 1990. 2,3,7,8-TCDD, and 2,3,7,8- TCDF and PCB's in Marine Sediments and Biota: Laboratory and Field Studies. Final Report of USEPA, Narragansett Lab to US Corps of Engineers New York District.
- Safe, S. 1990. Health effects of dioxins and related compounds. *Am. Assoc. Advan. Sci. Ann. Meeting*, New Orleans, LA, 15-20 Feb 1990.
- Seafood Business Report. 1985. The Seafood Marketing Guide. Journal Publications, Cambridge MA. 71 pp.
- Silbergeld, E.K. and T.A. Gasiewicz. 1989. Dioxins and the Ah receptor. *Am. J. Industrial Med.* 16: 455-474.
- Swanson, S.E. 1988. Dioxins in the Bleach Plant. Institute of Environmental Chemistry, Univ. of Umea, Umea, Sweden. 401pp.
- Taylor, T, and K.M. Rudo. 1990. Risk Assessment of Dioxin Contaminated Fish. *Environ. Epidemiol., Div. Epidemiol., Dept. Env. Health Nat. Res.*, Raleigh, NC. 27pp.
- Virginia Marine Products Board. 1990. "1989-1990 The Virginia Seafood Industry Fact Sheet. Unpublished data. Marine Products Board, Newport News, VA

Wilcher, L.S. 1990. Letter from EPA Assistant Administrator, L.S. Wilcher, to Water Management Division Directors, regarding water quality standards for dioxin; Jan. 5, 1990.

DIOXIN CRITERIA ADOPTION BY OTHER STATES

EDF POSITION

The dioxin standard will be based on water quality considerations, which will vary for each state; thus, final criteria will be different for some states. While it may be useful to know what other states are doing, what other states adopt is not determinative for Virginia since Virginia's standard must protect Virginia waters, regardless of the impact of other state actions. Presently, dioxin standard adoption by other states is in flux. States without paper mills have generally adopted the EPA criteria; some states without mills have not adopted any dioxin standard. States with mills have adopted or proposed numbers ranging from below the EPA criteria to as high as 7.2 ppq. Only one state, New York, adopted and got approval for a standard as high as 1 ppq. Four states have adopted or propose numbers more stringent than the EPA criteria, 19 states and 3 territories have adopted or will adopt the EPA criteria, 6 states have adopted or will adopt in the 0.x range; and 13 states, the District and two territories have not proposed any standard (most have no paper mills). Only 4 states: Maryland, Georgia, Alabama and Tennessee now propose a 1.0 ppq or higher criteria (none have been approved or denied at this time). EDF believes that other state actions offer mixed guidance at this time, and Virginia should not be guided by the unsettled positions that exist today. To the extent that Virginia does wish to consider other state standard proposals, the weight of the adoptions and proposals to date are heavily skewed to values at or near the EPA criteria.

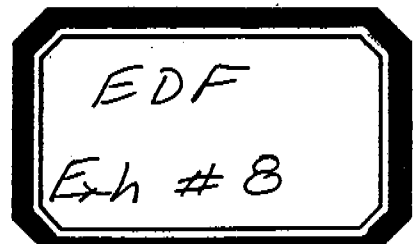
SUMMARY

In order to get the latest possible information on other state activities for dioxin standard adoption, we conducted a phone survey of states and EPA regional offices during the week of March 5, 1990. EPA also now maintains their own record of dioxin actions in the states (Contact David Moon, 202-475-7328). Because the states are in the development process, like Virginia, almost every state situation needs some comment. The information gained is reported below:

EPA Region 1

Contact EPA: Bill Butler, 617-565-3538; David Cochran, 617-565-3505

In Region 1 only two states have paper mills, Maine and New Hampshire; Maine has 6 mills, New Hampshire 1. Both states are non-delegated permit states and EPA will issue the NPDES permit. EPA plans to issue the EPA criteria at 10^{-6} for all six Maine mills, as Maine has adopted the EPA criteria number as its state standard.



New Hampshire officials could not be reached (George Berlandi, 603-271-2983). According to EPA, New Hampshire is considering a 10^{-5} risk factor but has not yet acted on it. However, EPA noted that the New Hampshire plant is located above, and flows into Maine waters. Thus, even if New Hampshire adopts a higher dioxin level, and even if approved by EPA, the paper mill discharge limit must still meet Maine's dioxin standard at the state line. This may result in EPA requiring the New Hampshire mill to achieve the same number as Maine's plants.

EPA Region 2

Contact EPA: Keith Tingberg, 212-264-9894

New York adopted a 1.0 ppq dioxin standard in 1985, and is enforcing it against the only mill in New York. The Standard uses a 10^{-6} risk and a BCF of 10,000, based on a 1981 State Health Department study. EPA approved the standard after its adoption in 1985 and there seems to be no interest by EPA in revisiting the standard at this time. New York thus stands as the only state to have adopted a dioxin standard, with EPA approval, at the 1 ppq level. The standard was, however, partially based on a limit of detection, which traditionally has not been acceptable to EPA.

New Jersey, Puerto Rico and the Virgin Islands do not propose to adopt a dioxin standard at this time.

EPA Region 3

Contact EPA: Randy Waite, 215-597-3425

West Virginia and the District of Columbia have no paper mills and have not adopted any dioxin standard. Pennsylvania has adopted the EPA criteria, rounded off to 0.01 ppq. Delaware has adopted three levels of dioxin standards: freshwater, 0.016; freshwater for fish consumption, 0.017 (uses 5.2 gm/day); marine fish consumption, 0.0024 (uses 37 gm/day). Maryland has a proposed standard of 1.2 ppq.

EPA Region 4

Contact EPA: Larinda Gronner, 404-347-2126

South Carolina has not yet proposed any standard. Florida and Kentucky have proposed the EPA criteria and risk level, while Mississippi has proposed the EPA criteria at 10^{-5} (0.13-ppq). North Carolina has adopted the EPA criteria but has agreed to review it in the present triennial review. Tennessee has proposed 1 ppq; Alabama has adopted a 1 ppq standard but has not yet submitted it to EPA for review. Georgia adopted a 7.2 ppq by emergency order and will hold hearings to confirm or amend during the week of March 12, 1990. The Georgia standard is the highest to be adopted to date and is under intense fire: NRDC has petitioned for disapproval. EPA is non-committal until

after the hearings but is not believed to support the standard. The justification for 7.2 ppq may not be supportable.

EPA Region 5

Contact EPA: David Moon, 202-475-7328

The states in this region have been very active. Indiana and Ohio adopted standards early in 1990 using the EPA criteria and a 10^{-5} risk level, yielding a 0.13 ppq standard.

Wisconsin adopted a standard on February 1, 1989, with various levels of protection for different kinds of use. The coldwater fishery criteria, based on a 20 gram/day fish consumption, is 0.03 ppq; the other criteria are slightly higher.

Illinois and Michigan both are using a translator mechanism. Illinois uses a 20 gm/day fish consumption; Michigan uses a 6.5 gm/day factor, a 10^{-5} risk and a BAF of 51,000. The result is an Illinois standard predicted to be about the EPA criteria, and a Michigan number at 0.014 ppq.

Minnesota has the most stringent standard of all the states. Using a 30 gm/day fish consumption, BAF's of 336,346 for salmon waters and 84,000 for other waters, the proposed standard is 0.0003 ppq.

EPA Region 6

Contact EPA: David Moon, 202-475-7328

Although this region has a number of mills, the development of dioxin criteria has been slow. New Mexico and Oklahoma have no mills, and have proposed no standard. Texas, Louisiana and Arkansas have 13 mills between them but none of the three states has a firm proposal for dioxin at this time.

EPA Region 7

Contact EPA: David Moon, 202-475-7328

Kansas has proposed the EPA criteria, 0.013 ppq; Missouri adopted the EPA criteria of 0.013 ppq in December, 1987. Iowa has no proposal and no mills. Nebraska adopted 0.13 ppq, using a 10^{-5} risk level in February, 1990; EPA has not approved it yet.

EPA Region 8

Contact EPA: Bill Worthal, 303-293-1586

Three of the six states in Region 8, Wyoming, North Dakota and Utah have not adopted any criteria for dioxin. EPA is of the opinion that all

three states will adopt by reference all the gold book criteria, including dioxin. South Dakota adopted the gold book criteria by reference but failed to make a risk level selection, apparently an oversight. EPA has requested clarification. Colorado has adopted a water consumption based dioxin standard of 0.22 ppq; EPA has informed the State that must also adopt a fish consumption standard. Colorado apparently believes that they have no dioxin in any of their fish and thus need no standard.

Montana is the only state in the Region with a bleach-kraft mill. They have adopted the EPA criteria by reference.

EPA Region 9

Contact EPA: Phil Woods, 415-351-8653

Arizona, with one mill, is currently proposing the 0.013 EPA criteria. California has nine mills and has proposed to adopt a marine and freshwater standard based on food consumption. The ocean fish consumption standard is based on 23 gm/day, yielding a standard of 0.0039 ppq; the freshwater and estuarine standard is based on a 6.5 gm/day consumption factor, yielding a 0.013 ppq standard. Hawaii also has a dual standard: ocean fish at 19.9 gm/day, yielding a standard of 0.005 ppq; and a freshwater value of 0.003 ppq. Both Hawaii standards were adopted in January, 1990. Nevada, which has no mills, originally proposed the EPA criteria, but is not currently pushing adoption.

The remaining three territories, Somoa, Guam and the Mariannas have all adopted or are proposing to adopt the EPA criteria by reference.

EPA Region 10

Contact EPA: Rick Albright, 206-442-8514

This region is loaded with paper mills and is the focus of heavy industry pressure. Nonetheless, every state is using the EPA criteria number. Alaska (2 mills) and Idaho (1 mill) are using the EPA criteria through a narrative standard but apply 0.013 ppq. Oregon, with 3 mills, has adopted a 0.013 ppq standard and has confirmed that it will not change. In fact, Oregon has indicated that if EPA changed the cancer potency factor, Oregon would reevaluate their fish consumption data. Washington State, with 9 mills, also uses a narrative standard with the EPA factors and a 10^{-6} risk level. Washington has been under severe fire from the paper industry which has tried to separate Washington from Oregon but so far to no avail. Washington, like many other states, is concerned only that EPA will hold the line and not create a competitive disadvantage for Washington mills.

RISK LEVEL

EDF POSITION

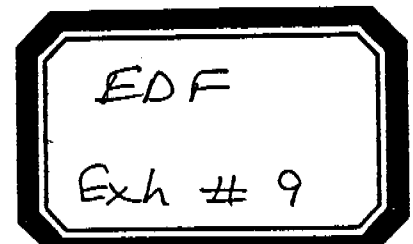
EDF recommends that the Board utilize a risk level of one additional cancer risk per one million persons. This recommendation is based on advice of the Virginia Department of Health, the population affected, the amount of dioxin already in the environment and the assumed selection of a less conservative cancer potency factor than that recommended by EPA. This risk level is also the only appropriate level for application to statewide standards.

SUMMARY

The level of risk for dioxin exposure chosen by the Board is a matter of policy.¹ It is not likely to be an issue reviewed by the courts as the Board is entitled to difference on technical issues affecting public policy.² Because this decision is not reviewable by a court, and involves a critical social choice to be made on behalf of all citizens of the Commonwealth, the level of risk chosen is the most important decision to be made by this Board. Fortunately, the Board is not without guidance in this area. The Virginia Department of Health has recommended a one additional cancer per one million person risk level.³ Since this is primarily a health question, the Board must give great weight to this recommendation.

It is also critical that the Board realize how the Department of Health uses risk levels in its determinations. A case in point is the recent health advisory on dioxin issued by the State Health Department. In determining whether to issue an advisory based on potential fish consumption, the Department considered the amount of fish likely to be consumed, the nature of the fishery and its location.⁴ The Health Department also considered the involuntary versus involuntary risk involved in fishing.⁵ In the present situation, considering the voluntary nature of fishing, especially sportfishing, and the likelihood of consumption of dioxin contaminated fish, the Health Department used a 10^{-5} level of risk to issue its health advisory.^{6,6a} Significantly for Virginians, the Department still felt obliged to warn the public against consumption of fish from several Virginia rivers based on even this risk level.

But there is a world of difference (and exposure) when one looks at an ambient standard designed to protect the general population. Here, involuntary risks are involved, huge and unknown subpopulations (children, the old, the sick, etc.) and a general risk from multiple uses and exposures. In this case, the Health Department falls squarely on a one cancer per million persons risk.⁷ While no set rules determine how State health officials



handle such evaluations, the actions by our Health Department in this case follow general guidelines established by EPA.⁸

Viewing these examples, which are on point here, the industry request for a 10^{-5} risk level is clearly excessive and unprotective of the general population. They are already getting a 10^{-5} risk when the Health Department determines health advisories in specific areas; now they would seek to lower the general risk level for all citizens of Virginia as well.

The effect of the industry proposed risk level is even greater when subpopulations are evaluated. If the general population is protected at a 10^{-5} level, then what about those who do not fit the "general" population statistic? The fisherman, the poor rural population that supplements its diet with fish, the young kids who ask Mom to prepare the catch (often catfish or suckers); what level of risk are these citizens exposed to? Their consumption of fish could easily be 5-10 times the amount of the general population, and the fish that they consume may be 10 times higher in dioxin content than the general fish population. Their level of risk could well be 10^{-4} or less. Is their legitimate use of state waters protected? Can we really say that they knowingly assume the risk? And even if so, should the State impose such risk?

Unfortunately, we are not able to shape this policy decision around a single opportunity for dioxin exposure. Even industry admits that dioxin is also found in air emissions from incinerators, secondary copper smelters, fuel and wood combustion, forest fires and magnesium production.⁹ EPA and numerous other published documents have acknowledged that dioxin contamination also comes from pesticide contamination (recall agent orange?) and other miscellaneous sources. The paper industry has even attempted to minimize their contribution of this highly toxic substance by implying that other sources should be removed first: "Approximately 87 pounds of dioxin currently are estimated to be released in the United States in the course of one year. The entire amount attributable to all of the 104 bleach kraft mills combined (before recent improvements) is approximately 11.4 ounces, which corresponds to one-tenth of an ounce per mill per year."¹⁰

The plain and unfortunate reality is that the citizens of Virginia are already exposed to unknown levels of dioxin. Not only from the industrial sources that the paper industry notes above, but from their own products which they neglect to mention: paper plates, cups, towels, coffee filters and milk cartons.^{11,11a,11b,11c,11d} The level of already existing contamination is best stated by an EDF study which found that human mother's milk contained so much dioxin that FDA could not allow its sale in interstate commerce.^{12,12a,12b} What risk are our new born babies taking in suckling their own mothers?

The level of risk is under fire here not because of appropriate risk assessment, but because of the impact the risk level has on the dioxin numerical standard as derived from the formula used by the staff and EPA. The number can be lowered or raised by one order of magnitude using the risk

factor alone. The Board, however, must look beyond the effect of the risk level on the standard, and determine what is the appropriate policy choice for Virginia. The rest is calculation. If the Board looks first to the impact of its risk level decision, then it is not a policy decision at all but merely a pawn in the standard setting formula.

RISK LEVEL BIBLIOGRAPHY

1. U. S. Environmental Protection Agency. Memorandum from L. S. Wilcher to Water Management Division Directors (Jan. 5, 1990).
2. Va. Code 9-6.14:7 (1989).
3. Virginia Department of Health. Letter to R. N. Burton, Virginia Water Control Board, from C. M. G. Buttery (Dec. 8, 1989).
4. Virginia Department of Health. Memorandum from C. W. Armstrong and K. Wasti to C. M. G. Buttery (Sept. 20, 1989).
5. Dr. C. Armstrong, Virginia State Department of Health. Personal Communication, February 28, 1990.
6. Ibid.
- 6a. Virginia Department of Health. "Public health advisory - dioxin; Jackson and James rivers." News release (Dec. 13, 1989).
7. Dr. C. Armstrong, Virginia State Department of Health. Personal Communication, February 28, 1990.
8. Ibid.
9. National Council of the Paper Industry for Air and Stream Improvement, Inc. Memorandum from I. Gellman to Corporate Correspondents (June 28, 1989).
10. Comments of Weyerhaeuser Company to proposed NPDES permit by North Carolina, as contained in a transmittal from R. C. DeYoung, Ward and Smith, P.A., Counsel, to C. Wakild, North Carolina Department of Environment, Health and Natural Resources (Jan. 2, 1990).
11. U. S. Congress. Office of Technology Assessment. "Technologies for Reducing Dioxin in the Manufacture of Bleached Wood Pulp, OTA-BP-O-54." (May, 1989).
- 11a. Environmental Defense Fund. "Dioxins in Paper." Undated.
- 11b. Travis, C. C., H. A. Hattemer-Frey and E. Silbergeld. "Dioxin, Dioxin Everywhere." Environ. Sci. Technol. 23, 1061 (1989).
- 11c. Beck, H., et al. "Occurrence of PCDD and PCDF in Different Kinds of Paper." Chemosphere, Vol 17, No 1, pp51-57, 1988.

11d. Raloff, J. "Dioxin: Papers Trace." Science News, Vol 135, p 104.
(Feb. 1989)

12. Schecter, A and T.A. Gasiewicz. "Health Hazard Assessment of Chlorinated Dioxins and Dibenzofurans Contained in Human Milk." Chemosphere, Vol. 16, No. 8/9, 2147 (1987).

12a. Environmental Defense Fund. "Summary of Some reported Values of PCDDs/PCDFs in Human Adipose Tissue and Breastmilk Samples." Memo (1988).

12b. Schector, A., et al. "Levels of Polychlorinated Dibenzofurans, Dibenzodioxins, PCBs, DDT and DDE, Hexachlorobenzene, Dieldrin, Hexachlorocyclohexanes and Oxychlorane in Human Breast Milk from the United States, Thailand, Vietnam and Germany." Chemosphere, in Press, 1988.

SEAFOOD CONSUMPTION LEVEL

EDF POSITION

The EPA suggested fish consumption factor for dioxin water quality criteria is not appropriate nor protective of Virginia seafood consumption. Data from both local and national seafood industry indicate that eastern coastal states consume seafood at a rate several times higher than the national average. The Board must consider Virginia seafood consumption patterns and adjust the dioxin criteria accordingly. The correct seafood consumption value for Virginia is 25 grams per person per day for the coastal regions and 6.5 grams per person per day (the EPA criteria) for freshwater fish or, alternatively, 18 grams per person per day statewide.

SUMMARY

Water quality standard calculations for toxic or carcinogenic substances are based on the anticipated or known exposure of the population to that substance. The procedures take into consideration whether the substance occurs in drinking water, food, air or a consumer product. The expected dose for human exposures is then based on the quantitative contact with the contaminated material. Dioxin in pulp mill effluents is released via water¹, and then concentrated in the tissues of animals (and plants) that reside in those waters for all or part of their life history. Finfish, molluscs (clams, oysters, mussels, snails, conch, etc.) and crustaceans (crabs, shrimp, crayfish, etc.) will be exposed to dioxin directly from the water, via the sediments where dioxin may be attached to settled particulate matter, and by consuming contaminated material (animals, plants or sediments).

There is ample scientific data showing that several species of finfish take up and concentrate dioxin in various tissues^{2,2a,2b,3}. Dioxin is known to concentrate in the tissues of fish³, and recent literature indicates that the bioconcentration factor of 5000 used by EPA is far too conservative: actual bioconcentration may be as high as 40,000. Although there is less information on dioxin accumulation in shellfish, we know that soft clams and blue crabs accumulate dioxin^{3,3b,3c}. Thus, the fish and seafood consumption factor for Virginia should include a consumption factor for all these foods.

EPA uses a seafood consumption factor of 6.5 grams/person/day, based on the national average¹. The national average is derived from a survey of nationwide consumption patterns for all seafood; it is not representative of those regions that consume substantially more or less fish or seafood, and does not account for recreational consumption. EPA has recognized that fish/seafood consumption will vary from state to state, and their criteria guidance requires an adjustment where fish/seafood consumption is likely different from the national average. In such cases, the state must consider and include such data in the record⁴. An accurate fish/seafood consumption

factor is very important, as it has a direct effect on the calculated standard.⁵ Other states, using EPA guidance, have adopted fish consumption rates up to 36 grams/person/day (New Jersey, Delaware, Wisconsin, Illinois, Minnesota, California and Hawaii)⁶.

According to data from the Virginia Marine Products Board, the average national seafood consumption in 1988 was 18.7 grams/person/day.⁷ This value, also not corrected for regional or local differences, reflects an increase in seafood consumption that has accompanied national public health concerns and a growing population of Asian people that traditionally consume greater quantities of seafood. Other surveys have confirmed the increased consumption of fish/seafood, especially in coastal areas.^{2b} As recently as 1985, the state of New Jersey selected a 15.7 grams/person/day factor for dioxin risk assessment.^{2b}

The National Seafood Survey, last completed in 1981 and upon which the EPA factor of 6.5 gm/person/day is based, acknowledges that the South Atlantic coast (Virginia, North and South Carolina, Delaware, Georgia and Florida) accounts for 20% of all seafood consumption, yet only 15% of the population resided in that region at the time of the survey.⁸ This ratio of seafood consumption/population ($20/15 = 1.33$) is a good conservative index to correct for the observed regional differences in consumption patterns. Using this ratio, the adjusted seafood consumption for Virginia is 25 grams/person/day, which equates to one eight ounce serving of fish or seafood about every nine days. This value is well within similar values adopted by other fish/seafood consuming states: Delaware (37 gm); Wisconsin (20 gm); Illinois (20 gm); Minnesota (30 gm); California (23 gm); Hawaii (19.9 gm).⁶

There are other inherent assumptions and errors in the calculations of dioxin dose based on fish and seafood consumption that warrant a very conservative approach. One of the major assumptions is that no grossly contaminated seafood will be consumed. That assumption has no validity for dioxin, which contaminates fish/seafood over broad areas not usually considered "polluted".

Another assumption is that the consumption rates are within certain ranges. However, due to dioxin contamination of both fresh and estuarine areas, exposure may vary considerably based on the food consumed. Concentrations of dioxin in blue crabs, for example, indicate an unequal distribution among tissues and, as in other species, the highest concentrations are found in the tissues with the highest fat content.³ In both shellfish and finfish, dioxin is found in all tissues of contaminated animals, albeit at lower levels in skeletal muscle tissue. In spite of health advisories, it is not possible nor feasible to guarantee that contaminated tissue will be removed before consumption, nor is there any way to prevent contamination among tissues. And, as is obvious, many people consume the whole body of some fish/seafood. Finally, there is no convincing scientific evidence that dioxin is lost or broken down during the food preparation process. It is clear then, that any fish/seafood consumption factor, and

standard adopted pursuant thereto, should protect against dioxin levels at the whole body level.

In addition to affecting the calculation of the dioxin standard, seafood consumption factors reflect the importance of the entire seafood industry to the Commonwealth. According to the Virginia Marine Products Board, the dockside value of the state's fishery is worth \$104 million, and the total value to the state is estimated at \$442 million.⁷ Perhaps lesser known, but even more astounding, is the Virginia seafood industries' national market: Virginia is the nation's third largest producer of marine products (only Alaska and Louisiana were greater); Virginia processors handle nearly 25% of the fresh seafood business in the nation's grocery chains, and international markets are increasing.⁷

Fish consumption factors also indirectly impact another major consideration for Virginia's seafood industry: credibility. Should the Board select an arbitrarily low consumption value, the message would be "don't eat Virginia seafood: the assumption is low consumption". An action of this nature could be devastating in the long range to an industry that is currently struggling with nationwide criticism for quality control. The quality control issue is so important to Virginia's seafood industry that the industry includes in its fact sheets information on Virginia's water pollution control program, including the state's latest "finger printing" techniques.⁷

With respect to Virginia's freshwater systems, the correct factor is EPA's current national average number, 6.5 gm/person/day. While a very few states have reduced their freshwater food consumption factor, Virginia's situation does not support such reductions. Virginia's waters are nationally known; the Shenandoah River is part of our national heritage, not to mention an excellent fishery; the James River, above and including Richmond, is nationally known, classed as one of the best smallmouth bass streams on the East Coast and is often the subject of national fishing magazine articles. The national Bassmasters Association will hold its third straight national fishing tournament in Richmond this year, the first time the organization has ever gone to the same place three consecutive years. And, not only is Virginia's freshwater fishery nationally known (which is desirable to retain), Virginia is mostly rural in many of these river areas; it is common knowledge that many of our citizens use freshwater fish to supplement their food needs, an important subpopulation that warrants protection.

The Board has an obligation to protect not only the health of its citizens, but also to preserve the valuable seafood and fishery assets of the state for the future.

Bibliography

1. Environmental Protection Agency. "Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. EPA 440/5-84-007 (1984). [Incorporated by reference]
2. Muir, D.C.G. and A.L. Yarechewski. "Dietary accumulation of four chlorinated dioxin congeners by rainbow trout and fathead minnows." *Environmental Toxicology and Chemistry* 7, 227-236 (1988).
 - 2a. Mehrle, P.M. et al. "Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-Tetrachlorodibenzofuran in rainbow trout." *Environmental Toxicology and Chemistry* 7, 47-62 (1988).
 - 2b. Belton, T.J. et al. "A study of dioxin (2,3,7,8- Tetrachlorodibenzo-p-Dioxin) contamination in select finfish, crustaceans and sediments of New Jersey waterways. Office of Science and Research, N. J. Dept. of Env. Prot., Trenton, N.J. (October 30, 1985).
3. Cristini, A., et al. "The distribution of dioxins in the tissues of juvenile blue crabs; Callinectes sapidus." Abstract from Toxicologist. Undated.
 - 3a. Cooper, K.R. et al. "Histopathologic and chemical analysis for dioxins and dibenzofurans in Mya arenaria in Newark Bay." Abstract from Toxicologist. Undated.
 - 3b. Brown, R.P. et al. "The adsorption and tissue distribution of 3H-TCDD in the soft shell clam, Mya arenaria." Abstract from Toxicologist. Undated.
4. Environmental Protection Agency. Memorandum from L. S. Wilcher to Water Management Division Directors (Jan. 5, 1990). (enclosed as EDF exhibit 9-1).
5. Virginia Department of Health. Letter to R. N. Burton, Virginia Water Control Board, from C. M. Buttery (Dec. 8, 1989). (enclosed as EDF exhibit 9-3).
6. Dioxin Criteria Adoption by Other States. EDF Exhibit No. 8.
7. Virginia Marine Products Board. "1989-1990 The Virginia Seafood Industry Fact Sheet. Unpublished data (February 1990). Virginia Marine Products Board, 97 Main Street, Suite 103, Newport News, Virginia 23601.
8. Seafood Business Report. "The Seafood Marketing Guide." (1985)



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WATER RESOURCES BOARD

July 19, 1990

Edwin B. Erickson
Regional Administrator
U.S. Environmental Protection Agency
Region III
841 Chestnut Building
Philadelphia, PA 19107

Dear Mr. Erickson,

On June 25, 1990, NRDC submitted comments to Region III on the Maryland dioxin water quality standard, requesting EPA disapproval of the 1.2 picograms per liter (or parts per quadrillion (ppq)) standard. (These comments consisted primarily of our comments on Virginia's nearly identical proposal.) We appreciated the opportunity to meet with you on June 27th to discuss in more detail why the Maryland dioxin standard must be disapproved. We wish to supplement the record of comments with this letter and its attachments. Information enclosed describes the ramifications of the Maryland dioxin standard, if approved, and points to the desirability of returning to the EPA recommended dioxin standard of 0.013 ppq.

Maryland Has Not Met the Standards for Approval Under EPA Rules

As we discussed in our meeting, Maryland has not met its burden of demonstrating that the proposed dioxin standard is scientifically defensible. Under 40 CFR § 131.11(b), state criteria must be based on 304(a) guidance, or such guidance modified to reflect site-specific conditions, or other scientifically-defensible methods. Here, Maryland has met none of these tests. The fundamental import of this regulation is that the state bears the burden of proving that any standard that varies from EPA guidance meets the minimum requirements of the Clean Water Act in all respects. Maryland has not even come close to doing so here.

First, Maryland has chosen to weaken the overall dioxin criterion by selectively reviewing the assumptions used by EPA to derive its 304(a) guidance. In essence, Maryland weakened the assumptions that define risk while leaving in place EPA's assumptions regarding exposure. This procedure is not defensible as a matter of good science and good public policy.

Maryland's theory appears to be that, while it modified and purports to defend the risk assumptions in the equation, it has no duty to evaluate the exposure assumptions because it has used

the same assumptions as EPA. As explained in our earlier comments, we disagree with Maryland's conclusions regarding the risk assumptions. But even if these assumptions are accepted, the accompanying suggestion that there is no duty to defend the remainder of the analysis is incorrect and unacceptable. Public health and the environment are not protected by a series of assumptions -- they are protected by the resulting standard.¹ Any lack of conservatism in EPA's exposure assumptions is compensated for, in part, by its use of relatively more conservative assumptions in the risk analysis. Maryland's weakening of the risk assumptions without reviewing the exposure assumptions as well, to defend the overall degree of protection afforded by its proposed standard, is thus inappropriate. More important, having selected a standard that deviates from 304(a) guidance, Maryland is obligated under 40 CFR § 131.11(b)(iii) to demonstrate that its scientific analysis as a whole is defensible. It has made no such showing.

In fact, had Maryland evaluated the exposure assumptions in its analysis, it would have learned that they are not appropriately conservative, especially when coupled with weakened assumptions regarding risk and potency.

A review of recent literature and public statements, both within and without EPA, shows that the EPA exposure factors used by Maryland lack conservatism and a sound scientific basis. These factors include the bioconcentration factor and the fish consumption rate. We believe that substituting more realistic and scientifically-defensible values for these factors will result in a Maryland dioxin standard that is far more protective of human health than the 1.2 ppq standard now under review by Region III, and will meet minimum CWA requirements.

Table 1 (attached) shows the range of values for the bioconcentration factor. We believe that a BCF of at least 50,000 is more scientifically defensible than 5,000, is used or recommended for use by State officials in Michigan and Maine, and is a moderate value compared with the values Dr. Cook obtained for carp, lake trout, and fathead minnow.² Furthermore, the 5,000 BCF value is at the low end of the range of values by

¹ As demonstrated in the next section, the proposed standard as applied will not protect human health.

² Cook, P.M. 1987. Memorandum titled, "2,3,7,8-TCDD in Aquatic Environments" to J. Cummings of the Office of Solid Waste and Emergency Response, from P. Cook, U.S. Environmental Research Lab, Duluth, MN, dated Feb. 4, 1987.

admission of EPA's Region IV Administrator, Greer Tidwell (see Table 1 quote).

As for fish consumption, no data specific to the State of Maryland is available³. As discussed above, we believe that by proposing to weaken its dioxin criterion so severely beyond that recommended by EPA, the State assumed the heavy burden, which it made no attempt to meet, of demonstrating actual fish consumption patterns in Maryland. However, we have attached Table 2 showing available fish consumption data for recreational fishers from around the country.⁴ These data show that sportfishers consume in the range of 10 to 60 grams per day. Furthermore, even EPA's own risk assessment manual for fish contamination calls the 6.5 grams/day a "low" value, and 20 grams/day a "moderate" consumption rate for the purposes of performing a risk assessment for the general population.⁵ Thus, we believe that, at a minimum, 20 grams per day is both a more realistic and a more protective assumption for fish consumption for Maryland than the 6.5 grams per day rate employed by Maryland in its dioxin risk assessment.

Looking beyond Maryland's flawed dioxin exposure assumptions, there are further problems with Maryland's approach to setting a dioxin standard. EPA's 304(a) guidance for dioxin is based on the assumption that human health risks due to carcinogenicity are the limiting factor in developing the dioxin criterion. By substantially weakening this criterion, Maryland calls this assumption into question. For example, adverse effects on fish and wildlife (such as reproductive effects on Maryland's recovering Bald Eagle population based on consumption of dioxin-contaminated fish and subsequent bioaccumulation) may occur at levels between .013 ppq and 1.2 ppq; yet Maryland failed to analyze such effects. Similarly, EPA's Criteria Document and Risk Assessment Documents for dioxin indicate health effects due to reproductive toxicity at approximately 2 ppq based on short-

³ Noreen Eberley, Seafood Marketing Division, Maryland Department of Agriculture, personal communication, 7/10/90.

⁴ Compiled by Dr. Leon Olson for the State of Wisconsin. The State of Wisconsin, for whom this survey was compiled, adopted a fish consumption rate of 20 grams/day based on the survey results.

⁵ EPA/OMEP, September 1989, "Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual." EPA-503/8-89-002. Relevant page is attached.

term exposure.⁶ Maryland's 1.2 ppq standard applied at a less conservative streamflow than 7Q10 (or similar measure of low flow) will not protect against such human health threats based on short-term exposure. Again, Maryland has not met its burden under 40 CFR § 131.11(b) of showing that its alternative to EPA's 304(a) guidance as a whole is scientifically defensible, and meets the minimum requirements of the Clean Water Act.

Maryland's Proposed Standard Will Prevent Attainment of the Clean Water Act's Mandatory Fishable Waters Use

The Clean Water Act mandates that all waters meet a minimum water quality goal "which provides for the protection and propagation of fish, shellfish, and wildlife and provides for recreation in and on the water...." CWA §101(a)(2). This so-called "fishable and swimmable" goal means not only that fish survive, but that they are healthy and safe to eat. Section 303(c)(2)(A) requires that State water quality standards "protect the public health or welfare, enhance the quality of water, and serve the purposes of [the] Act," including the minimum "fishable" use described above. See also 40 CFR, Part 131. Thus, data from EPA's own Bioaccumulation Study show that fish in Maryland rivers downstream of paper mills already have unacceptably high dioxin levels (Table 3 and Figure 1). These dioxin levels, for segments of the Potomac River in particular, are so high that EPA has been pressuring the State of Maryland to issue fish consumption advisories.⁷ Obviously, such consumption advisories demonstrate that the minimum requirements of the Act are not being met, and Maryland's revised water quality standards must be designed to correct this noncompliance.

If the 1.2 ppq Maryland dioxin standard is approved, however, dioxin levels in fish will increase significantly. EPA will be in the awkward (and illegal) position of approving a standard that will create the need for future fish consumption advisories and even fishing bans. EPA may not approve a standard that will prevent the attainment of the mandatory Clean Water Act "fishable waters" use for Maryland waters.

We have performed calculations in order to show the effect of the Maryland 1.2 ppq dioxin standard on the State's fisheries and fish consumers, and to compare it with the EPA-recommended dioxin

⁶ We believe this number itself is too weak for many of the reasons cited in our earlier comments regarding cancer risk. In particular, it is based on faulty assumptions about fish consumption, bioaccumulation, and other factors.

⁷ Baltimore Sun, Thursday, June 21, 1990, attached.

standard of 0.013 ppq (Table 4 and Figure 2). (Attached to Table 4 is a sheet showing how the calculations were performed, and which assumptions were used.) Table 4 shows fish-flesh dioxin levels using 5,000 and 50,000 as bioconcentration factors. Figure 2 shows dioxin levels that will result from implementation of the Maryland 1.2 ppq standard vs. the EPA 0.013 ppq guideline, compared with Region III's internal fish tolerance level for dioxin. Our calculations, using the 50,000 BCF, show that EPA Region III's own internal fish tolerance level of 7 parts-per-trillion (Randy Waite, Region III, personal communication, 7/18/90) -- which corresponds to a highly nonconservative 10^{-4} cancer risk level -- will be exceeded if the Maryland standard is approved. The 1.2 ppq standard, even with the use of the nonconservative 5,000 BCF, would cause an exceedance of the dioxin tolerance level of 0.7 ppt if a 10^{-5} cancer risk goal is applied. Table 4 also shows human-body dioxin accumulation rates for two different fish consumption rates: the "low" estimate of 6.5 gram/day, and the "moderate" estimate of 20 gram/day.

Figures 3 and 4 show predicted human fatty tissue dioxin concentrations for a 70-year (lifetime) exposure, for the 6.5 and 20 grams/day fish consumption rate, respectively. The graphs show the dramatic difference that approval of the 1.2 ppq standard will make over a lifetime of exposure, even for the nonconservative 6.5 gram/day fish consumption rate. Schecter et al. (1987) state that the mean value in the U.S. and Canada, for adipose tissue concentration, is 7 ppt for 2,3,7,8-TCDD in adults⁸. Using the least-conservative exposure assumptions of 6.5 gram/day fish consumption and a 5,000 BCF, the 1.2 ppq dioxin standard, if approved, would result in a lifetime fatty tissue dioxin accumulation over ten times the current U.S. mean. Using the more realistic assumptions of 20 gram/day fish consumption and a 50,000 BCF, the lifetime dioxin accumulation would be 2,184 ppt, or over three hundred times the current U.S. mean. Clearly, such dioxin body-burdens are unacceptable, and EPA should act to avoid this scenario.

Based on the EPA Region III fish tolerance level, then, the Maryland dioxin criterion of 1.2 ppq, if approved, will allow further contamination of Maryland fisheries with dioxin such that future fishing advisories and bans are a virtual certainty. This is true particularly in light of the fact that, by using the 1.2

⁸ Schecter, A.J., Gross, M. and Ryan, J.J. (1987) "Biological markers of exposure to chlorinated dioxins and dibenzofurans in the United States and Vietnam," in Hazardous Materials Disposal: Siting and Management. Edited by Manas Chatterji. Avebury publishers, Aldershot, England.

NRDC Letter to Edwin B. Erickson
Re: The Maryland Dioxin Standard

6

ppq standard, the revised NPDES permit for the treatment plant used by the Westvaco facility will preserve the status quo in terms of Westvaco's allowable dioxin discharges. The Maryland criterion will clearly result in further dioxin bioaccumulation and exceedance of EPA's threshold dioxin level for fish, and thus will prevent the attainment of minimum Clean Water Act goals of fishable waters and protection of human health and welfare.

NRDC requests that EPA disapprove the Maryland dioxin criterion because it will result in a loss of fishery resources for Marylanders. EPA should act to avoid the blatant inconsistency of approving a water quality standard for fisheries that will assuredly cause the loss or restriction of those same fisheries, while at the same time urging Maryland to issue fish consumption advisories for those waters.

Sincerely,

Robert W. Adler *(signature)*

Robert W. Adler
Senior Attorney
Clean Water Program

Diane M. Cameron *(signature)*

Diane M. Cameron
Environmental Engineer
Clean Water Program

Table 1: Range of Values for Bioconcentration/Bioaccumulation of Dioxin in Fish

Value	Comments
5,000	"More recent data indicates that this value is at the low end of the range of uncertainty." (Region IV Administrator Greer Tidwell in letter to Leonard Ledbetter, Director of the Georgia Department of Natural Resources, March 27, 1990)
• 50,000	recommended by Dr. Robert Frakes, State Toxicologist, for use in setting the Maine dioxin water quality criterion
51,000	used by the State of Michigan in setting dioxin water quality criteria
66,000 (carp)	Cook, P.M. 1987. Memorandum titled, "2,3,7,8-TCDD in Aquatic Environments" to J. Cummings of the Office of Solid Waste and Emergency Response, from P. Cook, U.S. EPA Environmental Research Lab, Duluth, MN, dated Feb. 4, 1987.
140,000 (lake trout)	Cook, P.M. and Lodge, K.B. (1989). Chapter 6. Laboratory Study of TCDD Bioaccumulation by Lake Trout from Lake Ontario Sediments, Food Chain and Water. Draft manuscript.
97,000 (fathead minnow)	Cook, 1987 (as cited above).
159,000 (fathead minnow)	" " "

Table 2.

Fish Consumption Rate Literature Survey Compilation by Leon John Olson, Ph.D.

WI DHSS/DOH March 9, 1988

FISH CONSUMPTION RATES (FRESH WATER, COMMERCIAL AND TOTAL)
(grams per day)

	Freshwater or Recreational	Commercial or saltwater	Total
FDA and EPA National Average (from NPD survey, in Kleiman)	6.5	18.7	
USDA, 1965, in Kleiman			9
USDA (1977-78 Survey. 1985) may not include recreational or restaurant fish (Cordle, 1978)			9.5 (15 for ages 23-34)
USDA (1982 Survey) (mean, in Guidance Man and Kleiman) 90% = 96 99% = 221			54.0
USDA (1983, in Kleiman)		6.4 (unfrozen only)	
USDA (1985 Survey) (in guidance manual rept.			21.0
Wisconsin DOH Survey (mean) (1986) 95% = 37.3 (caught) 95% = 63.4 (all meals)	12.3		26.1
Ontario (Cox, 1983 Survey)	21.75	14.40	36.15
Ontario (1984, Kleiman)	13.8		
Ontario (1986, Ontario MOE)	19.4	16.24	35.64
Columbia River, Wash. (Rupp) (1966-67 study) maximum = 90.4	7.8		

Commencement Bay, Tacoma, WA
sport caught only, Nicola,
1987 (salt water)

1 lb. (452 gms)

Great Lakes Fish Consumption 15.7 (90%)
(in Kleiman) 36.8 (99%)

Lake Michigan (1973-74, 47.8 (ave. of high consumers only)
Humphrey, Kleiman, Foran)

New York (in guidance manual) 32.4 (90% of rec. caught consumers)

Seafood consumption Survey, 1.7
1981? (NMFS, cited by Ohio)

Seafood Consumption Survey, East- 9.8 27.9
North Central Region, NMFS, 1973
in Rupp, 1980 (fish eaters only)
in Rupp, 1980)
90% = 32.6
99% = 91.5

NMFS Markets Facts Survey, 1969-70 16.8
(99% = 77, in guidance manual)

NMFS, 1970, recreation only 3.7 - 5.3
caught fish only, in guidance
manual, Kleiman

National Dairy Purchase, 1973-74 14.3
(95% = 41.7, in guidance manual,
Kleiman)

National Dairy Purchase, 1973-74 17.6
99% = 85.4, according to
Tollefson, 1986
(fish eaters only)

Cordle, 1978: same data = 18.7

Kleiman: 14.3 total fish
consumption
95% = 41.7

Kleiman:
consumers of FW trout = 12.3 g/day

Sonstegard Lake Ontario, 1985, 31 - 62 (high of 373)
1987, Fishermen, in guidance
manual and Kleiman, Intensive

lake Ontario fishing

Fisherman in American Samoa ate up to 9 ounces of fish per day for 22 months (Tollefson)

Relevant Studies to WI and Great Lakes, with daily intake averages (grams/day) derived from the above data (note; the NMFS-North Central region data of 1973 is not included because it is very probably outdated for the average fish consumer):

	A	B
WI DOH	12.3 (mean)	12.3
Ontario	21.8 "	21.8
Ontario	13.8 "	13.8
Ontario, 1986	19.4 "	19.4 not averaged in column B)
L. Ontario	31.0 "	31.0
(Sontesgard)		
G.L. Fish Comm	15.7 (90%)	
Lake Michigan	47.8 (high)	
(Humphrey)		
New York	32.4 (90%)	
AVERAGE:	24.97 grams/day	19.73 grams/day

DRSS recommends the use of 20.0 grams per day as the average daily intake of sportcaught fish for WI fish consumers, based on the average of the four studies in column B, above, and the fact that the WI DOH survey was not designed to specifically answer the question of daily intake of sportcaught fish in Wisconsin.



Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish:

A Guidance Manual

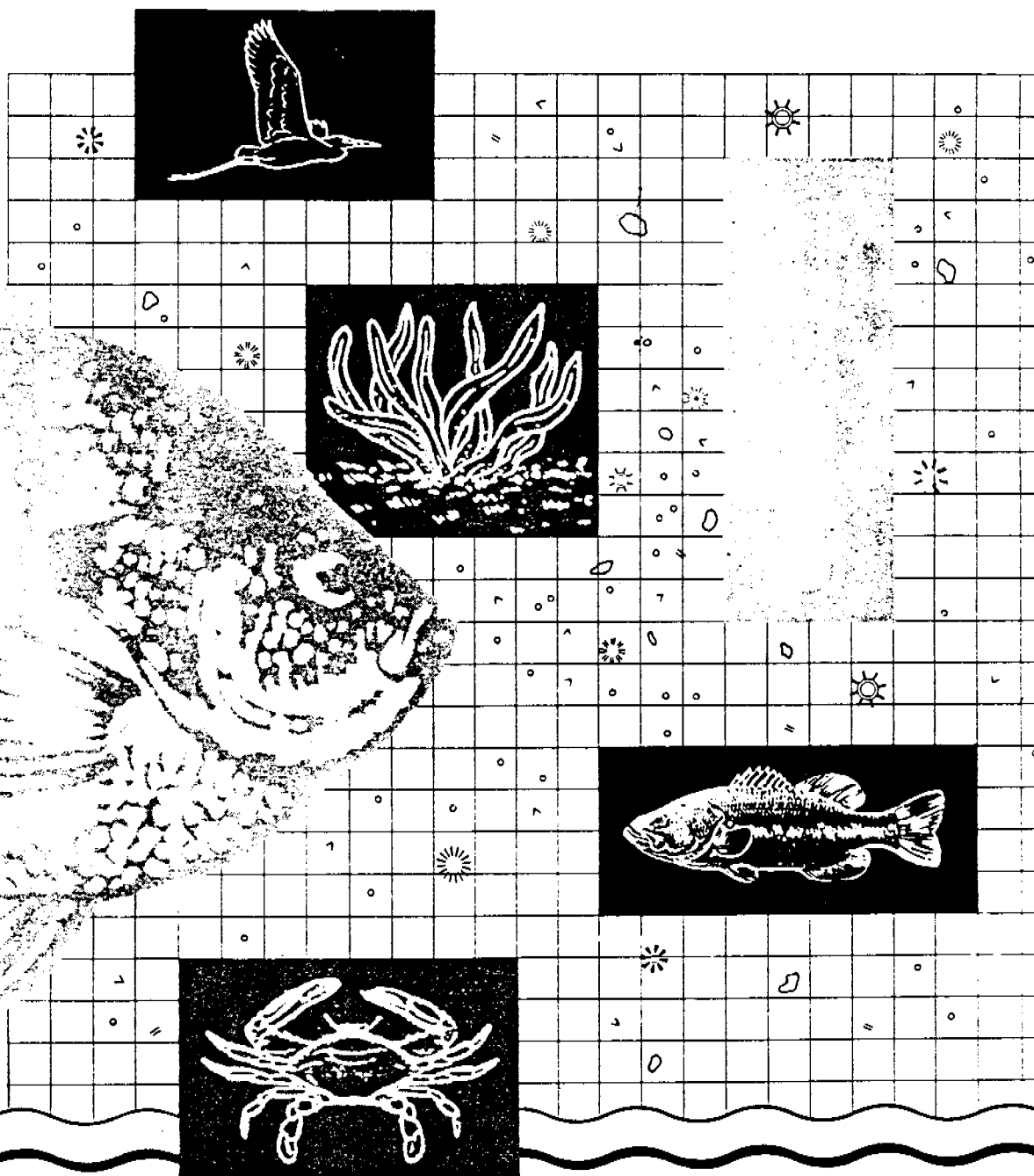


TABLE 7. Summary of Assumptions and Numerical Estimates Used in Risk Assessment Approach

Reference	Assumption/Estimates	Parameter
Worst case for parent compounds. Net effect on risk is uncertain.	No effect on cooking.	Exposure Assessment: Contaminant concentrations in tissues of indicator species
Low, moderate, and high values for U.S. population (see text).	6.5 g/day, 20 g/day, 165 g/day	Average consumption rate ^a
U.S. EPA 1980b; 1986a,b	1.0. Assumes efficiency of absorption of contaminants is same for humans and bioassay animals.	Gastrointestinal absorption coefficient
U.S. EPA 1980b; 1986a,b	70 years	Exposure duration
U.S. EPA 1986a,b	70 kg (= average adult male)	Human body weight
U.S. EPA 1980b, 1986a, 1987a	Linearized Multistage. At risks less than 10 ⁻⁶ : Risk = Exposure x Potency	Risk Characterization: Carcinogenic risk model
U.S. EPA 1987a	Potency factors are based on low-dose extrapolation from animal bioassay data. Upper bound of 95 percent confidence interval on potency is used.	Carcinogenic potency
U.S. EPA 1987a	RfDs for noncarcinogens compared with estimated exposure.	Noncarcinogenic risk

^a Estimates of consumption for local population should be used in place of values shown for U.S. population whenever possible.

Other assumptions, such as general approaches or assumptions underlying models that are commonly used to estimate risk, can be summarized in the text of a risk assessment document. Some additional assumptions involved in applying the risk assessment approach described in this manual include the following:

- Adverse effects in experimental animals are indicative of adverse effects in humans (e.g., lifetime incidence of cancer in humans is the same as that in animals receiving an equivalent dose in units of mg per surface area)
- Dose-response models can be extrapolated beyond the range of experimental observations to yield plausible-upper-bound estimates of risk at low doses
- A threshold dose does not exist for carcinogenesis
- A threshold dose (e.g., NOAEL) exists for noncarcinogenic effects
- The most sensitive animal species is appropriate to represent the response of humans
- Cumulative incidence of cancer increases in proportion to the third power of age (this assumption is used to estimate lifetime

Table 3.

Mean Dioxin Concentrations in Fish Fillets-
Combined EPA and Westvaco 1988 Data Sets

Sample Location	Number of Composites	Dioxin (ppt)		
		Arith Average	Weighted Average	Geometric Mean
Savage River	6	0.57	0.71	0.10
Potomac, Above Savage	5	0.09	0.08	0.09
Potomac, Luke-Keyser	4	7.80	5.29	5.46
Potomac, Cumberland	7	11.59	9.59	5.98
Potomac, Paw Paw	5	4.93	3.69	1.21
Potomac, Hancock	6	0.34	0.35	0.08
Potomac, Point of Rocks	4	0.94	0.81	0.81
Luke-Keyser to Paw Paw	16	8.56	6.85	3.54

From: the draft Support Document
"Health-Risk Assessment for Consumption
of Dioxin-Contaminated Fish From the
Potomac River"

PREPARED BY:

Jeffrey M. Paull, MHYG, MPH

Roger C. Cochran, PhD

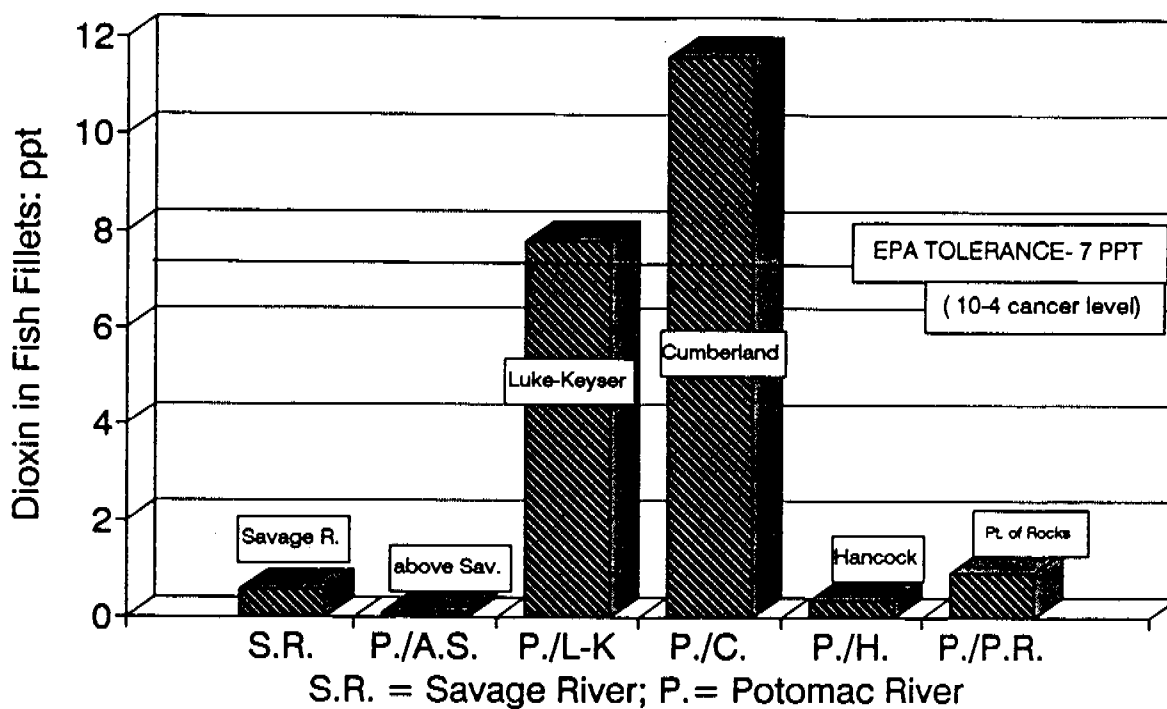
Environmental Toxicology and Risk Assessment Section
Center for Environmental Health

March 31, 1989

Figure 1.

Mean Dioxin Concentrations/Fish Fillets

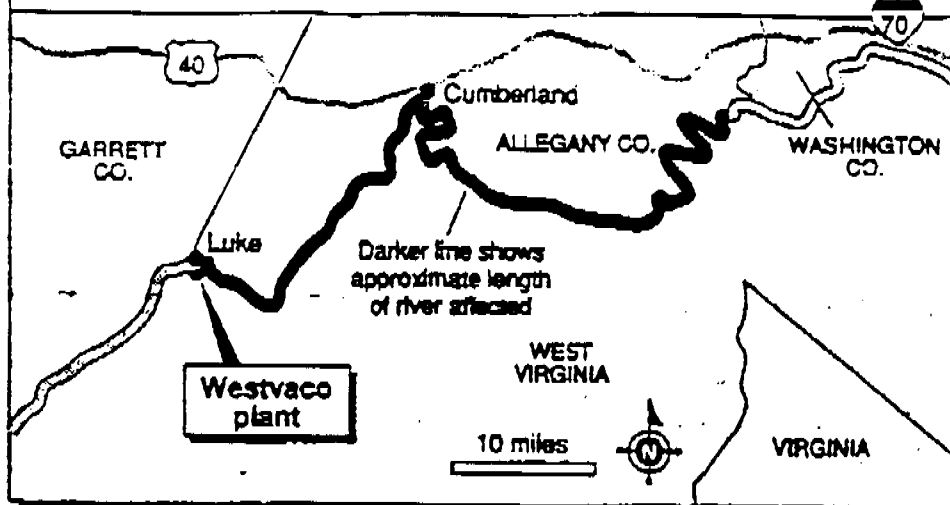
(From Paull and Cochran, 1989)



THURSDAY, JUNE 21, 1990

Warning on eating fish caught in the Potomac River

West Virginia advises residents to limit consumption of fish caught within 70 miles below Westvaco paper mill at Luke. EPA is urging Maryland to issue a similar warning.



Maryland urged to warn of dioxin danger in fish

By Liz Bowle

The Environmental Protection Agency is pressing Maryland to warn residents that eating fish from the northern branch of the Potomac River will increase their risk of getting cancer.

Such a warning would be a major shift in policy for the Maryland Department of the Environment.

For the past year, the department has maintained that dioxin contamination of fish below the Westvaco paper mill at Luke is not great enough to warrant any warnings to the public.

However, the EPA disagrees. In the draft of a letter given to Environment Secretary Martin W. Walsh Jr. about two weeks ago, the federal agency said, EPA is of the opinion that a significant health risk exists

and that a fish consumption advisory is needed."

In the letter, recently obtained by *The Sun*, the EPA advised Mr. Walsh that it would be holding a news conference in July to say that a health risk exists and asked him to issue a public health advisory before that.

"I haven't made a decision yet," Mr. Walsh said in a telephone interview yesterday. "I am going to be putting out an advisory in the next seven to 10 days."

He declined to disclose what the public statement would say.

Edwin Erickson, EPA administrator for the region that includes Maryland, said the letter he "shared" with Mr. Walsh was a working document and was not final. However, he said

See DIOXIN, 2A, Col. 5

will not change.

Maryland's current position on dioxin disregards the advice of its own scientists and EPA estimates of public health risk.

It also runs contrary to the position taken by neighboring West Virginia, which advised residents last summer to limit consumption of fish caught within 70 miles below the paper mill.

The contamination of fish with dioxin — a probable carcinogen — was first discovered a year and a half ago when the EPA tested fish caught below the paper mill for dioxin. The chemical, discharged into the river, is a byproduct of bleaching paper products. Although the Westvaco mill has reduced its dioxin discharges in the past year, scientists say the chemical has accumulated in the bottom mud, where it can contaminate fish for years.

Maryland and the EPA measured dioxin in fish samples from the Potomac in the fall of 1988, then estimated what a person's lifetime risk would be if he consumed fish from different locations in the river.

The analysis indicated that the cancer risk was greater than considered acceptable by most scientific standards. The EPA has estimated that in Paw Paw, W.Va., some 53 miles downstream of the plant, the lifetime risk of getting cancer would be more than one in 10,000.

The lead scientist in the Department of Environment's toxicology division suggested a year ago that the state advise fishermen not to eat fish that feed on the bottom, and that residents shouldn't eat more than seven meals a year of other fish, such as bass and sunfish, caught between the mill and Cumberland, according to documents obtained by a request through the federal Freedom of Information Act.

But top Maryland officials said no such advisory was necessary. Instead, the department issued a statement saying that the fish were safe to eat but recommending that people remove the fatty portions, such as the pancreas, intestines, liver and skin, where dioxin accumulates.

Department of the Environment officials said at the time that the risks from dioxin did not warrant scaring the public with an advisory.

And yesterday, Mr. Walsh pointed to considerable differences of opinion on dioxin between the EPA and the U.S. Food and Drug Administration.

State officials based their conclusion on the FDA's, which in theory would allow higher levels of dioxin in

to revisit their decision
in view of
the high risk.

ROBERT KRAMER
EPA regional official

fish because it assumes that the chemical is less potent and that a person would not make a steady diet of the fish.

Sports fishermen in Western Maryland have said they rarely eat the bass they catch because of the water pollution just below the mill. But they say some local residents do eat the fish, particularly those who are poor and fish for a source of food or who are simply uninformed.

"We want Maryland to revisit their decision in view of the high risk," said Robert Kramer, acting deputy director of the EPA's regional environmental services division.

Mr. Kramer said the EPA turned up the heat on Maryland recently, not because of any new data that have been collected but because the agency realized it needed a national approach to reducing the cancer risks from dioxin.

The EPA has spent several years collecting and analyzing data from 104 paper mills around the country that discharge dioxin. The study found that the most significant risk to the public is from contaminated fish below the plants.

In many cases, states have issued health advisories for waters below the mills. For instance, Virginia has issued advisories below another Westvaco mill that discharges into the James River, and Pennsylvania has issued an advisory for fish caught from Dordorus Creek.

"To be honest, the Maryland situation sort of stuck out as somewhere where we should be a little more forceful," Mr. Kramer said. Since the EPA has no regulatory authority to issue public health advisories, the strongest action it can take is to issue a public statement saying what it believes the risks are.

No commercial fishing is done in contaminated sections of the Potomac, and no fish caught there is sold in stores. The only risk is to those who eat the fish they catch.

Westvaco Corp. has spent \$15 million in the past year to make changes in the bleaching process and reduce the dioxin in its discharge by 96 percent. The plant is in compliance with current water-quality standards.

Procedures Used in Generating Table 4: Fish-Tissue Dioxin Levels and Human Dioxin Accumulation Rates Based on Varying Bioaccumulation and Fish Consumption Assumptions.

1. Fish-Tissue Dioxin Calculations

A. BCF = 5,000

Fish-tissue concentration = 5,000 X dioxin water concentration. For example, for the Maryland 1.2 ppq standard, the fish-tissue concentration is

$$\text{Fish conc.} = 5,000 \times 1.2 \text{ ppq} = 6000 \text{ ppq} = 6 \text{ ppt.}$$

B. BCF = 50,000

The procedure would be the same as performed above, only

$$\text{Fish conc.} = 50,000 \times 1.2 \text{ ppq} = 60,000 \text{ ppq} = 60 \text{ ppt.}$$

2. Human Accumulation Rate for Dioxin

The human accumulation rate depends upon the assumption used for fish consumption.

A. Fish consumption = 6.5 grams/day

human accumulation = 6.5 g/d X fish tissue conc. (pg/g) = picograms per day, divided by human body weight (70 kg).

For the 6 ppt fish tissue concentration from 1.A. above,

$$\text{human accumulation} = 6.5 \text{ g/day} \times 6 \text{ pg/g} = 39 \text{ pg/day divided by } 70 \text{ kg} = 0.56 \text{ pg/kg/day.}$$

B. Fish consumption = 20 grams/day

The procedure would be the same as performed above, except that we use 20 g/d instead of 6.4 g/day.

For example,

$$\text{human accumulation rate} = 20 \text{ g/day} \times 6 \text{ pg/g} = 120 \text{ pg/day divided by } 70 \text{ kg} = 1.7 \text{ pg/kg/day.}$$

Table 4: Fish-Tissue Dioxin Levels, and Human Dioxin Intake Rates Based on Varying Bioaccumulation and Fish Consumption Assumptions.

i. U.S. EPA Recommended Water Quality Criterion for Dioxin:

0.013 parts per quadrillion

		BCF	
		5,000	50,000
Fish Cons. Rate (g/d)	6.5	0.065 pg/g 0.0060 pg/kg/day	0.65 pg/g 0.060 pg/kg/day
	20.0	0.065 pg/g 0.018 pg/kg/day	0.65 pg/g 0.18 pg/kg/day

fish-tissue level / human intake rate

ii. Maryland State Water Quality Criterion for Dioxin

1.2 parts per quadrillion

		BAF/BCF	
		5,000	50,000
Fish Cons. Rate (g/d)	6.5	6 pg/g 0.56 pg/kg/day	60 pg/g 5.6 pg/kg/day
	20.0	6 pg/g 1.7 pg/kg/day	60 pg/g 17.1 pg/kg/day

Figure 4.

70-Year Dioxin Concentration in Human Fatty Tissue

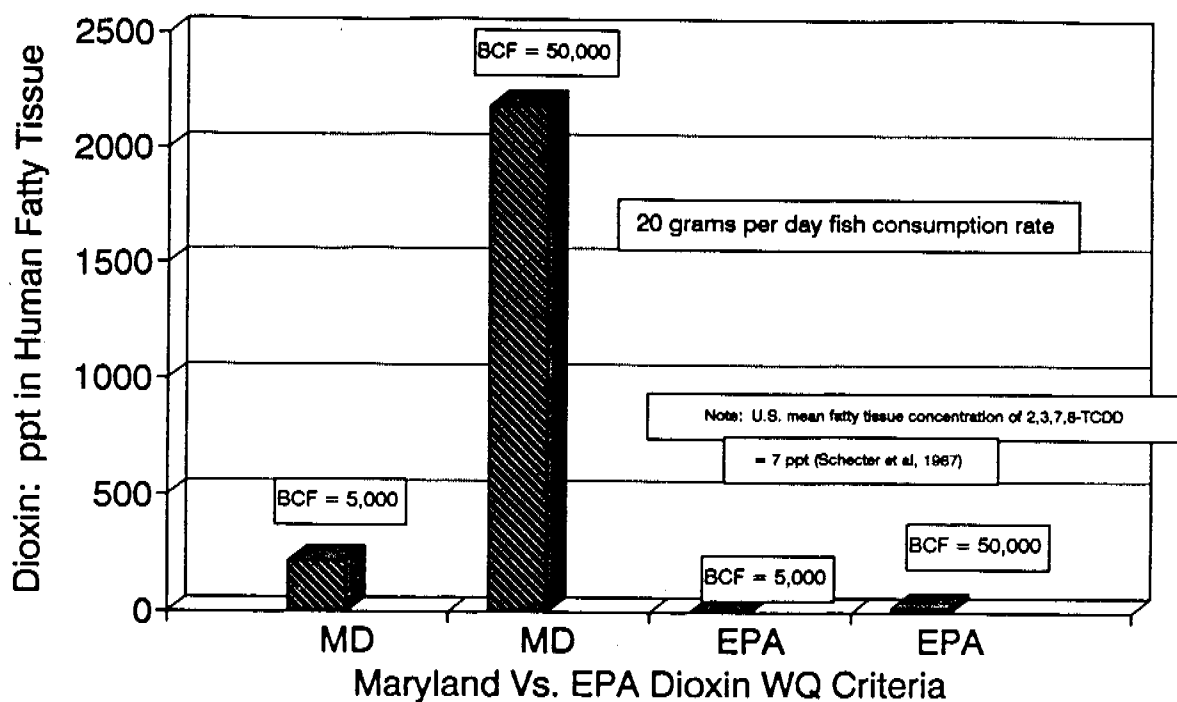


Figure 2.

Dioxin Levels in Maryland Fish

Predicted Levels/ BCF = 5,000 or 50,000

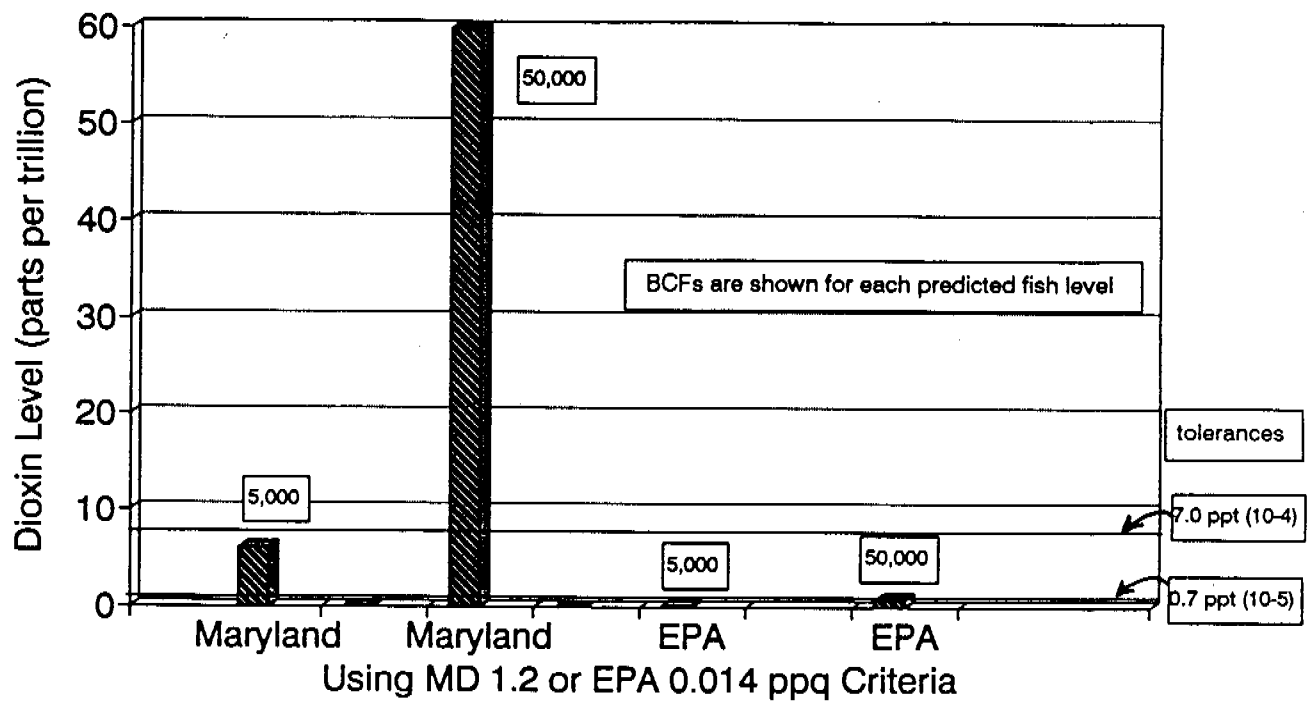
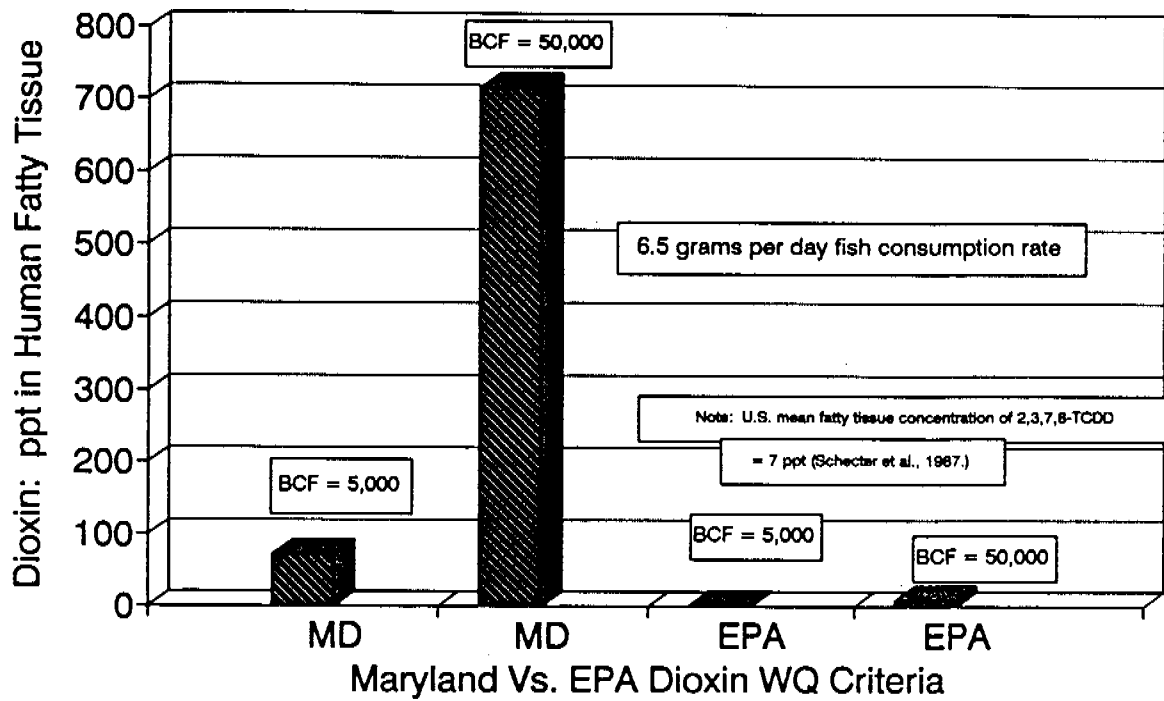


Figure 3.

70-Year Dioxin Concentration in Human Fatty Tissue



BIOACCUMULATION FACTORS

EDF POSITION

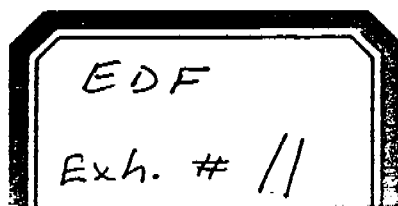
The EPA criteria document recommends the use of a bioaccumulation factor (BAF) of 5,000. EDF agrees with EPA, but only, as EPA acknowledges, if this factor is considered to be non-conservative. The scientific literature developed since the 1984 EPA dioxin criteria document contains many studies pointing to higher bioconcentration (BCF) and bioaccumulation values. Using a BAF factor of 5,000 means that the Board limits its choices of other factors in the dioxin criteria formula. Additionally, EDF strongly recommends that the Board require a specific monitoring program for all affected paper mills to determine the actual BAF for each mill, taking into account the specific environmental circumstances at each location.

SUMMARY

The EPA criteria document of 1984 includes a specific section on BAF, and acknowledges in that report that BCF values for dioxin have been reported from 3,000 up to 900,000¹. Measured BCF's have been reported from 390 to 13,000.¹ EPA resolved this question not through the use of any particular scientific technique, but rather assumed that a BCF of 5,000 would not violate the FDA action level in fish.¹ EPA carefully conditioned this conclusion by stating that if actual field results demonstrated a different BAF then the number should be adjusted.¹

This "guidance" has been of little help to states faced with the dioxin problem. First of all, many states reject the FDA action level of 50 parts per trillion (ppt), which was developed before widespread dioxin contamination was recognized. With what we know about dioxin today, many states believe that public safety depends on much lower values in fish tissue. For example, Minnesota restricts fish consumption at fish concentrations of 32 ppt; Michigan advises at 10 ppt; Maine banned consumption on the Androscoggin River at 6 ppt; and Minnesota issues advisories at 0.6 ppt. Some states believe that any level of dioxin contamination presents some health risk. Virginia's Health Department issued a health advisory on the Jackson/James river with fish values under 10 ppt.² Thus, the "assumption" of meeting the FDA action level is now meaningless.

Since the publication of the EPA document, the scientific literature has produced some rather startling results. It has been clearly shown, and now beyond debate, that dioxin accumulates in fish tissue through food, water and exposure to environmental contamination in sediments, etc.^{1,3,4,5}. The most significant study and the one causing the greatest concern among fishery scientist today is the report by the U.S. Fish and Wildlife Service.⁴ In this 1987 study, one of the few long term, carefully controlled laboratory



studies, researchers found a measured BCF for rainbow trout of 26,000; the estimated BCF at steady state conditions was 39,000 for the lowest exposure concentration where fish were affected. The authors noted, however, that the BCF values had to be calculated since the fish did not reach steady state conditions during the 28 day test period.

It is also known that dioxin concentration will vary among fish species.^{1,4,5} Commonly, it has been noted that bottom feeders accumulate large amounts of dioxin, probably because of their physical proximity to contaminated sediment and possibly diet. However, high levels of dioxin contamination are not limited to suckers and carp, but may occur in trout⁴ and other salmonid species. Data for shellfish and crabs also indicate that dioxin accumulation can be significant.⁵

Given the above data, there is really very little support for the EPA 5,000 BCF number; unfortunately, the available data are either too sparse or too confused because of local variations to provide consistent guidance. This has not prevented a number of states, however, from using higher BAF values in the dioxin formula to protect resident fish species: New York uses 10,000; Illinois 51,000; Minnesota uses 336,346 for salmonids, 84,000 for other species.⁶ Other states have also compensated by using higher fish consumption numbers: Delaware, Wisconsin, Illinois, Michigan, Minnesota, California and Hawaii.⁶

EPA has recognized this problem. The criteria adoption guidance from EPA recognizes (and states that FDA concurs) that some of the assumptions made in deriving FDA action levels are not appropriate for EPA's water quality protection mandates.⁷ Further, EPA states that while the states have some flexibility in setting their individual standards, the use of factors less stringent than the suggested EPA factors requires strong proof.⁷ There is no proof that a BAF of less than 5,000 could be used; the only question is whether a higher value should be applied. The paper industry has proposed using the 5,000 BAF factor.

EDF can agree with the use of a 5,000 factor in Virginia only because the reported fish data (so far, and limited as it is) does not imply a higher number in Virginia waters (imply is the best word we can use because there isn't enough ambient data--practically none--to calculate a BAF). The data base is so poor that, in using the 5,000 value in spite of scientific literature to the contrary, the state is taking the chance of a serious long term mistake. To correct this situation, and accurately evaluate the correct BAF figure for Virginia (especially as paper mills reduce dioxin levels), EDF urges the Board to require that every VPDES permit issued with dioxin limitations contain a specific bioaccumulation study requirement. This requirement would be separate from, and in addition to, the general fish monitoring requirements, since a BAF cannot be calculated from ambient fish data without ambient water quality data, which we will not get in the foreseeable future. These studies should be based on effluent exposure of known dioxin concentrations for long periods (at least 30 days) to fish exposed to these controlled (or at least known) conditions. We offer the

following specific language to be adopted as part of the proposed dioxin standard, or adopted as an implementation policy:

"Each VPDES permit controlling the discharge of dioxin shall contain, in addition to other appropriate dioxin monitoring requirements, a specific program to determine the bioaccumulation factor for the discharge effluent, appropriately diluted to reflect ambient conditions, with respect to resident fish species."

BIOACCUMULATION BIBLIOGRAPHY

1. Environmental Protection Agency. "Ambient Water Quality Criteria for 2,3,7,8-Tetrachloro-dibenzo-p-dioxin." Office of Water Regulation and Standards, EPA 440/5-84-007 (1984). [Incorporated by reference]
2. Virginia Department of Health. "Public Health Advisory- dioxin; Jackson and James Rivers." News Release (Dec. 13, 1989). [Copy enclosed as EDF Exhibit 9-6a]
3. Muir, D. C. G. and A. L. Yarechewski. "Dietary Accumulation of Four Chlorinated Dioxin Congeners by Rainbow Trout and Fathead Minnows." Environmental Toxicology and Chemistry, Vol 7, pp. 227-236 (1988). [Copy enclosed as EDF Exhibit 10- 2]
4. Mehrle, P.M., et al. "Toxicity and Bioconcentration of 2,3,7,8-Tetrachlorodibenzodioxin and 2,3,7,8- Tetrachlorodibenzofuran in Rainbow Trout." Environmental Toxicology and Chemistry, Vol 7, pp. 47-62 (1988). [Copy enclosed as EDF Exhibit 10- 2a]
5. Belton, T. J., et al. "A Study of Dioxin (2,3,7,8- Tetrachloro-dibenzo-p-dioxin) Contamination in Select Finfish, Crustaceans and Sediments of New Jersey Waterways." Office of Science and Research, N.J. Dept. of Env. Prot., CN409. Trenton, NJ 08625 (Oct. 30, 1985). [Copy enclosed as EDF Exhibit 10- 2b]
6. EDF exhibit 8.
7. Environmental Protection Agency. Memorandum from L.S. Wilcher to Water Management Division Directors (Jan. 5, 1990). [Copy enclosed as EDF Exhibit 9-1]

Carcinogenicity and Other Human Health Effects

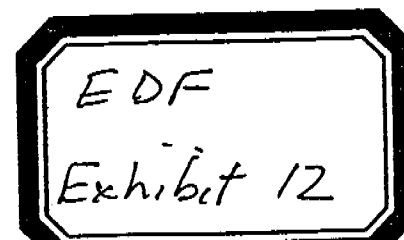
EDF POSITION

The Commonwealth must adopt a dioxin standard that uses a scientifically defensible body of knowledge based on the fact that this chemical is the most potent and toxic substance known. Any argument that focuses on which factor to use to adjust for cancer potency ignores the underlying basis for using such a factor: the mechanism of action and the effects of this compound. The most protective cancer potency factor, used in the EPA criteria, is designed to protect from all environmental contamination, including the specific health risk of cancer. Deviating from the EPA recommended criteria raises the probability of other health risks. EDF recommends using the EPA cancer potency factor because it is the most protective, and acknowledges while other cancer potency factors may be scientifically defensible, they may not be as protective.

SUMMARY 1. CARCINOGENICITY

The potential for dioxin, or any other toxic substance to cause cancer in humans is a difficult issue to address by applying direct data. The reason for this difficulty is that humans are not used in experiments on cancer causing potential of chemicals; nor will humans ever be used as experimental animals. Thus, the cancer causing potential for humans exposed to low doses over long periods must be gleaned from data on other exposures and on experimental animals. The existing data concern the accidental (including occupational) exposure of humans, and laboratory exposure of experimental animals to dioxin. Therefore, any determination of the cancer causing potential of dioxin must use some extrapolation from exposures typical of accidents (usually high doses over short periods of time), unknown exposures in the workplace, or exposure of laboratory animals. The goal is to predict the effects from the anticipated exposures to chronic releases by pulp mills (usually low doses over very long periods of time). The mechanisms used in such extrapolations have become the source of great controversy in setting standards for release of dioxin, and other toxic compounds.

The necessity in using experimental animals for determining cancer causing potential of dioxin is obvious, but the underlying scientific basis is not always appreciated. The cancer causing potential of any substances can be assessed using experimental animals because of the basic common properties of all animal cells. Most, if not all aspects of cell function are so basic that original research on nerve, muscle, hemoglobin, heart and kidney function can be applied to humans regardless of the experimental species used. This applicability is true for cancer causing potential of specific chemical agents. Thus, Safe¹ concluded that the pattern of toxicity, not simply cancer causing potential, is the same in all species not only for dioxin, per se, but also the entire group of compounds to which dioxin belongs (the chlorinated hydrocarbons). The argument regarding the toxicity and cancer



causing potential of dioxin is still based on the fact that dioxin is a highly toxic substance, exposure to which causes cancer in animals².

Thus, the cancer causing potential of dioxin must be determined from a larger body of information using data for experimental animals, and herein lies the controversy. The experimental animals used thus far have different levels of sensitivity to dioxin. The Toxicological Profile for Dioxin³ (p14) graphically compares the exposure levels among studies and this comparison indicates that the range of 1-10 parts per trillion (ppt) is the dose (or concentration) at which effects are most commonly observed. Thus, use of that work as the basis for determining cancer causing potential is scientifically defensible on the basis of repeatability.

The three federal agencies that have used such results from animal experiments to determine a cancer potency factor for humans have interpreted the results in slightly different ways, leading to variation in the resulting factor. The first scientific disagreement centers on whether to use early mortalities as non-cancerous individuals, or to remove them from the experimental group; EDF favors the latter strategy. The second issue is whether to correct for animal size on the basis of body weight or body surface area (both of which vary with animal "size"). EDF recognizes the validity of each method. The different cancer potency factors were compared by the Virginia Department of Health (exhibit 9-3). An important point that has been overlooked is that there is no disagreement over whether or not exposure to dioxin increases the cancer risk for humans or animals. EDF's position is that the final decision on the dioxin standard must be based on the fact that exposure to even low levels of dioxin cause cancer.

II. HUMAN HEALTH EFFECTS

The data for accidental and occupational exposure of humans to dioxin have been tabulated and summarized by several authors in order to further understand the cancer causing potential and other toxic effects of dioxin. The cases of exposure include some residences in Missouri, manufacturing facilities such as the one at Nitro, West Virginia, exposure during spraying of Agent Orange in Vietnam, accidental spraying with herbicide, and the accidental large dose exposure in Italy. None of the cases of human exposure yet examined presents a situation that clearly permits the type of straightforward conclusions that many regulators and administrators would like to have. What is consistent from the data on human exposure is that the following symptoms are characteristic in persons exposed to dioxin or dioxin containing materials:

- skin lesions and irritation, known as chloracne (All cases)
- impaired liver function (4,5)
- impairment of peripheral nervous system function (5)
- increased white blood cell counts (4)
- reduced responses of the immune systems (immunosuppression) (4)
- some type of impairment of reproductive function (6,7)

This is not to say that the effects of dioxin exposure on humans is well understood, quite the contrary. Silbergeld and Mattison⁶ noted that exposures and doses are not well known for humans, a conclusion reached by others². The need identified by the authors is for sufficient data (probably epidemiological) to provide numerical assessment of human effects from the few known cases of human exposure. In other words, researchers know there is an effect, but they do not know how much dioxin causes which effect.

An excellent example of such work is in the paper by Suskind and Hertzberg⁴, investigating the health of a group of workers exposed to dioxin for approximately 20 years in the manufacture of the herbicide 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) to determine the long term health effects. The project included clinical examinations during June 1979 and medical history reviews. The results are not atypical of other studies on dioxin or other occupational toxic chemical exposures. Suskind and Hertzberg concluded that while uniform exposures could not be guaranteed, there was no reason to doubt the exposure to dioxin. There were carcinogenic (but not statistically significant) and other toxic effects apparent in the workers exposed to dioxin in the workplace. The most common effects were increases in chloracne (skin lesions), respiratory disfunction (breathing depth, volume, etc.) and upper gastrointestinal tract irritations, including ulcers. In another such study, the exposure of residents of Times Beach, MO caused alteration of liver function and depression of normal immune responses of the body⁴, neither of which is specifically carcinogenic, but both of which are long term non-carcinogenic effects.

In two recent reviews^{1,7}, Safe pointed out that there are numerous non-carcinogenic toxic effects of dioxin, due at least in part to the way in which dioxin acts on animal cells. These effects fall into three basic categories: developmental, reproductive, and immunological. Developmental effects are those that change the growth of organs and tissues in fetuses (fetotoxicity) or immature animals as they grow and mature. Fetotoxicity and teratogenicity (causing birth defects) occur in rats, rabbits, monkeys and mice ^{4a} exposed to dioxin, with a fetal toxicity (death was the end point) of 1 ppb. Using the bioconcentration factor of 5000, consumption of fish with even low parts per trillion levels of dioxin will expose a fetus to ppb levels of dioxin. The Toxicological Profile³ reviews some of the recent work on developmental effects, noting again the difficulties in interpreting epidemiological data on humans with uncertain exposure levels, and that the point remains controversial. Safe⁷, however, as recently as February 1990, concluded, on the basis of the most recent experimental data^{9c}, that developmental effects were the most worrisome and dangerous of human health effects.

Reproductive effects occur in rats, mice, hamsters and primates (monkeys), and include reduced litter size, internal bleeding, reduced reproductive organ size, and spontaneous abortion (in rhesus monkeys)^{3,9,9a,9b,10}. There is even a demonstrated effect on some wild bird species⁶. Once again, the difficulty in making a clear conclusion for human effects is the uncertainty of actual dose and the total absence of studies using humans as experimental animals. As with other effects, there is no

doubt that dioxin has an adverse effect on reproductive function in non-humans, the doubt is in how to apply this information to human exposure. It is not known, for example, if exposure to 2 ppq dioxin will cause spontaneous abortion in humans, as it does in some other animals³. If dioxin is allowed to accumulate in humans to levels used in experiments on rats, then we can expect an impact on reproductive behavior^{9,9a,9b}, and loss of reproductive function from the levels reported in mother's milk.

Immunological effects on humans are more clear because the level of action of dioxin can be related to the animal, organ, tissue and cellular levels of function in various species, including humans^{1,2,3,4,5,10}. Furthermore, immunologic effects do not require the sort of extrapolations among species that characterize the carcinogenicity issues described above. These studies show that exposure to dioxin in the parts per trillion to parts per billion range in experimental animals causes atrophy of the thymus, delayed immune responses, decreased antibody production, and increases in identified cell products¹¹. It is important to note single doses as well as chronic administration of dioxin elicit immunological responses in experimental animals, although somewhat higher doses (ppb) are required. The significance of this finding is that the dioxin standard must be protective of single high dose exposures, in addition to the chronic low dose exposure.

The accepted role for dioxin in affecting the immune system is within the cell, at the level of a specific protein¹¹. According to current understanding, dioxin enters a cell and binds to a receptor protein and then the dioxin-receptor complex affects DNA function. This mechanism means that it is possible for individual molecules of dioxin to exert an effect on the immune system (and on other cells). The danger for human health is that the direct effect on the immune system does not have to be great in order to seriously impair the health of large numbers of people, as in other immune disorders.

The immunotoxic effect of dioxin demonstrates the extreme level of sensitivity of animals and the multiple levels at which sublethal effects can be observed. Furthermore, new information on the immune response to dioxin clearly shows that this compound can act in more than one way^{12,13}. Medical College of Virginia researcher Dr. Michael Holsapple has provided clear and convincing evidence that the cellular receptor mechanism of dioxin action on immune systems does not account for all the observed effects. Although this point was at one time a controversial issue, it is now generally accepted. Dr. Holsapple has also demonstrated that similar actions on immune systems result from exposure to other chlorinated dioxins¹⁴, not just the one compound that is under intense investigation and regulation. Thus, it is imperative to strictly control dioxin release at a conservative level to prevent increasing body burdens to the point where diverse toxic effects are manifested.

The scientific community will continue to focus research energy and resources on the health of dioxin and the mechanism of action, because of its applicability to other systems and other compounds. As researchers uncover new

information, the existing theories and models will be modified so as to incorporate the latest information. This change in theories and models does not indicate scientific uncertainty about the effects of dioxin, only on how those effects are brought about. The nature of science requires a continual modification of theories to account for the latest data. For the purposes of regulation, the specific mechanism of action of dioxin does not change the fact that the mechanism results in cancer or a suppression of the immune system, or a failure of reproductive behavior, or birth defects. Other authors,^{4a,11} reviewing this issue agree that current dioxin standards must use conservative estimates in order to provide unqualified protection for the environment and the human population.

Bibliography

1. Safe, S. "Comparative toxicology and mechanism of action of polychlorinated dibenzo-p-dioxins and dibenzofurans. Annual Review of Pharmacology and Toxicology, 26, 371-399 (1986).
2. National Toxicology Program, "Fifth Annual Report on Carcinogens, Summary 1989." NTP 89-239, National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709.
3. "Toxicological Profile for 2,3,7,8- tetrachlorodibenzo-p-dioxin." Agency for Toxic Substances and Disease Registry, U.S. Public Health Service and Environmental Protection Agency (1989).
4. Hoffman, R.E. et al., " Health effects of long-term exposure to 2,3,7,8- tetrachlorodibenzo-p-dioxin." Journal of the American Medical Association Vol. 255 (no. 15), 2031-2038 (1986).
- 4a. Wilson, J.A. et al., "The Toxicity of Dioxins." Environmental Institute for Waste Management Open File Report No. 20, University of Alabama, P.O.Box 2486, Tuscaloosa, AL 35486.
5. Suskind, R.R. and V.S. Hertzberg "Human health effects of 2,4,5-Y and its contaminants." Journal of the American Medical Association 251, 2372-2380 (1984).
6. Silbergeld, E.K. and D.R. Mattison. "Experimental and clinical studies on the reproductive toxicology of 2,3,7,8- tetrachlorodibenzo-p-dioxin." American Journal of Industrial Medicine 11, 131-144 (1987).
7. Safe, S. "Health effects of dioxins and related compounds." American Association for the Advancement of Science Annual Meeting, New Orleans, LA. 15-20 February 1990.
8. Hoffman, D.J. et al., "Embryotoxicity, teratogenicity, and aryl hydrocarbon hydroxylase activity in Forster's terns on Green Bay, Lake Michigan." Environmental Research 42, 176-184 (1987).

9. Moore, R.W. et al., "Effects of perinatal 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) exposure on the development of male rats and their androgenic status." *Toxicologist* 10, 247 (1990).

9a. Mably, T.A. et al., "Altered sexual behavior in male rats exposed perinatally to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)." *Toxicologist* 10, 247 (1990).

9b. Bjerke, D.L. et al., "Effects of perinatal exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on reproductive function in male rats." *Toxicologist* 10, 313 (1990).

9c. Fine, J.S. et al., "Impaired prothymocyte activity following TCDD exposure." *Toxicologist* 9, 39 (1989).

10. Sharma, R.P. and P.J. Gehring. "Effects of 2,3,7,8-tetrachlorodibenzo-p- repeated exposures." *Annals of the New York Academy of Sciences* 487-497 (1979).

11. Silbergeld, E.K. and T.A. Gasiewicz. "Dioxins and the Ah receptor." *American Journal of Industrial Medicine* 16, 455-474 (1989).

12. Holsapple, M.P. et al., "Direct suppression of antibody response by chlorinated dibenzodioxins in cultured spleen cells from (C57BL/6 X C3H)F1 and DBA/2 mice." *Immunopharmacology* 12, 175-186 (1986).

13. Dooley, R.K. and M.P. Holsapple. "Elucidation of cellular targets responsible for tetrachlorodibenzo-p-dioxin (TCDD)- induced suppression of antibody responses: I The role of the B lymphocyte." *Immunopharmacology* 16, 167-180 (1988).

14. Holsapple, M.P. et al., "Suppression of humoral antibody production by exposure to 1,2,3,6,7,8- hexachlorodibenzo-p-dioxin." *Journal of Pharmacology and Experimental Therapeutics* 231, 518-526 (1984).

APPLYING THE DIOXIN STANDARD:

STREAM FLOW

EDF POSITION

EDF recognizes that the traditional stream flow application of 7Q10 may be overprotective for standards based on lifetime exposures to carcinogenic materials. EDF rejects, however, the paper industries' proposed "mean annual" flows as under-protective. To provide acceptable dilution for lifetime risks, as well as ensuring dioxin levels do not inadvertently "bump" other dioxin effects, such as reproductive impairment, immunological toxicity and aquatic life toxicity, EDF recommends the use of an "harmonic mean", calculated using monthly data to produce a single annual number, and where warranted, a seasonal tier to adjust where necessary for localized effects, as the appropriate stream flow application.

SUMMARY

Traditionally, the Board has applied a minimum flow requirement for all water quality standards: 7Q10.¹ This flow protected all beneficial stream uses, including aquatic life, at a minimum low flow applicable to all dischargers.¹ As the Board proceeds with human health standards, however, the applicable period for protection is no longer a low stream flow, but rather a long term, often lifetime risk of cancer. For the protection of such long term risks, the application of 7Q10 may be over-protective since these flows, by definition, occur only once in 10 years.

The paper industry has proposed using "mean annual" flows for the lifetime public health risks of dioxin. While such a proposal may appear reasonable for lifetime exposures, it is in fact fraught with problems. Mean annual flow, or the simple average of annual flows, produces a number that is skewed by both high winter/spring flood flows and low summer extremes. The average, however, does not "balance" these two extremes but is disproportionately affected by the high spring and flood flows. This problem is easily demonstrated by looking at the distribution of flows for the Jackson, Blackwater and Mattaponi Rivers, using data from the U.S. Geological Survey Office.²

The mean annual flow for the Jackson River, adjusted for the effect of Lake Moomaw, is 588 cfs.³ Even though this flow represents a leveling of flow extremes, the mean annual flow is almost twice as high as the mean flow for each summer month.⁴ Exhibit 13-4 also demonstrates the impact of monthly maximum flows on the arithmetic mean. Any standard set by this method would be exceeded significantly during the summer periods when aquatic life uptake is at its peak and public contact with the state's waters are highest.

Perhaps more importantly, however, from a scientific viewpoint, mean annual flows are less accurate in representing daily, weekly, or monthly fluctuations. This is demonstrated in EDF exhibit 13-5.⁵ The harmonic mean is a better "fit" for the various flow ranges expected over a life time period, as shown by the maximum and minimum flows by month, as well as the last flow year (1989), a low flow year (1988), the first flow year (1975) and the highest flow year (1984). Exhibit 13-5 again shows the "pull" that high flows have on the annual mean, even when means of each month are used to calculate annual means (means of means). From the scatter of points around the mean, the predictive or relative value of the mean annual flow is much weaker, even when the annual mean is computed for each month. Computing the mean from annual means for the entire period of record is even worse: the mean annual flow from the U.S.G.S. data for the period of record 1975-1989, using the mean of the annual means, is 717 cfs. This flow value exceeds the individual monthly flow averages of the record period 62% of the time. During the critical summer and fall low flow months, June-September, 717 cfs exceeds the monthly average flows for the period of record 87% of the time. Any water quality standard, even a lifetime health risk, based on a mean annual flow value calculated in this manner would be at best only a rough approximation of exposure.

The harmonic mean number is also improved by calculating the mean not from annual flow means but rather monthly averages over the period of record. For example, the annual harmonic mean calculated using the annual harmonic means for the period of record is 658 cfs (without adjustments). The same annual harmonic mean calculated using monthly harmonic means is 366 cfs. The more data computed into the mean results in a generally lower number, which better reflects the flow numbers without the disproportionate high flows.

Another serious problem arises with a mean annual flow value. Because the number is skewed so strongly to higher flows, the dilution it contemplates is often not available, with the result that any discharge permitted with such assumed dilution actually results in much higher levels in the ambient water during many months of the year. For example, using the 3.8 ppq aquatic effects value discussed in the aquatic toxicity section⁶, or even the toxic effect number reported in the literature of 38 ppq,⁷ or the 2 ppq reproductive number or immunological effects discussed in the "carcinogenic and other effects"⁸ as compared to industries' proposed 1 ppq ambient level at mean annual flow, it can be readily seen that protection against all of these toxic effects would be lost during summer low periods. During these periods, dioxin toxicity, not carcinogenicity, is the limiting factor.

EPA warns against such possibilities in its dioxin guidance.⁹ EPA properly concludes that the possibility of the EPA criteria causing other effects is unlikely; however, such impacts may result where any state relaxes the EPA criteria. The burden then falls upon the state to justify the continued validity of the new standard.⁹

The draft EPA technical support document will likely recommend the use of "harmonic means".¹⁰ The harmonic mean calculation differs from the simple

average mean by using the inverse number. The effect, as shown above, is to reduce the disproportionate strength of high flow instances. As harmonic means produce lower numbers, especially if calculated using monthly data instead of annual, there is less likelihood that other toxic effects of dioxin will be encountered.

The arithmetic mean gives equal "weight" to the high and low values, despite the fact that the high flows are not typical and are relatively infrequent. Most of the flow values are below the arithmetic mean: they cluster around low flow values. Because the distribution of flow values is not random or uniform, the measure of "central tendency" (typically the mean) must be appropriate for the type of distribution that actually occurs. The harmonic mean, as does the geometric mean, corrects for this unequal distribution of values, especially the power of a few large values to alter the measure of central tendency.

STREAM FLOW BIBLIOGRAPHY

1. Virginia State Water Control Board. "Water Quality Standards" (1988, as amended). [Incorporated by reference]
2. U.S.G.S. "Gaging station data from the Jackson, Mattaponi and Blackwater Rivers". (1990).
3. Letter. J.P. Godfrey to P. deFur, February 23, 1990.
4. Environmental Defense Fund. "Comparison of arithmetic, harmonic and monthly mean flow calculations on the Jackson River." Chart (1990).
5. Environmental Defense Fund. "Comparison of various mean flow data on the Jackson River." Chart (1990).
6. EDF Exhibit 14.
7. EDF Exhibit 10-2a.
8. EDF Exhibit 12.
9. U.S. Environmental Protection Agency. Memorandum from L.S. Wilcher to Water Division Directors. (Jan 5, 1990) [enclosed as EDF exhibit 9-1]
10. Personal Communication. Bill Diamond, EPA; Durwood Willis, State Water Control Board.

AQUATIC LIFE TOXICITY

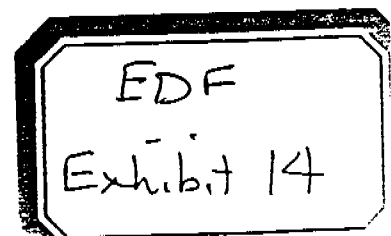
EDF Position

A substantial body of research exists to show that dioxin is the most toxic compound known. The paper industry proposal to set an ambient water quality criteria for the protection of public health at 1.2 parts per quadrillion (ppq), applied at mean annual flow, would allow dioxin concentrations to reach levels that cause chronic toxic effects to aquatic life during low flow periods above 7Q10 flow. EPA has no dioxin criteria for the protection of aquatic life; the lack of such criteria does not, however, mean that such effects may be ignored. For the purposes of limiting aquatic toxicity, and protecting beneficial uses of state waters, EDF recommends that the Board use 3.8 ppq as a guide to chronic toxicity to aquatic life.

SUMMARY

Although the carcinogenic potential of dioxin is usually considered controlling for all toxic effects, and the EPA criteria of 0.013 ppq accordingly protects all aquatic life, proposals to relax the EPA criteria through various means (risk level, mean annual flow, cancer potency, etc) threaten to reduce the protection afforded by the EPA criteria to the point that other toxic effects of dioxin occur. The paper industry proposal of 1.2 ppq is 100 times less stringent than the EPA criteria and, if applied at mean annual flows, could result in dioxin concentrations in state waters that are 5-7 times higher than the 1.2 ppq standard¹. EPA specifically cautions against these effects².

There is a considerable body of literature on the toxicity of dioxin to fish, shellfish and even on birds that feed primarily on fish or seafood. Rainbow trout, yellow perch, striped bass, goldfish, minnows, other finfish, blue crabs, soft clams and Forsters terns take up dioxin from water and/ or contaminated sediments and concentrate it in body tissues^{3,4,5,5a,5b,6,7}. Several fish species suffer toxic effects when exposed to dioxin in the water^{7,8,9}, including both lethal and sublethal effects. Most work has utilized relatively short exposure periods of hours to a few days, or injection of the dioxin using an oil based medium to deliver the dioxin. These experiments consistently show that dioxin administered at levels of parts per billion are lethal and result in observable histopathologic effects. Spitsbergen^{7,8,9} has also shown that immunotoxicity occurs in these species as it does in the mammalian models used, raising the probability that long term accumulation will manifest itself in sublethal effects. Similarly, blue crabs not only take up dioxin and concentrate it in tissues, but also suffer from delayed molting and inability to regenerate claws when exposed to dioxin⁵. In most of these experiments, the mechanism of administering dioxin has been artificial, via injection or in high ambient doses, rather than via a normal uptake mechanism such as in food or in contaminated sediment exposure.



The most significant study to date on the toxicity of dioxin in aquatic animals is that of Mehrle at the National Fisheries Contamination Research Center of the U.S. Fish and Wildlife Service at Columbia, MO¹⁰. Mehrle exposed rainbow trout to ppq levels of dioxin (38-789) in the water with a normal feeding regime, with the possibility for uptake directly from the water or from dioxin that might adsorb to the food in the experimental tanks. The exposure period was 28 days with a 28 day depuration period following. After 28 days, significant mortality occurred at 176 ppq and higher exposure groups, and only those in the lower concentrations were continued in the depuration phase.

Significant mortality continued during the depuration phase of the experiment at all test concentrations. By the end of the 28 day depuration phase, 83 % of the trout exposed to 79 ppq and 45 % of those in the 38 ppq exposure had died. There was no evidence of recovery from the exposure to dioxin. Dioxin caused decreased survival, diminished growth, and alteration of normal behavior during both the exposure and depuration phases.

Because the authors continued to observe toxic effects at the lowest exposure, the no observable effect level was lower than the lowest experimental exposure of 38 ppq. Given the nature of the sublethal effects reported at 38 ppq, and the nature of the sublethal immunologic effects, it is clear that dioxin's toxic effects would be felt on rainbow trout, and other species, at much lower levels.

Without additional data, EDF can only predict possible safe levels by using adjustment factors, the method used before EPA arrived at present procedures of tests and calculations. Continued mortality of fish during depuration clearly indicates that dioxin's persistent sublethal effect has a long delay, but is very powerful.

For demonstrated chronic toxicity, a factor of 1/20 or at least 1/10 is usually chosen. Since the test period exposure was longer (28 day) than other test periods, EDF will use a factor of 1/10. Thus, in the absence of a formal EPA criteria, EDF believes that no more than 3.8 ppq dioxin should be permitted in order to protect aquatic life. Aquatic life protection is required at 7q10 flows as an established beneficial use under state law.

While admittedly a non-precise number, the use of application factors has been successful in many cases in predicting safe levels. The new EPA criteria methods, once all data requirements are met, are often more stringent than the numbers predicted by application factors. EDF believes that is the case here, i.e., the eventual EPA criteria will contain a dioxin criteria less than 3.8 ppq. Until such time as the EPA criteria is completed, however, each state must consider possible aquatic toxicity if that state chooses to apply a less stringent health criteria than EPA's.

BIBLIOGRAPHY

1. EDF Exhibit # 13.
2. U.S. Environmental Protection Agency. Memorandum from L. S. Wilcher to Water Management Division Directors (Jan.5, 1990) (enclosed as EDF exhibit 9-1).
3. Spitzenbergen, J.M. et al., "Toxicity of 2,3,7,8- tetrachlorodibenzo-p-dioxin (TCDD) in freshwater fish." Toxicologist 4, 190 (1984).Abstract.
4. Kleeman, J.M., et al., "Uptake, metabolism and elimination of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in fish. Toxicologist 4, 189 (1984). Abstract.
5. EDF exhibit 10
 - 5a. O'Keefe,P., et al., " Tetrachlorodibenzo-p-dioxins and tetrachlorodibenzofurans in Atlantic coast striped bass and in selected Hudson River fish, waterfowl and sediments." Chemosphere 13, 849-860. (1984).
 - 5b. EDF exhibit 12-8.
6. Kuehl, D.W., et al., "Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp." Chemosphere 16, 667-679. (1987).
7. Spitzbergen, J.M. "2,3,7,8- tetrachlorodibenzo-p-dioxin toxicity in yellow perch (*Perca flavescens*). Journal of Toxicology and Environmental Health 23, 359-383. (1988).
8. Spitzbergen,J.M. et al., "Interactions of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) with immune responses of rainbow trout." Veterinary Immunology and Immunopathology 12, 263-280 (1986).
9. Spitsbergen,J.M. "Morphologic lesions and acute toxicity in rainbow trout (*Salmo gairdneri*) treated with 2,3,7,8-tetrachlorodibenzo-p-dioxin." Journal of Toxicology and Environmental Health 23, 333-358. (1988).
10. EDF exhibit 10, Mehrle enclosed as EDF 10-2a.

June 21, 1991

Charles I. Carroll, Jr.

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JUN 26 1991

WATER RESOURCES BOARD

West Virginia Water Resources Board
State Capital
Charleston, WV.

Please Refuse permit to polute Ohio River.

In the June 20, 1991 Herald-Dispatch it reads that alabama river pulp has ask for permission to increase dioxin a highly toxic compound suspected of causing cancer from .013 parts per quadrillion to 1.2 parts per quadrillion 91 times greater amounts.

This location is approximately 50 miles above where I take hundreds of diver diving. These divers will have traveled from as far away as Mexico, Canada, Maryland, New Jersey, Michigan, Pennsylvania, Illinois, Florida, and many other states. I do not know what dangers these toxic chemicals will pose for these tourist divers.

I know that our river is polluted because we have been advised not to eat the fish because they are full of toxins.

I am also afraid that the endangered species I refer to as the Ohio River Mussel a prehistoric family of creatures that are having a hard time adapting to the poisons we are now feeding the river will not survive this new additional to toxin river poisoning that is being proposed.

I also have this last fear my family and I live below the proposed dump site and Huntington City Water treated by WV-American Water acquires the drinking water from the Ohio River will the toxin levels be within safe drinking levels to guarantee that neither I nor my family will develop a cancer associated with these dioxin. Will it be assured that the Alabama River Pulp post Bonds of large enough amounts to cover medical expenses incurred by the families who have family members that develop a cancer that associated to exposure to dioxin.

Is a possible 1500 jobs worth the possible early death of all animal life (including human animal) below Mason County, West Virginia worth any business either large or small.....

Yours Truly,

Charles I. Carroll, Jr.
Charles I. Carroll, Jr.

C
The Herald-Dispatch
THURSDAY, JUNE 20, 1991

Water board to hear debate on toxin level

Environmentalists oppose firm on proposed mill standards

By MONTY FOWLER
Of The Herald-Dispatch staff

CHARLESTON — Environmentalists will argue tonight against recommendations to increase the allowable levels of dioxin in the Ohio River to accommodate a proposed paper mill in Mason County.

Small amounts of dioxin, a highly toxic compound suspected of causing cancer, would be released into the Ohio River at Apple Grove by Alabama River Pulp Co.'s proposed \$1.1 billion plant.

The state Water Resources Board has recommended that U.S. Environmental Protection Agency guidelines on the allowable amount of dioxin released in rivers be followed.

However, Alabama River Pulp has asked that the limit be raised from .013 parts per quadrillion to 1.2 parts per quadrillion, a 91 percent increase. State officials say even the higher level is undetectable with their current equipment.

A public hearing at 7 tonight in Building 7, Conference Room C of the Capitol Complex in Charleston, is scheduled as part of the board's triennial review of state water quality standards.

"The question is will the state government prostitute itself to get this economic development at the expense of its health and people or will it hold firm and say, 'Alabama, these are the rules,'" said Norma Steenstra, environmental coordinator of the West Virginia-Citizen Action Group.

Other groups, including the Ohio Valley Environmental Coalition and Tri-State Building and Construction Trades Council, are concerned about the potential downstream impact on cities like Huntington, which get drinking water from the Ohio.

Stuart Caldwell, a Charleston attorney representing the trades council, said their chief concern is how the allowable pollution levels are calculated and the fact that the Water Resources Board is relying on an EPA rule of thumb instead of actual river flow measurements in evaluating the requested increase.

"The state has not done any of these calculations," said Caldwell, whose group hired an environmental firm to do so. "If this rule change goes into effect, (pollution) dischargers will be able to dump 7.4 times the amount of carcinogens into the Ohio that they're currently dumping."

EPA rules allow states to use one of two methods to calculate wastewater discharges. Caldwell said, but the way state law is written, all an applicant has to do to get the higher allowed method — the harmonic mean flow — is ask.

"It doesn't take a rocket scientist to see what this means. The harmonic mean flow is much higher than the 7Q10 flow," the other method, which measures the lowest flow of a river on seven days over a 10-year period.

If industries request the harmonic mean flow method be used to calculate discharges, they could increase discharges of potential cancer-causing chemicals by 7.4 times on the Ohio, 5.4 times on the Kanawha and 7.8 times on the Greenbrier rivers, Caldwell said.

► River

From C1

"The 7Q10 method is the safest, most conservative method of measuring carcinogens; we think it needs to be looked at before the water board goes any further," Caldwell said.

Alabama River's owners, Parsons & Wittemore Inc. of Ryerbrook, N.Y., are proposing a \$700 million pulp mill and a \$400 million news print plant at Apple Grove but recently asked the Division of Natural Resources to delay public hearings on its water discharge permit.

Jan Taylor, technical adviser to the Water Resources Board, said the state has never set a dioxin level and has followed the EPA's recommended level of .013 parts per quadrillion.

The board has recommended to continue following that level but won't make a final decision until after a public comment period ends June 28. Its decision must then be approved by the Legislature, the governor and the EPA before it would go into effect in 1992.

The EPA has approved similar levels in Alabama, Maryland and Virginia, but environmental groups have filed lawsuits challenging those changes.

"There's a fish advisory on the Ohio and Kanawha rivers that say don't eat them, which should tell us right there that we have a problem," Steenstra said. "There are indications, thank God, the Water Resources Board will hold firm on this one. It depends what kind of lobby they get on the other side."

The Associated Press contributed to this report.

JUNE 26, 1991

To the WV Water Resources Board:

We the undersigned are not against any industry that will bring 2,000 jobs into our state. However, we are against raising dioxin levels to be released in our rivers from .013 parts per quadrillion to 1.2 parts to accomodate such an industry. We are also against self regulation of any industry handling such toxic chemicals.

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JUNE 26, 1991

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LEGAL DEPARTMENT

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Monongahela Power Company

Part of the Allegheny Power System

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JUN 27 1991

WATER RESOURCES BOARD

June 26, 1991

West Virginia Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

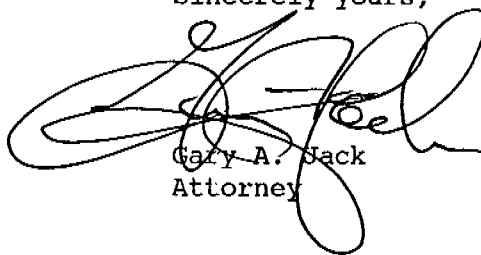
RE: Triennial Regulatory Review - 1991

Dear Water Resources Board:

Monongahela Power Company has contributed to and fully supports the joint comments and recommendations of the West Virginia Chamber of Commerce and the West Virginia Manufacturers Association which are submitted under separate cover in connection with the Water Resources Board's revisions to Series I. Accordingly, please consider Monongahela's comments to be included within that joint submittal, except for one additional comment noted below.

Monongahela Power Company is disappointed that the Board did not propose many of the revisions submitted by the Chamber and the Manufacturers on February 22. These proposals, many of which have been endorsed by EPA and adopted by neighboring states, represent a more flexible approach to water quality management. Accordingly, we find our state being placed in a competitive disadvantage vis a vis our neighbors until the Board considers and implements such revisions. We are not asking for the Board to diminish or compromise environmental quality; we are just asking that our proposals be given due consideration and proposed for public comment rather than to be just summarily rejected.

Sincerely yours,



Gary A. Jack
Attorney

GAJ:drf

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June 27, 1991

Frances E. Hunter
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

-Hand Delivered-
June 27, 1991

Re: Public Comments On Proposed
Amendments And Revisions To
State Water Resources Board
46 CSR 1 Title 46 Legislative
Rule Series 1 Requirements
Governing Water Quality Standards
CMP File No. 3671

Dear Ms. Hunter:

Enclosed is a copy of the results of a study commissioned by Tri-State Building and Construction Trades Council pertaining to the proposed changes in Rule 8.2.C. of requirements governing water quality standards. I submit this study as a "public comment" in conjunction with the presentation I made on behalf of my clients before the Water Resources Board on June 20, 1991. Please see to it that the Board is provided a copy of the enclosed study results.

Again, I urge the Board, on behalf of my clients, to evaluate the impact the proposed Rule change will have on the quantity of carcinogens discharged to the rivers and streams of West Virginia prior to recommending the change.

Very truly yours,



Stuart Calwell

SCjks
Enclosure

cc w/encl: Steve Burton
Allen Fisher

C
E
A

Carpenter Environmental Associates, Inc.

70 Hilltop Road, 1st Floor
Ramsey, New Jersey 07446
Telephone (201) 818-4844
Fax (201) 818-4853

RECEIVED
JUN 27 1991
WATER RESOURCES DIV.

June 26, 1991

Stuart Calwell, Esq.
Calwell, McCormick & Peyton
405 Capital Street
Charleston, WV 25301

**Re: Study of Proposed Rule 8.2.C of West Virginia's "Requirements
Governing Water Quality Standards."
CEA # 91015**

Dear Mr. Calwell:

Carpenter Environmental Associates, Inc. has carried out the study described below to determine the effect of the proposed rule change to allow the use of harmonic mean flow as the design flow for wasteload allocation for carcinogens.

Proposed Rule 8.2.C. of West Virginia's "Requirements Governing Water Quality Standards" (46 CSR 1 Title 46 Legislative Rule) would allow the harmonic mean flow of a river to be used as the critical design flow in the wasteload allocation model that determines the quantity of a carcinogen that can be discharged into the river by a facility. Utilizing the harmonic mean flow would represent a change from West Virginia's usual method of wasteload allocation which uses the 7 day mean low flow with a 10 year return period (7Q10) expected in a river as the critical design criteria. Use of the harmonic mean flow rather than a measure of the low flow as the critical design flow would result in an increase in the amount of carcinogens that could be discharged to the waters of West Virginia. The amount of the increase can be estimated by comparing the harmonic mean flow of a river, or river segment, to the low flow.

Carpenter Environmental Associates, Inc. (CEA) has developed the harmonic mean flow (Q_h) for locations on three rivers in West Virginia. These rivers are the Ohio, Kanawha, and the Greenbrier Rivers. These rivers were selected due to their size (large, medium, and small, respectively) and nature (controlled or uncontrolled). Daily stream flow data for the available periods of record were obtained on floppy disk for three United States Geological Survey (USGS) flow gaging stations, located on these rivers. The data were extracted from these disks and incorporated into a spreadsheet program to perform the required calculations. To

calculate the harmonic mean flow, the total number of record flows (N) was divided by the summation of the inverse of each daily flow (1/Q). The harmonic mean flow was calculated as follows:

$$Q_h = \frac{N}{\sum 1/Q}$$

Q_h = harmonic mean flow
N = Total number of recorded daily flows
1/Q = Inverse of daily flow

Due to the large quantity of data used for the analysis, two independent computations for the harmonic mean flow were performed. The results of these computations were compared to assure an accurate result.

The 7Q10's were obtained from the USGS Water Resources Division located in West Virginia. The 7Q10's for the gaging stations of interest are as follows: Ohio River at Huntington - 4500 cubic feet per second (CFS) (estimated), Kanawha River at Charleston - 1250 CFS (estimated), and the Greenbrier River at Hilldale - 55 CFS. Due to the regulation of the Ohio and Kanawha Rivers by locks and dams, there are only estimated design 7Q10's available. CEA was informed by the USGS that these estimated 7Q10's are currently appropriate to use as the critical design flow. The results of the analyses follow:

River	USGS Station Name	USGS #	Period of Record (Years)	N	$\Sigma 1/Q$	Q_h (CFS)	7Q10 (CFS)	$\frac{Q_h}{7Q10}$ Ratio
Ohio	Huntington	03206000	53	16319	0.48908	33,367	4500	7.4
Kanawha	Charleston	03198000	52	18410	2.7465	6,703	1250	5.4
Greenbrier	Hilldale	03184000	55	19530	45.65	428	55	7.8

Each ratio developed above represents the factor by which the quantity of carcinogens discharged to West Virginia's waters will be increased if the harmonic mean flow is used as the design flow rather than the 7Q10. That is, promulgation of Proposed Rule 8.2.C. would result in 5.4 to 7.8 times more carcinogens being discharged in the river segments studied. Similar analyses must be

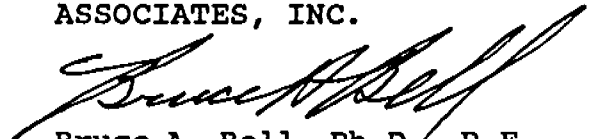
performed for other river segments in West Virginia to determine the factor by which discharges of carcinogens would be increased in each river.

Based on the analyses performed, the proposed change to using the harmonic mean flow as the critical design flow for determining wasteload allocations will result in significant increases in the amounts of carcinogens that are discharged to West Virginia waters. These increased discharges will result in increased cancer risks, the magnitude of which depends upon the particular carcinogen. For some carcinogens, the impact of the proposed rule change on risk of cancer may be small, for others the increase in risk of cancer may be many times the increase in discharge amount. The risk of cancer may be further increased if requests before the Board to increase the water quality standards for carcinogens such as dioxin are granted. Given these conclusions, it is prudent and appropriate that the West Virginia State Water Resources Board determine the increased quantities of carcinogens the proposed rule would allow to be discharged to all river segments in West Virginia and determine the increased cancer risk to West Virginia's citizens these discharges would represent for each carcinogen. With this information in hand, a fruitful public debate, weighing the human health impacts of the proposed rule can commence.

Please contact me if you have any questions or if we may provide any additional information.

Sincerely,

CARPENTER ENVIRONMENTAL
ASSOCIATES, INC.



Bruce A. Bell, Ph.D., P.E.
Sr. Vice President

BAB/mw

ALABAMA RIVER PULP COMPANY INC.



P. O. Box 100
Claiborne Mill
Perdue Hill, Alabama 36470
Phone (205) 575-2000

June 27, 1991

West Virginia State Water Resources Board
1260 Greenbriar Street
Charleston, West Virginia 25311

Dear Sir,

Our sister company, Apple Grove Pulp and Paper Company, Inc. submits the following documents for your consideration on the establishment of a water quality standard for 2,3,7,8 - Tetrachloro-Dibenzo-p-Dioxin:

- (1) Support Document for the Establishment of a Water Standard for Dioxin for the State of West Virginia

by: ChemRisk
A Division of McLaren/Hart
Stroudwater Crossing
1685 Congress Street
Portland, Maine 04102

- (2) West Virginia Ambient Water Quality Standard for 2,3,7,8-TETRACHLORO-DIBENZO-p-DIOXIN

by: Joseph V. Rodricks, Ph.D.,
Senior Vice President of
EVIRON Corporation
Arlington, Virginia

The two consulting firms conclude that a dioxin standard of 1.2 ppq is conservative and protective of human health. Apple Grove Pulp and Paper Company, Inc. respectfully requests that the West Virginia State Water Resource Board adopt 1.2 ppq as their dioxin standard.

Sincerely,

Donald B. Morris
Manager of Environmental Affairs

DBM:ab

Hand Deliver 7/28/91

RECEIVED

JUN 28 1991

WATER RESOURCES BOARD

U.S. official revises stand on dioxin

ST. LOUIS (AP) — A federal health official who recommended that Times Beach be evacuated because of contamination by the chemical dioxin now says he no longer views it as the deadly substance it was believed to be.

That change of opinion could influence pending or future lawsuits involving dioxin, parties to the suits say.

Dr. Vernon N. Houk of the Centers for Disease Control told the St. Louis Post-Dispatch that if he knew in 1982 what he knows now about the dangers of dioxin, he never would have recommended that the community in southwestern St. Louis County be evacuated.

"I would not be concerned about the levels of dioxin at Times Beach," said Dr. Houk.

Times Beach was abandoned in December 1982 and bought by the government. The cleanup of dioxin-tainted soil in the town began last month. The dioxin was contained in waste oil spread on streets to control dust.

Experts estimate that the cleanup at Times Beach, once a town of 2,242 people, will cost \$200 million by the time of completion in 2000.

Dioxins, a class of chemicals often formed as unwanted byproducts of chemical processes, once were considered by the U.S. Environmental Protection Agency to be "the most toxic man-made chemical."

But Dr. Houk now says that if dioxin is a carcinogen "it is, in my view, a weak one that is associated only with high-dose exposures."

Dr. Houk is an assistant surgeon general and director of the Center for Environmental Health and Injury Control at the CDC. He spoke Tuesday at the 25th annual Conference on Trace Substances in Environmental Health. Dioxin was a major topic at the conference.

Dr. Houk said studies now show dioxin in low doses to be a minimal health risk in humans. Thus, he said, the evacuation order was an overreaction.

Asked in an interview what he would tell Times Beach residents whose lives were uprooted by the order, Dr. Houk said:

"I would tell them when we originally got involved with you, we were very concerned about the substance. But the data we've accumulated in the last 10 or 12 years show we should not be that concerned — so for that, thank God.

"We should have been more upfront with Times Beach people and told them: 'We're doing our best with the estimates of the risk, but we may

"I think we never added 'but we may be wrong.'"

Dr. Houk said it is up to the EPA to decide whether to continue with the cleanup.

Karen Webb, director of the division of environmental and occupational health at the St. Louis University School of Medicine, said studies of Times Beach's residents found "no significant chronic illness."

"The data seem to point out that if TCDD [one type of dioxin] is a carcinogen at all, it is not a very potent one," she said.

Dr. Houk's comments could affect legal actions.

"I don't think this kind of decision [by Dr. Houk] can hurt," said Dan R. Bishop, director of corporate communications for Monsanto Co. Monsanto is appealing a \$16.25 million award to 65 plaintiffs in the town of Sturgeon, who sued the company for exposure to dioxin, a byproduct of its chemical manufacturing.

"It may serve to ease people's minds ... and potentially may be a deterrent against future lawsuits," Bishop said.

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MINI SUMMARY

WEST VIRGINIA AMBIENT WATER QUALITY STANDARD FOR 2,3,7,8-TETRACHLORO-DIBENZO-p-DIOXIN

Prepared by:

Joseph V. Rodricks, Ph.D.,
Senior Vice President of
ENVIRON Corporation
Arlington, Virginia

Dr. Rodricks conclusion was, "The discussion in the preceding sections demonstrates that the EPA recommendation about a water quality standard for TCDD is based on outdated information and substantially conservative assumptions. Using less conservative but supportable assumptions, we have concluded that a water quality standard in the vicinity of 20 pg/l (ppq) is fully supportable and protective of human health. Given the great disparity between 20 ppq and the EPA recommendation at 0.013 ppq, the standards in Alabama, Maryland, and Virginia at 1.2 ppq now themselves appear somewhat conservative. We urge West Virginia to use the TCDD data base fully in developing its final water quality standard for dioxin."

Basis of Calculation used:

Body weight - 70 Kg
Drinking water consumption - 1.5 L/Day
Fish consumption - 1 g/Day
Bioconcentration factor - 2 L/g

Donald B. Morris
Manager of Environmental Affairs

DBM:ab

**WEST VIRGINIA AMBIENT
WATER QUALITY STANDARD FOR
2,3,7,8-TETRACHLORO-
DIBENZO-p-DIOXIN**

Prepared for

Apple Grove Pulp and Paper Company, Inc.

Prepared by

Joseph V. Rodricks, Ph.D.
Senior Vice President
ENVIRON Corporation
Arlington, Virginia

June 25, 1991

CONTENTS

	<u>Page</u>
I. INTRODUCTION	1
II. CRITERION DOSE	4
A. EPA Non-Threshold Model	4
1. Risk Criterion	4
2. Slope Factor	5
B. Threshold Model	6
III. EXPOSURE ASSUMPTIONS	9
A. Body Weight	9
B. Consumption of Drinking Water	9
C. Consumption of Fish	9
D. Bioconcentration Factor	10
IV. CONCLUSIONS	13
V. REFERENCES	14

I. INTRODUCTION

The State of West Virginia is in the process of issuing an ambient water quality standard for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). The numeric standard to be issued will be designed to provide protection against potential health effects to people consuming TCDD-containing drinking water or fish and, at the same time, to protect aquatic resources. The U. S. Environmental Protection Agency (EPA) has been advising the states to set the numeric standard at 0.013 parts per quadrillion (ppq) in water (or more precisely, at 0.013 picograms per liter of water), based on a risk assessment developed in the early 1980s and a conservative risk acceptance factor. This concentration is not measurable by currently available techniques, and even discharge limitations calculable from the EPA proposal may not be measurable.

Although other sources of TCDD exist and may contribute the substantial majority of TCDD entering the general environment, one important source of TCDD in surface waters are the mills that bleach wood pulp with chlorine during papermaking. Parsons and Whittemore, Inc. is contemplating the construction of a bleached pulp mill on the Ohio River near Ashton, West Virginia, and so has a corporate interest in West Virginia's TCDD standard. Apple Grove Pulp and Paper Company requested ENVIRON Corporation to examine the recent science on TCDD and propose a more justifiable and achievable standard.

In laboratory animals, a wide variety of adverse effects related to TCDD exposures have been reported, including the development of tumors (Kociba 1978), effects on reproduction (Bowman et al. 1987) and development (Sparschu et al. 1971a,b) and on the immune system (Hong et al. 1987) as well as various forms of chronic organ toxicity (Kociba 1979, NTP 1982) and a generalized wasting syndrome (Allen et al. 1977). In humans, the effects of TCDD exposure are substantially uncertain. Although chloracne, a skin condition, has definitely been associated with human exposure to TCDD, the evidence for all other health effects is equivocal (Gough 1991). Because tumors were produced in laboratory animals treated with TCDD, several epidemiological investigations have been launched to see if exposure to TCDD can be linked with cancer in humans. Some of these studies have reported statistically significant relationships between TCDD exposure and various cancers (e.g., Fingerhut 1991), but the studies all suffer from various methodological problems, not least of which is the fact that exposures to TCDD usually occur in people who could have

been exposed to other chemicals that might be the causative agent. In any case, cancer is the effect of most interest in developing a water quality standard for TCDD, because protection against cancer should provide protection against all other effects of TCDD. In this document, therefore, our comments will refer principally to cancer as the endpoint of concern in risk assessment for TCDD and subsequent standards development.

A numeric water quality standard is intended to be a concentration in water that can be reached without adverse effects on human health or the environment. When the effect of concern is acute, it may be necessary to determine a short-term averaging time over which compliance with the standard must be met. For a substance potentially affecting the development of cancer in humans, however, the period of interest is long, effectively a lifetime, and the standard refers to the long-term average concentration that must be achieved.

For the standard to be health protective, it must assure with a reasonable degree of confidence that long-term average human exposures, expressed as daily doses, do not exceed dose criteria established from research on the dose-response relationship for the chemical in question, in this case TCDD. To derive a standard, then, we need a dose criterion for TCDD and the values of factors that connect the ambient water concentration of TCDD with the dose that could be received. These latter factors include body weight, daily consumption rates for food and drinking water, and the ratio of the concentration in edible fish tissues to the concentration in the water in which the fish live.

EPA's risk assessment for dioxin in surface water was conducted in 1984 and featured many choices that were reasonable at the time but are now being challenged as new information becomes available. EPA (1984) derived a criterion dose beginning with the assumption that TCDD's dose-response relationship was linear without threshold at low doses, meaning that any exposure to TCDD above zero would increase cancer risk (probability of developing cancer over a lifetime of exposure) in proportion to the amount of daily dose received. Under that concept of assessing cancer risk, no "safe" dose can be defined through science alone. Instead, EPA needed to select some risk criteria that expressed the Agency's degree of concern about the risks taken. EPA solved this issue by selecting three criteria (one in one hundred thousand or 10^{-5} , one in a million or 10^{-6} , and one in ten million or 10^{-7}) as spanning the range it was willing to grant to the states for setting water quality standards (EPA 1984).

The criterion dose can be derived from the risk criterion through dividing it by the slope factor for TCDD carcinogenicity, where the slope factor is the constant of proportionality between dose and risk in the low-dose region of the relationship that relates risk (response) to

dose. EPA usually calls such a number a "risk specific dose" or RSD. For TCDD, EPA's choice for slope factor is 156,000 in units of kilograms body weight (kg) times days per milligram (mg). Using 1 picogram (pg) = 10^{-9} mg, the RSD at the 10^{-6} risk level is 0.0064 pg/kg/day.

To translate a criterion dose or reference dose to an ambient water quality standard, EPA assumed that people on average weighed 70 kg, drank 2 liters (l) of water per day, and ate 6.5 g of fish per day. It further assumed that the ratio of TCDD concentrations in fish tissue to TCDD concentrations in ambient water was 5,000 l/kg. (The unusual units result from the conventional way of expressing concentrations in fish and water.)

With these assumptions, EPA calculated the water quality standard as a concentration according to the formula

$$C_{wqs} = RSD \times BW / (WC + FC \times BCF), \quad (1)$$

where C_{wqs} = concentration limit for the water quality standard in mg/l,
RSD = risk specific dose in mg/kg/day,
BW = body weight in kg,
WC = drinking water consumption in l/day,
FC = fish consumption in kg/day, and
BCF = bioconcentration factor in l/kg.

Substituting the values cited above and converting units properly, EPA derived a recommended water quality standard for TCDD of 0.013 pg/l or 0.013 parts per quadrillion (ppq). In the following sections, we discuss each of the factors that goes into the equation and propose alternative values for consideration by the West Virginia Water Resources Board.

II. CRITERION DOSE

Two alternatives to the EPA criterion dose of 0.0064 pg/kg/day for TCDD are proposed. The first alternative uses the basic EPA methodology but uses different values for the risk criterion and the slope factor. The second alternative abandons the linear non-threshold model favored by EPA and replaces with a threshold model consistent with all recent scientific findings about the mechanisms of toxic and carcinogenic action for TCDD.

A. EPA Non-Threshold Model

EPA assumes that any dose of TCDD higher than zero can raise the risk of cancer in exposed humans. It uses the so-called linearized multistage model to estimate the slope of the dose-response relationship in the low-dose region and then uses that slope factor with a risk criterion to derive a risk specific dose to be used as a criterion dose in the process of setting water quality standards.

1. Risk Criterion

The risk criterion used by EPA in its recommendations for a TCDD water quality standard is a one in a million (10^{-6}) lifetime risk. This value is the middle of three values for which EPA calculated water quality criteria in its 1984 document (EPA 1984). EPA also made it clear that it would approve standards based on any risk criterion within the range 10^{-7} to 10^{-5} .¹ On an arithmetic scale, 10^{-6} is near the lower end of the range and is not a mid value in the sense of an unbiased average.

We propose the alternative value of 10^{-5} as the risk criterion for TCDD if West Virginia chooses to utilize the linearized multistage model in its risk assessment for TCDD. This value has adequate precedent in other regulatory actions at both the state and federal levels and is particularly appropriate to use when other assumptions of the risk assessment are markedly conservative, as will shown to be the case for TCDD.

¹ Note that the Clean Water Act does not give EPA the authority to mandate even this range of risks, let alone a specific value from within the range. States may obtain EPA approval for their standards as long as they provide adequate scientific support.

A risk criterion of 10^{-5} has been used for the TCDD water quality standard in the neighboring states of Maryland, Ohio, and Virginia as well as in our sponsor's home state of Alabama. The standards in Maryland and Virginia are among the most recent TCDD standards approved by EPA (e.g., EPA 1991). EPA's own proposed drinking water standard of 50 ppq for TCDD (the MCL) corresponds to a risk criterion of 2.5×10^{-4} (EPA 1990). In California, the Safe Drinking Water and Toxic Enforcement Act uses the 10^{-5} cancer risk level as a trigger for enforcement of prohibitions against discharges to water and of requirements for warning (CHWA 1987).

Travis et al. (1987) have analyzed 137 federal decisions involving cancer risk and have shown that risks of up to 10^{-4} or even higher have been accepted under certain circumstances, as when the number of people at risk is small or the economic consequences of reducing risk are great, as would be the case for the TCDD standard in West Virginia. Even if we assumed that *all* West Virginians would be at maximum risk of TCDD-related cancer and that the risk estimates were accurate instead of conservative upper bounds, it is unlikely that even one person out of the 2 million West Virginians would be affected in any given year.

For all these reasons, a risk criterion of 10^{-5} is appropriate and protective of public health when applied to the water quality standard for dioxin.

2. Slope Factor

EPA uses a slope factor of 156,000 kg-day/mg derived from the tumor data reported by Kociba (1978) in female rats. In deriving this value, EPA applied a correction factor of about 1.7 for early mortality in the rats and projected from rats to humans using a scaling factor based on the assumption that rats and humans are as sensitive to TCDD if the dose is scaled by surface area. The Food and Drug Administration (FDA) also used the Kociba data as a starting point, but did not correct for early mortality and used a scaling factor based on the assumption that rats and humans are as sensitive to TCDD if the dose is scaled by body weight. These two differences yielded a slope factor approximately 9 times lower, at 17,500 kg-day/mg. The Centers for Disease Control (CDC) also used the Kociba data to derive a slope factor, but by a different method that yielded an estimate between the EPA and FDA values.

Both empirical evidence and theoretical considerations suggest that the body weight scaling rule is better for TCDD. Allen et al. (1987) analyzed the slope factors in humans and animals for all chemical carcinogens for which estimates of the slope factor were available in both humans and at least one animal species. Although both the surface area

rule and the body weight rule showed correlation between human and animal potencies, the body weight rule was superior in predicting human slope factors from animal data. For TCDD in particular, the likely mechanism of action for carcinogenesis involves a cytosolic receptor that would be distributed with volume and weight, not with surface area, and the body weight rule is also more attractive on theoretical grounds.

Alabama, Maryland, and Virginia have all adopted water quality standards for TCDD based on the FDA slope factor, and EPA has approved the standards with the comment that the FDA potency has sufficient scientific support. West Virginia would be consistent with two of its neighbor states and with a competing paper producing state if it adopted 17,500 as the slope factor.

An even lower slope factor is supported by the most recent scientific evidence. At the urging of the Maine Scientific Advisory Panel, a pathology working group organized by Pathco, Inc. reviewed the original Kociba slides using pathology classifications established in 1986 (Sauer 1990). With these classifications, many of the lesions counted as potential tumors by Kociba and later by EPA would not be so classified today. If the revised tumor count is used in the linearized multistage model, a slope factor for rat carcinogenicity at least 2 times lower than the original is generated. If we now adjust the EPA slope factor for this difference and for the body weight scaling rule for an overall adjustment of 10.8, the resulting slope factor is 14,400 kg-day/mg.² At the 10^{-5} cancer risk level, this slope factor produces a criterion dose of 0.7 pg/kg-day. We believe this value is the most supportable at the present level of scientific understanding if the non-threshold model of carcinogenesis is used for risk assessment.

B. Threshold Model

Even in 1987, when the association of TCDD with the bleached pulp industry had just become known, the understanding of the toxicity of TCDD was understood much more fully than in 1984 when EPA issued its water quality criteria document (EPA 1984). By that time, it had become evident that TCDD did not damage DNA, that is, it was not genotoxic. Experiments had shown that TCDD promoted tumors when administered after a known initiator of cancer, but did not promote tumors, or even provided protection against tumors, when administered before the initiator. Other evidence had accumulated about TCDD's interaction with the aryl hydrocarbon (Ah) receptor, which would stimulate enzyme

² Using the detailed pathology findings from the Pathco reanalysis, Keenan and his colleagues have suggested that a potency below 10,000 kg-day/mg can be supported (publication in press).

production only if a sufficient fraction of available receptor sites were occupied. In a report prepared for the National Council of the Paper Industry on Air and Stream Improvement (NCASI 1987), ENVIRON supported other scientist's conclusions to the effect that TCDD was likely to exhibit a threshold for carcinogenesis, a daily dose below which no risk of cancer whatsoever was likely to occur. At that time, we estimated (based on the no-effect level in the Kociba experiment and a safety factor of 100) that 10 pg/kg-day would be a protective dose level in humans.

Since that time, data have continued to accumulate in support of that position (NCASI 1989, ENVIRON 1989) and against the non-threshold hypothesis. The Pathco reanalysis (Sauer 1990) not only resulted in a lower tumor count but also found that all tumors occurred in rat livers that also showed signs of overt toxicity, implying that damage to the liver and subsequent cell proliferation, a threshold phenomenon, may be necessary for tumor development. The information base on dioxin toxicity has now become so extensive that in the fall of 1990, a group of internationally recognized TCDD experts met at the Banbury Dioxin Conference and concluded, with surprisingly little dissent, that a receptor-mediated threshold model best described what was known about TCDD (Roberts 1991). Scientists at the conference speculated that the threshold dose for humans would be in the range 1 to 10 pg/kg-day (see, e.g., Birnbaum 1990). Shortly thereafter, a group of scientists assembled by the World Health Organization (WHO 1990) issued a press release recommending that 10 pg/kg-day be used for regulatory purposes. Several countries other than the United States have been using the threshold model for some years (see Table 1). EPA has recently organized a working group to develop a new cancer model for TCDD that is likely to show a threshold (Reilly 1991).

Recently, a competing explanation for the toxicity of TCDD has been advanced by Rozman (1989) that invokes another enzyme system that controls the synthesis of glucose in the body. Although Rozman speculates that the Ah receptor may not be as important in TCDD toxicity as commonly thought, the "gluconeogenesis" mechanism would also produce a threshold behavior in the dose-response relationship.

Given the strength of evidence for the threshold model, West Virginia would be better served by adopting the threshold model than by wrestling with the twin difficulties of choosing a risk criterion and a slope factor for use with the non-threshold model. A criterion dose as high as 10 pg/kg-day can certainly be supported, and a criterion dose of 1 pg/kg-day should prove protective at a high level of confidence.

TABLE 1 Dioxin Regulation by Threshold Model	
Country/Province	Criterion Dose (pg/kg/day)
Ontario	10
Netherlands	4
Denmark	5
Germany	1-10
United Kingdom	1
Source: Carlo 1990	

III. EXPOSURE ASSUMPTIONS

Once a criterion dose has been selected, it must be translated to a numeric water quality standard expressed as a concentration. The principal quantities involved in this translation are the body weight for average humans, their typical consumption rates for drinking water and fish, and the ratio that relates the concentration in water to the concentration in edible fish tissues. The values for these quantities are discussed in this section.

A. Body Weight

Although body weight varies from person to person and for the same person with age, the uncertainty about the average body weight over a lifetime is relatively low. Because consumption of fish and water tend to correlate with body weight to some degree, the effect of the variation on risk is even smaller. Therefore the use of 70 kg for the average person is adequately accurate for the risk assessment of TCDD.

B. Consumption of Drinking Water

In most risk assessments of the 1980s, drinking water consumption has been assumed to be 2 l/day. Upon examination of the underlying data for this assumption, EPA (1989) has concluded that this figure is probably a reasonable maximum, while the average consumption is about 1.4 l/day. Because use of a TCDD-containing water supply as a sole source of water intake is rare, use of the 1.4 l/day figure is justified for setting a water quality standard for dioxin.

C. Consumption of Fish

EPA's fish consumption value was derived from a report by SRI (1980) that used data from the National Marine Fisheries Service to conclude that consumption of freshwater and estuarine fish and shellfish averaged 6.5 g/day. Because estuarine fish are not at risk of TCDD contamination in West Virginia, only the consumption of freshwater fish is relevant to the water quality standard. ENSR (1989) reanalyzed the SRI data and adjusted for increasing fish consumption in the 1980s. With these corrections, freshwater fish consumption in the Mid Atlantic and South Atlantic regions characteristic of West Virginia averaged 0.87 and 1.05 g/day, respectively. Furthermore, in a letter to the West Virginia Division of Natural

Resources, Miles (1991) estimates that on a stretch of the Ohio River near Ashton, even the regular fishermen would consume only 12 fish per year at a maximum. Therefore, a value of 1 g/day as an average is more supportable than 6.5 g/day. An additional margin of safety is provided by the fact that fish lose TCDD when cooked; the loss has been estimated to be 25% or greater (ENSR 1989).

D. Bioconcentration Factor

Based on limited experimental data available in 1984, EPA chose a value of 5,000 l/kg as a reasonable choice for the ratio between TCDD concentrations in fish and those in ambient water (EPA 1984). EPA described this ratio as a bioconcentration factor (BCF) because the experiments measured concentrations in fish in comparison to measured or computed concentrations in water as prepared by the experimenter.

Since that time, a contentious debate has emerged on the proper interpretation of laboratory and field data on the concentration of TCDD in fish tissues from a source in ambient water. Using data from highly contrived laboratory conditions and inedible or juvenile fish, and sometimes from measurements of whole fish rather than edible tissues (e.g., fillets), some scientists have claimed that the factor to be used in Eq. (1) should be much higher than 5,000, perhaps 50,000.

This claim has largely overlooked several important facts:

- if even a small load of suspended organic materials is present in the ambient water, the vast majority of the TCDD will be attached to the solids rather than dissolved in the water;
- many experimental values for "BCF" have been derived in systems with very low suspended solids in comparison to typical ambient waters;
- most of the TCDD in a fish will have arisen through feeding and intake of solids (described by the bioaccumulation factor, BAF) instead of through the gills as is technically the case for a BCF;
- fish with low lipid (fat) content will not accumulate as much TCDD as will fish with higher lipid content;

- systems for measuring BCFs or BAFs may not be in equilibrium and therefore may underestimate or overestimate the steady-state ratios; and perhaps most importantly
- compliance with a water quality standard for TCDD will be monitored by assaying total TCDD in effluents, including TCDD on suspended solids as well as the minor fraction dissolved in water.

These facts have been taken into account by Sherman (1991) in a reanalysis of several existing studies of bioaccumulation of TCDD in fish. When normalized to a lipid content of 2.5% in fish and adjusted for suspended solids and other factors, virtually all of the "regulatory BAFs" are at or below 5,000 l/kg. According to a West Virginia survey conducted by the State Department of Natural Resources cited in the submission by the West Virginia Chamber of Commerce and other parties, typical fish caught for consumption show lipid content of around 1%, implying that the value to be used for "BCF" in Eq. (1) should be about 2,000 l/kg or equivalently 2.0 l/g. Without site-specific fish and water concentration measurements, the State would be justified in using this value in setting the TCDD standard.

Eq. (1) was developed specifically for use with the nonthreshold model but can be generalized to represent the threshold model by replacing the RSD by the generalized criterion dose, CD:

$$C_{wqs} = CD \times BW / (WC + FC \times BCF). \quad (2)$$

Using the values of 70 kg for body weight, 1.4 l/day for water consumption, 1 g/day for fish consumption, 2 l/g for BCF, and either 0.7 pg/kg day (nonthreshold model) or 1.0 pg/kg/day (threshold model) for the criterion dose, the water quality standard for dioxin becomes either

$$C_{wqs} = 0.7 \times 70 / (1.4 + 1.0 \times 2.0) = 15 \text{ pg/l or} \quad (3)$$

$$C_{wqs} = 1.0 \times 70 / (1.4 + 1.0 \times 2.0) = 21 \text{ pg/l,} \quad (4)$$

where the lower end of the threshold range has been used. Note that using the upper end of the threshold range (10 pg/kg-day) with the standard EPA assumptions about exposure also results in a similar standard:

$$C_{wqs} = 10 \times 70 / (2.0 + 6.5 \times 5.0) = 20 \text{ pg/l.} \quad (5)$$

IV. CONCLUSIONS

The discussion in the preceding sections demonstrates that the EPA recommendation about a water quality standard for TCDD is based on outdated information and substantially conservative assumptions. Using less conservative but supportable assumptions, we have concluded that a water quality standard in the vicinity of 20 pg/l (ppq) is fully supportable and protective of human health. Given the great disparity between 20 ppq and the EPA recommendation at 0.013 ppq, the standards in Alabama, Maryland, and Virginia at 1.2 ppq now themselves appear somewhat conservative. We urge West Virginia to use the TCDD data base fully in developing its final water quality standard for dioxin.

V. REFERENCES

- Allen, B.M., A.M. Shipp, K.S. Crump, B. Killian, M.L. Hogg, J. Tudor, and B. Keller. 1987. Investigation of cancer risk assessment methods: Summary. Prepared by Clement Associates, Inc. for the U.S. Environmental Protection Agency. EPA/600/6-87/007a. Washington, DC: EPA.
- Allen, J.R., D.A. Barsotti, J.P. Van Miller, L.J. Abrahamson, and J.J. Lalich. 1977. Morphological changes in Monkeys consuming a diet containing low levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Fd. Cosmet. Toxicol.* 15:401-410.
- Agency of Toxic Substances and Disease Registry (ATSDR). 1988. Toxicological profile for 2,3,7,8-tetrachlorodibenzo-p-dioxin.
- Birnbaum, L.S. Consensus at Banbury dioxin conference. Memorandum to Erich Bretthauer, EPA Assistant Administrator for Research and Development. October 31.
- Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross, and D.A. Barsotti. 1987. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 and 25 parts per trillion in the monkey: TCDD kinetics and dose-effect estimate of reproductive toxicity. Presented at Dioxin '87: Seventh International Symposium on Chlorinated Dioxins and Related Compounds, October 4-9, Las Vegas. (Abstract).
- California Health and Welfare Agency (CHWA). 1987. No significant risk levels. Article 7 of Safe Drinking Water and Toxic Enforcement Act of 1986, Chapter 3. 22 Calif. Code of Regulations Division 2. Sacramento, CA: Health and Welfare Agency.
- Carlo, G.L. 1990. Letter to Harold F. Reheis, Georgia Department of Natural Resources. December 18.
- DeCaprio, A.P., D.M. McMartin, P.W. O'Keefe, R. Rej, J.B. Silkworth, and L.S. Kaminsky. 1986. Subchronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the guinea pig: Comparisons with a PCB-containing transformer fluid pyrolysate. *Fund. Appl. Toxicol.* 6:454-463.
- ENSR Consulting and Engineering (ENSR). 1989. Technical support document for a proposed Alabama water quality standard for dioxin. Boston, MA: ENSR.
- ENVIRON Corporation. 1989. An update on the literature on dioxin. Washington, DC: ENVIRON Corporation.

Environmental Protection Agency (EPA). 1984. Ambient water quality criteria for 2,3,7,8-tetrachlorodibenzodioxin. EPA-440/5-84-007. Washington, DC: EPA.

Environmental Protection Agency (EPA). 1989. Exposure factors handbook. EPA/600/8-89/043. Washington, DC: EPA.

Environmental Protection Agency (EPA). 1990. Proposed MCLs for synthetic organic chemicals. Fed. Reg. 55:30425 (July 25).

Environmental Protection Agency (EPA). 1991. EPA approves Virginia dioxin standard. EPA Environmental News, Region 3. February 25.

Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piacitelli, P.A. Honghar, M.H. Sweeney, A.L. Greife, P.A. Dill, K. Steenland, and A.J. Suruda. 1991. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. New England J. Med. 324:212-218.

Gough, M. 1991. Human health effects: What the data indicate. Ambio. In press.

Hong, R., K. Taylor, and R. Abonour. 1987. Immune abnormalities associated with chronic TCDD exposure in rhesus monkeys. Presented at Dioxin '87: seventh International Symposium on Chlorinated Dioxins and Related Compounds, October 4-9, Las Vegas. (Abstract).

International Commission on Radiological Protection (ICRP). 1984. Report of the task group on Reference Man. ICRP Publication No. 23. New York: Pergamon Press.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreson, C.E. Wade, D.A. Dittenber, P.O. Kalmins, L.F. Franson, P.N. Park, S.D. Barnard, P.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-TCDD in rats. Toxicol. Appl. Pharmacol. 46:279-303.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreson, and P.J. Gehring. 1979. Long-term toxicologic studies of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in laboratory animals. Ann. NY Acad. Sci. 320:397-404.

Miles, Bob. 1991. Memorandum to Director Hamrick regarding fish consumption near the proposed Ashton pulp plant. June 10, 1991.

National Council of the Paper Industry for Air and Stream Improvement (NCASI). 1987. Dioxin: A critical review of its distribution, mechanism of action, impacts on human health, and the setting of acceptable exposure limits. Technical Bulletin No. 524. New York, NY: NCASI.

- National Council of the Paper Industry for Air and Stream Improvement (NCASI). 1989. Biological risk assessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin. Technical Bulletin No. 567. New York, NY: NCASI.
- National Toxicology Program (NTP). 1982. Bioassay of 2,3,7,8-tetrachlorodibenzo-p-dioxin for possible carcinogenicity (gavage study). DHHS Publ. No. (NIH) 82-1765.
- Reilly, W.K. 1991. Dioxin: Follow-up to briefing on scientific developments. Memorandum to Erich W. Bretthauer and E. Donald Elliott. April 8.
- Rodricks, J.V., S.M. Brett, and G.C. Wrenn. 1987. Significant risk decisions in federal regulatory agencies. Reg. Toxicol. Pharmacol. 7:307-320.
- Roberts, Leslie. 1991. Dioxin risks revisited. Science 251:624-626. February 8.
- Rozman, K. 1989. A critical view of the mechanism(s) of toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Implications for human safety assessment. Dermatosen in Beruf und Umwelt 37:81-92.
- Sauer, Robert M. 1990. Pathology Working Group: 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Sprague-Dawley rats. Report prepared by PATHCO, Inc. for the Maine Scientific Advisory Panel. March 13.
- Sherman, William R. 1991. Letter to Philip M. Cook, US Environmental Protection Agency dated April 22. S.D. Warren Company.
- SRI International (SRI). 1980. Seafood Consumption Data Analysis. Report prepared by SRI International for the U.S. Environmental Protection Agency. Menlo Park, CA: SRI.
- Sparschu, G.L., Jr., F.L. Dunn Jr., and V.K. Rowe Jr. 1971a. Study of the teratogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the rat. Fd. Cosmet. Toxicol. 9:405-412.
- Sparschu, G.L., F.L. Dun, R.W. Lisowe, and V.K. Rowe. 1971b. Effects of high levels of 2,4,5-trichlorophenoxyacetic acid on fetal development in the rat. Fd. Cosmet. Toxicol. 9:527-530.
- Squire, R.A. 1990. Letter from Robert A. Squire to Robert A. Michaels, RAM TRAC Corporation, dated January 8.
- Travis, C.C., S.A. Richter, E.A.C. Crouch, R. Wilson, and E.D. Klema. 1987. Cancer risk management: A review of 132 federal regulatory decisions. Environ. Sci. Technol. 21:415-420.

Universities Associated for Research and Education in Pathology, Inc. (UAREP). 1988.
Human health aspects of environmental exposure to polychlorinated dibenzo-p-dioxins and
polychlorinated dibenzo furans. Bethesda, MD: UAREP.

World Health Organization (WHO). 1990. Working Group on Dioxin. Press Release.
December 7.

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ESTABLISHED 1802

E. I. DU PONT DE NEMOURS & COMPANY

INCORPORATED

BELLE, WEST VIRGINIA 25015

AGRICULTURAL PRODUCTS DEPARTMENT

BELLE PLANT, 901 W. DU PONT AVENUE

June 27, 1991

WV Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Dear Sir:

Comments on the proposed revisions to the West
Virginia Quality Standards are attached for your consideration.

Sincerely,

R. F. SHERMAN
ENVIRONMENTAL COORDINATOR

RFS/jss
3.34

JUNE 28, 1991

SUBMITTED TO

WEST VIRGINIA WATER RESOURCES BOARD

COMMENTS ON PROPOSED REVISIONS TO
WEST VIRGINIA WATER QUALITY STANDARDS

SUBMITTED BY

E. I. DU PONT DE NEMOURS & CO.

RECEIVED

JUN 28 1991

WATER RESOURCES

o Section 46-1-4.1.3 Anti-degradation Policy

The proposed language requires full intergovernmental coordination as part of the process in proposing lower water quality of a stream. However, we feel this section should explicitly identify all federal/state/local government agencies to ensure that all appropriate governmental agencies are involved. If left as is, a governmental agency which felt that it should have been part of the coordination but was not included may disrupt the water quality review process and cause excessive delays.

o Section 46-1-5 Mixing Zones Specifically 5.2.a.4

The proposed language to be added to Subsection 5.2.a.4 "Mixing Zones shall:.....nor exhibit acute toxicity to aquatic organisms"; is overly restrictive. Existing regulations empower the state to appropriately size acute mixing zones for wastewater discharges. Thus, there is no need to add additional regulatory constraints to limit the permittee and the permitting regulatory authority in establishing environmentally acceptable acute mixing zones.

It appears that the new language will require that discharges pass an acute toxicity test of 100% effluent at the end of pipe. This does not allow flexibility for site and facility specific conditions such as the acute toxicity of the effluent, discharge velocity, instream-dilution, geometric configuration of the stream or outfall which may affect mixing, designated use of stream and other factors.

The recently published Environmental Protection Agency's "Technical Support Document (TSD) for Water Quality-based Toxics Control" (MARCH 1991) discusses the use and criteria for establishing mixing zone boundaries. The TSD discusses some of the relevant site specific criteria which may need to be considered in establishing an area of initial dilution and mixing zone. The initial dilution is an area within the mixing zone for which neither acute or chronic toxic criteria apply. However, if promulgated as proposed, Section 46-1-5 additions could effectively prohibit use of areas of initial dilution and eliminates any flexibility for sizing of acute mixing zones for wastewater treatment systems and/or install a diffuser with very little corresponding environmental benefit.

o Section 46-1-6 Water Use Categories

The proposed language states "Unless otherwise designated by these rules, at a minimum all waters of the State are designated for the Propagation and Maintenance of Fish and Other Aquatic Life (Category B) and Water Contact Recreation (Category C) consistent with Clean Water Act goals. When a discharge permit is to be issued all uses shall be assumed present unless the applicant demonstrates that the designated uses do not apply to the stream segment in question".

The intent of the proposed language is not totally clear. The State generates several reports such as the 305(b) report, Section 304(a) report, and Section 319 Nonpoint Source report and collects data through the State's primary and secondary monitoring stations (STORET data base) on its streams which the state should be able to utilize in determining if the stream segment meets its standards.

Is it the intent to allow flexibility for a discharger, above and beyond the states evaluation to demonstrate that a designated use does not apply? Does a use attainability study need to be done if a designated use is not maintained? Additional clarification is needed on how the State plans to implement the above.

Appalachian Power Company
PO Box 2021
Roanoke, VA 24022-2121
703 985 2300



OVERNIGHT

David E. Samuel, Ph.D., Chairman
West Virginia Water Resources Board
1206 Greenbrier Street
Charleston, West Virginia 25311

RECEIVED

JUN 28 1991

June 27, 1991

WATER RESOURCES BOARD

Dear Dr. Samuel:

In response to the Public Notice issued May 17, 1991, regarding the West Virginia Water Resources Board's intention to amend and revise regulations entitled, 46 CSR Title 46 Legislative Rule - Series 1, Requirements Regarding Water Quality Standards, Appalachian Power Company offers the following comments.

General Issues

Appalachian Power and American Electric Power participated in the development of the comments to be submitted on behalf of the West Virginia Chamber of Commerce, et al. We recommend that the Board give these comments due consideration.

Sections 2.8 and 7.3.d - Outstanding State Resource Waters:

The amendments to these two sections apparently only involve the addition of the word "Outstanding" to the category previously termed "National Resource Waters." However, if this is intended to conform the state's treatment of waters so designated to the EPA policy regarding waters listed as Outstanding Natural Resource Waters, the effects could be far-reaching.

EPA policy regarding Outstanding National Resource Waters is stated at 40 CFR 131.12(a)(3):

"Where high quality waters constitute an outstanding National resource, such as waters of National and State parks and wildlife refuges and waters of exceptional recreational or ecological significance, that water quality shall be maintained and protected."

This contrasts with 40 CFR 131.12(a)(2), which deals with "high quality" waters in that in Section 3 there is no allowance for the regulatory agency to permit any lessening of water quality for any reason. This is usually interpreted as equivalent to a ban on new or increased discharges. Once designated, the state loses the authority to determine that important social and

Dr. David E. Samuel
June 27, 1991
Page Two

economic development outweighs the strict preservation of water quality in such a water body. The Board can not grant a variance to this requirement.

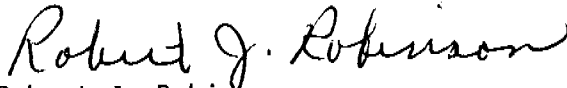
Since the inclusion in this category places a stream in an essentially untouchable category and can have significant economic impact, the Board should carefully consider whether it wishes to automatically extend EPA's Tier III designation to all streams designated according to Sections 2.8 and 7.3.d.

Appendix C - Category E-3 Power Production:

On page 1 of the appendix, the John E. Amos Power Plant is listed as being located in Kanawha County. This should be listed as Putnam County.

We appreciate this opportunity to comment and look forward to continued cooperation with the Board.

Sincerely,

A handwritten signature in cursive script that reads "Robert J. Robinson".

Robert J. Robinson
Environmental Affairs Director

RJR:d



West Virginia-American Water Company

P.O. Box 1906 • Charleston, WV 25327 • (304) 345-3474

Thomas W. Holbrook
Operations Manager -
Production & Water Quality

June 28, 1991
File No. 382

RECEIVED

JUN 28 1991

Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

WATER RESOURCES BOARD

Dear Board Members:

The West Virginia-American Water Company is an investor owned water utility providing service to 120,000 customers in thirteen (13) districts throughout the state. This corresponds to a population of about 375,000 which represents about 21% of the residential population within the State of West Virginia.

The Water Company's goal is to provide the best quality water at the most reasonable cost. It is from that viewpoint that we present the following comments with regard to the new/revised stream standards under the Code of West Virginia, Chapter 20-5A.

West Virginia-American Water Company provides quality water through strict adherence to the water quality regulations as promulgated by the U.S. Environmental Protection Agency and administered by the State of West Virginia which has accepted primacy for these regulations.

The U.S. EPA in its revisions to the Safe Drinking Water Act have promulgated Maximum Contaminant Levels (MCL's) and Maximum Contaminant Level Goals (MCLG's) for a number of organic and inorganic parameters for the protection of the health of water consumers. The U.S. EPA believes that the health data is sufficient to support the establishment of these MCL's and MCLG's.

With respect to the issue of dioxin, the U.S. EPA, in its Phase V SOC/IOC Rule published January 25, 1990, has proposed an MCLG of ZERO for this compound based on the strong evidence of carcinogenicity. In addition, an MCL in drinking water of 50 parts per quadrillion (ppq) was proposed for this compound. The final Phase V Rule and final regulatory limit for dioxin is expected to be promulgated in May, 1992.

Also, the Phase II SOC/IOC Rule, finalized by the EPA on January 30, 1991, contains a requirement that water utilities begin monitoring for dioxin in drinking water for surveillance purposes.

The West Virginia-American Water Company is concerned about the upstream discharge of dioxin and other contaminants into the Ohio River since it operates the water treatment facility at Huntington which provides drinking water to nearly 100,000 persons in and about the Huntington area. The source of water for this treatment facility is solely the Ohio River.

The proposed paper mill site in Mason County is just 25 miles upstream from the Huntington water intakes and has the potential to degrade source water quality. Both the water treatment plant, and the proposed paper mill, lie in the same pool system behind the Greenup Locks and Dam. As hydroelectric facilities are added along the Ohio River, the tendency to pool or store water behind these dams becomes greater and river flows may be affected and further degradation of the stream is possible. As new manufacturing facilities are located along the Ohio River and limits are set for stream discharges, one cannot assume that these limits will be met at all times; thus, further degradation of the stream is possible.

Water plant operating costs may increase if additional treatment is necessary of if levels of contaminants build up in the granular activated carbon filter media or in the treatment residuals which accumulate in the water plant waste lagoons. Increased laboratory analysis using sophisticated equipment would be necessary to monitor both source and finished water conditions for any discharged contaminants upstream.

All these additional costs would be borne by our customers throughout the state and could potentially be borne by other water utility customers downstream who must also use the Ohio River as a source water for their drinking water supplies.

A second concern is the proposal to base stream standards on harmonic mean flows in the streams. Due to the changing weather patterns over the past ten years, many parts of the state have experienced dry conditions and low stream flows.

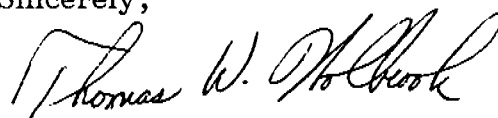
Unless the stream standards are set at zero for those compounds, such as dioxin, which are regulated or promulgated for regulation by the U.S. EPA and for which MCLG's or proposed MCLG's are set at zero; then the Water Company feels that it is inappropriate to use the harmonic mean flow. By retaining the use of the ten year low flow (7Q 10 flow) the greatest protection would be afforded the water utility customers who must use state streams as drinking water supplies.

The West Virginia-American Water Company, therefore, urges the members of the State Water Resources Board to set stream standards at zero for all potentially carcinogenic compounds for which the U.S. EPA maintains an MCLG of zero. For all other contaminants, the stream standards should be of levels consistent with the U.S. EPA Safe Drinking Water Regulations and at levels in the best interests of the

Page 3.

water utilities, and their customers throughout the State of West Virginia. Where technology in manufacturing is available to produce the lowest discharge levels of contaminants, or even eliminate discharges altogether, the stream standards should be set by the Board at the lowest possible levels to encourage the use of the most environmentally conscious technology.

Sincerely,

A handwritten signature in cursive script, reading "Thomas W. Holbrook". The signature is written in dark ink and is positioned above the typed name.

Thomas W. Holbrook
Operations Manager, Production

TWH:rp

cc: T. Jones, Jr.
M. Bugher
J. Perry
J. Routt

JUNE 28, 1991

SUBMITTED TO

WEST VIRGINIA WATER RESOURCES BOARD

COMMENTS ON PROPOSED REVISIONS TO
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o Section 46-1-5 Mixing Zones Specifically 5.2.a.4

The proposed language to be added to Subsection 5.2.a.4 "Mixing Zones shall:.....nor exhibit acute toxicity to aquatic organisms"; is overly restrictive. Existing regulations empower the state to appropriately size acute mixing zones for wastewater discharges. Thus, there is no need to add additional regulatory constraints to limit the permittee and the permitting regulatory authority in establishing environmentally acceptable acute mixing zones.

It appears that the new language will require that discharges pass an acute toxicity test of 100% effluent at the end of pipe. This does not allow flexibility for site and facility specific conditions such as the acute toxicity of the effluent, discharge velocity, instream-dilution, geometric configuration of the stream or outfall which may affect mixing, designated use of stream and other factors.

The recently published Environmental Protection Agency's "Technical Support Document (TSD) for Water Quality-based Toxics Control" (MARCH 1991) discusses the use and criteria for establishing mixing zone boundaries. The TSD discusses some of the relevant site specific criteria which may need to be considered in establishing an area of initial dilution and mixing zone. The initial dilution is an area within the mixing zone for which neither acute or chronic toxic criteria apply. However, if promulgated as proposed, Section 46-1-5 additions could effectively prohibit use of areas of initial dilution and eliminates any flexibility for sizing of acute mixing zones for wastewater treatment systems and/or install a diffuser with very little corresponding environmental benefit.

o Section 46-1-6 Water Use Categories

The proposed language states "Unless otherwise designated by these rules, at a minimum all waters of the State are designated for the Propagation and Maintenance of Fish and Other Aquatic Life (Category B) and Water Contact Recreation (Category C) consistent with Clean Water Act goals. When a discharge permit is to be issued all uses shall be assumed present unless the applicant demonstrates that the designated uses do not apply to the stream segment in question".

The intent of the proposed language is not totally clear. The State generates several reports such as the 305(b) report, Section 304(a) report, and Section 319 Nonpoint Source report and collects data through the State's primary and secondary monitoring stations (STORET data base) on its streams which the state should be able to utilize in determining if the stream segment meets its standards.

Is it the intent to allow flexibility for a discharger, above and beyond the states evaluation to demonstrate that a designated use does not apply? Does a use attainability study need to be done if a designated use is not maintained? Additional clarification is needed on how the State plans to implement the above.

Subject: Proposed amendments and revisions to Series 1 of the
Legislative Rules "Requirements Governing Water
Quality Standards"

From: Dr. William C. Beard, Ph.D.

To: West Virginia Water Resources Board
1260 Greenbrier St.
Charleston, WV 25311

The following comments relate to the request of several organizations (1) that a risk factor of 1×10^{-5} and a water quality standard of 1.2 ppq for dioxin (2,3,7,8-tetrachlorodibenzo-para-dioxin) be adopted.

I am especially concerned about evidence cited in support of the request as contained in the text (p. 4) and Appendix (pp. 4,5). In both citations, reference was made concerning a "consensus" having been reached about the levels of safe doses and carcinogenicity of dioxin at a recent Banbury Conference in October 1990, in Cold Spring Harbor, NY.

Attached documents (2,3) speak to the "trans-scientific objectives" of the organizers and sponsors of the conference and the way in which communication was done--through the public relations firm representing the Chlorine Institute.

Several participants in the conference refuted statements concerning agreement in terms of risk assessment and that dioxin is much less toxic to humans than originally believed.

The use of such materials in support of requests for laxer standards calls into question, in my mind, the soundness of other evidence cited. Therefore, I urge that the Water Resources Board establish a

1 Supplemental Proposals for Revisions to West Virginia Water Quality Standards, submitted on behalf of West Virginia Chamber of Commerce, West Virginia Manufacturers Association, West Virginia Oil and Gas Association, Independent Oil and Gas Association of West Virginia and West Virginia Farm Bureau, March 22, 1991.

2 Letter from Ellen Silbergeld to Jan Witkowski, Director, Banbury Center, January 29, 1991.

3 "A Press Release Sets the Record Wrong," Scientific American, 264 (4) 24, April 1991.

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concentration limit of 0.013 ppq TCDD.

Furthermore, regarding the achievability of permit levels, the EPA states, (4) "EPA has no convincing evidence that the industry will be unable to comply with appropriate permit limits based on risk levels of $10E-5$ or $10E-6$ by the 1992 and 1993 statutory deadlines of section 304 (1). Therefore, it is inappropriate and premature to assume at this time that the statutory deadlines are unachievable" (emphasis added).

Also, I propose that the total 2,3,7,8-TCDD toxic equivalency of all dioxins and furans present in surface water effluent be measured in applying the 0.013 ppq water quality standard.

In conclusion, I again urge that the Water Resources Board adopt a risk assessment factor of $1 \times 10E-6$ and a water quality standard of 0.013 ppq TCDD.

Respectfully submitted,

William C. Beard
William C. Beard, Ph.D.
Rt. 1, Box 260-101
Gallipolis, OH 45631
(614) 367-0593

Enclosures: 2

Date: 26 June 1991

4 EPA Memorandum: "State Policies, Water Policies Standards and Permit Limitations Related to 2,3,7,8-TCDD in Surface Water" from LaJuana S. Wilcher, EPA Assistant Administrator to Water Management Division Directors, EPA, January 5, 1990.



UNIVERSITY OF MARYLAND AT BALTIMORE

The University Program in Toxicology
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Baltimore, Maryland 21201-1596
(301) 328-8196

January 29, 1991

Dr. Jan Witkowski
Director
Banbury Center
Cold Spring Harbor Laboratory
P O Box 534
Cold Spring Harbor, NY 11724

Dear Dr. Witkowski:

I was a participant in the recent Banbury Conference on "Biological Basis for Risk Assessment of Dioxins and Related Compounds" held at the Banbury Center in October 1990. I am writing you because I have just been informed of a very disturbing result of that conference; a press release sent out by a public relations firm along with statements by Drs Scheuplein, van der Heiden, and Gallo purporting to represent the "consensus" views of the participants at that conference with respect to regulatory conclusions related to risk assessment of dioxins. I only learned of this press release from a reporter who called me last week (Marguerite Holloway of Scientific American).

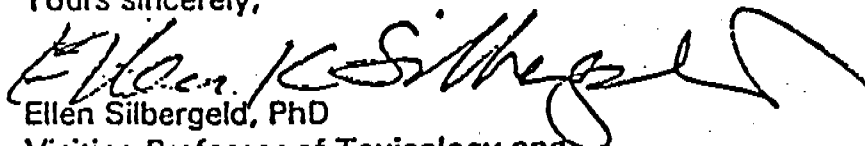
This press release, copy enclosed, was never shown to me or to most of the participants in the conference, as far as I know. Thus, in terms of process alone, it should not be represented as a "consensus" document. Moreover, its contents do not accurately reflect the views of all participants, or even a consensus of those views, as best I can determine. I resent the circulation of this press release as reflecting the views of a meeting in which I was a participant, and I feel that my name attached to it somehow implies my agreement with it.

I am in fact rather astounded by such a product from a Banbury Conference. While it was rather obvious to some of us that the organizers, and some of the sponsors, of this conference had some trans-scientific objectives in mind related to regulations concerning dioxin, I had expected that the Banbury Center would be able to keep these motives under control. The press releases and statements imply that a major focus of the conference was a discussion of the regulatory risk assessments that have been applied to the dioxins; this was not the focus of this meeting. I agreed to participate based upon my previously held high regard for Banbury and Cold Spring Harbor. I did not expect to be manipulated by industry and government spokespeople

(who are not dioxin researchers, incidentally) to be made into a supporter of their political views on dioxins and risk assessment. This is particularly annoying to me because I was invited to present the main conference paper on the topic of the scientific basis for dioxin risk assessment. In this paper, I have attempted to present the complexity of integrating the basic molecular biology of dioxins into a receptor-based model. I do not feel that the state of knowledge on this complex topic can be reduced to a simplistic press release.

The preparation and release of these documents by Drs Scheuplein, van der Heijden, Carlo, and Gallo, with the assistance of a public relations firm, discredits all of us. It challenges the precious institution of free scientific discussion, epitomized by such places as Banbury, Dahlem, and the Gordon conferences. I hope you believe that I would be just as angry if this action had been taken by an environmental group. I trust you will take action to dissociate Banbury from this attempt to manipulate science and scientists. Because these people have acted without consulting the rest of us, and because I have heard about this only through the press, I am with great regret also sending this letter to the persons shown under my signature, as well as to my colleagues at the conference, an action not taken by these people.

Yours sincerely,



Ellen Silbergeld, PhD

Visiting Professor of Toxicology and
Adjunct Professor of Pharmacology
and Experimental Therapeutics

cc: Leslie Roberts, Science
Marguerite Holloway, Scientific American
Cristine Russell, Washington Post
Chris Joyce, New Scientist
Judy Randall, The Economist
Betty Mushak, NIEHS
William Farland, EPA

attendees, Banbury Conference on Dioxins

warfare might become obsolete any way—if the tentative spread of democracy worldwide continues.

These conclusions are both supported by the Correlates of War project, a

war, is one of the most frequent precursors of it. At the very least, Singer says, such a finding suggests that the U.S. policy of supplying arms to na-

tistical correlation between warfare in a given region and prior unchecked aggression, Singer says.

A somewhat more hopeful finding

A Press Release on Dioxin Sets the Record Wrong

When the Chlorine Institute shopped around for a place to hold a scientific conference, they did not want just any host. "We were looking for an organization that was squeaky clean, that would not in any way, shape or form be questioned about the conference," says Robert G. Smerko, president of the Washington, D.C.-based institute, which is supported by some 170 chemical, paper and other manufacturers.

Smerko seemed to have met his requirements when he finally landed Cold Spring Harbor Laboratory. Last October the laboratory's respected Banbury Center held a conference—jointly sponsored by the Chlorine Institute and the Environmental Protection Agency—on the toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, or TCDD. That chlorinated compound achieved notoriety during the Vietnam War, when it was identified as a contaminant of the defoliant Agent Orange. It remains controversial because it is found in some commercial herbicides and is produced in other chemical processes, such as paper bleaching.

Cold Spring Harbor Laboratory may have been squeaky clean, but the conference apparently was not. And the outcome of that meeting—attended by 38 of the world's dioxin experts, few of whom say they knew it was industry sponsored—is every bit as controversial as the substance that was the topic of discussion.

The issue is a press release sent out at the conclusion of the meeting by the Chlorine Institute's public relations firm, Daniel J. Edelman, Inc. It announced that the experts had agreed on a model for the toxicity of dioxin that "allows for the presence of a substance in the environment, with no risk experienced below a certain level of exposure." The release said that the scientists had rejected a linear exposure model, in which any level of exposure would have a biological effect, in favor of a receptor-based model that implies a threshold level. (This part of the release was approved by Cold Spring Harbor Laboratory, says the Banbury Center's director, Jan A. Witkowski—although he now says Edelman made several changes after he saw it.)

Such a consensus, of course, would have implications for setting permissible levels of the substance in the environment. But those at the conference insist that no such

agreement was reached. "There was no consensus in terms of risk assessment," says George W. Lucier of the National Institute of Environmental Health Sciences. In addition, none of the scientists saw the press release, although their names accompanied it. "We were being used, clearly, and that's unfortunate," declares Arnold J. Schechter, professor of preventive medicine at the State University of New York at Binghamton. "Political layering is not particularly good, especially when it is unbeknownst," Lucier adds.

Few of the participants seem to dispute that the receptor-based mechanism of dioxin is relevant to human exposure. Nor did they before the conference, observes Alan P. Poland of the University of Wisconsin at Madison, who discovered the receptor in 1976. "The basic tenets were all known since 1981 or 1982," Poland says. But Lucier notes that now "we are at the point where we can reevaluate the linear model."

Indeed, the EPA intends to explore the question of whether there is a threshold response. The agency will investigate the receptor-based model with Michael A. Gallo, one of the conference organizers and a professor of toxicology at the University of Medicine and Dentistry of New Jersey—Robert Wood Johnson Medical School. But Gallo and others agree that discussion of thresholds in a regulatory context may be premature. At the conference, "some regulators got real excited by back-of-the-envelope calculations" and thought dioxin standards could be eased, says Linda S. Birnbaum, director of the EPA's environmental toxicology division. "Clearly, we don't know that."

Although many of the Banbury attendees were the last to know about the consensus they reportedly reached, news about the conference traveled quickly in political circles. At a recent hearing on dioxin standards in Alabama, expert witness for the pulp and paper industry Russell E. Keenan invoked the Banbury results in his testimony. "There was general agreement among the attending scientists that dioxin is much less toxic to humans than originally believed," Keenan claimed. Obviously, "it is not useless to tout Banbury results if you have a political ax to grind," comments Cate Jenkins, a chemist in the EPA's hazardous waste division.

—Marguerite Holloway



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Region III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

JUN 27 1991

Dr. Jan Taylor
Technical Advisor
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Dear Dr. Taylor:

Enclosed are comments on the proposed revisions to West Virginia's Water Quality Standards which were made available for public comment on May 17, 1991. We commend the State Water Resources Board for proposing revisions regarding narrative biological criteria, antidegradation, and wetlands.

We appreciate the opportunity to comment on these revisions and look forward to receiving the final, amended Water Quality Standards for approval. If you have any questions concerning these comments, please feel free to contact Ms. Linda Holst of my staff at (215) 597-0133.

Sincerely,

Victoria P. Binetti

Victoria P. Binetti, Chief
Program Support Branch

Enclosure

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ENVIRONMENTAL PROTECTION AGENCY'S (EPA'S) COMMENTS ON
PROPOSED REVISIONS (MAY 17, 1991) TO
WEST VIRGINIA'S WATER QUALITY STANDARDS

Page no.

Comment

- 4 Section 3.2.1. EPA commends the State for proposing a narrative biological criterion. However, it is difficult for EPA to determine if the suggested language is consistent with EPA's April 1990 Biological Criteria: National Program Guidance for Surface Waters (EPA-440/5-90-004) since the phrase "no significant adverse impact" is undefined. If the Board intends to evaluate the biological integrity of potentially impacted sites relative to the biological integrity of reference sites (i.e., waters least impaired by human activities), then the proposed narrative biological criterion appears to be consistent with EPA guidance. In other words, the narrative biological criterion should promote water quality to protect the most natural community possible for the applicable designated use rather than just maintain the status quo. EPA requests that the Board clarify whether it intends the proposed narrative biological criterion to be interpreted consistent with EPA's guidance, and identify what procedures will be used to implement the proposed language. If implementation procedures have not been developed, EPA requests that the Board provide a schedule for when the procedures will be completed and submitted to EPA for review and approval.
- 4 Section 4.1. Section 131.12(a) of EPA's Water Quality Standards Regulation (40 CFR 131) requires States to "develop and adopt a statewide antidegradation policy and identify the methods for implementing such policy...." EPA is aware that the Board has plans to revise the State's existing implementation procedures. EPA fully supports this activity and offers to provide technical assistance. EPA requests that the Board provide a schedule for when the procedures will be completed and submitted to EPA for review and approval.
- 5 Section 4.1.a. EPA commends the Board for proposing language which defines "existing uses" as:
- those uses actually attained in the water body on or after November 28, 1975, whether or not they are included as designated uses within these water quality standards.

Defining "existing uses" in this manner is completely consistent with the definition contained in 40 CFR §131.3(e). Furthermore, 40 CFR §131.12(a)(1) requires States to maintain and protect "existing instream water uses and the level of water quality necessary to protect the existing uses." Therefore, with the proposed definition for existing uses, Section 4.1.a of West Virginia's Water Quality Standards conforms with EPA's Water Quality Standards Regulation.

- 7 Section 4.1.g. This section requires that "waters which constitute an outstanding national resource... shall be maintained and protected and improved where necessary." This language is consistent with 40 CFR §131.12(a)(3). EPA's national interpretation of 40 CFR §131.12(a)(3) is that this section means no new or increased discharges are allowed to waters which constitute an outstanding national resource. The only exception to this prohibition is discussed in the preamble to the Water Quality Standards Regulation which indicates that States may allow some limited activities which result in temporary and short-term changes in water quality. In order to be consistent with the intent of 40 CFR §131.12(a)(3), the Board should add clarification to Section 4.1.g which specifies that no new or increased discharges will be allowed to outstanding national resource waters.

- 7 Section 5.2.a.4. The Board has proposed language that prohibits "acute toxicity" within mixing zones. EPA requests that the Board clarify whether this prohibition is limited to lethality or encompasses other acute responses. In addition, EPA requests that the Board identify in the water quality standards the procedures that will be used in establishing mixing zones. If procedures have not been developed, EPA suggests that the Board add a statement which says mixing zones will be established in accordance with EPA's March 1991 Technical Support Document for Water Quality-based Toxics Control (TSD) EPA/505/2-90-001.

- 8 Section 6.1. EPA commends the Board for proposing the default use designations of Categories B and C for the protection of aquatic life and water contact recreation for all waters in West Virginia.

- 10 Sections 6.3.d and 6.5.c. EPA commends the Board for adopting use designation categories for wetlands. With regard to Section 6.3.d, the Board has proposed language which says "stream criteria may not be appropriate for application to wetlands." It is unclear what this section means. Does it mean that narrative criteria, numeric aquatic life criteria, numeric human health criteria, or a combination of any of the three types of criteria will not be applicable to wetlands? Narrative criteria must apply to all waters at all times. With regard to numeric criteria, EPA's Environmental Research Laboratory (ERL) in Duluth has said that human health criteria are directly applicable to wetlands. In addition, ERL-Duluth is finalizing a report entitled An Approach for Evaluation of Numeric Water Quality Criteria for Wetlands Protection. The report suggests that most numeric aquatic life criteria are applicable to most wetland types.

The Board should clarify what criteria are to be adopted for wetlands at this point in time, and what criteria the Board believes may not be applicable to wetlands.

- 14 Section 7.2.c. This section should be rewritten to indicate that numeric water quality standards will not apply when the conditions expressed in the succeeding subsections occur. Narrative criteria must apply in all waters at all times in order to comply with Section 303(c)(2)(A) of the Clean Water Act.
- 14 Section 7.2.c.2. This section waives the application of water quality standards for wet weather streams, or for intermittent streams when they are dry or have no measurable flow. The State must identify which streams fall under these categories. In addition, if the discharge of an effluent to a water body provides sufficient volume of water to support uses (e.g., aquatic life, recreation, etc.), then the water body must be protected for those uses.
- 14 Section 7.2.c.4. EPA interprets this section as allowing criteria to be automatically "reset" when "naturally-occurring" background concentrations exceed the State-wide criteria contained in Appendix E. EPA requests the State to provide clarification regarding whether naturally occurring conditions also means waters which are unaffected by nonpoint source loadings. In addition, the State must identify which

water bodies fall under this category and what the applicable criteria are for these waters.

15

Section 7.2.d.7.B. EPA has a concern which was raised previously in EPA's September 29, 1989 letter from Dr. Alvin R. Morris to Dr. David E. Samuel regarding West Virginia's last triennial water quality standards revisions. Numerous site-specific criteria (e.g., suspended solids, oil and grease, ammonia-nitrogen, phenols, cyanide, zinc and copper) were adopted for an unnamed tributary to the Monongahela River. Some or all of these criteria may not be protective of aquatic life uses (e.g., the criteria for copper, zinc, and cyanide are approximately 55, 13 and 2 times greater than EPA's acute criteria, respectively). Apparently the criteria are interim measures put in place until the Superfund status of Sharon Steel has been decided.

Section 131.11(a)(1) of EPA's Water Quality Standards regulation requires States to adopt criteria which protect designated uses. Since the State's aquatic life and water contact recreation use categories would apply to this unnamed tributary, the criteria applicable to this stream should protect for aquatic life and all other applicable uses. Therefore, the Board must take one of the following actions to make this section of the standards consistent with 40 CFR 131: 1) provide justification for the existing (or revised) site-specific criteria which demonstrates that the aquatic life use (and all other applicable uses) will be protected; 2) remove the site-specific criteria for the unnamed tributary and restore the State-wide criteria; 3) remove the aquatic life use (assuming a use attainability analysis is conducted and demonstrates that attaining the use is not feasible because of one of the conditions expressed in 40 CFR 131.10(g) is met); or 4) grant Sharon Steel a variance from meeting certain water quality standards because controls more stringent than those required by Sections 301(b) and 306 of the Clean Water Act would result in substantial and widespread economic and social impact.

16&17

Sections 7.2.d.16.A and 7.2.d.20.B. EPA substantially revised its aquatic life criteria for selenium in 1988. As expressed in EPA's September 29, 1989 letter, the site-specific selenium criteria for Connors Run and Little Scary Creek should be reevaluated in light of EPA's revised criteria.

16 Section 7.2.d.16.B. As requested in EPA's September 29, 1989 letter, the Board must provide EPA with an updated economic assessment for Weirton Steel since this facility was basically granted a variance from meeting water quality standards for temperature, iron and fluoride. Since variances are temporary and are considered to be changes to water quality standards, they must be reexamined at least once every three years like any other standards revision.

17 Section 7.2.d.20-D. The Board has proposed site-specific temperature criteria of 100°F (daily maximum) and 93°F (monthly average) for Simmons Creek. EPA is still not convinced that the proposed temperature criteria will be protective of aquatic life uses, will allow for the free passage of aquatic life, or will be protective of downstream uses of the Kanawha River. The maximum daily temperature of 100°F (38°C) exceeds the lethal threshold of probably every fish species that could occur in this stream, absent DuPont's high temperature discharge. Although the proposed criteria may maintain existing uses of Simmons Creek (e.g., an impaired aquatic life use), the criteria do not support the Section 101(a) goals of the Clean Water Act nor do they comply with Section 303(c)(a) of the Clean Water Act.

Therefore, the Board must take one of the following options: 1) provide justification for the existing (or revised) site-specific criteria which demonstrates that the aquatic life use (and all other applicable uses) will be protected; 2) remove the site-specific criteria for Simmons Creek and restore the State-wide temperature criteria; 3) remove the aquatic life use (assuming a use attainability analysis is conducted and demonstrates that attaining the use is not feasible because of one of the conditions expressed in 40 CFR 131.10(g) is met); or 4) grant DuPont a variance from meeting certain water quality standards because controls more stringent than those required by Sections 301(b) and 306 of the Clean Water Act would result in substantial and widespread economic and social impact.

19 Section 8.2.c. EPA supports the Board's proposal of allowing the harmonic mean flow to be used as the design flow for carcinogens. Use of the harmonic mean flow is consistent with EPA's TSD.

- 21 Section 9.4. The references for bioassay testing should be modified. The reference to the EPA document EPA-660/3/75/009 should be deleted. This document was superseded by EPA 600/4-85/013 and EPA 600/4-89/011, both of which are cited in Section 9.4. These two documents should be identified as the primary references for bioassay testing, whereas the Standard Methods and ASTM manuals should be cited as secondary references.

Comments on Appendix E - Specific Water Quality Criteria

1. EPA commends the Board for adopting human health criteria for some pollutants (e.g., 1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, vinyl chloride, and 2,3,7,8-tetrachlorodibenzo-p-dioxin) which are the same as existing or updated EPA criteria recommendations.
2. The Board has chosen not to adopt a two-criteria approach (i.e., acute and chronic criteria) for maintaining water quality. EPA encourages the Board to include both acute and chronic criteria, where appropriate, to better reflect the different tolerances of aquatic systems to the inherent variability in pollutant concentrations and the toxicological characteristics of pollutants. If West Virginia's "chronic" criteria are applied as "not to be exceeded" criteria, then they will provide adequate acute and chronic protection. EPA requests that the Board describe how the criteria are applied and how acute and chronic protection is provided.
3. There appears to be inconsistent application of criteria for the protection of humans from consuming contaminated fish. Since the use category "recreation" includes "fishing" as one of the uses to be protected, "fish consumption" criteria must be adopted for all waters designated for recreation. The Board has adopted "fish consumption" criteria for many pollutants. However, some of these criteria (e.g., criteria for hexachlorobenzene, carbon tetrachloride, and chloroform) are applied to the warmwater and trout water designations, rather than to the recreation category. The Board should resolve these inconsistencies and ensure that fish consumption criteria apply to all waters of the State which support aquatic life.
4. The Board has chosen not to adopt criteria for all pollutants for which EPA has issued Section 304(a) guidance (see #4a and #4b below). As a consequence, for these pollutants, EPA requires the Board to submit justification for why the

discharge or presence of those pollutants could not reasonably be expected to interfere with the designated uses assigned to the waters of the State.

EPA's position on the adoption of toxics criteria is clearly stated in EPA's 1988 "Guidance for State Implementation of Water Quality Standards for CWA Section 303(c)(2)(B)" and in EPA's TSD. These documents indicate that the discharge or presence of pollutants in surface waters strongly suggests that such pollutants could be interfering with attaining designated uses and is sufficient justification for the adoption of numeric criteria. On April 25, 1991, EPA submitted to the Board computer printouts and a candidate list of pollutants for criteria adoption. On May 7, 1991, Jan Taylor responded to EPA and said "errors in the data bases make much of the information unusable." Most of Dr. Taylor's comments were regarding the retrievals of information contained in NPDES permit applications or the pollutants predicted to be in effluents, rather than the ambient data. The data retrievals indicate that many pollutants have been detected in the ambient water column, sediment or fish tissue samples (see #4c below), which EPA believes provides sufficient justification for adopting criteria.

- a. **Pollutants for which the Board has not adopted aquatic life criteria but for which EPA has issued Section 304(a) criteria recommendations.**

trivalent chromium
lindane
endosulfan
heptachlor

- b. **Pollutants for which the Board has not adopted human health criteria (fish consumption and/or public water supply) but for which EPA has issued Section 304(a) criteria recommendations.**

trivalent chromium (fish consumption and public water supply)
antimony (fish consumption and public water supply)
certain phthalate esters (i.e., diethyl phthalate, dimethyl phthalate, and bis(2-ethylhexyl) phthalate) (fish consumption and public water supply)
thallium (fish consumption and public water supply)
acrolein (fish consumption and public water supply)
chlorobenzene (public water supply)
1,3-dichloropropylene (fish consumption and public water supply)
ethylbenzene (fish consumption and public water supply)

1,2-trans-dichloroethylene (fish consumption and public water supply)
 1,1,2-trichloroethane (fish consumption and public water supply)
 benzidine (fish consumption and public water supply)
 certain haloethers (i.e., bis(2-chloroethyl) ether and bis(2-chloroisopropyl) ether) (fish consumption and public water supply)
 3,3'-dichlorobenzidine (fish consumption and public water supply)
 diphenylhydrazine (fish consumption and public water supply)
 fluoranthene (fish consumption and public water supply)
 hexachloroethane (fish consumption and public water supply)
 hexachlorobutadiene (fish consumption and public water supply)
 hexachlorocyclopentadiene (public water supply)
 isophorone (fish consumption and public water supply)
 nitrobenzene (public water supply)
 nitrosamines (i.e., n-nitrosodimethylamine, n-nitrosodi-n-propylamine, and n-nitrosodiphenylamine) (fish consumption and public water supply)
 alpha-hexachlorocyclohexane (fish consumption and public water supply)
 beta-hexachlorocyclohexane (fish consumption and public water supply)
 lindane (gamma-hexachlorocyclohexane) (fish consumption and public water supply)
 endosulfan (fish consumption and public water supply)
 heptachlor (fish consumption and public water supply)
 nitrophenols (2,4-dinitrophenol and 4,6-dinitro-2-methylphenol) (fish consumption and public water supply)

c. Pollutants for which the Board has not adopted all necessary criteria but which have been detected in ambient water column, in sediment or in fish tissue samples. The specific pollutants and the type of criteria which should be adopted are identified below.

alpha-hexachlorocyclohexane (fish consumption and public water supply)
 beta-hexachlorocyclohexane (fish consumption and public water supply)
 antimony (fish consumption)
 phthalate esters (public water supply)
 chlorobenzene (fish consumption and public water supply)
 trivalent chromium (aquatic life, fish consumption, and public water supply)
 ethylbenzene (fish consumption and public water supply)

lindane (aquatic life, fish consumption, and public
 water supply)
 heptachlor (aquatic life, fish consumption, and public
 water supply)
 4,6-dinitro-2-methylphenol (fish consumption and public
 water supply)
 thallium (fish consumption)
 fluoranthene (fish consumption and public water supply)

5. The Board has proposed aquatic life criteria for some of the pollutants which are less stringent than EPA's Section 304(a) criteria recommendations. The Board must provide justification that demonstrates that the proposed criteria will protect aquatic life uses or adopt revised criteria which will protect aquatic life uses.

cadmium (trout water criteria exceed EPA's recommended
 criteria when the water hardness is between 75 and
 101 mg/L CaCO_3)
 chloride (warmwater and trout water)
 iron (warmwater)
 toxaphene (warmwater and trout water)
 pH (warmwater and trout water)
 silver (at certain hardness ranges, the warmwater and
 trout water criteria exceed EPA's proposed chronic
 criterion of 0.12 ug/L and EPA's acute criteria)
 zinc (warmwater criteria exceed EPA's recommended
 chronic criteria when the water hardness is less
 than 41 mg/L, between 301 and 340 mg/L, and
 between 401 and 772 mg/L CaCO_3)

6. The Board has proposed aquatic life criteria for some pollutants for which EPA has not developed recommended criteria and which may exceed the lowest observed effect levels (LOEL). EPA requests the Board to provide information regarding how these criteria were derived.

beryllium (warmwater and trout water)
 1,2-dichlorobenzene (warmwater and trout water)
 1,3-dichlorobenzene (warmwater and trout water)
 1,4-dichlorobenzene (warmwater and trout water)
 1,1,1-trichloroethane (warmwater and trout water)
 toluene (warmwater and trout water)
 phthalate esters (warmwater and trout water)

7. The Board has proposed some human health criteria either for fish consumption or for public water supplies which are less stringent than EPA Section 304(a) human health criteria recommendations. The Board must provide justification that demonstrates that the proposed criteria will protect human health or adopt revised criteria which will protect human health.

iron (public water supply)
 manganese (public water supply)
 acrylonitrile (warmwater and trout water)
 trichloroethylene (warmwater, trout water, and public water supply)
 phenolic materials (warmwater, trout water, and public water supply)

8. The Board has proposed some human health criteria either for fish consumption or for public water supplies which are less stringent than EPA criteria updated using the latest information contained in EPA's Integrated Risk Information System (IRIS). EPA sent the updated criteria recommendations to Jan Taylor on April 11, 1991. EPA suggests that the Board include these updated criteria in its final water quality standards.

acrylonitrile (warmwater and trout water are less stringent)
 trichloroethylene (public water supply is less stringent)
 toluene (warmwater, trout water, and public water supply are less stringent)
 phenolic materials (public water supply is less stringent)
 butylbenzyl phthalate (absent)
 di-n-butyl phthalate (absent)
 endosulfan sulfate (absent)
 endrin aldehyde (absent)
 heptachlor epoxide (absent)

9. EPA strongly encourages the State to replace its fecal coliform criteria with either EPA's recommended bacteriological criteria for Escherichia coli or enterococci.

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WRITER'S DIRECT DIAL NO. 347-8358

June 28, 1991

Mrs. Frances E. Hunter
Executive Secretary
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Re: Comments Of Howes Leather Company
Regarding Triennial Review Of
The West Virginia Water Quality
Standards.

Dear Mrs. Hunter:

Enclosed please find the original and seven copies of the
Comments Of Howes Leather Company Regarding Proposed Changes To The
West Virginia Water Quality Standards.

If you have any questions, please do not hesitate to contact
me.

Sincerely yours,

David Yaussy
David L. Yaussy

DLY:shb

Enclosure

RECEIVED

JUN 28 1991

WATER RESOURCES BOARD

COMMENTS OF HOWES LEATHER COMPANY
REGARDING PROPOSED CHANGES TO
THE WEST VIRGINIA WATER QUALITY STANDARDS

The West Virginia Water Resources Board, as part of its triennial review of the West Virginia water quality standards, has proposed deleting the water quality criteria for ammonia of 50 ug/l for warmwater B1 and B3 warmwater fish uses and substituting a chart which reflects the criteria developed by the EPA in its 1986 ammonia criteria document. Howes Leather Company ("Howes") urges the Board to withdraw that proposal and retain the 50 ug/l limit until such time as better-reasoned criteria is developed.

The new criteria is found in Chart A to Appendix E and includes a footnote which was also in the EPA criteria document. That footnote reads as follows:

Site-specific criteria development is strongly suggested at temperatures above 20° C because of the limited data available to generate the criteria recommendation, and at temperatures below 20° C because of the limited data and because small changes in criteria may have significant impact on the level of treatment required in meeting the recommended criteria.

In effect, the footnote disclaims the general applicability of the criteria in the chart for temperatures below or above exactly 20°

C. In its ammonia criteria document, EPA explained that:

The very limited amount of data regarding effects of pH on chronic NH₃ toxicity also indicates increasing NH₃ toxicity with decreasing pH, but the data are insufficient to derive a broadly applicable toxicity/pH relationship. Data on temperature effects on acute NH₃ toxicity are limited and somewhat variable, but indications are that NH₃ toxicity to fish is greater as temperature decreases. There is no information available

regarding temperature effects on chronic NH3 toxicity.

Quality Criteria for Water 1986. Furthermore, test results showed no clear trend among fish for temperature and pH-adjusted chronic toxicity values, and revealed a wide range of acute-chronic ratios. Id. Accordingly, there is insufficient support for establishing reliable chronic ammonia values by direct testing or extrapolation from acute toxicity data.

EPA has acknowledged that more information regarding ammonia's effect on aquatic life is needed. It indicated in the 1986 criteria document that it would be conducting additional research on the effect of temperature on ammonia toxicity. Development of new criteria before obtaining these new research results would be premature.

Given the current unsettled state of criteria development, the Board should not change its existing standard until better data is available. In the event that the new criteria are adopted, Howes urges the Board to recognize that site-specific variances will be necessary to properly adjust these limits for any particular discharger, based upon the quality of the receiving stream, the type of discharge, the average temperatures and pH, and other factors. Criteria which will force each discharger to develop a site-specific criterion will be very costly, create an administrative burden, and may well create serious interim compliance problems.

Howes appreciates the opportunity to offer these comments and urges the Board to retain its present ammonia standard until such time as EPA is able to provide valid guidance.

Respectfully submitted this 28th day of June, 1991.

Howes Leather Company
Star Route 3, Box 100
Frank, WV 24920-9706

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JUN 28 1991

WATER RESOURCES BOARD



WEST VIRGINIA MINING AND RECLAMATION ASSOCIATION 1624 KANAWHA BOULEVARD, EAST • CHARLESTON, WEST VIRGINIA 25311 • (304) 346-5318

June 28, 1991

State Water Resources Board
1260 Greenbrier Street
Charleston, West Virginia 25311

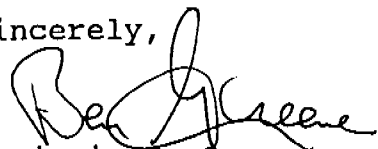
Gentlemen:

RE: Comments to Proposed Legislative Rules for Requirements
Governing Water Quality Standards - 1991 Title 46, Series 1

The attached comments are offered on behalf of the more than 350 member companies of the West Virginia Mining and Reclamation Association and the member companies of the West Virginia Coal Association. The majority of our member companies are concerned with the impact of these proposed changes on the mining of coal throughout West Virginia. In addition, many of our member companies hold NPDES permits for activities other than coal mining. These companies are similarly concerned with the impact of these proposed changes on their operations and businesses.

We would appreciate your consideration of these comments and should any of the members of the Board or any of its technical staff have questions or a need for additional information, do not hesitate to notify me immediately.

Sincerely,


Benjamin C. Greene
President

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WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46, Series 1 June 28, 1991

On Page 6, Section 46-1-4.1.e., the proposed new language "full intergovernmental coordination" should be deleted.

RATIONALE: This term is not defined in these proposed regulations or the applicable laws. Such a nebulous, undefined term being placed in a regulation governing determinations to be made by the permitting authorities will create disruption of established and effective permitting procedures.

The generalized requirement is not supported by federal or state statute. As written, the requirement is subject to a wide variety of interpretations by all branches and levels of government. At any point in the permitting and enforcement process, an aggrieved party could claim they were not included in such a broad consideration as "full intergovernmental."

On Page 8, Section 46-1-6.1, the proposed new language should be deleted.

RATIONALE: This language is not supported by or required by federal or state law. The lack of stream data or water quality information should not be used as justification for a presumption which will become a practical requirement for every permit. This is of particular concern to coal mining operations which are generally located in the headwater portion of streams for which such a presumption is many times inaccurate. To require a yet to be defined "demonstration" by every applicant in these type areas is an unnecessary mandate which has little, if any, relevance to water quality.

On Page 1, Appendix E, 8.1, ALUMINUM. The proposed changes should be deleted.

RATIONALE: The proposed change in the aluminum standard is not supported by data or natural conditions. A letter from Dr. Jan Taylor, Technical Adviser to the Water Resources Board to the US EPA, is attached as a partial and accurate explanation of the concern with the proposed change in the Al standard. The US EPA's response is also attached which recommends site-specific criteria be adopted, which reflects local conditions. That statement is consistent with actual field data collected on undisturbed West Virginia streams.

Although the warmwater criteria is not as limited as the coldwater, it will remove areas from coal production due to natural aluminum content associated with the overburden in some parts of the state. It will also restrict the use of aluminum as

WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA
COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR
REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46,
Series 1 June 28, 1991

a flocculant in drinking water plants because the aluminum concentrations of the backwash waters will be too high to discharge without violating the instream standard limit of 0.748 ppm.

A typical central West Virginia warmwater stream was selected (Amos Run, Braxton County) to define the background limits of aluminum concentrations occurring for a period of 21 months. The flow ranged from low to extremely high. The aluminum were as follows:

max:	0.75 ppm
Min:	<0.10 ppm
Mean:	0.24 ppm

As can be seen, the range of aluminum values encountered ranged from below detection (<0.10 ppm) to slightly above the MAC of (0.748 ppm). These values were naturally occurring. The average aluminum concentrations were 0.24 ppm which is well below the maximum, but the proposed regulations do not indicate an average value would be acceptable as compliance with the proposed stream standards. The maximum background aluminum concentration, which is a natural condition, was above the proposed maximum allowable stream standard.

Troutwaters (coldwaters) have an even stricter criteria of 0.087 mg/l MAC of aluminum. Two problems present themselves with this limit. The first is that a large number of troutwaters will have aluminum values that naturally exceed this limit. The second problem is that accepted methods of metal analysis rules out the use of flame AA and forces either graphite furnace methodology (detection limit at .005 ppm) or ICP methodology (detection limit at .05 ppm). These detection limits are not possible with flame Atomic Absorption spectroscopy unless concentration or extraction techniques are used. Although the Eriochrome Cyanine R calorimetric procedure is sensitive enough, it is dependent on careful control of reaction time, temperature, plus alkalinity. Furnace AA and ICP can obtain these limits, but the cost of the instrumentation lies beyond the means of most labs. The troutwater limit lies below the flame AA detection limit. The analytical lab will have to concentrate or extract the samples in order to determine aluminum at this level by flame AA. Again, Furnace AA and ICP can accomplish the task, but are expensive.

WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA
COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR
REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46,
Series 1 June 28, 1991

Data collected on Pendleton Creek, a tributary of Blackwater River over a thirty month period, shows the following aluminum concentrations.

Max: 1.19 ppm
Min: < .10 ppm
Mean: 0.30 ppm

Although Pendleton Creek is not classed as troutwaters by the West Virginia WRB, it does empty into the Blackwater River at the start of the troutwater area at Davis (Tucker County). These values were collected above any mining activities and, therefore, are not influenced by surface disturbance. The average value of natural aluminum is about five (5) times the proposed new standard of .087 ppm, but well within the current standard of 0.50 mg/l. If the proposed standard maximum concentration of .087 ppm is adopted, then the natural background aluminum would violate established water quality standards and any permitted discharge into Pendleton Creek would have to meet a discharge limit of zero aluminum.

Data collected on a seventeen month baseline on Shavers Fork Mainstream adjacent to and including the Shavers Fork "Fish for Fun" area shows the following average for aluminum.

Max: 1.05 ppm
Min: < .10 ppm
Mean: .28 ppm

These values were collected from seven points along Shavers Fork Mainstream and clearly show that the average aluminum values are three times the proposed troutwater standard. Shavers Fork, "Fish for Fun" area, holds citation size trout and is stocked on a regular basis.

In addition to the specific concern with the analytical procedures for coldwater standards, there is a general concern regarding the availability of instruments which can accurately distinguish between 748 micrograms/liter and 749 micrograms/liter.

Also, the implementation of this change does not recognize the practical conflict with achieving the manganese standard set forth in 8.17 of Appendix E. To again reference the letter of

WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA
COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR
REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46,
Series 1 June 28, 1991

August 13, 1990, from the US EPA to Dr. Jan Taylor, these proposed standards would be realistic and should only be implemented if applied to dissolved Al, as opposed to total Al, as noted on pages 3 and 4 of that letter.

On Page 1, Appendix E, 8.2; 8.2.1; and 8.2.2, AMMONIA. The proposed changes to this standard should be deleted.

RATIONALE: According to a high ranking official of US EPA's Region III office in Philadelphia, speaking to the 12th Annual Surface Mine Drainage Task Force Symposium in Morgantown, WV on April 3, 1991, efforts by state officials and persons working with ammonia in the state's mining industry had resulted in EPA recognizing West Virginia as having the most advanced program for the use of this chemical of any state in the nation. That EPA official also complimented the accomplishments of those in West Virginia who had compiled the Handbook For Use of Ammonia in Treating Mine Waters (enclosed).

Because of that extensive experience with the use of ammonia, the Association has great concern with the proposed changes in these standards. The Association offers the following supplemental field data and practical discussions to support our request that these proposed changes be deleted.

The formula, as given, yields un-ionized ammonia values in mg/l, yet the limits are expressed in ug/l. Unless the formula is modified, most labs will be reporting results that are off by a factor of 1000. Ammonia is almost always measured and reported as ammonia-nitrogen, but the limits are expressed as "ammonia." For the sake of consistency, the Total Ammonia (NH3) values in Charts A & B should be changed to Ammonia-Nitrogen (NH3-N). If the Board chooses to leave the values in the present form and include a coefficient to convert between these values, the note at the bottom of the chart should read; "To convert concentrations of un-ionized ammonia (NH3) to un-ionized ammonia-N (NH3-N), multiply by 0.822."

Further, the concentration of un-ionized ammonia is calculated by an empirical formula where total ammonia-N is determined by analytical methods, and field temperature (in degrees Celsius) and field pH are known. The chart or description should indicate "field pH and temperature."

WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA
COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR
REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46,
Series 1 June 28, 1991

The equation listed in the proposed rules at 8.2 is incorrect. The superscript "a" following the number 273.2 should be deleted.

Sections 8.2.1 and 8.2.2 refer to Chart A (B2 or trout streams) and Chart B (B1 and B3 streams), which combine pH and temperature to determine the criteria for these streams. The intention of these charts appears unclear. One assumes that given a certain temperature and pH, the concentration shown at the intersection of the chart cannot be exceeded. Since the calculation of the toxic portion of total ammonia is tedious and difficult to calculate, a chart is a welcome addition to the rules.

However, it is difficult to determine if the attempt is to clarify the criteria by means of an understandable chart or to limit the un-ionized ammonia concentration in streams depending on pH and temperature. The present chart fails both objectives.

As an example, if one takes a sample at pH 7.0 and 20 degrees C and the analysis indicates 4.0 mg/l total ammonia, the empirical formula would indicate 0.01896 mg/l un-ionized ammonia in the trout stream. This would indicate compliance (less than 0.02 mg/l). Yet Chart A indicates only 1.49 mg/l total ammonia is allowed, or 0.0059 mg/l of un-ionized ammonia is allowed. Which is it? The formula produces 0.007 mg/l of un-ionized ammonia under these conditions for 1.49 mg/l total ammonia. The values also do not agree with the chart abstracted from Thurston et.al. in the EPA Red Book. The table is incorrect, misleading and ambiguous.

If the objective of revising the regulations was to include an easy to use chart, the Board is to be commended. However, the only dependable way to insure compliance is to maintain numerical limits such as 0.02 and 0.05 mg/l, and use the formula or a chart similar to the one attached for calculating the values.

If the objective of revising the regulations is to further limit the level of ammonia being discharged to the waters of the state, this action is unwarranted. There is no reason for more stringent standards. Trials in WV, at mining operations using anhydrous ammonia, indicate the substance can be used safely and in an environmentally sound manner. Where present limits have been met, there have been no problems. Further limiting ammonia

WEST VIRGINIA MINING & RECLAMATION ASSOCIATION AND WEST VIRGINIA
COAL ASSOCIATION COMMENTS TO THE PROPOSED LEGISLATIVE RULES FOR
REQUIREMENTS GOVERNING WATER QUALITY STANDARDS -1991 - Title 46,
Series 1 June 28, 1991

concentrations, below the national norm, would only discourage responsible operators from its use and cause further stream problems due to fluctuating effluents and non-compliance.

As indicated on the "errata" sheet, deleting the lines on the charts is counterproductive to either objective. If the charts were correct, it would distinguish compliance levels from noncompliance levels.

On Page 1, Appendix E, 8.42, ARSENIC.

The proposed regulations call for limits for Trivalent Arsenic. Why "Trivalent Arsenic"? Why not "Total Arsenic"? The total recoverable digestion referenced in 40 CFR 136 makes it impossible to differentiate the oxidation states.

On Page 5, Appendix E, 8.15, IRON.

This section has several misspelled words. Also, this parameter, although not proposed to be changed, does not appropriately recognize that existing and/or natural conditions in many streams in West Virginia already exceed this standard. That is in direct conflict with the proposed changes to section 6.1 of these rules.

On Page 6, Appendix E, 8.18.2, MERCURY.

The mercury limit of 0.012 ug/l is for methyl mercury. This is 12 parts per trillion methyl mercury. Is this realistic?

Attachments:

CHART FOR DETERMINING UN-IONIZED AMMONIA

for
Total Ammonia (N) = 1 PPM

Temperature in Degrees C°

pH	0	5	10	15	20	25
4.0	0.00000	0.00000	0.00000	0.00000	0.00000	0.00001
4.5	0.00000	0.00000	0.00001	0.00001	0.00002	0.00002
5.0	0.00001	0.00001	0.00002	0.00003	0.00005	0.00007
5.5	0.00003	0.00005	0.00007	0.00010	0.00015	0.00022
6.0	0.00010	0.00015	0.00022	0.00033	0.00048	0.00068
6.5	0.00031	0.00047	0.00071	0.00104	0.00150	0.00215
7.0	0.00099	0.00150	0.00223	0.00327	0.00474	0.00679
7.5	0.00313	0.00472	0.00702	0.01030	0.01488	0.02120
8.0	0.00983	0.01481	0.02194	0.03196	0.04582	0.06457
8.5	0.03056	0.04561	0.06673	0.09558	0.13384	0.18291
9.0	0.09158	0.13328	0.18836	0.25783	0.34099	0.43503
9.5	0.24859	0.33985	0.44471	0.55670	0.66792	0.77117
10.0	0.54292	0.66653	0.78071	0.87855	0.95853	1.02054

Un-ionized Ammonia for other values of Total Ammonia (N) may be determined by multiplying the chart value by the Total Ammonia (N) desired.

Un-ionized Ammonia limits for trout streams = 0.02 -----

Un-ionized Ammonia limits for non-trout streams = 0.05 =====

The chart is based on a Total ammonia value of 1 ppm. Read the un-ionized ammonia value for the pH and temp. of your sample, and multiply this value by your actual total ammonia value. (If your total ammonia value is 0.5 ppm,

BEN B. FAULKNER

environmental consultant

June 26, 1991

BRATTON FARM

Rt. 1, Box 673

Princeton, WV 24740

Phone/Fax (304) 487-2886

State Water Resources Board
1260 Greenbrier St.
Charleston, WV 25311

Dear Members of the Board,

As a member of the Water Quality Advisory Committee and the WV Mine Drainage Task Force, I wish to comment on the proposed Title 46 Legislative Rules Series 1 (1991).

Appendix E 8.1 Aluminum

Establishing stream standards of 748 $\mu\text{g/l}$ for warmwater streams and 87 $\mu\text{g/l}$ for trout streams is too stringent. Background (sites upstream of any mining or other activity) water quality in some trout streams exceeds 87 $\mu\text{g/l}$ aluminum and many warmwaters have concentrations higher than 748 $\mu\text{g/l}$ in undisturbed areas from natural sources.

I am also particularly concerned about lowering limits where individuals must treat their effluent for aluminum. As you are aware, aluminum exhibits the unusual characteristic of redissolving at higher pH levels. As operators elevate pH by the addition of neutralizers to precipitate iron, manganese and aluminum, it is more likely that overtreatment (and subsequent redissolution of aluminum) will occur when these lower limits are being sought. I am not aware of a specific problem in the mining industry in WV where aluminum alone has voided life in a once productive stream, although I do know of many alkaline streams with fairly high (>2 mg/l) concentrations of aluminum from natural sources.

Appendix E 8.2 Ammonia

The concentration of un-ionized ammonia is calculated by an empirical formula where total ammonia-N is determined by analytical methods, and field temperature (in degrees Celsius) and field pH are known.

The equation listed in the referenced rules is incorrect. The superscript "a" following the number 273.2 should be deleted.

Sections 8.2.1 and 8.2.2 refer to Charts A (B2 or trout streams) and Chart B (B1 and B3 streams) which combine pH and temperature to determine the criterion for these streams. The intention of these charts appears unclear. One assumes that given a certain temperature and pH, the concentration shown at the intersection of the

chart cannot be exceeded. Since the calculation of the toxic portion of total ammonia is tedious and difficult to calculate, a chart is a welcome addition to the rules.

I cannot determine if the attempt is to clarify the criterion by means of an understandable chart or to limit the un-ionized ammonia concentration in streams depending on pH and temperature. The present chart fails either objective.

As an example, if one takes a sample at pH 7.0 and 20°C and the analysis indicated 4.0 mg/l total ammonia, the empirical formula would indicate 0.01896 mg/l un-ionized ammonia in the trout stream. This would indicate compliance (less than 0.02 mg/l). Yet Chart A indicates only 1.49 mg/l total ammonia is allowed, or 0.0059 mg/l of un-ionized ammonia is allowed. Which is it? The formula produces 0.0071 mg/l of un-ionized ammonia under these conditions for 1.49 mg/l total ammonia. The values also do not agree with the chart abstracted from Thurston et.al. in the EPA Red Book. The table is incorrect, certainly misleading and ambiguous.

If the objective of revising the regulations was to include an easy to use chart, I commend the board. The only sure way to ensure compliance is to maintain numerical limits such as 0.02 and 0.05 mg/l, and use the formula or a chart similar to the one attached for calculating the values.

If the objective of the revising the regulations is to further limit the level of ammonia being discharged to the waters of the state, this action is unwarranted. There is no reason for more stringent standards. Trials in WV at mining operations using anhydrous ammonia indicate the substance can be used safely and in an environmentally sound manner. Where present limits have been met, there have been no problems. Further limiting ammonia concentrations below the national norm would only discourage responsible operators from its use and cause further stream problems due to fluctuating effluents and non-compliance.

Lowering limits will not accomplish the objective we all seek - improved or protected water quality and uses. As an example, If the speed limit on a highway is 35 mph, and many accidents occur when a few individuals drive 65 mph, does it really help to lower the limit to 25 mph? Individuals using anhydrous ammonia find it gives them better control, more efficient and economical results, and that it is more environmentally sound than many other ameliorative agents. Taking away this valuable tool from responsible operators will only result in less compliance for the very parameters you wish to improve.

Please examine the field record of the use of anhydrous ammonia in West Virginia. Become more familiar with anhydrous ammonia. The WV Mining & Reclamation Association has published a booklet to help operators meet their goal of compliance when using ammonia. I also invite

you to examine EPA's field and laboratory studies in WV where anhydrous ammonia is being used. Their benthic and bioassay studies do not indicate a lower limit is warranted.

Deleting the lines on the charts (a correction) is counterproductive to either objective. If the charts were correct, a delineation would distinguish compliance levels from noncompliance levels.

Further, the ammonia criterion should be stated as un-ionized ammonia-nitrogen ($\text{NH}_3\text{-N}$); not as un-ionized ammonia (NH_3). If you wish to leave the values in the present form and include a coefficient to convert between these values, the note at the bottom of the chart should read; "To convert concentrations of un-ionized ammonia (NH_3) to un-ionized ammonia-N ($\text{NH}_3\text{-N}$), multiply by 0.822."

Finally, the chart or description should indicate "field pH and temperature".

Appendix E 8.1 Iron

Several misspelled words appear in this parameter description. Additionally, iron limits are presently sufficiently strict to protect stream uses. Many warmwater streams and trout streams exhibit concentrations of total iron higher than the proposed limits upstream to any disturbances or man-made activity.

As you finalize these regulations I pray you will scrutinize not only the laboratory studies referenced to these criteria, but will examine the field trials and performance of individuals in WV who have been diligently meeting the present strict effluent limits. These limits do not need to be lowered either directly or as a result of a change in stream criteria.

Sincerely,



CHART FOR DETERMINING UN-IONIZED AMMONIA

for
Total Ammonia (N) = 1 PPM

Temperature in Degrees C°						
pH	0	5	10	15	20	25
4.0	0.00000	0.00000	0.00000	0.00000	0.00000	0.00001
4.5	0.00000	0.00000	0.00001	0.00001	0.00002	0.00002
5.0	0.00001	0.00001	0.00002	0.00003	0.00005	0.00007
5.5	0.00003	0.00005	0.00007	0.00010	0.00015	0.00022
6.0	0.00010	0.00015	0.00022	0.00033	0.00048	0.00068
6.5	0.00031	0.00047	0.00071	0.00104	0.00150	0.00215
7.0	0.00099	0.00150	0.00223	0.00327	0.00474	0.00679
7.5	0.00313	0.00472	0.00702	0.01030	0.01488	0.02120
8.0	0.00983	0.01481	0.02194	0.03196	0.04582	0.06457
8.5	0.03056	0.04561	0.06673	0.09558	0.13384	0.18291
9.0	0.09158	0.13328	0.18836	0.25783	0.34099	0.43503
9.5	0.24859	0.33985	0.44471	0.55670	0.66792	0.77117
10.0	0.54292	0.66653	0.78071	0.87855	0.95853	1.02054

Un-ionized Ammonia for other values of Total Ammonia (N) may be determined by multiplying the chart value by the Total Ammonia (N) desired.
Un-ionized Ammonia limits for trout streams = 0.02 -----
Un-ionized Ammonia limits for non-trout streams = 0.05 =====

The chart is based on a Total ammonia value of 1 ppm.
Read the un-ionized ammonia value for the pH and temp. of your sample, and multiply this value by your actual total ammonia value. (If your total ammonia value is 0.5 ppm, multiply by 0.5; if it is 3 ppm, multiply the value by 3.)



ISLAND CREEK CORPORATION

250 WEST MAIN STREET P.O. BOX 11430 LEXINGTON, KY 40575-1430 TELEPHONE 606/266-3000

CERTIFIED #P513 198 143
RETURN RECEIPT REQUESTED

June 26, 1991

State Water Resources Board
STATE OF WEST VIRGINIA
1260 Greenbrier Street
Charleston, West Virginia 25311

Re: Comments Regarding Proposed Amendments to Title 46,
Series 1 -- Water Quality Standards

Gentlemen:

Island Creek Corporation appreciates the opportunity to comment on the proposed changes to the West Virginia Water Quality Standards, Title 46, Series 1. Island Creek subsidiaries operate throughout West Virginia, producing over seven million tons of coal per year and employing over nine hundred personnel in the state. We are directly and significantly affected by the subject regulations.

The West Virginia Mining and Reclamation Association and West Virginia Coal Association, of which we are a member, will also be submitting comments on these proposed rules. We concur with and refer you to the additional details which will be contained in those comments. We offer, however, the following comments to outline our position.

At 46-1-4.1(a), the Board, at EPA's request, is proposing to change the language to read: "Existing uses are those uses actually attained in the water body on or after November 28, 1975, whether or not they are included as designated uses within these water quality standards."

There are two significant problems with this approach. The first and most obvious is that applying a retroactive use to stream conditions which may have existed 16 years ago is highly problematic in its own right. In 16 years a number of changes could have occurred which may have changed the complexion of the stream dramatically in terms of quality, quantity and physical conditions so that uses a stream once may have had may never be recaptured. Natural phenomena such as floods, West Virginia has

State Water Resources Board
Proposed Regulations to Title 46, Series 1
June 26, 1991
Page 2

experienced quite a few in 16 years, droughts, hurricanes and other natural phenomena have had significant impacts on many streams in the State. Man made changes due to farming, highway construction, mining, timbering, dam construction, etc. also have had a large role to play. A retroactive use application will prove to be cumbersome, unweildy and argumentative.

Secondly, the retroactive date is not mandated by the anti-degradation provision of the U.S. EPA regulations required to be adopted by States. If the Board wishes to clarify "an existing use" we recommend it make a provision that existing uses are those actually attained in the water body on or after the effective date of the proposed regulations.

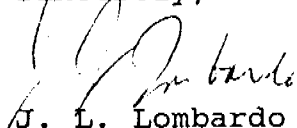
At Appendix E, 8.1 Aluminum, the proposed change should be deleted.

As allowed by EPA, States have the prerogative to modify the recommended standards to more closely fit such standards to State conditions. A review of the criteria document for aluminum quickly shows that the basis for EPA's recommendations is highly subjective. Without belaboring the point, EPA's recommendation could just as easily have been 1,496 $\mu\text{g/l}$ if not for fuzzy rationale which says a final acute-chronic ratio can never be less than 2. The ratio in this case could have been 0.9958, otherwise.

The comments point out that the criteria document for aluminum leaves much to be desired. We believe the Board has every justification to ignore its recommendations (which are fuzzy in their own right) and retain its present standard for aluminum. After all, the original standard of 0.5ppm for trout streams is based on past State studies, using State facilities, State expertise, State funds, State waters and State fish. This should be sufficient to justify retaining the standard until the EPA and/or the State can justify changes based on further State studies in State waters.

If we can be of further service in this regard, please don't hesitate to contact us.

Sincerely,


J. L. Lombardo
Director - Environmental Affairs

cc: G. White (WVCA)
B. Greene (WVMRA)



SIERRA CLUB
WEST VIRGINIA CHAPTER

P. O. Box 4142
Morgantown, WV 26504

RECEIVED

JUN 28 1991

WATER RESOURCES BOARD

June 27, 1991

Dr. Jan Taylor
State Water Resources Board
1260 Greenbrier St.
Charleston, WV 25311

Dear Jan:

Please consider the following comments on the Proposed Amendments and Revisions to the West Virginia Water Quality Standards as submitted on behalf of the 1300 members of the West Virginia Chapter of Sierra Club.

1. Section 2.8: We concur with the addition of the word "outstnading."
2. Section 3.2.i: We concur with the altered language.
3. Section 4.1.a: We concur with this change (see Section 6.1 below).
4. Section 4.1.e and g: We concur with these changes.
5. Section 5.2.a.4: We strongly support this change.
6. Section 6.1: We have problems with how the second sentence of the proposed changes in this section will be applied and adjudicated, and we see a significant potential for abuse resulting in the effective lowering of water quality standards. This second sentence should be deleted.
7. Section 6.3.d: We concur with this addition, however, how will B4 and D3 wetlands be addressed in the water quality criteria table? We see no reference to it, and do not feel that it fits in with the "all other uses" categories which are generally less stringent. We recommend that, until the appropriate regulations are promulgated, all wetlands be included under the B1,B3 standards unless directly impacting trout waters in which case B2 would apply.
8. Section 6.5.c: We concur with the addition, but see point (7) above.
9. Section 7.2.b: We strongly support the additional language.

10. Section 7.2.d.2.B: We support the deletion of this exception.

11. Section 7.2.d.20.D: We question the proposed change to such a high temperature which will seriously impact the aquatic life of the stream and potential for water contact recreation (the Clean Water Act goals). We recommend additional public review of this proposed exception prior to inclusion.

12. Section 7.3.d: We concur with the addition.

13. Section 8.2.c: We are opposed to the addition of this section in dealing with carcinogens. We recommend that it be deleted. The proposed change would allow a standard to be exceeded in a given stream a significant proportion of the time compared to setting the standard using 7Q10 flows. During periods of very low flow, like we are seeing now and in 1988, carcinogen concentrations would dramatically exceed the recommended standard.

14. Section 9.4: We concur with the additions.

15. Standards. In general, why are "X's" sometimes used and sometimes not? Some consistency improvement could be done.

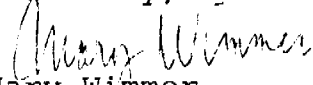
a) We question the dramatic (!) increase in the Arsenic standard. Why such a great change (greater than 20,000-fold)? And why are there no standards for other than public drinking waters?

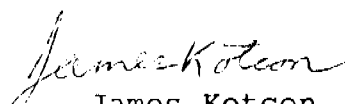
b) We strongly support the proposed dioxin standard. Any alternation to less stringent standard should await completion of the ongoing review by EPA. One comment on the industry group claim that the Banbury Conference produced consensus. As scientific researchers ourselves who attend numerous scientific conferences, we are sympathetic to the Banbury Conference criticism raised by University of Maryland Professor Ellen Silbergeld that "consensus" was not the purpose or intent of the conference. That industry would use such a scientific setting for a PR ploy to promote their special interest (relaxation of dioxin standards) we find irresponsible and disappointing.

c) Other changes in the standards we have no comment on. We are, however, concerned about the small incremental increases in a number of criteria such as beryllium and total mercury. Why such small changes? Is the science that accurate?

We appreciate the opportunity to comment on the proposed water quality regulations.

Sincerely,


Mary Wimmer and
Conservation Committee


James Kotcon

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JUL 08 1991

WATER RESOURCES BOARD



VIRGINIA POWER

July 5, 1991

Dr. David Samuels
Chairman, Water Resources Board
West Virginia State Water Resources Board
1206 Greenbrier Street
Charleston, WV 25311

Dear Dr. Samuels:

Virginia Power supports the comments on proposed revisions to the West Virginia Water Quality Standards as submitted by the West Virginia Chamber of Commerce and Manufacturers Association on June 28, 1991. Virginia Power owns and operates the Mount Storm Power Station in Grant County, West Virginia, which operates under the terms and conditions of an NPDES Permit. We are concerned about these regulations and believe the aforementioned comments will help improve the effectiveness of the water quality standards.

We would be happy to answer any questions that you may have.

Sincerely,

B. M. Marshall, P.E.
Manager
Water Quality

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WRITER'S DIRECT DIAL NO. 347-8358

June 28, 1991

Mrs. Frances E. Hunter
Executive Secretary
State Water Resources Board
1260 Greenbrier Street
Charleston, WV 25311

Re: Triennial Review of West Virginia
Water Quality Standards

Dear Mrs. Hunter:

Enclosed please find the original and seven copies of the Comments On Proposed Revisions To The West Virginia Water Quality Standards 46 C.S.R. Series 1.

If you have any questions, please do not hesitate to contact me.

Sincerely yours,


David L. Yaussy

DLY:shb

Enclosure

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JUN 28 1991
WATER RESOURCES BOARD

**BEFORE THE
WEST VIRGINIA WATER RESOURCES BOARD**

**COMMENTS ON PROPOSED REVISIONS TO THE
WEST VIRGINIA WATER QUALITY STANDARDS
46 C.S.R. SERIES 1**

SUBMITTED ON BEHALF OF

**WEST VIRGINIA CHAMBER OF COMMERCE
WEST VIRGINIA MANUFACTURERS ASSOCIATION
WEST VIRGINIA OIL AND NATURAL GAS ASSOCIATION
INDEPENDENT OIL AND GAS ASSOCIATION OF WEST VIRGINIA
WEST VIRGINIA FARM BUREAU**

JUNE 28, 1991

TABLE OF CONTENTS

	<u>Page</u>
I. INTRODUCTION	1
II. COMMENTS ON PROPOSED WATER QUALITY STANDARDS	3
A. Measuring Bioavailable Forms Of Metals.	3
B. Analytical Variability.	7
C. Instream Biomonitoring And Narrative Biocriteria	10
D. Prohibition Of Acute Toxicity In Mixing Zones . .	12
E. Designated Uses	14
F. Time Extensions To Comply With Water Quality Based Effluent Limits When Treatment Technology Is Not Available	16
G. Water Use Categories\Special Criteria	17
H. Regulating Only Substances Harming Designated Uses.	18
I. Regulating Human Health Constituents At The 10 ⁻⁶ Risk Level	20
J. Narrative Criteria For Wetlands	22
K. Harmonic Mean Flow.	22
L. Specific Criteria	27
1. Selenium	
2. Aluminum	
3. Mercury	
4. Arsenic	
M. New Criteria - Dioxin	33
1. Background.	33
2. Elements Of A Water Quality Standard For Dioxin.	36
a. Cancer Potency	36
b. Risk Level	44

	<u>Page</u>
c. Bioaccumulation/Bioconcentration Factors	45
d. Fish Consumption Rate.	47
e. Water Ingestion Rate	50
f. Fish Cooking Losses	50
3. Summary.	50
III. <u>CONCLUSION</u>	51
IV. <u>ENDNOTES</u>	52

**COMMENTS ON PROPOSED REVISIONS TO THE
WEST VIRGINIA WATER QUALITY STANDARDS
46 C.S.R. SERIES 1**

I. INTRODUCTION

On May 17, 1991 The West Virginia Water Resources Board ("the Board") proposed amendments to the West Virginia water quality standards, 46 C.S.R. Series 1. In response to those proposals, comments have been prepared by the following organizations:

WEST VIRGINIA CHAMBER OF COMMERCE

WEST VIRGINIA MANUFACTURERS ASSOCIATION

WEST VIRGINIA OIL AND NATURAL GAS ASSOCIATION

INDEPENDENT OIL AND GAS ASSOCIATION OF WEST VIRGINIA

WEST VIRGINIA FARM BUREAU

The West Virginia Chamber of Commerce is a non-profit state trade organization, headquartered in Charleston, West Virginia. It represents over 1,000 business members, approximately 56 local chambers of commerce (with aggregate memberships exceeding 25,000 businesses), and several business, trade and professional associations. Substantially all of the private employment in West Virginia has been created by businesses represented, either directly or indirectly (through local chambers and various businesses, trade and professional associations), by the Chamber.

The West Virginia Manufacturers Association is a non-profit state trade organization headquartered in Charleston, West Virginia. It represents over 250 companies in West Virginia, including manufacturers of chemicals, metal, glass and many other

wholesale and consumer products. Its membership ranges from small family-owned businesses to large multi-national corporations.

The West Virginia Oil and Natural Gas Association, headquartered in Charleston, West Virginia, is a non-profit state trade association representing producers, contractors, and those businesses that are directly related to the oil and gas industry. It has a membership of approximately 55 companies. The organization is charged with the object of fostering and advancing the common interests of those engaged in the industry by all beneficial or helpful legal actions and to disseminate pertinent data and information useful or necessary in the operations of the oil and gas business.

The Independent Oil and Gas Association of West Virginia is a non-profit state trade association representing more than 170 companies engaged in the extraction and production of oil and natural gas in West Virginia. In addition, its membership includes nearly 400 drilling, contracting, or supply companies, professional firms, and royalty owners. The organization is dedicated to promoting and protecting a strong, competitive and capable independent oil and natural gas producing industry in West Virginia, and on the national level.

The West Virginia Farm Bureau is a private, non-profit organization of farmers and rural property owners, with approximately 12,500 members statewide. The organization is wholly financed and controlled by its membership, which ranges from large-scale farming operations to family-owned farms.

These organizations are referred to collectively herein as "the Commentors". The Commentors respectfully request that the Board consider the following comments regarding its proposed changes to West Virginia water quality regulations.

II. COMMENTS ON PROPOSED WATER QUALITY STANDARDS

A. Measuring Bioavailable Forms Of Metals.

The Series 2 NPDES regulations require that permit limits for metals and metalloids be expressed and measured as "total recoverable." This means that an effluent sample is acidified to preserve it in the field and is then digested with nitric acid to one-quarter volume. After digestion, the sample is subjected to analysis. Chemically, this technique measures both the dissolved metals as well as the portion adsorbed to suspended matter. This adsorbed portion is basically biologically inert, in that it is not readily available to aquatic life.

The Series 2 requirement that total recoverable metals be used to set permit limits is derived from 40 C.F.R. §122.45. However, there is no similar requirement that water quality standards be expressed as total recoverable metals. In fact, EPA, in the agency's oral presentation at the July 16, 1990 public hearing on the Board's proposed water quality regulations, encouraged the use of bioavailable speciation in setting the water quality standard for aluminum. In addition, in the national criterion documents EPA has recently produced, the Agency has recommended the use of a method to determine the acid-soluble form of constituents. For example, EPA's Selenium Criterion Document

contains a description of its acid soluble method and 11 toxicological and practical advantages the agency cites for the method. This description is included in Appendix A.

Currently, EPA states (40 C.F.R. 136.3, Table 1B) that use of the total recoverable, less vigorous acid digestion (Method 4.1.4 in "Methods for Chemical Analysis of Water and Wastes, 1979, EPA-600/4-79-020) is preferred in most cases over the much more vigorous "total" acid digestion (Method 4.1.3). The rigorous Method 4.1.3 digestion strips ions from their bound, biologically unavailable states (for example, on clay particles), and thus overestimates the bioavailable concentrations of the analyte in natural water. Even the total recoverable acid digestion will overestimate the concentration of bioavailable metals in natural waters since it still entails a hot, nitric acid digestion. Consequently, Method 4.1.4, while the better approximation of the bioavailable metal, is nevertheless an inaccurate predictor of the amount of metal available to life forms.

In addition to providing excessively stringent testing methods, EPA criteria development is suspect because it may measure the wrong form of the element. The criteria for metals are derived from bioassays where only filtered laboratory water and the chemical to be tested are present. Consequently, the criteria may bear no direct relationship to the most relevant toxic form of the element and cannot adequately protect aquatic ecosystems without being unrealistically stringent. Recent research has shown that the toxicity of many trace elements is a function of the chemical

form of the element. For example, copper toxicity is related directly to the free metal ion C^{+2} . In the case of copper, the free ion concentration depends on the formation of copper complexes with inorganic ions and organic complexing agents and generally comprises less than 1% of the total dissolved copper. [1] The EPA's recent water quality criteria documents recognize the importance of determining the metal form, such as the free copper ion, for determining toxicity, but criteria continue to be established based on analysis of total or total recoverable metals.

EPA's ambivalence about the propriety of the total recoverable method can be seen in its on-again, off-again program to develop a more biologically relevant measurement method. Early in 1986, EPA issued a draft acid-soluble metals method upon which industry commented. Although EPA ceased work on the protocol due to budget constraints, others have pressed ahead with this approach and at least two EPA regions (III and VI) have endorsed a metals measurement technique allowing permittees to reduce the measured value by an amount calculated on the basis of metal shown to be bound upon the solids fraction of the effluent. Now EPA is apparently reconsidering the issue of metals measurement methods used to determine attainment of state water quality standards. A draft of an EPA document that the Commentors believe is being circulated among the states discusses these methods, including the approaches used by Texas and Virginia to relate dissolved metals to total recoverable metals. This document is found in Appendix B.

Until EPA accepts an acid-soluble or dissolved metals method for determining the toxicological form of a pollutant, the Board should establish its criteria in terms of dissolved metals and allow permit limits, if expressed in terms of total recoverable metals, to be adjusted accordingly. EPA has recognized that states may set water quality standards in one measurement form, while permit limits are established using another form, and has encouraged states to develop a procedure reconciling the two. If not, EPA recommends that permit writers adopt one of four approaches to setting permit limits:

First, the permit writer could assume no difference between the dissolved or acid soluble phases and the total recoverable phase. This is the most stringent approach and would be most appropriate in waters with low solids, where the discharged form of the metal was mostly in the dissolved phase, or where data to use the other options are unavailable. Second, the permit writer could develop a site-specific relationship between the phases of metals by developing a relationship through review of information on instream metal concentrations. This approach requires concurrent sampling of both metal phases during periods reflective of the environmental conditions used to determine the WLA. Third, the permit writer could use a relationship developed by EPA from national data; this relationship is described in the national guidance for determining WLAs for toxic metals in rivers. This relationship requires knowledge of instream concentrations of total suspended solids at the environmental conditions used to determine the WLA. Fourth, the permit writer could use a geochemical model, such as the equilibrium metal speciation model MINTEQA2 [2].

To calculate a permit adjustment procedure appropriate for West Virginia, the Board could direct the use of the dissolved

metals method which has been approved for use in Texas or develop a scientifically defensible method for calculating the amount of metal adsorbed to solids and unavailable to aquatic life, such as the method proposed for adoption and use in Virginia. Both of these approaches are summarized in Appendix B. The Texas method, is set out in TAC Title 31, §307.6(c)(7), which provides:

Specific numerical criteria for metals and metalloids apply to dissolved concentrations, which can be estimated by filtration of samples prior to analysis or by converting from total recoverable measurements in accordance with procedures approved by the commission - such as in the latest revision of the EPA document, Water Quality Assessment: A Screening Procedure for Toxic and Conventional Pollutants in Surface and Ground Water (Revised 1985) (EPA/600/6-85/002 a and b).

The Virginia proposal provides a method for calculating dissolved metals from total recoverable metals by following specified statistical procedures and certain analytical requirements. Both the Texas regulation and the Virginia proposal acknowledge the crucial distinction between total recoverable metals and dissolved metals, and the greater relevance of the dissolved metals measurement to aquatic life. The Board should consider doing the same.

B. Analytical Variability

As the Board continues to lower the criteria in Appendix E, analytical detection limits and analytical variability become extremely important. When the criteria are translated into water quality-based effluent limits in NPDES permits, permittees are required to report in their Discharge Monitoring Reports the levels

of pollutants that are discharged and whether the detected levels exceed the effluent limits established in their permits. However, permittees cannot guarantee the accuracy of these reports when measuring very low concentrations. Each of the approved EPA procedures in 40 C.F.R. 136 has some inherent variability due to matrix interactions, instrument differences, single and multiple operator differences and other factors. These test methods, when applied to a complex industrial effluent, may not be able to accurately detect analytes in such matrices at the level of the water quality standard. Therefore, it is imperative that a regulated discharger be allowed to demonstrate compliance with a water quality standard by reporting results as below the practical quantification level (PQL) when the PQL is larger than the water quality standard.

The PQL is a concept recognized by EPA [3] which reflects or estimates the variability of test results from multiple laboratories. EPA defines the PQL of an analyte as the lowest concentration of that analyte that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions. In the absence of interlaboratory performance data for a given analyte, EPA has proposed that the PQL be equal to five to ten times the method detection limit ("MDL").

[3] The Commentors propose that the PQL, for purpose of determining permit compliance, be no less than a value ten times the mean of MDL values supplied by dischargers within an industry for a similar effluent matrix.

An MDL is defined as the minimum concentration of a substance that can be measured and reported with 99% confidence that the true value is greater than zero. [4] The MDL is derived from single laboratory, single operator determinations using approved methods in 40 C.F.R. 136. However, EPA has stated that: "... MDLs are a variable affected by the performance of a given measurement system. MDLs are not necessarily reproducible over time in a given laboratory, even when the same analytical procedures, instrumentation and sample matrix are used". [3] Thus, MDLs achievable under ideal conditions (i.e., single operator/single laboratory using distilled water matrix), as are sometimes given in the approved test procedures, may not be achieved for industrial effluent. Therefore, regulated dischargers must be allowed to establish MDLs that are specific to their individual discharges and laboratories.

The Board should provide that test results which establish that an analyte is not present at the PQL constitute compliance with permit limits for that parameter. Until the requisite testing for setting individual PQLS is done, the Board should establish a "regulatory" PQL to be applied in the following fashion:

Failure to detect any pollutant at a level 10 times the method detection limit for that pollutant, as determined in Methods 624, 625 or 40 C.F.R. 136, as appropriate, will be considered compliance with the permit limit for that pollutant.

C. Instream Biomonitoring And Narrative Biocriteria

Rather than establish criteria which apply to all waterbodies which are designated for a certain use, the Board should set water quality-based limits on a more site-specific basis. The best method of doing that is found in Section 9.1 of Series 1, which allows the setting of safe concentration values based upon "relevant aquatic field studies." The Commentors interpret the provisions of Section 9.1 to allow in-stream biomonitoring as a kind of toxicity test, to determine if an actual aquatic life use is attained and to appropriately defer or adjust water quality-based effluent limits in an NPDES permit to protect the attained use. Currently, the Board and Water Resources Section presume laboratory-derived criteria will protect designated uses, but in many cases, the actual use may be different from the designated use, and require different criteria than are provided by the Board's general criteria for the designated use. Because tens of millions of dollars could be unnecessarily spent treating an effluent that is not affecting stream life, the Board and Water Resources Section should not rely solely on chemical-specific data or whole-effluent bioassay results. EPA's Water Quality Standards Handbook states that:

EPA's laboratory-derived criteria may not always reflect the bioavailability and/or toxicity of a pollutant because of the effect of local, physical and chemical characteristic or varying sensitivities of local aquatic communities. [5]

Studies performed by some of the Commentors show that bioassay results can misleadingly suggest aquatic life uses are not

being attained when population monitoring shows that they are attained. By evaluating the status of aquatic life in a waterbody, the Division can more accurately assess the aquatic life use and determine whether effluent treatment is really necessary.

Directly monitoring indigenous aquatic communities is endorsed by EPA:

EPA is supporting those states using in-stream biological survey data to establish and measure water quality criteria for toxic and conventional pollutants, and is encouraging those states not active in this process to develop program plans for implementation. [6]

Many states have adopted such in-stream biomonitoring in varying degrees. For example, Ohio has recently proposed revisions to its water quality regulations which provide for biological criteria and in-stream biomonitoring to supersede chemical-specific and whole-effluent bioassay criteria. [7]

The Board's proposed revision of section 3.2.i incorporates satisfactory narrative biological criteria into water quality standards. The adoption of this narrative biological criteria represents Phase I of a State's eventual implementation of numeric biological criteria, according to EPA guidance. Principal goals of Phase I include:

. . . the development and adoption of narrative biological criteria into state standards for all surface waters . . . Definitions of terms and expressions in the narratives must be included in these standards . . . Adoption of narrative biological criteria in State standards provides the legal and programmatic basis for using ambient biological surveys and assessments in regulatory actions. [8]

Fully implementing Phase I requires studies of waterbodies to identify such things as natural levels of biological activity, which would lead to a thorough understanding of the biological performance of state surface waters. Until the appropriate studies are done, we believe the Board should defer the setting of more specific biological criteria for categories of uses or waterbodies.

To implement the biocriteria standard it has proposed, the Board should encourage the Water Resources Section to use in-stream biomonitoring results where sufficient data is available, as provided by the regulated community or the Chief's own staff, to determine whether aquatic life uses are being attained. Water quality standards, which are less precise measures of use attainment, could be curtailed in favor of the more specific evidence of use attainment represented by in-stream biological activity. To encourage this use of biocriteria in permits, the Board should add the following language to its section 6.1:

6.1. ...In-stream biological survey data can be used to assess the aquatic life use of a waterbody and determine appropriate concentration levels of constituents to support a designated or existing use.

D. Prohibition Of Acute Toxicity In Mixing Zones

The Commentors oppose the prohibition against acute toxicity to aquatic organisms in mixing zones that has been proposed for section 5.2.a.4. The Board's proposal contains no guidance as to what constitutes acute toxicity, or what organisms are considered in making that determination. Acute toxicity is

usually expressed in terms of lethality to organisms exposed to pollutants in the mixing zone. [9] Some states, such as Idaho, regulate acute toxicity in mixing zones by prohibiting exceedance of the 96 hour LC50, or the concentration which kills 50% of the organisms exposed to it for a 96 hour period. [10] The Board's proposed rule should be restated to clarify that acute effects are those which cause death, and that exceedance of the criteria found in Appendix E does not represent acute toxicity.

Another objectionable feature of the Board's proposal is the failure to identify the organisms to be protected against acute toxicity. It would be unfair to measure an effluent's toxicity based on test species that will never be exposed to that effluent or for test exposure times which are far greater than exposure time the organism will actually experience. For example, Idaho prohibits concentrations of effluent in the mixing zone which exceed the LC50 for "biota significant to the receiving water's aquatic community". [10] Without this clarification, acute toxicity might be determined using sensitive test species which are not indigenous to the waterbody in question. For non-native species, the stream water used as a diluent may contain substances that are as toxic as anything in the effluent.

The Board's proposed changes to §5.2.a.4 should be deleted, and a new §5.2.b.7 created, to read as follows:

Concentrations of pollutants within the mixing zone must not exceed the LC50 for the expected exposure duration for biota significant to the receiving water's aquatic community.

E. Designated Uses

The Board has proposed in section 6.1 that "[w]hen a discharge permit is to be issued all uses shall be assumed present unless the applicant demonstrates that the designated uses do not apply to the stream segment in question." This places the onus on every NPDES permit applicant to prove that it is not discharging into a trout stream and is not affecting a public water supply. Such an approach is excessively burdensome and inconsistent with Appendices A through D, which specifically identify waterbodies which qualify as waters designated for trout, public water supply, power production, and recreational water contact uses. These lists are unnecessary if each of these uses is presumed present for every state stream.

The general uses that could reasonably be assigned to the waters of the state are the national water quality goal uses of protection and propagation of fish, shellfish, wildlife and recreation in and on the water, or the "fishable/swimmable" uses. This was evidently the Board's intent in proposing in section 6.1 that all waters of the state be designated for Aquatic Life (Category B) and Water Contact Recreation (Category C) unless the standards provide otherwise. Other uses may apply, based on the Board's lists of waterbodies that are designated for particular uses or the permit writer's belief that other uses must be protected. However, there is no reason to presume that all uses apply in every situation.

The Board's designation of all uses for waterbodies receiving point source discharges is also objectionable because it is inherently unfair. Designated uses should be determined based on achievable stream quality, not on who is discharging into the stream. There is no reason that NPDES permit holders should be held to a different standard than non-point dischargers. Under the Board's proposed language, point source discharges might be required to perform a use attainability study or an analysis of uses and water quality such as that ordinarily required to obtain a Series 6 variance. Furthermore, Series 6 variances obtained from the Board one year will presumably have to be reestablished to a permit writer's satisfaction the next year if the permit is due for reissuance. Such disproving of uses will be expensive and time consuming.

Eliminating the proposed presumption in favor of all uses applying to each state waterbody would not interfere with a permit writer's ability to establish permit limits based on the actual or designated uses of a particular waterbody. It would, instead, relieve permittees of establishing that clearly inapplicable uses do not apply to waterbodies into which it discharges. The designation of waterbodies for fishable/swimmable uses is sufficient to meet national water quality goals, and still allows for protection of other uses as appropriate. The Board should withdraw the second sentence of its proposed amendment to section 6.1.

F. Time Extensions To Comply With Water Quality-Based Effluent Limits When Treatment Technology Is Not Available.

Many of the existing and proposed water quality criteria are at or below the analytical detection level. At such low levels, not only is it impossible to accurately measure the concentration, but in some cases wastewater treatment technology is not available to consistently and efficiently remove pollutants to the low levels required to meet the criteria. An example of this problem is selenium. The criterion of 5 ug/L is lower than any demonstrated treatment technology has been able to consistently achieve. Research sponsored by the Electric Power Research Institute has identified coprecipitation with amorphous iron oxyhydroxide as a potential technology for selenium removal. In pilot tests of this technology, the process removed 80-95% of Se^{+4} but only 5-15% of Se^{+6} . [11]

A provision is necessary to allow for an extension of time to meet a water quality-based effluent limit when wastewater treatment technology is not available to achieve the limits. The Chief presently has statutory authority to grant compliance schedules for persons not able to meet water quality standards. See W.Va. Code §20-5A-3a. This should be reflected in regulations which specify those situations in which this relief from water quality-based limits will be granted, such as inability to meet water quality-based limits due to a lack of adequate removal technology.

G. Water Use Categories\Special Criteria

Category B of the water use categories of Section 6.3 should be revised to include a category for "other warmwater streams" consisting of very small creeks and stream segments which typically flow for only a portion of the year, mainly in response to precipitation or snow-melt. This category of stream would have a more limited flow than B3 Small Non-Fishable streams, but would not run only in direct response to precipitation as do wet weather streams. Because the flow of water in such streams is intermittent, stream use by fish is incidental and normally limited to minnows, darters, and the like. Benthics are sparse or nonexistent. The survival and reproductive stages of benthic invertebrates reflect the short term availability of water. Emergence periods are very short when water is available and resting stages resistant to desiccation are very long when no water is present. These strategies are vastly different from the relatively longer emergence periods and shorter resting periods of benthic invertebrates inhabiting warmwater waterbodies in categories B1 and B3. For this reason, a new category for such intermittent streams should be added to Section 6.3, as follows:

6.3.e Category B5 - Other Warm Water Streams -

Streams or stream segments which, because of their size, water flow patterns, or recurrent lack of natural sources of water, do not offer sport fishing and are used by minnows, darters, and other aquatic life only on an incidental basis. Criteria in Appendix E apply to this use category as indicated, provided that the designated uses of downstream waters are not adversely affected.

The Board may also wish to eliminate the definition of "intermittent streams" in Section 2.5 and related references in favor of references to the B4 category.

The criteria in Appendix E of Series 1 should be revised to reflect those found in Appendix E of these comments, which are based on EPA calculations for Final Acute Values ("FAVs") except for iron and chloride. Chloride criteria was derived from a report done by the University of Kentucky, while the BPA-reactive ferrous iron criteria was derived from a study done for the Board, after approval by ASTM of the analytical method found in Appendix B. These FAVs reflect toxicity data from laboratory bioassays using warm water species. The calculations are greatly influenced by the lowest available bioassay data value, resulting in FAVs which are often overprotective of the biota in the water body. Because these criteria can be overprotective, state-wide criteria should be resolved site-specifically as necessary under the Board's administrative regulations in Series 4.

H. Regulating Only Substances Harming Designated Uses.

The Board must establish criteria for all parameters on the basis of harm to designated uses. Whenever a state revises its water quality standards, it must review all available information and data to first determine whether the discharge or the presence of a toxic pollutant is interfering with or is likely to interfere with the attainment of the designated uses of any stream segment. A state must adopt criteria for all listed toxic pollutants "the discharge or presence of which in the affected waters could

reasonably be expected to interfere with those designated uses adopted by the State". Clean Water Act section 303(c)(2)(B). If a state does not expect the pollutant to interfere with the designated use, section 303(c)(2)(B) does not require a numeric standard for that pollutant. It does not need to add parameters to Appendix E simply because EPA has developed criteria for them.

The Board has proposed establishing numerical water quality standards for several pollutants for which criteria were not previously established. Before establishing such standards, the Board is obligated to justify its choice of pollutants under federal and state statutory standards. Federal water quality standards regulations provide that "States must adopt those water quality criteria that protect the designated use". 40 C.F.R. §131.11(a). Therefore, the fundamental legal standard for water quality criteria is that they support certain "uses" that have been designated by the state. If the discharge or presence of a particular pollutant cannot reasonably be expected to interfere with a designated use, then the authority of the state to impose numerical criteria does not exist.

The Board cannot conclude that a particular pollutant will interfere with designated uses unless it possesses and considers some evidence to support that conclusion. Therefore, the Board should explain why the pollutants for which it has developed criteria could reasonably be expected to interfere with designated uses of waters of the state. Until then, developing numerical

criteria for those pollutants is not consistent with the general policy of the State Water Pollution Control Act.

I. Regulating Human Health Constituents At The 10^{-6} Risk Level.

The Board should reconsider its selection of the 10^{-6} risk level for known or suspected carcinogens. The choice of a risk level, as EPA has recognized, is a policy matter rather than a scientific or technical decision. Nevertheless, it is a decision the state must make in a rational manner. Information the Board should consider in making this decision includes the risk decisions made by other state and federal authorities. For example, California uses 10^{-5} as the risk criterion for limiting discharges of pollutants to surface and groundwater under its Safe Drinking Water and Toxic Enforcement Act (Proposition 65). [12] Many federal decisions on risk have allowed residual individual risks of 10^{-4} or even more. [13]

Use of a 10^{-5} risk level is also supported by a more fundamental argument. The current rate of cancer mortality in the United States approaches 20%. Even after removing the most obvious lifestyle contributions to the cancer rate (tobacco and alcohol), the rate would be in the vicinity of 10% (10^{-1}). A reasonable public health goal would be to limit the contribution of industrial sources to 1% of that value (10^{-3}), and a reasonable rule of single hazard would be to limit it to 1% of that value, or 10^{-5} . This argument takes into account the fact that any one person is very unlikely to be simultaneously exposed to a vast array of cancer hazards, all at the maximum permitted levels. [14]

In the context of the federal Safe Drinking Water Act, EPA has described what it considers to be "safe" levels of contaminants:

As mentioned above, for contaminants in drinking water, the target reference risk range for carcinogens is 10^{-4} to 10^{-6} and the MCLs EPA is promulgating in this notice generally fall in this range. EPA considers these to be safe levels and protective of public health. This is supported by the concept expressed by the WHO 1984 Guidelines for Drinking Water Quality, where it selected a 10^{-5} guideline value, and then explained that the application could vary by a factor of ten (i.e., 10^{-4} to 10^{-6}).

52 Fed. Reg. 25,700-01 (July 3, 1987). As the table below shows, EPA's risk levels for proposed drinking water standards have been in the 10^{-4} to 10^{-5} range.

RISK LEVELS FOR PROPOSED EPA DRINKING WATER STANDARDS

Chlordane	7.43×10^{-5}
Ethylene Dibromide	1.21×10^{-4}
Heptachlor	5.14×10^{-5}
PCBs	1.01×10^{-4}

Virginia has chosen risk levels ranging from 10^{-3} down to 10^{-5} . In the most recent such decision, establishing a water quality standard for dioxin, Virginia chose a 10^{-5} risk level. In light of these precedents, the choice of 10^{-6} is inconsistent with the risk level chosen by other states and should be reconsidered for West Virginia.

In light of the evidence, 10^{-5} rather than 10^{-6} is a more appropriate risk level for across-the-board application. But whatever the ultimate decision, the Board is required to assess the

available evidence that bears on such policy decisions and make a rational choice, rather than choose an arbitrary number. If the Board were to insist on an across-the-board 10^{-6} risk level, then it certainly ought to incorporate flexibility by making that level only a rebuttable presumption. A different risk level might then be established for a particular water body, based on economics, technical considerations, or other relevant factors.

J. Narrative Criteria For Wetlands

The Commentors support the Board's establishment of a new use category for wetlands. Criteria developed for open bodies of water are inappropriate for wetlands, which are significantly different from a biological and hydrological standpoint. The Board should proceed carefully in setting criteria which will reflect actual uses and existing water quality.

K. Harmonic Mean Flow

West Virginia has included in its proposed water quality standards a provision for use of the harmonic mean flow as the critical design flow for carcinogens. Section 8.2.c (Specific Water Quality Criteria - Criteria for Toxicants), as proposed, provides:

If a permit applicant requests and provides the necessary information, critical design flow for carcinogens shall be the harmonic mean flows. In-stream compliance with carcinogen standards shall be determined by computing the arithmetic mean of all samples analyzed for the carcinogen.

In evaluating this proposal, it is helpful to examine the EPA's position regarding use of harmonic mean flow and why it, instead of the mean annual flow, the 7Q10, or the 30Q5, is the

proper flow when dealing with carcinogens. EPA has defined and explained harmonic mean flow as a long term mean flow value calculated by retrieving several years of daily flow records, taking the reciprocal of each value, calculating the average, and taking the reciprocal of the average.[15]

In discussing the relation of harmonic mean flow to waste load allocations, EPA states:

The duration of exposure assumed in deriving the reference ambient concentration (RAC) [defined as the concentration of a bioconcentratable chemical in water which will not cause adverse impacts to human health], should affect the selection of the design conditions and may affect the point of application of a criterion. This duration of exposure or averaging period underlying the derivation of the water quality criterion or RAC is the time period required to obtain an adverse effect or an unacceptable risk from marginally exceeding the RAC or criterion. The duration should have a logical connection with the design flow. Often, however, the flow averaging period only approximates the criterion averaging period, as for example when the 7Q10 is coupled with the 4-day average aquatic life criterion.

The duration of the averaging period for determining the WLA [waste load allocation] should be consistent with the assumptions used to derive the criterion or RAC being applied. For example, the human health criteria for carcinogens are based on the assumed risks associated with a lifetime exposure which can consist of a series of short term exposures or a continuous exposure over a 70-year period.

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Therefore, the averaging period underlying the RAC is 70 years for carcinogens, but may be pollutant specific or unknown for noncarcinogens.

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The appropriate dilution design conditions to use in calculating a WLA depend on the averaging period of the exposure. **For averaging periods greater than one year, as is the case for carcinogens, the harmonic mean flow should be used as the critical design flow.** For averaging periods of one month to one year, the 30-day

flow with an appropriate recurrence interval (e.g. five or ten years) should be used; for averaging periods less than one month, the 7Q10 as the design flow should be used.

. . . .

The design flow for calculating average receiving water concentrations over a several-year period is selected as the flow that results in the average dilution obtained over that period. Since the dilution obtained by a given flow is proportional to the reciprocal of the flow (the instream concentration is equal to the discharge load divided by the stream flow), a simple mean of the daily flows does not characterize the average dilution obtained. The harmonic mean flow, is the flow at which average dilution is obtain, and this flow should be used as the design flow for multiple-year averaging periods as is the case for carcinogens.

The recommendation of the mean flow is the best estimator of the lifetime exposure to carcinogenic pollutants. The adverse impact of carcinogenic pollutants is estimated in terms of receptors' (human) lifetime intakes. To be within the acceptable level of a lifetime exposure of any carcinogen, such intakes should not exceed the RAC during "the lifetime" of the receptor. A lifetime for carcinogenic pollutants is defines by EPA as 70 years or approximately 365 (days/years) x 70 years. In estimating the lifetime exposure, EPA assumes that people will catch and consume fish at random times spaced over a year. Consumption over 70 years of random fishing can be best estimated by calculating the exposure of fish to the average concentration in the receiving water which in turn is best calculated by using the harmonic mean flow. Once fish tissue concentrations approach an equilibrium with the average receiving water concentrations, variations in the tissue concentrations will reflect variations in the receiving water, but the average will not change.

. . . .

The harmonic mean flow represents the long-term average based flow of a stream minimizing the rainfall-induced spikes of high flow which can skew the annual arithmetic average flow. This average base flow is the flow which aquatic life is subjected to for the majority of the year and is more appropriate for relating to bioconcentration. The difference between the arithmetic and harmonic mean flow is greatest in rivers characterized by long periods of low base flow followed by short periods of high flows.

. . . .

In order to develop some quantitative sense of how a long-term harmonic mean flow of any stream compares with its 7-day 10-year low flow, EPA's Assessment and Watershed Protection Division and the Risk Reduction Engineering Laboratory at Cincinnati, Ohio analyzed flow records of 60 streams. These are the same stream flow records which had been analyzed for stream design flow condition for aquatic life protection criteria as listed in Book VI, Design Conditions: Chapter 1, Stream Design Flow for Steady-State Modeling [EPA's Office of Water Regulations and Standards, August, 1986]. Based on the long-term harmonic flow and 7-day 10 year low flow estimated for these sixty streams, the long-term harmonic mean flows of all 60 streams were equal to or greater than 2 times the 7Q10 low flow. Fifty-four of the streams' harmonic mean flows were equal to or greater than 2.5 times their 7Q10 low flows. Finally, 40 of the 60 streams' harmonic mean flows were equal to or greater than 3.5 times the 7Q10. [16] (emphasis added)

In its Technical Support Document For Water Quality-based Toxics Control issued in March, 1991, EPA pointed out:

The harmonic mean is always less than the arithmetic mean. The harmonic mean is the appropriate design flow for determining long-term exposures using steady-state modeling of effluents. The arithmetic mean flow is not appropriate as the design flow since it overstates the dilution available. Extreme value statistics (such as 7Q10 or 30Q5) are also not appropriate since they have not consistent relationship with the long-term mean dilution.... [17] (Emphasis added)

It should be noted that although EPA recommendation for use of the harmonic mean flow contrasts both higher and lower flows, the overall focus of the documents strongly implies that the only real choice is between use of the harmonic mean or the mean average flow. [18] Significantly, Virginia and Maryland both used the mean average flow for determining their water quality standard for dioxin, which EPA approved as adequately protective of human

health. [19] Use of a lesser flow, such as 7Q10, for carcinogens is clearly inappropriate.

Kentucky used the harmonic mean flow its water quality standards in the following manner:

. . . .

(2) On occasion surface water quality may be outside of the limits established to protect designated uses because of natural conditions. When this condition occurs during periods when stream flows are below the flow which is used by the cabinet to establish effluent limits for wastewater treatment facilities, a discharger shall not be considered a contributor to instream violations of water quality standards, provided that treatment in compliance with permit requirements is maintained.

(3) Governing flows for water quality-based permits. The following stream flows are to be utilized when deriving KPDES permit limitations for the protection of surface waters for the listed uses and purposes.

...

(c) Domestic water supply protection - Harmonic mean for cancer-linked substances, 7Q10 for noncancer-linked substances, determined at points of withdrawal.

(d) Human health protection from fish consumption only - Harmonic mean for cancer-linked substances, 7Q10 for noncancer-linked substances. [20]

. . . .

Clearly this board has sufficient support and precedent for use of either the harmonic mean flow or the mean average flow in establishing water quality standards for carcinogens in West Virginia. Equally as clearly, use of the 7Q10 in such situations is not justified.

L. Specific Criteria

Sufficient data is available to allow revision of certain numeric limits established in Appendix E of Series 1. The justifications for some of these revisions are set forth below.

1. Selenium

Because of certain deficiencies in EPA's derivation of the selenium limit in its national criterion document, the Board should revise the 5 ug/L selenium limit. A review of the national criterion document for selenium indicates that the reduction of the national criterion was based on two site-specific field studies - Belews Lake, North Carolina, and Kesterson Reservoir in California. [21] Belews Lake is a large impoundment with long retention time. EPA admits that the Belews Lake studies ". . . cannot establish a cause-effect relationship [with selenium] because a variety of other inorganic and organic materials undoubtedly entered the lake with the selenium." [22] In spite of this conclusion, EPA unsuccessfully tries to establish a cause-effect relationship by citing Finley's 1985 feeding experiment with a total of eight bluegill fish. [23] U.S. EPA cites the death of three of four bluegills (the other four were controls) fed mayfly nymphs as evidence that selenium contaminated the food chain in Belews Lake and eventually caused effects in fish populations. [24] However, EPA effectively discredits the Finley study, by admitting that ". . . the nymphs also contained one or more toxicants in addition to selenium." [25]

Selenium bioconcentrated in Kesterson Reservoir to such an extent that the fish community experienced mortality and reproductive failure. The most likely bioconcentration mechanism was methylation in the sediments, with transfer up the food chain. In contrast to lakes, bioconcentration in streams and rivers is unlikely. Sediments rarely become anoxic and methylation does not occur. Most selenium would be flushed out of the stream system with little impact to aquatic life.

Other field studies show aquatic life uses are attained at selenium concentrations much greater than 5 ug/L. For instance, with selenium input into Adair Run, Virginia, a mountain stream averaging 42 ug/L Se in a nine-month period (Range: 14 to 85 ug/L Se), no detrimental impact on structural and/or functional responses of aufwuchs, invertebrates, or fish was noted. [26, 27] The abundance, diversity, and faunistic composition of fish in the stream segment receiving selenium were similar to the abundance, diversity, and composition in the uninfluenced reach. [28]

In Crooked Run, a first and second order West Virginia stream, the aquatic life use of the creek has been maintained in spite of selenium inputs averaging 14 and 19 ug/L at two biological sampling stations. [29] Over a four year time period, selenium in the creek ranged from 0 to 75 ug/L (N=47) while benthic macroinvertebrate monitoring data on Hills diversity measures, evenness, species, richness, and proportional numerical dominance of the most abundant tax on show a healthy macroinvertebrate community. Reproduction and growth of benthic macroinvertebrates

and fish are within normal ranges. Nevertheless, NPDES permittees must reduce the selenium concentration in the creek to the 5 ug/L criterion established in the Board's water quality standards. Such a reduction is needless and, since the creek was dry in the 1988 drought, compliance with the Board's criterion will actually degrade the stream's use.

Data exists which confirms that selenium in excess of water quality standards will not necessarily affect biological activity in a stream, as established by the justification provided to this Board for the 62 ug/L selenium criterion for Little Scary Creek, a small tributary to the lower Kanawha River. [30] In fact, it was demonstrated that a 92 ug/L selenium limit, as a 30-day average, would support the uses of that creek.

Better justified state-wide selenium criteria can be derived. As shown in the table below, EPA has already taken steps to provide these criteria in the national criterion document [31], although EPA deviated from its normal derivation procedures to arrive at the 5 ug/L total selenium criteria by interpreting the effects of forms of selenium. EPA also specifically derived criteria using bioassay data for Se^{+4} and Se^{+6} .

	<u>Se⁺⁴</u> <u>ug/L</u>	<u>Se⁺⁶</u> <u>ug/L</u>	<u>Existing National</u> <u>Total Se Criterion</u>
Criterion Maximum Concentration	186	12.8	19.98
Average (Criterion Continuous Concentration) (non-trout stream)	44.7	9.7	5
Average (Criterion Continuous Concentration) (trout stream)	44.6	9.7	5
Final Acute Value	371.8	25.6	39.96

If the available laboratory bioassay data for Se⁺⁴ and Se⁺⁶ are carefully reviewed, and only quality data points used, ample justification is available for substantially increasing Se⁺⁴ and Se⁺⁶ criteria. For instance, by using only quality data points, the maximum, average and final acute values for Se⁺⁴ should be 660, 84 and 1329 ug/L, respectively. [31, 32, 33]

Because the Board's existing selenium criterion is not scientifically defensible, the Board should take the following action:

- a) increase its statewide selenium criterion of 5 ug/L for warmwater aquatic life. This revision should, at a minimum, apply to lotic (flowing) water bodies.
- b) Derive a statewide criterion using only quality data points. The Commentors offer their technical expertise to work with the Board to derive such a criterion.
- 3) Recognize that aquatic life uses at even higher selenium concentrations are possible to justify on a site-specific basis.

2. Aluminum

The Commentors support the changes that have been proposed for the aluminum criteria. The change to 748 ug/L is eminently justifiable based on available data, [34] and frees dischargers from having to comply with a criterion that is often lower than background concentrations in state streams.

3. Mercury

The Commentors support the changes proposed by the Board, which recognize that methylmercury is the proper form of mercury for which warmwater fish and trout use criteria should be set.

4. Arsenic

The Commentors support the change in the arsenic criteria for public drinking water uses, and urge deferral of further changes until the review of EPA's IRIS database and other information concerning arsenic is completed. The potential dangers posed by arsenic have been overstated. For example, arsenic occurs naturally in the environment and is commonly found as arsenic sulphide or as an impurity in various minerals and as arsenate or arsenite. It has been found in both groundwater and surface water and its presence can result from both natural processes and industrial activities. [35]

The Board's current human health standards for arsenic are based on EPA's 1980 Ambient Water Quality Criteria Document for Arsenic. These proposed values were calculated based upon a 1968 study of increased incidence of skin cancers in humans in Taiwan. [36] Accordingly, the standards are based on the assumption that

arsenic is carcinogenic. However, in the National Academy of Science Safe Drinking Water Committee's analysis of the issue, which was done subsequent to development of the water quality criterion, it was determined that high levels of exposure to pentavalent arsenic for long periods of time are required to produce carcinogenicity. This type of exposure does not occur via drinking water. Other studies have also questioned the carcinogenicity of arsenic. [37, 38, 39] For a detailed technical analysis of this issue see Appendix C to these comments.

EPA is now reviewing the drinking water maximum contaminant level (MCL) for arsenic in light of a number of controversial issues. These include:

- a. the carcinogenicity of arsenic;
- b. whether or not arsenic is an essential nutrient; and
- c. the determination of the threshold level below which no adverse health effects occur.

There is evidence that arsenic is an essential element in certain animal species and potentially in humans. [40] Arsenic has been found to be a normal constituent of food; seafoods tend to have particularly high arsenic levels. In fact, seafoods dominate total arsenic intake by humans. [41] It has also been known for many years that the arsenic found in seafoods is present in a relatively non-toxic form compared to inorganic arsenic, and arsenic at low concentrations in the diet enhances some parameters of growth and development. Though arsenic has not been documented as an essential element, the issue remains controversial and deserves the Board's attention. [42]

To date, human health authorities have published standards for arsenic, in both waters and foods, which are related only to total arsenic, despite the fact that it is known that different arsenicals exhibit very distinct toxicological properties. In reference to the determination of an appropriate threshold level for arsenic, it has been learned that the dose-response curve for skin cancers may be sublinear, or even biphasic. [43] If this is the case, cancer potency factors selected for arsenic will overestimate the risks.

The data indicate that there is substantial doubt about the carcinogenicity of arsenic, its role as an essential nutrient, and the appropriateness of currently proposed threshold values. The Board is wise to refuse to adopt standards which are based on questionable scientific information and instead adopt EPA's drinking water maximum contaminant level for arsenic of 50 ug/l as a standard for water and fish tissue consumption. In the Toxicological Profile for Arsenic it is stated that 27 of the 50 states have established specific water quality standards for arsenic. [44] The majority of these states have set the water quality standards for arsenic at 50 ug/l; however, for a few states, the values range from 5 to 1000 ug/l.

M. New Criteria - Dioxin

1. Background

In the late 1970's, at the request of the U. S. Environmental Protection Agency, Dr. Richard Kociba conducted laboratory tests exposing rats to high levels of dioxin. Using

1975 criteria, Dr. Kociba and Dr. Robert Squire then independently calculated the tumor incidents for the EPA. Based on those calculations, which were in basic agreement, and using a model that assumed a cancer risk at any dose, EPA established its estimate of the cancer potency for dioxin. That factor and an acceptable risk factor were used in arriving at a hypothetical dose level at which cancer risk would increase at no more than the chosen rate. EPA then made certain assumptions and calculations regarding accumulation of dioxin in water and fish and thereby established its water quality standard. Interestingly, EPA and the Food and Drug Administration have reached different conclusions in estimating the cancer potency of dioxin, and therefore different acceptable dosage levels, partly because EPA based its calculations on surface area of the body while FDA utilized body weight. FDA's cancer potency factor shows a 5.4-fold decrease when compared with the EPA cancer potency factor.

In late 1989, Dr. Squire and panel of pathologists re-examined the pathologic slides from the Kociba study, but used current criteria for tumor determinations. They found a substantially reduced tumor count and classified dioxin as a much weaker cancer threat than assumed by EPA originally. Their studies indicate that, contrary to EPA's original assumption, there is indeed a threshold level of exposure which must be reached before any carcinogenic responses are to be found.

Based on that reevaluation, in October, 1990, a conference of leading scientists from around the world meeting at

Cold Spring Harbor Laboratory, Long Island, New York, reached the same consensus that there is a minimum threshold level for exposure to dioxin below which no adverse human effects will occur.

In calculating water quality criteria, EPA utilized a risk level of one possible increased incident of cancer in one million people exposed to a dose level assumed to result from a water quality standard for dioxin of 0.014 ppq. That risk level, stated as 1×10^{-6} , does not mean that one person out of every million who is exposed will contract cancer but only that the risk of cancer is increased by one in a million. To put this in perspective, we note the background risk of cancer in the United States today is approximately one in four, which means that of a population of a million people, 250,000 are at risk to develop some form of cancer in their lifetimes. Using a dosage that results in a risk of one out of 100,000 (1×10^{-5}) instead of one out of one million (1×10^{-6}) for setting a water quality standard for dioxin would mean the current background risk would be increased from 25% to 25.001% (at 1×10^{-5}) instead of 25.0001% (at 1×10^{-6}). That risk level (1×10^{-5}) has been utilized by EPA, the Food and Drug Administration, and other federal and state agencies in establishing regulatory limits for various situations.

As EPA stated last month while approving a 1.2 ppq water quality standard for dioxin for Virginia, "EPA's methodology for estimating the upper limit of the cancer risk of [dioxin] has involved making a number of specific scientific assumptions in the absence of a clear scientific understanding."

2. Elements Of A Water Quality Standard For Dioxin

There are six basic elements that should be considered in deriving a water quality standard for dioxin. These elements are:

- cancer potency factor (or acceptable daily intake)
- risk level
- bioaccumulation/bioconcentration factors
- fish consumption rate
- water ingestion rate
- dioxin cooking losses

Each of these elements is addressed in detail below.

a. Cancer Potency

The principal element in deriving a water quality standard for dioxin is its carcinogenic potency, expressed as a cancer potency factor ("CPF"). [45] The CPF is developed from laboratory animal data. All of the modern human risk assessments for dioxin are based upon the research data developed by Dr. Richard Kociba in the late 1970s. Dr. Kociba exposed Sprague-Dawley rats to high levels of dioxin, and then extrapolated the resulting dose-response data to yield a predicted human dose-response at much lower levels of exposure.

Based upon the research data, Dr. Kociba calculated tumor incident in the exposed rats. EPA asked Dr. Robert Squire (currently of Johns Hopkins University) to independently examine the Kociba tissue slides. The tumor incidence calculated by Dr. Kociba and Dr. Squire, based on 1975 criteria, were similar. From the tumor incidence, EPA utilized its linearized multistage model

("LMS") to estimate human carcinogenicity. [46] The LMS model assumes that any dose, no matter how small, poses some level of risk from carcinogenesis (the "nonthreshold" assumption). In other words, EPA's model forces the dose-response correlation line to pass through the origin (zero dose and zero incremental risk). [47] The LMS model calculates the maximum possible linear slope, at a 95 percent upper confidence limit, yielding a maximum estimate for potency. This estimate was based upon tumors in the liver (the most sensitive organ in the test animals) of the female specimens (the more sensitive gender). [48] The EPA cancer potency estimate was 1.56×10^5 (mg/kg-day)⁻¹. [49] EPA's CPF is equivalent to a risk-specific dose ("RsD") at a 1×10^{-5} risk level of approximately 0.06 picograms/kilogram body weight/day ("pg/kg/day"). The risk-specific dose is the average exposure level which is predicted, based upon the nonthreshold assumption, to increase the cancer incidence rate by no more than the chosen risk level.

The FDA cancer potency estimate is based upon more scientifically defensible assumptions than the assumptions used by EPA, but is still very conservative. The FDA extrapolated the Kociba data from rats to humans on the basis of body weight, which results in a 5.4-fold decrease in the CPF when compared with the EPA CPF. The FDA utilized a linear extrapolation model and obtained a CPF of 1.75×10^4 (mg/kg/day)⁻¹. [50]

i. The recent reevaluation of the Kociba data demonstrates that dioxin is, at worst, a weak carcinogen.

The original pathological examination of the Kociba female rat liver slides was based on 1975 criteria. Since that

time, much has been learned about both carcinogenic and noncarcinogenic tumors. Some lesions that were believed to be tumors in the 1970s are now known to be relatively common spontaneous benign lesions in laboratory animals. The current National Toxicology Program criteria are the result of additional experience in animal testing and research conducted by many investigators in recent years on the specific lesions and tumors involved. The classification system is now accepted by U.S. government agencies.

In late 1989, Dr. Squire reexamined the Kociba slides at the request of the Scientific Advisory Panel ("SAP") to the Maine Board of Human Services. Dr. Squire concluded that, based upon the current criteria, there would be a substantial change in the tumor count in the Kociba test animals. However, rather than announcing the results of his own review upon which regulatory decisions might be based, Dr. Squire recommended that an impartial Pathology Working Group ("PWG") be convened.

Under the auspices of PATHCO, an independent organization which frequently performs pathological evaluations, a panel of highly respected pathologists, all of whom have special expertise in this area, was convened in March of 1990. The PWG findings were consistent with Dr. Squire's earlier reevaluation. [51]

Dr. Squire summarized the PWG findings in a May 24, 1990 letter to LaJuana S. Wilcher, EPA's Assistant Administrator for Water, as follows. [52]

Based upon currently accepted [National Toxicology Program] criteria, the PWG findings

indicated fewer total numbers of hepatocellular tumors and fewer malignant hepatocellular tumors than originally reported. There was unanimous agreement on malignancy for only one tumor.

....

The new incident figures are....based upon current scientific evidence and will be the most accurate and appropriate for risk assessment procedures.

The PWG characterized dioxin as a weak oncogen (a weak carcinogen). [53] The PWG findings are in contrast to EPA's continuing position that dioxin is a strong or potent carcinogen. EPA, in approving Virginia's 1.2 ppq standard, acknowledged the "reevaluation would reduce FDA's and EPA's cancer risk estimate by a factor of approximately 3." [54]

ChemRisk, a consulting group in toxicology with widely-recognized expertise in risk assessment, has utilized EPA's LMS model to reevaluate carcinogenic potency based upon the PWG results. The LMS model calculates a RSD of 1.0 pg/kg/day at a 1×10^{-5} risk level. [55]

- ii. The PWG findings go beyond the calculation of a CPF and demonstrated that there is a safe human exposure level for dioxin.

The implications of the PWG findings go beyond tumor incidence. The PWG found that increased tumorigenicity only occurred where there was overt hepatotoxicity (liver toxicity). PATHCO prepared two reports on the hepatotoxicity observed in the Kociba test animals. [56] Those reports demonstrated that increased tumor incidence is closely correlated with hepatotoxicity.

Toxicity does not occur at low levels. Since any human exposure to dioxin would be at levels far below the exposure levels which produced liver toxicity in the Kociba studies, the Kociba data do not predict a human carcinogenic response to dioxin exposure. [57]

The most important indication from the findings is that there is a safe human exposure level for dioxin. The PWG findings contradict the linearity of dose-response assumed by EPA's LMS modeling approach at the low doses characteristic of human exposure. The occurrence of tumorigenicity only at overtly toxic doses shows that dioxin demonstrates a threshold of exposure, below which no carcinogenic endpoint is expected. [58] The existence of a threshold is inconsistent with the nonthreshold nature of the LMS model and demonstrates that the application of that model is very conservative, irrespective of what CPF is chosen.

iii. There is a growing consensus among leading experts, including some EPA scientists, that dioxin does not represent a human health hazard at levels encountered through environmental exposure.

The October, 1990 Banbury Conference at Cold Spring Harbor Laboratory, Long Island, New York, addressed dioxin risk assessment. A number of leading scientists from around the world participated. The results of the Banbury Conference lead to the conclusion that a safe dose in the 1-10 pg/kg/day range is fully protective of human health.

Dr. Linda S. Birnbaum, Director of EPA's Environmental Toxicology Division, notes in an internal EPA memorandum [59] that consensus was reached at the Banbury Conference on several issues

concerning dioxin and its effects upon humans. In particular, her memorandum points out there was a consensus that there is a minimum threshold level below which no adverse human effect will occur. She notes that "[preliminary] calculations lead to agreement that exposure to humans of 1-3 pg/kg/day [picograms/kilogram body weight/day] would be essentially a [no observed effect level.]"

Although some controversy has arisen about possible abuse of the Banbury results for publicity purposes by both sides of the dioxin issue, it should be noted that Dr. Birnbaum's memorandum was an internal agency working document, not a publicity release, and therefore is untainted by that controversy.

It is apparent that the conclusions from the Banbury Conference are in close agreement with the RSD of 1.0 pg/kg/day derived from the PWG results.

iv. Recent scientific developments lead to a better understanding of the biochemical effects of dioxin and support the conclusion that dioxin acts by a threshold mechanism.

The Banbury Conference produced agreement that the possible carcinogenicity of dioxin, as well as other toxic effects, appears to be mediated through the Ah receptor and the induction of cytochrome P450. This means that for dioxin to have an effect it must reach a specific site, bind to a receptor, move into the nucleus, and bind to the genetic material. Several thousand receptors must be occupied before any biological or toxic effect is seen. At doses which are too low to cause induction, no effects can occur. Therefore, a "safe" dose for dioxin, otherwise known as a threshold dose, can in principle be established. [60] Thus, new

scientific information on the specific biological mechanism for the possible human carcinogenic response is in close agreement with the conclusion from the Kociba bioassay data that dioxin acts through a threshold mechanism.

- v. Further information from the human epidemiological database supports the conclusion that there is no risk to human health from environmental exposure to dioxin.

Epidemiological work on dioxin has been based upon studies of human exposure from occupations in chemical manufacturing, exposure data relating to herbicide sprayers, studies of human exposure due to a 1976 chemical plant accident in Seveso, Italy, exposure of residents of Times Beach, Missouri, and Viet Nam veterans' exposure to Agent Orange in Operation Ranch Hand. In these studies, the concentrations of dioxin to which people were exposed were much greater than would typically be encountered in the environment. In addition, the populations evaluated in many of these studies were exposed to multiple chemicals, thereby complicating the assessment of possible carcinogenicity due to dioxin exposure. Throughout these studies, no credible carcinogenic human effect has been observed. [61]

- vi. Evaluation of possible non-carcinogenic endpoints yields similar results.

In addition to possible human carcinogenicity of dioxin, some have expressed concern about possible non-carcinogenic endpoints. ChemRisk has summarized the available data on human neurotoxic, immunotoxic, and reproductive effects. [62] Tests done on the Rhesus monkey (the most sensitive species found for reproductive toxicity) yield a no observed adverse effects level

(NOAEL) of 127 pg/kg/day. The application of a safety factor of 100 yields a safe human exposure level of 1.3 pg/kg/day.

The use of FDA's CPF in the calculation of a 1.2 ppq water quality standard for dioxin correlates to a human exposure level of 0.6 pg/kg/day. [63] The foregoing is obviously very conservative when compared to the safe level for possible reproductive toxicity.

vii. The best available current science supports a safe dose in the range of 1-10 pg/kg/day.

In summary, the PWG reevaluation of the Kociba data requires a substantial lowering of tumor counts utilized to predict a human carcinogenic response to dioxin. More significantly, the occurrence of a tumorigenic response only with overt liver toxicity lead the PWG to characterize dioxin as a weak carcinogen and refutes the linearity of dose-response at low doses assumed by EPA's LMA model.

The convergence of safe exposure level for the reproductive toxicity endpoint; safe exposure level based upon a classical safety factor approach for carcinogenicity; and the RsD derived using EPA's LMS model allows confidence in the safety of exposure in the range of 1-10 pg/kg/day. Comparison of this exposure range with the 0.6 pg/kg/day exposure level reflected in FDA's CPF at a 1×10^{-5} risk level demonstrates the extreme conservatism built into the RsD in the calculation of a 1.2 ppq dioxin standard.

b. Risk Level

The 1.2 ppq standard is based upon a risk level of 1×10^{-5} , which is within the range of risk levels that EPA has stated it will approve, and has approved for Maryland and Virginia, [64] when reviewing state dioxin standards. [65] It is also consistent with risk levels used by other states such as Michigan, Ohio, Alabama, South Carolina, Indiana and Georgia in development of their water quality standards for dioxin. In so doing, they have, in essence, recognized that a risk level of 1×10^{-5} (1 in 100,000) is negligible when large population exposures are involved. [66]

Leading experts in toxicology and risk assessment point out that it is common for the public to erroneously assume that all occupational and environmental regulations are based on a maximum cancer risk of 1×10^{-6} (1 in 1,000,000) and that any risk above this level poses a serious threat to public health. [67] In fact, risks at and below 1×10^{-6} are so small as to be negligible, and environmental regulatory decisions are frequently based on risks of 1×10^{-5} and higher. [68]

A 1×10^{-6} risk level is considered negligible because it represents only an insignificant incremental increase in the level of risk above background. The current background for cancer in the United States is one in four to one in three, meaning that in a population of one million people, 250,000 to 333,000 will develop some form of cancer in their lifetimes. The difference between 1×10^{-6} and 1×10^{-5} means that no more than one additional cancer case will be above the background risk of developing cancer over a

70 year lifetime. Using one in four as the background, this translates to a statistical calculation of each person's risk of contracting cancer as follows:

Background	0.250000	(25 percent risk for each person)
1×10^{-6}	0.250001	(25.0001 percent risk)
1×10^{-5}	0.250010	(25.0010 percent risk)

In the final analysis, selection of a risk level is a policy decision, and the Board would be clearly justified in using 1×10^{-5} in setting its dioxin standard, particularly in view of the extremely conservative assumptions built into the potency factor. [69]

c. Bioaccumulation/Bioconcentration Factors

Although bioaccumulation factor ("BAF") and bioconcentration factor ("BCF") are different concepts, they are both used to predict concentrations of environmental contaminants in fish tissues and in the same way to calculate a water quality standard for dioxin. [70] Briefly, BAF is a value representing the degree at which an organism accumulates a substance through ingestion of food and water, while BCF is a value representing the degree at which an organism concentrates in its tissue a substance dissolved in water directly through contact with the water. [71]

Many site-specific factors affect the potential of chemical compounds such as dioxin to bioaccumulate in fish tissue, and scientists generally agree that where sufficient data is available, water quality standards should be developed using site-specific BAFs. In the absence of such data, however, regulators

have used BCFs derived from laboratory tests to develop water quality standards. [72]

In establishing Ambient Water Quality Criteria for dioxin, the EPA recommended a BCF of 5,000 for dioxin citing several studies which indicated BCFs ranging from 2,000 for channel catfish to 9,270 for rainbow trout. In the past few years, laboratory-derived BCFs greater than 5,000 have been published in the literature or reported in conference proceedings. There has been some suggestion that the BAF be increased to 50,000 or more based on these laboratory data. However, proponents of BCFs higher than 5,000 fail to take into account critical adjustments that must be made to laboratory-derived BCFs before they can be used to calculate a water quality standard for dioxin. These adjustments relate to the fact that bioconcentration is related to the fat or lipid content of fish. Accordingly, adjustments must be made to account for (1) differences in lipid content of laboratory-raised and naturally occurring fish, even fish of the same species; (2) differences in lipid content between different species, and (3) the effects of cleaning fish since people generally eat only the muscle meat of the fish which have a lower fat content than whole fish. [73]

In addressing the choice of a bioconcentration factor of 50,000, Dr. Philip Cook, the EPA Environmental Research Laboratory - Duluth scientist who performed those studies, said that based on the available BCF measurements 50,000 would provide a mid-range prediction of bioaccumulation potential for TCDD in a variety of

freshwater exposure situations for fish tissues averaging 7% lipid. [74] Dr. Cook confirmed there is an association of BCF with percent lipid content in the test species. However, the species used in Dr. Cook's studies, lake trout, have lipid contents in fillets which range from 1.9% to 19.4% and are generally in excess of 10%. [75]

In contrast, a Department of Water Resources survey of the lipid content of fillets of West Virginia fish revealed thirteen largemouth bass averaged .9 percent, fourteen smallmouth bass averaged .78 percent, three bluegill averaged .24 percent, and even thirty-eight channel catfish averaged only 3.8 percent.

Based on the foregoing, in the absence of site-specific BAFs, 5,000 is a reasonable and realistic BCF to use in the calculation of a water quality standard for dioxin. Since data are not available from which to calculate site-specific BAFs for West Virginia waters, there is a valid scientific basis for the Board's use of a BCF of 5000 in the calculation of its water quality standard for dioxin. [76]

d. Fish Consumption Rate

Another factor in establishing a safe water quality standard is the amount of dioxin in fish consumed by people. Many site-specific factors affect the potential of chemical compounds such as dioxin to bioaccumulate in fish tissue, and scientists generally agree that where sufficient data is available, water quality standards should be developed using those site-specific bioaccumulation factors. In the absence of such data, however, EPA

and state regulators have used bioconcentration factors derived from laboratory tests to estimate the level of dioxin in water that would be necessary for dioxin to bioaccumulate to a given level in fish. EPA, in developing its original water quality standard, utilized a bioconcentration factor of 5,000. Although some recent scientific reports have questioned this number, EPA as late as last month said it lacked sufficient scientific basis for increasing that number.

Dioxin tends to concentrate in lipids, a type of fat, and for setting the bioconcentration factor utilized by EPA, a study was done of whole fish tissue for fish averaging 7% lipid content. Significantly, that study utilized all of the edible and inedible portions of lake trout instead of just using fillets. The lipid content in the fillets of lake trout range from 1.9% to 19.4% (and generally in excess of 10%), whereas a government survey of the lipid content of fillets of several popular West Virginia fish, bluegill, smallmouth bass and largemouth bass, revealed fillet lipid contents below 1%. Further, while EPA's standard utilized a national average per capita consumption rate for all fish of 6.5 grams per day, a National Marine Fishery Service study done in 1973-74 indicates West Virginia and the other South Atlantic states have an adult freshwater fish consumption rate of less than 2 grams per day. Therefore, the real threat of exposure to dioxin in West Virginia through fish consumption is substantially lower than assumed by EPA in establishing its water quality standard.

There is general agreement in the scientific community that, where available, local or regional fish consumption rates should be used in calculating water quality standards instead of EPA's national average per capita consumption rate for all fish (freshwater, estuarine, and shellfish) of 6.5 grams per day. Therefore, the available data on local and regional fish consumption rates should be examined.

Turning first to regional fish consumption rates, a review of the available data indicates that the most useful and valid data were collected in 1973 and 1974 by the National Marine Fisheries Service ("NMFS"). [77] The results of the NMFS survey are based on responses from 24,562 individuals who over a one month period recorded the number of fish meals they ate and the size of the meal for each species of fish. The results of the survey can be broken down by region and species of fish. West Virginia is in the South Atlantic region, together with Delaware, Maryland, District of Columbia, Virginia, North Carolina, South Carolina, Georgia, and Florida. In the NMFS study the South Atlantic region had an adult freshwater fish consumption rate of 1.1 grams per day. Although the NMFS survey was conducted in the mid 1970s, even if one factors in an assumed increase of 33.3 percent in the rate of fish and frozen fish consumption in the United States since then, the per capita freshwater fish consumption rate for West Virginia is increased only from 1.1 to 1.5 grams per day.

e. Water Ingestion Rate

While Commentors have no substantial objection to the water ingestion rate used by EPA in the computation of its water quality criteria, [78] they do note the 2.0 L/day is based on the daily ration of water required by United States Army field personnel and therefore probably high for the average civilian population. This adds another element of conservatism.

f. Fish Cooking Losses

Few, if any, people eat fish raw. Since dioxin is fat-soluble, and since much of the oils and fat in fish are lost during cooking, it would be appropriate to utilize a factor in the calculation of a water quality standard for dioxin which takes into account cooking losses. [79]

3. Summary

The real issue, for dioxin or any other contaminant, is whether a given water quality standard will result in harmful levels of exposure. Even though it has not changed its own criteria, EPA has approved water quality standards for dioxin of 1.2 ppq for Maryland and Virginia which utilized a risk factor of 10^{-5} and the FDA cancer potency estimate; Georgia has adopted the same standard, effective March, 1991 (EPA has indicated it plans to approve Georgia's standard but has not yet done so); and, the same standards have been officially proposed in South Carolina and Alabama. In approving Virginia's standard for dioxin, the EPA noted in its technical support document that "the basis for EPA approval of a State water quality criterion is not whether the

State used EPA's recommended criterion, but whether the State's decision is scientifically defensible and protective of designated [water] uses."

Based on these comments and the appendix, it is appropriate for the Board to depart from EPA's water quality criteria for dioxin and adopt a water quality standard for dioxin of 1.2 ppq. There is ample scientific evidence in support of a 1.2 ppq standard, and its adoption would be consistent with the Board's proper role in the water quality standard setting process under the Clean Water Act. The Commentors urge the Board to fully consider these comments and recommendations.

III. CONCLUSION

The Commentors respectfully request that the Board give full consideration to these suggestions and comments regarding the West Virginia water quality standards. If needed, we are willing to provide further information or clarification on any of the matters discussed herein.

Respectfully submitted this 28th day of June, 1991.

WEST VIRGINIA CHAMBER OF COMMERCE

WEST VIRGINIA MANUFACTURERS ASSOCIATION

WEST VIRGINIA OIL AND NATURAL GAS ASSOCIATION

INDEPENDENT OIL AND GAS ASSOCIATION OF WEST VIRGINIA

WEST VIRGINIA FARM BUREAU

III. Endnotes

1. D'elia, C.F. et. al., 1989. Analytical Chemistry for Environmental Sciences. Env. Sci. and Tech. 23(7): 768.
2. Technical Support Document for Water-Quality Based Toxics Control, March, 1991, p. 111.
3. U.S EPA has defined PQLs for Drinking Water Supplies as the lowest level that can reliably be achieved within specified limits of precision and accuracy during routine laboratory operating conditions (U.S. EPA, July 8, 1987, 52 Fed. Reg. 25689). See also March 29, 1990, 55 Fed. Reg. 11798. Hazardous Waste Management System, Identification and Listing of Hazardous Waste; Toxicity Characteristics Revisions.
4. U.S. EPA, November 13, 1985, 50 Fed. Reg. 46906.
5. U.S. EPA, December 1983. Water Quality Standards Handbook. p. 1-6.
6. U.S. EPA, December, 1988. Proceedings of the First National Workshop on Biological Criteria, Lincolnwood, Ill. December 2-4, 1987. EPA-905/989/003. p.iv.
7. Ohio EPA, October 19, 1989. Proposed Revisions to Ohio Water Quality Standards 2745-1. Water Use Designations and Criteria 3745-1-07(A)(5).
8. U.S. EPA, 1990. Biological Criteria-National Program Guidance for Surface Waters. EPA-440/5-90-004.
9. The Technical Support Document for Water Quality-Based Toxics Control, March, 1991, p. 70, provides that mixing zones should

be established to ensure that there is "no lethality to organisms passing through the mixing zone."

10. Idaho Department of Health and Welfare Rules, Section 01.2400,03(h).
11. Electric Power Research Institute. July 1985. Trace Element Removal by Coprecipitation with Amorphous Iron Oxyhydroxide: Engineering Evaluations. EPRI CS-4087, Project 910-2.
12. CHWA 1987.
13. TRAVIS et al 1988.
14. Environ. Corp. March 12, 1990. Comments on the Water Quality Standard for Dioxin Proposed by the Commonwealth of Virginia.
15. Assessment and Control of Bioconcentratable Contaminants in Surface Waters (Draft), EPA Office of Water, March 1991, Page X.
16. Assessment and Control of Bioconcentratable Contaminants in Surface Waters (Draft), EPA Office of Water, March 1991, pages V-8, V-9, and V-10.
17. Technical Support Document For Water Quality-Based Toxics Control, EPA Office of Water, March, 1991, Page 88.
18. Technical Support Document for EPA's September 12, 1990 Approval/Disapproval of Maryland's Water Quality Standards Revisions, Page 25.
19. Technical Support Document for EPA's February 25, 1991 Approval of Virginia's Water Quality Standards Revisions, Pages 20 - 24. Technical Support Document for EPA's September

- 12, 1990 Approval/Disapproval of Maryland's Water Quality Standards Revisions, Pages 25 - 27.
20. Kentucky WQS (401 KAR 5:031) (Surface water standards) Section 3.
21. U.S. EPA, 1987. Ambient Water Quality Criteria for Selenium. EPA-440/5-87-006. p. 29.
22. U.S. EPA, 1987. Ambient Water Quality Criteria for Selenium. EPA-440/5-87-006. pp. 26, 27.
23. Finley, K.A. 1985. Observations of Bluegills Fed Selenium-Contaminated *Hexagenia* nymphs collected from Belews Lake, NC. Bull. Environ. Contam. Toxicol. 35:816-825.
24. See Reference 21.
25. U.S. EPA, 1987. Ambient Water Quality Criteria for Selenium. EPA-440/5-87-006. p. 28.
26. Specht, W.L., D.S. Cherry, R.A. Lechleitner and J. Cairns, Jr., 1984. Structural, Functional and Recovery Responses of Stream Invertebrates to Fly Ash Effluent. Can. J. Fish. Aquatic Sci. 41:8484-896.
27. Nicholson, R.B., 1982. An Assessment of the Environmental Effects of Coal Ash Effluents Using Structural and Functional Parameters of Aufwuchs Communities. M.S. Thesis. Biology Dept. VPI&SU, Blacksburg, VA, Cited in: Cherry et al. 1986.
28. Whitaker, J.B., 1982. Field and Laboratory Determined Behavioral Avoidance and Alternations of Fish Gill Histological Responses to Acidic and Alkaline pH Conditions.

- M.S. Thesis. Biology Dept. VPI&SU, Blacksburg, VA Cited in:
Cherry et al. 1986.
29. Monogahela Power Company, 1991. Effects of Selenium in Crooked Run, Fort Martin, West Virginia. 5 pp.
 30. Appalachian Power Company and AEP Service Corporation. March 28, 1990. Rejustification of the Selenium and Copper Water Quality Criteria for Little Scary Creek and Application for Amendment to the Site-Specific Criteria. 70 pp. and 16 Figures, 20 Table, 4 Appendices.
 31. U.S. EPA, 1987. Ambient Water Quality Criteria for Selenium. EPA-440/5-87-006. p. 60.
 32. Cherry, D.S., S.E. Belanger and J.L. Farris, July 22, 1986. Critique of U.S. EPA's Draft Ambient Aquatic Life Water Quality Criteria for Selenium(IV). p. 23.
 33. Upon request, the Company will provide a recalculation description from Cherry et al. July 22, 1986.
 34. U.S. EPA, 1988. Ambient Water Quality Criteria for Aluminum.
 35. Phillips, D.J.H. 1990. Arsenic in aquatic organisms: a review, emphasizing chemical speciation. Aquatic Toxicol. 16:151-186.
 36. Tseng, et al. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453.
 37. Brown, C.C. and K. C. Chro. 1983. A new method for the analysis of chart studies: implications of the multistage

- theory of carcinogenesis applied to occupational arsenic exposure. *Environ Health Perspect* 50:253-308.
38. Chen, C.J., T.L. Kuo and M.M. Wu. 1988. Arsenic and cancer. *Lancet* 1 (8582) 414-415.
 39. Brown, K.G., K.E. Boyle, C.W. Chen and H.J. Gibb. 1989. A dose-response analysis of skin cancer from inorganic arsenic in drinking water. *Risk Anal.* 9:519-528.
 40. 50 Fed. Reg. 4690 (Nov. 13, 1985).
 41. MAFF. 1982. Survey of arsenic in food. The eighth report of the steering group on food surveillance. The working party on the monitoring of foodstuffs for heavy metals. Her Majesty's Stationary Office, London.
 42. Uthus, E.O., W.E. Cornatzer and F.H. Nielsen. 1983. Consequences of arsenic deprivation in laboratory animals. In: Arsenic: industrial, biomedical, environmental perspectives, edited by W.H. Lederer and R.J. Fensterheim, Van Nostrand Reinhold, N.Y. pp 173-189.
 43. VERSAR Inc. February 1990. A critical examination of the evidence for a threshold for cancer risk in humans from trivalent inorganic arsenic. Prepared for Safe Drinking Water Task Force, Edison Electric Institute, Washington, DC.
 44. Agency For Toxic Substances and Disease Registry (ASTDR), Public Health Service. November 1987. Draft Toxicological Profile For Arsenicity. 120 pp. and Appendixes.
 45. Comments on the Georgia Department of Natural Resources' Proposed Water Quality Standard for TCDD (T. Farber, 1989).

46. Technical Support Document for a Proposed Alabama Water Quality Standard for Dioxin (ENSR Consulting and Engineering, 1989).
47. Comments on the Georgia Department of Natural Resources' Proposed Water Quality Standard for TCDD (T. Farber, 1989), at 15.
48. Technical Support Document for a Proposed Alabama Water Quality Standard for Dioxin (ENSR Consulting and Engineering, 1989), at 3-9.
49. Dioxin Development Document; White Paper Report on Dioxin, Northwest Pulp and Paper Association (ChemRisk, 1989), at 18.
50. Technical Support Document for a Proposed Alabama Water Quality Standard for Dioxin (ENSR Consulting and Engineering, 1989).
51. Pathology Working Group, 2,3,7,8-Tetrachlorodibenzo-p-Dioxin in Sprague-Dawley Rats (PATHCO, Inc., Mar. 13, 1990).
52. Letter to LaJuana S. Wilcher, Assistant Administrator, U.S. EPA from Robert A. Squire, D.V.M., Ph.D. (May 24, 1990).
53. Pathology Working Group, 2,3,7,8-Tetrachlorodibenzo-p-Dioxin in Sprague-Dawley Rats (PATHCO, Inc., Mar. 13, 1990).
54. Technical Support Document for EPA's February 25, 1991 Approval of Virginia's Water Quality Standards Revisions, at 10.
55. Critical Factors in Establishing a Health-Based Water Quality Standard for Dioxin (ChemRisk, Sept. 4, 1990), at 7.

56. Hepatotoxicity in Female Sprague-Dawley Rats Treated with 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) (PATHCO, Apr. 27, 1990); Review of the Liver Lesions Reported in the Carcinogenesis Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) (PATHCO, Apr. 27, 1990).
57. Id.
58. Statement of Russell E. Keenan, Ph.D. before the Arkansas Commission of Pollution Control and Ecology (ChemRisk, Jan. 3, 1990).
59. Memorandum to Erich Brettheuer, Assistant Administrator, ORD from Linda S. Birnbaum, Ph.D., D.A.B.T., Director, Environmental Toxicology Division, HERL, OHR (EPA, Oct. 31, 1990).
60. Statement of Russell E. Keenan, Ph.D. before the Arkansas Commission of Pollution Control and Ecology (ChemRisk, Jan. 3, 1990).
61. Effects of Dioxin on Humans and Regulatory Policies About Dioxin (M. Gough, 1989) at 33; Comments Related to the Development of a Water Quality Standard for Dioxin (R. Hartung, 1989) at 5; Comments on the Proposed Georgia Water Quality Standard for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (A. Robertson, et al- 1989) at 8; Comments on the Georgia Department of Natural Resources' Proposed Water Quality Standard for TCDD (T. Farber, 1989) at 4; Biological Risk Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin at 7-8 (Cantox, Apr. 1989); A Review of Quantitative Estimates of the

- Carcinogenicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin at 2 (Envirologic Data, 1989).
62. Critical Factors in Establishing a Health-Based Water Quality Standard for Dioxin (ChemRisk, Sept. 4, 1990).
 63. Technical Support Document for a Proposed Alabama Water Quality Standard for Dioxin (ENSR Consulting and Engineering, 1989), at 3-17.
 64. Letter to Richard N. Burton, Executive Director, Virginia State Water Control Board, from Edwin B. Erickson, EPA Regional Administrator, Region III, (February 25, 1991), and Technical Support Document for EPA's February 25, 1991 Approval of Virginia's Water Quality Standards Revisions.
 65. Memorandum to Water Management Division Directors from LaJuana S. Wilcher, EPA Assistant Administrator (EPA, Jan. 5, 1990).
 66. Critical Factors in Establishing a Health-Based Water Quality Standard for Dioxin (ChemRisk, Sept. 4, 1990).
 67. Id.
 68. Cancer Risk Management, in Environmental Science and Technology 21(5):415-420 (Travis C.C., et al., 1987); Human Exposure to 2,3,7,8-TCDD, in Chemosphere 16 (10-12): 2331-2342 (Travis, C.C. & Hattemer-Fray, H.A., 1987).
 69. For an extensive discussion supporting the use of a 1×10^{-5} risk level, see Comments in Support of a Dioxin Water Quality Standard for South Carolina. (Anderson, ENSR, Aug. 7, 1990).
 70. Critical Factors in Establishing a Health-Based Water Quality Standard for Dioxin (ChemRisk, Sept. 4, 1990).

71. For a detailed discussion of the distinction between BAFs and BCFs, see *Id.* at pp. 11-13.
72. *Id.* at 12.
73. *Id.* at 13.
74. Memorandum to Alison Greene, EPA Water Quality Analysis Branch, from Philip Cook, Senior Research Chemist, EPA-Duluth (Aug. 1990).
75. R. J. Henderson and D. R. Tocher, The Lipid Composition and Biochemistry of Freshwater Fish. Prog. Lipid Res. 26:281-347 (1987).
76. Technical Support Document for EPA's February 25, 1991 Approval of Virginia's Water Quality Standards Revisions, at 13.
77. Some Results of Recent Surveys of Fish and Shellfish Consumption by Age and Region of U.S. Residents, in *Health Physics* 39:165-75 (Rupp, E.M., et al., 1980).
78. Critical Factors in Establishing a Health-Based Water Quality Standard for Dioxin (ChemRisk, Sept. 4, 1990).
79. The EPA criteria do not take into account the loss of dioxin during cooking of fish. However, studies establish that dioxin is, in fact, lost during cooking. N. C. Stachiw, et al., Tetrachlorodibenzo-p-dioxin Residue Reduction Through Cooking/Processing of Restructured Carp Fillets (*J. Agri. Food Chem.* 36:848-852, 1988); see also H. W. Puffer & R.W. Gossett, PCB, DDT, and Benzo(a)pyrene in Raw and Pan-Fried

White Croaker (Bull. Environ. Contam. Toxicology 30:65-73,
1983).

APPENDIX A

Pages 35-38 of the Environmental Protection Agency's
Ambient Water Quality Criteria for Selenium - 1987.
EPA-440/5-87-006

the one-hour average concentration does not exceed 300 $\mu\text{g/L}$ more than once every three years on the average. If selenium is as toxic to saltwater fishes in the field as it is to freshwater fishes in the field, the status of the fish community should be monitored whenever the concentration of selenium exceeds 5.0 $\mu\text{g/L}$ in salt water.

Implementation

Because of the variety of forms of selenium in ambient water and the lack of definitive information about their relative toxicities to aquatic species, no available analytical measurement is known to be ideal for expressing aquatic life criteria for selenium. Previous aquatic life criteria for metals and metalloids (U.S. EPA 1980b) were expressed in terms of the total recoverable measurement (U.S. EPA 1983a), but newer criteria for metals and metalloids have been expressed in terms of the acid-soluble measurement (1985b). Acid-soluble selenium (operationally defined as the selenium that passes through a 0.45 μm membrane filter after the sample has been acidified to a pH between 1.5 and 2.0 with nitric acid) is probably the best measurement at the present for the following reasons:

1. This measurement is compatible with nearly all available data concerning toxicity of selenium to, and bioaccumulation of selenium by, aquatic organisms. It is expected that the results of tests used in the derivation of the criteria would not have changed substantially if they had been reported in terms of acid-soluble selenium.
2. On samples of ambient water, measurement of acid-soluble selenium will probably measure all forms of selenium that are toxic to aquatic life or can be readily converted to toxic forms under natural conditions. In addition, this measurement probably will not measure several forms, such

- as selenium that is occluded in minerals, clays, and sand or is strongly sorbed to particulate matter, that are not toxic and are not likely to become toxic under natural conditions.
3. Although water quality criteria apply to ambient water, the measurement used to express criteria is likely to be used to measure selenium in aqueous effluents. Measurement of acid-soluble selenium is expected to be applicable to effluents. If desired, dilution of effluent with receiving water before measurement of acid-soluble selenium might be used to determine whether the receiving water can decrease the concentration of acid-soluble selenium because of sorption.
 4. The acid-soluble measurement is expected to be useful for most metals and metalloids, thus minimizing the number of samples and procedures that are necessary.
 5. The acid-soluble measurement does not require filtration of the sample at the time of collection, as does the dissolved measurement.
 6. For the measurement of total acid-soluble selenium the only treatment required at the time of collection is preservation by acidification to a pH between 1.5 and 2.0, similar to that required for the total recoverable measurement.
 7. Durations of 10 minutes to 24 hours between acidification and filtration of most samples of ambient water probably will not substantially affect the result of the measurement of total acid-soluble selenium. However, acidification might not prevent oxidation or reduction of selenium(-II), selenium(VI), or selenium(IV) (May and Kane 1984). Therefore, measurement of acid-soluble selenium(IV) and/or acid-soluble selenium(VI) might require separation or measurement at the time of collection of the sample or special preservation to prevent conversion of one oxidation state of selenium to the other.

8. Ambient waters have much higher buffer intensities at a pH between 1.5 and 2.0 than they do at a pH between 4 and 9 (Stumm and Morgan 1981)
9. Differences in pH within the range of 1.5 to 2.0 probably will not affect the result substantially.
10. The acid-soluble measurement does not require a digestion step, as does the total recoverable measurement.
11. After acidification and filtration of the sample to isolate the acid-soluble selenium, the analysis for total acid-soluble selenium can be performed using either furnace or hydride atomic absorption spectrophotometric or ICP-atomic emission spectrometric analysis (U.S. EPA 1983a), as with the total recoverable measurement. It might be possible to separately measure acid-soluble selenium(IV) and acid-soluble selenium(VI) using the methods described by Oppenheimer et al. (1984), Rotberecht and Van Grieken (1982), and Uchida et al. (1980).

Thus, expressing aquatic life criteria for selenium in terms of the acid-soluble measurement has both toxicological and practical advantages. The U.S. EPA is considering development and approval of a method for a measurement such as acid-soluble.

Metals and metalloids might be measured using the total recoverable method (U.S. EPA 1983a). This would have two major impacts because this method includes a digestion procedure. First, certain species of some metals and metalloids cannot be measured because the total recoverable method cannot distinguish between individual oxidation states. Second, in some cases these criteria would be overly protective when based on the total recoverable method because the digestion procedure will dissolve selenium that is not toxic and cannot be converted to a toxic form under natural conditions. Because no measurement is known to be ideal for expressing aquatic life criteria for

selenium or for measuring selenium in ambient water or aqueous effluents measurement of both acid-soluble selenium and total recoverable selenium in ambient water or effluent or both might be useful. For example, there might be cause for concern when total recoverable selenium is much above an applicable limit, even though acid-soluble selenium is below the limit.

In addition, metals and metalloids might be measured using the dissolved method, but this would also have several impacts. First, whatever analytical method is specified for measuring selenium in ambient surface water will probably also be used to monitor effluents. If effluents are monitored by measuring only the dissolved metals and metalloids, the effluents might contain some selenium that would not be measured but might dissolve, due to dilution or change in pH or both, when the effluent is mixed with receiving water. Second, measurement of dissolved selenium requires filtration of the sample at the time of collection. Third, the dissolved measurement is especially inappropriate for use with such metals as aluminum that can exist as hydroxide and carbonate precipitates in toxicity tests and in effluents. Use of different methods for different metals and metalloids would be unnecessarily complicated. For these reasons, it is recommended that aquatic life criteria for selenium not be expressed as dissolved selenium.

As discussed in the Water Quality Standards Regulation (U.S. EPA 1983b) and the Foreword to this document, a water quality criterion for aquatic life has regulatory impact only after it has been adopted in a State water quality standard. Such a standard specifies a criterion for a pollutant that is consistent with a particular designated use. With the concurrence of the U.S. EPA, States designate one or more uses for each body of water or segment thereof and adopt criteria that are consistent with the use(s) (U.S. EPA

APPENDIX B

Relating Metals Measurements to Water Quality
Criteria for Protection of Aquatic Life

RELATING METALS MEASUREMENTS
TO WATER QUALITY CRITERIA
FOR PROTECTION OF AQUATIC LIFE

DEC 26 1983

Background

EPA recognizes four methods of sample preparation for metals analysis. These lead to measurement of: (a) total metals, (b) total recoverable metals, (c) acid soluble metals, and (d) dissolved metals. Ordinarily, the four methods should measure all of the dissolved metal present at the time of sampling. They differ in the amount of particulate metal that they measure.

The total metals procedure and the total recoverable metals procedure (EPA 1983, sections 4.1.3 and 4.1.4) allow measurement of all metal that can be dissolved in hot acid (pH near zero for total recoverable). The total procedure involves a longer, hotter, and more acidic digestion period than the total recoverable procedure. The results from both procedures are reported as "total", although not all solids can be digested by these procedures. The 1980 water quality criteria documents expressed metals criteria in terms of total recoverable metals.

The acid soluble procedure (Martin et al. 1986) allows measurement of metal that can be dissolved at pH 1.5-2.0 at room temperature. The milder extraction conditions are intended to result in measuring less particulate metal than the total or total recoverable methods. Recent criteria documents have expressed metals criteria in terms of the acid soluble procedure, despite it not being a formally approved EPA method.

The dissolved procedure (EPA 1983, section 4.1.1) allows measurement of metal that can pass through a 0.45 μm filter at the time of sample collection and can be dissolved at pH < 2 (the sample preservation pH). The results from this procedure are reported as "dissolved", although it may include metal that was bound to micro-particles (<0.45 μm) at the time of sample collection.

All the above methods simply prepare the sample for analysis. Actual analysis is generally done by direct aspiration or graphite furnace atomic absorption or by inductively coupled plasma spectroscopy. For some important metals, such as copper, lead, and cadmium, only the graphite furnace instruments usually provide sufficient sensitivity to reliably measure typical ambient levels.

Metals criteria documents issued in 1980 expressed the criteria in terms of total recoverable metal. Beginning in 1984 the metals documents have expressed the criteria in terms of acid-soluble metal. While a draft method for acid-soluble metal has existed for several years, the method has not been made final

and may continue to exist only in draft form, due to unresolved questions about its appropriateness. In the absence of an approved acid-soluble method, the criteria documents have been recommending the total recoverable method, which they acknowledge may be overly protective.

The Nature of the Problem

To exert an effect within the tissues of the target organism, a metallic toxicant must be in a dissolved form to pass through biological membranes and react chemically at the site of action. In some cases, the particular form of dissolved metal (e.g., divalent cation) may be an important determinant of toxicity. Metal that is already in the dissolved state within the aquatic environment has a much greater potential for contributing to a toxic effect than does metal in the particulate phase. Nevertheless, the toxic potential for ambient particulate metal is not zero. For example, some metal that is in the particulate phase in the surface water environment may become dissolved at the slightly reduced pH within the gill environment.

In most (but not all) of the toxicity tests forming the basis for water quality criteria, the fraction of particulate metal is fairly low. For example, typically 10-15 percent of the copper is in a particulate phase during flow-through tests with rather clean freshwater.

In ambient waters, on the other hand, the fraction particulate is often higher. STORET stream data suggest that roughly 50 percent of the arsenic, and 60-90 percent of the copper, lead, nickel, and zinc, and 90-95 percent of the aluminum is typically in a particulate phase measured by the total recoverable method but not by the dissolved method.

The fraction of metal that is biologically available is thus often expected to be lower under ambient field conditions than under laboratory conditions. Although the relationship between water quality criteria and measurements of total, total recoverable, acid soluble, or dissolved metal can be discussed in general terms, it is not currently possible to quantify the errors involved in relating the results from each of these methods to the water quality criteria under different field situations. While we intend to work toward resolving the technical questions, it is necessary in the interim to have a policy on use of these methods for criteria purposes.

Possible Direction of EPA Policy

EPA is reassessing its position on metals analyses used for evaluating the attainment of ambient criteria and determining the need for water quality-based controls. The following policy has

been drafted and is currently under review in EPA Laboratories and Regional Offices:

For purposes of evaluating the attainment of ambient criteria and determining the need for water quality-based controls, the following techniques would be acceptable, in the absence of information to the contrary: total, total recoverable, acid soluble, or dissolved.

Nevertheless, as indicated by 40 CFR 122.45, only the total recoverable method would be used to determine compliance with NPDES permit limitations.

Before such an interim policy was adopted, it would be widely reviewed outside EPA.

Total and total recoverable metals:

The total or total recoverable metals measurements provide greatest degree of safety when comparing to water quality criteria. Nevertheless, the criteria documents acknowledge that use of these measurements "may be overly protective". Thus, their use may result in identifying problems that do not in reality exist.

Dissolved metals:

The dissolved metal measurement may often provide the most accurate and appropriate comparison with water quality criteria. However, there are three significant problems with this approach.

First, the water quality criteria are generally based on the reported dissolved plus particulate concentrations in the toxicity tests. If interpreted as dissolved concentrations, the criteria values should be downwardly adjusted to account for the fraction dissolved typically occurring during the tests. For example, assuming that 85-90 percent of the copper were typically dissolved during freshwater toxicity tests, the current freshwater copper criterion would be adjusted to 85-90 percent of its current value in order to serve as a dissolved criterion. For aluminum, the adjustment would be much larger, to account for the much lower percentage dissolved typically occurring during toxicity tests.

Second, by measuring comparatively little of the particulate fraction, the dissolved method may at times understate the toxicologically effective concentration that may occur. This is more likely to be a concern if the dissolved concentration is only a small percentage of the particulate concentration.

Third, the dissolved method usually cannot appropriately be applied to effluents to determine compliance with NPDES limitations. A common method of removing metals from wastewaters

is to chemically precipitate the metal and settle the resulting particles. Expressing a metals limitation in terms of dissolved metal would imply little concern about the effectiveness of the settling process. Furthermore, as the chemical conditions in ambient surface waters are likely to be different than those in the effluent, there is no assurance that the particulate metal would not rapidly dissolve after discharge. Consequently, NPDES regulations (40 CFR 122.45) require that permit limitations ordinarily be expressed as total recoverable metal.

Determining the total recoverable limitation corresponding to a dissolved criterion would involve an assessment of the fraction of discharged metal that would be soluble under ambient chemical conditions. Where such information cannot be obtained readily, it is fully acceptable to assume that 100 percent of the total recoverable metal may become dissolved.

Where greater accuracy is desired, the dissolved fraction of total recoverable metal could be determined by direct measurement of dissolved and total recoverable metal in the affected ambient waters, or possibly by geochemical model calculations or other estimation methods. Whenever possible, the discharger should be given the responsibility for obtaining and analyzing any needed ambient or effluent samples.

Acid soluble metals:

This approach is a compromise between total recoverable and dissolved. It was originally conceived with the intent to apply it to both effluent and ambient waters. Its advantages were intended to be that (a) it would exclude from measurement the strongly particulate-bound and therefore relatively unavailable metal that would be measured by the total recoverable method, (b) it would provide greater safety than the dissolved method, and (c) it would allow use of a single method for both effluent and ambient waters.

To be a good measure of metals that were available or nearly available biologically, an extraction pH of around 4 was originally thought desirable. The extraction pH was reduced to the current 1.5-2.0 to enhance reproducibility. It is not presently known how the method relates to biologically available metals. Nor is it known how the method compares with the dissolved and total recoverable methods when applied to ambient samples. The Agency has not yet done an adequate evaluation of the method.

Lacking sufficient information on the behavior of the method, it is not now considered suitable for expressing effluent limitations. For comparison to water quality criteria, the method is acceptable, as it is intermediate between the dissolved and total recoverable methods.

APPENDIX

DETERMINING THE RATIO BETWEEN DISSOLVED AND TOTAL METAL

Two methods are currently being considered or being used to relate ambient dissolved standards to effluent total recoverable limits: (a) national average ratios based on available ambient data (e.g., State of Texas procedure) and (b) site-specific measurements (e.g., proposed State of Virginia procedure). Both rely on simultaneous measurement of dissolved and total (recoverable) metal.

Using National Average Values

The STORET data analysis done by HydroQual (1982), presented in whole by Delos et al. (1984), and later summarized by Mills et al. (1985), could serve as a means of specifying the ambient percentage dissolved. Using data for samples where both dissolved and total metal were detected, and where suspended solids concentrations was simultaneously measured, HydroQual determined metals partition coefficients and related them, by regression, to the suspended solids concentration. HydroQual also examined the relationship of partitioning to measured pH and alkalinity but found little correlation with these parameters. Table 1 summarizes the results.

Using Site-Specific Measurements

The concept is to measure the dissolved-total metal ratio downstream of the discharger. Although it sounds simple, it may not be so easy to put into practice.

Because the chemical properties of an effluent (particularly an industrial effluent) may be much different than the chemical properties of the receiving water downstream of the discharger, it is much more appropriate to measure the ratio in the stream rather than in the effluent. To specify precisely how to measure dissolved and total metal in the downstream waters requires dealing with a number of issues.

- How near or far downstream should samples be taken?
- Can samples be taken at any flow? Should it be restricted to lower flows?

An alternative approach to downstream sampling is to sample the effluent and the upstream waters and mix samples at the desired dilution. In that case one must decide:

- What dilution should be used? If the metal is not detected, should the dilution be lowered?
- How long should the effluent and ambient samples be mixed before filtering for dissolved metal?

With either approach it must be decided:

- Should sampling be restricted to time periods when suspended solids concentrations are not too high?
- Should sampling be restricted to time periods when the pH is not too high? Should the sample pH be adjusted artificially before filtering the sample?

The above questions can be answered in a number of different ways. It is not known how much the different possible answers would affect the final result.

The most important constraint on the feasibility of carrying out site-specific measurements is the capability of analytical laboratories to detect and accurately quantify the metal. The ratio between dissolved and total metal of course cannot be determined unless both dissolved and total metal can be detected.

Direct aspiration (flame) atomic absorption (AA) methods will often not be sensitive enough. Graphite furnace (flameless) AA techniques will usually be needed. Even so, EPA criteria for a few metals are below the detection limits of any of the EPA approved methods. While the analytical state of the art is ahead of the approved methods in general use, the accuracy of results even for the approved methods may be governed as much by amount of meticulous drudgery put into cleaning sampling vessels and other glassware, as by the technology in the instrument or skill of the operator.

Finally it should be noted that the *actual* dissolved-total ratio can be expected to vary somewhat over time. Due to measurement imprecision, the *measured* ratio is likely to vary substantially more than the *actual* ratio. It must then be decided:

- Should the mean dissolved-total ratio be used? Or should a less frequently occurring (more protective) ratio be used?
- If something other than the mean or median ratio is used, how should the analytical variability be controlled or accounted for?

References

- Delos et al. 1984. Technical Guidance Manual for Performing Waste Load Allocations. Book II Streams and Rivers. Chapter 3 Toxic Substances. U.S. EPA. OWRS. Washington, DC. EPA-440/4-84-012.
- HydroQual, Inc. 1982. Water Sediment Partition Coefficients for Priority Metals. Report to EPA Grosse Ile Lab.
- Martin, T.D., J.W. O'Dell, E.R. Martin, and G.D. McKee. 1986. Evaluation of Method 200.1 Determination of Acid Soluble Metals. U.S. EPA, Environmental Monitoring and Support Laboratory, Cincinnati, OH.
- Mills et al. 1985. Water Quality Assessment: A Screening Procedure for Toxic and Conventional Pollutants in Surface and Ground Water - Part 1. U.S. EPA, Athens, GA. EPA/600/6-85/002a.
- U.S. Environmental Protection Agency. 1983. Methods for Chemical Analysis of Water and Wastes. Environmental Monitoring and Support Laboratory, Cincinnati, OH. EPA-600/4-79-020.

Table 1: Metals partitioning from STOFET data: mean partition coefficient and corresponding fraction dissolved.

The partition coefficient, K_p [L/kg], equals the pollutant concentration [$\mu\text{g/kg}$] attached to particles divided by the dissolved concentration [$\mu\text{g/L}$]. At a particular suspended solids concentration, SS [mg/L], K_p is given by the following regression formula:

$$K_p = K_{p0} \cdot SS^a$$

The corresponding fraction dissolved is given by:

$$\text{Frac diss} = 1/(1 + 10^{-K_p \cdot SS})$$

Metal	<u>a</u>	<u>K_{p0}</u>	Corresponding Fraction Dissolved	
			SS=10 mg/L	SS=100 mg/L
Arsenic	-0.7286	0.48×10^6	0.53	0.37
Cadmium	-1.1307	4.00×10^6	0.25	0.31
Chromium	-0.9304	3.36×10^6	0.20	0.17
Copper	-0.7436	1.04×10^6	0.35	0.23
Lead*	-0.8	2.8×10^6	0.18	0.12
Mercury	-1.1356	2.91×10^6	0.32	0.39
Nickel	-0.5719	0.49×10^6	0.43	0.22
Zinc	-0.7038	1.25×10^6	0.29	0.17

- * For lead, due to error in the original document, the regression slope, a , and intercept, K_{p0} , were graphically re-estimated to apply at SS less than or equal to 100 mg/L.

Note: for $a=-1.0$, increasing the suspended solids concentration decreases the partition coefficient at the same rate at which it increases the mass of sorbing solids. The net result is that for $a=-1.0$ the fraction dissolved does not change with suspended solids concentration.

APPENDIX C

**Environ. Corp. Aug. 6, 1990. Comments on the
Water Quality Standards for Arsenic
Proposed by the Commonwealth of
Virginia. Prepared for Westvaco. 19pp.**

COMMENTS ON THE
WATER QUALITY STANDARD
FOR ARSENIC PROPOSED BY THE
COMMONWEALTH OF VIRGINIA

Prepared for

Westvaco
Covington, VA

Prepared by

ENVIRON Corporation
Washington, DC

August 6, 1990

CONTENTS

	<u>Page</u>
I. INTRODUCTION	1
II. DERIVATION OF PROPOSED STANDARD	3
III. COMMENTS ON ASSUMPTIONS	5
IV. CONCLUSIONS AND RECOMMENDATIONS	14
V. REFERENCES	17

I. INTRODUCTION

To meet requirements under the 1987 amendments to the federal Clean Water Act administered by the U.S. Environmental Protection Agency (EPA), the Commonwealth of Virginia has proposed a slate of ambient water quality standards for surface waters of the Commonwealth. These standards will be used as the basis for limiting discharges to surface water by point sources under the National Pollutant Discharge Elimination System (NPDES), taking into account any site-specific conditions that affect the relationship between discharges and ambient water quality.

Among the substances for which Virginia has issued proposed standards is arsenic. Virginia's principal concern is with ingested arsenic and its potential to cause cancer. Several epidemiological studies conducted outside the United States (Geyer 1898; Arguello et al. 1939, Borgono et al. 1977, Zaldivar 1974, Tseng 1977 and Tseng et al. 1968) reveal an association between high levels of arsenic in drinking water and certain unusual forms of readily treatable skin cancers. These observations have not been confirmed in animal studies or in epidemiologic studies conducted in the United States, but the International Agency for Research on Cancer (IARC) and the EPA have listed ingested arsenic as a human carcinogen.

Humans can be exposed to arsenic entering surface waters by using those waters as a source of drinking water or by consuming fish caught in those waters. It appears, however, that the Commonwealth of Virginia has proposed regulations based on water from public water supplies, fish and water consumption, and fish consumption alone. Some fish concentrate arsenic in their tissues to concentrations somewhat higher than those in the water in which they live. By making assumptions about the average quantities of water and fish consumed by a person over a lifetime and the extent of bioconcentration in fish, Virginia can estimate the average daily dose that a person would receive for any concentration of arsenic in the surface water body in question. If the estimated dose for any concentration of arsenic in surface water is combined with the estimate of the cancer dose-response relationship for inorganic arsenic, the risk of human cancer can be estimated. Finally, Virginia can choose a level of protection from risk that it considers adequate, given the uncertainty whether arsenic can cause any human cancer at very low doses.

The Commonwealth of Virginia has adopted a public water supply standard for arsenic of 50 $\mu\text{g/l}$ [50 parts per billion (ppb) by weight]. The Commonwealth has

proposed a standard for water and fish consumption of $0.0022\mu\text{g/l}$ (2.2 ppt), and for fish consumption only of $0.0175\mu\text{g/l}$ (17.5 ppt).

Westvaco Corporation, which operates a paper mill in western Virginia, has requested ENVIRON Corporation review and offer comments on the contemplated standards.

II. DERIVATION OF PROPOSED STANDARD

The Commonwealth of Virginia appears to have made the following assumptions in the derivation of the proposed arsenic standards:

1. Arsenic can cause cancer in humans at any level of exposure. The risks of cancer do not depend on the details of arsenic exposure over time, but instead on the average level of arsenic exposure over a lifetime.
2. The relationship between risk and dose at low levels of exposure is approximately linear; that is, the number of cancers expected in a given exposed population increases in proportion to the level of dose.
3. Cancer risks from ingestion of arsenic are based upon a "potency factor" (also called a "slope factor"). The proposed CPF (cancer potency factor) of $15 \text{ (mg/kg/day)}^{-1}$ appears to be based on an out-of-date EPA document. The CPF is based on a linear extrapolation model with the added assumption that there is no threshold in the dose-response relationship.
4. Water consumption is based on an EPA estimate of 2.0 l/day.
5. The consumption of fish containing arsenic averages 6.5 g/day over a lifetime.
6. The concentration of arsenic in the fish averages 44 times the concentration of arsenic in the surface water in which the fish lives; that is, the bioconcentration factor is 44.
7. The body weight of the exposed person at risk averages 70 kg.
8. All forms of cancer are equally lethal.
9. All forms of arsenic are equally carcinogenic.

10. Industrial effluent should contain less arsenic than the surface water source into which it is discharged.
11. Exposures to arsenic should be limited so that the computed lifetime risk of cancer does not exceed one in one million (10^{-6}).

With these assumptions, the ambient water quality standard (concentration) for consumption of drinking water and fish, WQS, in mg/l, is given by the equation:

$$WQS = BW \times RL / CPF \times [WC + (FC \times BCF)] ,$$

where:

BW is body weight (70 kg)
 RL is the risk limit (10^{-6})
 CPF is the cancer potency factor [$15 \text{ (mg/kg bw/day)}^{-1}$]
 WC is water consumption (2 l/day)
 FC is fish consumption (6.5 g/day = 0.0065 kg/day)
 BCF is the bioconcentration factor (44)

Substituting these values into the equation, the standard is computed to be:

$$\begin{aligned} WQS &= 70 \times 10^{-6} / 15 \times [2 + (0.0065 \times 44)] , \\ &= 2.2 \times 10^{-6} \text{ mg/l} \\ &= 0.0022 \text{ } \mu\text{g/l} \end{aligned}$$

If only fish are consumed, the factor WC does not appear in the equation and the standard is computed to be $0.0175 \text{ } \mu\text{g/l}$.

III. COMMENTS ON ASSUMPTIONS

The comments below are keyed to the eleven assumptions listed in the previous section.

1. **Arsenic can cause cancer in humans at any level of exposure. The risks of cancer do not depend on the details of arsenic exposure over time, but instead on the average level of arsenic exposure over a lifetime.**

Arsenic is ubiquitous in the human environment, and consequently all members of the United States population ingest arsenic daily, from both food and water sources. The wide-spread occurrence of arsenic may suggest its importance as an essential nutrient. According to EPA (1988), arsenic is a "possible but not proven nutritional requirement in animals. If arsenic is in fact an essential nutrient in animals, it is likely to be essential in humans." The FDA does not consider arsenic ingested through foods to represent a human cancer risk. Food exposure is estimated to average 60 $\mu\text{g}/\text{day}$, of which up to 20 $\mu\text{g}/\text{day}$ is probably inorganic (EPA 1984). Organically bound arsenic is a chemical form that is not as suspect, although there is no unequivocal proof that organically bound arsenic is not carcinogenic.

Exposure to some level of arsenic in drinking water is extensive. According to government reports, the average arsenic level in drinking water in the United States is 2.5 $\mu\text{g}/\text{l}$ (EPA 1981, ATSDR 1987). EPA (1984) reports that 0.8 percent of rural households exceed the federal 50 $\mu\text{g}/\text{l}$ arsenic maximum contaminant level (MCL) for drinking water, with the proportion exceeding the MCL in the West and North Central States being 2.1% and 1.8%, respectively. Significantly higher drinking water levels have been found in areas in the Western U.S. and Alaska that are associated with geochemical enrichment of arsenic. For example, arsenic levels in Lane County, Oregon have ranged up to 2.2 ppm (Morton et al 1976), while arsenic levels in Fairbanks, Alaska have been reported as high as 10 ppm (Harrington 1978).

3. Cancer risks from ingestion of arsenic are based upon a "potency factor" (also called a "slope factor"). The proposed CPF (cancer potency factor) of $15 \text{ (mg/kg/day)}^{-1}$ appears to be based on an out-of-date EPA document. The CPF is based on a linear extrapolation model with the added assumption that there is no threshold in the dose-response relationship.

The value for the cancer potency factor for ingested arsenic proposed by the state of Virginia is $15 \text{ (mg/kg/day)}^{-1}$ and would appear to be based on an obsolete EPA number (EPA 1980). The current EPA cancer potency factor for arsenic is $1.75 \text{ (mg/kg/day)}^{-1}$ and is based on a reanalysis of the Tseng et al. study conducted in Taiwan (EPA 1988).

There is historical evidence linking chronic exposure to high levels of arsenic with a variety of skin diseases including cancer. The studies that have been relied upon for cancer risk assessment are those by Tseng, who correlated a high prevalence of skin cancer and a circulatory disorder of the extremities (blackfoot disease) in Taiwan with the arsenic content of the drinking water. Almost half of the Taiwanese wells had arsenic levels in excess of 400 ppb. Unfortunately, Tseng did not include analysis of drinking water constituents other than arsenic. This omission is especially relevant because another Taiwanese study (Lu 1978) reported the presence of ergot alkaloids in Taiwanese well water associated with blackfoot disease. The possibility of other confounding chemicals being present in the Taiwanese wells has lowered EPA's confidence in the association of arsenic and skin cancer (EPA 1984).

In addition, three epidemiological studies conducted in the United States found no correlation between high levels of arsenic in drinking water and adverse health effects. Communities with significantly higher drinking water levels have been found in areas in the Western U.S. and Alaska that are associated with geochemical enrichment of arsenic. In Lane County, Oregon, Morton et al. (1976) found no increase in skin cancers among the residents exposed to elevated arsenic levels in water. Almost 5% of the population drank water with arsenic levels in excess of 100 ppb. Harrington et al. (1978) could find no increase in skin disorders among more than 200 residents of Fairbanks, Alaska who consumed drinking

water with 224 ppb of arsenic. Especially relevant is a study performed in Utah (Southwick et al. 1981) by EPA and the Utah Department of Health. In this study, residents from two towns, consuming water with average arsenic concentration of 0.18-0.21 mg/l, were compared to residents of a community where drinking water arsenic averaged 0.02 mg/l. Estimated mean and median total daily doses from drinking water for the exposed towns were approximately 0.42 and 0.33 mg/day, respectively, over a 10 year period. No symptoms of arsenic intoxication were noted among the inhabitants and the incidence of cancer and cancer death rates were comparable between the exposed and control inhabitants.

The U.S. epidemiological studies suggest that the current EPA cancer potency factor of $1.75 \text{ (mg/kg/day)}^{-1}$ is conservative. If the EPA's CPF is to be believed, the excess lifetime cancer risk associated with the average ($2.5 \text{ } \mu\text{g/l}$) U.S. exposure to arsenic through drinking water (EPA 1981, ATSDR 1987) is 1.25×10^{-4} (one in 8,000); the average excess risk associated with average food intake of inorganic arsenic ($20 \text{ } \mu\text{g/day}$ from EPA 1984) is up to 5.0×10^{-4} (one in 2000); and the excess lifetime cancer risk associated with the EPA's maximum contaminant level ($50 \text{ } \mu\text{g/l}$) is 2.50×10^{-3} (one in 400). Obviously, therefore, EPA's CPF is implicitly assumed to be excessively conservative.

4. Water consumption is based on an EPA estimate of 2.0 l/day.

In its Exposure Factors Handbook (EPA 1989), EPA uses a value of 2 l/day as a reasonable worst case. This value includes drinking water consumed in the form of coffee, juices and other beverages made with tap water. EPA states that this value probably overestimates the water intake for most individuals and is used to estimate long-term consumption; EPA suggests that 1.4 l/day is more representative of average consumption. The Commonwealth of Virginia is consistent with the EPA and other federal agencies in adopting the higher number as a conservative choice for daily water intake.

5. The consumption of fish containing arsenic averages 6.5 g/day over a lifetime.

Any assumption regarding fish consumption is very difficult to defend as a generality. Fish consumption can differ markedly from person to person, with many people eating essentially no fish and others being nearly "subsistence fishermen." Moreover, consumption habits can vary greatly over a person's lifetime. The statistic used (6.5 g/day) is the national average consumption of freshwater and estuarine fish and shellfish, as determined from a survey conducted by the National Marine Fisheries Service in the mid 1970s (SRI 1980), and excludes marine fish. Estimated average freshwater fish consumption (ENSR 1989) for the South Atlantic, including the Commonwealth of Virginia, is 1.05 g/day (Table 1). This suggests that the 6.5 g/day value is an extremely conservative choice.

6. The concentration of arsenic in the fish averages 44 times the concentration of arsenic in the surface water in which the fish lives; that is, the bioconcentration factor is 44.

Similarly, many arguments could be made on the propriety of various possible values for the bioconcentration factor relating concentrations of arsenic in fish to the concentrations in water. The bioconcentration factor used by EPA (1980) in its derivation of the water quality criteria for arsenic was 44, based on one measurement in bluegills ($BCF = 4$) and one in oysters ($BCF = 350$). Another six studies showed essentially no bioconcentration of arsenic in aquatic organisms. Therefore, the EPA value is likely conservative for the overall consumption of freshwater and estuarine fish and shellfish. Virginia's neighboring state of Maryland has used this information as a basis for declining to set a human health criterion for arsenic for consumption of fish alone (MDE 1990).

7. The body weight of the exposed person at risk averages 70 kg.

The International Commission on Radiological Protection (ICRP 1984) is the source of the 70-kg value for the body weight of "standard man," which is widely used in risk assessments. EPA (1989) has recently suggested that

adult U.S. males average closer to 78 kg, but that children and adult females weigh less. Fish and water consumption also differ by age and sex, of course. Although it could probably be demonstrated that the lifetime average weight for the whole U.S. population is slightly lower than 70 kg, the standard is not very sensitive to such small differences, and Virginia is justified in continuing with the traditional value.

8. **All forms of cancer are equally lethal.**

Because of their high degree of visibility and low rate of metastasis, skin cancers are one of the most treatable forms of cancer. Arsenic-induced skin lesions in the Taiwanese study were of various types (hyperpigmentation, hyperkeratosis, skin cancer). Hyperpigmentation is the most common, but only the skin cancers and a portion of the hyperkeratoses are malignant. These skin conditions were the type of lesion most frequently associated with the high levels of arsenic ingestion. Of the arsenic-induced cancers (squamous cell carcinoma, basal cell carcinoma, Bowen's disease) little is known of their metastatic potential. However, the EPA (1988) has noted that historical evidence for the potential for these types of cancers to metastasize is extremely low.

In its review of the Taiwanese study, EPA (1988) attempted to review case fatality rates for arsenic-induced skin cancer within the U. S. The level of health care in the U.S. versus Taiwan was believed to be significant in determining the outcome of skin cancer. Since no arsenic-induced skin cancer is verified in the U.S., EPA used data primarily from sunlight-induced carcinomas. These nonmelanoma skin cancers are the most common malignant neoplasms among caucasians and have a case-fatality rate of < 2%. EPA estimates that the case-fatality rate for arsenic-induced skin cancers in the U.S. is <1 %. On the basis of this low fatality rate associated with arsenic induced skin cancers, EPA has suggested that a 10-fold higher acceptable risk level may be appropriate for inorganic arsenic ingestion exposures.

9. All forms of arsenic are equally carcinogenic.

Arsenic is a chemical element that can combine with other elements to form a variety of chemical compounds or "species." Many of the toxic effects of arsenic are associated with species that come from industrial processes rather than those that occur in the environment. Toxicologists generally view the trivalent species of arsenic (arsenites) as more toxic than the pentavalent species (arsenates) (NAS 1977, IARC 1980). The trivalent forms are not as frequently found in the general environment, and surface waters in particular, as are the pentavalent forms (Boyle and Jonasson 1973). In those industries for which sources of arsenic in effluents are natural, the species may be largely in the more benign pentavalent state. EPA has acknowledged this distinction in its water quality standards for aquatic life (EPA 1980). The Commonwealth of Virginia should regulate only trivalent forms and require dischargers to assay for trivalent rather than total arsenic. The trivalent and pentavalent forms can be separated by chemical techniques and then the trivalent fraction analyzed for total arsenic (Mushak et al. 1977). The cost for such an analysis is perhaps triple that of total arsenic alone (e.g., \$150 vs \$50).

10. Industrial effluent should contain less arsenic than the surface water source into which it is discharged.

Arsenic is a natural constituent of surface and groundwater. The EPA estimates that the average drinking water source contains 2.5 $\mu\text{g/l}$ (EPA 1981, ATSDR 1987). In the state of Virginia, pristine surface water may contain 1 to 2 $\mu\text{g/l}$ (USGS 1984) of arsenic.

11. Exposure to arsenic should be limited so that the computed lifetime risk of cancer does not exceed one in one million (10^{-6}).

The choice of a risk limit is a policy decision, not a scientific issue. The risk level of one in a million (10^{-6}), used by the Commonwealth of Virginia in its proposed standard, was developed with a specific application in mind, namely the practical definition of "no risk" for food additives under the

Delaney clause of the Food, Drug, and Cosmetic Act (Rodricks et al. 1987). EPA (1989) suggests, but does not have the authority to mandate, that the range 10^{-7} to 10^{-5} is appropriate for setting water quality standards.

The State of California uses 10^{-5} as the risk criterion for limiting discharges of pollutants to surface and ground water under its Safe Drinking Water and Toxic Enforcement Act (Proposition 65)(CHWA 1987). Travis et al. (1987) have shown that many federal decisions on risk have allowed residual individual risks of 10^{-4} or even more.

These precedents are supported by a more fundamental argument. The current rate of cancer mortality in the United States approaches 20%. Even after removing the most obvious lifestyle contributions to the cancer rate (tobacco and alcohol), the rate would be in the vicinity of 10% (10^{-1}). A reasonable public health goal would be to limit the contribution of industrial sources to 1% of that value (10^{-3}), and a reasonable rule for a single hazard would be to limit it to 1% of that value, or 10^{-5} . This argument takes into account the fact that any one person is very unlikely to be simultaneously exposed to a vast array of cancer hazards.

The state of Virginia's choice of 10^{-4} is thus near the lower limit of risks that attempt a balance between protection of the individual and unreasonable overregulation. The Commonwealth of Virginia has acknowledged these precedents by setting its ambient water quality standard for dioxin on the basis of a 10^{-5} risk factor. The above precedents, furthermore, are all based on the assumption that any cancer caused would carry a high potential of lethality. The only cancers associated with arsenic exposure in water are skin cancers, which as stated above are rarely fatal. An EPA proposal (1988) suggested that concern about arsenic might be ten-fold lower than for other carcinogens. A value of 10^{-4} for a carcinogen of low lethal potential is therefore more comparable with the bulk of regulatory decisions in similar circumstances and consistent with decisions regarding arsenic clean-up levels at several Superfund sites. In suggesting this, we emphasize that nearly all of the assumptions used by Virginia in calculating a proposed arsenic water quality standard are conservative. Thus, the assumptions applied do not

take into account the data suggesting that, at low doses, arsenic is largely detoxified; consequently, the risks calculated based on the EPA cancer potency factor may substantially overestimate the true risk at low doses.

IV. CONCLUSIONS AND RECOMMENDATIONS

The assumptions used by the Commonwealth of Virginia are conservative. For example, ENVIRON believes that the weight of the scientific evidence indicates that arsenic is even less potent a carcinogen than is indicated by the present EPA CPF of $1.75 \text{ (mg/kg/day)}^{-1}$. In risk assessment, each successive conservative assumption compounds those preceding it. The EPA MCL for arsenic is $50 \text{ }\mu\text{g/l}$. That standard has been adopted as an acceptable water quality standard in numerous states (see Table 2). It does not seem reasonable to require a surface water body that is a direct source of public water to contain less arsenic than can be supplied by a municipal water supply. Thus, $50 \text{ }\mu\text{g/l}$ appears to be an acceptable water quality standard for arsenic.

TABLE 1

**Regional Freshwater Fish Consumption
1980s Per Capita Average, All Ages
(g/day)**

United States	1.38
New England	0.26
Mid Atlantic	0.87
S. Atlantic	1.05
E.S. Central	2.24
E.N. Central	1.79
W.N. Central	2.05
W.S. Central	2.69
Mountain	1.47
Pacific	1.05

TABLE 2

**Water Quality Standards for Arsenic
in Eastern and Southern U.S.
for the Protection of Human Health**

Arkansas	50 $\mu\text{g/l}$
Connecticut	50 $\mu\text{g/l}$
Delaware	50 $\mu\text{g/l}$ fish and water
Florida	50 $\mu\text{g/l}$
Georgia	50 $\mu\text{g/l}$ freshwater 36 $\mu\text{g/l}$ coastal & estuarine
Kentucky	50 $\mu\text{g/l}$
Maine	50 $\mu\text{g/l}$
Maryland	50 $\mu\text{g/l}$
Mississippi	50 $\mu\text{g/l}$
New Jersey	50 $\mu\text{g/l}$
North Carolina	50 $\mu\text{g/l}$
Pennsylvania	50 $\mu\text{g/l}$
Rhode Island	50 $\mu\text{g/l}$
South Carolina	50 $\mu\text{g/l}$

V. REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1987. Toxicological profile for arsenic. ORNL TP-ATSDR-4228AR.
- Allen, B.M., A.M. Shipp, K.S. Crump, B. Killian, M.L. Hogg, J. Tudor, and B. Keller. 1987. Investigation of Cancer Risk Assessment Methods: Summary. Prepared by Clement Associates, Inc. for the U.S. Environmental Protection Agency. EPA/600/6-87/007a. Washington, DC: EPA.
- Arguello, R.A., E.E. Tello, and D.D. Cengel. 1939. Cancer and regional endemic chronic arsenicalism. *Br. J. Dermatol.* 51:548.
- Borgono, J.M., P. Vicent, H. Venturino, and A. Infante. 1977. Arsenic in the drinking water of the city of Antofagasta: Epidemiological and clinical study before and after the installation of a treatment plant. *Environ. Health Perspect.* 19:103-105.
- Boyle, R.W., and I.R. Jonasson. 1973. The geochemistry of arsenic and its use as an indicator element in geochemical prospecting. *J. Geochem. Explor.* 2:251-296.
- California Health and Welfare Agency (CHWA). 1987. No Significant Risk Levels. Article 7 of Safe Drinking Water and Toxic Enforcement Act of 1986, Chapter 3. 22 Calif. Code of Regulations Division 2. Sacramento, CA: Health and Welfare Agency.
- ENSR Consulting and Engineering (ENSR). 1989. Technical support document for a proposed Alabama water quality standard for dioxin. Boston, MA:ENSR.
- Environmental Protection Agency (EPA). 1980. Ambient water criteria for arsenic. Office of water regulation and standards. EPA 440/5-80-021. October.
- Environmental Protection Agency (EPA). 1981. An exposure and risk assessment for arsenic. Final Draft Report. Office of Water Regulation Standards.
- Environmental Protection Agency (EPA). 1984. Health assessment document for inorganic arsenic. Office of Health and Environmental Assessment. EPA-600/8-83-021F. March.
- Environmental Protection Agency (EPA). 1988. Special report on ingested inorganic arsenic: Skin Cancer; Nutritional Essentiality. Risk Assessment Forum. EPA/625/3-87/013. July.

- Environmental Protection Agency (EPA). 1989. Exposure factors handbook. EPA/600/8-89/043. Washington, DC: EPA.
- Geyer, L. 1898. Ueber die chronischen Hautveränderungen beim Arsenicismus und Betrachtungen ueber die Massenerkrankungen in Reichenstein. Arch. Dermatol. Syphilol. 43:221-280.
- Harrington, J.M., J.P. Middaugh, D.L. Morse, and J. Houseworth. 1978. A survey of a population exposed to high arsenic in well water in Fairbanks, Alaska. Amer. J. Epidemiol. 108:377-385.
- International Agency for Research on Cancer (IARC). 1980. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Vol. 23: Some metals and metallic compounds. Lyon, France: World Health Organization.
- International Commission on Radiological Protection (ICRP). 1984. Report of the Task Group on Reference Man. ICRP Publication No. 23. New York: Pergamon Press.
- Lu, F.-J. 1978. Study on fluorescent compounds in drinking water of endemic areas of Black-foot disease and reinvestigation of the causes of Blackfoot disease. K'o Hsueh Fa Chan Yueh K'an. 6:388-403.
- Maryland Department of the Environment (MDE). 1990. Issue paper on "The Necessity of a Fish Consumption Criterion for Arsenic. January 4. Baltimore: MDE.
- Morton, W.G., G. Starr, D. Pohl, J. Stoner, S. Wagner, and P. Weswig. 1976. Skin cancer and water arsenic in Lane County, Oregon. Cancer 37:2523-2532.
- Mushak, P., K. Dessauer, and E.L. Walls. 1977. Flameless atomic absorption (FAA) and gas-liquid chromatographic studies in arsenic bioanalysis. Environ. Health Perspec. 19:5-10.
- National Academy of Sciences (NAS). 1977. Drinking water and health. Safe Drinking Water Committee, National Research Council. National Academy of Sciences. Washington, D.C.: National Academy of Press.
- Rodricks, J.V., S.M. Brett, and G.C. Wrenn. 1987. Significant risk decisions in federal regulatory agencies. Reg. Toxicol. Pharmacol. 7:307-320. SRI International (SRI). 1980. Seafood Consumption Data Analysis. Report prepared by SRI International for the U.S. Environmental Protection Agency. Menlo Park, CA: SRI.

- Southwick, J.W., A.E. Western, M.M. Beck, T. Whitley, R. Isaacs, J. Petajan, and C.D. Hansen. 1981. Community health associated with arsenic in drinking water in Millard County Utah. Health Effects Research Laboratory, U.S. Environmental Protection Agency, Cincinnati, OH. EPA Report No. EPA-600/1-81-064. NTIS PB82-108374.
- Tseng, W.-P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. *Environ. Health Perspect.* 19:109-119.
- Tseng, W.-P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin, and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. *J. Natl. Cancer Inst.* 40:453-463.
- Travis, C.C., S.A. Richter, E.A.C. Crouch, R. Wilson, and E.D. Klema. 1987. Cancer risk management: A review of 132 federal regulatory decisions. *Environ. Sci. Technol.* 21:415-420.
- United States Geological Survey (USGS). 1984. Water resources data, Virginia, Water year 1984. U.S. Geological Survey Water-Data Report VA-84-1.
- Zaldivar, R. 1974. Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning. *Beitr. Pathol. Bd.* 151:384-400.

APPENDIX D

Specific Water Quality Criteria
for New Use Designation -
Warmwater B4

Specific Water Quality Criteria

For New Use Designation Warmwater B4

(Add New Subsections and Column to Existing Table)

Parameter	Units	Use Designation Warmwater B4
8.1 Aluminum		--
8.2.2 Ammonia (New subsection) The concentration of unionized ammonia shall not exceed values in Table 1 (attached)	ug/L	X
8.3.2 Arsenic Not to exceed 440 ug/L Trivalent Arsenic	ug/L	440
8.4 Barium		--
8.5.4 Cadmium (New subsection)		X
Hardness mg/L as CaCO_3	Total Recoverable Cadmium ug/L Calculate from equation	
0 - 100		
101 - 150	46	
151 - 200	64	
201 - 300	103	
301 - 400	144	
8.6.1 Chloride (New subsection) Not to exceed 1200 mg/L chloride	mg/L	1,200
8.7.2 Copper (New subsection)		X
Hardness mg/L as CaCO_3	Total Recoverable Copper ug/L	
50	9.2	
100	18	
200	34	
300	42	
400	55	
500	67	
600-700	Calculate from equation	

8.8.1 Cyanide (New subsection) As free cyanide HCN + CN ⁻): Not to exceed 38 ug/L	ug/L	38
8.9.4 Dissolved Oxygen (New subsection) Not less than 2.0 mg/L at any time	mg/L	2.0
8.10 Fecal Coliform		--
8.11.2 Fluoride		--
8.12.3 Hexavalent Chromium (New subsection) Not to exceed 19 ug/L	u/gL	19
8.13.3 Iron (New subsection) Not to exceed 4.0 mg/L total iron or 3.7 mg/L EPA-reactive ferrous iron	mg/L	X
8.14.2 Lead (New subsection)		--
8.15.2 Manganese (New subsection) Not to exceed 2.0 mg/L	mg/L	2.0
8.16 Mercury (Total)	ug/L	0.5
8.16.1 (New subsection) Total Mercury Concentration. In any unfiltered water sample shall not exceed 2.2 ug/L	ug/L	2.2
8.17 Nickel Not to exceed 50 ug/L		--
8.18 Nitrate (as Nitrate - N) Not to exceed 10 mg/L		--
8.19 Nitrite (as Nitrite - N) Not to exceed 1.0 mg/L		--
8.20 Organics		X
8.21 pH		X
8.22.1 Phenolic Materials (New subsection) To be determined on a case-by- case basis		X
8.23 Radioactivity		X
8.24.2 Selenium (New subsection) Not to exceed 260 ug/L Selenite	ug/L	260
8.25.1 Silver		X

8.26.4	Temperature (New subsection)	°F	98
8.27	Threshold Odor		X
8.28.3	Total Residual Chlorine (New subsection)		
	Not to exceed 19 ug/L as measured by the amperometric or equivalent method	ug/L	19
8.29	Turbidity		X
8.29.1	Turbidity		X
8.30.3	Zinc (New subsection)		X

Hardness	Zinc (ug/L)
mg/L as CaCO ₃	
0-149	Calculate using equation
150	390
151-200	495
201-300	693
301-400	880
401-500	1,060
500-	calculate using equation