WEST VIRGINIA

SECRETARY OF STATE

KEN HECHLER

ADMINISTRATIVE LAW DIVISION

Form #3

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OFFICE OF WEST VIRGINIA
SECRETARY OF STATE

NOTICE OF AGENCY APPROVAL OF A PROPOSED RULE AND FILING WITH THE LEGISLATIVE RULE-MAKING REVIEW COMMITTEE

AGENCY: State Water Resources Board TITLE NUMBER: 46

CITE AUTHORITY Chapter 20-5 & 20-5A

AMENDMENT TO AN EXISTING RULE: YES X NO

IF YES, SERIES NUMBER OF RULE BEING AMENDED: 1

TITLE OF RULE BEING AMENDED: Legislative Rules Water Resources Board Requirements Governing Water Quality Standards

IF NO, SERIES NUMBER OF NEW RULE BEING PROPOSED:

TITLE OF RULE BEING PROPOSED:

THE ABOVE PROPOSED LEGISLATIVE RULE HAVING GONE TO A PUBLIC HEARING OR A PUBLIC COMMENT PERIOD IS HEREBY APPROVED BY THE PROMULGATING AGENCY FOR FILING WITH THE SECRETARY OF STATE AND THE LEGISLATIVE RULE MAKING REVIEW COMMITTEE FOR THEIR REVIEW.

Frances E. Hunter

STATE WATER RESOURCES BOARD

1260 Greenbrier Street
Charleston, WV 25311
(304) 348-4002

August 21, 1991

The Honorable Ken Hechler
Secretary of State
State Capitol Bldg. # 1
Charleston, West Virginia 25305

Dear Mr. Secretary:

Enclosed find a copy of the State Water Resources Board's approved proposed rule Title 46 - Series 1 Legislative Rules - Water Resources Board - Requirements Governing Water Quality Standards along with the required filing form. On this date fifteen (15) copies of this proposed rule was also filed with the Legislative Rule-Making Review Committee.

If you have any question do not hesitate to contact this office.

Respectfully submitted,

Frances E. Hunter

Frances E. Hunter
Executive Secretary

c
Legislative Rule-Making
Review Committee
John M. Ranson, Cabinet
Secretary

FISCAL NOTE FOR PROPOSED RULES

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1991 AUG 21 PM 4:18

OFFICE OF WEST VIRGINIA SECRETARY OF STATE

Rule Title: Title 46 - Series 1 - Requirements Governing Water Quality Standards

Type of Rule: X Legislative ____ Interpretive ____ Procedural

Agency Dept. of Commerce, Labor & Environmental Resources-Water Resources Board Address 1260 Greenbrier Street Charleston, WV 25311

1. Effect of Proposed Rule	ANNUAL Increase	ANNUAL Decrease	FISCAL YEAR Current	FISCAL YEAR Next	FISCAL YEAR Thereafter
Estimated Total Cost	$	$	$	$	$
Personal Services	NONE				
Current Expense					
Repairs and Alterations					
Equipment					
Other					

2. Explanation of above estimates:

N/A

3. Objectives of these rules:

Trienniel review to meet federally-mandated changes in water quality criteria which insure the protection and maintenance of the State's water resources.

4. Explanation of Overall Economic Impact of Proposed Rule.

A. Economic Impact on State Government.

None.

B. Economic Impact on Political Subdivisions; Specific Industries; Specific groups of citizens.

The proposed rule may increase wastewater treatment costs for some industries including coal mining and electric power production.

C. Economic Impact on Citizens/Public at Large.

Cost of improved wastewater treatment may be passed on to utility customers. Improved water quality may enhance the attractiveness of West Virginia as a travel destination and thereby increase the State's economic base.

Date: August 21, 1991

Signature of Agency Head or Authorized Representative

Frances E Hunter

PROPOSED
TITLE 46
LEGISLATIVE RULES
STATE WATER RESOURCES BOARD

REQUIREMENTS GOVERNING WATER QUALITY STANDARDS
SERIES 1
1991

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STATE WATER RESOURCES BOARD
1260 Greenbrier Street
Charleston, WV 25311
(304)348-4002

PROPOSED
TITLE 46
LEGISLATIVE RULES
WATER RESOURCES BOARD

REQUIREMENTS GOVERNING WATER QUALITY STANDARDS
Series 1
1991

46-1-1. General

1.1 Scope - These rules establish requirements governing the discharge or deposit of sewage, industrial wastes and other wastes into the waters of the State and establish water quality standards for the waters of the State standing or flowing over the surface of the State. 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, and other 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. (See W. Va. Code 20-5A-1.)

1.2 Authority - W. Va. Code 20-5A-3.

1.3 Filing Date - ~~May 9, 1991.~~ _____.

1.4 Effective Date - ~~May 9, 1991.~~ _____.

1.5 Repeal of Former Rule - This Legislative rule repeals West Virginia Administrative Regulations, State Water Resources Board, Chapter 20-5 and 5A, Series I (1991), "Requirements Governing Water Quality Standards", filed May 9, 1991.

46-1-2. Definitions

The following definitions in addition to those set forth in Chapter 20, Article 5A, Section 2, shall apply to these rules unless otherwise specified herein, or unless the context in which used clearly requires a different meaning:

2.1 "Conventional treatment" is the treatment of water as approved by the State Health Department to assure that

the Water is safe for human consumption.

2.2 "Cumulative" means a pollutant which increases in concentration in an organism by successive additions at different times or in different ways (bio-accumulation).

2.3 The "Federal Act" means the Clean Water Act (also known as the Federal Water Pollution Control Act) Public Law 92-500, as amended by Public Law 100-4, 33 U.S.C. 1251, et seq.

2.4 "High quality waters" are those waters whose quality is equal to or better than the minimum levels necessary to achieve the national water quality goal uses. Included are those streams or stream segments which receive annual stockings of trout but which do not support year-round trout populations.

2.5 "Intermittent streams" are streams which have no flow during sustained periods of no precipitation and which do not support aquatic life whose life history requires residence in flowing waters for a continuous period of at least six (6) months.

2.6 National Resource Waters" are those whose unique character, ecological or recreational value or pristine nature constitutes a valuable national or State resource. (See Section 7.3.)

2.7 "Natural" or "naturally occurring" values or " natural temperature" shall mean for all of the waters of the State:

2.7.a Those water quality values which exist unaffected by -- or unaffected as a consequence of -- any water use by any person; and

2.7.b Those water quality values which exist unaffected by the discharge, or direct or indirect deposit, of any solid, liquid or gaseous substance by any person.

2.8 "Non-point source" shall mean any source other than a point source from which pollutants may reach the waters of the State.

2.9 "Persistent" shall mean a pollutant and its transformation products which under natural conditions degrade slowly in an aquatic environment.

2.10 "Point source" shall mean any discernible, confined and discrete conveyance, including, but not limited to,

any pipe, ditch, channel, tunnel, conduit, well, discrete fissure, container, rolling stock, or vessel or other floating craft, from which pollutants are or may be discharged. This term does not include agricultural stormwater discharges and return flows from irrigated agriculture.

2.11 "Representative important species of aquatic life" shall mean those species of aquatic life whose protection and propagation will assure the sustained presence of a balanced aquatic community. Such species are representative in the sense that maintenance of water quality criteria will assure both the natural completion of the species' life cycles and the overall protection and sustained propagation of the balanced aquatic community.

2.12 The "State Act" or "State Law" shall mean the West Virginia Water Pollution Control Act, section one, article five(a), chapter twenty, et seq. of the West Virginia Code.

2.13 "Total recoverable" refers to the digestion procedure for certain heavy metals as referenced in 40 CFR 136, as amended June ~~30~~ 15, ~~1986~~ 1990 , Guidelines Establishing Test Procedures for the Analysis of Pollutants Under the Clean Water Act.

2.14 "Trout waters" are streams or stream segments which sustain year-round trout populations. Excluded are those streams or stream segments which receive annual stockings of trout but which do not support year-round trout populations.

2.15 "Water quality criteria" shall mean levels of parameters or stream conditions that are required to be maintained by these regulations. Criteria may be expressed as a constituent concentration, levels, or narrative statement, representing a quality of water that supports a designated use or uses.

2.16 "Water quality standards" means the combination of water uses to be protected and the water quality criteria to be maintained by these rules.

2.17 "Wetlands" include such areas as swamps, marshes, bogs, and other land subject to frequent saturation or inundation, and which normally support a prevalence of vegetation typically found where wet soil conditions prevail.

2.18 "Wet weather streams" are streams that flow only in direct response to precipitation or whose channels are at

all times above the water table.

46-1-3. Conditions Not Allowable in State Waters

3.1 Certain characteristics of sewage, industrial wastes and other wastes cause pollution and are objectionable in all waters of the State. Therefore, the State Water Resources Board does hereby proclaim that the following general conditions are not to be allowed in any of the waters of the State.

3.2 No sewage, industrial wastes or other wastes present in any of the waters of the State shall cause therein or materially contribute to any of the following conditions thereof:

3.2.a Distinctly visible floating or settleable solids, suspended solids, scum, foam or oily slicks;

3.2.b Deposits or sludge banks on the bottom.

3.2.c Odors in the vicinity of the waters;

3.2.d Taste or odor that would adversely affect the designated uses of the affected waters;

3.2.e Materials in concentrations which are harmful, hazardous or toxic to man, animal or aquatic life;

3.2.f Distinctly visible color;

3.2.g Concentrations of bacteria which may impair or interfere with the designated uses of the affected waters;

3.2.h Requiring an unreasonable degree of treatment for the production of potable water by modern water treatment processes as commonly employed; and

3.2.i Any other condition, including radiological exposure, which adversely alters the ~~chemical, physical or biological~~ integrity of the waters of the State including wetlands ~~.~~; no significant adverse impact to the chemical, physical, hydrologic, or biological components of aquatic ecosystems shall be allowed.

46-1-4. Anti-degradation Policy

4.1 It is the policy of the State of West Virginia that instream water uses shall be maintained and protected as follows:

4.1.a Existing instream water uses and the level of water quality necessary to protect the existing uses shall be maintained and protected. 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. Waste assimilation and transport are not recognized as designated uses. The classification of the waters must take into consideration the use and value of water for public water supplies, protection and propagation of fish, shellfish and wildlife, recreation in and on the water, agricultural, industrial and other purposes including navigation. Subcategories of a use may be adopted and appropriate criteria set to reflect varying needs of such subcategories of uses, for example to differentiate between trout water and other waters. (See 4.1.d).

4.1.b At a minimum, uses are deemed attainable if they can be achieved by the imposition of effluent limits required under Sections 301(b) and 306 of the Federal Clean Water Act and use of cost-effective and reasonable best management practices for non-point source control. Seasonal uses may be adopted as an alternative to reclassifying a water body or segment thereof to uses requiring less stringent water quality criteria. If seasonal uses are adopted, water quality criteria will be adjusted to reflect the seasonal uses; however, such criteria shall not preclude the attainment and maintenance of a more protective use in another season. A designated use which is not an existing use may be removed, or subcategories of a use may be established if it can be demonstrated that attaining the designated use is not feasible because:

4.1.b.1 Application of effluent limitations for existing sources more stringent than those required pursuant to Section 301(b) and Section 306 of the Federal Act in order to attain the existing designated use would result in substantial and widespread adverse economic and social impact; or

4.1.b.2 Naturally-occurring pollutant concentrations prevent the attainment of the use; or

4.1.b.3 Natural, ephemeral, intermittent or low flow conditions of water levels prevent the attainment of the use, unless these conditions may be compensated for by the discharge of sufficient volume of effluent discharges to enable uses to be met;

or

4.1.b.4 Human-caused conditions or sources of pollution prevent the attainment of the use and cannot be remedied or would cause more environmental damage to correct than to leave in place; or

4.1.b.5 Dams, diversions or other types of hydrologic modifications preclude the attainment of the use, and it is not feasible to restore the water body to its original condition or to operate such modification in a way that would result in the attainment of the use; or

4.1.b.6 Physical conditions related to the natural features of the water body, such as the lack of a proper substrate, cover, flow, depth, pools, riffles, and the like, unrelated to water quality, preclude attainment of aquatic life protection uses.

4.1.c The State shall take into consideration the quality of downstream waters and shall assure that its water quality standards provide for the attainment of the water quality standards of downstream waters.

4.1.d In establishing a less restrictive use or uses, or subcategory of use or uses, and the water quality criteria based upon such uses, the Board shall follow the requirements for revision of water quality standards as required by section three(a), article five A, chapter twenty of the West Virginia Code and Section 303 of the Federal Act and the regulations thereunder. Any revision of water quality standards shall be made with the concurrence of EPA. The Board's administrative procedural regulations for applying for less restrictive uses or criteria shall be followed.

4.1.e With the exception of the provisions of Section 7.2.c of this series, the existing trout and other high quality waters of the State must be maintained at their existing high quality unless it is determined after consultation with EPA and the Chief and opportunity for public comment and hearing that allowing lower water quality is necessary to accommodate important economic or social development in the area in which the waters are located. If limited degradation is allowed, it shall not result in injury or interference with existing stream water uses or in violation of State or Federal water quality criteria that describe the base levels necessary to sustain the national water quality goal uses of protection and propagation of fish, shellfish and wildlife and recreation in and on the water.

4.1.f The Board and the Chief shall assure that all new and existing point sources shall achieve the highest established statutory and regulatory requirements applicable to them and shall assure the achievement of cost-effective and reasonable best management practices for non-point source control.

4.1.g In all cases, waters which constitute an outstanding national resource as designated in Section 7.3.d shall be maintained and protected and improved where necessary.

4.1.h All applicable requirements of Section 316 (a) of the Federal Act shall apply to modifications of the temperature water quality criteria provided for in these rules.

46-1.5. Mixing Zones

5.1 In the permit review and planning process or upon the request of a permit applicant or permittee, the Chief may establish on a case-by-case basis an appropriate mixing zone.

5.2 The following criteria shall be applied to the establishment of mixing zones:

5.2.a Mixing zones shall:

5.2.a.1 Be kept as small as practical in area and length;

5.2.a.2 Not be used for, or considered as, a substitute for waste treatment;

5.2.a.3 Provide for as rapid a mixing as practical;

5.2.a.4 Not prevent the free passage of aquatic species or include spawning or nursery areas;

5.2.a.5 Not overlap a public water supply intake;

5.2.a.6 Not cause or contribute to any of the conditions prohibited in Section 3; and

5.2.a.7 Not interfere with any designated water use category.

5.3 The boundaries of the mixing zone shall

reflect:

5.3.a Receiving water body characteristics such as:

5.3.a.1 Water quality,

5.3.a.2 Local meteorology,

5.3.a.3 Flow regime (including low-flow records),

5.3.a.4 Magnitude of water exchange at point of discharge,

5.3.a.5 Stratification phenomena,

5.3.a.6 Waste capacity of the receiving system including retention time,

5.3.a.7 Turbulence and speed of flow;

5.3.a.8 Morphology of the receiving system as related to plume behavior, and biological phenomena;

5.3.a.9 Designated water use categories; and

5.3.b Discharge characteristics such as:

5.3.b.1 Flow regime,

5.3.b.2 Volume,

5.3.b.3 Design,

5.3.b.4 Location,

5.3.b.5 Rate of mixing and dilution, and

5.3.b.6 Plume behavior and mass-emission rates of constituents including knowledge of their persistence, toxicity, and chemical or physical behavior with time..

5.4 Where the seven (7) day, ten (10) year return frequency is 5 cfs or less, no mixing zone may be established.

5.5 In order to facilitate a determination or assessment of a mixing zone pursuant to this section, the Chief may require a permit applicant or permittee to submit such information as deemed necessary.

46-1-6. Water Use Categories

6.1 These rules establish general Water Use Categories and Water Quality Standards for the waters of the State. 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 for Water Contact Recreation (Category C) consistent with Clean Water Act goals. Incidental utilization for whatever purpose may or may not constitute a justification for assignment of a Water Use Category to a particular stream segment.

6.2 Category A - Water Supply, Public - This category is used to describe waters which, after conventional treatment, are used for human consumption. This category includes:

6.2.a All community domestic water supply systems;

6.2.b All non-community domestic water supply systems, (i.e. hospitals, schools, etc.);

6.2.c All private domestic water systems; and

6.2.d All other surface water intakes where the water is used for human consumption, and

6.2.e Shall apply to the stream segment extending upstream from the intake for a distance as defined in Section 7.2.a.2 of this series. (See Appendix B for partial listing).

6.3 Category B - Propagation and maintenance of Fish and Other Aquatic Life. This category includes:

6.3.a Category B1 - Warm Water Fishery Streams. Streams or stream segments which contain a fish population composed overwhelmingly of warm water species. (These are primarily sport fisheries and may be stocked with trout seasonally.)

6.3.b Category B2 - Trout Waters - As defined in Section 2.14 (see Appendix A for a representative list).

6.3.c. Category B3 - Small Non-Fishable Streams. Streams or stream segments which because of their size or flow patterns do not offer sport fishing; they generally contain only minnows, darters, etc.

6.3.d. Category B4 - Wetlands. As defined in Section 2.17; numeric stream criteria may not be appropriate for application to wetlands (see Appendix E).

6.4 Category C - Water Contact Recreation. This category includes swimming, fishing, water skiing and certain types of pleasure boating such as sailing in very small craft and outboard motor boats.

6.5 Category D - Agriculture and Wildlife Uses.

6.5.a Category D1 - Irrigation. This category includes all stream segments used for irrigation.

6.5.b Category D2 - Livestock Watering. This category includes all stream segments used for livestock watering.

6.5.c Category D3 - Wildlife. This category includes all stream segments and wetlands used by wildlife.

6.6 Category E - Water Supply Industrial, Water Transport, Cooling and Power. This category includes cooling water, industrial water supply, power production, commercial and pleasure vessel activity, except those small craft included in Category C.

6.6.a Category E1 - Water Transport. This category includes all stream segments modified for water transport and having permanently maintained navigation aides.

6.6.b Category E2 - Cooling Water. This category includes all stream segments having one or more users for industrial cooling.

6.6.c Category E3 - Power Production. This category includes all stream segments extending from a point 500 feet upstream from the intake to a point one half (1/2) mile below the wastewater discharge point. (See Appendix C for representative list.)

6.6.d Category E4 - Industrial. This category is used to describe all stream segments with one or more industrial users. It does not include water for cooling.

46-1-7. WEST VIRGINIA WATERS

7.1 Major River Basins and Their Alphanumeric System. All streams and their tributaries in West Virginia shall be individually identified using an alphanumeric system as identified in the "Key to West Virginia Stream Systems and Major Tributaries" (1956) as published by the Conservation Commission of West Virginia and revised by the West Virginia Department of Natural Resources, Division of Wildlife (1985).

7.1.a J - James River Basin. All tributaries to the West Virginia-Virginia State line.

7.1.b P - Potomac River Basin. All tributaries of the main stem of the Potomac River to the West Virginia-Maryland-Virginia State line to the confluence of the North Branch and the South Branch of the Potomac River and all tributaries arising in West Virginia excluding the major tributaries hereinafter designated:

7.1.b.1 S - Shenandoah River and all its tributaries arising in West Virginia to the West Virginia-Virginia State line.

7.1.b.2 PC - Cacapon River and all its tributaries.

7.1.b.3 PSB - South Branch and all its tributaries.

7.1.b.4 PNB - North Branch and all tributaries to the North Branch arising in West Virginia.

7.1.c M - Monongahela River Basin. The Monongahela River Basin main stem and all its tributaries excluding the following major tributaries which are designated as follows:

7.1.c.1 MC - Cheat River and all its tributaries except those listed below:

7.1.c.1.A MCB - Blackwater River and all its tributaries.

7.1.c.2 MW - West Fork River and all its tributaries.

7.1.c.3 MT - Tygart River and all its tributaries except those listed below:

7.1.c.3.A MTB - Buckhannon River and all its tributaries.

7.1.c.3.B MTM - Middle Fork River and all its tributaries.

7.1.c.4 MY - Youghigheny River and all its tributaries to the West Virginia-Maryland State line.

7.1.d O Zone 1 - Ohio River - Main Stem. The main stem of the Ohio River from the Ohio-Pennsylvania-West Virginia State line to the Ohio-Kentucky-West Virginia State line.

7.1.e O Zone 2 - Ohio River - Tributaries. All tributaries of the Ohio River excluding the following major tributaries:

7.1.e.1 LK - Little Kanawha River. The Little Kanawha River and all its tributaries excluding the following major tributary which is designated as follows:

7.1.e.1.A LKH - Hughes River and all its tributaries.

7.1.e.2 K - Kanawha River Zone 1. The main stem of the Kanawha River from mile point 0, at its confluence with the Ohio River, to mile point 72 near Diamond, West Virginia.

7.1.e.3 K - Kanawha River Zone 2. The main stem of the Kanawha River from mile point 72 near Diamond, West Virginia and all its tributaries from mile point 0 to the headwaters excluding the following major tributaries which are designated as follows:

7.1.e.3.A KP - Pocatalico River and all its tributaries.

7.1.e.3.B KC - Coal River and all its tributaries.

7.1.e.3.C KE - Elk River and all its tributaries.

7.1.e.3.D KG - Gauley River. The Gauley River and all its tributaries excluding the following major tributaries which are designated as follows:

7.1.e.3.D.i KG-19 - Meadow River and all its tributaries.

7.1.e.3.D.ii KG-34 - Cherry River and all its tributaries.

7.1.e.3.D.iii KGC - Cranberry River and all its tributaries.

7.1.e.3.D.iv KGW - Williams River and all its tributaries.

7.1.e.3.E KN - New River. The New River from its

confluence with the Gauley River to the Virginia-West Virginia State line and all tributaries excluding the following major tributaries which are designated as follows:

7.1.e.3.E.i KNG - Greenbrier River and all its tributaries.

7.1.e.3.E.ii KNB - Bluestone River and all its tributaries.

7.1.e.3.E.iii KN-60 - East River and all its tributaries.

7.1.e.3.E.iv K(L)-81-(1) - Bluestone Lake.

7.1.e.4 OG - Guyandotte River. The Guyandotte River and all its tributaries excluding the following major tributary which is designated as follows:

7.1.e.4.A OGM - Mud River and all its tributaries.

7.1.e.5 BS - Big Sandy River. The Big Sandy River to the Kentucky-Virginia-West Virginia State lines and all its tributaries arising in West Virginia excluding the following major tributary which is designated as follows:

7.1.e.5.A BST - Tug Fork and all its tributaries.

7.2 Applicability of Water Quality Standards. The following shall apply at all times unless a specific exception is granted in this section:

7.2.a Water Use Categories as described in Section 6.

7.2.a.1 Based on meeting those Section 6 definitions, tributaries or stream segments may be classified for one or more Water Use Categories. When more than one use exists, they shall be protected by criteria for the use category requiring the most stringent protection.

7.2.a.2 Each segment extending upstream from the intake of a water supply public (Water Use Category A), for a distance of five (5) miles or to the headwater, must be protected by prohibiting the discharge of any pollutants in excess of the concentrations designated for this Water Use Category in Section 8. Provided, however, that within a zone extending one half (1/2) mile above the intake, the Chief, Water Resources Section, Division of Natural Resources, may establish for any discharge,

effluent limitations for the protection of human health that require additional removal of those pollutants. (If a watershed is not significantly larger than either of the two (2) zones above the intake, the water supply section may include the entire upstream watershed to its headwaters.)

7.2.b In the absence of any special application or contrary provision, water quality standards shall apply at all times when flows are equal to or greater than the minimum mean seven (7) consecutive day drought flow with a ten (10) year return frequency (7Q10). NOTE: Exceptions do not apply to Trout Waters~~.~~ nor to the requirements of Section 3.

7.2.c Exceptions: Water quality standards shall not apply: (See Section 7.2 for site specific revisions.)

7.2.c.1 When the flow is less than 7Q10;

7.2.c.2 In wet weather streams (or intermittent streams, when they are dry or have no measurable flow) provided that the designated uses of downstream waters are not adversely affected;

7.2.c.3 In any mixing zones which are established pursuant to Section 5 of these rules;

7.2.c.4 Where lesser quality is due to natural conditions. In such cases the naturally-occurring values shall be the applicable criteria.

7.2.d Site-specific applicability of water use categories and water quality criteria - State-wide water quality standards shall apply except where site-specific water quality standards have been adopted for the State's waters as follows:

7.2.d.1 James River - (Reserved)

7.2.d.2 Potomac River

7.2.d.2.A Except for the unnamed tributary of the South Branch of Buzzard Run above and below Prather Pond shall not have Water Use Category A; therefore may contain fluoride not to exceed 2.0 mg/l.

~~7.2.d.2.B Except for Turkey Run, a small tributary of Opequon Creek (P-4) of the Potomac River, shall not have Water Use Category A, and therefore may contain fluoride not to exceed 8 mg/l, ammonia not to exceed 4 mg/l, chlorides not to exceed 530~~

~~mg/l, cyanide (as free cyanide HCN + CN) not to exceed 50 ug/l and hexavalent chromium (total) not to exceed 100 ug/l.~~

7.2.d.3 Shenandoah River - (Reserved)

7.2.d.4 Cacapon River - (Reserved)

7.2.d.5 South Branch - (Reserved)

7.2.d.6 North Branch

7.2.d.6.A Except that the Stony River downstream from the limit of the thermal mixing zone (as established by Board Order of 11/20/75) for the Mount Storm Lake wastewater treatment facility to its confluence with the North Branch of the Potomac River is exempt from the 5°F above natural temperature rise; however, the maximum temperature outside the mixing zone shall not exceed 87°F at any time during the months of May through November and not to exceed 73°F at any time during the months of December through April.

7.2.d.7 Monongahela River

7.2.d.7.A Except that flow in the main stem of the Monongahela River, as regulated by the Tygart Reservoir, operated by the U. S. Army Corps of Engineers, is based on a minimum flow of 345 cfs at Lock and Dam No. 8, river mile point 90.8. This exception does not apply to tributaries of the Monongahela River.

7.2.d.7.B Except for an unnamed tributary to the Monongahela River mainstem at approximately 3700 feet upstream of mile point 125, which may contain suspended solids not to exceed 60 mg/1, oil and grease not to exceed 15 mg/1, Ammonia-Nitrogen not to exceed 30 mg/1, total phenols not to exceed .10 mg/1, total cyanide not to exceed .05 mg/1, total manganese not to exceed 4 mg/1, total zinc not to exceed 1.5 mg/1, total copper not to exceed 1.0 mg/1, Benzene not to exceed .05 mg/1, Napthalene not to exceed .05 mg/1 and Benzo (a) Pyrene not to exceed .05 mg/1 and iron not to exceed 4 mg/1 for the months June through November and 7 mg/1 for the months of December through May.

7.2.d.8 Cheat River - (Reserved)

7.2.d.9 Blackwater River - (Reserved)

7.2.d.10 West Fork River - (Reserved)

7.2.d.11 Tygart River - (Reserved)

7.2.d.12 Buckhannon River - Reserved)

7.2.d.13 Middle Fork River - (Reserved)

7.2.d.14 Youghiogheny River

7.2.d.14.A Water Use Categories A and E are excluded from the tributaries of the Youghiogheny River in West Virginia which flow into Maryland.

7.2.d.15 Ohio River Main Stem - (Reserved)

7.2.d.16 Ohio River Tributaries

7.2.d.16.A Except the stretch of Conners Run (O-77-A), a tributary of Fish Creek, from its mouth to the discharge from Conner Run impoundment, shall not have the Water Use Category A and may contain arsenic not to exceed 200 ug/l; selenium not to exceed 62 ug/l; and, iron not to exceed 3.5 mg/l as a monthly average and 7 mg/l as a daily maximum.

7.2.d.16.B Except for that segment of Harmon Creek (O-97) from its confluence with the Ohio River to a point 2.2 miles upstream shall not have the Water Use Category A designation. Therefore, at any time the temperature shall not exceed 100°F, total iron shall not exceed 4.0 mg/l and total fluoride shall not exceed 2.0 mg/l, each as thirty (30) day average values to be determined from four (4) weekly samples.

7.2.d.16.C Except in the stretch of Cow Creek (O-55) from its mouth to a point approximately 2,300 feet upstream, the Water Use Category A shall not apply.

7.2.d.17 Little Kanawha River - (Reserved)

7.2.d.18 Hughes River - (Reserved)

7.2.d.19 Kanawha River Zone 1 - Main Stem

7.2.d.19.A For the Kanawha River main stem, Zone 1, Water Use Category A shall not apply; and

7.2.d.19.B The minimum flow shall be 1,960 cfs at the Charleston gauge.

7.2.d.20 Kanawha River Zone 2 and Tributaries

7.2.d.20.A For the main stem of the Kanawha River only, the minimum flow shall be 1,896 cfs at mile point 72.

7.2.d.20.B Except the stretch between the mouth of Little Scary Creek (K-31) and the Little Scary impoundment shall not have Water Use Category A or B1 and shall have Water Use Category B3; therefore may contain arsenic not to exceed 200 ug/l and selenium not to exceed 62 ug/l; and copper not to exceed 105 ug/l as a daily maximum nor 49 ug/l as a 4-day average..

7.2.d.20.C Except for Ward Hollow (K-39-A), a small tributary of Davis Creek which may contain chlorides not to exceed 540 mg/l.

7.2.d.20.D Except for Simmons Creek (K-54) from its mouth to a point 1200 feet upstream which shall have a maximum daily temperature not to exceed 38°C (100°F) nor a monthly average temperature to exceed 34°C.

7.2.d.21 Pocatalico River - (Reserved)

7.2.d.22 Coal River - (Reserved)

7.2.d.23 Elk River - (Reserved)

7.2.d.24 Gauley River - (Reserved)

7.2.d.25 Meadow River - (Reserved)

7.2.d.26 Cherry River - (Reserved)

7.2.d.27 Cranberry River - (Reserved)

7.2.d.28 Williams River - (Reserved)

7.2.d.29 New River

7.2.d.29.A Except the stretch of Laurel Creek (KN-5), a tributary of the New River, from the confluence of Dempsey Branch and Laurel Creek to a point 1.7 miles below, where the specific criterion for iron shall be 2.0 mg/l total iron, and from that point to the confluence of Laurel Creek and the New River, the specific criterion for iron shall be 1.0 mg/l total iron.

7.2.d.30 Greenbrier River

7.2.d.30.A Water Use Category A and B2 shall not apply to that segment of the East Fork of the Greenbrier River (KNG-78)

from the reservoir located at the tannery to the confluence with the West Fork; provided that all trout water (B2) standards shall not be violated in the mainstem Greenbrier River.

7.2.d.31 Bluestone River - (Reserved)

7.2.d.32 Bluestone Lake

7.2.d.32.A Category E Water Uses are deleted in Bluestone Lake and temperature rise shall be limited to no more than 3°F above natural not to exceed 81°F at any time during the months of May through November and not to exceed 73°F at any time during December through April.

7.2.d.33 East River - (Reserved)

7.2.d.34 Guyandotte River - (Reserved)

7.2.d.35 Mud River - (Reserved)

7.2.d.36 Big Sandy River - (Reserved)

7.2.d.37 Tug Fork River - (Reserved)

7.3 Special Waters of the State

7.3.a High Quality Waters. High quality waters shall include but are not limited to all waters as defined in Section 2.4.

7.3.b All streams designated by the West Virginia Legislature under the West Virginia Natural Streams Preservation Act, Section One, Article Five B, Chapter 20, et seq. of the West Virginia Code.

7.3.c West Virginia High Quality Streams Fifth Edition, prepared by the Wildlife Resources Division, Department of Natural Resources (1986).

7.3.d National Resource Waters. National Resource Waters shall include but are not limited to the following waters of the State:

7.3.d.1 All Federally designated rivers under the "Wild and Scenic Rivers Act", Public Law 95-542, as amended, 16 u.S.C. 1271, et seq.

7.3.d.2 All naturally reproducing trout streams.

7.3.d.3 All streams and other bodies of water in State and National Forests and Recreation Areas.

7.3.e National Rivers. "National Parks and Recreation Act of 1978." Public Law 95-625, as amended, 16 U.S.C. 1, et seq.

46-1-8. Specific Water Quality Criteria

8.1 Charts of specific water quality criteria are included in Appendix E.

8.1.a. Specific state (i.e. total, total recoverable, valence, etc.) of any parameter to be analyzed shall follow 40 CFR 136, Guidelines Establishing Test Procedures for Analysis of Pollutants Under the Clean Water Act, ~~June 30, 1986~~ as amended, June 15, 1990. (see also Series II, Section 7.3).

8.1.b An "X" or numerical value in the use columns of Appendix E shall represent the applicable criteria.

8.1.c Charts of water quality criteria in Appendix E shall be applied in accordance with major stream and use applications, Sections 6 and 7.

8.2 Criteria for Toxicants

8.2.a Toxicants which are carcinogenic have human health criteria (Water Use Category A) based upon an estimated risk level of one additional cancer death per one million persons (10^{-6}) and are indicated in Appendix E by an asterisk (*).

8.2.b For Water Use Category B, the criteria for organic carcinogens are for the protection against accumulation of those carcinogens in fish flesh in excess of the amount that would produce a cancer risk level of one in one million (10^{-6}) humans.

8.2.c. If a permit applicant requests and provides the necessary informationto the chief, critical stream flow for carcinogens shall be the harmonic mean flow (as defined and calculated in Technical Support Document for Water Quality-based Toxics Control, EPA/505/2-90-001, March 1991). Harmonic mean flows are appropriate when criteria are derived from cancer risk assessment based on lifetime exposure to contaminated fish flesh and drinking water (Use Categories B & A). In-stream compliance with carcinogen standards may be assessed by computing the arithmetic mean of all samples analyzed for the carcinogen on an annual basis.

46-1-9. Establishment of Safe Concentration Values

When a specific water quality standard has not been established by these rules and there is a discharge or proposed discharge into waters of the State, the use of which has been designated as Category B1 and B2, such discharge may be regulated by the chief where necessary to protect State water through establishment of a safe concentration value as follows:

9.1 Establishment of a safe concentration value shall be based upon data obtained from relevant aquatic field studies, standard bioassay test data which exists in substantial available scientific literature, or data obtained from specific tests utilizing one or more representative important species of aquatic life designated on a case-by-case basis by the chief and conducted in a water environment which is equal to or closely approximates that of the natural quality of the receiving waters.

9.2 In those cases where it has been determined that there is insufficient available data to establish a safe concentration value for a pollutant, the safe concentration value shall be determined by applying the appropriate application factor as set forth below to the 96-hour LC 50 value. Except where the chief determines, based upon substantial available scientific data that an alternate application factor exists for a pollutant, the following appropriate application factors shall be used in the determination of safe concentration values:

9.2.a Concentrations of pollutants or combinations of pollutants that are not persistent or cumulative shall not exceed 0.10 (1/10) of the 96-hour LC 50.

9.2.b Concentrations of pollutants or combinations of pollutants that are persistent or cumulative shall not exceed 0.01 (1/100) of the 96-hour LC 50.

9.3 Persons seeking issuance of a permit pursuant to these rules authorizing the discharge of a pollutant for which a safe concentration value is to be established using special bioassay tests pursuant to subsection 9.1 of this section shall perform such testing as approved by the chief and shall submit all of the following in writing to the chief:

9.3.a A plan proposing the bioassay testing to be performed.

9.2.b Such periodic progress reports of the testing as may be required by the chief.

9.2.c A report of the completed results of such testing including, but not limited to all data obtained during the course of testing, and all calculations made in the recording, collection, interpretation, and evaluation of such data.

9.4 Bioassay testing shall be conducted in accordance with the methodologies outlined in the following documents: Environmental Monitoring Series Publication, Methods for Measuring the Acute Toxicity of Effluents to Freshwater and Marine Organisms (600/4-85/013) 3rd Edition, March 1985) or Short Term Methods for Estimating Chronic Toxicity of Effluents and Receiving Waters to Freshwater Organisms (EPA/600/4-89/001), March 1989; Standard Methods for the Examination of Water and Wastewater (17th Edition); or ASTM Practice E 729-88 for Conducting Acute Toxicity Tests with Fishes, Macroinvertebrates and Amphibians as published in Volume 11.04 of the 1988 Annual Book of ASTM Standards. Test waters shall be reconstituted according to recommendations and methodologies specified in the previously cited references or methodologies approved in writing by the chief.

APPENDIX A
CATEGORY B-2 - TROUT WATERS

This list contains known trout waters and is not intended to exclude any waters which meet the definition in Section 2.14.

River Basin	County	Stream
James River		
J	Monroe	South Fork Potts Creek
Potomac River		
P	Jefferson	Town Run
P	"	Rocky Marsh Run
P	Berkeley	Opequon Creek
P	"	Tuscarora Creek (Above Martinsburg)
P	"	Middle Creek (Above Route 30 Bridge)
P	"	Mill Creek
P	"	Hartland Run
P	"	Mill Run
P	"	Tillance Creek
P	Morgan	Meadow Branch
PS	Jefferson	Flowing Springs Run (Above Halltown)
PS	Jefferson	Cattail Run
PS	"	Evitt's Run
PS		Big Bullskin Run
PS	"	Long Marsh Run
PC	Hampshire	Cold Stream
PC	"	Edwards Run and Impoundment
PC	"	Dillons Run
PC	Hardy	Lost River
PC	"	Camp Branch
PC	"	Lower Cove Run
PC	"	Moores Run
PC	"	North River (Above Rio)
PC	"	Waites Run
PC	"	Trout Run
PC	"	Trout Pond (Impoundment)
PC	"	Warden Lake (Impoundment)
PC	"	Rock Cliff Lake (Impoundment)
PSB	Hampshire	Mill Creek
PSB	"	Mill Run

PSB	Hardy	Dumpling Creek
PSB	Grant-Pendleton	North Fork South Branch
PSB	Grant	North Fork Lunice Creek
PSB	"	South Fork Lunice Creek
PSB	"	South Mill Creek (Above Hiser)
PSB	"	Spring Run
PSB	Pendleton	Hawes Run (Impoundment)
PSB	"	Little Fork
PSB	"	South Branch (Above North Fork)
PSB	"	Seneca Creek
PSB	"	Laurel Fork
PSB	"	Big Run
PNB	Mineral	North Fork Patterson Creek
PNB	"	Fork Ashby (Impoundment)
PNB	"	New Creek
PNB	"	New Creek Dam 14 (Impoundment)
PNB	"	Mill Creek (Above Markwood)

Monongahela River

M	Monongalia-Marion	Whiteday Creek (Above Smithtown)
MC	Monongalia	Morgan Run
MC	"	Coopers Rock (Impoundment)
MC	"	Blaney Hollow
MC	Preston	Laurel Run
MC	"	Elsey Run
MC	"	Saltlick Creek
MC	"	Buffalo Creek
MC	"	Wolf Creek
MC	Tucker	Clover Run
MC	"	Elklick Run
MC	"	Horseshoe Run
MC	"	Maxwell Run
MC	"	Red Creek
MC	"	Slip Hill Mill Branch
MC	"	Thomas Park (Impoundment)
MC	"	Blackwater River (Above Davis)
MC	Randolph	Camp Five Run
MC	"	Dry Fork (Above Otter Creek)
MC	"	Glady Fork
MC	"	Laurel Fork
MC	"	Gandy Creek (Above Whitmer)

	MC	"	East Fork Glady Fork (Above C & P Compressor Station)
	MC	Randolph	Shavers Fork (Above Little Black Fork)
	MC	"	Three Spring Run
	MC	"	Spruce Knob Lake (Impoundment)
	MW	Harrison	Dog Run (Pond)
	MW	Lewis	Stonecoal
	MT	Barbour	Brushy Fork (Above Valley Furnace)
	MT	"	Teter Creek Lake (Impoundment)
	MT	"	Mill Run
	MT	Taylor-Barbour	Tygart Lake Tailwaters (Above Route 119 Bridge)
	MT	Preston	Roaring Creek (Above Little Lick Branch)
	MT	Randolph	Tygart River (Above Huttonsville)
	MT	"	Elkwater Fork
	MT	"	Big Run
	MTB	Upshur-Randolph	Right Fork Buckhannon River
	MTB	Upshur	Buckhannon River (Above Beans Mill)
	MTB	Upshur	French Creek
	MTB	Upshur-Randolph	Left Fork Right Fork
	MTM	Upshur	Right Fork Middle Fork River
	MTM	Randolph	Middle Fork River (Above Cassity)
	MY	Preston	Rhine Creek
Little Kanawha River			
	LK	Upshur	Left Fork-Right Fork Little Kanawha River
	LK	Upshur-Lewis	Little Kanawha River (Above Wildcat)

Kanawha River

KE	Braxton	Sutton Reservoir
KE	"	Sutton Lake Tailwaters (Above Route 38/5 Bridge)
KE	Webster	Back Fork
KE	"	Desert Fork
KE	"	Fall Run
KE	"	Laurel Fork
KE	Pocahontas	Laurel Run
KE	Webster	Left Fork Holly River
KE	"	Sugar Creek
KE	"	Elk River (Above Webster Springs)
KC	Raleigh	Stephens Lake (Impoundment)
KC	"	Marsh Fork (Above Sundial)
KG	Nicholas	Summersville Reservoir (Impoundment)
KG	"	Summersville Tailwaters (Above Collison Creek)
KG	Nicholas	Deer Creek
KG	Randolph-Webster	Gauley River (Above Moust Coal Tipple)
KG	Fayette	Glade Creek
KG	Nicholas	Hominy Creek
KG	"	Anglins Creek
KG	Greenbrier	Big Clear Creek
KG	"	Little Clear Creek and Laurel Run
KG	Greenbrier	Meadow Creek
KG	Fayette	Wolf Creek
KG	Nicholas	Cherry River
KG	Greenbrier-Nicholas	Laurel Creek
KG	Greenbrier-Nicholas	North Fork Cherry River
KG	Greenbrier	Summit Lake (Impoundment)
KG	Greenbrier-Nicholas	South Fork Cherry River
KGC	Pocahontas-Webster-Nicholas	Cranberry River
KGC	Pocahontas	South Fork Cranberry River
KGW	Pocahontas	Tea Creek

KGW	Pocahontas-Webster	Williams River (Above Dyer)
KN	Raleigh	Glade Creek
KN	Summers	Meadow Creek
KN	Fayette	Mill Creek
KN	"	Laurel Creek (Above Cotton Hill)
KN	Raleigh	Pinch Creek
KN	Monroe	Rich Creek
KN	"	Turkey Creek
KN	Fayette	Dunloup Creek (Downstream from Harvey Sewage Treatment Plant)
KN	Mercer	East River (Above Kelleysville)
KN	"	Pigeon Creek
KN	Monroe	Laurel Creek
KNG	Monroe	Kitchen Creek (Above Gap Mills)
KNG	Greenbrier	Culverson Creek
KNG	"	Milligan Creek
KNG	Greenbrier-Monroe	Second Creek (Rt. 219 Bridge to Nickell's Mill)
KNG	Greenbrier	North Fork Anthony Creek
KNG	"	Spring Creek
KNG	"	Anthony Creek (Above Big Draft)
KNG	Pocahontas	Watoga Lake
KNG	"	Beaver Creek
KNG	"	Knapp's Creek
KNG	"	Hills Creek
KNG	"	North Fork Deer Creek (Above Route 28/5)
KNG	"	Deer Creek
KNG	"	Sitlington Creek
KNG	"	Stoney Creek
KNG	"	Swago Creek
KNG	"	Buffalo Fork (Impoundment)
KNG	"	Seneca (Impoundment)
KNG	"	Greenbrier River (Above Hosterman)
KNG	"	West Fork-Greenbrier River above the impoundment at the tannery
KNG	"	Little River-East Fork
KNG	"	Little River-West Fork
KNG	"	East Fork, Greenbrier River

KNG	"	Five Mile Run
KNG	"	Mullenax Run
KNG	"	Abes Run
KNB	Mercer	Marsh Fork
KNB	"	Camp Creek
OG	Wyoming	Pinnacle Creek
BST	McDowell	Dry Fork (Above Canebrake)

APPENDIX B
CATEGORY A - WATER SUPPLY PUBLIC

This list contains known waters used as public water supplies and is not intended to exclude any waters as described in Section 6.2.

River Basin	County	Operating Company	Source
Shenandoah River			
S	Jefferson	Charlestown Water	Shenandoah River
Potomac River			
P	Jefferson	3-M Company	Turkey Run
P	"	Shepardstown Water	Potomac River
P	Jefferson	Harpers Ferry Water	Elk Run
P	Berkeley	DuPont Potomac River Works	Potomac River
P	Berkeley	Berkeley County PSD.	Le Feure Spring
P	"	Opequon PSD	Quarry,Spring
P	"	Hedgesville PSD	Speck Spring
P	Morgan	Paw Paw Water	Potomac River
PSB	Hampshire	Romney Water	South Branch Potomac River
PSB	Hampshire	Peterkin Conference Center	Mill Run
PSB	Hardy	Moorefield Municipal Water	South Fork River
PSB	Pendleton	U.S. Naval Radio Sta.	South Fork River
PSB	"	Circilville Water Inc.	North Fork of South Branch, Potomac River
PSB	Grant	Mountain Top PSD	Mill Creek, Imp.
PSB	"	Petersburg Municipal Water	South Branch, Potomac River
PNB	Grant	Island Creek Coal	Impoundment
PNB	Mineral	Piedmont Municipal Water	Savage River, Maryland
PNB	Mineral	Keyser Water	New Creek
PNB	"	Fort Ashby PSD	Lake
Monongahela River			
M	Monongalia	Morgantown Water Comm.	Colburn Creek & Monongahela R.

M	Monongalia	Morgantown Ordinance Works	Monongahela R.
M	Preston	Preston County PSD	Deckers Creek
M	Monongalia	Blacksville # 1 Mine	Impoundment
M	"	Loveridge Mine	Impoundment
M	"	Consolidation Coal Co.	Impoundment
M	Preston	Mason Town Water	Block Run
MC	Preston	Fibair Inc.	Impoundment
MC	Monongalia	Cheat Neck PSD	Cheat Lake
MC	"	Lakeview Country Club	Cheat Lake-Lake Lynn
MC	Monongalia	Union District PSD	Cheat Lake-Lake Lynn
MC	Monongalia	Cooper's Rock State Park	Impoundment
MC	Preston	Kingwood Water	Cheat River
MC	"	Hopemount State Hospital	Snowy Creek
MC	Preston	Rowlesburg Water	Keyser Run & Cheat River
MC	Preston	Albright	Cheat River
MC	Tucker	Parsons Water	Shavers & Elk Lick Fork
MC	Tucker	Thomas Municipal	Thomas Reservoir
MC	"	Hamrick PSD	Dry Fork
MC	"	Douglas Water System	Long Run
MC	"	Davis Water	Blackwater River
MC	"	Hambleton Water System	Roaring Creek
MC	Tucker	Canaan Valley State Park	Blackwater River
MC	Pocahontas	Cheat Mt. Sewer	Shavers Lake
MC	"	Snowshoe Co. Water	Shavers Fork
MC	Randolph	Womelsdorf Water	Yokum Run
MW	Harrison	Lumberport Water	Jones Run
MW	"	Clarksburg Water Bd.	West Fork River
MW	"	Bridgeport Mun. Water	Deecons & Hinkle Creek
MW	Harrison	Salem Water Bd.	Dog Run
MW	"	West Milford Water	West Fork River
MW	Lewis	W.V.Water-Weston District	West Fork River
MW	"	Jackson's Mill Camp	Impoundment
MW	"	West Fork River PSD	West Fork River
MW	"	Kenndy Comprssor	West Fork River

	MW	Lewis	Jane Lew Water Comm.	Hackers Creek
	MW	Harrison	Bel-Meadow Country Club	Lake
	MW	Harrison	Harrison Power Station	West Fork River
	MW	Harrison	Oakdale Portal	Impoundment
	MW	"	Robinson Port	Impoundment
	MT	Marion	Fairmont Water Comm.	Tygart River
	MT	"	Mannington Water	Impoundment
	MT	"	Monongah Water Works	Tygart River
	MT	"	Eastern Assoc. Coal Corp.	Impoundment
	MT	Marion	Four States Water	Impoundment
	MT	Harrison	Shinnston Water Dept.	Tygart River
	MT	Taylor	Grafton Water	Tygart River-Lake
	MT	Barbour	Phillippi Water	Tygart River
	MT	"	Bethlehem Mines Corp.	Impoundment
	MT	Barbour	Belington Water Works	Tygart River & Mill Run Lake
	MT	Randolph	Elkins Municipal Water	Tygart River
	MT	"	Beverly Water	Tygart River
	MT	"	Valley Water	Tygart River
	MT	"	Huttonsville Medium Security Prison	Tygart River
	MT	Randolph	Mill Creek Water	Mill Creek
	MTB	Upshur	Buckhannon Water Board	Buckhannon River
Ohio River				
O	Zone 1	Hancock	Chester Water & Sewer	Ohio River
O	Zone 1	Brooke	City of Weirton	Ohio River
O	" "	Brooke	Weirton Steel Division	Ohio River
O	" "	Ohio	Wheeling Water	Ohio River
O	" "	Tyler	Sistersville Municipal Water	Ohio River
O	" "	Pleasants	Pleasants Power Station	Ohio River
O	" "	Cabell	Huntington Water Corp.	Ohio River
O	" "	Marshall	Mobay Chemical Co.	Ohio River
O	" "	Wood	E. I. DuPont	Ohio River
O	Zone 2	Marshall	Cameron Water	Glass House Hollow

O	"	"	Marshall	New Urindahana Water System / Wheeling Creek
O	"	"	Wetzel	Pine Grove Water / North Fork, Fishing Creek
O	"	"	Marshall	Consolidated Coal Co. / Impoundment
O	"	"	Tyler	Middlebourne Water / Middle Island Creek
O	"	"	Doddridge	West Union Municipal Water / Middle island Creek
O	"	"	Mason	Hidden Valley Country / Lake/Impoundment
O	"	"	Jackson	Ripley Water / Mill Creek
O	"	"	Wayne	Wayne Municipal Water / Twelve Pole Creek
O	Zone 2		Wayne	East Lynn Lake / East Lynn Lake
O	"	"	"	Monterey Coal Co. / Impoundment

Little Kanawha

LK	Wood	Claywood Park PSD	Little Kanawha River
LK	Calhoun	Grantsville Municipal Water	Little Kanawha River
LK	Gilmer	Glenville Utility	Little Kanawha River
LK	Gilmer	Consolidated Gas Compressor	Steer Creek
LK	Braxton	Burnsville Water Works	Little Kanawha River
LK	Roane	Spencer Water	Spring Creek & Mile Tree Reservoir
LK	Wirt	Elizabeth Water	Little Kanawha River
LKH	Ritchie	Cairo Water	North Fork Hughes River
LKH	Ritchie	Harrisville Water	North Fork Hughes River
LKH	Ritchie	Pennsboro Water	North Fork Hughes Fork

Kanawha River

K	Putnam	Buffalo Water	Cross Creek
K	"	Winfield Water	Poplar Fork & Crooked Creek

K	Putnam	South Putnam PSD	Poplar Fork & Ceooked Creek
K	Kanawha	Cedar Grove Water	Kanawha River
K	"	Pratt Water	Kanawha River
K	Fayette	Armstrong PSD PO-Kl-CO-EL	Kanawha River & Gum Hollow
K	Fayette	Kanawha Water Co.-Beards Fork	Unnamed Tributary Kanawha River
K	Kanawha	Midland Trail School	Impoundment
K	"	Cedar Coal Co.	Impoundment
K	Fayette	Elkem Metals Co.	Kanawha River
K	"	Deepwater PSD	Kanawha River
K	"	Kanawha Falls PSD	Kanawha River
K	"	W.V. Water-Montgomery	Kanawha River

Pocatalico River

KP	Kanawha	Sissonville PSD	Pocatalico River
KP	Roane	Walton PSD	Silcott Fork Dam

Coal River

KC	Kanawha	St. Albans Water	Coal River
KC	"	Washington PSD	Coal River
KC	Lincoln	Lincoln PSD	Coal River
KC	Boone	Coal River PSD	Coal River
KC	"	Whitesville PSD	Coal River
KC	Raleigh	Armco Mine 10	Marsh Fork
KC	"	Armco Steel-Montc. Stickney	Coal River
KC	Raleigh	Peabody Coal	Coal River
KC	"	Stephens Lake Park	Lake Stephens
KC	Boone	W.V.Water-Madison Dist.	Little Coal River
KC	Boone	Van PSD	Pond Fork
KC	Raleigh	Consol. Coal Co.	Workmans Creek
KC	Boone	Water Ways Park	Coal River

Elk River

KE	Kanawha	Clendenin Water	Elk River
KE	"	W.V. Water-Kanawha Valley Dist.	Elk River
KE	Kanawha	Pinch PSD	Elk River
KE	Clay	Clay Waterworks	Elk River
KE	"	Procious PSD	Elk River
KE	Braxton	Flatwoods-Canoe Run PSD	Elk River

KE	Braxton	Sugar Creek PSD	Elk River
KE	"	W.V.Water-Gassaway Dist.	Elk River
KE	Braxton	W.V.Water-Sutton District	Elk River
KE	Webster	W.V.Water-Webster Springs	Elk River
KE	Webster	Holly River State Park	Holly River

Gauley River

KG	Nicholas	Craigsville PSD	Gauley River
KG	"	Summersville Water	Impoundment/ Muddlety Creek
KG	Nicholas	Nettie-Leivasy PSD	Jim Branch
KG	Webster	Cowen PSD	Gauley River
KG	Nicholas	Wilderness PSD	Anglins Creek & Meadow River
KG	"	Richwood Water	North Fork Cherry River

New River

KN	Fayette	Ames Heights Water	Mill Creek
KN	"	Mt. Hope Water	Impounded Mine (Surface)
KN	Fayette	Ansted Municipal Water	Mill Creek
KN	"	Fayette Co. Park	Impoundments
KN	"	New River Gorge Campground	Impoundment
KN	Fayette	Fayetteville Water	Wolfe Creek
KN	Raleigh	Beckley Water	Glade Creek
KN	"	Westmoreland Coal Co.	Farley Branch

Bluestone River

KNB	Summers	Jumping Branch-Nimitz	Mt. Valley Lake
KNB	"	Bluestone Conf. Center	Bluestone Lake
KNB	"	Pipestem State Park	Impoundment
KNB	Mercer	Town of Athens	Impoundment
KNB	"	Bluewell PSD	Impoundment
KNB	"	Bramwell Water	Impoundment
KNB	"	Green Valley-Glenwood PSD	Bailey Reservoir
KNB	Mercer	Kelly's Tank	Spring
KNB	"	W.V.Water Princeton	Impoundment-Brush Creek

KNB	Mercer	Lashmeet PSD	Impoundment
KNB	"	Pinnacle Water Assoc.	Mine
KNB	"	W.V.Water Bluefield	Impoundment

Greenbrier River

KNG	Summers	W.V.Water Hinton	Greenbrier River & New River
KNG	"	Big Bend PSD	Greenbrier River
KNG	Greenbrier	Alderson Water Dept.	Greenbrier River
KNG	"	Ronceverte Water	Greenbrier River
KNG	"	Lewisburg Water	Greenbrier River
KNG	Pocahontas	Denmar State Hospital Water	Greenbrier River
KNG	Pocahontas	City of Marlinton Water	Knapp Creek
KNG	Pocahontas	Cass Scenic Railroad	Leatherbark Creek
KNG	Pocahontas	Upper Greenbrier PSD	Greenbrier River
KNG	"	The Hermitage	Greenbrier River

Guyandotte River

OG	Cabell	Salt Rock PSD	Guyandotte River
OG	Lincoln	West Hamlin Water	Guyandotte River
OG	Logan	Logan Water Board	Guyandotte River
OG	"	Man Water Works	Guyandotte River
OG	"	Buffalo Creek PSD	Buffalo Creek/ Mine/Wells
OG	Logan	Chapmanville	Guyandotte River
OG	Logan	Logan PSD	Whitman Creek/ Guyandotte River
OG	Mingo	Gilbert Water	Guyandotte River
OG	Wyoming	Oceana Water	Laurel Fork
OG	"	Glen Rogers PSD	Impoundment
OG	"	Pineville Water	Pinnacle Creek/ Guyandotte River
OG	Wyoming	Mullens Water Works	Slab Fork Creek
OG	Raleigh	Raleigh Co. PSD-Amigo	Tommy Creek
OGM	Cabell	Milton Water Works	Guyandotte River
OGM	"	Culloden PSD	Indian Fork Creek
OGM	Putnam	Hurricane Municipal Water	Impoundment
OGM	Putnam	Lake Washington PSD	Lake Washington

Big Sandy River

BS	Wayne	Kenova Municipal Water	Big Sandy River
BS	"	Fort Gay Water	Tug Fork
BST	Mingo	Kermit Water	Tug Fork
BST	"	Matewan Water	Tug Fork
BST	"	A & H Coal Co., Inc.	Impoundment
BST	"	Williamson Water	Impoundment
BST	McDowell	City of Welch	Impoundment/ Wells
BST	McDowell	City of Gary	Impoundment/ Mine

APPENDIX C

CATEGORY E-3 - POWER PRODUCTION

This list contains known power production facilities and is not intended to exclude any waters as described in Section 6.6.c.

River Basin	County	Station Name	Operating Company
Monongahela River			
M	Monongalia	Fort Martin Sta.	Monongahela Power
M	Marion	Rivesville Sta.	Monongahela Power
MC	Preston	Albright Sta.	Monongahela Power
MW	Harrison	Harrison Sta.	Monongahela Power
Potomac	Grant	Mt. Storm Power Station	Virginia Electric & Power Company
Ohio River			
O Zone 1	Wetzel	Hannibal (Hydro)	
O Zone 1	Marshall	Kamer	Ohio Power
O " "	"	Mitchell	" "
O " "	Pleasants	Pleasants Sta.	Monongahela Power
O " "	Pleasants	Willow Island Station	Monongahela Power
O " "	Mason	Phillip Sporn Plant	Central Operating (AEP)
O " "	Mason	Racine (Hydro)	Ohio Power
O " "	Mason	Mountaineer	Appalachian Power Company
K	Putnam	Winfield (Hydro)	Appalachian Power Company
K	Kanawha	Marmet (Hydro)	Appalachian Power Company
K	Kanahwa	London (Hydro)	Appalachian Power Company
K	Kanawha	Kanawha River	Appalachian Power Company
K	Putnam	John E. Amos	Appalachian Power Company

APPENDIX D
CATEGORY C - WATER CONTACT RECREATION

This list contains waters known to be used for water contact recreation and is not intended to exclude any waters as described in Section 6.4.

River Basin	Stream Code	Stream	County
Shenandoah	S	Shenandoah River	Jefferson
Potomac	P	Potomac River	Jefferson
	P	" "	Hampshire
	P	" "	Berkeley
	P	" "	Morgan
	P-9	Sleepy Creek & Meadow Branch	Berkeley
	P-9-G-1	Noeth Fork of Indian Run	Morgan
South Branch	PSB	South Branch of Potomac River	Hampshire
	PSB	" "	Hardy
	PSB	" "	Grant
	PSB-21-X	Hawes Run	Pendleton
	PSB-25-C-2	Spring Run	Grant
	PSB-28	North Fork South Branch Potomac River	Grant
North Branch	PNB	North Branch of Potomac River	Mineral
	PNB-4-EE	North Fork Patterson Creek	Grant
	PNB-7-H	Linton Creek	Grant
	PNB-17	Stoney River-Mt. Storm Lake	Grant
	PC	Cacapon River	Hampshire
Monongahela			
Cheat	MC	Cheat Lake/Cheat River	Monongalia/ Preston
	MC	Alpine Lake	Preston
	MC-6	Coopers Rock Lake/ Quarry Run	Monongalia
	MC-12	Big Sandy Creek	Preston

	MSC	Shavers Fork	Randolph
	MTN	Middle Fork River	Barbour/ Randolph/ Upshur
	MW	West Fork River	Harrison
	MW-38	Stonecoal Creek/ Stonecoal Lake	Lewis
Ohio	O	Ohio River	Brooke/Cabell/ Hancock/Jackson/ Marshall/Mason/ Ohio/Pleasants/ Tyler/Wayne/Wood/ Wetzel
	O-2-H	Beech Fork of Twelvepole Creek/ Beech Fork Lake	Wayne
	O-2-Q	East Fork of Twelvepole Creek/ East Lynn Lake	Wayne
	O-3	Fourpole Creek	Cabell
	O-21	Old Twon Creek/ McClintic Ponds	Mason
	OMi	Middle Island Creek/ Crystal Lake	Doddridge
	OG	Guyandotte River	Cabell
	OG	Guyandotte River/ R D Bailey Lake	Wyoming
	OGM	Mud River	Cabell
Little Kanawha	LK	Little Kanawha River/Burnsville Lake	Braxton
Kanawha	K	Kanawha River	Fayette/Kanawha/ Mason/Putnam
	K-1	Unnamed Tributary Krodel Lake	Mason
	KC	Coal River	Kanawha
	KC-46-Q	Stephens Branch/ Lake Stephens	Raleigh
	KE	Elk River	Kanawha/Clay/ Braxton/Webster/ Randolph
	KE	Sutton Lake	Braxton
	KN	New River	Fayette/Raleigh/

KN-26-F	Little Beaver Creek	Summers Raleigh
KNG	Greenbrier River	Greenbrier/ Pocahontas/ Summers
KNG-23-E-1	Little Devil Creek/ Moncove Lake	Monroe
KNG-28	Anthony Creek	Greenbrier
KNG-28-P	Meadow Creek/Lake Sherwood	Greenbrier
KNB	Bluestone River/ Bluestone Lake	Summers
KNB	North Fork Brush Creek	Mercer
KG	Gauley River	Webster
KG	Gauley River/ Summersville Lake	Nicholas
KGW	Williams River	Webster

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.1 ALUMINIM: Not to exceed: ~~0.5 mg/l.~~	ug/l ~~mg/l~~	748 ~~---~~	87 ~~-X-~~	—	—	—
8.2 AMMONIA: The concentration of un-ionized ammonia (NH_3) shall not exceed 50 ug/l. Un-ionized ammonia shall be determined from values of total ammonia-N, pH and temperature according to the following equation: Un-ionized ammonia = $\frac{1.2(\text{total ammonia-N})}{1 + 10^{(pk_a - pH)}}$ where $pk_a = 0.0902 + 2730/(273.2 + T)$ and T = temperature in degrees C.	ug/l	50	—	—	50	—
8.2.1 The concentration of un-ionized ammonia shall not exceed 20 ug/l.	ug/l	—	20	—	—	—
8.3 ANTIMONY: Not to exceed 14 ~~146~~ ug/l.	ug/l	—	—	—	14 ~~146~~	—
8.4 ARSENIC: Not to exceed ~~2.2 ng/l~~ 50 ug/l.	ug/l ~~ng/l~~	—	—	—	50 ~~2.2~~	—
8.4.1 Not to exceed 100 ug/l (Category D Uses).	ug/l	—	—	—	—	100
8.4.2 Not to exceed 190 ug/l trivalent arsenic.	ug/l	X	X	X	—	—
8.5 BARIUM: Not to exceed 1.0 mg/l.	mg/l				1	
8.6 *BERYLLIUM:	ng/l	130 ~~117~~	130 ~~117~~	— —	7.7 ~~6.8~~	

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B 2	Recreation C	Public A	All Other Uses
8.7 CADMIUM: Hardness mg/l as $CaCO_3$ — Soluble cadmium (ug/l) 0-35 — 1 36-75 — 2 76-150 — 5 greater than 150 — 10	ug/l	___	___	___	X	___
8.7.1 Not to exceed 10 ug/l in the Ohio River (O Zone 1) main stem (See Section 7.1.d)	ug/l		___		10	
8.7.2 Not to exceed 0.4 ug/l where hardness is less than 75 mg/l as $CaCO_3$ and 1.2 ug/l in water where hardness is greater than 75 mg/l as $CaCO_3$.	ug/l	___	X	___	___	___
8.7.3 The concentration of cadmium shall not exceed the criterion determined by the equation: Cd (ug/l) = $e^{(0.7852[\ln(\text{hardness})]-3.490)}$ For example: Hardness mg/l as $CaCO_3$ — Total Recoverable Cadmium (ug/l) 50 — 0.7 100 — I.I 200 — 2.0 300 — 2.7 400 — 3.4	ug/l	X	___	X	___	
8.8 CHLORIDE: Not to exceed 250 mg/l.	mg/l	250	250	250	250	___
8.9 COPPER: Not to exceed 1000 ug/l.	ug/l	___	___	___	1000	___

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.9.1 COPPER (Cont'd) Not to exceed the criterion determined by the equation: $Cu\ (ug/l) = e^{(0.8545[\ln(Hardness)]-1.465)}$						
Hardness mg/l as $CaCO_3$ / Total Recoverable Copper ug/l						
50 — 6						
100 — 11						
200 — 20						
300 — 29						
400 — 38						
500 — 46						
600 — 55						
700 — 63	ug/l	X	X	—	—	
8.10 CYANIDE: (As Free cyanide HCN + CN^-): Not to exceed 5 ug/l.	ug/l	5	5	5	5	
8.11 DISSOLVED OXYGEN: Not less than 5.0 mg/l at any time.°	mg/l	X	—	X	X	
8.11.1 Not less than 4.0 mg/l at any time in the Kanawha River main stem, Zone 1.	mg/l	X	—	—	—	—
8.11.2 Concentration shall average 5.0 mg/l per calendar day and shall not be less than 4.0 mg/l at any time or place outside any established mixing zone, provided that a minimum of 5.0 mg/l at any time is maintained during the April 15-June 15 spawning season. Ohio River Main stem.	mg/l	X				
8.11.3 Not less than 7.0 mg/l in spawing areas and in no case less than 6.0 mg/l at any time.	mg/l	—	X	—	—	—

°may not be applicable to wetlands (B4) - site-specific criteria are desirable.

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.12 FECAL COLIFORM: Maximum allowable level of fecal coliform content for Primary Contact Recreation (either MPN or MF) shall not exceed 200/100 ml as a monthly geometric mean based on not less than 5 samples per month; nor exceed 400/100 ml in more than ten percent of all samples taken during the month.	counts	—	—	X	X	—
8.12.1 For the Ohio River main stem (zone 1). For the non-recreational season November through April only, the maximum allowable level of fecal coliform for the Ohio River (either MPN or MF) shall not exceed 2000/100 ml as a monthly geometric mean based on not less than 5 samples per month.	counts	—	—	X	—	—
8.13 FLUORIDE: Not to exceed 1.4 mg/l.	mg/l	—	—	—	1.4	
8.13.1 Not to exceed 2.0 mg/l for D Uses.	mg/l	—	—	—	—	2.0
8.14 HEXAVALENT CHROMIUM: Not to exceed 50 ug/l.	ug/l	—	—	—	50	—
8.14.1 Not to exceed 10 ug/l.	ug/l	10 :	—	—	—	—
8.14.2 Not to exceed 7.2 ug/l.	ug/l	—	7.2	—	—	—

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.15 IRON:						
Not to exceed 1.5 mg/l.°	mg/l	1.5	___	___	1.5	
Effluent limitations which may result in a concentration of up to 3.5 mg/l total iron in the stream are allowable upon a demonstration to the Chief by the applicant that such concentration will not have an adverse impact upon designated stream uses. This demonstration is subject to EPA approval and must show either; (1) that the stream is supporting designated uses while containing total iron concentrations higher than the applicable criteria or (2) the stream does not have an aquatic life use to protect. Notwithstanding Series 1, Section 4 of the board's rules, this demonstration shall be the only demonstration required before the Chief and the Board with respect to water quality related effluent limitations. This exception does not apply to Trout Waters.	mg/l	X	___	___	___	___
°May not apply to certain wetlands (B4) - site-specific criteria are desirable.						
8.15.1 Not to exceed 0.5 mg/l.	mg/l	___	0.5	___	___	___
8.16 LEAD:						
Not to exceed 50 ug/l.	ug/l	___	___	___	50	___
8.16.1 The concentration of lead shall not exceed the criterion determined by the equation: $Pb\ (ug/l) = e^{(1.273[\ln(\text{hardness})]-4.705)}$ For example:						
Hardness mg/l as $CaCO_3$ / Lead ug/l: 50 / 1.3; 100 / 3.2; 200 / 7.7; 300 / 12.9; 400 / 18.6; 500 / 24.7; 600 / 31.1; 700 / 37.9	ug/l	X	X	___	___	___

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.17 MANGANESE: Not to exceed 1.0 mg/l.	mg/l	1.0	1.0		1.0	—
8.17.1 Effluent limitations which may result in a concentration up to 2.0 mg/l manganese in the stream are allowable upon a demonstration to the Chief by the applicant that such concentration will not have an adverse impact upon designated stream uses. This demonstration is subject to EPA approval and must show either; (1) the stream is supporting designated uses while containing manganese concentrations higher than the applicable criteria, or (2) the stream does not have an aquatic life use to protect. Notwithstanding Series 1, Section 4 of the Board's rules, this demonstration shall be the only demonstration required before the Chief and the Board with respect to water quality related effluent limitations. This exception does not apply to Trout Waters.	mg/l	X	—	—	—	—
8.18 MERCURY: ~~(total)~~ The total organism body burden of any aquatic species shall not exceed 0.5 ug/g as ~~total mercury.~~ methylmercury.	ug/g	0.5	0.5	—	—	—
8.18.1 Total mercury concentration in any unfiltered water sample shall not exceed ~~0.14~~ 0.20 ug/l.	ug/l	—	—	X	X	
8.18.2 Not to exceed 0.012 ug/l. ~~methylmercury.~~	ug/l	X	X			
8.19 NICKEL: Not to exceed 510 ug/l.	ug/l				X	
8.19.1 Not to exceed 50 ug/l.	ug/l	—	X	—	—	—

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.19.2 Not to exceed the concentration determined by the following equation: $Ni\ (ug/l) = e^{(0.846\ [\ln(hardness)]+1.1645)}$ For example: Hardness mg/l as $CaCO_3$ / Nickel ug/l: 50 / 88; 100 / 160; 200 / 280; 300 / 399; 400 / 509; 500 / 615; 600 / 718	ug/l	X				
8.20 NITRATE: (as Nitrate -N) Not to exceed 10 mg/l.	mg/l	—	—	—	10	—
8.21 NITRITE: (as Nitrite -N) Not to exceed 1.0 mg/l.	mg/l	1.0	—	—	—	
8.21.1 Not to exceed 60 ug/l.	ug/l	—	60	—	—	—
8.22 ORGANICS:						
*Chlordane (Criteria 0.46; Body Burden ug/l 1.0)	ng/l	X	X	X	X	X
*DDT (0.024; 0.1)	ng/l	X	X	X	X	X
*Aldrin-Dieldrin (0.071; 0.3)	ng/l	X	X	X	X	X
Endrin (.0023; 0.3)	ug/l	X	X	X	X	X
*Toxaphene (0.71; 1.0)	ng/l	X	X	X	X	X
*PCB (0.079; 2.0)	ng/l	X	X	X	X	X
Methoxychlor (.03; —)	ug/l	X	X	X	X	X
*2,3,7,8-TCDD (Dioxin) (6.4 pg/g)	pg/l	1.0	1.0	1.0	0.96	1.0
The body burden criteria shall not be exceeded in edible tissues of fish.						

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
*Acrylonitrile	ug/l	0.77	0.77		.059	
*Benzene	ug/l	40	40	-	0.66	-
1,2-dichlorobenzene	mg/l	17.0	17.0		2.7	
1,3-dichlorobenzene	mg/l	2.6	2.6		0.4	
1,4-dichlorobenzene	mg/l	2.6	2.6		0.4	
*2,4-dinitrotoluene	ug/l	9.1	9.1		0.11	
*Hexachlorobenzene	ng/l	0.74	0.74	-	0.72	-
*Carbon Tetrachloride	ug/l	4.4	4.4	-	0.25	-
*Chloroform	ug/l	15.7	15.7	-	0.19	-
*Other Halomethanes	ug/l	15.7	15.7	-	0.19	-
*1,2-dichloroethane	ug/l	98.6	98.6	-	0.035	-
1,1,1-trichloroethane	mg/l	67.3	67.3	-	1.2	-
*1,1,2,2-tetrachloroethane	ug/l	10.7	10.7	-	0.17	-
*1,1-dichloroethylene	ug/l	1.9	1.9	-	0.03	-
*Trichloroethylene	ug/l	92.4	92.4	-	3.1	-
*Tetrachloroethylene	ug/l	8.9	8.9	-	0.8	-
Toluene	mg/l	424	424	-	14.3	-
*Polynuclear Aromatic Hydrocarbons	ng/l	31.1	31.1	-	2.8	-
Phthalate Esters	ug/l	3.0	3.0	-	-	-
*Vinyl Chloride (chloroethene)	ug/l	525	525		2.0	
The organic chemicals listed above shall not exceed the specified water quality criteria. When the specified criteria are less than the practical laboratory quantification level, instream values will be calculated from discharge concentrations and flow rates, and from fish body burden, where applicable.						
8.23 pH: No values below 6.0 nor above 9.0. Higher values due to photosynthetic activity may be tolerated.° °May not apply to certain wetlands (B-4) - site-specific-criteria are desirable.	unit	X	X	X	X	X
8.24 PHENOLIC MATERIALS: Not to exceed 5 ug/l.	ug/l	5	5	—	5	—
8.25 RADIOACTIVITY: Gross Beta activity not to exceed 1000 picocuries per liter (pCi/l), nor shall activity from dissolved strontium-90 exceed 10 pCi/l, nor shall activity from dissolved alpha emitters exceed 3 pCi/l.	pCi/l	X	X	X	X	X

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B 2	Recreation C	Public A	All Other Uses
8.25.1 Gross total alpha particle activity (including radium-226 but excluding radon and uranium) shall not exceed 15 pCi/l and combined radium-226 and radium-228 shall not exceed 5 pCi/l; provided that the specific determination of radium-226 and radium-228 are not required if dissolved particle activity does not exceed 5 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 in the Ohio River main stem.	pCi/l	X	X	X	X	X
8.26 SELENIUM: Not to exceed 10 ug/l.	ug/l	—	—	—	10	—
8.26.1 Not to exceed 5 ug/l.	ug/l	X	X	—	—	—
8.27 SILVER:						
Hardness / Silver ug/l: 0-50 / 1; 51-100 / 4; 101-200 / 12; greater than 201 / 24	ug/l	—	X	—	X	—
8.27.1 0-50 / 1; 51-100 / 4; 101-200 / 12; 201-400 / 24; 401-500 / 30; 501-600 / 43	ug/l	X	—	—	—	—

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B 2	Recreation C	Public A	All Other Uses
8.28 TEMPERATURE: Temperature rise shall be limited to no more than 5°F above natural temperature, not to exceed 87°F at any time during months of May through November and not to exceed 73°F at any time during the months of December through April. During any month of the year, heat should not be added to a stream in excess of the amount that will raise the temperature of the water more than 5°F above natural temperature. In lakes and reservoirs, the temperature of the elipimnion should not be raised more than 3°F by the addition of heat of artificial origin. The normal dialy and seasonable temperature fluctuations that existed before the addition of heat due to other than natural causes should be maintained.	°F	X				
8.28.1 For the Kanawha River Main Stem (K-1): Temperature rise shall be limited to no more than 5°F above natural temperature, not to exceed 90°F in any case.		X				
8.28.2 For the Bluestone R. (KNB), Bluestone Lake, (KN-60), East River (KNE), New River (KN), Gauley R. (KG) and Greenbrier River (KNG): Temperature rise shall be limited to no more than 5°F above natural temperature, not to exceed 81°F at any time during the months of May through November and not to exceed 73°F at any time during December through April.		X				

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

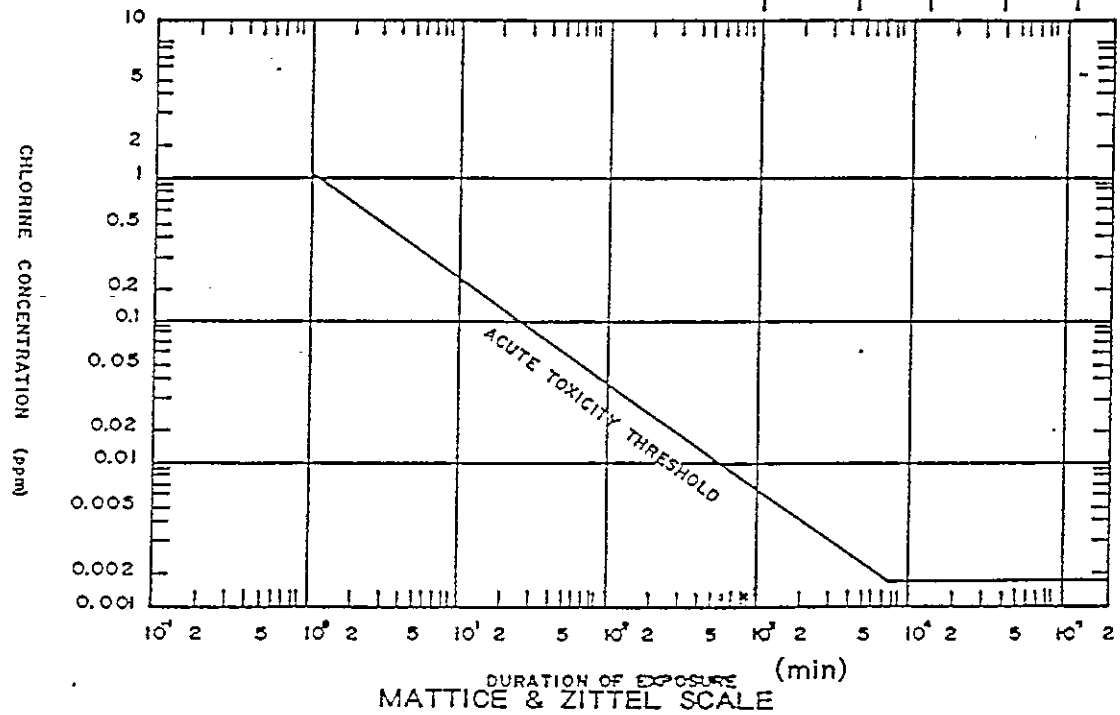
USE DESIGNATIONS

PARAMETER	UNITS	Warmwater B 1,3,4	Troutwater B 2	Recreation C	Public A	All Other Uses
8.28.3 No heated effluents will be discharged in the vicinity of spawing areas. The maximum temperatures for cold waters are expressed in the following table:						
Oct. - April: Daily Mean 50°F; Hourly Maximum 55°F						
Sept. - May: Daily Mean 58°F; Hourly Maximum 62°F						
Transition Period						
June - August: Daily Mean 66°F; Hourly Maximum 70°F			X			
8.28.4 For Ohio River Main Stem (01)(Section 7.1.d)						
January 1-31: Period Average 45°F; Inst. Max. 50°F	°F					
February: 45; 50						
March 1-15: 51; 56						
March 16-31: 54; 59						
April 1-15: 58; 64						
April 16-30: 64; 69						
May 1-15: 68; 73						
May 16-31: 75; 80						
June 1-15: 80; 85						
June 16-30: 83; 87						
July 1-31: 84; 89						
August 1-31: 84; 89						
September 1-15: 84; 87						
September 16-30: 82; 86						
October 1-15: 77; 82						
October 16-31: 72; 77						
November 1-30: 67; 72	°F	X	—	—	—	
December: 52; 57						
8.29 THALLIUM: Not to exceed ~~43~~ 1.7 ug/l.	ug/l	—	—	—	1.7 ~~43~~	—

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.30 THRESHOLD ODOR: Not to exceed a threshold odor number of 8 at 104°F as a daily average. °May not apply to certain wetlands (B4).	t.o.n.	X	X	X	X	
8.31 TOTAL RESIDUAL CHLORINE: Not to exceed 10 ug/ as measured by the amperometric or equivalent method.	ug/l	10	—	10	10	
8.31.1 No chlorinated discharge allowed.			X			
8.31.2 The following chart may be used to derive the criteria instead of the above fixed (10 ug/l) figure:						



MATTICE & ZITTEL SCALE

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B1,3,4	Troutwater B2	Recreation C	Public A	All Other Uses
8.32 TURBIDITY: No point or non-point source to West Virginia's waters shall contribute a net load of suspended matter such that the turbidity exceeds 10 NTU's over background turbidity when the background is 50 NTU's or less, or have more than a 10 percent increase in turbidity (plus 10 NTU minimum) when the background turbidity is more than 50 NTU's. This limitation shall apply to all earth disturbance activities and shall be determined by measuring stream quality directly above and below the area where drainage from such activity enters the affected stream. Any earth disturbance activity continuously or intermittently carried on by the same or associated persons on the same stream or tributary segment shall be allowed a single net loading increase.	NTU's	X	X	X	X	
8.32.1 This rule shall not apply to those activities at which Best Management Practices in accordance with the State's adopted 208 Water Quality Management Plan are being utilized, maintained and completed on a site specific basis as determined by the appropriate 208 cooperative or an approved Federal or State Surface Mining Permit is in effect. This exemption shall not apply to Trout Waters.	NTU's	X	—	X	X	

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. submitts 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. Incidently, 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

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June 25, 1991

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Support Document for the Establishment of a Water Quality Standard for Dioxin in the State of West Virginia

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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. Incidently, 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.

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 cased 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 tumors 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. (1900) 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) intratrachaelly 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 AAH 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

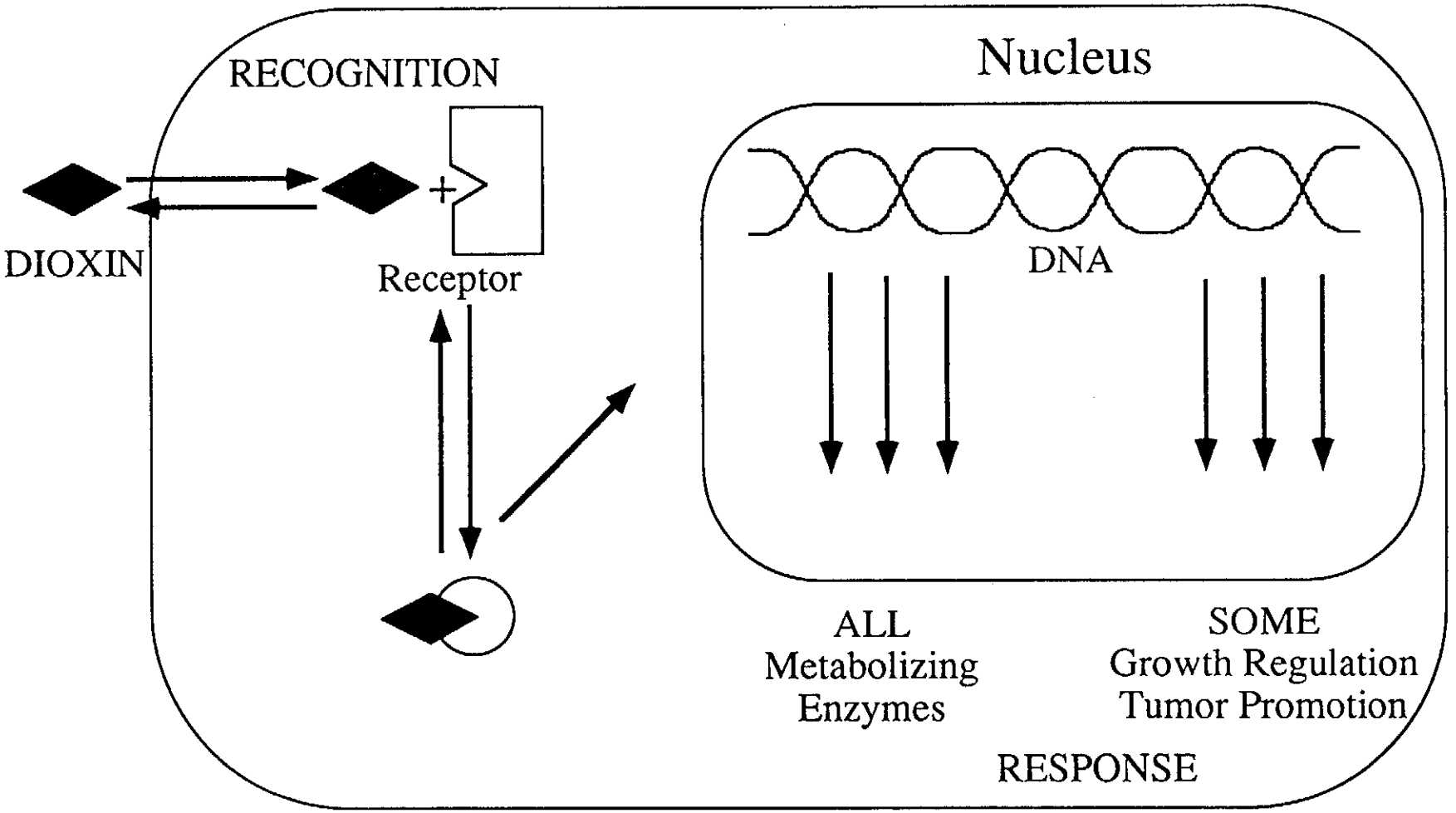
associated with exposure to dioxin, including immunotoxicity, reproductive effects, and cancer, appear to be mediated through the Ah receptor (Birnbaum, 1990; Gallo, 1990; Scheuplein, 1990a; van der Heijden, 1990). According to Gallo (1990), for dioxin to have an effect, it must reach a specific site, 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 censuring 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 $(mg/kg\text{-}day)^{-1}$ 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 $(mg/kg\text{-}day)^{-1}$ (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 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 $(mg/kg\text{-}day)^{-1}$, 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 LD_{50} (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 LD_{50} 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 f_0 litters, and neonatal survival was reduced in the f_1 generation. At 0.01 μg/kg-day, fertility was significantly reduced in f_1 and f_2 generations but not in the f_0 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 f_1 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 μg/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 μg/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 μg/kg dose group. None of the four mothers given 0.2 μg/kg in divided doses showed signs of toxicity and the three surviving offspring in the 0.2 μg/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 μg/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 (Zingeser, 1979). Maternal toxicity, effects or 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 resistent 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; Nessal 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 $(mg/kg\text{-}day)^{-1}$ 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

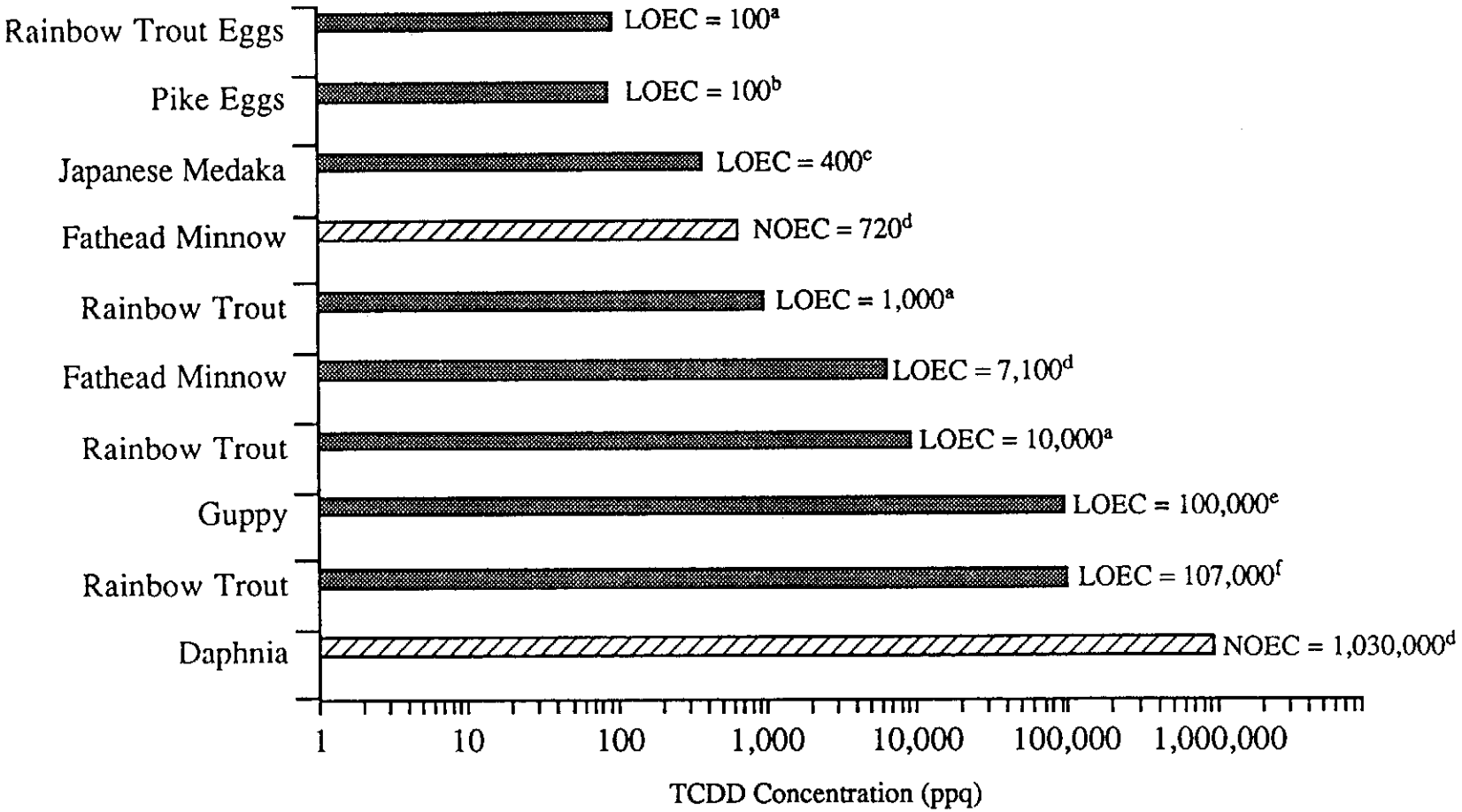
Species	Life Stage Exposed	Exposure Duration (hours)	Test Duration (days)	Carrier Solvent	TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration[a] (ppq)	Effects[a]	Reference
Fathead minnow *Pimephales promelas*	juvenile (1 - 2 g)	24 - 96	150	acetone	0; 120; 720 7,100; 82,000	720	7,100	Mortality[b]	Adams et al., 1986
Guppy *Poecilia reticulatas*	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 *Oryzias latipes*	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 *Oryzias latipes*	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 *Esox lucius*	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 *Salmo gairdneri*	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 [a] (ppq)	Effects [a]	Reference
Rainbow trout *Salmo gairdneri*	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 *Salmo gairdneri*	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 *Salmo gairdneri*	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 *Daphia magna*	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

LOEC
NOEC

Table 3-2. Effects of Chronic Exposure of Fish and Other Aquatic Organisms to TCDD in Ambient Water

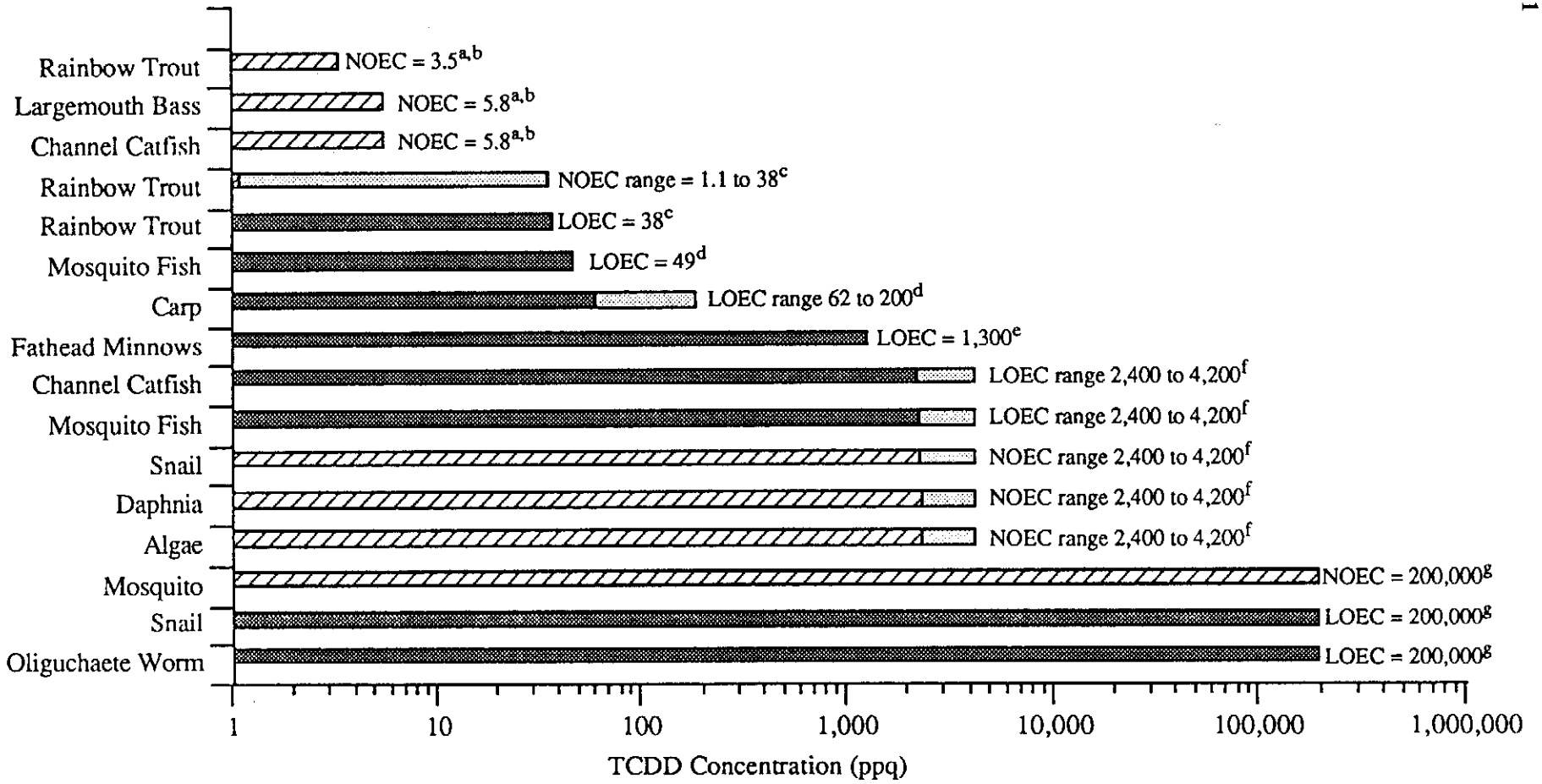
Species	Life Stage Exposed	Exposure Duration (days)	Test Duration (days)	Carrier Solvent	Nominal (Measured) TCDD Concentration in Water (ppq)	No-Observed-Effect-Concentration (ppq)	Lowest-Observed-Effect-Concentration[a] (ppq)	Effects[a]	Reference
Carp *Cyprinus carpio*	1-yr old (15 g)	71	132	dimethyl-formamide	0; 200 (62)	ND	62 - 200	Mortality[b], signs of overt toxicity[b], extensive pathology[b]	Cook et al., 1991
Channel catfish *Ictalurus punctatus*	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 *Pimephales promelas*	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 *Pimephales promelas*	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 *Gambusia affinis*	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 *Salmo gairdneri*	fry (0.38 g)	28	56	acetone	0 (1.1); 115 (38); 231 (79); 463 (176); 925 (382); 1,850 (789)	Between 1.1 and 38, according to authors	38	Reduced growth and survival[c], altered behavioral responses[b]	Mehrle et al., 1988
Algae *Oedogonium cardiacum*	NR	32	32	benzene	(2,400 - 4,200)	2,400 - 4,200	ND	No apparent adverse effects[b]	Yockim et al., 1978
Mosquito *Aedes aegypti*	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 *Paranais sp.*	adult	55	55	acetone	0; 200,000	ND	200,000	Reduced number of worms[c]	Miller et al., 1973
Snail *Helosoma sp.*	NR	32	32	benzene	(2,400 - 4,000)	2,400 - 4,200	ND	No apparent adverse effects[b]	Yockim et al., 1978
Snail *Physa sp.*	adult	36	48	acetone	0; 200,000	ND	200,000	Reduced snail hatch[d]	Miller et al., 1973
Water flea *Daphnia magna*	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

LOEC
NOEC

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	Lowest-Observed-Effect-Concentration or Level [a]	LD50	Effects [a]	Reference
Bluegill *Lepomis macrochirus*	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 *Ictalurus melas*	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 *Cyprinus carpio*	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 [c], fin necrosis [d], cutaneous hemorrhage [d], cutaneous hyper-pigmentation [d]	Kleeman et al., 1988
Coho Salmon *Oncorhynchus kisutch*	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 [c] and reduced growth [c]	Miller et al., 1979
Guppy *Poecilia reticulatus*	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	Lowest-Observed-Effect-Concentration or Level [a]	LD50	Effects[a]	Reference
Lake trout *Salvelinus namaycush*	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 *Micropterus salmoides*	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[e], fin necrosis[d], cutaneous hyperpigmentation[d]	Kleeman et al., 1988
Rainbow trout *Salmo gairdneri*	fingerling (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 *Salmo gairdneri*	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 *Salmo gairdneri*	juvenile (7.8 cm)	105 days	105 days	feeding	0; 2.3; 2,300; 2,300,000 pg/g diet (avg. intake of 0, 3.2×10^{-8}, 3.6×10^{-5}, 2.1×10^{-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	Lowest-Observed-Effect-Concentration or Level [a]	LD50	Effects [a]	Reference
Rainbow trout *Salmo gairdneri*	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 *Salmo gairdneri*	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 *Perca flavescens*	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 *Perca flavescens*	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 hemorrhage [d]	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 *Perca flavescens*	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], hemorrages[d], ascites[d], lymphomyeloid lesions[d], cardiac lesions[d], epithelial lesions including hepatocyte, lipidosis[d]	Spitsbergen et al., 1988b
Frog *Rana catesbiana*	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 *Rana catesbiana*	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 exopthalmia. 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 [^{3}H]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.

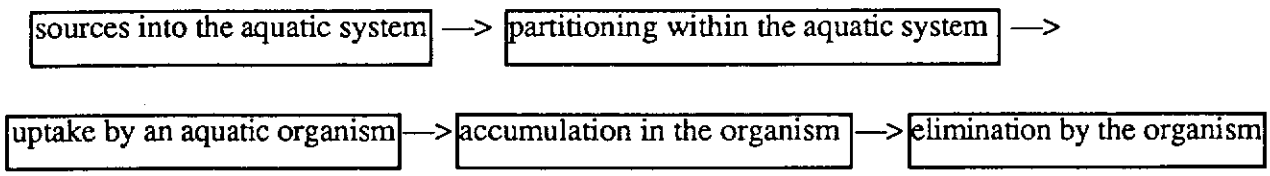
Although field research is still in progress, the bioaccumulation factor (BAF) approach and a variant of the BAF, which we have defined as the Regulatory Bioaccumulation Multiplier (RBM) (Sherman and Keenan, 1991), is the best available method for predicting dioxin uptake by fish while allowing regulatory 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:

$$\frac{dC_f}{dt} = k_1C_w - k_2C_f \qquad (1)$$

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:

$$C_f = k_1/k_2 \times C_w \times (1 - e^{-k_2t})$$

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:

$$C_w = C_e \times V_e / V_r \qquad (1)$$

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:

$$BAF = C_f / CE \quad (3)$$

where the C_f represents the concentration in fish tissue and CE is the environmental concentration of TCDD.

Solving for the environmental concentration:

$$CE = C_f / BAF \quad (4)$$

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),

$$C_f / BAF = (C_e \times V_e) / V_r \qquad (6)$$

Solving in terms of BAF:

$$BAF = \frac{C_f}{C_e \times V_e / V_r} \qquad (7)$$

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:

$$\begin{matrix}\text{Regulatory} \\ \text{Bioaccumulation} \\ \text{Multiplier}\end{matrix} = RBM = \frac{C_f}{(C_e \times V_e / V_r)} = \frac{C_f}{C_N}$$

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 $(mg/kg\text{-}day)^{-1}$[d]	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^{-8} [b]	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 x 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 x 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 x 10^{-5} or 4 x 10^{-4}, respectively (Crouch and Wilson, 1982). Workers at gas stations are exposed to benzene vapors from volatilizing fuel levels ranging up to 32 $\mu g/m^3$ (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 x 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 $\mu g/m^3$ (Wallace, 1990). The risk associated with exposure to a midrange level of 15,000 $\mu g/m^3$ for a worker's occupational lifetime can be calculated to be approximately 3 x 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 transporation-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 µg	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 µg	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 x 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 x 10^{-5} to 3.7 x 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:

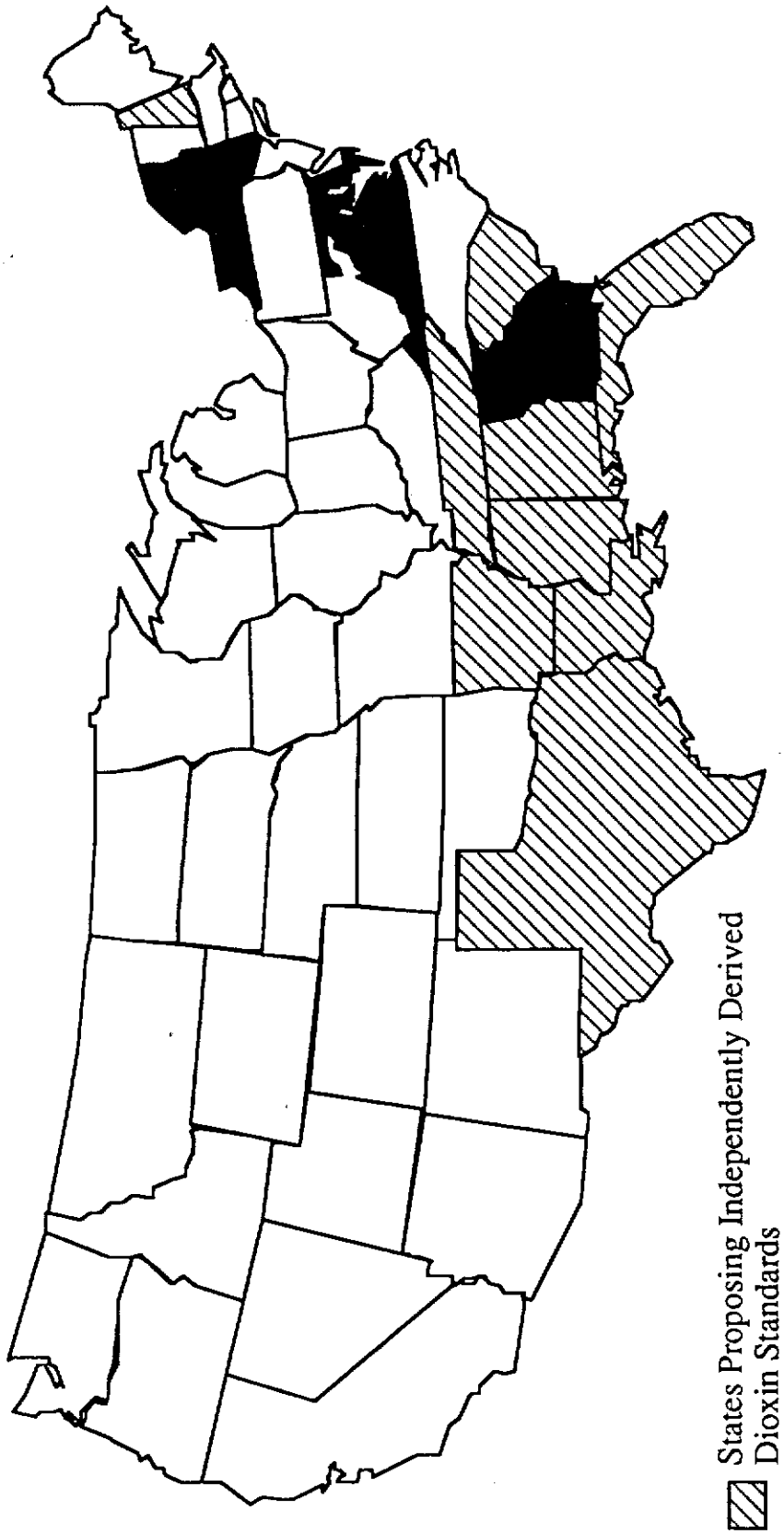


Figure 7-1. States Proposing Independently Derived Dioxin Standards

$$WQS = (ADI \times BW) / (BCF \times FCR)$$

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 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.

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APPENDIX A

Review of Reported Fish

Bioaccumulation/Bioconcentration Factors

APPENDIX A

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 m^3/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

West Virginia State Water Resources Board
1260 Greenbriar Street
Charleston, West Virginia 25311

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

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.

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 <u>incorrectly</u> 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 <u>zero</u>, since EPA policy assumes that <u>some</u> 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 <u>not</u> 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

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

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,

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. submitts 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
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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,

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 <u>not</u> 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 <u>(and all)</u> 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 <u>all</u> 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.

Innsbrook Technical Center
5000 Dominion Boulevard
Glen Allen, Virginia 23060

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.

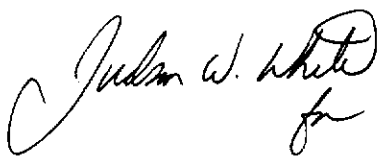
7. Instream biomonitoring as a method to verify lack of environmental impact. Ohio uses the results of such studies in-lieu of whole effluent toxicity tests.

8. Separate selenium criteria for lake and stream systems.

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,



B. M. Marshall
Manager
Water Quality

TWL/jmh

Mark J. Reasor, Ph.D.

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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.

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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

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

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 carcinogen chemicals throughout the lifetime is difficult to asse and may contribute to an increased cancer rate. A recent review of the epidemiological studies on diox carcinogenicity in humans (Bond et al., 1990) report that there is insufficient evidence to conclude tha 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

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.

Mark J. Reason

Equation for the calculation of a reference ambient concentration (RAC) (US EPA 1991):

$$RAC\ (mg/l) = \frac{(RL \times WT)}{ql^* \ [WI + FC \times L \times (FM \times BCF)]}$$

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.

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US EPA. (1991), Technical Support Document for Water Quality-based Toxics Control. EPA/505/2-90-001, PB91-127415, March.

APPENDIX E

SPECIFIC WATER QUALITY CRITERIA

PARAMETER	UNITS	USE DESIGNATIONS: Warmwater B 1,3,4	Troutwater B 2	Recreation C	Public A	All Other Uses
8.33 ZINC:						
Hardness mg/l as $CaCO_3$ / Zinc ug/l						
0-150 / 50						
151-300 / 100						
301-400 / 300						
greater than 401 / 600	ug/l	—	—	—	X	—
8.33.1						
0-50 / 40						
51-80 / 75						
81-120 / 90						
121-160 / 110						
161-200 / 130						
201-240 / 150						
241-280 / 175						
281-300 / 220						
301-320 / 270						
321-340 / 320						
341-400 / 370						
greater than 401 / 600	ug/l	X	—	—	—	—
8.33.2 Not to exceed 47 ug/l.	ug/l	—	X	—	—	—