APPENDIX I
WATER QUALITY PROGRAMS AND FUNDING SOURCES

NOTE TO READER: Because of ongoing changes in programs and funding sources, the information in this Appendix has been omitted. The most up-to-date information on funding sources can be found at the web site of the Massachusetts Nonpoint Source Management Plan: http://www.state.ma.us/dep/brp/wm/nonpoint.htm and by contacting other agencies and entities or consulting their web sites.

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APPENDIX II - REGULATIONS

NOTE: Regulations may change over time, and readers are urged to contact the regulatory agency to verify current regulatory requirements

The summary of the regulations presented here is an updated and expanded summary from the DEM Lake and Pond Management Coursebook (HWH, 1990b), the Clean Lakes Permit Guide (DEQE 1988) and the Wetlands Protection Program Policies (DEP 1995). All work in waterbodies or in their tributaries, outlets or bordering wetlands is subject to regulatory review.

The site location, project funding sources and scope of a proposed project defines the regulatory requirements that must be complied with. There are three regulatory levels: local, state and federal. Some environmental laws originate in the state, but are locally implemented (e.g. Wetlands Protection Act). Other environmental laws originate at the federal level, but are implemented at the state level (e.g. 401 Water Quality Certificate).

Environmental impacts are reviewed at two levels: the local conservation commission (ConComm), which has jurisdiction over the Wetlands Protection Act (WPA), and the Massachusetts Environmental Policy Act (MEPA) Unit, which has jurisdiction over state funded or authorized projects of a certain size or scope. The Massachusetts Department of Environmental Protection (DEP) may also review projects as part of the WPA and other regulations. A detailed guide to the Wetlands Protection Act is available (Colburn, 1995). *[Also see the* Environmental Handbook for MA Conservation Commissions, Dawson & Zielinski, 1997 – on the MACC web site, w/ updates for 2000 and 2002..]

The regulations may be broken down into two types: regulations that are concerned with protecting a specific site (e.g. sites which have unique environmental, rare species, historic, navigational, ownership, jurisdiction or other characteristics) and regulations that are concerned with activities and the types of pollution or water use (e.g. permits or licenses to apply chemicals, discharge wastes, dredging, draining or withdrawing water etc.).

In many cases, issuance of some permits is dependent upon successful review and issuance of other related permits. The more complicated a proposed project is the more complicated and intricate the regulatory flow chart becomes. For simple projects such as hand removal of vegetation on small (less than 10 acre) ponds a submission of a Request for Determination to the local ConComm and a Negative Determination of Applicability may be all that is required. Projects such as harvesting or herbicide application generally require the submission of a Notice of Intent (NOI) and the issuance of an Order of Conditions (OOC) from the ConComm along with the required permits from the state agencies. If the project involves some state agency action and is large (10 acres) or the site has certain characteristics (e.g., designation as an Area of Critical Environmental Concern (ACEC)) the Massachusetts Environmental Policy Act (MEPA) process is triggered, an ENF must be filed, and approval gained from MEPA before state permits can be issued. Dredging and filling in waterways generally require the most permits and may involve the issuance of a 401 Water Quality Certification, a Waterways (C.91) permit or license and a section 404 permit from federal ACOE. Such projects may also require a MEPA Environmental Impact Report (EIR). Large federal projects may require a federal Environmental Impact Statement (not discussed here).

The following sections briefly introduce the key regulations, criteria and timing for successful lake remediation, development and maintenance strategies. Acronyms are listed in Table II-1. Some of the more common permits for various lake management methods are summarized in Table II-2. Additional approval, permits and regulations that are site specific are listed in Table II-3.

Note that this summary is not intended to be a complete listing of the regulations and it remains the responsibility of the reader to comply with the regulations that are in effect at the time of application as they apply to the specific site and scope of work. Further information on general policies is contained in the publication "Wetlands Protection Program Policies" (DEP, 1995) and from the addresses listed in each section.

Table II-1. List of Acronyms.

ACEC	Area of Critical Environmental Concern
ACOE	U.S. Army Corps of Engineers
CMR	Code of Massachusetts Regulations
Con Comm	Conservation Commission
CZM	Coastal Zone Management
C.91	Chapter 91.
DEIR	Draft Environmental Impact Report
DEP	Department of Environmental Protection
DFA	Department of Food and Agriculture
DFW	Division of Fisheries and Wildlife
DOA	Determination of Applicability
DWM	Division of Watershed Management within DEP
EIR	Environmental Impact Report
ENF	Environmental Notification Form
EOEA	Executive Office of Environmental Affairs
EPA	U.S. Environmental Protection Agency
FEIR	Final Environmental Impact Report
LAC	License to Apply Chemicals
MDC	Metropolitan District Commission
MEPA	Massachusetts Environmental Policy Act
MHC	Massachusetts Historical Commission
MWRA	Massachusetts Water Resources Authority
NHESP	Massachusetts Natural Heritage Endangered Species Program
NOI	Notice of Intent
NPDES	National Pollutant Discharge Elimination Program
ODS	DEM Office of Dam Safety
OOC	Order of Conditions
PBL	Pesticide Bureau License
RDA	Request for Determination of Applicability
RPA	Rivers Protection Act
SECP	Sewer Extension Connection Permit
SOOC	Superseding Order of Conditions
SSC	State Sanitary Code
STP	Sewage Treatment Plant
USDA	U.S. Department of Agriculture
WMA	Water Management Act
WPA	Wetlands Protection Act
WPP	Wetlands Protection Program
WRP	Wetlands Restriction Program
ZC	Zoning Commission

It may be appropriate to add the Watershed Protection Act administered by MDC to the table below.

Table II-2 Potential Permits and Regulations for Lake Management.

Agency, Regulation	State EOEA MEPA	State DEP WPA	State DEP DWM	State DEP C.91	State DFG Fish	State DEP 401	Other misc.	Federal ACOE	Federal EPA
NUTRIENT CONTROL									
Nonpoint source, Best Man. Pract.		X					g		
Point source, sewage treatment	X	X	a,d,e	X			g		X
Dilution, flushing		X	b	X				X	
Hypolimnetic withdrawal		X	b	X			h	X	
Wetland, filter, detention	X	X		X		X	g,h	X	
Alum, P inactivation tech.		X	c	X					
Artificial circulation, aeration		X		X					
Dredging, hydraulic, dry	X	X	a, b	X	X	X	f,g,h	X	
Dredging, reverse layer	X	X		X		X	g	X	
WEED AND ALGAE CONTROL									
Water level draw down		X	b	X	X		g,h	X	
Harvesting, commercial, etc.		X							
Harvesting, hydroraking		X							
Biocontrol		X			X		I		
Sediment covers		X		X					
Dyes		X							
Herbicides, algicides		X	c				j		

KEY: X=Regulation may apply. a=DEP Surface Discharge Permit (NPDES). b=Water Management Act. c=DEP OWM License to Apply Chemicals. d=DEP Ground Water Discharge Permit. e=DEP DWPC Sewer Ext. or Connect permit. f=DEP Solid Waste Permit. g=Local zoning and/or restrictions may apply. h. DEM ODS permit. I=USDA and/or DFG and/or DAR permits for import and release of organisms to Massachusetts. j=DAR Pesticide license and Pesticide registry.

Note: Depending on site location, conditions and management method, other permits and regulations may apply (see Table II-3 and text).

Table II-3 Site Specific Regulations. The following table includes regulations that are directed at specific sites rather than specific treatments or methods of lake management. See text for more information.

Agency, Regulation (Abbreviation)	Description of applicable site.
Zoning Regulations, (Zoning)	Local towns may restrict construction and use of land within the borders of the town. Check with local officials.
Wetlands Restriction Program (WRP)	Selected wetlands may have permanent restriction orders restricting alteration of the resource. Check with Registry of Deeds.
Areas of Critical Environmental Concern (ACEC)	Unique environmental areas designated by the Secretary of EOEA are to be protected by EOEA agencies. Maps, designations, and information available from DCR ACEC Program. Protection is implemented through various state regulations and programs (DCR, DEP, MEPA, CZM).
Outstanding Resource Waters (ORW)	Exceptionally valuable waters, Class A water supplies, certified vernal pools are protected. List is available (DEP, 1993).
Massachusetts Waterways Regulations (C.91)	Tidelands, Great Ponds (over 10 acres) and certain rivers and streams are protected for public access and use. See section II.6.
Natural Heritage Program (NHESP)	Sites which contain rare or threatened species or their habitats. Almanac is available from the NHESP.
Coastal Zone Management Act (CZM)	Projects which require federal permits (401, NPDES) for work in coastal areas will require CZM review.
Massachusetts Historical Commission (MHC)	Projects funded, licensed or approved by State or Federal agencies which may impact a historic site. Sites are listed in the Registry of Historic Places.
Metropolitan District Commission (MDC) now the Department of Conservation and Recreation	Lands and waters under the supervision of the MDC are regulated by the MDC. These include lands around the Quabbin Reservoir. Abutters seeking to perform work may need a permit from the MDC.

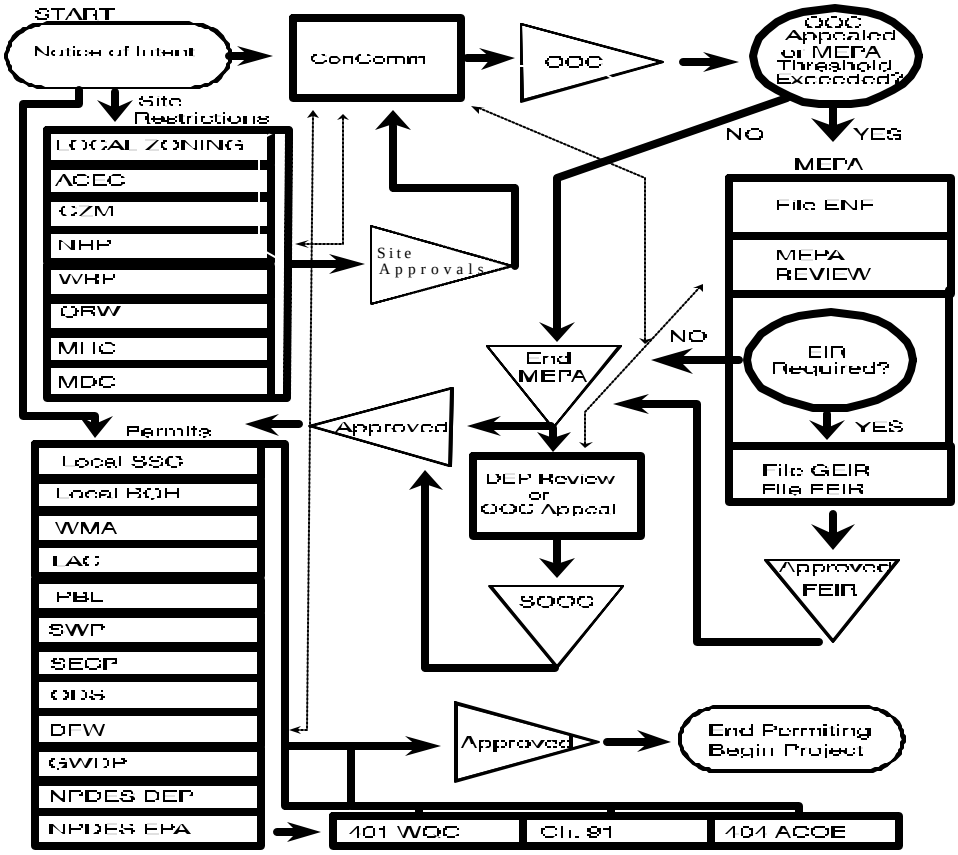

Figure II.1 Permit Flowchart. Dotted lines indicate agency interactions.

LOCAL PERMITS AND REGULATIONS

In accordance with Massachusetts Home Rule Provisions each city and town in the Commonwealth may enact local zoning, board of health and resource protection ordinances, bylaws and regulations. Depending upon the type of work proposed and the locality in which a waterbody is located, different levels of local regulatory review may be triggered. Three local agencies that may regulate work relative to lake or pond management are the Conservation Commission (ConComm), the Board of Health (BOH) and the Zoning Commission (ZC). These boards may be known by other names in certain towns.

II.1 SSC- STATE ENVIRONMENTAL CODE and TITLE 5 (BOH)

Jurisdiction: Public Health, Safety and Welfare relative to sewage disposal, bathing beaches.

Authority: MGL Chapter 111 and MGL c 21A s13.

Regulations: Under state law, local Boards of Health, by Board of Health vote, have the authority to adopt local bylaws that regulate disposal of sewage and that regulate bathing beaches.

State Sanitary Code
310 CMR 15.000 (Title 5)
105 CMR 445.00

Application: Title 5 regulates on-site subsurface sewage disposal (septic systems) from small (generally less than 10,000gpd) facilities. For domestic septic systems, the management techniques are detailed in Title 5 of the State Environmental Code 310 CMR 15.000 et. seq. Any new septic systems must comply with design and construction standards given in 310 CMR 15.00 etc. which specifies the leach field must be setback a minimum of 50 feet from surface waters. Current regulations do not require relocation of systems that are within 50' of surface waters which existed when setback requirement became effective in 1995. Several alternative technologies are approved under Title 5 for use in upgrades of existing systems and new construction [See DEP's Web Page for details].

Good water quality including a four foot visibility is to be maintained at swimming beaches (see 105 CMR 445.10).

Permit: Established locally.

Timing: Established locally.

Appeal: Superior Court

Inter-Agency Requirements: If subject to MEPA, the MEPA process must be complete.

Information: Local Boards of Health Officer.

II.2 Zoning- ZONING REGULATIONS (ZC)

Depending upon the type of work proposed and the locality in which a waterbody is located, different levels of local regulatory review may be triggered.

Jurisdiction: Construction and land use within the town.

Authority: In accordance with Massachusetts Home Rule Provisions each city and town in the Commonwealth may enact local zoning.

Regulations: Established locally.

Application: Established locally.

Permit: Established locally.

Timing: Established locally.

Appeal: Superior Court

Information: Local Zoning Commission. Zoning Board of Appeals.

II.3 WPA- WETLANDS PROTECTION ACT (ConComm)

Jurisdiction: See section II.4- process by which activities affecting Areas Subject to Protection Under the Act are to be regulated in order to contribute to the following eight interests:

- protection of public and private water supply
- protection of ground water supply
- flood control
- storm damage prevention
- prevention of pollution
- protection of land containing shellfish
- protection of fisheries
- protection of wildlife habitat

The purpose of the regulations is to define and clarify that process by establishing standard definitions and uniform procedures by which conservation commissions and the Department of Environmental Protection (DEP) may carry out their responsibilities under the Act. Further information is provided by the DEP Wetlands Policy

Authority: Massachusetts Wetlands Protection Act
MGL Chapter 131, Section 40
310 CMR 10.00
Local Wetlands Ordinances or Bylaws

Regulations: The Massachusetts Wetlands Protection Act sets forth a public review and decision see section II.5 below.

Local ordinances, bylaws or regulations may establish more stringent protection of wetlands or other natural resource areas (e.g. for purposes of aesthetics, wildlife, soils, etc.)

The state regulations establish "Areas Subject to Protection Under the Act" and from these areas establish "Wetland Resource Areas." Activity proposed within a resource area is subject to regulation. Activity proposed within one hundred (100) feet of a resource area (except for Bordering Land Subject to Flooding and Isolated Land Subject to Flooding) which may alter an Area Subject to Protection is subject to regulation as work within the Buffer Zone.

The following areas are subject to protection under the Act:

(a) Any bank, the ocean, any freshwater wetland, any estuary, any coastal wetland, any creek, any beach, bordering any river, any dune, on any stream, any flat, any pond, any marsh, or any lake or any swamp
(b) Land under any of the waterbodies listed above
(c) Land subject to tidal action
(d) Land subject to coastal storm flowage
(e) Land subject to flooding

Note: the Rivers Protection Act (see II.4 below) adds the “riverfront area” as a resource area under the Wetlands Protection Act.

The definition of "Activity" and "Alter" are broad and will potentially result in the regulation of all lake/pond remediation and maintenance projects.

> *Activity* means any form of draining, dumping, dredging, damming, discharging, excavating, filling or grading; the erection, reconstruction or expansion of any building or structures; the driving of pilings; the construction or improvement of roads and other ways; the changing of run-off characteristics; the intercepting or diverging of ground or surface water; the installation of drainage, sewage and water systems; the discharging of pollutants; the destruction of plant life; and any other changing of the physical characteristics of land.
>
> *Alter* means to change the condition of any Area Subject to Protection Under the Act. Examples of alterations include, but are not limited to, the following:
>
> (a) the changing of pre-existing drainage characteristics, flushing characteristics, salinity distribution, sedimentation patterns, flow patterns and flood retention areas;
> (b) the lowering of the water level or water table;
> (c) the destruction of vegetation;
> (d) the changing of water temperature, biochemical oxygen demand (BOD) and other physical, biological or chemical characteristics of the receiving water.

The regulations establish inland and coastal resource areas and establish performance standards to which all activity must conform. There are 11 coastal resource areas and 4 inland resource areas. Those most pertinent to lake and pond projects include

Coastal	Inland
Salt Marshes	Bank
Land Under Salt Ponds	Bordering Vegetated Wetland
Banks of or Land Under Ponds, Streams, Rivers, Lakes or Creeks that Underlie an Anadromous/ Catadromous Fish Run	Land Under Waterbodies and Waterways
	Land Subject to Flooding

Presumption of significance (why they are regulated) and performance standards (how to work within them) are established for each. In developing the Order of Conditions the ConComm considers site specific characteristics to which special regulations may apply, e.g. ACEC, ORW, CZM, MHC etc. See Table II-3 for a more complete list. ACEC designations may have Secretarial findings that wetlands resource areas within the ACEC are significant, and for coastal wetlands resource areas, this finding requires that significance shall be presumed by the local ConComm and by DEP. Within an ACEC, performance standards are raised. For coastal resource areas within an ACEC, the performance standard is raised to one of no adverse effect. For the inland wetlands resource area, Bordering Vegetated Wetland (BVW), within an ACEC, potential projects are prohibited that would result in the loss of up to 5,000 square feet or, in some cases, up to 500 square feet, of BVW. However, ACEC designation does not prohibit work affecting BVW if such work can be authorized under any other section of the Wetlands Protection Regulations, such as the "limited projects" section, 310 CMR 10.53(3). *Note:* Within an ACEC, an appeal of a local OOC requires the filing and review of an ENF at MEPA before DEP can act on the Superseding Order of Conditions, with the exception of projects for a single family dwelling.

Application: Request for Determination of Applicability (RDA): to identify jurisdiction and regulatory requirements.

Notice of Intent (NOI): Full application form for complete project description, analysis of impacts, discussion of mitigating measures. Notice of the NOI must also be delivered by hand or certified mail to all abutters (see Appendix VIII for details). A Notice of Intent must be sent to the Conservation Commission with a copy to the Department of Environmental Protection Regional Office. Additional copies must be sent to the Massachusetts DFW Natural Heritage and Endangered Species Program if rare or endangered species habitat is present. The NOI copies must be submitted at the same time as the NOI is sent to the Conservation Commission.

Permit: Determination of Applicability (DOA)
Order of Conditions (OOC)

Fees: Range from $55.00 to $1,000 for NOI and from $50.00 to $4,000 for DEP actions.

Information: Contact DEP Regional Office or Local Conservation Commission. Colburn (1995) provides "A Guide to Understanding and Administering the Massachusetts Wetlands Protection Act". 2nd. Edition, E.A. Colburn, (Ed.). Massachusetts Audubon Society, Lincoln, MA.

Guidance is also provided in the DEP (1990) publication: Wetlands Protection Program Interim Technical Guidance 90-TG1: Review of Lake and Pond Drawdown Projects for Aquatic Plant Control Under 10.53(4). Department of Environmental Protection. Boston, MA. Additional information can be found in S. Jackson, 1995. Delineating Bordering Vegetated Wetlands Under the Massachusetts Wetlands Protection Act. Department of Environmental Protection. Boston, MA. ***[see MACC web site & publications]***

Timing: Public Hearing held within 21 days of filing Notice of Intent. Permit issued/denied within 21 days of close of Public Hearing and local timing requirements.

Appeal: To DEP Regional Office within 10 days of issuance of order or denial. See section II.5 below.
To Superior Court if under local ordinance or bylaw.

NOTE: See "Guidance for Aquatic Plant Management in Lakes and Ponds as it Relates to the Wetlands Protection Act", DEP Policy/SOP/Guidance # BRP/DWM/WW/GO4-1, effective April 8, 2004.

II.4 RPA- RIVERS PROTECTION ACT (ConComm)

Jurisdiction: The act contains amendments to the Wetlands Protection Act (See II.3 above), which adds a new resource area with its performance standards to the Wetlands Protection Act.

Authority: River Protection Act
Chapter 258 of the Acts of 1996.
See also:
Massachusetts Wetlands Protection Act
MGL Chapter 131, Section 40
310 CMR 10.00
Local Wetlands Ordinances or Bylaws

Regulations: The resource area is called the "riverfront area" and extends 200 feet (25 feet in municipalities with large populations and in densely developed areas and 100 feet in areas of new agricultural or aquacultural activities) on each side of perennial rivers and streams throughout the Commonwealth. Applicants proposing work in the riverfront area must obtain an Order of Conditions as specified in the Wetlands Protection Act.

Regulations are included within the Wetlands Protection Act Regulations, 310 CMR 10.00.

STATE PERMITS AND REGULATIONS

II.5 ACEC- AREAS OF CRITICAL ENVIRONMENTAL CONCERN (DEM)

Authority: M.G.L c. 21A, s. 2(7)

Regulations: 301 CMR 12.00

Areas of Critical Environmental Concern (ACECs) are places in Massachusetts that receive special recognition because of the quality, uniqueness, and significance of their natural and cultural resources. These areas are identified and nominated at the community level and are reviewed and designated by the state's Secretary of Environmental Affairs. The Department of Environmental Management (DEM) administers the ACEC Program on behalf of the Secretary. As of February 2003, there are 28 ACECs in 73 municipalities covering approximately 241,000 acres.

The ACEC Regulations (301 CMR 12.00) describe the purpose and procedures for the nomination, review, and designation of ACECs by the Secretary. The designation works through the existing state environmental regulatory and review framework. (See sections in this appendix for MEPA and DEP.) The ACEC Regulations direct EOEA agencies to take actions, administer programs, and revise regulations in order to preserve, restore, and enhance the natural and cultural resources of ACECs. All EOEA agencies must:

1. acquire useful data on the ACEC,
2. preserve, restore, or enhance the resources of the ACEC,
3. ensure activities in or impacting such areas are carried out so as to minimize adverse effects on marine and aquatic productivity, surface and groundwater quality, habitat values, storm damage prevention or flood control, historic and archeological resources, scenic and recreational resources and other natural resource values of the area.

All EOEA agencies shall subject projects to the closest scrutiny to assure the above standards are met for any action subject to their jurisdiction. Descriptions and maps of ACECs, .a "Guide to State Regulations & Programs Regarding ACECs (July 2001)," and other publications are available from the ACEC Program at DEM.

Information: ACEC Program
EOEA/DCR
251 Causeway St., Ste. 700
Boston, MA 02114
617-626-1250
www.state.ma.us/dem/programs/acec

II.6 ODS- OFFICE OF DAM SAFETY (DEM)

Jurisdiction: Safety and inspection of dams greater than 6 feet in height or which can store 15 acre-feet of water or which, upon breach, could endanger property or public safety.

The definition of dam is established at 302 CMR 10.06:

> "Dam shall mean any artificial barrier, including appurtenant works, which impounds or diverts water and which is (1) twenty-five feet or more in height as defined herein and (2) has an impounding capacity at maximum water storage elevation of fifty acre-feet or more. Dam shall also mean any other artificial barrier, including appurtenant works, the breaching of which could endanger property or safety, as determined by the Commissioner, and is greater than six (6) feet in height or impounds more than fifteen (15) acre feet of water; or any structure classified as a roll dam."

Hazard Potential is defined as "the potential for causing property damage or loss of human life in the event of failure or improper operation of a dam."

Authority: M.G.L. Chapter 253, sections 44-50

Regulations: 302 CMR 10.00

Application Form: Dam Registration Forms, Notice of Intent of determine whether Chapter 253 Permit is required.

Permit: Chapter 253 Permit to Construct, Repair, or Remove a Dam ("Chapter 253 Permit"), Certificate of Compliance or Non-Compliance

Timing: A Chapter 253 Permit will be issued within 30 days from the time the final design report is received and approved.

Inter-Agency Requirements: Work in the vicinity of a dam may require permit filings relative to wetland alteration, e.g. Chapter 91, Wetlands Protection Act.

Information: Office of Dam Safety
Department of Environmental Management
251 Causeway St., 02114.
Boston, MA

II.7 DRINKING WATER PROTECTION (DEP)

Jurisdiction: The Department of Environmental Protection has regulations that promote the public health and general welfare by ensuring that public water systems in Massachusetts provide safe drinking water to its users. The regulations set forth standards and requirements of general application and future effect.

Authority: M.G.L. Chapter 111, section 160A

Regulations: The Massachusetts Drinking Water Regulations, 310 CMR 22.00 address land use controls at Class A surface water supplies and their tributaries. 310 CMR 22.20(B) applies to all reservoirs. Examples of land use controls in Section B include the following.

(6) No person shall swim, wade or bathe in any public surface water source and no person shall, unless permitted by written permit by the Board of Water Commissioners or like body having jurisdiction over such source , fish in; enter or go in any boat, seaplane, or other vehicle ; enter upon the ice for any purpose, including the cutting or taking of ice; or cause or allow any animal to go into, or upon, any surface water source or tributary thereto.

(8) No person shall apply herbicides to any surface water body including but not limited to any reservoir and their tributaries, which serve as a source of public water supply without a permit issued by the Department pursuant to M.G.L. c. 111, § 5E. This requirement does not apply to the application of algaecides containing copper by the public water system. However, the public water system shall notify the Department in writing prior to the application of such algaecides.

310 CMR 22.20C addresses land use controls at Class A surface water supplies and their tributaries that are implemented through local bylaw or regulation. Examples of land use controls in Section C include new:

(b) facilities that, through their acts or processes, generate, treat, store or dispose of hazardous waste that are subject to M.G.L. c. 21C and 310 CMR 30.000, except for the following:

1. very small quantity generators, as defined by 310 CMR 30.000;
2. treatment works approved by the Department designed in accordance with 314 CMR 5.00 for the treatment of contaminated ground or surface waters;

(c) sand and gravel excavation operations; and

(l) land uses that result in the rendering impervious of more than 15%, or more than 20% with artificial recharge, or 2500 square feet of any lot, whichever is greater.

These requirements apply to all land and watercourses used as or tributary to a public water supply system except:

(a) Rivers and streams designated as Class B waters pursuant to 314 CMR 4.00 which are used as drinking water sources and are not impounded at some point by means of a dam or dike to create a reservoir at which the water supply intake is located and

(b) Emergency sources approved by the Department under the provisions of M.G.L. 21G.

II.8 GWDP- GROUND WATER DISCHARGE PERMIT (DEP)

Jurisdiction: Discharge of greater than 10,000 gallons per day (gpd) of pollutants into groundwaters from point sources.

Authority: Massachusetts Clean Water Act MGL Chapter 21 S 42, 43.

Regulations: 314 CMR 2.00, 5.00, 6.00

Application: Public and private wastewater treatment systems discharging in excess of 10,000 gpd. Applications must include submission of a hydrogeologic study of affected area as well as plans and specifications for the proposed wastewater treatment scheme.

Permit: Ground Water Discharge Permit.

Timing: Application must be submitted at least 180 days before the date on which discharge is to commence.

Fees: Variable $1550 - $8600 with annual compliance fee.

Inter-Agency Requirements: If subject to MEPA, the MEPA process must be complete.

Information: Regional Office of the DEP

II.9 SECP- SEWER EXTENSION OR CONNECTION PERMIT (DEP)

Jurisdiction: Connection or extensions of sewer lines to existing Publicly Owned Treatment Works (sewage treatment plants). Discharges of less than 15,000 GPD of sanitary sewage are exempt.

Authority: Massachusetts Clean Water Act MGL Chapter 21 S 26-53.

Regulations: 314 CMR 7.00 and 314 CMR 12.00

Application: Application for Permit for Sewer System Extension or Connection. Forms are available from the Regional Offices of the DEP. Apply to Bureau of Waste Prevention for industrial wastes and the Division of Water Pollution Control for municipal (sewage) permits.

Permit: Permit for Sewer System Extension or Connection.

Timing: Not Specified.

Fee: Ranges from $200.00 to $1450.00. Industrial permits are more expensive.

Inter-Agency Requirements: Permits may require MEPA review.

Information: Regional Office of the DEP.

II.10 WMA- WATER MANAGEMENT ACT (DEP)

Jurisdiction: Withdrawal of water from surface or ground water sources for consumptive use in excess of threshold volumes. Current threshold is 100,000 GPD. Users withdrawing water for nonconsumptive use must still submit a notification.

Authority: Massachusetts Water Management Act, M.G.L. Chapter 21G

Regulations: 310 CMR 36.00

Application: Permit application form available from the Drinking Water Program.

Permit: Water Withdrawal Permit (Permit Code: BRPWM03).

Timing: Not specified. Permits valid for periods of time less than 20 years.

Information: Regional Office of the DEP.

II.11 NPDES- NATIONAL POLLUTANT DISCHARGE ELIMINATION SYSTEM PERMIT (DEP)

Jurisdiction: Point Source discharges to surface waters with proper wastewater treatment. This would apply if a treatment plant were proposed in association with lake remediation or management or if a point source discharge of stormwater from certain types of land uses were proposed. This permit is issued jointly by the EPA and the Department. A permit is submitted to the EPA and reviewed jointly by both agencies.

Authority: Massachusetts Clean Water Act, M.G.L. Chapter 21, Section 43; Federal Clean Water Act.

Regulations: 314 CMR 2.00, 3.00, 5.00 and 7.00

Application: Application for Permit to Discharge to Waters of the Commonwealth, Application to Discharge to Ground and Application for Permit for Sewer System Extension or Connection.

Permits: Permit for each of the above.

Timing: File at least 180 days prior to discharge (314 CMR 3.09 and 5.09)

Appeal: Within 30 days following issuance of a Permit, a request for an adjudicatory hearing may be filed 314 CMR 2.08).

Inter-Agency Requirements: If subject to MEPA, the MEPA process must be complete.

Information:

Department of Environmental Protection
Watershed Permitting Program
One Winter Street
Boston, MA 02108
617/292-5673

II.12 SWP- SOLID WASTE PERMIT (DSW)

Jurisdiction: Disposal of Dredged Material in approved landfill

Authority: M.G.L. Chapter 21A, sects. 2 and 8, M.G.L. Chapter 111, Section 150A

Regulations: 310 CMR 19.00

Application Form: If the dredge spoils are not deemed acceptable for sale or use as a daily landfill cover, soil conditioner or embankment fill, Request for Determination whether dredged material or other lake remediation solid waste is a special waste per 310 CMR 19.061.

Permit: Issuance of written determination.

Timing: Not specified

Inter-Agency Requirements: Any work within wetland areas will require compliance with all wetlands related permitting programs.

Information: Division of Solid Waste Management
One Winter Street
Boston, MA 02108
(617) 590-5961

II.13 ORW - OUTSTANDING RESOURCE WATERS (OWM)

Regulations: 314 CMR 4.04

The Massachusetts Surface Water Quality Standards contain antidegradation provisions to maintain existing uses of surface waters. Waters with exceptional socio-economic, ecological and/or aesthetic values are designated as Outstanding Resource Waters (ORW). These waters have more stringent requirements than other waters including the virtual prohibition of new or increased discharges of pollutants. In particular the 401 Water Quality Certification has more stringent requirements for ORWs.

Outstanding Resource Waters include all Class A designated public water supplies and vernal pool certified as such by the Natural Heritage Program of the Department of Fisheries and Wildlife. Other waters designated ORW may include National Parks, State Forests and Parks and Areas of Critical Environmental Concern. The most recent listing of ORW is found in the publication, "Designated Outstanding Resource Waters of Massachusetts, 1990" (DEP, 1993)

II.14 LAC - LICENSE TO APPLY CHEMICALS (DWM)

Jurisdiction: Application of chemicals to water body for ~~weed~~ nuisance vegetation control. A permit is not required for state or federal agencies while in the conduct of their official duties. No permit is required for privately owned (single owner) ponds from which there is no flowing outlet.

Authority: M.G.L. Chapter 111, Section 5E

Regulations: None

Application: Application for License to Apply Chemicals for Control of Nuisance Aquatic Vegetation.

Permit: License to Apply Chemicals for Control of Nuisance Aquatic Vegetation. Must include OOC or negative DOA, an accurate map of the lake and sites to be treated and information about the lake, lake usage, algae and/or plants and chemical to be applied.

Timing: License application must be completed and submitted at least thirty (30) days prior to the proposed date(s) of treatment. Within 14 days of treatment the licensee must submit a certified report on the treatment. The license is valid for nine months.

Fee: $60.00 (fee waived for town or municipal governments.)

Inter-Agency Requirements: . A NOI must be filed with the local Conservation Commission(s) in accordance with the Wetlands Protection Act (MGL c. 131, s. 40) and Wetlands Protection Regulations (310 CMR 10.00). Licensee shall obtain either a Final Order of Conditions or negative Determination of Applicability prior to application of chemicals.

Additionally, chemical application shall only be conducted by an applicator currently licensed by the Massachusetts Department of Food and Agriculture Pesticide Bureau in the aquatic weed category. Only products registered by the Pesticide Bureau (see BPL) may be applied in the state. ~~A NOI must be filed in accordance with the WPA.~~

Information: Bureau of Resource Protection, ~~OWM~~ DWM
Department of Environmental Protection
One Winter Street, 5th floor
Boston, MA 02108
(617)292-5500

II.15 WPP- WETLANDS PROTECTION PROGRAM (DWW)

This program has been described fully in the Local Regulation Section II.3, as local Conservation Commissions are responsible for the implementation of the Wetlands Protection Act and the regulations are 310 CMR 10.00. However, upon appeal, the DEP becomes the issuing authority.

The DEP Wetland Program Policy recommends that Conservation Commissions request an applicant to compile a minimum amount of information to assess whether plant species composition will be successfully effected by the treatment. This information may include the following:

- description of existing resource condition;
- goals of the project;
- why a need exists to improve resource area;
- how project will improve resource area;
- description of positive and negative effects;
- description of proposed mitigation efforts;
- what impacts will occur in relation to the eight interests of the act;
- what permits are required;
- description of the follow up monitoring plan.

Appeal Provisions: Any appeal of an action by the Conservation Commission must be filed with the DEP Regional Office within 10 business days of the issuance of the local decision. Upon appeal of a local action, the Request for Determination of Applicability or Notice of Intent and appealed Determination or Order of Conditions are reviewed by the Department of Environmental Protection (DEP) for issuance of a Superseding Determination of Applicability or Superseding Order of Conditions. The Department may issue a Superseding Determination of Applicability within 35 days from receipt of a request and may issue a Superseding Order of Conditions within 70 days of a request (310 CMR 10.05 (3) and (7).

Inter-Agency Requirements: Once an appeal is filed, the issuance of a Superseding Order of conditions constitutes a state action subject to MEPA. Any project proposing the alteration 5,000 square feet of Bordering Vegetated Wetland, 500 feet or more of Bank or of 1/2 acre of any other wetland resource area must file an ENF (301 CMR 11.03(3)(b)). Any project proposing alteration of one acre of Bordering Vegetated Wetland or 10 acres of wetland resource area or Great Pond is categorically included and must file both an ENF and EIR (301 CMR 11.03(3)(a)). Within an ACEC, any project appealed to DEP, regardless of size, except for a single-family dwelling, is subject to filing an ENF with MEPA (301 CMR 11.03(11)). The requirement to file with MEPA may be waived if the Secretary of the Executive Office of Environmental Affairs has found a Generic Environmental Impact Report (GEIR) sufficient to waive the requirement.

Information: Regional DEP Office or
Boston Office: Department of
Environmental Protection
Div. of Wetlands and Waterways
One Winter Street
Boston, MA 02018
(617) 292-5695

II.16 WRP- WETLANDS RESTRICTION PROGRAM (DWW)

Jurisdiction: The purpose of the Wetlands Restriction Program is to map all the state's wetlands and select the most important wetlands for deed restrictions which prohibit activities that would impair their functions. A list of communities having wetlands restrictions is available from the DWW. Depending on an individual deed restriction, lake management and remediation work may not be allowed. Typically, destruction of natural vegetation, alteration of existing patterns of flow, alteration of natural contours, discharge of pesticides and draining and dredging have been prohibited.

To place a restriction on a wetland area, public hearings and site visits must be held.

Authority: M.G.L. Chapter 130, section 105, M.G.L. Chapter 131, Section 40A

Regulations: 310 CMR 12.00 and 13.00

Application: Notice of Intent, per 310 CMR 10.00

Permit: Order of Conditions, per 310 CMR 10.00

Timing: Same as 310 CMR 10.00

Inter-Agency Requirements: Orders placed on restricted wetlands within the boundary of the Massachusetts coastal zone will be consistent with the policies and regulations of the Office of Coastal Zone Management (301 CMR 20.00, S. 21.00). The regulations for both coastal and inland restrictions (sections 12.01(4) and 13.01(4)) require the administering state agency to prepare a schedule for restricting inland and coastal wetlands located within designated ACECs. Many, but not all, ACECs have Orders of Restriction in place. However, the DEP Wetlands and Waterways Program has no current plans for placing additional wetlands restrictions in any communities.

Information: Program Coordinator
Wetlands Restriction Program
Division of Wetlands & Waterways
One Winter Street
Boston, MA 02108

II.17 C.91- WATERWAYS REGULATION CHAPTER 91 (DWW)

Jurisdiction:

(1) all waterways, including all flowed tidelands and all submerged lands lying below the high water mark of:
   (a) Great Ponds;
   (b) the Connecticut River
   (c) the section of the Westfield River in the Towns of West Springfield and Agawam lying between the confluence of said river with the Connecticut River and the bridge across said river at Suffield Street in said Town of Agawam;
   (d) the non-tidal river or stream on which public funds have been expended for stream clearance, channel improvement, or any form of flood control or prevention work, either upstream or downstream within the river basin, except for any portion of any such river or stream which is not normally navigable during any season, by any vessel including canoe, kayak, raft, or rowboat; the Department may publish, after opportunity for public review and comment, a list of navigable streams and rivers; and

(2) all filled tidelands, except for landlocked tidelands and all filled lands lying below the natural high water mark of Great Ponds (310 CMR 9.04).

Authority: M.G.L. Chapter 91

Regulations: 310 CMR 9.00

Application Form: Application Form 1; Dredging Addendum Application Form 2; Municipal Zoning Certification Application Form 3; and Municipal Planning Board Notification Application Form 4; To identify whether a project is subject to the regulations, one may submit a formal Determination of Applicability (310 CMR 9.05).

Permit: Chapter 91 License and Chapter 91 Dredge Permit

Timing: The Department shall issue a license, permit, etc., after an application is determined to be complete (310 CMR 9.14). The DEP recommends that the proponent asks for pre-application consultation per 310 CMR 9.11.

Inter-Agency Requirements: No license or permit will be issued until the MEPA process is complete, a final Order of Conditions is issued, a Water Quality Certification is issued, a Certificate of Compliance with local zoning and copies of all other state regulatory approvals that may apply are submitted. (310 CMR, 9.11, 9.33 and 9.34) Additionally, CZM policies and ACEC criteria may need to be complied with, as appropriate. The Waterways Regulations do not allow new fill in ACECs. Within an ACEC, other restrictions apply concerning private water-dependent structures. Higher standards are also required regarding dredging and disposal activities within ACECs (310 CMR 9.40(1)(b)). Improvement dredging, except for the sole purpose of fisheries or wildlife enhancement, is prohibited within an ACEC. Maintenance dredging remains eligible for a permit. Also, the regulations prohibit the disposal of dredged material within an ACEC, except for the purposes of beach nourishment, dune stabilization with proper vegetative cover, or the enhancement of fishery or wildlife resources.

Appeal: A Notice of Claim for an adjudicatory hearing must be filed within 21 days of the date of issuance of a written determination, draft license or permit. (310 CMR 9.17)

Information: Department of Environmental Protection
Wetlands and Waterways Program
Waterways Regulation Program
One Winter Street
Boston, MA 02108
617/292-5695

II.18 401- 401 WATER QUALITY CERTIFICATION (DWW)

Jurisdiction: A 401 Water Quality Certification is required under the federal Clean Water Act for projects that must obtain federal licenses or permits and that result in a discharge to state waters. The 401 review is to ensure the project will comply with the state water quality standards. This includes any project that results in a discharge or disposal of dredged material greater than 100 cubic yards to waters subject to federal agency jurisdiction (see ACOE) and projects that propose to fill or excavate more than 5,000 square feet of wetlands. Projects which are not in an ORW and which are smaller than 100 cubic yards or 5,000 square feet may be reviewed by the Conservation Commission and if approved under the WPA, the Order of Conditions will act as the 401 WQC certification. The Department of Environmental Protection will review the Notice of Intent to determine if the thresholds are exceeded.

Authority: Section 27 (12) of the Massachusetts Clean Waters Act, M.G.L. C. 21, sections 26-53.

Regulations: 314 C.M.R. 9.00 and 314 CMR 4.00

Application: 401 Water Quality Certification for Fill and Excavation Projects in Waters and Wetlands and;

Major Project Certification BRP WW10 (>5,000 sq. ft.)
Minor Project Certification BRP WW11 (<5,000 sq. ft.)

401 Water Quality Certification for Dredging and Dredged Material Disposal

Major Project Certification BRPWW07
Minor Project Certification BRP WW08
Amendment for Certification for Dredging BRPWW09

Permit: Water Quality Certification

Timing: For projects categorically excluded under the MEPA regulations, applicants shall file an application form with the DEP Division of Wetlands and Waterways. For all projects which are not categorically excluded under the MEPA regulations, the applicant shall submit to the MEPA Unit a completed application form with the ENF. The Division shall act on an application for a water quality certification in accordance with 310 CMR 4.00, the Department's Timely Action Schedule and Fee Provisions Regulations.
Inter-Agency Requirements: a) Should a project be subject to MEPA regulations, a Standard Application Form must be filed as part of the ENF process. (314 CMR 9.02) b) The regulations are intended to be consistent with and form part of the Coastal Zone Management Program, insofar as a they may apply to a project. c) Section 401 of the Federal Water Pollution Control Act provides that any applicant for a federal license or permit for a project which may result in a discharge into the navigable waters of a state must provide the Federal licensing agency with a certification from that state's water pollution control agency. d) A water quality certification is required prior to the issuance of a Chapter 91 license.

Appeal: A notice of claim for an adjudicatory hearing must be accompanied by a filing fee (310 CMR 4.06) and sent by certified mail or hand delivered to the Office of Administrative Appeals of the Department of Environmental Protection postmarked within 21 days of the date of certification. The right to appeal is limited to certain persons including, the applicant or property owner, any person aggrieved by the decision who has submitted comments during the public comment period, any ten persons of the Commonwealth where a member has submitted comments during the public comment period, and any government body or private organization with a mandate to protect the environment which has submitted written comments during the public comment period. Further information is provided in 314 CMR 9.10.

Information: Department of Environmental Protection
Division of Wetlands and Waterways
One Winter St. 8th Floor
Boston, MA 02108
(617) 292-5695
or Regional Office of the DEP

II.19 PESTICIDE BUREAU LICENSE (DAR)

Jurisdiction: The purpose of the Pesticide Bureau regulations is to establish the standards, requirements and procedures for the certification and licensing of pesticide applicators, including specific requirements for aquatic pest control. No person shall use a pesticide that has been classified by the Pesticide Board as being restricted or state limited use unless he is an appropriately certified private or commercial applicator. (333 CMR 10.03) All certifications and licenses shall be for a period not to exceed one year. Categories of Commercial Applicators are established at 333 CMR 10.04. The Pesticide Bureau also registers ~~chemicals~~ pesticides for use in the state through the Pesticide Board Subcommittee. Pesticides may not be distributed, purchased or used in the state, unless they are ~~which are not~~ registered by the Subcommittee ~~DFA~~ (333 CMR 8).

Authority: M.G.L. Chapter 132 B, sections 6A and 10.

Regulations: 333 CMR 10.00

Pesticide Exams~~:~~ Competence in the use of pesticides shall be determined on the basis of written examinations and performance testing. (333 CMR 10.05) In the case of aquatic pest control, the applicators shall demonstrate practical knowledge of the principals of limited area treatments and the potential for adverse effects on fish, birds, beneficial insects and other non-target organisms that may be present. (333 CMR 10.05 (2) (e)) Procedures are established at 333 CMR 10.09.

Appeal: Any person aggrieved by a determination of the Board to issue, deny, revoke, modify or suspend any certification may, within 21 days request an Adjudicatory Hearing before the Pesticide Board (333 CMR 10.16).

Information: Pesticide Bureau, Department of Agricultural resources, 251 Causeway Street, Suite 500, Boston, MA 02114. www.mass.gov/dar 617-626-1700.

II.20 DFW - DIVISION OF FISHERIES AND WILDLIFE (DFG)

Jurisdiction: Review of drawdown activities and impact on fisheries and wildlife interests.

Authority: M.G.L. 131 Chapter 42 and 48

Regulations: None

Application: Written Notification to Division, coordination with local Conservation Commission is recommended.

Permit: Conditions recommended by Division to local Conservation Commission

Timing: Notification prior to filing a Notice of Intent under the Wetlands regulations (310 CMR 10.00) is recommended.

Appeal: Under the provision of the Wetlands regulations at 310 CMR 10.00.

Information: Division of Fisheries and Wildlife
Westborough, MA
508/366-4470

II.21 NATURAL HERITAGE ENDANGERED SPECIES PROGRAM (DFG)

The Natural Heritage & Endangered Species Program (NHESP), through the Massachusetts Division of Fisheries & Wildlife (DFW), has the regulatory authority to protect all state-listed rare plants and animals (see Appendix V) under the Massachusetts Endangered Species Act and they have the responsibility of providing their expert opinion on the impact of certain projects which are subject to the Wetlands Protection Act regulations. This section will address these responsibilities separately as two distinct evaluation processes exist for each.

A. MASSACHUSETTS ENDANGERED SPECIES ACT (under NHESP)

Jurisdiction: The Massachusetts Endangered Species Act (MESA) prevents the "taking" of any rare plant or animal species listed as Endangered, Threatened, or of Special Concern unless specifically permitted for scientific, educational, propagation, or conservative purposes. The Act also directs state agencies to "use all practicable means and measures to avoid or minimize danger to such species", as well as protect habitat for these rare species in areas specifically designated as "significant habitats". The NHESP has the authority to issues MESA related permits. The NHESP is charged with the inventory, research and protection of rare plant and animal species as well as other features of the state's biological diversity.

Authority: Massachusetts Endangered Species Act MGL c.131A (MESA)

Regulations: MESA regulations 321 CMR 10.00

Application: Written request for rare species information.

Permit: In the case of lakes and ponds with rare species, a Conservation/Management Permit likely will be required, or an Alteration Permit if the project is within a formally designated "significant habitat".

Appeal: Appeals of Alteration Permits may be sent to the Secretary of EOEA.

Timing: A written request for rare species information must be submitted at any time prior to the initiation of a project.

B. WETLANDS PROTECTION ACT (under NHESP)

Jurisdiction: The permit-issuing authorities under the Wetlands Protection Act are the Conservation Commission and the Department of the Environmental Protection (DEP). The NHESP is responsible for providing an opinion on whether a project being reviewed by a conservation commission and DEP is within the actual wetland habitat of a state-listed rare wetlands wildlife species (vertebrate and/or invertebrate) and whether that project as proposed will have any short or long term adverse impact on the wetland habitat of state-listed rare wetlands wildlife species. Projects subject to NHESP review are those projects requiring the filing of a notice of intent (NOI) which fall within the most recent Estimated Habitat Map of State-listed Rare Wetlands Wildlife (if any) published by the NHESP.

Authority: Massachusetts Wetland s Protection Act, M.G.L., Chapter 131, Section 40 (WPA)

Regulations: WPA regulations pertaining to the rare species : 310 CMR 10.37 and 10.59.

Application: Not filed with the NHESP.

Permit: NHESP opinion provided to Conservation Commission and DEP.

Appeal: The opinion of the NHESP is presumed correct. but is rebuttable and may be overcome by a clear showing to the contrary.

Timing: The NOI must be filed with the NHESP by U.S. Postal Service, express mail, priority mail (or otherwise sent in a manner which guarantees delivery within two days), being sent no later than the date of the filing of the NOI with the Conservation Commission. The conservation commission shall not issue a permit for at least 30 days after the filing of the NOI (received in a timely manner by the NHESP) unless the NHESP before that time period has made a determination.

Information: Natural Heritage & Endangered Species Program (NHESP)
Division of Fish & Wildlife
Route 135
Westborough, MA 01581
ph. (508)792-7270 x200

II.22 CZM- MASSACHUSETTS COASTAL ZONE MANAGEMENT (EOEA)

Jurisdiction: Compliance of projects requiring federal permits to coastal zone enforceable policies in the Massachusetts coastal zone. The regulations are promulgated to comply with the requirements of the Federal Coastal Zone Management act of 1972, as amended. Any lake remediation or management project which requires a permit from the U.S. Army Corps of Engineers (e.g. filling in wetlands, dredging) or the U.S. Environmental Protection Agency (e.g. NPDES permit), when conducted in the coastal zone will require a CZM federal consistency review.

Authority: M.G.L. Chapter 21A, 552, 4A 16U.S.C. 1451 et seq.

Regulations: 301 CMR 20.00 and 21.00. 15 CFR 930, as amended.

Application Form: Federal Consistency Certification, federal permit applications (as appropriate), final decision of the MEPA process (as appropriate), other information, as deemed necessary.

Permit: Concurrence with Consistency Certification

Timing: A CZM Consistency Concurrance must be issued prior to the issuance of any federal permit. In all cases, the CZM Office shall issue its decision within six months of commencement of its final review. CZM Office will not issue a concurrence decision until EOEA Agencies have completed action on the license or permit applications.

Inter-Agency Requirements: The CZM Consistency Certification is a requirement prior to the issuance of federal permits for projects in the Massachusetts coastal zone. Additionally, the CZM Office participates in any coastal MEPA review so as to alert an applicant to any inconsistencies in the proposed activity to the CZM Policies at an early stage.

Information: Office of Coastal Zone Management
251 Causeway Street, Suite 900
Boston, MA 02114-1219
617/626-1200

II.23 MEPA- MASSACHUSETTS ENVIRONMENTAL POLICY ACT (EOEA)

Jurisdiction: MEPA provides overall environmental review of projects requiring state permits, funding or other state action. The Massachusetts Environmental Policy Act regulations provide a uniform system for compliance with the Massachusetts Environmental Policy Act, M.G.L. Chapter 30, sections 61 through 62H. MEPA applies to the activities of all agencies of the Commonwealth, to all activities carried out with financial assistance from agencies and to all activities which require permits granted by agencies. The regulations establish thresholds, a procedure and a timetable for a two-level review process, which generally includes filing of an Environmental Notification Form (ENF) and, if required, an Environmental Impact Report (EIR).

If the project exceeds review thresholds (301 CMR 11.25, 11.26 & 11.27) an ENF must be filed. This is reviewed by agencies, the public and, based on the review, the Secretary of Environmental Affairs must determine if an EIR is required. An EIR is an informational planning document which is intended to inform project proponents, public decision makers and the general public of the environmental effects of proposed activities, to enable environmental damages and benefits to be fully disclosed and to consider strategies to avoid or minimize environmental impacts.(301 CMR 11.01)

For lake and pond remediation and management work, the MEPA process may be triggered in several ways. A project is subject to MEPA if it requires any agency action, financial assistance, or permit. Lake management projects require both an ENF and an EIR if the project involves specific areas, funding or uses which are summarized below:(301 CMR 11.25)

a) Dredging or altering one or more acres of bordering vegetated wetlands or salt marsh, or 10 or more acres of other wetland resource areas including lakes (excluding the buffer zone)
b) Stream channelization or relocations of 2,000' or more.
d) New impoundments of one 1,000,000 or more gallons of water.

Projects require an ENF and may require an EIR if:(301 CMR 11.26 & 11.27)

a) An agency undertakes a project within its own permit jurisdiction and the project exceeds the review thresholds.(301 CMR 11.25, 11.26, & 11.27)
b) A Order of Conditions is appealed to the Department of Environmental Protection for a Superseding OOC and more than a specified area of wetlands resource area is being altered. These limits are more strict than those listed above. Specifically, the following require an ENF and may require an EIR if the project involves any dredging, filling, altering or removal of: 1,000 square feet of saltmarsh; 5,000 square feet of bordering vegetated wetland; 500 feet of bank; or one half acre of any other resource area subject to WPA.
c) A variance from the Wetlands Regulations is required.(310 CMR 10.00)
d) A release of Wetlands Restrictions is required.
e) A variance of Waterways regulations is required.
f) Dredging and/or disposal of 10,000 cubic yards of materials is involved.
g) 500 feet of bank will be altered.
h) Chapter 253 approval for construction or alteration of dams, or changing dam capacity by 20% is required.
I) Water Pollution Control Water Quality Certification related to discharges is required.(301 CMR 11.26(7)(c))
j) State projects, or state funding are involved and the cost (excluding land acquisition) is $1,000,000 or more.

Additionally, within an Area of Critical Environmental Concern (ACEC), any project involving a state permit or state funding, or a project undertaken by a state agency, regardless of size (except for projects for single family dwellings)

requires filing of an ENF (301 CMR 11.03(11)). The regulations listed above are not inclusive and the applicant should consult the details listed in 301 CMR 11.00.

Authority: M.G.L. Chapter 30, Sections 61 through 62H, inclusive.

Regulations: 301 CMR 11.00.(available through the Statehouse Bookstore 617-727-5834)

Application Form: The Environmental Notification Form (ENF) - a 10 page form with questions asking for project description, descriptions of site environment and regional environment, an analysis of potential environmental impacts and mitigation measures and proposed project benefits.(301 CMR 11.28)

Timing: 1. File the ENF by the 15th or last day of the month, any time prior to, but not later than 10 days after the filing of the first application for the state permit or financial assistance for the project.

2. Secretary publishes the first page of each ENF in the Environmental Monitor, generally within 7 to 21 days after receipt. Such publication commences the ENF review period involving:
a. A twenty day public comment period.
b. A MEPA public consultation session, usually at the project site.

3. On or before the last day of the 30 day ENF review period, the Secretary issues a Certificate establishing either that no EIR is required or establishing the need for and Scope of an EIR.

4. If no EIR is required, each state agency may act as soon as it has a copy of the Certificate. If an EIR is required, the proponent must prepare and file a Draft EIR, again by the 15th or last day of a month.

5. Secretary publishes Notice of Availability of the DEIR in the Environmental Monitor within 7 to 21 days of its receipt. Such publication commences a 30 day public comment period.

6. Within seven days of the close of the 30 day public comment period, the Secretary issues a Certificate on the Adequacy of the DEIR.

7. If DEIR is determined adequate, the proponent prepares and submit the Final EIR, again by the 15th or last day of the month. If the DEIR is declared inadequate, the proponent may have to prepare a Supplemental DEIR.

8. Secretary publishes Notice of Availability of the FEIR in the Environmental Monitor within 7 to 21 days of its receipt. Such publication commences the 30 day public comment period.

9. Within seven days of close of 30 day public comment period, the Secretary issues a Certificate on the Adequacy of the FEIR.

10. If the FEIR is declared adequate, state agencies may not act on the project until 60 days after the availability of the FEIR (the date the FEIR was noticed in the Environmental Monitor) (unless there has been an appeal). If the FEIR is declared inadequate, the proponent may have to prepare a Supplemental FEIR.(301 CMR 11.04-11.10)

Inter-Agency Requirements: The MEPA process must be completed prior to the issuance of any state permit, release of state funds, or initiation of activities by a state agency.(301 CMR 11.10) If a Division of Water Pollution Control Water Quality Certification is required, the Standard Application form must be submitted with the ENF. (314 CMR 9.04).

Appeal Provisions: Notice of Intent to Commence an Action filed with the Attorney General, the Secretary and the Proponent. (301 CMR 11.20, 11.29)

Information: Massachusetts Environmental Policy Act
Executive Office of Environmental Affairs
251 Causeway St. etc.
Boston, MA 02114

II.24 MDC- METROPOLITAN DISTRICT COMMISSION (MDC) now the Department of Conservation and Recreation

Jurisdiction: Use of reservoirs, roadways, driveways, bridges, dams and land within watershed reservations under the care and control of the Metropolitan District Commission are regulated. The regulations establish general regulations relative to use of land within watershed reservations. (350 CMR 8.01) All acts which pollute the water supply are prohibited. No person shall wade or swim unless authorized. Similarly, picnicking, cooking, ball playing, etc. is only allowed where specifically posted or designated. Special regulations for Quabbin Reservoir are established at 350 CMR 8.01 (2) and specify fishing and boating regulations. Abutters seeking to perform construction or other work may need a permit from the MDC.

Authority: M.G.L. Chapter 92, sections 10-19

Regulations: 350 CMR 8.00

II.25 MHC- MASSACHUSETTS HISTORICAL COMMISSION (MHC)

Jurisdiction: Elimination, minimization or mitigation of impact to properties listed in the State Register of Historic Places. This may be applicable to lakes management and remediation projects should dredging, drawdown, or other forms of landscape modifications be required that significantly impacts an archaeological or historic site that is listed on the State Register of Historic Places. The regulations establish a standardized procedure to protect the public's interest by directing state bodies to notify the Massachusetts Historical Commission as early as possible in the planning process of any project either undertaken by the state body or prior to the state body's funding or licensing a private project. Note that underwater archaeological resources over 100 years old are also protected under 312 CMR 2.00 Board of Underwater Archaeological Resources.

Authority: Chapter 254 of the Acts of 1988 amends M.G.L. Chapter 9, sections 26-27C

Regulations: 950 CMR 71.00

Application Form: Project Notification Form (PNF filed w/ MHC) or Environmental Notification Form (ENF), depending upon whether MEPA is triggered by the project.

Permit: Determination of Adverse Effect: either No Effect, Determination of No Adverse Effect of Determination of Adverse Effect. (95-CMR 71.07)

Timing: For projects which require review under MEPA and these regulations, project proponents will find it most convenient to follow the procedures established in the regulations at the time the MEPA reports are filed. (950 CMR 71.04) The MHC will issue a written determination of effect within 30 days of receipt of an adequately documented Project Notification Form or Environmental Notification Form. (950 CMR 71.07)

Inter-Agency Requirements: Compliance with MEPA. Note that projects involving Federal agencies (401 or NPDES permits or Federal funding) may also be restricted if the site is listed, or eligible to be listed in the National Register of Historic Places.

Appeal: If the MHC issues a Determination of Adverse Effect, the MHC, the state body and the project proponent shall consult to establish alternatives to the project as proposed that could eliminate, minimize or mitigate adverse effects. Should no agreement be reached the MHC will issue a Memorandum of Agreement outlining MHC's conclusions. The Agreement will be addressed at a meeting of the MHC. Funding or licensing of a project may not proceed until this process is complete. (950 CMR 71.07)

Information: Massachusetts Historical Commission
220 Morrissey Boulevard

Boston, MA 02125
617/727-8470

FEDERAL PERMITS AND REGULATIONS

II.26 NPDES- NATIONAL POLLUTANT DISCHARGE ELIMINATION SYSTEM PERMIT, U.S. ENVIRONMENTAL PROTECTION AGENCY -(EPA)

Jurisdiction: Discharge of a point source discharge in waters of the United States. This permit is issued jointly by the EPA and the Division of Water Pollution Control. A permit is submitted to the EPA and reviewed jointly by both agencies.

Authority: Federal Clean Waters Act, section 301 and 402

Regulations: 40 CFR Parts 122-125

Application: NPDES permit application

Permit: NPDES Permit

Timing: File application at least 180 days prior to discharge

Information: Olga Vergara WCP-2109
Environmental Protection Agency
JFK Federal Bldg.
Boston, MA 02203
617/565-3529

II.27 404- U.S. ARMY CORPS OF ENGINEERS, SECTION 404 PERMIT (ACOE)

Jurisdiction: Any activity which results in the dredge and fill in waters of the United States, including wetlands and waterways. No activity is authorized within 0.25 mile of a Wild and Scenic River (parts of W.Br. Farmington, Sudbury, Assabet, Concord, Westfield Rivers). Generally, the ACOE does not require application or notification for category I projects (less than 5,000 sq. ft.) although the 401 permit and CZM review may still apply. Category II projects (5,000 sq. ft. to 1 acre) are required to submit an application for screening for a General Permit. Category III (greater than one acre) require an Individual Permit application and require a Federal EIS.

Authority: Rivers and Harbors Act, Clean Waters Act of 1977, Marine Sanctuaries Act

Regulations: 33 CFR parts 320-330

Application: Standard Application Form 33 CFR 325, in addition to a Notice of Intent under the Massachusetts Wetlands Protection Act and regulations at 310 CMR 10.00.

Permit: U.S. Army Corps Permit

Timing: Generally, a decision issued no later than 60-90 days after receipt of a completed application (33 CFR 325).

Inter-Agency Requirements: In order for the ACOE to issue a permit, the federal agency must have a 401 Water Quality Certification for the Project. In Order for a Water Quality Certification to have been issued, an Order of Conditions must have been issued for the project, either by the local Conservation Commission or by the Department of Environmental Protection. For dredging less than 100 cubic yards the Order of Conditions will serve as the Water Quality Certificate. If applicable, a Waterways license may be required (C.91). Additionally, under section 404 (b) (1), the U.S. Environmental Protection Agency has veto authority over the Corps permit process. Full consideration of Fish and Wildlife Service views is required. Additionally, compliance with MEPA, The Endangered Species Act, CZM and National Historic Preservation Act is required.

Appeal: District Court

Information: Regulatory Branch
U.S. Army Corps of Engineers
424 Trapelo Road
Waltham, MA 02254-9149
617/647-8156
800/362-4367

REFERENCES FOR APPENDIX II

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APPENDIX III -- AQUATIC HERBICIDE TOXICOLOGICAL AND ENVIRONMENTAL FATE PROFILES

Office of Research and Standards
Massachusetts Department of Environmental Protection

NOTE TO READER

The information contained in Appendix III originally was compiled in 1997. The scientific information has not been updated; however, a number of editorial changes were made in 2004, including the removal of product names from the document. The intent of this section is to focus on the active ingredient rather than the product name since product names may change frequently. For the most up-to-date information, the reader is advised to visit the Massachusetts Department of Agricultural :Resources Aquatic Vegetation Management website at:
http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html

III.0 INTRODUCTION

This appendix contains comprehensive summaries of the known toxicological and ecological effects and properties of each of the aquatic herbicides addressed in this document. These herbicides include copper, diquat, 2,4-dichlorophenoxyacetic acid, glyphosate, fluridone, endothall, aquashade and triclopyr.

The profile for each compound includes information on uses and applications of the various herbicides, mechanism of action, environmental fate/transport, pharmacokinetics, toxicological effects, available toxicity criteria and ecological toxicity. The information summarized in these profiles was obtained from a variety of sources, including U.S. Environmental Protection Agency (EPA) summaries as well as information from manufacturers.

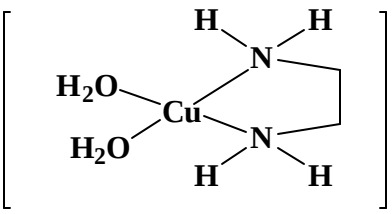
Much of the toxicological information presented for each chemical is derived from the results of studies conducted by the manufacturer to support a request to register a particular herbicide with EPA. As part of the registration process, EPA requires that laboratory animal studies addressing a number of toxicological endpoints be submitted including acute and chronic toxicity, eye and skin irritation, skin sensitization, neurotoxicity, teratogenicity, reproductive toxicity, mutagenicity and oncogenicity (carcinogenicity) as well as studies addressing the metabolism of the compound. EPA then identifies any data gaps in the submitted information and may require the manufacturer to conduct additional studies. As part of the process to register a compound for aquatic use, EPA also requires that the herbicide manufacturers submit a number of environmental fate studies addressing the following endpoints: hydrolysis, photodegradation, biodegradation, adsorption, volatility and dissipation studies.

In addition, EPA requires the manufacturer to submit the results of a series of studies addressing ecological toxicity to fish, aquatic invertebrates and birds. Required tests include acute toxicity tests (i.e., LD50s and LC50s) with the possibility of longer-term lifecycle studies and bioaccumulation studies in fish on a case-by-case basis if EPA determines it to be necessary. More specific information on EPA's data submission requirements for registration can be found in 53 FR 15993 of the Code of Federal Regulations.

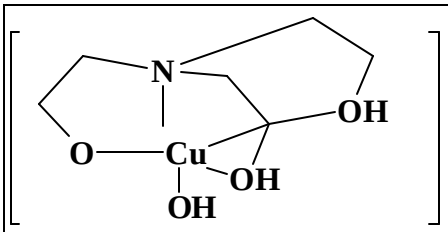
III.1 COPPER and COPPER COMPLEXES

$CuSO_4$

COPPER SULFATE



COPPER ETHYLENEDIAMINE COMPLEX



COPPER TRIETHANOLAMINE COMPLEX

SUMMARY

Copper is an element used as an aquatic herbicide in several different formulations. All products have been used to control algae and other aquatic vegetation in slow-moving or quiescent bodies of water including golf courses, ornamental fish and irrigation ponds, lakes and rivers (WSSA, 1994).

Copper is naturally occurring and is found in soil and water often in the form of complexes, both organic and inorganic. Removal of elemental copper from an aqueous system occurs through binding to sediments and dissolved organic compounds.

Copper is an essential element in humans, animals and plants, but in high enough concentrations it can be harmful to biota. Toxicity of copper is related to water hardness; copper in water with a hardness of less than 20 mg/l $CaCO_3$ is more toxic to fish than copper in the water at a greater hardness.

Copper complexes have been developed that serve to decrease the availability of the copper ion in the water column through chelation. Ionic copper in solution normally complexes with carbonates. The chelated copper complexes prevent these copper carbonates from forming, thereby decreasing the toxicity to non-target organisms including humans and fish (Ross and Lembi, 1985). Four chemical formulations of copper discussed in this report include copper sulfate, a mixed ethanolamine complexan ethylene diamine complex) and a triethanolamine complex. There is little information available for the formulated products of copper primarily because the USEPA only recently began requiring toxicity and environmental fate and persistence data on these products (Orr, pers. comm., 1995). These formulations contain varying percentages of copper along with a variety of proprietary inert ingredients. Data on the copper ion, considered to be the active ingredient in all of the formulations, are available.

The copper ion (copper II) is responsible for the toxicity of all of the formulations. Although the mechanism has not unequivocally been elucidated, it is believed that high levels of copper interfere with photosynthesis. Inhibition of photosynthesis leads to plant death.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing copper that are registered in Massachusetts can be accessed at http://www.mass.gov/eea/agencies/agr/pesticides/aquatic-vegetation-management.html at the Department of Agricultural Resources (DAR) Aquatic Herbicide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

COPPER AND COMPLEXES USES AND APPLICATIONS

Copper and chelated copper complexes are used to control the growth of algae and other aquatic vegetation that are considered a nuisance. The carbonate activity of the water in which it is found greatly influences copper sulfate activity. Copper sulfate ionizes in water. The ionic copper forms complexes with carbonates which precipitate out of solution. Carbonate concentration (or hardness) is directly related to alkalinity. Thus, in harder, more alkaline waters the copper rapidly precipitates out of solution which reduces the toxicity to algae. Alternatively, in more acidic, softer waters most of the copper stays in solution for a longer period of time. Thus, copper sulfate is generally more effective in soft water (although it is also more toxic to fish in soft water). Chelated copper complexes prevent the precipitation of copper from solution and therefore are longer lasting, particularly in harder, alkaline waters (Ross and Lembi, 1985).

Copper sulfate is used primarily to control algal growths in impounded waters, lakes, ponds, reservoirs and irrigation and irrigation drainage conveyance systems. In impounded waters, copper sulfate can be applied by spraying the water surface from a boat. Finer crystals can be dusted on the water surface. Large crystals can be put into a burlap bag and towed behind a boat. In irrigation conveyance systems, large or granular crystals are dumped into the ditch at required dosages at intervals, or for pondweed control, finer crystals are continuously metered into the flowing water by a specially designed feeder.

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/.

MECHANISM OF ACTION

High levels of copper inhibit the growth of algae and other plants by causing an imbalance in cofactors involved with enzyme function, especially those involved with photosynthesis. Inhibition of photosynthesis leads to a diminished ability to thrive and eventually to plant death (WSSA, 1994).

ENVIRONMENTAL FATE/TRANSPORT

The environmental fate and persistence of copper is dependent upon how it is distributed in the environment. Copper occurs naturally in the aqueous environment in the +1 oxidation state, also called the cuprous ion and in the +2 oxidation state, also called the cupric ion (WSDOE, 1992). The +3 oxidation state is uncommon and found only in the solid state. Copper is a transition metal which means it has the ability to form coordination complexes with a number of ligands including clays and humic substances, the amine and sulfhydryl groups of proteins, ethanolamines and ethylene diamine (EDA). These complexes are highly water soluble (WSSA, 1983) and largely of low toxicity (USEPA, 1980). The amount of copper that is bound to these compounds is dependent on the pH, metal concentration and the humic content of the water. Table III.1-2 (at the end of the copper section) lists selected

physicochemical characteristics of formulations of copper sulfate, copper ethylene diamine complexes, copper triethanolamine complexes and copper ethanolamine complexes.

Since copper is an element, biodegradation does not occur in aquatic ecosystems. Copper in aquatic ecosystems can be present in soluble form or it may be associated with particles in such ways as sorption, chelation, co-precipitation and biological accumulation by plankton. Removal from an aqueous phase occurs primarily through the adsorption of copper to sediments and is therefore not removed from the environment (WSSA, 1983). Copper can remain in the environment indefinitely if it is not physically removed. Types and kinds of organic matter in an aqueous system are important determinants of the free copper concentrations (WSDOE, 1992). Dissolved aqueous copper half-lives were reported from studies done in six Manitoba lakes (Wageman and Barica, 1979). In five of the six lakes the half-lives were between 1 and 2 days. In the other lake the half-life was 7 days.

Copper bioconcentrates in some aquatic organisms as shown in Table III.1-1.

Table III.1-1. Copper Bioconcentration Factors for Several Aquatic Species

ORGANISM	BCF
Hard shell clam (*Mercinaria mercinaria*)	88[1]
Green alga (*Chlorella vulgaris*)	2,000[1]
Fathead minnow (*Pimephales promelas*)	290[1]
Water flea (*Daphnia magna*)	1200 to 7100[2]

1 USEPA, 1980
2 Winner, 1985

PHARMACOKINETICS

Absorption of ingested copper occurs mainly in the upper portion of the gastrointestinal tract, with appearance of copper bound to albumen and amino acids in the blood within 1-2 hours of administration. The absorbed copper accumulates in the liver reducing blood copper levels. Blood copper levels then begin to rise slowly as a result of the hepatic production and release of copper-bound ceruloplasmin. Copper is also incorporated into several other proteins including the enzymes cytochrome oxidase, monoamine oxidase, tyrosinase and erythrocyte superoxide dismutase. Low molecular weight copper complexes are excreted rapidly with increasing amounts of high molecular weight copper complexes being formed over time. Copper elimination occurs primarily by the fecal route with relatively little excreted through urine, sweat or menstrual fluid (USEPA, 1987).

HEALTH EFFECTS

Mammalian:

While limited information on the pharmacokinetics and health effects of elemental copper exists, there is very little information on these subjects for the copper complexes. Since copper is considered to be the active component of the copper complexes, a summary of the toxicity of elemental copper is presented here.

Copper

Copper is an essential element in humans and other animals therefore most of the adverse health effects associated with copper are a result of its deficiency. Efficient homeostatic mechanisms generally protect mammals from the adverse effects of copper excess (USEPA, 1987). Copper is a component of enzymes that are vital in hematopoiesis, maintenance of vascular and cellular integrity and structure and function of the central nervous system (O'Dell, 1976 as cited in USEPA, 1987). Several enzymes use copper as a coenzyme including tyrosinase, cytochrome oxidase, superoxide dismutase and amine oxidase. Menke's disease and Wilson's disease are two genetic diseases associated with the increased sequestration of copper in brain and other tissues. Infants and children have increased susceptibility to the toxic effects of copper probably because of the normally high hepatic copper levels in early life and the fact that homeostatic mechanisms are not fully developed at birth.

Acute:

Thirteen of 53 patients died after ingesting 6-637 mg/kg copper in attempted suicides. These doses are much higher than doses from ingestion of water containing copper applied at recommended label application rates. Cause of death was reported to be shock and liver and/or kidney complications (Chuttani *et al.*, 1965 as cited in USDHHS, 1990). Increased mortality was reported in rats fed 4,000 ppm of copper for 1 week (Boyden *et al.*, 1938 as cited in USDHHS, 1990) and in weanling rats exposed to 6,000 ppm for 2 weeks (Haywood, 1985 as cited in USDHHS, 1990). The deaths were attributed to liver effects (i.e., centrilobular necrosis). Acute systemic effects observed upon consumption of contaminated water or copper sulfate in attempted suicides include gastrointestinal, liver and kidney effects (USDHHS, 1990). In animals, in addition to the ones noted in humans, effects on the blood, musculoskeletal and cardiovascular systems were also noted as well as changes in body weight (USDHHS, 1990).

Subchronic/Chronic:

Copper produces liver damage as the liver is the main storage depot for copper. The specific mechanism of liver toxicity has not been elucidated, but administration of copper compounds to laboratory animals has resulted in hepatocellular necrosis, regenerative activity, cirrhosis, Kupffer cell mobilization and hepatocellular pigment formation (Barka *et al.*, 1964 as cited in USEPA, 1987). Excessive subchronic ingestion of copper also produced kidney damage in rats (Rana and Kumar, 1980; Haywood, 1980 as cited in USEPA, 1987) but this only occurred after the liver began to accumulate high levels of copper (Haywood, 1980 as cited in USEPA, 1987). Blood levels of copper rise after copper accumulates in the liver.

There are few chronic copper toxicity studies available (except for in ruminant animals) and those that are available are characterized by serious experimental flaws (USDHHS, 1990). Ingestion of 150 mg copper/kg/day (i.e., 500 ppm dietary copper) by rats for 1 week resulted in no observable effects (e.g., no liver accumulation and no adverse kidney or liver changes). Administration of this dose to rats for six weeks caused severe kidney and liver damage in rats. Continued administration of this dose for up to 15 weeks resulted in no further damage but instead produced a regeneration of liver and kidney tissues (Haywood, 1980 as cited in USEPA, 1987). Liver and kidney necrosis occurred in rats fed 25.4 mg copper/kg/day for 20 days (Rana and Kumar, 1980 as cited in USEPA, 1987).

In rats fed 500 ppm copper in the diet for 27 days (Boyden, et al, 1938 as cited in USEPA, 1987) and 50 ppm copper for 35 days (Miranda *et al.*, 1981 as cited in USEPA, 1987), increased liver copper concentrations were noted. Higher levels resulted elevated copper levels in the liver and spleen, growth reduction and reduced dietary intake, resulting in death (Boyden *et al.*, 1938 as cited in USEPA, 1987).

Adverse effects were reported in pigs given copper supplements of 600 ppm in the diet for 48 days and 250 ppm for 79 days (Kline *et al.*, 1971 as cited in USEPA, 1987) whereas beneficial effects were reported in pigs dosed with 150-200 ppm for 61-88 days. A dose of 500 ppm administered in the diet for 61 days caused adverse effects including growth reduction, reduced hemoglobin and increased hepatic copper (Kline *et al.*, 1971 as cited in USEPA, 1987).

Developmental/Reproductive:

Copper compounds produced teratogenic effects at approximately 2 mg copper/kg when injected into female hamsters on the eighth day of pregnancy (DiCarlo, 1980; Ferm and Hanlon, 1974 as cited in USEPA, 1987); however, injection is not a normal route of exposure so these results do not provide any conclusive information on effects via oral exposures. In mice fed greater than 104 mg copper/kg/day as copper sulfate, increased fetal mortality was observed; developmental abnormalities were observed at greater than 155 mg copper/kg/day (Lecyk, 1980 as cited in USDHHS, 1990). In mink administered dietary copper sulfate levels greater than 3 mg copper/kg/day, an increased mortality rate in offspring was noted. 3 mg/kg/day was identified as the LOAEL for developmental effects in mink (Aulerich, *et al.*, 1982 as cited in USDHHS, 1990). A significant increase in testes weight was observed in rats treated with 130 mg copper/kg/day as copper acetate administered via the diet (Llewellyn *et al.*, 1985 as cited in USDHHS, 1990). A NOAEL of 13 mg/kg/day for reproductive effects was reported for mink (Aulerich *et al.*, 1982 as cited in USDHHS, 1990).

Mutagenicity:

Copper was generally found to produce negative results in microbial mutation assays. Low concentrations of copper have produced some mutagenic activity in cell culture assays. Copper sulfate produced an increase in the frequency of recessive lethal mutations in *D. melanogaster* at high concentrations (Law, 1938 as cited in USEPA, 1987).

Carcinogenicity:

Limited information available on the carcinogenicity of copper is equivocal. Tumors were induced in mice administered copper by subcutaneous injection (BRL,1968 as cited in USEPA, 1987). Copper-induced cancer was not reported to occur in rats or mice exposed to copper at dietary concentrations ranging from 5-1,000 mg/kg/day (Greene *et al.*, 1987, Kamamoto *et al.*, 1973 as cited in USDHHS, 1990). Based on the limited information, the U.S. EPA Office of Pesticide Programs (OPP) has designated copper as a Group E carcinogen under the old EPA cancer classification system. Under the new EPA classification system using descriptors, this classification corresponds to a descriptor of "Data are inadequate for the assessment of human carcinogenic assessment".

Other Information on Specific Copper Compounds:

Copper Sulfate

The toxic properties of copper sulfate are mainly attributable to its caustic properties (NRC, 1977). Symptoms of acute copper toxicity include metallic taste in the mouth, burning epigastric pain, vomiting, diarrhea, nausea and depending on the severity, jaundice, hemolysis, blood and urine effects. In severe cases, anuria, hypotension and coma can occur (USEPA, 1980). Skin contact with copper sulfate results in eczema; contact with the eyes causes conjunctivitis, edema of the eyelids, and ulceration and turbidity of the cornea (Patty, 1963). One infant fatality was reported following exposure to copper sulfate at a concentration of 6.75 mg/l in drinking water for 14 months (NRC, 1977).

Copper Ethanolamine Complexes

The acute oral LD50 in rats for a formulation of copper ethanolamine complexes has been reported to be between 650 and 2420 mg/kg (Applied Biochemists, 1994; WSSA, 1994). An oral LD50 has been reported in rats of 498 mg/kg. In rabbits, the acute dermal LD50 is greater than 2,000 mg/kg (WSSA, 1994).

Copper Triethanolamine Complexes

In an acute inhalation study done in Sprague-Dawley rats exposed to a formulation of copper triethanolamine complexes, LC50 values of 0.27 mg/l for male rats and 1.15 mg/l for female rats were derived (Griffin Corporation, 1992). An oral LD50 of 1200-2400 mg/kg and a dermal LD50 of 9600 mg/kg were reported in rabbits (WARF Institute, 1972; WSSA, 1994).

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) has developed a health-based Maximum Contaminant Level Goal (MCLG) of 1.3 mg/l for copper in drinking water. The EPA determined that it would not be feasible to set a Maximum Contaminant Level (MCL) standard for copper and therefore they established an Action Level of 1.3 mg/l accompanied by a treatment approach involving corrosion control, source water reduction, public education and copper service line replacement designed to achieve the public health goals of the Safe Drinking Water Act. The EPA also established a secondary MCL of 1 mg/l based on odor and taste considerations of drinking water (USEPA, 1992).

ECOLOGICAL TOXICITY

Aquatic Organisms :

Invertebrates:

The toxicity of copper to aquatic invertebrates generally decreases as hardness increases. Additional data for several species indicate that toxicity also decreases with increases in alkalinity and total organic carbon (USEPA AWQC, 1980). There is a wide range of acute toxicity responses (LC50, EC50) for invertebrates. Concentrations for crustaceans ranged from 5 to 300 µg/l; for annelids, the range was from 6 to 900 µg/l; and for mollusks the range was 40 to 9,000 µg/l (WSDOE, 1992). In another study, acute LC50 values ranged from less than 10 to 9,000 µg/l in crustaceans and from 39 to 2600 µg/l in mollusks (Harrison, 1985).

There is a large body of data for the fresh water crustacean *Daphnia* spp. In four species of *Daphnia*, LC50 values ranged from 68 to 87 µg/l (Speara and Pierce cited in Harrison, 1986). *D. magna* are capable of developing tolerance to copper but *D. pulex* are not (LeBlanc, 1985 in Harrison, 1986). However, *D. pulex* can be sensitized to copper toxicity (LeBlanc, 1985).

Four species of *Daphnia* experienced decreased survivorship when exposed to 0.040 mg/l of copper under laboratory conditions using a static method with water at 100-119 mg/l alkalinity, 130-160 mg/l hardness and 8.2-9.5 mg/l of dissolved oxygen (WSDOE, 1992). In a continuous flow bioassay (41 mg/l hardness, pH 7.7 and 43 mg/l alkalinity), the survival of the snail (*Physa integra*), amphipod (*Gammarus pseudolineus*) and operculate snail (*Campeloma decisum*), was reduced at 0.0148 and 0.028 mg/l copper (Arthur and Leonard, 1970). No growth inhibition was observed at 0.008 mg/l or less.

Vertebrates:

The copper (II) ion is believed to be the toxic form of copper to fish (WSDOE, 1992). The amount of the copper (II) form is dependent upon pH and therefore copper toxicity is related to pH (Chapman, 1977).

In addition, several studies have demonstrated that copper toxicity is related to water hardness. In water with a hardness of approximately 20 mg/l (as calcium carbonate), copper concentrations of 0.040 mg/l are reported to be toxic to salmonid eggs, fry, fingerling, juveniles and adults (Chu *et al.*, 1978). Fish tested in water with concentrations of $CaCO_3$ greater than 20 mg/l (harder water) were less sensitive.

There are several studies available that characterize toxicity of copper to fish. Large differences are seen in the sensitivities of different species to copper. Acute toxicity (48h to 96h LC50 or EC50) data for freshwater fishes range from 10-900 µg/l for Salmonidae, 700-110,000 µg/l for Centrarchidae and 20-2,000 µg/l for Cyprinidae (WSDOE, 1992).

In bluegill sunfish, LC50 values for a formulation of copper triethanolamine complexes ranged from 1.2 mg/l in soft water to 7.5 mg/l in hard water. In channel catfish the 96 hour LC50 value of 6 mg/l has been reported. The 96 hour LC50 value for rainbow trout is less than 0.2 mg/l in soft water and 4 mg/l in hard water (WSSA, 1994). Specific toxicity information for copper triethanolamine formulations was not found.

Death in fish from acute exposures may be due to the disruption of the respiratory process caused by damage to the gill epithelium. The effects of copper sulfate and copper nitrate were studied in the chinook salmon (Holland *et al.*, 1960). At concentrations of 0.178 to 0.318 mg/l between 42 and 96 hours, fifty percent mortality was reported. Total kills occurred in 18 hours when fish were exposed to 1 mg/l of copper and in less than 42 hours at concentrations of 0.563 mg/l.

Available Toxicity Criteria:

The EPA has developed a number of Ambient Water Quality Criteria (AWQC) for copper for both acute and chronic exposures of both freshwater and marine organisms. These include freshwater and marine acute values of 9.2 µg/l and 2.9 µg/l respectively as well as a freshwater chronic value of 6.5 µg/l (USEPA, 1992). (All of the AWQC values are dependent on the hardness of the water.)

Plants:

Copper has been used to control nuisance vegetation for many years. Concentrations of copper of 1-2 µg/l inhibit photosynthesis and plant growth (WSDOE, 1992). Toxicity data for individual plant species are lacking (USEPA, 1980).

The effects of pH on the toxicity of copper to algae can be important. Laboratory results demonstrated that changes in metal toxicity with pH resulted from competition between the hydrogen ion and the copper II ion for cellular binding sites at the lower pH range. At higher pH copper was still toxic because of the decreased competition of the hydrogen ion (WSDOE, 1992).

Table III.1-2. Physicochemical Properties of Formulations of Various Copper Compounds

Parameter	Copper Sulfate	Copper Ethylene Diamine Complex[1]	Copper Triethanolamine Complexes[2]	Mixture of Copper Carbonate, Monoethanolamine and Triethanolamine
Molecular Formula	$CuSO_4$	$C_2H_{12}CuN_2O_2$	$C_6H_{15}CuNO_4$	Proprietary
Molecular Weight (g/mol)	161.39	255.73	228.74	n/a
Density (mg/l)	2.28 (Sax)	1.22	1.20	1.1-1.3
Boiling Point (degrees C)	n/a	102	n/a	212
Water Solubility	completely miscible	completely miscible	completely miscible	completely miscible

n/a not applicable

1. WSSA, 1994.
2. Applied Biochemists, 1995.

Copper References

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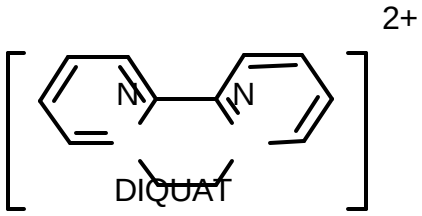
III.2 DIQUAT



SUMMARY

Diquat (6,7-dihydrodipyridol[1,2-a:2',1'-c] pyrazinediium ion) is a water-soluble contact type, nonselective herbicide that is used to control many submerged and floating aquatic macrophytes and some types of filamentous algae in static and low-turbidity water (Klingman, Aston and Noordhoff, 1975 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat binds very strongly and rapidly to sediments and once bound, it is very persistent (Reinert and Rodgers, 1987). When used as an aquatic herbicide at recommended application rates, diquat residues in water decrease rapidly to essentially undetectable levels within 7-14 days (State of Washington, 1984). The rate of diquat bioconcentration in fish is negligible (Reinert and Rodgers, 1987).

The common name, diquat, refers to the cation, which is responsible for the herbicidal action of the salt. The associated anion (i.e., bromide) has no effect on the herbicidal activity.

Many studies have been conducted using diquat addressing both toxicity and environmental fate and persistence. The EPA approved a Reregistration Eligibility Decision (RED) for diquat dibromide in July, 1995.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing diquat that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html on the DAR website for updates on this list. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

DIQUAT USES AND APPLICATION

~~Diquat can be used to control both s~~ubmerged and floating weeds. For submerged weeds, the diquat can be injected below the water surface or it can be applied directly into the water while moving slowly over the water surface in a boat. For floating plants, the foliage should be thoroughly wetted with diquat using either surface or aerial spraying (Herbicide Handbook, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988). Turbid or muddy water or mud-coated vegetation greatly reduces the effectiveness of diquat as the herbicide becomes adsorbed to particles (Aquatic Plant Identification and Herbicide Use Guide, 1988). Improved efficacy of diquat can often be achieved when applied in a mixture with complexed copper formulations (Aquatic Plant Identification and Herbicide Use Guide, 1988). In some cases it is recommended that diquat be applied with water carrier, thickener or invert emulsion carrier. The following adjuvants are recommended for use with diquat: for aerial applications, a nonionic surfactant to improve the ability of diquat to penetrate waxy plant cuticles (e.g., Ortho X-77 Spreader); for submersed growth, a polymeric thickener to improve sinking, herbicide confinement and contact properties (e.g., Nalquatic) (Aquatic Plant Identification and Herbicide Use Guide, 1988).

A formulation of diquat dibromide aquatic herbicide) targets the list of aquatic plants in Table III-2-1 (Zeneca, 1994). This herbicide controls the aquatic plants in Table III 2-1.

Diquat can be used at anytime during the growing season although control of early growth is recommended. Treatment of dense weed areas may result in oxygen loss from decomposition of dead weeds. The loss of oxygen may cause fish suffocation. Therefore, treat only 1/3 to 1/2 of the dense weed areas at a time and wait 14 days between treatments (Zeneca, 1994).

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. A list of the weeds that these products control, which has been compiled from the Environmental Protection Agency (EPA) registration labels for these products, is contained in Table III.2-1.

MECHANISM OF ACTION

Diquat's herbicidal activity and organic chemical reactions of diquat formulations are dependent only on the diquat cation and are not influenced by the nature of the associated anion, since the salts are mostly dissociated in aqueous solution (Herbicide Handbook, 1983). Diquat is absorbed readily by foliage through the cuticle of the leaf. Absorption is rapid, resulting in concentrations in plant tissues well above that in surrounding water so that very low concentrations (i.e., 0.1-1.5 ppm) in water will give effective control (HSDB, 1994). No absorption through buried plant roots occurs due to the rapid binding and inactivation of diquat by sediments (Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat is translocated only locally in plant tissues (Aquatic Plant Identification and Herbicide Use Guide, 1988). Diquat's mode of action is not clear but it is known that the mechanism is light-dependent (USEPA, 1992a; HSDB, 1994). Diquat interferes with the photosynthetic process, releasing strong oxidizers that rapidly disrupt and inactivate cells and cellular functions (Aquatic Plants Management Program for Washington State, 1992). This action results in the rapid death of the foliar parts of practically all plant species (HSDB, 1994).

ENVIRONMENTAL FATE/TRANSPORT

The available database for diquat indicates that dissipation following application is very rapid, initially by mixing and subsequently by adsorption by plants and sediments (USEPA, 1994). Once diquat reaches the sediments, it is tightly bound and is biologically unavailable.

Diquat is stable in neutral or acid conditions but hydrolyzes in the presence of alkaline materials including alkaline waters (Herbicide Handbook, 1979 as cited in HSDB, 1994). Volatilization and oxidation of diquat are insignificant fate processes.

Diquat is subject to photochemical degradation in surface layers of water in 1-3 or more weeks when not adsorbed to particulate matter (Sanborn, 1977). A 50% loss of diquat was noted within 48 hours when exposed to a UV source (Simsiman *et al.*, 1976 as cited by Reinert and Rodgers, 1987). A photodecomposition half-life of 1.6 weeks was calculated from the results of a study in which diquat in 20-cm glass petri plates was subjected to natural sunlight (Smith and Grove, 1969 as cited in Reinert and Rodgers, 1987). Diquat has a reported photolysis half-life of 2-11 days (Reinert and Rodgers, 1987). Despite the above information, photodegradation is not considered a major fate process for diquat in aquatic environments (Simsiman *et al.*, 1976 as cited by Reinert and Rodgers, 1987).

The photochemical breakdown of diquat on plant surfaces and in water exposed to sunlight releases 1,2,3,4-tetrahydro-1-oxopyrido-[1,2-a]-5-pyrazinium ion (TOPPS) as the major degradation product.

Further irradiation produces picolinamide and then degrades further via picolinic acid to volatile fragments (Smith and Grove, 1969 as cited in Aquatic Plants Management Program for Washington State, 1992). When a solution containing 5 ppm of diquat was exposed to sunlight during May and June, 70% of the diquat was degraded in 3 weeks. Picolinic acid and TOPPS were major photodegradation products (Smith and Grove, 1969 as cited in HSDB, 1994). A secondary degradation pathway results in diones and, to a limited extent, to monopyridone (Aquatic Plants Management Program for Washington State, 1992).

The major fate process for diquat in water is its propensity for rapidly binding to sediments. This property is due to its double positively charged diquat cation and clay minerals present in soil. The diquat cation may also insert itself between the layer planes of certain minerals such as montmorillonite (Reinert and Rodgers, 1987). Diquat may also incorporate into humus and/or become physically adsorbed to organic matter and particles (Aquatic Plants Management Program for Washington State, 1992). About 80-95% of diquat introduced into a flask containing sediment/water was sorbed to the sediment within 2 days (Simsiman and Chesters, 1976). Diquat is characterized by a fairly high octanol-water (K_{ow}) partition coefficient of 603 and adsorption coefficients (K_{oc}) ranging from 205-691ml/g based on various sediment types (Reinert and Rodgers, 1987). Once bound, diquat is no longer bioavailable. See Table III.2-4 for a list of environmental parameters of diquat.

Studies have shown that unbound, biologically available diquat can be biodegraded by bacteria in the laboratory. However, because of the rapid adsorption of diquat to sediments in the environment which renders it unavailable to biodegradation, the opportunity for microbial decomposition is not very great (Calderbank, 1968 as cited in Hamer, 1994). Thus, while diquat may disappear relatively quickly from water, it does tend to persist in sediments. In one study conducted with diquat in pond water, diquat disappeared from the water within days of treatment but persisted in the sediments for over 160 days (Frank and Comes, 1967 as cited in Reinert and Rodgers, 1987). Nevertheless, it has been shown that biodegradation does occur in various sediment/water systems although at a very slow rate. After 65 days, only 0.88% and 0.21% of diquat was converted to CO_2 and water under aerobic and anaerobic conditions using water and sediment from a eutrophic lake and negligible using water from an oligotrophic lake (Simsiman and Chesters, 1976 as cited by HSDB, 1994).

Table III.2-1. List of Weeds Controlled by Diquat

Common Name	Scientific Name
SUBMERSED AQUATICS:	
Bladderwort	*Utricularia*
Coontail	*Ceratophyllum demersum*
Elodea	*Elodea* spp.
Naiad	*Najas* spp.
Watermilfoil	*Myriophyllum* spp.
Hydrilla	*Hydrilla verticillata*
Pondweeds	*Potamogeton* spp.
FLOATING AQUATICS:	
Salvinia	*Salvinia* spp.
Water Hyacinth	*Eichhornia crassipes*
Water Lettuce	*Pistia stratiotes*
Duckweed	*Lemna* spp.
Pennywort	*Hydrocotyle* spp.
MARGINAL WEEDS:	
Cattails	*Typha* spp.
ALGAE:	
Filamentous green algae	*Pithophora* spp.
	Spirogyra spp.

(Zeneca, 1994)

Diquat does not tend to bioconcentrate to an appreciable degree in fish and other aquatic organisms. No diquat residues were detected in channel catfish collected from pools five months after a single application or two months after a second treatment of 1 ppm diquat (HSDB, 1994). Diquat did not significantly accumulate in fish with bioconcentration factors of $\leq$2.5X with rapid depuration once fish are in pesticide-free water. In laboratory flow-through systems, diquat did not accumulate to a significant degree in *Daphnia*, mayfly nymphs and oysters, with maximum bioconcentration factors of 32X. Depuration was rapid for all organisms (USEPA, 1994). Reported bioconcentration factors for aquatic (non-plant) organisms range from <1 - 62 (USEPA, 1994).

When sprayed on the surface of ponds in a dissipation study conducted in Florida, diquat mixed quickly both laterally and by depth in the water column. Diquat was removed from the water column with a half-life of $\leq$ 2 weeks. Most of the recovered diquat was bound to the first five centimeters of soil, with small amounts recovered from the 5-10 cm layer. Diquat is very persistent but due to its strong soil absorptive properties, it is unlikely to be a groundwater contaminant. When applied to surface water systems, diquat will most likely be associated with the sediment (USEPA, 1994).

PHARMACOKINETICS

In rats given oral doses of ^{14}C-labeled diquat dibromide or diquat dichloride, absorption of diquat through the gastrointestinal tract was very low. About 4-11% of the original dose was excreted within 48 hours in the urine and about 84-97% of the original dose was excreted in the feces. Biliary excretion in rats administered an oral dose of diquat was less than 5% of the administered dose within 24 hours. Most of the recovered radioactivity in rats was found to be unchanged diquat. Metabolic breakdown products of diquat include diquat monopyridone and diquat dipyridone in the urine and diquat monopyridone in the feces (USEPA, 1992a).

Absorption of diquat in dogs was somewhat higher than in rats. 29-32% of the orally administered dose was recovered in the urine within 3 days after dosing. 51-62% was recovered in feces (USEPA, 1992a).

Absorbed diquat tends to preferentially accumulate in the kidney, although it was also detected in other tissues. Single oral doses of 116-230 mg diquat ion/kg/day of diquat in dogs yielded diquat tissue concentrations of less than 3 µg/g and kidney concentrations of up to 10 µg/g after 4 hrs. Four to 48 hours after the dose was administered, the diquat residues decreased (USEPA, 1992a).

In an 8-week feeding study with rats administered 12.5 mg diquat ion/kg/day, tissue concentrations of diquat were less than 1 µg/g in the brain, liver, lung, stomach and small and large intestines. During the latter part of the experimental exposure period, diquat concentrations in the kidney and large intestine increased to greater than 1 µg/g. Within one week of return to a control diet, no diquat was detected in any tissue (USEPA, 1992a).

In rats given 116-125 mg diquat ion/kg/day, absorbed diquat was relatively uniformly distributed among tissues. At 2-30 hours postexposure, concentrations were slightly higher in the kidney than in other tissues. In rats given an oral dose of 231 mg diquat ion/kg/day, elevated levels of diquat were found in heart and lung tissue 2 hours after dosing but by 24 hours these levels had decreased and the levels in the kidney had increased between 24-48 hours. *In vitro* studies indicate that diquat accumulates in the kidney but not in the other tissues (USEPA, 1992b).

HEALTH EFFECTS

Avian:

A series of lethal doses and lethal concentrations of diquat were identified for birds in acute toxicity studies. A number of these have been summarized below:

Table III.2-2. Acute Toxicity Studies with Diquat in Birds

SPECIES	TYPE	RESULTS	REFERENCE
3-4 mo. old mallard chicks	oral LD50	564 mg/kg	USDWFS, 1984
mallard	oral LC50	>5,000 ppm	USDIFWS, 1975
bobwhite chicks	oral LC50	2932 ppm	USDIFWS, 1975
14-day old Japanese quail chicks	oral LC50	1346 ppm	USDIFWS, 1975
10-day old ring-necked pheasants	oral LC50	3742 ppm	USDIFWS, 1975

Mammalian:

Acute:

Symptoms of diquat poisoning include vomiting, diarrhea, general malaise, possible kidney and liver damage, dyspnea and pulmonary edema. Tremor and convulsions may occur with very large doses (Herbicide Handbook, 1979 as cited in HSDB, 1994). Workers who have skin contact with concentrated diquat solutions have shown a change in color and softening of one or more fingernails. Inhalation of dust or mist of the compound has led to nosebleeds and the mists may also cause skin irritation, irritation of the mouth and upper respiratory tract, cough and chest pain (Booth and McDonald, 1982 as cited in HSDB, 1994). Ingestion of concentrated solutions of diquat can cause severe irritation to the mucous membranes of the mouth, pharynx, esophagus and stomach. Ulceration and perforation may follow (Arena, 1979 as cited in HSDB, 1994).

Diquat is known to have a profound effect on the distribution of body water. Oral exposure with diquat increases gastrointestinal water content and results in hemoconcentration (USEPA, 1992a). Ingestion of diquat results in dehydration and gastrointestinal ulceration resulting in the vomiting of blood. Acute tubular necrosis of the kidney has also been reported resulting in anuria and increased blood levels of BUN and creatinine (USEPA, 1982 as cited in HSDB, 1994). Dehydration usually plays a key role in causing death from ingestion of diquat (USEPA, 1992a). Diquat administered subcutaneously is expected to be up to 20 times more toxic than via the oral route (USEPA, 1992a).

A number of cases of acute diquat poisoning in humans were reported in the literature. Of ten cases involving ingestion of diquat, six resulted in death. All six cases involved ingestion of at least 15 ml diquat and were characterized by clinical symptoms of toxicity involving the gastrointestinal tract, the brain and the kidney. The quantities ingested by these individuals were much higher than the amounts individuals swimming in waters treated at recommended application rates would ingest or absorb. In the remaining cases, which were characterized by ingestion of no greater than 5 ml diquat, no deaths occurred but gastrointestinal and renal tract damage was observed (USEPA, 1992a).

The acute oral toxicity of diquat in mammals is moderate. Reported acute toxicity values for diquat in mammals include an oral LD50 of 430 mg diquat ion/kg/day in the rat and >26 mg diquat ion/kg/day in the dog. These relatively high levels are attributed to the poor absorption of diquat through the gastrointestinal tract (USEPA, 1992a).

Rats exposed to 100-200 mg/kg diquat ion/kg had minor histopathological changes in the gastrointestinal tract, kidney and liver. An oral Lowest Observed Adverse Effect Level (LOAEL) was determined to be 18.4 mg diquat ion/kg for a single dose of diquat (based on an increase in water content in the gastrointestinal tract) (USEPA, 1992a).

Monkeys that died after being exposed to 100-400 mg of diquat ion/kg showed distinct exfoliation of the gastrointestinal tract epithelium and distinct pathological changes in kidneys (USEPA, 1992a).

Rats that were administered an LD50 dose of 166 mg diquat ion/kg were lethargic, showed signs of piloerection and weight loss, uncharacteristic, off-color feces, gross abdominal swelling, muscular twitching, erratic gait and, the most notable effect, an increase in gastrointestinal water content and hemoconcentration (USEPA, 1992a).

At LD50 doses of 100-200 mg diquat ion/kg in dogs and 100 mg diquat ion/kg in rabbits, perforation of the stomach wall was noted (USEPA, 1992a).

A single oral dose of 99 mg diquat ion/kg produced a marked decrease in renal excretory function. At 166 mg ion/kg, hemoconcentration and a significant reduction in renal plasma flow were observed. At LD50 levels, minimal pathological changes in the kidney were observed in rats at LD50 dose levels. Researchers have concluded that effects on kidney function observed after exposure to diquat are mainly due to body fluid redistribution. Pathological changes were observed in kidneys of monkeys receiving single oral doses of 100-400 mg diquat ion/kg (USEPA, 1992a).

In rats receiving acute lethal doses of diquat intraperitoneally and in monkeys receiving oral doses of diquat, minimal effects on the liver were noted. An increase in liver glycogen and blood glucose appeared to be mediated by altered adrenal secretion. Selenium-deficient rats, given 3.6 mg/kg diquat via intraperitoneal exposure were characterized by rapid and massive liver necrosis accompanied by a marked increase in hepatic liver peroxidation (USEPA, 1992a). A series of other acute toxicity studies conducted with various species yielded the toxicity values summarized in Table III 2-3.

Subchronic:

In a 4-week dietary study, a No Observed Adverse Effect Level (NOAEL) of 6.7 mg diquat ion/kg/day was identified in Charles River CD female rats (USEPA, 1992a).

Oral exposure of rats with either 2.1 or 4.3 mg/kg/day of diquat for four and one-half months produced lung damage characterized by apparently dose-related papillomatous proliferations of the bronchial and bronchiolar epithelia. In addition, moderate to severe alveolar damage was produced in mice exposed either intratracheally or intraperitoneally (USEPA, 1992a).

Table III.2-3. Acute Toxicity Studies With Diquat

SPECIES	TYPE	RESULTS	REFERENCE
Rabbit	dermal LD50	>750 mg/kg	Hartley and Kidd, 1983 as cited in HSDB, 1994
Cattle	oral LD50	30 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
guinea pig	oral LD50	100 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Mouse	oral LD50	106-146 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Rabbit	oral LD50	72-138 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994
Rat	oral LD50	194-274 mg/kg	Clark and Hurst, 1970 as cited in HSDB, 1994

In rats exposed to 500 and 1,000 mg/l diquat in drinking water for 20 and 8 days respectively and in rabbits exposed to 100 and 500 mg/l for 6 and 10 days respectively, no irritation of the digestive mucosa was noted (USEPA, 1992a).

Chronic:

Chronic feeding studies conducted in dogs, guinea pigs and rats resulted in the formation of cataracts. Cataract formation is cited as the most sensitive toxic indicator of diquat exposure. Diquat-induced formation of cataracts was found to be both dose and time-dependent in laboratory animals. In rats exposed for two years or longer to doses as low as 1.8 mg diquat ion/kg/day, a high frequency of cataract formation was noted. The minimal effective dose in rats was 2 mg/kg/day in drinking water. In a two-year study conducted with rats, cataracts were noted in animals exposed to 3.28 and 17.16 mg diquat ion/kg/day. A NOAEL of 0.22 mg/kg/day was identified for rats in this study. In another study in rats exposed to concentrations up to 36 mg diquat ion/kg/day, an extensive examination of hematology, urinalysis and gross and microscopic pathological examination showed no effects (other than in the eye) at any treatment level. Animals exposed to higher doses experienced more serious effects sooner. No effects were noted in rats exposed to 0.36 mg diquat ion/kg/day. No cataracts were noted in dogs exposed to 1.2 mg/kg/day for 4 years or at a dose level of 0.58 mg diquat ion/kg/day for 3 years. A LOAEL of 3.6 mg diquat ion/kg/day for dogs was identified from this study . A NOAEL for guinea pigs was identified in another study as 0.1 mg diquat ion/kg/day (USEPA, 1992a).

Developmental/Reproductive Effects:

No reproductive or teratogenic effects were observed in mice, or rats after oral diquat administration. In a mouse study in which animals received five daily oral doses of 10 mg diquat ion/kg as well as in a three-generation rat study in which animals were given 25 mg diquat ion/kg, no reproductive effects were reported in the parental, F_1 or F_2 generations. No significant teratogenic effects were observed in mice, rats or rabbits. However, teratogenic effects were produced with diquat administered intraperitoneally or intravenously. In rats administered a single intravenous dose of 8 mg diquat ion/kg, an increase in the number of dead and resorbed fetuses was observed. In addition, skeletal abnormalities were found in the embryos of mice exposed to 1.4 and 5.9 mg diquat ion/kg and rats exposed to 7.5 mg diquat ion/kg after treatment of dams with single intraperitoneal doses (USEPA, 1992a).

Mutagenicity:

The potential mutagenicity of diquat was tested in a number of bacterial and eukaryotic systems with contradictory results. Both positive and negative results were found in *Salmonella* assay, unscheduled DNA synthesis and mitotic gene conversion assay. Diquat induced recessive lethal damage in *Aspergillus* but not in *Drosophila* (USEPA, 1992a).

Carcinogenicity:

In four feeding studies conducted in rats and one in mice (in which only doses of up to 75 mg diquat ion/kg were given for periods of up to two years) no tumors were detected. However, two of these studies had insufficient data upon which to base any final conclusions. Under the old EPA carcinogen classification system, the U.S. EPA OPP determined that diquat was an E carcinogen (i.e., having evidence of noncarcinogenicity in humans) based on a lack of tumor production in rats and mice. Under EPA's current classification system involving the designation of descriptors for summarizing weight-of-evidence, the old E designation corresponds to the descriptor, "not likely to be carcinogenic to humans".

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) Carcinogen Risk Assessment Verification Endeavor (CRAVE) RfD/RfC Workgroup has developed an oral Reference Dose (RfD) of 0.0022 mg/kg/day for diquat based upon a 1985 2-year dietary rat study. The RfD is an estimate, (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1992b). The World Health Organization (WHO) has also developed an RfD of 0.002008 mg/kg/day. The EPA Office of Pesticide Programs (OPP) has developed an RfD of 0.005 mg/kg/day based upon a 1-year feeding study in dogs. (USEPA, 1995).

In addition, the EPA has also developed a Maximum Contaminant Level Goal (MCLG) of 0.02 mg/l for drinking water and has promulgated this value as a Maximum Contaminant Level (MCL) standard (USEPA, 1992b; USEPA, 1995b). Massachusetts has adopted this value as a drinking water standard, known as a Massachusetts Maximum Contaminant Level (MMCL).

ECOLOGICAL TOXICITY

Aquatic Organisms:

Acute flow-through type bioassays have been conducted with a variety of freshwater and marine fish and invertebrates. Because dissipation of diquat is very rapid, a comparison of toxicity data generated in the laboratory to expected diquat concentrations following application and dissipation in the field, indicates that acute effects on organisms in the field are unlikely at rates used for vegetation control (Hamer, 1994; MacKenzie, 1971 as cited in Aquatic Plants Management Program for Washington State, 1992).

Invertebrates:

LC50 toxicity values for invertebrates show a range of sensitivities to diquat. The most sensitive organisms tested were *Daphnia* and *Hyalella* with 24-hour LC50 values of 1-2 ppm and 0.6 ppm, respectively (Hamer, 1994). Studies of estuarine organisms in Florida, showed no adverse effects on oysters, shrimp or fish (Wilson and Bond, 1969 as cited in Aquatic Plants Management Program for Washington State, 1992). In a pond study, diquat had no direct effect on aquatic insects but a decrease in pond weeds after treatment did result in migration of some species to shoreline vegetation (Hilsenhoff, 1966 as cited in Aquatic Plants Management Program for Washington State, 1992). An application with 0.5 ppm diquat in another pond led to loss of aquatic vegetation. The decaying vegetation appeared to benefit certain organisms such as Oligochaetes, indicated by an increased number (Tatum and Blackburn, 1962 as cited in Aquatic Plants Management Program for Washington State, 1992).

Dragonflies, damselflies and tendipedids exposed to diquat concentrations 40 times the maximum field application rate, survived. *Hyalella* was very sensitive to diquat as was *Cladocera* although *Cladocera* populations returned to normal levels after diquat concentrations disappeared from the water (Gilderhus, 1967 as cited in Aquatic Plants Management Program for Washington State, 1992.)

The species discussed above are all water column or epibenthic organisms (with the exception of the Oligochaetes). Because diquat is very persistent in sediments, it would seem that infauna or deposit-feeding organisms would have the highest potential for exposure to this compound. No specific studies pertinent to this issue were available.

Vertebrates:

The toxicity of diquat varies with the size and type of fish as well as the softness or hardness of the water. Reported LC50 values from one source ranged from 12-90 mg/l for 24-hour exposures, 6-44 mg/l for 48-hour exposures and 4-36 mg/l for 96-hour exposures (Calderbank, 1972 as cited in State of Washington, 1984). Another source reports acute toxicity values for specific fish ranging from a 96-hour LC50 value of 5 mg/l for rainbow trout to a 96-hour LC50 value of 140 mg/l for bluegill sunfish (Aquatic Plant Identification and Herbicide Use Guide, 1988). The EPA AQUIRE database contains the results of acute toxicity tests ranging from a 24-hour acute LC50 value of 1.0 mg/l in striped bass (an anadromous fish) to a 24-hour acute LC50 value of 5967 mg/l in grass carp (AQUIRE, 1995). In a survey of the results of diquat toxicity tests, the manufacturer has identified a range in toxicity of diquat to fish, with 96 hour LC50 values of 0.5-245 mg/l (Hamer, 1994).

The results of 13 experiments conducted with diquat indicate that diquat did not cause direct mortality to any fish species at 1.0 ppm and below. (MacKenzie, 1971 as cited in Aquatic Plants Management Program for Washington State, 1992). The highest concentration of diquat allowed by the manufacturer's label would equal an initial in-water diquat concentration of 1.5 ppm (Aquatic Plants Management Program for Washington State, 1992).

Studies conducted with fish exposed to solutions of diquat indicate that diquat concentrations in fish do not accumulate above the concentration of diquat in the surrounding water. In addition, when water concentrations decrease, fish diquat residues also decrease. In salmon, trout and goldfish kept in water containing a 1 µg/ml diquat concentration, diquat residues in fish were less than external water concentrations and were mostly found in the nonedible portion of the fish including skin and viscera (Valent U.S.A. Corporation, 1989 as cited in Aquatic Plants Management Program for Washington State, 1992).

Following 24-hour exposure to diquat, changes in rheotaxis and swimming speeds were noted in rainbow trout (Dodson and Mayfield, 1979 as cited in HSDB, 1994).

Trout immersed in water containing 0.5 ppm and 1.0 ppm diquat for 16 days had diquat residue levels of 0.4 and 0.6 ppm, respectively. When fish were returned to non-contaminated water, these levels slowly returned to non-detectable levels. Similar results were obtained with goldfish. (Aquatic Plants Management Program for Washington State, 1992).

Diquat is used to treat disease in fish at hatcheries and for the species tested did not affect the breeding rate in bluegills or cause mortality in young fish. 1 ppm diquat applied up to 3 times and 3 ppm applied once or twice, with 8-week intervals between applications did not affect hatching and growth rates of bluegills in seven different pools. Channel catfish fry were not affected at 10 ppm and bluegill fry were not affected at 4 ppm diquat. Largemouth black bass fry were affected at 22.5°C at levels greater than 1.0 ppm and at 26.0°C at levels greater than 0.5 ppm (Jones, 1965 as cited in Aquatic Plants Management Program for Washington State, 1992).

Decaying vegetation caused by treatment with diquat may deplete oxygen content in the water. For this reason, it is recommended that only 1/3 to 1/2 of an area containing dense vegetation be treated with diquat at a time with a 14-day waiting period in between (Aquatic Plants Management Program for Washington State, 1992).

Plants:

Since diquat is effective in treating a large range of plants, it may have a widespread effect on non-target plants. In addition to direct toxic effects of the herbicide, treatment of a pond with diquat may also cause indirect impacts including dissolved oxygen depletion and habitat loss. These impacts may cause general weakening and/or death of plants on a large scale (Aquatic Plants Management Program for Washington State, 1992).

Microorganisms:

Incubation with diquat caused rapid loss of potassium and phosphate from *Aspergillus niger*, *Penicillium frequentans*, *Mucor hiemalis* and *Zygorrhynchus heterogamus*. At higher concentrations of diquat, the rate of loss is greater, especially with *Zynchorrhynchus* and *Mucor*. Short-term incubation with diquat is followed by sustained loss of potassium when colonies of the above four species are transferred to water (Sahid *et al.*, 1981).

A 50% decrease in O_2 evolution was noted in the following algae organisms: *Chlorococcum sp*, *Dunaliella tertiolecta*, *Isochrysis galbana* and *Phaedactylum tricornutum* in water containing >500 ppm, >500 ppm, 15 ppm and 15 ppm of diquat dibromide, respectively. A 50% decrease in growth was noted in *Chlorococcum sp*, *Dunaliella tertiolecta*, *Isochrysis galbana* and *Phaedactylum tricornutum* in water containing 200 ppm, 30 ppm, 15 ppm and 15 ppm of diquat dibromide, respectively (Verschueren, 1983 as cited in HSDB, 1994).

Table III.2-4. Properties of Diquat

CAS #:	85-00-7
Synonyms	Dipyrido(1,2-a:2',1-c)pyrazinediium, 6,7-dihydro-dibromide; Deiquat; Diquat; Ethylene dipyridylium dibromide; 1,1-Ethylene 2,2-dipyridylium dibromide; 5,6-Dihydro-dipyrido(1,2a:2,1c)pyraziniu m dibromide; 6,7-Dihydropyrido(1,2-a:2',1'-c)pyrazinedium dibromide 9,10-Dihydro-8A,10A-diazoniaphenanthrene dibromide
Molecular formula (salt)	$C_{12}H_{12}N_2Br_2$
Molecular weight (salt)	344.07
Physical properties	yellow solid (pure salt monohydrate); aqueous solution is dark reddish-brown.
Melting point	salts decompose at high temperatures, charring rather than melting; decomposition temperature is >300^{o}C
Density	1.20-1.27 g/ml @20^{o}C/20^{o}C
Vapor Pressure	nonvolatile
Photolysis half-life	2-11 days
Hydrolysis half-life	insignificant
Biodegradation half-life	32 days
K_{ow}	603
K_{oc}	205-691 ml/g
BCF	<1-62
Water solubility	568 mg/l

(HSDB, 1994; Aquatic Plants Identification and Herbicide Use Guide, 1988; Herbicide Handbook, 1983)

Diquat References

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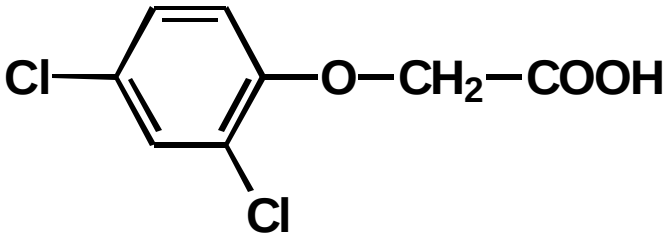
III.3 2,4-D



2,4- DICHLOROPHENOXYACETIC ACID

SUMMARY

2,4-D (2,4-dichlorophenoxyacetic acid) is a somewhat selective, systemic broadleaf herbicide that is used to control a variety of submersed, emersed and floating aquatic plants. 2,4-D exists in the acid form as well as in a variety of chemical forms. There are about 66 different formulations of 2,4-D, most of which are registered for terrestrial use. The 2,4-D acid form of this compound is not generally used for aquatic weed control (Reinert and Rodgers, 1987). The two categories of formulations which have been used most commonly for aquatic weed control include the butoxyethanol esters (2,4-D BEE) and the dimethylamine salts (2,4-D DMA) of 2,4-D. There are also a number of formulations being used for aquatic control containing the 2-ethylhexyl esters (2EHE), also known as 2-isooctyl esters (IOE), of 2,4-D (Reinert and Rodgers, 1987). 2,4-D formulations can exist as either emulsifiable concentrates, granulars, soluble concentrates, ready-to-use or pressurized liquids (DFA, 1988). The physical and chemical properties of 2,4-D are dependent on the chemical form of the active ingredient and vary dramatically.

In general, ester formulations of 2,4-D are more toxic to plants and fish than are amine salts. 2,4-D BEE formulations are generally not very soluble in water whereas 2,4-D DMA formulations have relatively high water solubility. Neither type of formulation is very volatile. Hydrolysis of 2,4-D BEE is a major fate process for this compound whereas it is not expected to be a significant fate process for 2,4-D DMA. Biotransformation and biodegradation are the major aquatic fate processes for both types of formulations. 2,4-D BEE tends to bioconcentrate to some degree in various organisms whereas 2,4-D DMA has a very low potential to bioconcentrate.

Many studies have been conducted with various formulations of 2,4-D addressing both toxicity and environmental fate and persistence. The U.S. Environmental Protection Agency (EPA) issued a registration standard for 2,4-D acid and all its chemical forms in September 1988 (USEPA, 1992). Since that time, the agency has been working with the industry to collect additional information under the mandate of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) amendments of 1988.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing 2,4-D that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html

on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

2,4-D USES AND APPLICATION

2,4-D can be used to control submersed, emersed and floating weeds. Liquid formulations of 2,4-D are only registered for the control of floating (e.g., waterhyacinth) and emergent vegetation. Surface applications can be made from a boat or from shore with dilute or concentrated product. Aerial applications can be made by spraying a dilute form of the product. Subsurface applications can be made with weighted trailing hoses from the boat (WSSA, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988).

Granular 2,4-D formulations can be distributed as either a surface application or as an aerial application using conventional mechanical spreaders or comparable equipment for large areas or a portable spreader for spot treatments (WSSA, 1983).

Treated water should not be used for irrigation, for agricultural sprays, for livestock watering or as a domestic water supply unless an approved assay indicates that the 2,4-D level does not exceed 0.1 mg/l 2,4-D acid-equivalent (Aquatic Plant Identification and Herbicide Use Guide, 1988). Alternatively, 2,4-D should not be used in waters with these uses. No swimming should take place for one day after treatment and fish should not be used from the treated waterbody for 3 days.

The best time to apply 2,4-D is in spring or early summer when young vegetation is actively growing. Application should be made in long strips separated by buffer zones. Application of liquid formulations should not be made during high wind or high water flow conditions. Aerial spraying should not be conducted if wind speed exceeds 8 km/hour. Drift control agents should be used when aerial spraying is conducted (Aquatic Plant Identification and Herbicide Use Guide, 1988).

For application of liquid formulations, especially when used on emersed or floating vegetation, use of invert emulsions or polymeric thickeners is recommended. For application of oil-soluble amine formulations, mixture with kerosene or other oil soluble solvent is recommended (Aquatic Plant Identification and Herbicide Use Guide, 1988).

Application rates of specific products vary due to the variation in the amount of active ingredient. For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. A list of the weeds that these products control, which has been compiled from the Environmental Protection Agency (EPA) registration labels for these products, is contained in Table III.3-1.

Table III 3-1. List of Aquatic Weeds Controlled by 2,4-D

Common Name	Scientific Name
Arrowhead	*Sagittaria* spp.
Bladderwort	*Utricularia* spp.
Bulrush	*Scirpus* spp.
Coontail or Hornwort	*Ceratophyllum demersum*
Creeping Waterprimrose	*Jussiaea repens*
Pickerelweed	*Pontederia* spp.
Spatterdock, Cow Lily, Yellow Water Lily	*Nuphar* spp.
Burreed	*Sparganium* spp.
Waterweed	*Elodea*
Waterchestnut	*Trapa natans*
Watermilfoil	*Myriophyllum* spp.
Water Smartweed	*Polygonum* spp.
White Waterlily	*Nymphaea* spp.
Naiad	*Najas flexilis*
Pondweed	*Potamogeton* spp.
Watershield	*Brasenia* spp.

(Riverdale Chemical Co.)

MECHANISM OF ACTION

2,4-D is readily translocated throughout the plant phloem, especially from foliage to roots, probably along with the products of photosynthesis (Aquatic Plant Identification and Herbicide Use Guide, 1988; Joyce and Ramey, 1986). It is a somewhat selective, systemic growth regulator with hormone-like activity. 2,4-D inhibits cell division of new tissue and stimulates cell division of some mature plant tissue, resulting in inhibition of growth, necrosis of apical growth and eventual total cell disruption and plant death. Low concentrations of 2,4-D may stimulate plant growth (Aquatic Plant Identification and Herbicide Use Guide, 1988). Introduction of saturation levels of artificial auxins (including 2,4-D) into growing plants disrupted the plants' delicate hormonal balance, causing reductions in root uptake of salts and water, phloem transport and photosynthesis, contributing to the death of the plant (White-Stevens, 1971 as cited in HSDB, 1995). 2,4-D also affects plant respiration and food reserves (Joyce and Ramey, 1986). Since 2,4-D produces many toxic responses, the primary mode of action has not been clearly established (Joyce and Ramey, 1986).

ENVIRONMENTAL FATE AND TRANSPORT

The environmental fate and transport of 2,4-D in aquatic environments is determined by the chemical formulation of the 2,4-D and the physical properties of the individual compounds.

2,4-D butoxyethyl esters (BEE) have low water solubility (estimated at approximately 12 mg/l) whereas 2,4-D diethylamines (DMA) have relatively high water solubility (about 3.0×10^6 mg/l) (Tables III 3-2 and III 3-3). The water solubility of the 2,4-D acid ranges from about 600-900 mg/l (Reinert and Rodgers, Aquatic Plant Identification and Herbicide Use Guide, 1988).

The 2,4-D acid has a Henry's Law (H) value of 6.2×10^{-3} indicating that it is somewhat volatile. The relative rate of volatilization is dependent on the formulation. In general, the acid, inorganic salts and amines are less volatile than the esters, which vary from high to low. The oil soluble amines are considered the least volatile (WSSA, 1983). Both 2,4-D BEE and 2,4-D DMA have relatively low volatility (Tables III 3-2 and III 3-3) (Reinert and Rodgers, 1987; Aquatic Plant Identification and Herbicide use Guide, 1988). A volatilization half-life of 895 days was calculated for 2,4-D BEE (Reinert and Rodgers, 1987).

The relatively high octanol:water partitioning coefficient (K_{OW}) and organic carbon partitioning coefficient (K_{oc}) for the BEE form (Table III 3-2) would suggest that this form would likely adhere to soils and or sediments with some organic content. The opposite would hold for the DMA form (Table III 3-3).

Estimates of the typical overall half-life of 2,4-D in water range from 10 to greater than 50 days. The primary fate process of 2,4-D in water is microbial biodegradation. The various chemical formulations of 2,4-D are also subject, to varying degrees, to breakdown via hydrolysis and photolysis.

There are a variety of microorganisms in both fresh and marine waters which are capable of degrading 2,4-D. The rate of biodegradation is dependent on a number of factors including the level of nutrients present, temperature, availability of oxygen and whether/not the water has had a prior history of contamination with 2,4-D or other phenoxyacetic acids. 2,4-D is generally more persistent in oligotrophic waters and in waters with high 2,4-D concentrations. Biodegradation half-lives in clear waters have been estimated to be from 18 to greater than 50 days. In muddy waters, biodegradation half-lives have ranged from 10-25 days (HSDB, 1995).

The significance of hydrolysis as a 2,4-D fate process varies with the chemical formulation. The 2,4-D acid is somewhat subject to hydrolysis. Hydrolysis is a significant fate process for 2,4-D BEE formulations but is not expected to be an important fate process for the 2,4-D DMA formulations (Reinert and Rodgers, 1987; Aquatic Plant Identification and Herbicide Use Guide, 1988).

There are conflicting reports as to the photolysis of 2,4-D and its derivatives in water. The relative significance of this fate pathway is dependent on the chemical formulation of the 2,4-D derivative. There are no available data which show direct photolysis of 2,4-D in the atmosphere upon exposure to natural sunlight. Most photolysis studies of 2,4-D have used high-intensity mercury lamps which emit large amounts of ultraviolet (UV) radiation (DFA, 1988). It has been shown, however, that 2,4-D exhibits an absorption maximum at 288 nm extending to greater than 290 nm. Sunlight reaching the earth is composed of wavelengths greater than 280 nm. These facts suggest that 2,4-D may be susceptible to direct photolysis (HSDB, 1995). Whereas some researchers do not believe that photolysis is a significant fate pathway for 2,4-D BEE (Aly and Faust, 1964 as cited in Reinert and Rodgers, 1987) others have calculated a photolysis half-life for these formulations (see Table III 3-2) (Zepp *et al.*, 1975 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1995). Photolysis is generally not expected to be a

significant fate pathway for 2,4-D DMA formulations (Reinert and Rodgers, 1987; Aquatic Plant Identification and Herbicide Use Guide, 1988).

Table III 3-2. Properties of 2,4-D BEE

CAS #:	1929-73-3
Molecular formula	$C_{14}H_{18}O_4Cl_2$
Molecular weight (g/mole)	321.2
Physical properties	colorless to amber oily liquid
Melting point	NA
Density	NA
Vapor pressure (mm Hg)	1.7×10^{-5} - 4.5×10^{-6}
Volatility [Henry's Law constant (atm m^3/mol)]	10^{-7}-10^{-5}
Photolysis half-life (days)	10-20
Hydrolysis half-life (days)	0.02-26
Biodegradation half-life (days)	0.11-2.3
K_{ow}	3400
K_{oc}	6607-6900 ml/g
BCF	162-408
Water solubility (mg/l)	12

(Reinert and Rodgers, 1987; Aquatic Plant Identification and Herbicide Use Guide, 1988; USEPA, 1988)

The overall fate of the 2,4-D BEE added to surface waters for Milfoil treatment in granular form is one of initial low concentrations of the ester in the water, appearance of the acid form of 2,4-D in the water shortly thereafter and relatively rapid subsequent decreases in concentration. The appearance of the ester in the water overlying sediments with the 2,4-D BEE associated granules is limited by the low water solubility of this compound, its uptake by water weeds and its rapid hydrolysis (Birmingham et al., 1981). Other recognized breakdown forms of the ester in water are butoxyethanol and 2,4-D acetate, and 2,4-dichlorophenol (Hoeppel and Westerdahl, 1983). After granular BEE application at the label rates to artificial ponds, maximum BEE concentrations in water were 0.16 and 0.68 mg/L one day after treatment and at or below detection limits of 0.01 mg/L after 13-15 days. Maximum concentrations of the acid form of 2,4-D occurred within 15 days and decreased 93% over the next 165 days (Birmingham et al., 1981).

Degradation of 2,4-D in aquatic sediments has been characterized as generally rapid (less than one day) and occurs mostly through microbial biodegradation (HSDB, 1995). Greater concentrations (~4x) of 2,4-D acid than ester occurred in the sediments of BEE treated artificial ponds (23 kg a.i./ha). Concentrations of the ester decreased by 94% over 42 days (Figure III 3-1) (Birmingham et al., 1981).

Table III 3-3. Properties of 2,4-D DMA

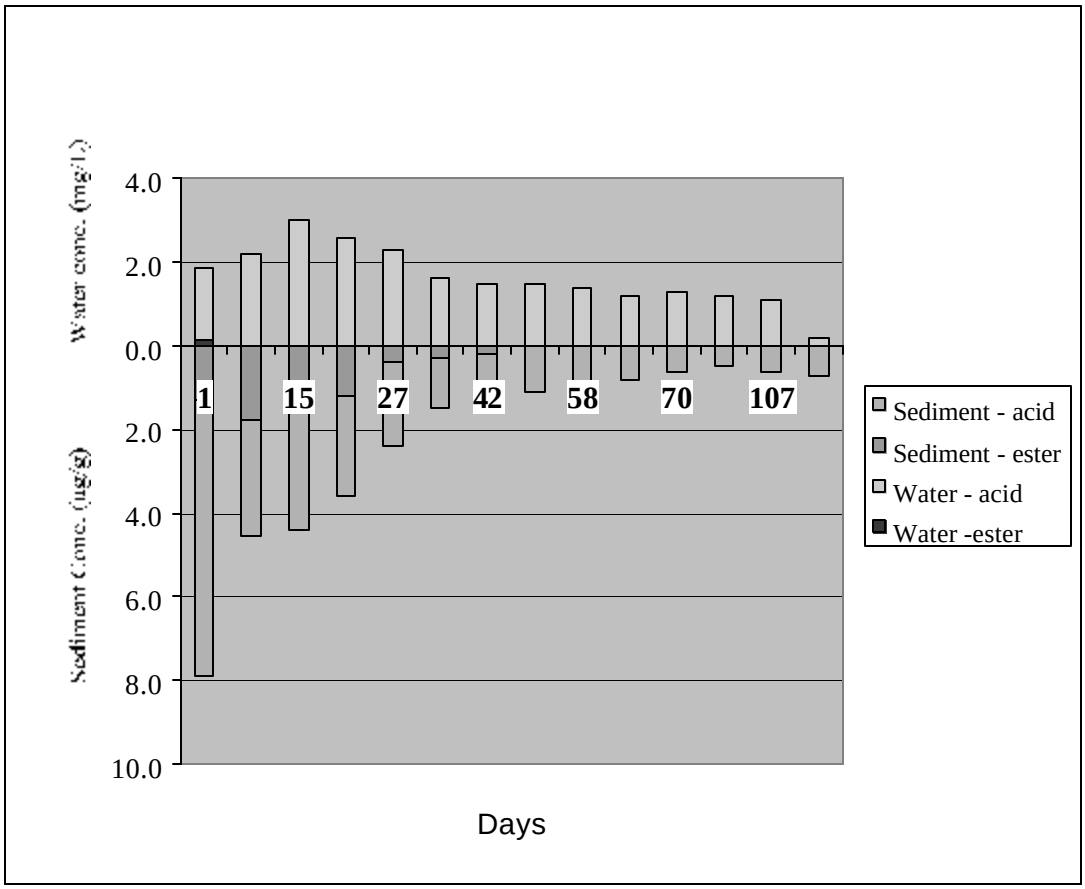
CAS #:	2008-39-1
Molecular formula	$C_{10}H_{13}O_3Cl_2N$
Molecular weight (g/mole)	266.12
Physical properties	white crystalline solid
Melting point (^oC)	85-87
Density	NA
Vapor pressure (mm Hg)	10^{-6} at 28^oC
Volatility [Henry's Law constant (atm m^3/mol)]	insignificant
Photolysis half-life (days)	insignificant
Hydrolysis half-life (days)	insignificant
Biodegradation half-life (days)	3.9-11 (based on overall half-life)
K_{ow}	low
K_p	0.13-0.25
BCF	1-7
Water solubility (mg/l)	3.0 x 10^6

(Reinert and Rodgers, 1987; Aquatic Plant Identification and Herbicide Use Guide, 1988; USEPA, 1988)

Mobility/Leaching:

A potential route of 2,4-D migration after application to lakes would be from the lake water into sediments and then into groundwater underlying the lake. This section evaluates the likelihood of such a process happening. Areas most likely to be subject to this flux are those where there is active recharge of the groundwater from the lake through the sediments and into underlying groundwaters. This situation may not exist in all lakes, only those that have significant contributions of lake water to groundwaters such as is found in many of the Atlantic Coastal Plain ponds on Cape Cod and possibly in southeastern Massachusetts. Some of these ponds receive the majority of water inflow from groundwater and have little surface water discharge, as Lake Water exits back through more porous pond sediments. Under these flow conditions, the likelihood of 2,4-D in sediments leaving with groundwater will be a function of the form of the 2,4-D, its water solubility and affinity for the sediments, and the organic content of the sediments.

Figure III 3-1. 2,4-D Decay and Partitioning in Experimental Ontario Ponds. Prepared from Data of Birmingham et al. (1981)



Information on water solubilty and soil (not sediment) affinity of 2,4-D can be used to make some conjectures about the likely fate of 2,4-D in situations where it has been applied to surface waters. The BEE form of 2,4-D is fairly water insoluble. The acid form of 2,4-D is quite water insoluble. A number of older studies summarized by Kelty (1980) demonstrated that organic carbon content of soils was a factor controlling leaching in terrestrial soils. Drenge (1969, cited in Kelty, 1980) found that 2,4-D acid or salt easily leached from permeable soils (sandy loam New Mexico soil). Opposing perspectives on leaching potential of 2,4-D acid were highlighted (Weber, 1970 and Liu and Cibes-Viade, 1973 cited in Kelty, 1980). 2,4-D acid movement in subsurface water was negligible (concentrations < 1 ug/L) after application of the acid to a small agricultural watershed in a sandy coastal plain soil. However, the author noted that the potential for losses in subsurface waters was greatest when it rained soon after herbicide application. Weber (1980) only cited one study in which 2,4-D application to ponds adversely affected water quality. The pond, groundwater and rivers in an area where 2,4-D had been used for weed control all had persistent (2.5 mo.) concentrations of 2,4-D. Groundwater concentrations did not appreciably decrease over the monitoring period.

Therefore, leaching from sediments into recharging groundwater would not be predicted based on the low water solubility of 2,4-D BEE and acid. In sandy, low organic content sediments, leaching would be facilitated by the low content and porous material. Field sediment and soil monitoring has documented 2,4-D migration into underlying groundwaters in such situations.

Biological Concentration:

The ability of 2,4-D and its derivatives to bioconcentrate in aquatic organisms is determined by its chemical formulation. One source indicates that there is little evidence that bioconcentration of 2,4-D acid occurs through the food chain. This conclusion was reached after a large-scale monitoring for 2,4-D residues in the many routes of metabolism and degradation that exist in ecosystems (Gray *et al.*, 1983 as cited in HSDB, 1995).

Bioconcentration factors for zooplankton after field exposure to either the butoxyethanol ester or the dimethylamine salt of 2,4-D ranged from 1 to 603 (Reinert and Rodgers, 1987). The maximum BCF in game and nongame fish after application of 2,4-D BEE at 22.5 and 45 kg/ha (~ maximum 0.68 mg/L 2,4-D in water) was approximately 22. Filter-feeding organisms may be more predisposed to accumulating 2,4-D in areas treated with granular BEE, but excretion is rapid and complete in less than 2 weeks (Hoeppel and Westerdahl, 1983).

Whole body 2,4-D BEE nonequilibrium BCF values were found to be very low, ranging from 2-14 in channel catfish (*Ictalurus punctatus*) and 6-12 in bluegill sunfish in aquaria. The ester was quickly hydrolyzed to the acid form and then rapidly excreted (Rodgers and Stalling, 1972 as cited in Reinert and Rodgers, 1987). Nevertheless, several estimates of the bioconcentration factor (BCF) for 2,4-D BEE formulations were made, varying by study and organism, ranging from 162-408 (Reinert and Rodgers, 1987). The bioconcentration potential for 2,4-D DMA formulations has consistently been shown to be low (Reinert and Rodgers, 1987).

MAMMALIAN TOXICITY OF 2,4-D ACID

The following subsections on mammalian toxicity are a summary of the detailed review of the toxic effects of 2,4-D by Harnois (1999). Harnois (1999) includes additional details including citations for the stated effects referenced in this summary.

Overview:

The extensive data from studies on the toxicity of 2,4-dichlorophenoxyacetic acid (2,4-D) and its derivative forms (e.g., salts, amines, esters) have been periodically reviewed by panels of experts. The most recent reviews were made during 1996 and 1997 by the United Nations Food and Agriculture Organization and World Health Organization (FAO/WHO), the United States Environmental Protection Agency (EPA) and the California Environmental Protection Agency (CALEPA).

Noncancer Effects:

In these recent reviews, noncancer effects were found to be similar in all species tested, although dogs were more sensitive for some effects. In almost all cases, derivatives of 2,4-D (amines, salts, esters) produced essentially the same effects as 2,4-D alone. The exception relates to exposure to the butanol portion of 2,4-D n-butyl ester, which was reported to lead to a form of ataxia not seen following exposure to 2,4-D or with other derivatives.

For noncancer effects, the Massachusetts Department of Environmental Protection (DEP) and EPA previously estimated that ingestion of 1 mg/kg/day over a lifetime would be without adverse effects in animals. The more recent reviews by EPA, FAO/WHO and CALEPA support this exposure level as an appropriate No Adverse Effects Level, or NOAEL, for 2,4-D. To derive a recommended human exposure limit protective against noncancer effects, all these agencies have applied a total uncertainty factor of 100 to

this NOAEL to account for differences between humans and animals and for differences in sensitivity between humans. This yields a recommended limit for human daily intake (Reference Dose) of 0.01 mg/kg/day for 2,4-D.

Cancer Effects:

Cancer effects have been studied in laboratory animals in controlled experimental studies using 2,4-D or its derivatives at several dose levels. Potential carcinogenic effects in humans have been investigated by studying populations that either produced or used 2,4-D or its derivatives. In all cases the people studied were also exposed to other potential carcinogens as well. Epidemiology studies now in progress may provide additional insight into any relationships between toxic effects and exposure to 2,4-D alone.

Animal Studies

Experiments on the potential carcinogenic activity of 2,4-D have been performed using mice, rats, and dogs. The results for studies in mice have been consistently negative. A single study in rats (reported in 1986) yielded results suggestive of a 2,4-D dose related increase in the incidence of brain tumors (astrocytomas). A more recent study using the same strain of rats (reported in 1995) and even higher doses showed no increase in total tumors or in tumors of any type. Beagle dogs fed 2,4-D for up to two years showed no indications of cancer effect. Because the normal lifespan of dogs is longer than 2 years, the exposure duration in this study was not adequate to demonstrate that 2,4-D is not carcinogenic in this species.

Based on these data, EPA has concluded that 2,4-D is not carcinogenic in technically acceptable rat and mice studies. No further studies were recommended.

Human Epidemiology Studies

Human epidemiology studies to date have been inadequate to causally relate 2,4-D and cancer effects. This is largely because the populations studied were also likely to have been significantly exposed to other potential carcinogens in addition to 2,4-D.

A potential association between 2,4-D exposure and the development of non-Hodgkins lymphoma has been reported. However, 2,4-D was not identified as the cause of this increase. Additional studies on the use of 2,4-D (and other chemicals) by farmers are in progress and may provide more specific data on chemicals associated with non-Hodgkin's lymphoma and other cancers.

At this time, the epidemiology data are inadequate to support a decision on 2,4-D's carcinogenic potential in humans.

Studies on Effects that May Be Related to Carcinogenicity

Many, but not all, genotoxic chemicals are also carcinogens. Genotoxicity and mutagenicity have been used as a predictor of carcinogenic potential. The results of genotoxicity tests on 2,4-D and derivative compounds vary, depending on the test endpoint. 2,4-D and similar compounds have been inconsistently mutagenic, but have shown more consistent evidence for interference with the distribution of chromosomes in several species. Some of these compounds were able to cause breaks in single strands of deoxyribonucleic acid (DNA) as well. EPA and its Carcinogenicity Peer Review Committee concluded that 2,4-D was not mutagenic but did note that some cytogenetic effects were seen; FAO/WHO concluded that 2,4-D was not genotoxic. None-the-less DEP believes that the current body of data indicate that 2,4-D is genotoxic due to its ability to alter DNA fine structure and composition in cells.

2,4-D is also a peroxisome proliferator. For some chemicals, this characteristic has been associated with a chemical's ability to enhance cellular proliferation and to increase the incidence of tumors in rodents, especially in the liver of mice. 2,4-D can induce peroxisomes in liver, but has not significantly increased the incidence of liver tumors in animal bioassays. It also does not exhibit an ability to transform cultured hamster cells or promote tumor growth in live mice also treated with urethane or naturally infected with mouse leukemia virus. The review panels noted that 2,4-D could enhance peroxisomal proliferation but concluded that the data were inadequate to relate this to carcinogenic potential in humans. Since 2,4-D is unlike the carcinogenic peroxisome proliferators in several respects, 2,4-D should not be considered a potential human carcinogen just because it is a peroxisome proliferator in rodents.

2,4-D has also been tested for ability to affect immune responses. The data from both animal tests and human studies on immune function indicate that there is no consistent effect associated with exposure to 2,4-D, either as a stimulant or as a suppressor. Additional testing for immunotoxic effect is needed since not all endpoints have been evaluated.

Conclusions on Potential for Carcinogenic Effect for Humans

- Technically adequate studies in rats and mice have now completed EPA's requirements for carcinogenicity testing. These studies failed to demonstrate a carcinogenic effect from exposure to 2,4-D.

- Human epidemiology studies have provided evidence that use of herbicides may be associated with increased tumors, but there is inadequate data to show that 2,4-D or its derivative forms are responsible for tumors in humans.

- EPA and WHO have concluded that 2,4-D is currently not classifiable with respect to human carcinogenic effect because of inadequate data from epidemiology studies.

Regulatory Implications

No increase in tumors or noncancer adverse effects were noted when animals were exposed to 100 times the human oral Reference Dose. Average daily intakes at this level are therefore not likely to increase risk of cancer in humans. Based on current information, the Reference Dose is recommended as a basis for deriving environmental guidelines expected to be protective against both noncancer and cancer effects.

MORE DETAILED SUMMARY OF PHARMACOKINETICS AND TOXIC EFFECTS

Data from the various animal tests and from human observations are summarized below (Harnois, 1999). Harnois et al, 1999 provides additional details including reference citations for the stated effects.

Pharmacokinetics:

2,4-D is well absorbed after oral, dermal, or inhalation exposure. 2,4-D is excreted in the original form, as are 2,4-D isopropylamine and 2,4-D triisopropylamine. The sodium salt and simpler amines are excreted as 2,4-D. The butoxyethyl ester and the ethylhexylesters are hydrolyzed to 2,4-D and the respective sidechains. The 2,4-D moiety is assumed to be a biochemically active element in the body since it is not metabolized further. The side chains of the esters are metabolized further and may contribute to the toxic effects seen in some cases.

At low exposure levels, absorbed 2,4-D is bound to carrier proteins (e.g., albumin, thyroxine carrier protein) in the blood; at higher doses, these carriers are saturated. Free 2,4-D is available to pass into the body tissues; in pregnant women it can also pass through the placenta into the fetus. Even at high concentrations, the fetus could still be protected if it had developed far enough to have its own carrier proteins in plasma.

2,4-D is more soluble in water than in oils and fats. The distribution in the body is related to this relative solubility. Two to eight hours after rodents were given an oral dose, 2,4-D was highest in blood, liver and kidneys of rodents of both sexes, and in ovaries of females. Intermediate quantities were present in skin, muscle, and in the male, the testes; fat and brain had the lowest concentrations.

Most of the 2,4-D is excreted through the kidney and in some cases, in perspiration. Lesser amounts are excreted in feces and milk. At doses higher than 50 mg/kg, mice and rats display a biphasic urinary clearance pattern, indicating that the clearance pathway in the kidney is saturable. After an oral exposure, about 50% of the administered dose was cleared in 1 hour (half-life equals 1 hour). In the second phase, the half-life for 2,4-D was 18 hours. At low doses (around 5 mg/kg), about 5% is transferred to bile in the liver and excreted in feces. At doses above 50 mg/kg, when urinary clearance is saturated, 15% or more was transferred to bile and excreted in feces. This biliary excretion pathway may be facilitated by conjugation of the 2,4-D with amino acids in the liver. None of the conjugate forms detected were associated with toxic effects.

At this time, the half-life of 2,4-D ingested by humans is not well defined; the few people tested have shown a high variability in response; the basis for this variability is not yet determined since few people (mostly young adult, physically active men) have been studied. The half-lives range from 4 to 48 hours, depending on the individual and on the administered dose. Even when a relatively low dose of 5 mg/kg was tested, one individual in the three subjects of one test showed a biphasic clearance pattern. The half-life for the first phase was 4 hours; for the second phase, 16 hours. Additional observations are needed if the 2,4-D half-life for humans is to be more accurately defined. The half-life of 2,4-D after dermal exposure (the usual route of human exposure) is even less well defined since it is affected by duration of contact and under working conditions, repeated exposures.

Acute Exposure Effects:

Acute exposures are one-time exposures that take place within a day. Acute exposure tests were used to determine if death or clinical signs could occur within a few weeks after absorption of 2,4-D into the body after ingestion, inhalation, or dermal exposure. Possible effects on skin and eyes from direct contact with 2,4-D were also tested.

Ingestion of 2,4-D results in effects within minutes; these effects include decreased activity, muscular weakness, uncoordinated movements, and possibly excess salivation and urination. At higher doses, the immediate effects can include vomiting, which can decrease the 2,4-D available for absorption into the body. Liver and kidney damage become evident within a few days; death may also occur within a few days. Those animals that do not die recover to function normally in time since 2,4-D is not stored in the body.

Oral doses which kill half the experimental animals in 14 days (LD50s) vary between species and show a broad range even within species. LD50s for beagle dogs ranged from 25 to 250 mg/kg; for rats, the LD50 range was 607 to 980 mg/kg. Human lethal doses vary, depending on the retained dose in suicide attempts. At least one death resulted from ingestion of 80 mg/kg but another person survived a dose of 700 mg/kg (plasma concentration, 392 mg/l) with the help of prompt medical attention, which included induced vomiting and alkaline diuresis therapy. This survivor recovered within 5 weeks and had no observed residual effects.

2,4-D on the skin is less toxic than ingested 2,4-D since absorption through skin is slower than absorption from the stomach. At low skin concentrations, the slower rate of absorption results in a more gradual increase in plasma concentrations of 2,4-D; the rate of removal from plasma could exceed this gradual rate of increase, resulting in no accumulation of 2,4-D in the plasma or body organs. In laboratory animals, the LD50 by this route is greater than 2000 mg/kg. Human exposure to 2,4-D in its various formulations is mainly by dermal exposure when workers are spraying it to control weeds. For humans, toxicity is enhanced when 2,4-D is allowed to remain on the skin or on clothing in contact with the skin. Clinical signs after a dermal exposure are the same as those resulting from ingestion of 2,4-D with the addition of skin irritation and lesions from prolonged skin contact. There are no reports that 2,4-D can cause allergic dermatitis or other types of sensitization.

Eye contact with 2,4-D (acid form) in laboratory animals resulted in corneal opacity, conjunctival edema and redness, ocular discharge, and inflammation of the iris; the effects persisted in rabbits for at least 20 days. Less toxicity and quicker recovery was noted after exposure to esters of 2,4-D.

Few studies are reported for effects after inhalation of 2,4-D. While it is not highly volatile, exposure by that route could occur when the herbicide is sprayed into the air to treat large land areas and in some industrial settings. Field workers applying 2,4-D to control weeds in crops have had symptoms of respiratory irritation (burning sensation in the throat and chest), loss of appetite, weight loss, and weakness.

Subchronic And Chronic Exposure Systemic Effects:

Experimental animal tests showed that 2,4-D is not accumulated or stored in the body but that exposure to low doses over several months can lead to progressive tissue damage, increasing the frequency and severity of effects seen in the liver, kidney, and brain. Additional adverse effects noted in animals included decreased serum thyroxine and hypertrophy of the thyroid, decreased serum glucose, cataracts, and retinal degeneration. Animals seemed to adapt in that the neurotoxic effects decreased with continued exposure. Systemic effects in surviving humans and animals were generally reversed within weeks when the 2,4-D exposure was stopped.

Genotoxic Effects:

Results of tests for genotoxicity were variable. Tests that measured mutations were usually negative; tests that measured breaks in individual DNA strands were positive; tests which detected defective distribution of chromosomes were often positive. Newly reported studies using exposure of larval forms of fruit flies to 2,4-D showed that both the treated insects and their offspring displayed new characteristics resulting from mutations and chromosome rearrangements. Since substantial evidence indicates that 2,4-D can induce chromosome changes in plant, insect, and mammalian cells, 2,4-D should be considered genotoxic.

Immunologic Effects:

Several epidemiology studies addressed possible immunological effects and exposure to 2,4-D. Although some effects were observed between use of herbicides and alterations in immunological parameters, no causal relationship was noted between 2,4-D and either overstimulation or suppression of the immune response. There are only limited data on the immunotoxic potential of 2,4-D and additional studies are needed to complete the current standard battery of immunotoxicity tests.

Neurotoxic Effects:

Observations made on neuromuscular endpoints in animals treated for a year showed that they were less able to relax their muscles after strongly gripping a test object (myotonia). Other signs of neurological

effects included sedation, uncoordinated movement, and decreased locomotor activity; these effects were also observed in subchronic and chronic exposure studies. These effects were reversed after treatment ended. In some studies, rats showed lesser effects as dosing continued, suggesting a possible adaptation.

Rats given 2,4-D-n-butyl ester showed increased foot splay when landing during the functional tests. This effect was also seen when n-butanol was administered, but not when other forms of 2,4-D or other butanols were administered. It was related to dose of n-butanol. This ester could have additional neurotoxic properties in humans not seen with other forms of 2,4-D.

Retinal degeneration was the most severe neurotoxic effect observed when rodents were given lower doses of 2,4-D in their diet for either a subchronic or chronic exposure duration. Females were more susceptible than males.

No human epidemiology studies were found to show a lasting specific 2,4-D effect on the nervous system.

Biochemical Effects:

Consistent effects of 2,4-D on the endocrine system have been reported including decreases in serum glucose and serum thyroxine.

Depletion of detoxifying molecules, such as glutathione and thiols, have also resulted from exposure to 2,4-D. These molecules are involved in the stabilization of cell microstructure by acting alternately as hydrogen donors and acceptors. One of the cell microstructures facilitates the distribution of chromosomes in cell division; interference with its normal function can result in genotoxic effects.

2,4-D stimulates the activity of oxidative enzymes associated with microsomes. It also stimulates activity of other small cytoplasmic particles, the peroxisomes. Increased peroxisome activity leads to a greater release of active oxygen, which can be genotoxic. Chemicals which enhance peroxisome proliferation are not necessarily carcinogens, but those which are carcinogens readily induce liver tumors in rodents. 2,4-D has not induced liver tumors in rodents or shown other activity which can enhance the incidence of tumors: it has not acted as a tumor promoter for pre-treated or virus-infected cells, nor has it stimulated uncontrolled proliferation of cultured normal cells. It is unlikely that 2,4-D would be carcinogenic because it is a peroxisome proliferator.

Reproductive Effects:

Exposure of rats for two generations resulted in no effect on ability to reproduce at doses that were otherwise nontoxic to the animals.

Most of the human epidemiology studies have studied effects in men; these studies have not shown that there were effects specifically from 2,4-D. Studies currently in progress do include significant numbers of women. Results from these studies, if specific to 2,4-D, would provide useful information on the potential of 2,4-D for reproductive effects.

Developmental Effects:

Pregnant rats and rabbits exposed to doses of 2,4-D which did not cause them to be severely ill were able to deliver normal litters, indicating that there were no teratogenic effects directly related to 2,4-D in these tests.

Children born to men who worked as pesticide applicators in the United States were observed for congenital abnormalities. Aborted fetuses and stillborn infants were not observed in the study, so all types of developmental effect were not included in the study. There was an increase in congenital abnormalities in live-borne children of couples residing in the farm area and a greater increase in children of pesticide appliers, especially for those who worked on multiple crops. As with other epidemiology studies, the association between chemical exposure and effects seen could not be attributed to 2,4-D alone.

Cancer Effects:

Animal Studies.

A very early study in mice (Innes, 1969) indicated that 2,4-D, 2,4-D isopropyl ester, 2,4-D butyl ester, 2,4-D isooctyl ester, alpha-(2,4-dichlorophenoxy)propionic acid, and 2-(2-4,-dichlorophenoxy)propionic acid were negative for cancer effect when tested at the maximum tolerable dose. These results for 2,4-D were confirmed in later, more detailed, cancer studies.

Tests in rats and mice on structurally related compounds were included in the review by the EPA Cancer Peer Review Committee (Rowland et al., 1997). 2,4-Dichlorophenoxy butyric acid, 2,4-dichlorophenoxy-2-propionic acid, 4-chloro-2-methylphenoxyacetic acid were not carcinogenic in rats and mice. 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) containing less than 0.05 ppm total dioxins increased the number of tumors in one strain of mice (XVII/G) but not another (C3Hf); dioxin-free 2,4,5-T was not carcinogenic in rats.

Results of a later study (1986) on 2,4-D administered to rats in their diet at doses of 0, 1, 5, 15, and 45 mg/kg/day suggested that the frequency of brain tumors (astrocytomas) was elevated in the high dose males (Table III 3-4). The incidences in males by dose group were 1/50, 0/50, 0/50, 2/50, and 6/50, respectively. There was a significant dose-related trend in the males.

The test was repeated (1995) in the same strain of rat at doses of 0, 5, 75, and 150 mg/kg/day. The frequency of astrocytomas was much lower in this study: 0/50 in male controls and 1/50 in high dose males; 1/50 in control females and 1/50 in high dose females. No tumors were found in the 5 and 75 mg/kg/day animals, but not all animals in the two intermediate groups were examined. The EPA Cancer Peer Review Committee has requested that brain tissue from all animals in the intermediate doses be examined histologically to complete the study. The results of this follow-up are not yet available.

Testing facilities record the observations on control group animals in a database. Observations in controls in subsequent studies are compared to this historical control database to ascertain if the source or lot of animals, or other conditions unrelated to treatment, have influenced the outcome of the study. No data were presented on animals at the testing facilities at which the 2,4-D feeding studies were conducted. However, EPA did compare the control and treated animals to the historical controls in the National Toxicology Program (NTP) database (Table III 3-4). The 2,4-D studies and the NTP studies were all done at different facilities. NTP rats were from various sources, but did not include the Charles River Laboratories, Portage MI, which was the sole source of the animals used in both of the 2,4-D feeding studies. This suggests that the background incidence in NTP rats may not be appropriate for use as historical controls. In this case, comparison should be to the historical control database at the testing facility, which are presently not available.

Adequate tests in two animal species are all EPA requires in its battery of animal toxicology studies used for regulatory purposes. EPA concluded that 2,4-D was not carcinogenic in the technically acceptable rat and mice studies. No further studies were recommended. There was no indication of

potential for cancer induction in the studies before 1986 on 2,4-D and on its derivative forms. The rodent studies thus provided no conclusive evidence that 2,4-D was a carcinogen.

Dogs allowed on lawns freshly treated with 2,4-D could have a relatively high intake of 2,4-D (maximum urinary excretion of 21.3 mg/liter urine two days after lawn treatment) and may be useful environmental sentinels under controlled conditions. Lymphoma frequency in dogs was studied in an effort to test this idea. Owners of dogs brought to veterinary clinics with lymphomas and other diseases (control dogs) were asked about use of pesticides and other environmental factors at their residence. Those dogs with lymphomas were found to have a possibly greater exposure to lawn treatment chemicals than other dogs, but the exposure to 2,4-D could not be separated out from exposure to other chemicals.

In laboratory studies, beagle dogs exposed to only 2,4-D in their diet for 2 years did not show evidence of tumor development.

Table III 3-4. Astrocytomas of the Brain in Rats Fed 2,4-D Acid

Astrocytomas of the brain in rats fed 2,4-D acid										
Sex	Males					Females				
1986 study at Hazleton Laboratories Dose (mg/kg/day)	0	1	5	15	45	0	1	5	15	45
Astrocytomas (after 2 years)	1/50 2%	0/50 0%	0/50 0%	2/48 4%	6/50 12%	0/50 0%	1/50 2%	2/50 4%	1/50 2%	1/50 2%
1995 study at Dow Chemical facility Dose (mg/kg/day)	0		5	75	150	0		5	75	150
Astrocytomas (after 2 years)	0/50 0%		0/26	0/18	1/50 2%	1/50 2%		0/14	0/11	1/50 2%
Background in historical controls (NTP: 3 other laboratories)										
Range			Males: 0-4.4%			Females: 0-4%				
Mean			Males: 0.4%			Females: 0.3				

Human Studies.

Some reports suggest a possible association between exposure to 2,4-D and the development of tumors. Different populations of agricultural workers who applied chemicals, including 2,4-D, to crops showed increased incidence of either soft tissue sarcoma or non-Hodgkin's lymphoma. However, none of the numerous epidemiological studies on the association between 2,4-D and various forms of cancer adequately demonstrate that the association is specific for 2,4-D. The populations studied had significant exposure to other chemicals, either as background exposures over their lifetimes living in rural agricultural areas or at the same time as the 2,4-D exposure. In many cases, increases in tumor incidence were not statistically significant.

Human epidemiology studies have provided evidence that use of herbicides may be associated with increased tumors, but there is inadequate data to show that 2,4-D or its derivative forms is responsible for these tumors. Additional studies are in progress to address deficiencies in those currently available for review.

EPA and WHO have concluded that 2,4-D is currently not classifiable with respect to human carcinogenic effect because of inadequate data from epidemiology studies.

Quantitative Evaluation:

Dose-response for Noncancer Effects:

In chronic exposure tests on animals, effects other than cancer were observed at lower average daily doses than were effects suggestive of cancer. The relationship between average daily dose and the effects seen (dose-response) can be estimated for noncancer effects.

EPA had previously accepted a NOAEL of 1 mg/kg/day 2,4-D (or equivalent) based on absence of liver, kidney, and hematopoietic effects which were seen at higher doses in a subchronic effect study on rats. Their current evaluation is based on a NOAEL of 1 mg/kg/day for absence of systemic effects in a 1-year study in dogs.

The WHO summary shows that the lowest NOAEL of 1 mg/kg/day 2,4-D is based on this same dog study and on absence of noncancer effects (kidney lesions) in the 1986 2-year rat study discussed above.

Both agencies used a total 100-fold adjustment or uncertainty factor to extrapolate to humans and to account for differences sensitivity between people. Based on this approach both agencies estimated that a human reference dose protective against noncancer effects would be 0.01 mg/kg/day 2,4-dichlorophenoxy acetic acid (or equivalent in the salts, amines, or esters).

Evaluation of Carcinogenic Potential:

The available data from animal carcinogenicity studies suggest that 2,4-D would not be a carcinogen for humans. Limited data indicate that several other phenoxy herbicides, including those having 2,4-D as a biologically active moiety, were not carcinogenic when tested in mice and rats.

2,4-D is both a genotoxin and a peroxisome proliferator in animals, but these characteristics are not always related to carcinogenic potential. 2,4-D has not caused a significant increase in the incidence of rodent liver tumors (a characteristic of carcinogenic peroxisome proliferators) or of having the capability to promote the carcinogenic effect of other chemicals or viruses.

Human epidemiological studies have not provided adequate data for evaluation of 2,4-D as a human carcinogen since they include exposure to other chemicals; other studies currently in progress could provide a more accurate characterization of 2,4-D's effect on both men and women. EPA has concluded that 2,4-D is not classifiable as to human carcinogenicity since the human epidemiology data are still being clarified.

Since cancer effects were not observed in animals at even 1 mg/kg/day 2,4-D, exposures at the Reference Dose of 0.01 mg/kg/day are not expected to cause a significant increase in the risk of human cancer.

Available Toxicity Criteria:

The EPA RfD/RfC Workgroup has developed an oral Reference Dose (RfD) of 0.01 mg/kg/day for 2,4-D based upon a 90-day rat oral bioassay and a 1-year interim report from a 2-year rat bioassay. The RfD is an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1992).

The EPA Office of Pesticide Programs (OPP) has developed an RfD of 0.003 mg/kg/day based on a two-year feeding study in rats (USEPA, 1995).

The EPA has developed a Maximum Contaminant Level (MCL) for drinking water for 2,4-D of 0.07 mg/l (USEPA, 1988). This drinking water level has been adopted by Massachusetts as a Massachusetts Maximum Contaminant Level (MMCL).

The EPA has also developed an Ambient Water Quality Criterion (AWQC) for 2,4-D of 0.001 µg/l. This concentration represents an acceptable level of 2,4-D in ambient water for a human who drinks the water and eats fish who inhabit the water (USEPA, 1988).

ECOLOGICAL TOXICITY

Avian:

A number of toxicity tests conducted in birds indicate that 2,4-D is only slightly toxic or practically nontoxic to most test birds. LC50 values (i.e., concentrations lethal to 50% of the test population) for ring-necked pheasant, Japanese quail, Bobwhite quail and mallard ducks to be greater than 5,000 ppm for 2,4-D, the butoxyethanol ester of 2,4-D and the dimethylamine salt of 2,4-D (USDA, 1984). Reported LD50 values for 2,4-D acid include an oral dose of 668 mg/kg for rock doves, an oral dose greater than 2,000 mg/kg for 4 month old mallards and an oral dose of 668 mg/kg for 2 month old mallards (HSDB, 1995). 2,4-D was found to be moderately toxic to 4 month old chukar (partridge) with a reported LD50 value of 200-400 mg/kg (HSDB, 1995).

In terms of reproductive or developmental effects, spraying chicken eggs with 2,4-D amine 29 hours before incubation at rates of 1, 10 and 20 times the recommended rates had no adverse effect on any parameter used to evaluate either incubation or subsequent live performance (USDA, 1984). Two studies indicated that spraying eggs of quail, pheasants and chickens with 2,4-D in concentrations up to 10 times the recommended doses, produced no effect on the hatching rate, body weight, sexual differentiation or reproductive performance (as adults) of number of malformed chicks (GEIR, 1985). In another study in which 2,4-D amine was sprayed at a concentration of 1.1 kg active ingredient per hectare on fertile eggs, 77% of ring-necked pheasant embryos, 43% of red partridge embryos and 77% of grey partridge embryos were dead on the nineteenth day of incubation. Surviving embryos were malformed or partially or completely deformed (GEIR, 1985).

Aquatic Organisms:

The toxicity of 2,4-D has been shown to vary with the formulation, the species of fish, the water quality and the environmental conditions (season and temperature). According to several sources, many of the formulations, especially the esters, are toxic to fish (GEIR, 1985). Hoeppel and Westerdahl (1983) cited numerous studies showing ester formulations of 2,4-D and butoxyethanol to be 50-200 times more toxic to fish than the free acid or dimethylamine salt of 2,4-D. 96-Hour LC50 values for fish are in the

0.6-7 mg/L range. The bluegill is the most sensitive of the species with compiled data and the salmonids are also quite sensitive with LC50s in the lower part of the noted range.

Numerous studies show that the 96-hour LC50s for cutthroat trout fingerlings ranged from greater than 50 ppm for the isooctyl ester down to 0.78 ppm for the butyl ester (USDA, 1984). 96-hour LC50s for the dimethylamine salt were reported as 64 ppm for cutthroat trout, 100 ppm for chinook salmon and 236 ppm for smallmouth bass. A 96-hour LC50 of 1313 ppm for grass carp was also reported for the amine salt (GEIR, 1985). 96-hour fish LC50s for the 2,4-D acid are generally higher, ranging from a slightly toxic 26.7 ppm in banded killifish to a practically nontoxic 358 ppm in rainbow trout.

Table III 3-5 lists the results of a number of 96-hour acute toxicity assays using 2,4-D acid or the butoxy ethyl ester. The acute lethal toxicity (96 hour LC50) of the BEE to a variety of salmonids increases by factors of 2.8-4 times as the water pH decreases (Wan et al., 1990).

Many studies conducted to assess the effects of 2,4-D on lower aquatic organisms suggest that toxicity varies with the different formulations of 2,4-D (GEIR, 1985). Again, it appears that some ester formulations are the most toxic. A 96-hour LC50 was reported as 6.1 ppm for scud and 2.6 ppm for sowbug exposed to the butoxyethanol ester of 2,4-D. Results of other lower aquatic organisms exposed to various 2,4-D esters were similar (USDA, 1984). 2,4-D was only slightly toxic to *Daphnia*, with a 48-hour acute LC50 of 25 ppm (Dow Elanco, 1995). 48-hour TL50s (concentrations at which there is some toxic effect to half the population) of 100 ppm were reported for many crustaceans exposed to the dimethylamine salt (USDA, 1984). A TL50 greater than 100 ppm was reported in crayfish (USDA, 1984).

The relatively rapid conversion of the BEE to the acid form in water makes interpretation of and application of laboratory toxicity study results to field exposures less than straightforward. In static exposure 96 hour tests much of the parent BEE will have changed to the less toxic acid form over the test duration so that recorded mortalities expressed as a nominal concentration of BEE actually reflect decreasing exposures. In study results from flow-through exposures continually delivering fresh BEE to test vessels over a study, mortality results more accurately reflect constant exposures to the nominal concentrations, but will have lessened quantitative relationship to likely exposures in the field.

It should be noted that LC50s are toxicological benchmarks but convey nothing about the sublethal toxicity characteristics of the chemical. Exposure concentrations in the environment that are less than a 96 hour LC50 value may not represent no risk values.

Observed 2,4-D BEE concentrations in the water after application of the 2,4-D BEE to ponds (see section above) peak within a day at less than 0.2 mg/L and decrease within 15 days to 0.01 mg/L (see Section on Environmental Fate and Transport). Both of these concentrations are less than the 96 hour LC50s of the more sensitive aquatic species, but possibly within the range where sublethal effects might occur as the concentration is only a third that of the LC50 of the lowest LC50 value that has been identified.

The acid form of 2,4-D is quickly formed from the ester and is present for much longer at higher concentrations. Highest acid form concentrations over any 96-hour period would be in the range of 3 mg/L. However these types of concentrations (2-3 mg/L) could persist in the water for four to five weeks, representing a considerably longer exposure than that used in standardized aquatic toxicity tests.

This information suggests that there would be the potential for some acute lethal and sublethal toxicity to aquatic species in the days immediately following application, due to the action of the butoxyethyl ester form of 2,4-D. Further lethal toxicity seems unlikely after that because the 2,4-D will have changed to the acid form in concentrations that should be from 10 to 100 times less than the 96 hour

LC50s for aquatic species. However, given that the exposures could persist for many days longer than those in laboratory toxicity tests, the possibility of mortalities or sublethal effects cannot be ruled out. This review has not sought to comprehensively identify the universe of data which may exist on chronic exposures and sublethal toxicity of aquatic species to 2,4-D; therefore the potential for this type of toxicity remains an open question. For comparison, the reproductive performance of female crabs was adversely affected after chronic exposure to 2,4-D isobutoxyethanol ester at a concentration of 50 mg/L which was near the incipient lethal level for this compound and species (Rodriguez et al. (1994). The observed decrease in oocyte sizes is a consistent reflection of the type of outcome which might be associated with the recognized mode of action of this chemical: uncoupling of oxidative phosphorylation in the respiratory chain.

Table III 3-5. 96 Hour LC50 Aquatic Toxicity Tests Using 2,4-D

Form	Species	Conc.(ppm)	Reference
Acid	rainbow trout	358	Dow Elanco, 1995
	fathead minnow	320	Dow Elanco
	american eel	300.6	HSDB, 1995
	bluegill	263	Dow Elanco, 1995
	carp	96.5	HSDB, 1995
	pumpkinseed fish	94.6	HSDB, 1995
	guppy	70.7	HSDB, 1995
	striped bass	70.1	HSDB. 1995
	cutthroat trout	64	USDA, 1995
	white perch	40	HSDB, 1995
	Banded killifish	26.7	HSDB, 1995
	Daphnia	25,36.4	Dow Chemical Co. , 1983a.
BEE	Rainbow trout	2.0	Dow Chemical Co., 1983b
	Bluegill	0.6	Dow Chemical Co., 1983b
	Daphnia	7.2	Dow Chemical Co., 1983b
	Fathead minnow	2.5	Dow Chemical Co., 1983b
	Coho salmon Chinook salmon Chum salmon Pink salmon Sockeye salmon Rainbow trout	0.6-4.3	Wan et al., 1990

Plants:

Treatment of a water body with 2,4-D may cause depletion of dissolved oxygen from decomposition of dead weeds as well as habitat loss (Riverdale Chemical Co., 1988). Benthic macroinvertebrate species diversity was significantly lower in BEE treated experimental ponds than in control ponds after 338 days (Stephenson and Mackie, 1986).

2,4-D References

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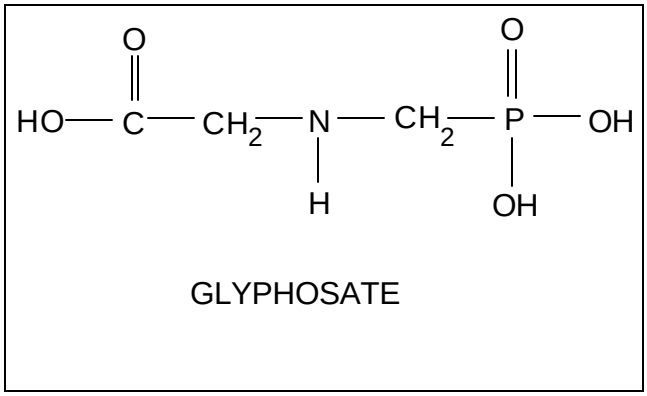
III.4 GLYPHOSATE



GLYPHOSATE

SUMMARY

Glyphosate ((N-phosphonomethyl)glycine) is a broad-spectrum herbicide used to control emersed aquatic grasses, broadleaf weeds and brush. It is not applied to submersed or mostly submersed vegetation. Glyphosate is not subject to hydrolysis or photolysis and is not expected to degrade by either route. It is not volatile. In natural waters, glyphosate dissipates in about 1.5-14 days. Breakdown of glyphosate in the aquatic environment occurs mostly through microbial degradation. Glyphosate is also rapidly inactivated by adsorption to soil. Its tendency to bioconcentrate in fish is very low. There are no restrictions on the use of glyphosate-treated water for irrigation, recreation, or domestic purposes. However, there are restrictions on the application of glyphosate within 0.5 mile upstream of potable water intakes and on the retreatment of an area within 24 hours (Monsanto, 1990). Available information indicates that glyphosate is of relatively low toxicity to mammals and aquatic organisms.

The Environmental Protection Agency (EPA) first registered glyphosate for use in 1974. The glyphosate registration was reviewed under EPA 1988 amendments to FIFRA (Federal Insecticide, Fungicide and Rodenticide Act). In 1993, the EPA issued a Reregistration Eligibility Decision (RED) on glyphosate along with a large number of products containing glyphosate as an active ingredient (USEPA, 1994).

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing glyphosate that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

GLYPHOSATE USES AND APPLICATION

Glyphosate can be used to control emergent aquatic weeds in freshwater lakes, ponds, reservoirs, canals, rivers, estuaries, seeps, irrigation and drainage ditches, wastewater treatment facilities and wildlife habitat restoration and management areas (McLaren/Hart, 1995).

Application of glyphosate may be made using a variety of methods. Broadcast sprays (either ground-rig or aerial) can be used for broad spectrum control over large areas. Handgun and backpack sprayers can be used for more localized application of the herbicide when the spray needs to be targeted away from desirable species. Wiper trunk injection, cut stem/cut stump and tree injection techniques can also be used for more localized control. The more selective methods are only practical for treating relatively small areas (McLaren/Hart, 1995).

The most effective time of application for most perennial and rhizome-bearing species (cattails, phragmites, etc.) is after the plant enters the reproductive stages of growth (ie., generally late August to October) (Kantrud, 1992 as cited in McLaren/Hart, 1995). In general, application should be made in times of low stress (e.g., drought, disease, nutrient depletion, infestation, etc.) and maximum translocation.

Glyphosate is effective for use on floating and emergent aquatic plants but not on submerged aquatic plants because it is diluted below an effective concentration in the treated water. In floating weeds, the effectiveness is reduced if wave action washes the product off before it can penetrate plant foliage (McLaren/Hart, 1995).

The application rate of glyphosate varies depending on the target species, the application method and the specific formulation used. The maximum rates are used for the most resistant target species or for high target weed infestations. Product labels should be consulted for recommended application rates and use restrictions (e.g., not to apply within specified distance from potable water sources).

The addition of a non-ionic surfactant is recommended to promote adhesion, spreading and penetration of the spray droplets through the plant cuticle on the leaves and to maximize absorption and effectiveness of treatment (WSDOE, 1992).

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/.

MECHANISM OF ACTION

Glyphosate penetrates the plant leaf cuticle shortly after contact and begins a cell by cell migration to the phloem, from which it is transported throughout the plants. The herbicidal action usually occurs within 7 days and up to 30 for woody plants (McLaren/Hart, 1995; Monsanto, 1990.)

Glyphosate's primary herbicidal mode of action is to block the synthesis of aromatic amino acids and the metabolism of phenolic compounds by disrupting the plant's shikimic acid metabolic pathway, leading to the inability of the plant to synthesize protein and produce new plant tissue. This is the only herbicide known to interfere with this particular pathway (McLaren/Hart, 1995). A secondary mode of action affects the photosynthetic process, synthesis, respiration and synthesis of nucleic acids by interacting with a complex series of enzymes which control synthesis of important molecules such as chlorophyll. The results of these interactions are a decrease in the rate of photosynthesis, an increase in respiration rate and a series of cellular changes (i.e., formation of granular bodies, deterioration of oil bodies, the endoplasmic reticulum and ribosomes and the vacuolation of the cytoplasm) leading to death (McLaren/Hart, 1995).

Table III.4-1. List of Aquatic Plants Controlled by Glyphosate

Alder	*Alnus* spp.
Ash	*Fraxinus* spp.
Barnyardgrass	*Echinochloa crus-galli*
Birch	*Betula* spp.
Cattail	*Typha* spp.
Cordgrass	*Spartina* spp.
Dogwood	*Cornus* spp.
Elder	*Sambucus* spp.
Elm	*Ulmus* spp.
Flatsedge, Chufa	*Cyperus esculentus*
Fleabane	*Erigeron* spp.
Foxtail	*Setaria* spp.
Foxtail, Carolina	*Alopecurus carolinianus*
Hemlock, Poison	*Conium maculatum*
Honeysuckle	*Lonicera* spp.
Hornbeam, American	*Caprinus caroliniana*
Lettuce, prickly	*Lactuca serriola*
Maple, red	*Acer rubrum*
Milkweed	*Asclepias* spp.
Monkey-flower, Common	*Mimulus guttatus*
Nutgrass	*Cyperus rotundus*
Oak, pin	*Ouercus palustris*
Panicum	*Panicum* spp.
Phragmites	*Phragmites* spp.
Poison Ivy	*Rhus radicans*
Poplar	*Populus* spp.
Purple Loosestrife	*Lythrum salicaria*
Salt cedar	*Tamarix* spp.
Saltbush, sea myrtle	*Baccharis halimifolia*
Smartweed, Pennsylvania	*Polygonium pennsylvanicum*
Smartweed, swamp	*Polygonum coccineum*
Spikerush	*Eleocharis* spp.
Sumac, poison	*Rhus vernix*
Sycamore	*Platanus occidentalis*
Tules, common	*Scirpus acutus*
Willow	*Salix* spp.
Waterhyacinth	*Eichornia crassipes*
Water-lettuce	*Pistia stratiotes*

McLaren/Hart, 1995

ENVIRONMENTAL FATE/TRANSPORT

The major fate process affecting glyphosate persistence in aquatic environments is biodegradation. Microorganisms in soil, water and sediment biodegrade glyphosate under both aerobic and anaerobic conditions (Reinert and Rodgers, 1987; McLaren/Hart, 1995). The main biodegradation product in soil and sediments is aminomethylphosphonic acid (AMPA). Other minor metabolites, including N-

methylaminomethylphosphonic acid, N,N-dimethylaminomethylphosphonic acid, hydroxymethylphosphonic acid and two unidentified metabolites. Residue levels of glyphosate and AMPA in the aquatic environment are low and dissipate rapidly over time (McLaren/Hart, 1995).

Absorption to sediment is another major contributor to the aquatic dissipation of glyphosate. The average half-life of glyphosate in soil is 60 days. In natural waters, dissipation half-lives of glyphosate range from 1.5-14 days. The dissipation half-life of glyphosate in waters not associated with sediments is much longer, (i.e., 7-10 weeks). In the presence of sediments, under either aerobic or anaerobic conditions, dissipation half-lives for glyphosate range from 6.5-21 days (McLaren/Hart, 1995; WSDOE, 1992; Reinert and Rodgers, 1987).

Glyphosate is an acid and bonds to soil with ionic interactions. It has a negligible vapor pressure and is nonvolatile. Glyphosate contains no photolyzable or hydrolyzable groups and is not expected to degrade in these ways (WSSA, 1983 as cited in Reinert and Rodgers, 1987).

The bioconcentration factor (BCF) for glyphosate in fish is low (Westerdahl and Getsinger, 1988 as cited in WSDOE, 1992). Glyphosate residuals are not typically found in fish because there is no affinity between the glyphosate molecule and (the typically lipophilic) fish tissue. Any glyphosate will pass unchanged through the mouth or gills of the fish, remaining either in solution or adsorbed to suspended particulates (McLaren/Hart, 1995). Exposure of experimental fish for 10-14 days to glyphosate concentrations 3 to 4 times the recommended levels resulted in BCF values of 0.2-0.3, which are considered insignificant (Brandt, 1984 as cited in WSDOE, 1992). Information submitted by the manufacturer of this compound also supports the finding of BCF values no higher than 0.3 (Monsanto, 1990 as cited in McLaren/Hart, 1995).

PHARMACOKINETICS

Rat studies indicate that oral doses of glyphosate are rapidly but poorly absorbed by rats, with female rats absorbing more than males (McLaren/Hart, 1995; USEPA, 1992). The glyphosate that is absorbed is rapidly excreted as unmetabolized glyphosate, with 90% of the absorbed dose being excreted within 48 hours (McLaren/Hart, 1995). Peak levels of glyphosate in the blood and bone marrow of rats dosed intraperitoneally occurred within 30 minutes. The concentration of glyphosate in blood had a half-life of one hour but remained relatively constant in bone marrow, with a half-life of 7.6 hours for males and 4.2 hours for females. Following intravenous doses of glyphosate administered to mice, 30-36% of the compound was eliminated unchanged in the urine and the rest in the feces. Traces (0.04%) of aminomethylphosphonic acid (AMPA) were found to be the only metabolites in the feces. Studies conducted with glyphosate administered in feed to chickens, cows and swine suggest that glyphosate does not accumulate in animal tissues during periods of oral exposure (USEPA, 1992). A series of residue and metabolism studies have shown that glyphosate is poorly absorbed across the gastrointestinal tract and there is minimal tissue retention and rapid elimination of residues in birds and fish in addition to mammals (Monsanto, 1993).

HEALTH EFFECTS

Avian:

A number of acute toxicity studies of technical grade glyphosate were conducted on ducks and quail. Five-day LC50 values were >3,850 mg/l for each or, practically nontoxic (Monsanto, 1988 and USEPA, 1986 as cited in WSDOE, 1992; AQUIRE, 1995).

Mammalian:

Acute:

There is very little information in the published literature on the acute toxic health effects of glyphosate. Glyphosate has very low mammalian acute oral or dermal toxicity (McLaren/Hart, 1995). Acute toxicity studies for a commercial formulation of glyphosate have produced oral LD50 values for Rodeo of 4,873 and 5,600 mg/kg in rats and 1,568 mg/kg in mice (USEPA, 1992). A dermal LD50 value of greater than 5,000 mg/kg (i.e., practically nontoxic) was reported for rabbits (USEPA, 1992). For technical glyphosate, an oral LD50 in the rat and a dermal LD50 in the rabbit were found to be greater than 5,000 mg/kg. The most prominent effect following glyphosate poisoning was reported to be hyperemia (i.e., an excess of blood) of the lungs, with severe stress, accelerated breathing, elevated temperature, occasional convulsive movements and rigor preceding death. A commercial formulation of glyphosate was found to be practically nonirritating to rabbit eye and skin whereas technical glyphosate was severely irritating to rabbit eye but practically nonirritating to rabbit skin (McLaren/Hart, 1995). Glyphosate was found to be a cumulative irritant in guinea pigs (USEPA, 1992). The EPA concluded that glyphosate is slightly irritating to skin and is not a dermal sensitizer (USEPA, 1993a).

Subchronic/Chronic:

Results of subchronic and chronic laboratory studies also indicate that glyphosate is not very toxic. In 90-day feeding studies conducted with rats at doses up to 1,000 mg/kg, no changes as compared with controls in body weight, behavior, mortality, hematology, blood chemistry, or urinalysis were noted. In dogs administered up to 60 mg/kg, a similar lack of changes was noted (USEPA, 1992). A 26-month chronic feeding study in which rats were administered doses of up to 31.5 mg/kg/day (males) and 34 mg/kg/day (females) produced no significant effects on body weight, organ weight, organ/body weight ratios or hematologic and clinical chemistry parameters (USEPA, 1992). In a 24-month chronic study in which rats were administered glyphosate at 2,000, 8,000 and 20,000 ppm for 24 months, a significant decrease in body weight in high-dose females was noted. The No Observed Adverse Effect Level (NOAEL) for glyphosate in this study is 8,000 ppm (McLaren/Hart, 1995). In a one-year dog feeding study, there was an apparent decrease in absolute and relative pituitary weights with no accompanying histopathologic changes. A NOAEL of greater than 500 was reported from this study (Monsanto, 1985 as cited in USEPA, 1992).

Developmental/Reproductive:

In a three generation reproductive study in which male and female rats were administered dietary concentrations of glyphosate corresponding to 0, 3, 10 and 30 mg/kg/day, there were no treatment-related systemic or reproductive effects noted in adults. One group of third generation male pups whose parents were exposed to the highest dose (30 mg/kg/day) showed an increase in the incidence of unilateral renal tubular dilation. The No Observed Adverse Effect Level (NOAEL) for glyphosate in this study is 10 mg/kg/day and the Low Observed Adverse Effect Level (LOAEL) is 30 mg/kg/day (Bio/dynamics, Inc., 1981a as cited in USEPA, 1992). In a subsequent two-generation reproductive study in rats, rats were administered glyphosate in the diet at levels up to 30,000 ppm (about 1,500 mg/kg/day). The only effects noted were very frequent soft stools in the F_0 and F_1 males and females, decreased food consumption and body weight gain of the F_0 and F_1 males and females during the growth (premating) period and decreased body weight gain of the F_{1a}, F_{2a} and F_{2b} male and female pups during the second and third weeks of lactation. Focal tubular dilation of the kidneys, observed in the previous study, was not observed in this study at any level. As a result, the EPA concluded that the presence of this effect in the three-generation study was a spurious rather than glyphosate-related

effect (USEPA, 1993a). Rabbits treated with 350 mg/kg/day during days 6-27 of gestation produced signs of maternal toxicity but did not exhibit developmental toxicity.

Mutagenicity:

Glyphosate was not found to be mutagenic in eight strains of bacteria and yeast evaluated in microbial test systems and in Chinese hamster ovary cells (USEPA, 1988; USEPA, 1993b). In addition, glyphosate also produced negative results for chromosomal aberrations in mouse dominant lethal test, the *in vivo* cytogenetics assay, the *Bacillus subtilis* rec assay and in the rat hepatocyte DNA repair assay. High concentrations of glyphosate have produced sister chromatid exchange in human lymphocytes *in vitro* (USEPA, 1992). However, the information from this study has been shown to be possibly erroneous (Slapikoff, 1983; Brusick, 1983).

Carcinogenicity:

No clear-cut dose-response relationship has been established between glyphosate exposure and tumor formation. In one study, male and female rats were administered glyphosate in the diet at doses up to 31.5 and 34.0 mg/kg/day, respectively, for 26 months. No increase in tumor formation was noted (Bio/dynamics, Inc., 1981b as cited in USEPA, 1992). In a 24-month chronic feeding study in mice exposed to levels up to 30,000 ppm glyphosate, no excess of tumors was noted. However, the EPA has classified this study as a chronic toxicity study rather than a cancer study because the study does not meet the specific guidelines for a cancer study established by EPA (USEPA, 1986 as cited in USEPA, 1992). Another cancer study, in which rats were fed glyphosate at concentrations of 2,000, 8,000 and 20,000 ppm for 24 months revealed an increased incidence of adenomas (i.e., benign tumors) of the pancreas, thyroid and liver. Although no dose-response relationship was established and the tumors did not progress from adenomas to carcinomas (malignant tumors), the EPA has recommended that the carcinogenic effects of glyphosate be addressed by a peer review committee (USEPA, 1992). In an 18-month carcinogenicity study, mice were fed diets containing 1, 150, 750 or 4500 mg/kg/day of glyphosate. No effects were observed in the low and mid-dose groups. Effects noted in the high-dose group included decreased body weight gain in males and females, various liver and kidney effects as well as slightly increased incidence of renal tubular adenomas, a rare tumor, in males. The EPA concluded that occurrence of these adenomas was spontaneous rather than compound-induced because the incidence of renal tubular adenomas in males was not statistically significant when compared with the concurrent controls. After extensive evaluation, an independent group of pathologists and biometricians concurred with this conclusion. Therefore, glyphosate was not considered to be carcinogenic in this study.

In 1988, an EPA Science Advisory Panel labeled glyphosate as a D carcinogen under the old EPA cancer classification system, indicating that it is “not classifiable as to human carcinogenicity” based on a lack of statistical significance and uncertainty as to a treatment-related effect (Doyle, 1996; USEPA, 1993b). Under the new EPA cancer classification system using descriptors, a designation of D corresponds to the descriptor, “Data are inadequate for an assessment of human carcinogenic potential”. On June 26, 1991, the EPA Office of Pesticide Programs (OPP) labeled glyphosate an E carcinogen (again, based on the old EPA cancer classification system) based on a lack of convincing evidence of carcinogenicity in adequate studies with two animal species, rat and mouse. An E classification is EPA's most favorable category and is given to compounds for which there is "evidence of noncarcinogenicity in humans" (McLaren/Hart, 1995). The EPA Integrated Risk Information System (IRIS) database still lists the 1988 D cancer classification. However, the most recent EPA classification is the OPP 1991 designation of E. Under the new EPA cancer classification system, a designation of E corresponds to the descriptor, “not likely to be carcinogenic to humans”.

Available Toxicity Criteria:

The EPA has developed several Drinking Water Health Advisories for glyphosate. Health Advisories are defined as concentrations of a substance in drinking water estimated to have negligible deleterious effects in humans, when ingested for a specified period of time. These values include a ten-day health advisory for a child of 20 mg/l as well as a lifetime health advisory of 1 mg/l for a child and 4 mg/l for a 70-kg adult (USEPA, 1988).

The EPA has also developed a Maximum Contaminant Level Goal (MCLG) for drinking water and has promulgated this value as a Maximum Contaminant Level (MCL) standard (USEPA, 1993b; USEPA, 1995). Massachusetts has adopted this value as a drinking water standard, known as a Massachusetts Maximum Contaminant Level (MMCL).

In addition, the EPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) RfD/RfC workgroup has developed an oral Reference Dose (RfD) of 0.1 mg/kg/day for glyphosate based on the three-generation rat reproduction study conducted by Monsanto cited earlier. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1993b). The EPA Office of Pesticide Programs (OPP) has developed an RfD of 2.0 mg/kg/day. The World Health Organization (WHO) has developed an RfD of 1.75 mg/kg/day (USEPA, 1995b).

ECOLOGICAL TOXICITY

Aquatic Organisms:

Glyphosate has very low toxicity in aquatic fish and invertebrates. A range of 96-hr LC50 values identified for fish exposed to a formulation of glyphosate were reported to be greater than 1,000 mg/l for a number of species including carp, rainbow trout, bluegill, sunfish and harlequin fish (WSDOE, 1992 as cited in McLaren/Hart, 1995). Another source cites an LC50 greater than 10,000 mg/l for carp. Values over 1,000 mg/l are considered an insignificant hazard (Christensen, 1976 as cited in McLaren/Hart, 1995). Reported 96-hour LC50s for technical grade glyphosate include values ranging from 86 mg/l for rainbow trout to 168 mg/l for harlequin fish. Reported LC50s for technical glyphosate for other invertebrate species include values ranging from >10 mg/l for American oyster larvae to 934 mg/l for a fiddler crab, with the LC50s for *Daphnia magna*, honeybee, shrimp and *Chironomus plumosus* falling in between (WSDOE, 1992; McKee, pers. comm., 1996). A value greater than 10 is considered only slightly toxic (Christensen, 1976 as cited in McLaren/Hart, 1995). The EPA AQUIRE database lists reported LC50s for unspecified forms of "glyphosate" ranging from a 4-hr LC50 value of 1.3 mg/l for rainbow trout to a 4-hr LC50 value of 25,605 mg/l for goldfish (EPA, 1995).

Plants:

Since glyphosate is a broad spectrum herbicide, it is effective on a large number of annual and perennial grasses, broadleaf weeds, sedges, rushes and woody plants as well as ditchbank or shoreline aquatic weeds. Glyphosate is not effective on plants that are completely submerged or which have most of their foliage under water (Monsanto, 1981 as cited in WSDOE, 1992). Because of its widespread effects, glyphosate may affect non-target plants. As with all herbicides, use of glyphosate should be coordinated as part of an overall management plan to control vegetation in an organized manner. Such a plan is particularly important when the objective is the control of large areas of vegetation such as phragmites, cattails or purple loosestrife due to the potential for simultaneous die-off. This die-off could result in oxygen depletion due to rapid decomposition of organic matter, resulting in widespread

nonspecific destruction of plant life in addition to fish kills and the proliferation of microfauna and flora which are harmful to waterfowl (WSDOE, 1992 as cited in McLaren/Hart, 1995).

Table III.4-2. Properties of Glyphosate

CAS #:	1071-83-6
Synonyms	isopropylamine salt; n-(phosphonomethyl)glycine
Molecular formula	$C_3H_8NO_5P$
Molecular weight	169.1
Physical properties	solid, white, odorless
Melting point	200^oC
Density	0.5 gm/cc for pure chemical
Vapor pressure	negligible
Photolysis half-life	stable
Hydrolysis half-life	stable
Biodegradation half-life	60 days (soil)
Dissipation half-life	1.5-14 days
K_{ow}	5.6×10^{-4}
K_{oc}	High
BCF	Low
Water Solubility	1.2×10^4

(WSSA, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988)

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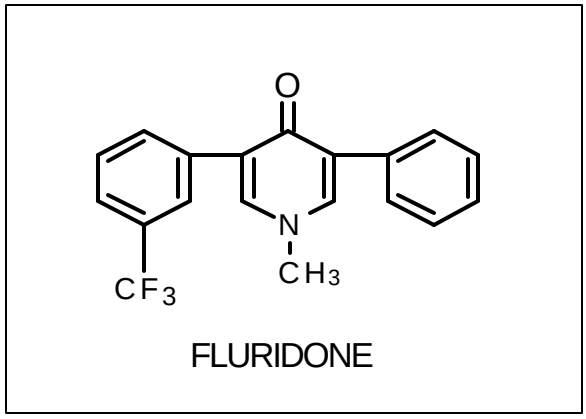
III.5 FLURIDONE



FLURIDONE

SUMMARY

Fluridone (1-methyl-3-phenyl-5-[3-(trifluoromethyl)phenyl]-4(1H)-pyridinone) is a selective systemic aquatic herbicide used to control primarily broad-leaved, submerged aquatic macrophyte species including Eurasian watermilfoil, curly-leaf pondweed as well as native pondweeds (McLaren/Hart, 1995). It is used to treat ponds, lakes, reservoirs, canals and rivers. Fluridone is stable to oxidation and hydrolysis (McCowen *et al.*, 1979 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Volatilization of fluridone is insignificant (Muir and Grift, 1982 as cited in Aquatic Plant Identification Guide, 1988). Breakdown of fluridone in the aquatic environment occurs mostly through photolysis. Other fate processes include plant uptake and adsorption to soil and suspended colloids (Joyce and Ramey, 1986). Some microbial degradation of fluridone has also been reported (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). Fluridone is taken up in fish but studies demonstrate that fish tissue concentrations generally reflect water concentrations and that fish concentrations rapidly clear when fluridone residues are removed from the water (West *et al.*, 1983 and Muir and Grift, 1982 as cited in McLaren/Hart, 1995). There are no restrictions on the use of fluridone to treat water used for swimming or domestic purposes. Care should be taken when applying Fluridone within one-fourth mile of any potable water intake (WSDOE, 1992).

The U.S. Environmental Protection Agency (USEPA) approved the label for Sonar on March 31, 1986 (McLaren/Hart, 1995).

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing fluridone that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

FLURIDONE USES AND APPLICATION

Fluridone is used to manage aquatic vegetation in fresh water ponds, lakes, reservoirs, canals and rivers (Cockreham, pers. comm., 1996). It is absorbed from the water by the shoots of submerged plants and from the hydrosoil by the roots of aquatic vascular plants. The effectiveness of fluridone depends on the degree to which the herbicide maintains contact with plants. Rapid water movement or any dilution of this herbicide in water will reduce its effectiveness (Dow Elanco, 1992; Aquatic Plant Identification and Herbicide Use Guide, 1988; WSDOE, 1992).

Application of fluridone may be made in several ways depending on the formulation used. The liquid suspension may be applied as a spray to the water surface, subsurface or along the bottom of the water body using specialized equipment. The pellet can be spread on the water surface (WSSA, 1983). Water should be used as a carrier during application of the liquid fluridone suspension. No surfactant is specified for use during application.

When treating ponds, application should be made to the entire water body. When treating lakes and reservoirs, plots no smaller than ten surface acres should be treated. In addition, areas with a large linear aspect (such as boat lanes and narrow shorelines) should not be treated (Aquatic Plant Identification and Herbicide Use Guide, 1988).

Application of fluridone may be made prior to active growth of aquatic weeds or any time during the spring or summer when weeds are visible (WSSA, 1983; Aquatic Plant Identification and Herbicide Use Guide, 1988).

Caution should be used when applying fluridone within one-fourth mile of any functioning potable water intake.

The plant selectivity of fluridone is dependent upon dose, application timing and formulation used. For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. A list of the weeds that these products control, which has been compiled from the Environmental Protection Agency (EPA) registration labels for these products, is contained in Table III.5-1.

MECHANISM OF ACTION

Fluridone produces its toxic effect in plants by inhibiting synthesis of carotenes (pigments that protect chlorophyll molecules from photodegradation). The absence of carotenes causes degradation or "bleaching" of chlorophyll by sunlight from plants. Plants become whitish-pink or chlorotic at growing points and die slowly. This slow dying-off of plants (i.e., 30-90 days) (Cockreham, pers. comm., 1996) reduces the instantaneous oxygen demand caused by plants dying off and decomposing all at once (Joyce and Ramey, 1986). The herbicidal effects of fluridone usually appear within 7-10 days. Species susceptibility to fluridone may vary depending on time of year, stage of growth and water movement (McLaren/Hart, 1995).

Table III.5-1. List of Aquatic Plants Controlled by Fluridone

Common Name	Scientific Name
American Lotus	*Nelumbo lutea*
Bladderwort	*Utricularia* spp.
Common Coontail	*Ceratophyllum demersum*
Common Duckweed	*Lemna minor*
Common Elodea	*Elodea canadensis*
Egeria, Brazilian Elodea	*Egeria densa*
Fanwort	*Cabomba caroliniana*
Hydrilla	*Hydrilla verticillata*
Naiad	*Najas* spp.
Pondweed (except Illinois)	*Potamogeton* spp.
Watermilfoil (including Eurasian Watermilfoil)	*Myriophyllum* spp. (including *M. spicatum)*
Spatterdock	*Nuphar* spp.
Waterlily	*Nymphaea* spp.
Waterprimrose (including Waterpurslane)	*Ludwigia* spp. (including *Ludwigia palustris*)
Watershield	*Brasenia schreberi*

(McLaren/Hart, 1995; SePRO, 1994)

ENVIRONMENTAL FATE/TRANSPORT

The major fate process affecting fluridone persistence in aqueous environments is photolysis. Thus any factors which affect sunlight intensity and/or penetration of light into the water column will affect the dissipation rate of fluridone (Joyce and Ramey, 1986). Other factors affecting dissipation include geographic location, date of application, water depth, turbidity, weather and weed cover (West *et al.*, 1983 as cited in McLaren/Hart, 1995). Microbial degradation is also reported to occur in laboratories, but photolysis generally occurs much more quickly (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). Other secondary fate processes include adsorption to soil and suspended colloids and plant uptake (Joyce and Ramey, 1986).

Fluridone will adhere to sediment particles/organics in the sediment. Eventually, the fluridone will desorb and photodegrade into the water column from the hydrosoil (Elanco, 1981 as cited in McLaren/Hart, 1995). The pH of the water can affect this rate (with the lower the pH, the higher the adsorption rate (Malik and Drennan, 1990 as cited in McLaren/Hart, 1995).

Fluridone is taken up in fish tissue. Fluridone fish concentrations generally reflect the concentrations of fluridone in the water (McLaren/Hart, 1995). When fluridone residues are removed from the water

column, the fluridone concentration from fish tissue clears (West *et al.*, 1983; Muir *et al.*, 1983 as cited in McLaren/Hart). Based on a low bioaccumulation rate in fish and high levels of fluridone necessary to produce toxic responses in mammals and birds, it is not expected that fish-eating animals would be affected by fluridone used at recommended (registered) application rates (McLaren/Hart, 1995).

The primary metabolite of fluridone degradation in fish was identified as 1-methyl-3-(4-hydroxyphenol)-5-[3-trifluoromethyl)phenyl]-4[1H]-pyridone (West *et al.*, 1983 as cited in McLaren/Hart, 1995). This compound was also identified as a minor metabolite in water and hydrosoil (Muir and Grift, 1982 as cited in McLaren/Hart, 1995). 1,4-dihydro-1-methyl-4-oxo-5-[3-(trifluoromethyl)phenyl]-3-pyridone was also identified as the major hydrosoil metabolite in hydrosoil studies conducted in the laboratory; however, this compound has not been identified in the hydrosoil of small ponds under natural conditions (West *et al.*, 1983 as cited in McLaren/Hart, 1995). A number of other metabolites including benzaldehyde, 3-(trifluoromethyl)-benzaldehyde, benzoic acid and 3-(trifluoromethyl)-benzoic acid were produced as photolytic breakdown products in one laboratory study (Saunders and Mosier, 1983, as cited in McLaren/Hart, 1995). N-methylformamide (NMF) was produced in another study. However, NMF has not been identified as a breakdown product under natural conditions (Saunders and Mosier, 1983 as cited in McLaren/Hart, 1995).

The half-life of fluridone in water of small, artificial ponds ranged from 1-7 days. In hydrosoils, the compound persisted for 8 weeks to one year (Joyce and Ramey, 1986; WSDOE, 1992). Fluridone has a water solubility of 12 mg/l and an octanol-water partition coefficient (K_{ow}) of 74.1 (Elanco Products Company, 1985 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988). Fluridone is stable to oxidation and hydrolysis (McCowen *et al.*, 1979). Volatilization of fluridone is not expected to be a significant process, (Muir and Grift, 1982 as cited in Aquatic Plant Identification and Herbicide Use Guide, 1988).

PHARMACOKINETICS

Metabolism and distribution studies have shown that fluridone is absorbed and excreted in the feces within 72 hours of oral administration to rats (McLaren/Hart, 1995). No bioaccumulation of fluridone was noted. 90% of the absorbed fluridone was cleared in 96 hours (USEPA, 1988).

HEALTH EFFECTS

Avian:

Fluridone has very low toxicity to birds. A number of acute toxicity studies were conducted in various bird species. An oral LD50 value of >2,000 mg/kg was obtained for bobwhite quail. The EPA considers this value to represent slight toxicity (USEPA, 1986). An LD50 of >2,000 was identified for mallard ducks (WSDOE, 1992). Oral LC50 values of > 5,000 ppm were identified for bobwhite quail and mallard duck (USEPA, 1986). No impairment on reproduction for the above species was noted up to a dietary exposure concentration of 1,000 ppm (USEPA, 1986). In other studies, an LC50 value of about 10,000 ppm was identified for bobwhite quail and an LC50 value of >20,000 ppm was identified for mallard duck (WSDOE, 1992).

Mammalian:

Acute:

Most of the available information on the toxic effects of fluridone comes from studies conducted by the industry on various formulations of the product. Generally, the acute toxicity of fluridone is low. The LD_{50} for both rats and mice exposed through ingestion to technical grade fluridone is

greater than 10,000 mg/kg. The oral LD_{50}s for cats and dogs exposed to technical grade fluridone are 250 mg/kg and 500 mg/kg, respectively. The LD_{50} for rabbits exposed through the skin to technical grade fluridone is greater than 2,000 mg/kg (Elanco, 1981 as cited in McLaren/Hart, 1995).

Fluridone was found to produce eye irritation in rabbits with effects including redness, corneal dullness and conjunctivitis. Fluridone was found to be neither irritating nor a sensitizer to rabbit skin at 2,000 mg/kg (USEPA, 1988).

Subchronic/Chronic:

In a three-week study in which fluridone was applied to rabbit skin daily at doses ranging from 192-768 mg/kg/day, dose-dependent skin irritation was produced at all doses. No systemic effects were noted at any dose. An increase in organ weight was noted at 384 mg/kg/day (USEPA, 1988).

In a three-month subchronic feeding study with fluridone, no treatment-related effects were noted in rats administered doses of 62 mg/kg or in mice administered doses of 330 mg/kg (Elanco, 1981 as cited in McLaren/Hart, 1995). A dietary level of fluridone of 16.5 mg/kg/day administered to mice for three months resulted in a partial enlargement of livers. A dietary level of 166 mg/kg administered to rats for three months resulted in an increase in liver weights. A No Observed Effect Level (NOEL) of 30 mg/kg/day was identified in rats administered fluridone in the diet for three months (USEPA, 1988). A concentration of 0.033% of fluridone fed to mice for three months produced morphologic changes in the liver and an increase in absolute liver weights in male mice (USEPA, 1988). In a study conducted with dogs, daily dietary fluridone levels up to 200 mg/kg/day did not result in any treatment-related effects (Elanco, 1978 as cited in USEPA, 1990).

In a one-year chronic study in which dogs were administered fluridone by capsule in food, a number of effects including weight loss, an increase in liver weight and an increase in liver enzymes were noted at a dose level of 150 mg/kg/day. A NOEL of 75 mg/kg/day was identified (USEPA, 1988). In a two-year feeding study in which mice were administered fluridone concentrations in the diet of up to 330 ppm fluridone, there was an increase in liver enzymes in males exposed at 330 ppm. No other toxic effects or lesions were noted at any of the doses (USEPA, 1988). In another two-year study, rats were exposed to doses of 0, 8, 25 and 81 mg/kg/day. At 25 mg/kg/day, rats experienced inflammation in the kidney, atrophy of the testes, inflammation of the cornea, weight loss and decreased organ weights (USEPA, 1988; USEPA, 1990).

Developmental/Reproductive:

In a study in which rats were exposed to up to 200 mg/kg/day of fluridone, neither maternal nor fetotoxic effects were noted (USEPA, 1988). In a three-generation study conducted in rats exposed to fluridone at a dose of 100 mg/kg/day, no teratogenic or maternal effects were noted. However, a dose of 100 mg/kg/day was found to be toxic to rat pups (USEPA, 1988; USEPA 1990). In a teratology study in which rabbits were exposed to fluridone doses of up to 750 mg/kg/day, a level of 300 mg/kg resulted in maternal effects including a decrease in body weight gain and abortion. Fetal effects, also noted at this level, included resorptions (USEPA, 1988). No teratogenic effects were noted (USEPA, 1990). In a pilot study in which rabbits were exposed to fluridone at doses of 0, 250, 500, 750 and 1,000 ppm, a maternal NOEL of 500 mg/kg was identified. A level of 750 mg/kg produced a maternal loss in body weight. A NOEL of 250 mg/kg/day was identified for fetal effects. At 500 mg/kg/day, fetal resorptions occurred (USEPA 1988). In another study, rats were administered doses by oral gavage of 0, 100, 300 and 1,000 mg/kg/day. A maternal NOEL of 100 mg/kg/day was identified. At 300 mg/kg/day, there was a decrease in maternal body weight. The NOEL for developmental effects was identified as 300 mg/kg/day. At 1,000 mg/kg/day, fetal effects included a

decrease in fetal weight and delayed ossification. The NOEL for teratogenic effects was greater than 1,000 mg/kg/day (USEPA 1988).

Mutagenicity:

Fluridone was not found to be mutagenic in several test assays. Fluridone produced negative results in the Ames assay and did not induce sister chromatid exchange in Chinese hamster bone marrow cells. In addition, fluridone did not promote unscheduled DNA synthesis in rat hepatocytes (USEPA, 1988).

Carcinogenicity:

Based on negative cancer findings in the two chronic toxicity studies discussed above, there is no evidence indicating that fluridone is carcinogenic. The EPA Health Effects Division has designated fluridone as a Group E carcinogen (i.e., having evidence of noncarcinogenicity for humans) by the old EPA classification system. Under the new cancer classification system (USEPA, 1995), an E classification would correspond to a weight-of-evidence descriptor of "not likely to be carcinogenic to humans"..

Available Toxicity Criteria:

The EPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) (RfD/RfC) workgroup has developed an oral Reference Dose (RfD) of 0.08 mg/kg/day for fluridone based on one of the two-year rat feeding studies conducted by Elanco cited earlier (USEPA, 1990). The EPA Office of Pesticide Programs (OPP) has calculated the same RfD value based on the same study (USEPA, 1995). The RfD is an estimate, (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1990).

The EPA has designated an acceptable residue level for fluridone in potable water of 0.15 ppm. This level is based on the maximum application rate for fluridone as registered under FIFRA (Federal Insecticide, Fungicide and Rodenticide Act) (USEPA, 1986 as cited in McLaren/Hart, 1995). The EPA has also established a tolerance of 0.5 ppm for residues of fluridone and its primary metabolites in fish and crayfish. In addition, EPA has established tolerances for crops irrigated with water containing fluridone residue concentrations at 0.15 ppm as well as for a number of raw, agricultural commodities (USEPA, 1986 as cited in McLaren/Hart, 1995).

ECOLOGICAL TOXICITY

Aquatic Organisms :

A number of studies have been conducted with fluridone to determine the LD50 or LC50 values for a variety of organisms. The LD50 (or LC50) is the dose (or concentration) to which a particular species is exposed, which results in the death of 50% of the test population. The EPA has cited the results of a number of these studies. EPA considers these studies to demonstrate moderate toxicity. These studies are listed in the Table III 5-2.

In addition, a Maximum acceptable theoretical concentration (MATC) value for fathead minnow (second generation fry) was calculated to be between 0.48 mg/l and 0.96 mg/l, meaning no treatment-related effects were noted at or below 0.48 mg/l. Total length of 3-day old fry was reduced at 2 mg/l fluridone (USEPA, 1986).

No adverse effects were noted on crayfish, bass, bluegill, catfish, long-neck soft-shelled turtles, frogs, water snakes and waterfowl from the use of 0.1 to 1.0 ppm fluridone during field experiments (Arnold, 1979, McCowen et al., 1979 as cited in WSDOE, 1992). Application of 1.0 ppm fluridone to zooplankton caused a reduction in population, but the population quickly recovered. Application of 0.3 ppm did not cause a change in the total number of benthic organisms whereas application of 1.0 ppm did cause a change (Parka *et al.*, 1978 as cited in WSDOE, 1992). An aqueous solution of fluridone caused a reduction in population of the amphipod *Hyalella azteca* when applied at a rate of 1.0 ppm but not when applied at a rate of 0.3 ppm (Arnold, 1979 as cited in McLaren/Hart, 1995). Fish abundance and community structure remained unchanged in ponds exposed to a fluridone concentration level of 0.125 ppm (Struve *et al.* 1991 as cited in McLaren/Hart, 1995). LC50 values for a variety of microscopic crustaceans including *Diaptomus*, sp., *Eucyclops* sp, *Alonella* sp., and *Cypria* sp., ranged from 8.0 - 13.0 ppm (Naqvi and Hawkins, 1989 as cited in McLaren/Hart, 1995).

Table III.5-2. Acute Toxicity Tests

SPECIES	TEST TYPE	VALUE
Daphnia magna	48-hr LC50	6.3 mg/l
Bluegill	96-hr LC50	12 mg/l
Rainbow trout	96-hr LC50	11.7 mg/l
Sheepshead minnow	96-hr LC50	10.91 mg/l
Oyster embryo larvae	48-hr LC50	16.51 mg/l

(USEPA, 1986)

One group of investigators conducted extensive acute toxicity tests on a variety of aquatic invertebrates including amphipods, midges, daphnids, crayfish, blue crabs, eastern oysters and pink shrimp. The average 48-hour or 96-hour LC50 or EC50 (concentration at which 50% of the organisms exhibit an effect) was calculated as 4.3 $\pm$ 3.7 ppm (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995). The same investigators also conducted studies with a variety of fish including rainbow trout, fathead minnows, channel catfish, bluegills and sheepshead minnows. A 96-hour LC50 value of 10.4$\pm$3.9 was calculated (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995).

Daphnids, amphipods and midge larvae exposed chronically to fluridone concentrations of 0.2, 0.6 and 0.6 ppm as well as catfish fry exposed to fluridone concentrations of 0.5 ppm showed no treatment-related significant effects. Exposure to concentrations of 1 ppm produced a decreased growth rate of catfish fry and concentrations of 0.95 and 1.9 ppm produced a decreased survival rate of fathead minnows within 30 days after hatching (Hamelink *et al.*, 1986 as cited in McLaren/Hart, 1995).

Plants:

Fluridone selectively controls a number of broad-leaved submerged and floating aquatic macrophyte species as specified by its EPA label. In addition, the literature contains reports of fluridone's variable efficacy in controlling other species. The efficacy of fluridone is very dependent on contact time with plants. Thus, fluridone should be applied during periods of minimum water movement. Factors related to fluridone's variable efficacy include temperature, pH and light levels (Wells *et al.* 1986 as cited in WSDOE, 1992). In addition, one investigator found that in *Hydrilla* exposed to fluridone at various concentrations for 1, 3 and 5 weeks, plant recovery was directly related to the concentration of active iron (Fe^{2+}) in the plant at the time of treatment (Spencer and Ksander, 1989 as cited in WSDOE, 1992).

Fluridone did not appear to adversely affect desirable phytoplankton but some reduction in population of the less desirable species given as *Anabaena* and *Anacystis* occurred upon application of fluridone at levels of 0.3 and 0.1 ppm (Parka et al, 1978 as cited in WSDOE, 1992). A drastic reduction in phytoplankton population in Greek ponds including the disappearance within two months of a population of Cyanophyceae (Cyanobacteria) occurred after fluridone application. Diatom populations, a more desirable species, increased significantly, especially epiphytic and benthic species (Kamarianos *et al.*, 1989 as cited in WSDOE, 1992). No sufficient reduction in phytoplankton densities was noted when they were consistently exposed to a fluridone concentration of 0.125 ppm (Struve *et al.*, 1991 as cited in McLaren/Hart, 1995).

An aqueous solution of fluridone applied at a concentration of 1.0 ppm produced a significant reduction in a zooplankton population whereas a concentration of 0.3 ppm had no effect. The 1.0 ppm population returned to pretreatment levels within 43 days (Arnold, 1979 as cited in McLaren/Hart, 1995).

Table III.5-3. Properties of Fluridone

CAS #:	59756-60-4
Synonyms:	1-methyl-3-phenyl-5-[3-(trifluoromethyl)phenyl-4(1H)-pyridinone;
Molecular formula	$C_{19}H_{14}F_3NO$
Molecular weight	329.3
Physical properties	white, crystalline solid
Melting point	154-155^{o}C
Vapor pressure	< 1 x 10^{-7} mm Hg at 25^{o}C
Photolysis half-life	1-6 days
Hydrolysis half-life	stable
Biodegradation half-life	2-60 days (based on overall half-life)
K_{ow}	74.1 at 20^{o} C
K_{oc}	~350-2460 ml/g
BCF	0.9-15.5
Water solubility	12 mg/l at 25^{o} C and pH 7

(Reinert and Rodgers, 1987; WSSA, 1983; Aquatic Plant Identification and Herbicide UseGuide, 1988; WSSA, 1994)

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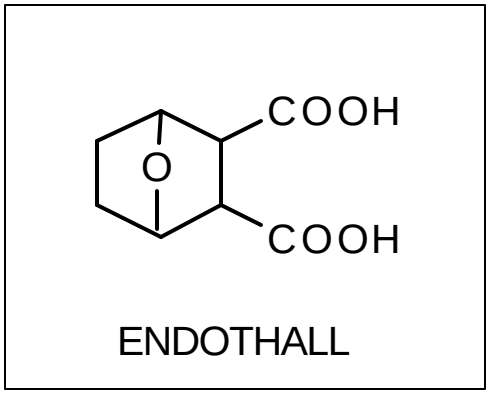
III.6 ENDOTHALL



SUMMARY

Endothall (7-oxabicyclo[2.2.1]heptane-2,3-dicarboxylic acid) is a relatively water-soluble contact herbicide, primarily used for the control of submersed weeds. Endothall exhibits a relatively short persistence time in the aquatic environment, usually undergoing complete degradation by microbial action in 30-60 days (USEPA, 1992a). Endothall does not adsorb to sediments nor does it bioconcentrate in aquatic organisms to any appreciable degree.

Two derivatives of endothall are available for aquatic weed control. These include the mono(N,N-dimethylalkylamine) salt and the dipotassium salt. Formulations containing the monoamine salt are particularly effective against filamentous algae but are more toxic to fish; thus they should not be used in areas where fisheries resources are important. Formulations containing the dipotassium salt exhibit a lower organism toxicity and are more appropriate for use in important fisheries areas.

The aquatic herbicidal properties of endothall were first suggested in the mid 1950s by its manufacturer at the time, Pennwalt Corporation. Actual development of endothall for this use started in 1958 (Elf Atochem, 1993a).

Endothall toxicity as noted in animal studies ranges from dermal and eye irritation, respiratory failure and hemorrhaging of the gastrointestinal tract upon exposure to high concentrations for a short period of time to effects on the liver and kidney upon longer-term exposure. There is no conclusive evidence indicating that endothall is either teratogenic, fetotoxic, mutagenic or carcinogenic.

Many studies have been conducted with the various endothall formulations addressing both toxicity and environmental fate and persistence. The U.S. Environmental Protection Agency (EPA) requires that endothall be reregistered under the 1988 amendments to FIFRA (Federal Insecticide, Fungicide and Rodenticide Act). Endothall is currently still under review. A number of studies relating to the toxicity of endothall to aquatic organisms as well as to its environmental persistence were submitted to EPA to fulfill some of the requirements of the reregistration process. The results of many of these studies are cited in this report; however, none of these studies has been critically reviewed by Massachusetts.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing endothall that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

ENDOTHALL USES AND APPLICATION

Both the monoamine salt formulation and the dipotassium salt formulation are manufactured for use in lakes and ponds to control aquatic vegetation. For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. A list of the weeds that these products control, which has been compiled from the Environmental Protection Agency (EPA) registration labels for these products, is contained in Table III.6-1.

Endothall applications should be made soon after emergence of new vegetative growth. Water temperature should be at least 65° F (18° C) prior to application. Although the EPA registration labels for these products do not recommend that any specific adjuvants be used during application, the following adjuvants have been suggested elsewhere: with salt formulations, polymeric adjuvants are recommended to aid in sinking the herbicide for underwater applications (e.g., with dipotassium salt formulations, Nalquatic and with monoamine salt formulations, Nalcotrol II). For the liquid formulations of these herbicides, invert emulsions are recommended to improve spreading and penetration of droplets, resist washoff and reduce evaporation and drift (Aquatic Plant Identification and Herbicide Use Guide, 1988).

MECHANISM OF ACTION

Endothall's herbicidal mode of action is not clear. Several mechanisms have been postulated. It is known that endothall interferes with plant protein synthesis in some way (Aquatic Plant Identification and Herbicide Use Guide, 1988). In addition, endothall affects lipid synthesis and dipeptidase and proteinase activities (Mann and Pu, 1968; Mann *et al.*, 1965 as cited in MacDonald *et al.*, 1993). 5 µg/l of endothall caused an approximate 40% inhibition of incorporation of malonic acid into the lipid fraction of hypocotyl segments of the hemp plant (*Sesbania exaltata*) (Mann and Pu, 1968 cited in USEPA, 1988). It has been suggested that endothall produces a number of cell membrane changes that cause drying and wilting of leaf tissue and an increased respiratory rate in plants (Maestri and Currier, 1966 cited in USEPA, 1988). It has also been postulated that endothall acts to inhibit respiration. This was noted in a study in which the effect from endothall is greater in the dark, indicating the mechanism of action is not light-dependent. Under light conditions, photosynthesis provides some energy for respiration; however, all energy under dark conditions is produced via respiration. Thus, it was suggested that this effect may be due to respiratory inhibition by endothall (MacDonald et al, 1993). It is also postulated that endothall interferes with metabolism of molecules involved in genetic coding (e.g., mRNA metabolism) (MacDonald et al, 1993).

ENVIRONMENTAL FATE/TRANSPORT

The fate and transport patterns of endothall in aquatic environments are similar for both the potassium and monoamine salt formulations (Aquatic Plant Identification and Herbicide Use Guide, 1988). Endothall is generally reported to be stable to oxidation, chemical hydrolysis and photolysis and not very volatile. However, in one study by the manufacturer, ^{14}C-labeled technical endothall, which was found to be stable to photolysis by xenon at pHs of 7 and 9, had a half-life of less than 24 hours at a pH of 5 (although the labeled endothall could not be accounted for). In another study by the manufacturer, the same compound exposed to xenon was stable at pH 5. In addition, the manufacturer also found that while technical endothall is stable to hydrolysis at pHs of 5 and 9, it breaks down with a half-life of 2825 days at pH 7. The above studies were all submitted by the manufacturer to the EPA to fulfill requirements for reregistration. These studies have not been reviewed by Massachusetts (Atochem, 1991a, 1991b, 1992a).

Endothall is also not expected to bioaccumulate or adsorb to suspended solids or sediments as indicated by very low octanol/water partition coefficients (K_{ow}). (See Table III.6-3: Properties of

Endothall). The dominant fate processes affecting endothall in the aquatic environment are biotransformation and biodegradation via microbial action. A three phase clearance mechanism for endothall in the environment has been postulated. These include an initial, rapid rate where the endothall is temporarily adsorbed to sediments. The second phase, involving microbial metabolism, is considerably slower. The third phase consists of an intermediate rate of disappearance attributed to the proliferation of microorganisms with the ability to degrade endothall (Sikka and Rice, 1973 as cited in Aquatic Plants Management Program for Washington State, 1992).

Under aerobic conditions, endothall biodegrades rapidly in the aquatic environment, with a half-life of about one week or less (HSDB, 1994). Under anoxic conditions, the biodegradation half-life is longer. The manufacturer has determined a half-life of 10 days for the biodegradation of endothall dipotassium salt in water under anaerobic conditions (Atochem, 1993a). Other factors that affect endothall biodegradation include the presence of organic matter, plant tissue and microorganism populations (State of Wisconsin, 1990 as cited in Aquatic Plants Management Program for Washington State, 1992). Biotransformation of endothall occurs mainly by the tricarboxylic acid cycle after splitting of the oxabicyclo ring. Glutamic acid is the primary breakdown product. Minor metabolites include aspartic and citric acids, alanine, phosphate esters (not positively identified) and an unidentified product (HSDB, 1994 as cited in Sikka and Saxena, 1973). The importance of microbial action on endothall breakdown was illustrated in a study in which 2 ppm of endothall added to pond water resulted in no apparent degradation of endothall in autoclaved (sterilized) water after 9 days; yet the same amount added to non-autoclaved water resulted in 50% degradation after 4 days (Sikka and Rice, 1973 as cited in HSDB, 1994).

Table III.6-1. List of Weeds Controlled by Endothall

Bass Weed *(Potamogeton amplexifolius)*	*Potamogeton diversifolius*
Bur Reed *(Sparganium, spp)*	*Potamogeton filiformis*
Coontail *(Ceratophyllum spp)*	*Potamogeton pusillus*
Milfoil *(Myriophyllum spp)*	Water Star Grass *(Heteranthera spp*)
Bushy Pondweed *(Najas spp)*	Water celery *(Vallisneria americana)*
Curly-leaf Pondweed *(Potamogeton crispus)*	Canadian Waterweed *(Elodea)*
Flat-Stem Pondweed *(Potamogeton zosteriformis)*	Filamentous Green Algae *(Cladophora, Pithophora, Spirogyra)*
Floating-Weed Pondweed *(Potamogeton natans)*	Stonewort, Muskgrass (*Chara)*
Horned Pondweed *(Zannichellia spp)*	
Sago Pondweed *(Potamogeton pectinatus)*	
Potamogeton nodosus	

Endothall applied to ponds at rates ranging from 0.3-10 ppm was undetectable after an average of 2.5 days and a maximum of 4 days (Simsiman *et al.*, 1976 as cited in HSDB, 1994). Fifty-five percent of a 1.2 ppm application of endothall added to another pond was removed after 12 days (Frank, 1972 as cited in HSDB, 1994). In other studies, an overall half-life of 4 days was reported in experimental greenhouse pools treated with 0.3 to 1.4 ppm endothall (Reinert *et al.*, 1985). In farm reservoirs, about 71% of

endothall applied at rates ranging from 0.3-1.4 ppm was removed after 12 days (Simsiman *et al.*, 1976 as cited in HSDB, 1994). Endothall added to the water of irrigation supply ponds at a concentration of 2 ppm decreased linearly with the predicted concentration of zero at 26 days (half-life 12 days) (Langeland and Warner, 1986 as cited in HSDB, 1994). Only 28% removal of endothall was achieved 30 days after addition of endothall to anoxic water (Simsiman *et al.*, 1976 as cited in HSDB, 1994).

Endothall does not significantly bioconcentrate in organisms. Consistently low endothall levels have been observed in many laboratory and field studies. Based on a water solubility of 100,000 mg/l at 20°C, a bioconcentration factor (BCF) of <1 was estimated for endothall as a function of its octanol-water partition coefficient (K_{ow}) (Lyman *et al.*, 1982). A BCF of 10 for mosquito fish was observed in a modified Metcalf model ecosystem (Insensee, 1976 as cited in Reinert and Rodgers, 1987). In a field study, a 5 mg/l dipotassium endothall water concentration resulted in BCFs ranging from 0.003-0.008 in bluegills. After 72 hrs in the above study, no endothall residue was detected in the fish flesh (Serns, 1977 as cited in Reinert and Rodgers, 1987). In several organisms, it was noted that endothall concentrations exceeded the water concentration of endothall by more than an order of magnitude. Calculated BCF values of 150 for the water flea, 63 for green algae and 36 for a snail); however, the residue concentrations were transient and were not passed along trophic levels (Insensee, 1976 as cited in Reinert and Rodgers, 1987).

PHARMACOKINETICS

Very little information exists regarding the pharmacokinetics of endothall in mammals. In rats given a single oral dose of about 5 mg/kg ^{14}C-labeled endothall, approximately 3% of the endothall was recovered as carbon dioxide in urine while 90% was recovered in the feces and 7% in the urine (Soo *et al.*, 1967 as cited in USEPA 1988). The rats had received 5 mg/kg of unlabeled endothall in the diet for two weeks prior to treatment with ^{14}C-labeled endothall. These results suggest that little gastrointestinal absorption took place. Studies in which deaths were induced in rabbits exposed to endothall directly in the eye or on the skin indicate the potential for absorption by these routes (Pharmacology Research, Inc., 1975a, 1975b as cited in USEPA, 1988, 1992a).

In rats receiving a single oral dose of 1.0 mg/kg ^{14}C-labeled endothall, the highest levels of ^{14}C after one hour were detected in the stomach and intestines (~ 95%), liver (~1.1%) and kidney (0.9%) and 0.02-0.1% in heart, lung, spleen and brain). Within 48-72 hours, endothall levels in all tissues fell to below detection. Total excretion of the ^{14}C was over 95% complete after 48 hours and over 99% complete after 72 hours. In addition, no radioactivity was detected in rat pups of dams who had been given oral doses of ^{14}C-endothall. Thus, endothall is not expected to accumulate. The metabolism of endothall has not been determined (Soo *et al.*, 1967 as cited in USEPA 1988). Another study also demonstrated that endothall was poorly absorbed via the oral route. In rats given a single oral dose of endothall, approximately 89-98% of the dose remained in the gut and was excreted in the feces unchanged (Hallifax, 1990 as cited in WSDOE, 2001). Regardless of whether rats received a single dose or a dose delivered subchronically for fifteen days, both the absorbed and unabsorbed chemical were not metabolized but were excreted in urine and feces. Bile was a very minor excretory route for endothall (Hallifax, 1990 as cited in WSDOE, 2001).

HEALTH EFFECTS

Avian:

Several acute or short-term toxicity studies have been conducted with endothall to fulfill EPA registration requirements. These studies have not been reviewed by this office. An oral LD50 value of 344 mg/kg was determined in a 21-day study conducted with mallard ducks and a formulation of the dipotassium salt (Atochem, 1992b). 8-day acute dietary studies conducted using a dipotassium salt

formulation with both bobwhite quail and mallard ducklings indicated that the acute oral LD50 and the NOEL values were both greater than 5,000 ppm (Elf Atochem, 1994c, 1994d). Two 20-week oral toxicity and reproductive studies conducted using the technical acid of endothall with bobwhite quails and mallard ducks yielded NOELs of 250 ppm for quail and 50 ppm for duck (Elf Atochem, 1992d, 1992e).

Mammalian:

Acute/Subchronic:

The only available information in the literature addressing acute health effects of endothall to humans is a case history of a young male suicide victim who ingested 7-8 g of endothall in solution (about 100 mg endothall ion/kg). Effects included repeated vomiting, focal hemorrhages and edema in the lungs and gross hemorrhages of the gastrointestinal tract (Allender, 1983 as cited in USEPA, 1988).

Effects noted in animals exposed to high levels of endothall for a short period of time include cardiac arrest or respiratory failure as causes of death in dogs and rabbits injected with endothall at a concentration of at least 5 mg/kg (Goldstein, 1952; Srensek and Woodard, 1951 as cited in USEPA, 1988). The acute toxicity of the endothall acid appears to be greater than that of the salt forms usually used in herbicides. Acute oral LD50s in rats were reported to be 35-51 mg/kg for the acid form and 182-197 mg/kg for the sodium salt (Simsiman at al., 1976; Tweedy and Houseworth, 1976 as cited in USEPA, 1988).

Rats receiving about 40 or 400 mg/kg/day endothall ion in food for four weeks had slight liver degeneration and focal hemorrhaging in the kidney. Most of the rats receiving 400 mg/kg/day endothall died within a week (Brieger, 1953a as cited in USEPA, 1988).

Dogs that received 1-50 mg of disodium endothall/kg/day (0.8-40 mg endothall ion/kg/day) for 6 weeks died within 11 days (Brieger, 1953b as cited in USEPA, 1988). In the group given 20 mg/kg/day, vomiting and diarrhea occurred. Other health effects including pathological changes in the gastrointestinal tract (congested and edematous stomach walls and edematous upper intestines) were seen in all dogs. Erosion and hemorrhages of the stomach were noted with doses of at least 20 mg/kg/day.

Application of a 1% solution of endothall to the skin of rabbits resulted in no effects in unbroken skin and mild skin lesions in scarified skin. Application of 10% and 20% endothall solutions resulted in more serious effects, including necrosis and some animal deaths (Goldstein, 1952 as cited in USEPA, 1988). Dermal exposure of 6 rabbits to 200 mg endothall technical/kg resulted in the deaths of all of the rabbits within 24 hours of treatment (Pharmacology Research, Inc., 1975a as cited in USEPA, 1988).

Application of technical endothall to the eyes of rabbits produced severe eye irritation. Several rabbits died upon treatment, indicating that absorption of endothall took place through the eye (Pharmacology Research, Inc. 1975b as cited in USEPA, 1988). A number of acute and shorter-term toxicity studies in mammals have been conducted to fulfill EPA registration requirements. These studies have not undergone review by the Department. The studies are listed in Table III.6-2.

Table III.6-2. Acute/Short-Term Studies Submitted to the U.S. EPA (Elf Atochem)

SPECIES	DURATION	TYPE	RESULTS	REFERENCE
Dipotassium salt formulation:				
Rat	one dose	acute oral	LD50 = 99.5 mg/kg	Atochem, 1991c
rabbit	one dose	acute dermal	LD50 > 2,000 mg/kg	Atochem, 1991d
Rat	one dose	acute inhal.	LC50 = 0.83 mg/l (liquid aerosol)	Atochem, 1992c
rabbit	one dose at 0.1 ml	eye irrit.	Class I irrit.; one death	Atochem, 1991e
rabbit	one dose at 0.5 ml	skin irrit. (intact)	not irrit. at 0.5 ml	Atochem, 1992d
guinea pig	3 6-hr applics	dermal hyper-sensitivity	delayed contact hypersensi-tivity when induced at 5%, challenged and re- challenged at 2% in 80% ethanol	Atochem,1991f
rat	1x/d; 5 d/wk; 21 d	dermal toxicity	40 mg/kg	Atochem, 1992e
rat	1x/d; 5 d/wk; 21 d	dermal toxicity	3 deaths at 80 mg/kg; 10 deaths each at 200 mg/kg and 500 mg/kg	Elf Atochem, 1993b
Monoamine salt formulation:				
rat	one dose	pharmaco-kinetic	half-life (blood) at 0.9 mg/kg - 1.8 hrs (M) and 2.5 hrs (F); at 4.5 mg/kg - 13.9 hrs (M)	Pennwalt Corp., 1990a
rat	one dose	dermal tox.	<7% of applied doses were absorbed into systemic circulation	Pennwalt Corp., 1990b

Chronic:

A 2-year toxicity study conducted with sodium endothall in beagle dogs yielded no adverse effect at 2 mg endothall ion/kg/day and an increase in organ weights and organ/body-weight ratios of the stomach and small intestine at the mid- and high-dose groups (6 and 16 mg ae/day for 24 months, respectively). The effect at the mid-dose was considered to be due to the irritation potential of the chemical (Keller, 1965 as cited in USEPA, 1988). A similar 1-year toxicity study in beagle dogs produced changes in the portal tract of the liver and dose-related changes in stomach mucosa at 14.4 mg ion/kg/day. At 4.8 mg ion/kg/day, there were no observed effects on the liver and marginal injury to the stomach (Greenough *et al.*, 1987 as cited in USEPA, 1992a).

No adverse effects were reported in female rats given 100 mg ion/kg/day for 2 years (Brieger, 1953b as cited in USEPA, 1988).

A number of longer-term toxicity studies using disodium endothall have been conducted to fulfill EPA registration requirements. These studies have not been reviewed by Massachusetts for this report and are summarized as reported by the manufacturer. A series of range-finding tests and a 2-year toxicity study were conducted in beagle dogs. A overall dose-response chart was developed by the authors of these studies with doses ranging from a NOEL of 4 mg/kg/day, to a LOEL of 6 mg/kg/day with increasingly severe health effects, up to a dose level of 120 mg/kg/day producing severe anorexia, vomiting, decreased body weight and decreased food consumption, leading to sacrifice of the animals after 4 days due to their poor condition (Elf Atochem, 1992e).

The selection of the NOEL in both the above study and another study on carcinogenicity discussed below are based on stomach changes in the dog (Elf Atochem, 1992c). Thickening of the stomach wall was not considered a significant effect by the toxicologist evaluating the results of these studies based on the fact that such thickening was consistent with stomach findings produced by long-term treatment with prostaglandins. The toxicologist concluded that the effect was an adaptive response to the irritating properties of the constant ingestion of endothall and not an adverse effect (Elf Atochem, 1992c).

In rats fed 0, 5.3, 10.5, 31.5 or 63 mg ae/kg/day disodium endothall (12.6% endothall acid equivalent) for 24 months, rats in the three highest dose groups had dose-related decreases in body weight and body weight gains and decreased glucose levels. Gross necropsy revealed an increased incidence of thickening of both the glandular and non-glandular stomach at the three highest dose groups. Acanthosis and keratosis were seen in the gross stomach lesions. The LOAEL was 31.5 endothall acid equivalent/kg/day and the NOAEL was 10.5 mg endothall ae/kg/day (Plankenhorn, 1990 as cited in WSDOE, 2001).

In an oral dietary study conducted in VD-1 6-week old mice over 92 weeks, NOELs of 100 ppm for males and 300 ppm for females were determined; however, the results from this study are questionable based on a possible miscalculation of dose calculations in feed (Atochem, 1990).

Developmental/Reproductive:

A three-generation study in rats was conducted in which groups of male and female rats were fed diets containing 0, 4, 12 or 100 mg endothall ion/kg/day until they were 100 days old and then mated. Three successive generations of offspring were kept on the same test diet and then mated to produce the next test generation of offspring. No adverse effect was noted in the 4 mg/kg/day pups. Pups in the 12 mg/kg/day group had decreased body weights. Pups in the 100 mg/kg/day group did not survive more than one week (Scientific Associates, 1965 as cited in USEPA, 1988).

A short-term teratology study in rats indicated that no developmental effects were produced in offspring at endothall concentrations that were lethal to dams. Groups of 25 or 26 female rats were mated and then orally dosed with 0, 8, 16 or 24 mg endothall ion/kg/day of aqueous endothall technical on days 6 to 19 of gestation. Two dams died at the 16 mg/kg/day dose and ten dams died at the 24 mg/kg/day dose. The study suggests that the dams are more susceptible to endothall than are embryos or fetuses (Science Applications, Inc., 1982 as cited in USEPA, 1988).

In another study conducted in mice, endothall was administered via gavage to 4 groups of 25 pregnant mice on gestation days 6 through 16 at doses of 0 (control), 5, 20 and 40 mg/kg bw/day. Two dams died at the 20 mg/kg/day dose and two dams died at the 40 mg/kg/day dose. The incidence of vertebral and rib malformations in the offspring was increased although it was not statistically significant. The authors suggested that the results of this study are nevertheless significant since the usual incidence of vertebral and rib malformations is low in their laboratory;

however, they acknowledged that the influence of maternal toxicity in producing the reported malformations could not be ruled out (IRDC, 1981 as cited in USEPA, 1992a).

A developmental/reproductive study was conducted by the manufacturer in which rats were dosed with disodium endothall in drinking water during organogenesis (days 6-15) of pregnancy and sacrificed on day 20. A NOAEL of 12.5 mg/kg/day was determined for maternal effects and a NOAEL of 25 mg/kg/day was determined for fetal effects (Elf Atochem, 1993c). In another study, rats were given disodium endothall in the diet until completion of breeding, a 2-generation reproductive study was conducted and a NOAEL of 150 ppm for maternal reproductive effects was identified (Elf Atochem, 1993d).

Another, more recent two-generation study was conducted in which rats were fed endothall disodium salt at 1.2, 6 or 36 mg ae/kg/day endothall. No treatment-related effects were noted in terms of pregnancy rates, fertility, reproductive performance or offspring via bility and survival. The only significant adverse effect noted was decreased body weight in parents and offspring in the high dose group. The NOAEL for the study was 6 mg ae/kg/day endothall (Trutter, 1993 as cited in WSDOE, 2001).

Mutagenicity:

A number of short-term mutagenicity studies have been conducted with various forms of endothall as the test agent. The results of these studies are mixed. Endothall was not mutagenic in studies conducted with bacteria, fungus, mammalian cells or *Drosophila*. Assays conducted with in vivo somatic or male germinal-cells using the disodium salt did not induce any mutagenic effects. Endothall did not produce aneuploidy in plants and dipotassium endothall did not induce the frequency of sister chromatid exchange in human lymphocytes. the dipotassium salt formulation did induce mutagenic effects in BALB/3T3 both in the presence and absence of rat primary hepatocytes; however the validity of these studies is questionable (USEPA, 1992a).

Several mutagenicity assays, conducted by the manufacturer, including two Ames assays, one assay in Chinese hamster ovary cells and one in vivo test conducted in mouse bone marrow erythropoietic cells yielded all negative and one set (Ames) of equivocal results (Elf Atochem, 1993e, 1993f, 1994e). Endothall produced positive results in a chromosome aberration study in *Allium cepa* (Mutation Research, 1982 as referenced in GENETOX, 1995).

Carcinogenicity:

Limited studies have been conducted which address the potential carcinogenicity of endothall. 10 male and 10 female rats were exposed to endothall in the diet at various concentrations up to 2,500 mg disodium endothall/kg food (about 100 mg endothall ion/kg/day) for 2 years. Two of the treated rats had lung tumors; however, based on the small sample size used for this investigation and the lack of information obtained on tumor type and dose group, the statistical validity of these findings was not evaluated (Brieger, 1953b as cited in USEPA, 1988). A 2-year oral cancer study in rats found no carcinogenic response at doses up to 1800 ppm (Atochem North America, 1990). The present database is inadequate to assess the animal or human carcinogenic potential of endothall. Based on a review of the available chronic feeding studies and results of mutagenicity tests, there are no definitive data indicating that endothall is carcinogenic. The primary histopathological findings have been attributed to the high irritation potential of endothall to the gasto-intestinal tract (WSDOE, 2001). The U.S. EPA Office of Pesticide Programs (OPP) has designated endothall as a Group E carcinogen under the old EPA cancer classification system. Under the new EPA cancer classification system using descriptors, a Group E carcinogen corresponds to the descriptor “not likely to be carcinogenic to humans”.

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) has developed several Drinking Water Health Advisories for endothall. Health Advisories are defined as concentrations of a substance in drinking water estimated to have negligible deleterious effects in humans, when ingested for a specified period of time. These values include a ten-day health advisory for a child of 0.8 ppm as well as a lifetime health advisory of 0.2 ppm for a child and 0.7 ppm for an adult (USEPA, 1988).

The EPA has also developed a Maximum Contaminant Level Goal (MCLG) of 0.1 mg/l for drinking water and has promulgated this value as a Maximum Contaminant Level (MCL) standard (USEPA, 1992b; USEPA, 1995a). Massachusetts has adopted this value as a drinking water standard, known as a Massachusetts Maximum Contaminant Level (MMCL).

In addition, the EPA Carcinogen Risk Assessment Verification Endeavor (RfD/RfC) Workgroup has developed an oral Reference Dose (RfD) of 0.02 mg/kg/day for endothall based upon the Keller (1965) two-year feeding study in dogs cited earlier. The EPA Office of Pesticide Programs (OPP) has calculated the same RfD value based on the same study (USEPA, 1995b). The RfD is an estimate, (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 1992b).

ECOLOGICAL TOXICITY

Aquatic Organisms :

Limited information indicates that, in contrast to endothall absorbed by mammals in which it is excreted largely as the bound form, endothall absorbed by plants and fish is completely metabolized (Simsiman *et al.*, 1976 as cited in HSDB, 1994). In acute and behavioral toxicity studies, goldfish did not avoid endothall at 0.17 ppm and 1.70 ppm, but avoided it at 17.0 ppm (Berry, 1984 as cited in HSDB, 1994). Rainbow trout avoided a dipotassium salt formulation at concentrations above 10 ppm (State of Wisconsin, 1990 as cited in WSDOE, 1992).

Most of the available information on the toxicity of endothall to aquatic organisms is based on acute exposure data obtained in laboratory studies. There are very few studies addressing longer-term exposures. One longer-term study evaluated the effects of a one-time application of dipotassium endothall over a three-year period on reproduction or survival of young-of-the-year bluegills. No difference in effects was noted but adult fish survival was found to be higher in the treatment pond. It was suggested that this finding might be due to slower growth in the treatment pond reflecting a greater biomass of fish present (State of Wisconsin, 1990 as cited in WSDOE, 1992).

Acute flow-through type bioassays have been conducted with a number of freshwater and marine fish and invertebrates. In general, the dipotassium salt formulation is the least toxic to aquatic organisms, the technical acid is somewhat more toxic and the monoamine formulation is much more toxic. Typical acute LC50 values obtained in 96-hr static bioassays using a 40.3% dipotassium salt of endothall formulation (i.e., as a liquid) range from >150 ppm for channel catfish to 230-450 ppm for rainbow trout to 343-450 ppm for bluegills to 313 ppm for scuds. Typical LC50 values obtained in similar assays using a 53% formulation of endothall monoamine salt (i.e., as a liquid) are much lower ranging from 0.05 ppm for grass shrimp and stoneflies to 0.49 ppm for channel catfish to 0.94 for bluegills (Phipps, G.L., 1984 as cited in HSDB, 1994). In a series of studies conducted with freshwater (rainbow trout, bluegill sunfish, water flea) and marine (oyster, sheepshead minnow and mysid shrimp) species: For tests conducted with a formulation of 40.3% active ae, the LC50 values ranged from 240-740 ppm. The least sensitive species was the bluegill and the most sensitive species were the mysid shrimp and the water flea. For the

technical acid, the LC50 values ranged from 39-110 ppm. The least sensitive species was the sheepshead minnow while the most sensitive species was the mysid shrimp. For tests conducted using a monoamine salt formulation, the LC50 values ranged from 0.19-2.0 ppm with the least sensitive species being the bluegill and sheepshead minnow and the most sensitive species being the mysid shrimp and the water flea (Elf Atochem, 1993a).

Thus, the toxicity of endothall to aquatic organisms depends on the formulation used. The dipotassium salt of endothall is generally not toxic to aquatic organisms at recommended application rates of 0.5-5 ppm, whereas the monoamine salt formulation is lethal to many organisms at the same recommended application rate.

Plants:

Since endothall is effective in treating a large range of plants, it may have a widespread effect on non-target plants, especially when applied as a whole-pond treatment. In addition to direct toxic effects of the herbicide, treatment of a pond with endothall may also cause indirect impacts including dissolved oxygen depletion and habitat loss. These impacts may cause general weakening and/or death of plants on a large scale (WSDOE, 1992).

Microorganisms:

No significant differences were seen in zooplankton population over a 5-month period in a pond treated with 5.0 ppm dipotassium endothall as compared to a control pond. No significant impacts were noted on aquatic bacteria from the dipotassium salt at 5ppm (WSDOE, 1992).

Table III.6-3. Properties of Endothall

CAS #:	145-73-3
Synonyms	Hexahydro-3,6-endo-oxy-phthalic acid; 3,6-endo-Epoxy-1,2- cyclohexanedicarboxylic acid
Molecular formula	$C_8H_{10}O_5$
Molecular weight	186.2
Physical properties	crystalline, white solid; odorless
Melting point	when heated rapidly at 144^o C, decomposes into the anhydride and water
Density	1.43 g/ml
Vapor pressure	negligible
Photolysis half-life	stable
Hydrolysis half-life	stable
Biodegradation half- life	8.35 days
K_{ow}	1.36 (potassium salt) 1.91 (acid)
K_{oc}	110-138 ml/g
BCF	<1-1.1
Solubility:	(g acid monohydrate/100 g solvent)
Acetone	7.0
Benzene	0.01
Dioxane	7.6
Ether	0.1
Isopropyl Alcohol	1.7
Methanol	28.0
Water	10.0

(Aquatic Plant Identification and Herbicide Use Guide, 1988; WSSA, 1983)

Endothall References

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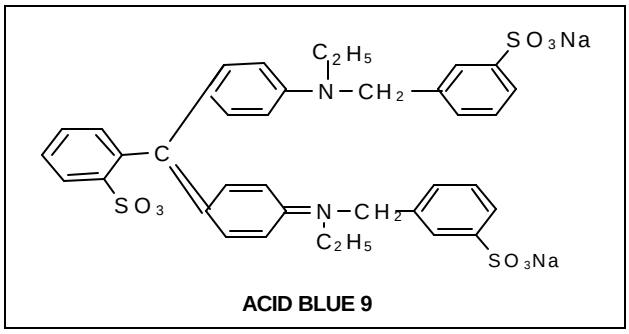
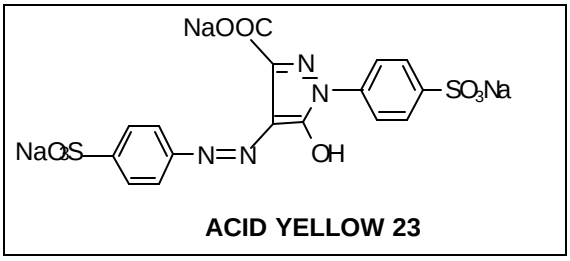
III.7 AQUATIC DYES

As of the time of publication of this document, only products containing the aquatic dye Aquashade are currently registered for use in Massachusetts.



ACID BLUE 9



ACID YELLOW 23

SUMMARY

Active ingredients in AQUASHADE

Aquashade is a water-soluble mixture of blue and yellow dyes, which is used as a nonselective herbicide to control young, bottom-growth of plants in contained lakes and ponds (Applied Biochemists, Inc., 1992a). The principle active ingredient in Aquashade is Acid Blue 9 (n-ethyl-n-[4-[[4-[ethyl[(3-sulfophenyl)methyl]amino]- phenyl](2-sulfophenyl)-methylene)]2,5-cyclohexadien-1-ylidene]-3-sulfobenzenemethanaminium hydroxide inner salt, disodium salt (also prepared as the diammonium salt) (The Merck Index, 1983). The other active ingredient is acid yellow 23 (4,5-dihydro-5-oxo-1-(4-sulfophenyl)-4-[(4-sulfophenyl)azo]-1H-pyrazole-3-carboxylic acid trisodium salt) (The Merck Index, 1983). Aquashade filters out the red-orange and blue-violet wavelengths of light from the sunlight spectrum, thus interfering with the photosynthetic process in plants. The half-life for Aquashade in water is about 4 weeks. Over time, Aquashade is removed from a water body through a combination of dilution, photodegradation, and some biodegradation (Applied Biochemists, Inc., 1992a).

Although Aquashade was registered by the Environmental Protection Agency (EPA), it did not receive an EPA Registration Standard prior to the effective date of the 1988 Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) (HSDB, 1995). FIFRA 1988 requires that by 1997, each registered pesticide product containing an active ingredient initially registered before November 1, 1984 be reregistered (USEPA, 1994). Pesticides for which EPA did not issue Registration Standards prior to FIFRA 1988 were divided into three lists based upon their potential for exposure and other factors, with list B being of highest concern and list D of least. Aquashade was placed on list D and its current status is "Awaiting Data/Data in Review" (USEPA, 1994).

Currently, very little data exist on the toxicological and/or environmental effects of Aquashade. Once the EPA registration process has been completed, the impacts of this pesticide on human health and the environment can be evaluated.

REGISTERED PRODUCTS IN MASSACHUSETTS

The current list of aquatic herbicides containing Aquashade that are registered in Massachusetts can be accessed at http://www.mass.gov/agr/pesticides/aquatic-vegetation-management.html on the Massachusetts Department of Agricultural Resources (DAR) Aquatic Pesticide Website. The DAR updates this list regularly with changes. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding these products.

AQUASHADE USES AND APPLICATION

Aquashade is intended to be used for the control of young plant growth on the bottom of contained bodies of water. Aquashade has also been shown to reduce the growth rate of algae (Applied Biochemists, Inc., 1992a). It is most effective in ponds with depths of two feet or greater.

The best time to apply Aquashade is at the beginning of the growing season, when young plants are just beginning to develop. Floating-leaf plants, which have already emerged, are not affected by Aquashade (Applied Biochemists Inc., 1992).

Application of Aquashade can be made in a number of ways. Manually, the herbicide can be poured along the water's edge, allowing it to disperse quickly with natural water movements to achieve even coloration. For quicker dispersion, the water can be applied over the surface of the water using a sprayer, can be injected into water pumps or can be applied near aerators or waterfalls (Applied Biochemists, Inc., 1992a).

Because Aquashade is a nonselective herbicide, it can affect a large number of young, bottom-growing plant species. In one study, nine species of aquatic plants were subjected to dye concentrations ranging from 0.5 to 15 ppm in 62 liter drums. Five of these plants, including elodea (*Elodea canadensis*), leafy pondweed, sago pondweed (*Potamogeton pectinatus*), curlyleaf pondweed (*Potamogeton crispus*) and brittle naiad (*Najas minor*), showed a significant reduction in weight over a four-week period at 1-5 ppm. One plant, whorled milfoil (*Myriophyllum verticillatum*), exhibited an increase in growth rate at concentrations of 1 ppm and above (White *et al.*, 1975).

Aquashade may be used in conjunction with other herbicides to achieve an effective year-round control and maintenance program for a variety of plant species at their various stages of development (Applied Biochemists Inc., 1992).

For specific information on recommended application rates, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. The product labels should also be consulted for any recommended use restrictions.

MECHANISM OF ACTION

Aquashade controls plant growth by competing with photosystem II pigments in plants. The blue and yellow dyes in Aquashade screen out the red-orange and blue-violet wavelengths of the sunlight spectrum which are required by plants and algae for photosynthesis (Applied Biochemists Inc, 1992; Spencer, 1984a). Aquashade is most effective in ponds which are greater than two feet deep since at this depth most of the critical wavelengths of sunlight can be absorbed by the dyes before they reach the bottom growth (Aquashade, Inc. (brochure)) .

ENVIRONMENTAL FATE/TRANSPORT

Information on the fate and transport of Aquashade is limited. The normal half-life of this product is reported to be four weeks and coloration is gradually lost by dilution, photodegradation and some biodegradation over time (Applied Biochemists Inc., 1992). However, as conditions among various bodies of water differ, there is a range of variation in this estimate. In one study in which Aquashade was applied to earthen catfish ponds with high seepage rates, it was estimated that about one-half of the dye applied initially was lost by seepage every 2 months (Boyd and Noor, 1982). In another study, the half-life of Aquashade in a test pond with no outlet was found to be about two months whereas in other test ponds, factors such as water outflow and evaporation affected the dye concentrations (White *et al.*, 1975).

In a pond to which Aquashade was applied to achieve a concentration of 3.0 ppm, the actual concentration varied from 1.5 to 3.5 ppm. The variation was attributed to dilution by heavy rains rather than to photo-oxidation. It was estimated that the dye holds its concentration for approximately three months during exposure to light (Osborne, 1979).

No information was found regarding the potential for Aquashade to bioconcentrate in organisms.

PHARMACOKINETICS

No information was found on the pharmacokinetics of Aquashade. Limited pharmacokinetic information was found for its principle active ingredient, acid blue 23. In male and female rats administered a single oral dose of either 30 or 3 mg/kg acid blue 9 dye, substantially all of the dose was excreted unchanged in feces within 72 hours. Similar findings were noted with mice and guinea pigs. When male rats were pretreated with unlabeled dye in the diet for 21 days and subsequently dosed with ^{14}C-labeled dye, no difference was noted in the route of excretion or the time taken to eliminate all of the label. In all three species noted, the lack of absorption and metabolism of the labeled dye in the gastrointestinal tract of the animals was confirmed by examining isolated loops of small intestine (HSDB, 1995).

Studies in rats, dogs and guinea pigs indicate that only a very small percentage of ingested dye is absorbed and that it is excreted mainly in the feces. Following its intravenous injection in rats, over 90% of the dye was excreted in the bile within 4 hours (IARC, 1978).

No information was located on the pharmacokinetics of acid yellow 23.

HEALTH EFFECTS

Very little information is currently available on the toxicity of Aquashade to plants and animals. Most of the limited available toxicological information was obtained from the manufacturer. Once FIFRA reregistration of Aquashade has been completed, additional data will become available. Limited information was also found on the toxicity of the principle active ingredient, acid blue 9. Very little information was found on the toxicity of acid yellow 23.

Avian:

Only two studies were located on the acute toxicity of Aquashade to birds. These include two LC50 studies conducted by the manufacturer, one in mallard ducks and the other in bobwhite quail. For both species, the acute dietary LC50 (i.e., concentration found to be lethal to half of the test population) was greater than 5620 ppm of Aquashade, indicating that the acute toxicity of this product to birds through ingestion is very low (Applied Biochemists, Inc. 1995a).

Mammalian:

Contact exposure to Aquashade may cause slight irritation and redness of the eyes and slight irritation of the skin. Inhalation of Aquashade may produce slight nausea. Ingestion may cause gastric disturbances (Applied Biochemists, Inc., 1992b).

No toxicological studies on the acute, subchronic or chronic health effects of Aquashade were located. However, limited information on the health effects of its principle active ingredient was obtained.

A subcutaneous LD50 of 4.6 g/mg for acid blue 9 was identified in rats. An oral LD50 for an unspecified salt of acid blue 9 was greater than 2.0 g/kg body weight. (Lu and Lavalle, 1974 as cited in IARC, 1978).

In 1978, the International Agency for Research on Cancer (IARC) concluded that subcutaneous injections of the disodium salt of acid blue 9 were carcinogenic to rats (IARC, 1978). They later determined that these data provided insufficient evidence of the carcinogenicity of Aquashade to animals (Applied Biochemists, Inc., 1992b).

Charles River albino rats and CD-1 mice were fed acid blue 9 in the diet over their lifetimes. The rat study had an in utero phase in which F0 generation rats were administered the dye in their diets at concentrations of either 0.0%, 0.1%, 1.0% or 2.0%. Randomly selected offspring of the F1 generation were exposed to the same concentrations for a lifetime. The maximum exposure times were 116 and 111 weeks for males and females respectively. No- observed-adverse-effect-levels (NOAELs) were identified as 2.0% for males (1,072 mg/kg/day) and 1.0% (631 mg/kg/day) for females based on a decrease in terminal body weight and decreased survival rate in the high-dose females as compared to controls. The CD-1 mice received dietary concentrations of either 0.0%, 0.5%, 1.5% or 5.0% for their lifetimes. The maximum exposure time for both males and female was 104 weeks. The NOAEL for this study was identified as 5.0% (7354 mg/kg/day and 8966 mg/kg/day for male and female mice respectively) (HSDB, 1995).

The FDA established a maximum acceptable daily intake for acid blue 9 of about 12 mg/kg/day. The U.S. FDA concluded that acid blue 9 is nonirritating when applied daily to intact or abraded skin. In addition, lifetime application of acid blue 9 to the skin of mice did not produce carcinogenicity (Fed. Regist. (47), 1982).

Both of the dyes contained in Aquashade have been reviewed by the Food and Drug Administration (FDA). Acid blue 9 has been approved by the FDA for use in food, drugs and cosmetics, excluding use in the eye area (The Merck Index, 1983; Fed. Regist.(47), 1982). Acid yellow 23 has been approved by the FDA for use in food and ingested drugs, and provisionally listed for externally applied drugs and cosmetics (The Merck Index, 1983; Fed. Regist.(44), 1979). The FDA requires that food containing acid yellow 23 be labeled accordingly, due to data indicating that an allergic reaction to this dye may result, especially in individuals who are allergic to aspirin (Fed. Regist. (44) 1979).

No health advisories or toxicity criteria have been developed for Aquashade.

ECOLOGICAL TOXICITY

Aquatic Organisms :

Limited studies on the toxicity of Aquashade to aquatic organisms have been conducted. Acute 96-hour toxicity studies were conducted by the manufacturer in bluegill sunfish and rainbow trout. LC50 values calculated from these studies were greater than 96 mg/l, indicating that the toxicity of Aquashade to these species is low (Applied Biochemists, Inc, 1995a). A 48-hour LC50 value greater than 97 mg/l was identified for the invertebrate, *Daphnia magna*, indicating that the toxicity of Aquashade to this organism is also very low (Applied Biochemists, Inc., 1995a). In another study, no differences were found in the oxygen consumption rates of crayfish (*Orconectes propinquus*) exposed to water containing 5, 10 or 15 ppm of Aquashade relative to that of control crayfish (Spencer, 1984b). In two channel catfish ponds treated with 4 mg/l of Aquashade, the average net fish production rate was calculated as 3,641 kg/hectare relative to an average of 3,010 kg/hectare in three control ponds. Because no improvement in water quality was noted from the treatment, the greater fish production in the treated ponds was attributed to random variation rather than to an effect by Aquashade. A third treated pond could not be included in

the calculation of net fish production due to a fish kill which was unrelated to the dye treatment (Boyd and Noor, 1982).

Plants:

Literature reports on the efficacy of Aquashade in controlling aquatic plants indicate a spectrum of effectiveness ranging from unsuccessful to very successful. In the study of channel catfish ponds discussed above, there was no difference in bottom coverage of underwater weeds between dye-treated and control ponds (Boyd and Noor, 1982). Application of Aquashade to small ponds did not prevent establishment of weeds at depths up to 2.5 m; however, the heaviest infestations occurred at depths of less than 1 m (White *et al.*, 1975). Very successful results with Aquashade have been obtained when Aquashade is applied following application of another herbicide or another treatment. A test pond in central Florida was initially treated with Hydrothol 191 herbicide in the fall at double strength (10 gal/acre) in order to remove the parent hydrilla (*Hydrilla verticillata Royle*) population. The pond was then immediately (i.e., two days later) treated with Aquashade at a concentration of about 2-3 ppm in order to reduce incoming red light to 1-3% of full sunlight intensity at a depth of 1 m. The percent frequency of occurrence of *Hydrilla* declined exponentially from November though May and no *Hydrilla* was found in the water at any depth from May through September. In comparison, pronounced *Hydrilla* growth was noted when only herbicide (with no subsequent Aquashade application) was applied to the pond during the previous year. The authors concluded that Aquashade can be used effectively to control *Hydrilla* regrowth from vegetative propagules when applied at a rate greater than 2 ppm before the spring, following an herbicide application (Osborne, 1979).

The Adirondack Lake Association in Indian Lake, New York successfully used Aquashade to control bass weed (*Potamogeton amplifolius*) in Adirondack Lake, after a series of unsuccessful attempts with other methods. The Aquashade was applied to the lake (which has an average depth of 7 feet) in May at a concentration of about 0.7-1.0 mg/l after a winter in which the lake water level was lowered 7 feet in an attempt to kill weed roots. Another touch-up Aquashade application was applied in August after the Aquashade water concentration was found to have fallen to 0.3 mg/l. The Adirondack Lake Association found that 3-5" weed sprouts that had already sprouted before the first Aquashade application grew to a height of 18-24", but they attributed this weed growth to the period of low dye concentration. The bass weed was reported to be well out of sight and out of the way in at least 90% of the lake, in contrast to past years when the weed heads broke the surface of the water, interfering with lake recreation (Purdue).

There is also evidence that Aquashade is effective against microalgae. One study found a 50% reduction in the photosynthetic rate of algal cultures exposed to 1-3 ppm Aquashade. In separate experiments, the same study found a reduced growth rate of microalgae at Aquashade application rates greater than 5 ppm (Spencer, 1984a). The manufacturer recommends an Aquashade application rate of 1 ppm for aquatic weed control (Spencer, 1984a). The fact that Aquashade may be effective against microalgae should be considered when making a decision as to its appropriateness for use in controlling macrophytes. In a natural water system that depends upon microalgae as a source of primary production, inhibition of microalgae growth may potentially disrupt the aquatic food chain, leading to an eventual reduction in food supply for the entire trophic structure of that water body.

The phytotoxicity of Aquashade to nontarget species (such as cattails (*Typha*) and spatterdock (*Nuphar*)) was low when poured directly onto plants due to the plants' waxy cuticles which repelled the dye droplets (White *et al.*, 1975). When the concentrated dye was sprayed onto foliage either with or without surfactant, it caused severe contact burns of the foliage. No translocation of the dye throughout the plant was noted (White *et al.* 1975).

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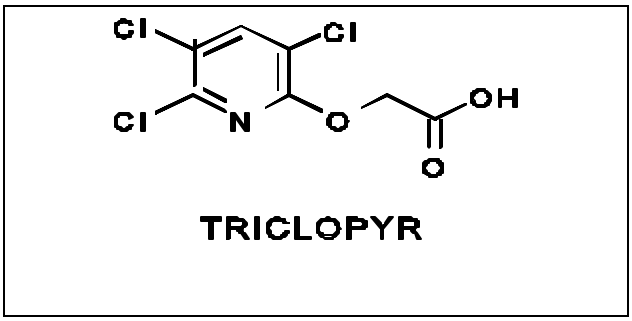
III.8 TRICLOPYR



SUMMARY

Triclopyr [(3,5,6-trichloro-2-pyridynyl) oxy] acetic acid is a synthetic herbicide that is used to control a wide variety of woody plants as a foliar spray or as a basal spray when applied to cut surfaces. There are three formulations of triclopyr commonly used to control nuisance vegetation: triclopyr acid (CASRN 55335063 (CASRN 057213691), the triethylamine salt (TEA) (CASRN 057213691); and the butoxyethyl ester of triclopyr (TBEE) (CASRN 008008206). Several authors have evaluated the use of triclopyr for control of Eurasian watermilfoil (Getsinger and Westerdahl, 1984; Green *et al.*, 1989; Netherland and Getsinger, 1992), but currently there are no formulations of triclopyr that are registered for aquatic uses in Massachusetts.

The U.S. EPA approved a Reregistration Eligibility Decision (RED) on September 30, 1997 for triclopyr which includes the triclopyr acid, as well as the TEA and TBEE formulations.

Photodegradation is the major pathway for the transformation of triclopyr in aquatic systems. The triethylamine salt and the butoxyethyl ester formulations are rapidly converted to the acid form in water (McCall and Gavit, 1986 and Solomon *et al.*, 1988; as cited in DFA/DEP Adhoc Committee, 1990).

REGISTERED PRODUCTS IN MASSACHUSETTS

As of the printing of this GEIR, there are currently no formulations of triclopyr registered for aquatic use in Massachusetts. However, this chemical is currently undergoing review by the Massachusetts Division of Agricultural Resources (DAR). The review of the environmental fate information has been completed and the DAR is still waiting to receive the results of many of the recent toxicity studies (Kennedy, pers. comm., 2004). The current list of aquatic herbicides that are registered in Massachusetts can be accessed at http://www.state.ma.us/dfa/pesticides/water/aquatic/ aquatic/profile.htm on the DAR Aquatic Pesticide Website. The DAR updates this list regularly with changes. The status of triclopyr can be followed by consulting this website. In addition, the DAR can be contacted directly at (617) 626-1700 for more specific questions regarding updates for this product.

TRICLOPYR USES AND APPLICATION

Triclopyr and its formulations, TBEE and the TEA salt, are often used to control unwanted woody plants and annual and perennial broadleaf weeds in rangeland, permanent pastures, forests and on non-crop areas including rights of way such as electric power lines, communication lines, pipelines, roadsides and railroads. There are also formulations of the TEA salt being marketed for aquatic weed control. The salt formulation is used to control nuisance aquatic vegetation.

For specific information on recommended application rates for a particular product, the product label should be consulted. The USEPA Office of Pesticide Programs (OPP) has a link to a database of product pesticide labels at http://www.epa.gov/pesticides/pestlabels/. Manufacturers' product labels should also be consulted for recommended product application rates and use restrictions.

MECHANISM OF ACTION

Triclopyr is an auxin type herbicide that is absorbed by the roots and leaves, is translocated through the plant and accumulates in the meristematic tissues of the root and shoot. The auxin-type response in plants associated with triclopyr interferes with normal growth processes, therefore, maximal effect occurs when applications are made soon after leaves are fully developed and when there is sufficient moisture for plant growth.

ENVIRONMENTAL FATE AND TRANSPORT

The triclopyr acid is short-lived in the aquatic environment with reported half-lives from 2.1 hours at the water's surface in the summer at 40° N latitude to 14 hr at 1 meter water depth in winter (McCall and Gavitt, 1986). In a study by Dow Elanco, half-lives for triclopyr were determined at 20, 30, 40 and 50° N latitude in summer and in winter (Batzer, 1994). In summer, from 20 to 50° N latitude, triclopyr had calculated half-lives of less than 1.2 days in both pH 7 buffered and natural waters.

Triclopyr acid is stable to hydrolysis in buffered solutions for periods of up to 9 months at pH 5, 7 and 8 at 15, 25 and 35°C (Hamaker, 1977). Therefore, hydrolysis is not expected to be a major route of degradation. Photolysis is the major degradation pathway for triclopyr acid in water. The rate of triclopyr photodegradation in water is rapid in both natural sunlight and in the laboratory. A photolysis half-life of 10 hours is reported by Hamaker (1977). In winter, the calculated photolysis half-life of triclopyr varied in latitude from 0.54 to 10 days in pH 7 buffered solutions and from 1.5 to 29 days in natural water. A half-life of 142 days has been reported for the metabolism of triclopyr conducted under dark conditions in a 30 day aquatic metabolism study (Woodburn and Cranor, 1987).

The principle decay product of the acid is the 3,5,6-trichloro-2-pyrindol (TCP), a transient metabolite in water with half-lives ranging from minutes to one day (Dilling *et al.*, 1984). Woodburn demonstrated that the triethylamine salt of triclopyr experimentally applied to a lake in Florida also provides useful comparative data on the persistence of triclopyr degradation products. TCP rapidly degrades into non-halogenated, low molecular weight organic acids (Woodburn, 1993), with phototransformation playing a larger role than hydrolysis in this process.

The fate of the butoxyethyl ester of triclopyr (TBEE) in water is summarized in Table III.8-1. This table shows the major degradation pathways for the ester in water but does not include the processes such as sediment and particulate adsorption. The fate of the ester in water has also been simulated with a modeling technique by McCall *et al.* (1988). The degradation pathway is believed to be TBEE to triclopyr acid to 3,5,6-trichloro-2-pyrindol (TCP) to non-halogenated organic acids.

TBEE degrades quite rapidly in water to triclopyr acid. Laboratory studies indicate that photolysis is the principal degradation pathway, with hydrolysis also contributing. Several studies indicate that the half-life of the ester in water can range from 1.5 to 6.6 days as a result of photolysis (McCall and Gavit, 1986; Solomon *et al.*, 1988; Havens and Shepler, 1993). Hydrolysis half-lives are dependent upon pH and temperature and range from 0.06 days to 208 days in natural waters. They decrease with increasing temperature and increasing pH. Acidic conditions increase the persistence of the ester substantially. The 208 day half-life was observed in natural waters at pH 5 at 15°C. Waters with this pH level occur in Massachusetts. One laboratory study produced contradictory results where the ester was stable to hydrolysis and little photodegradation of the ester occurred over 9 months (Hameker, 1977). This study

was performed with buffered, sterile water. Modeling results for the dissipation of the ester indicate that the decay should be fairly rapid with a half-life of 12 to 18 hours (McCall *et al.*, 1988).

A half-life of 3.8 to 4.3 days at 16 to 17°C was calculated for the degradation of the ester to TCP in an Ontario Lake (Solomon *et al.*, 1988). Woodburn (1993) added Triclopyr salt to a Florida lake and determined a half-life of 0.5 to 3.6 days at 30°C for the breakdown of the ester (or salt) to organic acids. With the exceptions of the Hameker (1977) study and slow breakdown at pH 5, most studies indicate that TBEE in water is degraded relatively rapidly.

Tables III.8-3a, III.8-3b and III.8-3c at the end of this triclopyr summary list the physical and chemical properties for the parent compound, triclopyr, the triethylamine salt and the butoxyethyl ester. The data indicate that triclopyr is relatively mobile and is not expected to bioaccumulate in aquatic organisms.

Table III.8-1. Aquatic Fate of Triclopyr

Pathway	Finding	Authors
Hydrolysis	insignificant route of degradation	Cleveland and Holbrook, 1991
Photodegradation	most significant route of degradation	Woodburn *et al*., 1990
Aerobic Aquatic Degradation	slow metabolism, half-life est. @ 4.7 months under dark conditions	Woodburn and Cranor, 1987
Anaerobic Aquatic Degradation	slow degradation, est. 3.5 years	Laskowski and Bidlack, 1984

Tables III.8-3a, III.8-3b and III.8-3c at the end of this triclopyr summary list the physical and chemical properties for the parent compound, triclopyr, the triethylamine salt and the butoxyethyl ester. The data indicate that triclopyr is relatively mobile and is not expected to bioaccumulate in aquatic organisms.

PHARMACOKINETICS

Both the triclopyr acid and the TBEE salt are readily absorbed from the gastrointestinal tract (Dryzga *et al.*, 1994). This same study indicated that both compounds are distributed similarly, have a similar plasma clearance (0.92 and 0.95 ml/min-kg^{-1}) and are both eliminated in 4.5 hours. Triclopyr is excreted primarily in the urine (Dow Elanco, 1992).

Triclopyr acid and triclopyr-BEE have been shown to be bioequivalent following a single low oral dose and a single high oral dose in rats (Dryzga *et al.*, 1994). There were no differences in plasma levels or pharmacokinetics between the triclopyr free acid and the TBEE under the conditions of the testing regime. Results of the high dose administration demonstrate that the tissue distribution was similar for both forms of triclopyr. The USEPA considers the triclopyr acid, the triclopyr butoxyethyl ester and the triclopyr triethylamine salt as bioequivalent (McMaster, 1995).

HEALTH EFFECTS

Avian:

The toxic effects of triclopyr on birds have been investigated in studies conducted by Dow Elanco. For mallard ducks, acute oral LD50 values are reported at 1648 mg/kg for unformulated triclopyr, 3176 mg/kg for Garlon 3A, and 4640 mg/kg for Garlon 4 (Pesticide Background Statement, USEPA, 1984). Eight day subchronic oral LC50 values are reported in Table III.8-2 below for the various triclopyr formulations:

Table III.8-2. Eight-day LC50 Values

Triclopyr	mallard duck bobwhite quail Japanese quail	LC50 = 5,000 ppm[1] LC50 = 2,935 ppm[1] LC50 = 3,278 ppm[1]
a formulation of the triethylamine salt (TEA)	mallard duck bobwhite quail	LC50 = 10,000 ppm[2] LC50 = 11,622 ppm[2]
a formulation of the butoxyethyl ester (TBEE)	mallard duck bobwhite quail bobwhite quail bobwhite quail	LC50 = 10,000 ppm[2] LC50 = 9,026 ppm[2] LD50 = 735 mg/kg[3] LC50 > 5401 ppm[4]

(1) WSSA, 1983
(2) Mc Call *et al.*,1988
(3) Campbell and Lynn, 1991
(4) Lynn *et al.*, 1991

The data summarized above indicate low acute and subchronic toxicity to bird species tested. No field studies on the toxic effects of triclopyr or its formulations in birds were available.

Mammalian

Acute:

The oral LD50 for triclopyr in rats is 729 mg/kg in males and 630 mg/kg in females (Pesticide Background Statements, 1984; Dow tech. data sheet). The rat LD50 for combined sexes has been reported as 713 mg/kg (WSSA, 1983; GEIR, 1985). Rabbits and guinea pigs are more sensitive via oral administration with LD50 of 550 and 310 mg/kg respectively. Both the TEA and TBEE formulations have oral LD50s of greater than 2,000 mg/kg.

The dermal LD50s are greater than 2,000 mg/kg in rabbits and greater than 3980 mg/kg in rabbits for the TBEE and TEA formulations respectively (Dow tech. data sheet; Dow MSDS for Garlon 3A; Dow MSDS for Garlon 4).

Triclopyr affects the eyes of rabbits and these effects are dependent on the chemical derivative involved: the TBEE formulation is essentially nonirritating (GEIR, 1985; Pesticide Background Statements, 1984; Dow MSDS for Garlon 4). The TEA formulation is not only an irritant but can cause serious injury (GEIR, 1985; Dow MSDS for Garlon 3A). These eye injuries include conjunctival irritation, moderate internal redness and moderate to severe corneal damage which may be permanent.

An inhalation study showed that 100% of the test rats survived a 1 hour exposure to 3 to 20 dilutions of the TEA formulation in air. Transitory nasal irritation to rats was noted after a 4 hour exposure to an aerosol formulation of the TBEE. (GEIR, 1985)

Subchronic/Chronic:

In subchronic studies, the 90 day dietary No Observed Effect Levels (NOELs) were 30 mg/kg/d and 20 mg/kg/d for rats and mice respectively. Dogs were more sensitive to dietary administration of triclopyr, demonstrating a decreased urinary excretion at 2.5 mg/kg/d (GEIR, 1985; Dow Tox profile for Garlon). In a one year study, dogs received doses of 0, 0.5, 2.5 or 5 mg/kg/d. Minimal kidney effects were observed at doses of 2.5 and 5.0 mg/kg/d. These were considered non-adverse effects by Dow making the No Observed Adverse Effect Level (NOAEL) 5.0 mg/kg/d and the NOEL 0.5 mg/kg/d (Quast., 1988).

Two studies in monkeys were conducted to investigate kidney effects in primates. In one of the studies the monkeys received 0, 10, 20 or 30 mg/kg/d in their diets for 28 days; no effects were reported (DOW, nd). In a second study, 4 monkeys received triclopyr at 5 mg/kg/d for 28 days. The dose was then increased to 20 mg/kg/d for 102 days (DFA/DEP Adhoc Comm., 1990). The effects reported from this study were stool softening and diarrhea.

Long-term bioasssays have been done with triclopyr in rats (Eisenbrandt *et al.*, 1987) and mice (Tsuda., 1987). Fischer 344 rats received 5, 20, 50 or 250 mg/kg/d in a preliminary 13 week study. There was a decrease in body weight gain at 50 and 250 mg/kg/d and kidney effects were seen in both sexes at doses of 20 mg/kg or greater. In the full two year study, the doses were 0, 3, 12 and 36 mg/kg/d. The dose related effects included increased body weight at 12 and 36 mg/kg/d in the males and increased pigmentation of the proximal tubules at 3, 12 and 36 mg/kg/d in females. Neither the weight increase in males or the hyperpigmentation in females was accompanied by morphological, histological or functional changes. The NOAEL for males and females was reported to be 3 mg/kg/d (Eisenbrandt *et al.*, 1987).

In the mouse bioassays, ICR-mice received triclopyr in their diets for 22 months. The doses were 0, 50, 250 and 1250 ppm (0, 5, 55, 28.6 and 143 mg/kg/d in males and 0, 5.09, 26.5 and 135 mg/kg/d in females). The range finding study included doses of 0, 200, 400, 800, 1600 or 3200 ppm. At the high dose there were decreases in body weight, anemia, changes in urine, increased cholesterol levels and multiple changes in liver function. Some of the liver changes were also noted at the 1600 and 800 ppm levels. There were decreases in body weights, changes in the kidney and urine and liver effects at the 1250 ppm dose. At 250 ppm there were mild kidney effects and the NOEL was reported as 10 ppm (5.55 mg/kg/d in males and 5.09 mg/kg/d for females) (Tsuda., 1987.)

Developmental/Reproductive:

The teratology of triclopyr was investigated using the rabbit as a model of human exposure. Doses in the range finding study were 0, 25, 50, 100 and 200 mg/kg. There was 50% and 71% mortality in the 100 and 200 mg/kg group respectively. The doses used in the full study were 0, 10, 25 and 75 mg/kg/d for days 6 to 18 of gestation. There were 16 rabbits per dose group. One dam in the 25 mg/kg/d dose group aborted and one dam in the 75 mg/kg/d dose group died. In the 25 mg/kg/d dose group, one fetus had hyperplasia of the aortic arch with pulmonary arterial semilunar valve stenosis. Another fetus had a missing gallbladder. There was a statistically significant but non-dose related increase in resorptions at 10 mg/kg/d. This increase is within historical control variability. The development of the NOEL was reported at 75 mg/kg/d with a slight increase in maternal mortality (WSSA, 1983).

Mutagenicity:

Triclopyr has been tested for mutagenicity in a variety of test systems and found to be weakly positive in one, the dominant lethal study in rats. Triclopyr was non-mutagenic in bacterial system assays, cytogenic assays and mouse dominant lethal studies (Pesticide Background Statement, USEPA, 1984).

Carcinogenicity:

There have been two chronic bioassays done for triclopyr. Rats received 0, 3, 12 or 36 mg/kg/d and mice received 0, 50, 250 or 1250 ppm (5.55, 28.6, 143 mg/kg/d for males and 5.09, 26.5, and 135 mg/kg/d for females). The only positive result was an increase in the combined incidence of mammary adenomas and adenocarcinomas in the female rats at the high dose. There was no evidence of multiple tumors and the effects were not dose related (Tsuda., 1987; Eisenbrandt *et al.*, 1987). The most recent information available at the time of publication of this GEIR indicates that the U.S.EPA Office of Pesticide Programs (OPP) has designated triclopyr as a Group D carcinogen. Under the new U.S.EPA cancer classification system using descriptors, a Group D carcinogen corresponds to the descriptor, "Data are inadequate for an assessment of human carcinogenic potential".

Available Toxicity Criteria:

The Environmental Protection Agency (EPA) Office of Pesticide Programs (OPP) has developed an oral Reference Dose (RfD) of 0.005 mg/kg/d for triclopyr based on a one-year feeding study in beagle dogs (Federal Register, 1995; USEPA, 1995).

ECOLOGICAL TOXICITY

Invertebrates:

Little data are available on the toxicity of triclopyr acid to invertebrates and microorganisms. Data are available for the TEA and triclopyr BEE formulations. Data for TEA indicate low acute lethal toxicity to organisms tested, with a 96h LC50 of between 326 and 895 ppm in grass shrimp (Ward *et al.*, 1992; DFA/DEP Adhoc Comm., 1990), 96h LC50 greater than 1,000 ppm in crabs and 48h LC50 ranging between 58 and 87 ppm in oysters (Boeri, 1993). The 48h LC50 in *Daphnia* is reported as 1,170 ppm (Pesticide Background Statement, 1984). After 72h of incubation with 500 ppm of triclopyr, no apparent effects on growth were observed in six soil microorganisms when compared with controls.

In crayfish (*Procambarus clarki*), a 96h LC50 of greater than 326 mg/l was determined for the TEA (Barron *et al.*,1989). Exposure of grass shrimp (*Palaemonetes pugio*) resulted in a 96 hour LC50 of 326 mg/l (Ward *et al.*, 1992). A NOEL of 132 mg/l was derived from the same study.

The BEE of triclopyr was tested under flow-through conditions for acute toxicity to grass shrimp (*Palaemonetes pugio*) and a 96-hour LC50 of 2.4 mg/l was determined (Ward and Boeri, 1991a). In oysters (*Crassostrea virginica*), a 96-h EC50 of 0.66 mg/l was reported for the triclopyr-BEE (Ward and Boeri, 1991b). Tidewater silversides that were exposed to the BEE of triclopyr in a 96-hour assay showed an LC50 value of 0.45 mg/l (Ward and Boeri, 1991c).

Vertebrates:

Triclopyr Acid:

The available information on triclopyr toxicity to fish indicate a wide response of fish to two formulations of triclopyr and to unformulated triclopyr. In fish, 96-hour LC50 values of 117 ppm and 148 ppm have been reported in rainbow trout and bluegill sunfish, respectively (WSSA, 1983).

Triethylamine salt:

The TEA salt is "slightly toxic" to fish with 96h LC50 values of 552 and 891 ppm for rainbow trout and bluegill sunfish respectively. The corresponding values for the unformulated triclopyr are 117 ppm for rainbow trout and 148 for bluegill sunfish. Both species were less sensitive to the TEA salt than to the active ingredient (DFA/DEP Adhoc Committee, 1990).

Butoxyethyl ester:

The BEE of triclopyr is "highly toxic to fish" with the 96h LC50 values for rainbow trout and bluegill sunfish of 0.74 and 0.87 ppm respectively (McCall *et al.*, 1988). The corresponding value for juvenile Coho salmon is 1.3 ppm (Mayes *et al.*, 1986). In 1993, Woodburn *et al*. reported LC50 values in blue gill sunfish (*Lepomis macrochirus*) of 0.63 mg/l (24-h), 0.44 mg/l(48-h), 0.40 mg/l (72-h) and 0.36 mg/l (96-h). Based on the categorization scheme used by USEPA, triclopyr-BEE is "highly toxic" to bluegill sunfish. No fish toxicity data are available for the 3,5,6-trichloro-2-pyrindol (TCP), the intermediate breakdown product of the triclopyr acid to the non-halogenated organic acid end product.

The persistence data described earlier and the simulation results of McCall et al (1988), provide a description of the probable fate of triclopyr in toxicity tank tests. The majority of the fish mortalities during the toxicity tests with bluegill sunfish and rainbow trout exposed to the ester occurred during the first 24 hours of the test, a pattern consistent with the change of the toxic ester form to a less toxic breakdown product during this period (McCarty *et al.* n.d.).

Plants:

The triclopyr-BEE has been tested for toxicity to non-target vegetation. Milazzo *et al*. (1993), reported an EC50 (i.e., a concentration at which 50% of the test organisms manifest effects in cell growth) of 2.2-3.7 mg/l in duckweed (*Lemna gibba*), 0.193 mg/l in the diatom *Navicula pelliculosa* (Hughes and Alexander, 1993) and an EC50 of 0.193 mg/l in the blue-green alga *Anabaena flos-aquae.*

Table III.8-3a. Properties of Triclopyr Acid

Molecular formula	$C_7H_4Cl_3NO_3$
Molecular weight (g/mol)	256.48
Vapor pressure (mm Hg @ 20° C)	1.26×10^{-6}
Water solubility (g/mol @ 24.5° C)	440
Octanol-water partition coefficient (at pH 7)	0.36
pKa[1]	2.93
K_{oc}[2]	59 ml/g
BCF (Bluegill sunfish)	0.03
BCF (Catfish)	0.04

1. Martin, E.J., 1988.
2. Woodburn *et al.*, 1988

Table III.8-3b. Properties of the Triethylamine (TEA) Salt of Triclopyr)

Molecular formula	$C_{13}H_{18}O_3N_2Cl_3$
Molecular weight	355
Vapor pressure (mm Hg)	$< 1 \times 10^{-8}$
Water solubility (g/l)	0.412
Octanol-water Partition coefficient (at pH 7)	0.196

(WSSA, 1994)

Table III.8-3c. Properties of the Triclopyr-Butoxyethyl Ester (TBEE)

Molecular formula	$C_{13}H_{16}Cl_3NO_4$
Molecular weight (g/mol)	356.69
Vapor pressure (mm Hg @ 33° C)	1×10^{-5}
Water solubility (mg/l)	5.75
Octanol-water partition coefficient	4.1×10^{-4}

(WSSA, 1994)

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III.10 ADJUVANTS

OVERVIEW

An adjuvant is any material added to a herbicide spray solution that modifies or enhances the action of that solution. Many herbicides already contain adjuvants as part of their formulations. Some of these formulations can be used directly whereas others need to be applied in conjunction with one or more adjuvants. There are over 3,000 adjuvants available for use. These can be grouped into three general types of adjuvants, including activators, spray modifiers and utility modifiers.

Activators increase the activity of herbicides by modifying certain herbicide characteristics, including particle size of the herbicide spray, distribution of the spray on the plant, spray viscosity, evaporation rate, rate of herbicide uptake or solubility of the herbicide in the spray solution. Activators can be either nonionic (producing little or no ionization in water) or ionic (having a positive or negative charge). It is generally recommended that a cationic (positively charged) herbicide should not be used with an anionic (negatively charged) adjuvant (and vice versa) because oppositely charged compounds could react, diminishing the effects of the herbicide. Most activators have no charge and thus can be mixed readily with any herbicide. There are three categories of activators including surfactants, wetting agents and oils.

Surfactants primarily influence the ability of herbicides to penetrate the leaf's waxy cuticle. Emersed and floating aquatic plants develop waxy cuticles similar to terrestrial plants, whereas submersed plants do not. Most herbicides are prepared in a solution of water. Water is a chemically polar material and thus can be repelled by the waxy surface of leaves. Activators reduce the surface tension of water on plants, and allow the herbicide formulation to wet leaf surfaces and enter into the plant.

Wetting agents increase the ability of water to displace air or liquids from the leaf surface, allowing it to be wet by the herbicide. (Surfactants also have wetting properties but they vary in the degree of wetting they provide.) Wetting agents help spread the solution more evenly over the leaf.

Oils are usually marketed at a concentration of about 80% oil/20% surfactant and are added to water to increase the retention time of a solution on leaves, allowing for an increase in herbicide uptake.

Spray modifiers influence the delivery and placement of spray solution. They confine or alter the characteristics of the spray solution. They include thickening agents (i.e., invert emulsions and polymers), stickers, spreaders, spreader-stickers and foaming agents, which reduce herbicide drift and allow for more exact placement of the herbicide.

Thickening agents modify the viscosity of formulations to reduce or control drift, aid in dispersal and promote sinking. Inverts and polymers are two types of thickening agents commonly used in aquatic herbicide applications.

Invert emulsions are mixtures of inverting oil and water, having a mayonnaise-like appearance on the water surface and a snowflake-like appearance under the water surface. Depending on their solubility, herbicides dissolve in either the oil or water component. The adjuvant/herbicide emulsion sticks to leaves and stems of plants, thus reducing drift and increasing herbicide contact time with plants.

Polymers are long-chain carbon molecules which are up to 40,000 carbons in length, forming a thick mucus-like material which helps to break the surface tension of water and enhance sinking of

herbicides. Higher molecular weight polymers are generally formulated as an emulsion and are used as sinking agents. Lower molecular weight polymers are usually formulated as solutions and are used for drift control. Polymers are not very effective in water with a flow rate of greater than 3 cm/sec. as the herbicides/adjuvant mixtures may be washed off leaves before effective contact time is achieved. However, polymers are effective in still waters.

Stickers are made of vegetable gels, resins, mineral oils, vegetable oils, waxes or latex polymers. They promote the sticking of a spray to the sprayed surface. Stickers are usually used for application of fungicides and insecticides rather than herbicides.

Spreaders are blends of primarily nonionic surfactants used for spreading and sticking a spray to plant leaves. They are not as cost-effective as most surfactants but they can increase the effectiveness of some herbicides.

Spreader-stickers are combinations of the above two materials which provide additional retention of herbicide in wet conditions. These adjuvants are more expensive than surfactants and are not used very much in herbicide applications but they are used with fungicides and insecticides.

Foaming Agents are surfactants which are used with specialized spray applicators to create foam for reducing drift and evaporation. These agents are used infrequently for drift control of herbicide applications.

Utility Modifiers are rarely used in aquatic plant control. The addition of modifiers to a herbicide formulation expands the range of conditions (e.g., pH, hardness, etc.) under which a formulation can be used. Types of modifiers include emulsifiers, dispersants and stabilizing agents (including buffering agents and anti-foam agents). Buffering agents and anti-foam agents are used for aquatic plant management. Buffering agents are used to increase the dispersion or solubility of herbicides in alkaline or acid waters used in making up an herbicide solution. Anti-foam agents are mostly silicone-based and are used to eliminate foam in the spray tank, especially useful when mixing herbicides with soft water which usually creates a foaming problem. (above information adapted from: Aquatic Plant Identification and Herbicide Use Guide, 1988; Langeland, 1991)

COMMONLY USED ADJUVANTS

Table III.10-1 contains a partial listing of adjuvants used with aquatic herbicides.

TOXICITY

The toxicity of adjuvants is not as well characterized as the toxicity of herbicides. Very commonly, a study of the toxicity of a herbicide focuses on the active ingredient in the herbicide and neglects to consider the toxicity of adjuvants used during application of that herbicide.

Part of the reason for the limited toxicity information of adjuvants is that the regulation of adjuvants is not very rigorous. Adjuvants which are used in the application of herbicides on food crops come under the jurisdiction of the Food and Drug Administration (FDA). If an adjuvant is included in a pesticide's formulation, unless it has herbicidal properties, it is listed together with all other additives under "inert ingredients". If a herbicide formulation specifies on its EPA label that a particular adjuvant should be used during application, then the adjuvant also falls under the jurisdiction of the EPA. It is obvious that many adjuvants are not regulated at all. As a result, there are very few toxicity testing requirements and unless the manufacturer has conducted their own testing, there is little or no data available o the toxicity of these compounds (Edwards, personal communication, 1995).

Table III.10-1. Commonly Used Adjuvants

Name	Type	Action
Big Wet (E,F)	activator	nonionic/anion spreader, wetting agent, penetrant
Cide-Kick (E,F,S)	activator	nonionic wetting agent, activator, penetrant
Cide-Kick II (E,F,S)	activator	nonionic wetting agent, activator, penetrant
Ortho X-77 Spreader (E,F)	activator	nonionic spreader, activator
Asgrow "403" Invert Emulsifier (E,F,S)	spray modifier-invert	invert emulsion, drift control, reduce evaporation, increase droplet spreading and penetration, resist washoff
Bivert (E,F,S)	spray modifier-invert	invert emulsion, chemical encapsulating, suspending agent, deposition and retention agent, reduce drift and washoff
I'vod Inverting Oil (E,F,S)	Spray modifier-invert	invert emulsion, drift control, reduce evaporation, increase droplet spreading and penetration, resist washoff. (Dilution with #2 diesel oil or water required.)
Spra-Mate Invert Emulsion (E,F,S)	Spray modifier-invert emulsion	invert emulsion, drift control, reduce evaporation, increase droplet spreading and penetration, resist washoff (Dilution with #2 diesel oil or xylene required.)
Visko-Rhap (E,F,S)	Spray modifier-inverting oil	invert emulsion, reduce drift. (Can be diluted with #2 fuel oil or kerosene, if necessary)
Nalquatic (S)	Spray modifier-polymer	improve sinking, herbicide confinement and contact properties
Nalco-Trol (E)	Spray modifier-polymer	drift control, developed for Rodeo (glyphosate), diquat and 2,4-D; sinking agent for Hydrothal 191 (endothall)
Nalco-Trol II (E,S)	Spray modifier-polymer	sinking agent developed for Hydrothol 191 (endothall) and drift control for RODEO (glyphosate)
Poly Control	Spray modifier-polymer	drift control, sticking agent, nonionic
Poly Control 2 (S)	Spray modifier-polymer	drift control, sticking agent, nonionic
Submerge (S)	Spray modifier-polymer	sinking agent, contact confinement of herbicides (manufactured in both anionic and nonionic forms)

E - emersed plants
S - submersed plants
F - floating plants

(Aquatic Plant Identification and Herbicide Use Guide, 1988)

A survey of several adjuvant manufacturers indicated that while limited information on acute ecological toxicity (i.e., fish and aquatic invertebrates) was available, there was practically no information available on longer-term ecological toxicity. In addition, there was very little information available on the toxicity of adjuvants to mammals. An occasional acute toxicity test result was available, which was based on a single active ingredient in the adjuvant mixture. Such studies are of very limited usefulness in terms of characterizing the toxicity of an adjuvant since the toxicity of individual active ingredients in an adjuvant formulation can vary depending on the nature of the other compounds in the mixture.

In addition, even the available ecological acute toxicity tests are of limited usefulness. Often, in these tests, the adjuvant is tested as a full-strength material whereas during actual field application, an adjuvant is used in dilute form. Acute toxicity tests are useful in that they allow for a comparison of the acute toxicities between various materials. However, they offer no information on toxicity upon longer-term exposures to these materials at actual concentrations used.

Table III.10-2 summarizes the available ecological toxicity information for a number of adjuvants. Although use of adjuvants in Massachusetts is not as common as it is in areas with milder climates, such as Florida where herbicide applications are much more prevalent, selected adjuvants are still occasionally used by applicators in the state (Smith, personal communication, 1995).

For each adjuvant, the type of adjuvant is specified as well as the 96-hour LC50 value for rainbow trout and/or bluegill sunfish as well as the 48-hour LC50 for *Daphnia*, an aquatic invertebrate. The LC50 is defined as the concentration in water (in mg/l) which will kill fifty percent of the organisms in a specific test situation. The table also includes a qualitative toxicity designation (Christenson, 1976) for each adjuvant for fish and for invertebrates as defined below:

LC50	Classification
<1 mg/l	HT (Highly Toxic)
1-10 mg/l	MT (Moderately Toxic)
10-100 mg/l	ST (Slightly Toxic)
100, 1,000 mg/l	PN (Practically Nontoxic)
>1,000 mg/l	IH (Insignificant Hazard)

Table III.10-2. TOXICITY STUDIES IN AQUATIC ORGANISMS FOR SELECTED ADJUVANTS

Adjuvant	Adjuvant Type	96-hr LC50 (mg/l) rainbow trout	96-hr LC50 (mg/l) bluegill sunfish	REF	TOX	48-hr LC50 (mg/l) Daphnia	REF	TOX
#4 Fuel Oil	surfactant	---	91.0	1	ST	---		
"403"	invert emulsifier	---	37.0	1	ST	---		
Activator 90	surfactant	1.4	2.0	2	MT	2.0	2	MT
Agri-Dex	surfactant	>1,000	>1,000	2	PN	>1,000	2	IH
Arbor Chem (same as X-77)[7]	nonionic spreader; activator	4.2	4.3	3	MT	2.0	2	MT
Big Wet	nonionic/anionic spreader; wetting agent; penetrant	---	112.0	3	PN	---		
Big Sur 90	wetter/spreader	---	112.0	1	PN	---		
Cide-Kick	nonionic wetting agent; activator; penetrant	---	5.2	1,3	MT	3.9	4	MT
Entry II	surfactant	4.2	1.3	2	MT	2.0	2	MT
Frigate	surfactant	3.6	2.4	2	MT	11.0	2	ST
Herbex	surfactant	---	8100.0	1,3	IH	---		
I'VOD	spray modifer/invert	---	37.0	1	ST	---		
Induce	surfactant	5.6	7.5	2	MT	18.0	2	ST
Latex Paint	surfactant	---	560.0	1	PN	---		
LI-700	surfactant	130.0	210	2	PN	170.0	2	PN
Liqua-Wet	surfactant	13.0	11.0	2	T	7.2	2	MT
Nalco-trol	spray modifier polymer	>1,000	---	5	IH	280	5	PN
Nalcotrol II	spray modifier/polymer	>1,000	---	5	IH	270	5	PN

Table III.10-2. TOXICITY STUDIES IN AQUATIC ORGANISMS FOR SELECTED ADJUVANTS

Adjuvant	Adjuvant Type	96-hr LC50 (mg/l) rainbow trout	96-hr LC50 (mg/l) bluegill sunfish	REF	TOX	48-hr LC50 (mg/l) Daphnia	REF	TOX
Nalcotrolb	spray modifier/polymer	---	>1,000	5		---		
Nalquatic	spray modifier polymer	---	200.0	1	PN	---		
No Foam A	surfactant	3.3	6.0	2	MT	7.3	2	MT
Passage	surfactant	52.0	75.0	2	ST	17	2	ST
Polysar Latex	surfactant	---	3600.0	1	IH	---		
R-11	surfactant	3.8	4.2	2	MT	19	2	ST
Spra-Mate	spray modifer/invert	---	0.96	1	HT	---		
Spreader-Sticker	surfactant	36.0	35.0	2	ST	48.0	2	ST
Super Spreader 200	surfactant	4.2	9.3	2	MT	24.0	2	ST
Widespread	surfactant	6.6	7.0	2	MT	16.0	2	ST
X-77	nonionic spreader; activator	4.2	4.3	3	MT	2.0	2	MT

Explanatory Notes for Table III.10-2

TOXICITY CLASSIFICATION [6]

LC50	Classification
<1 mg/l	HT (Highly Toxic)
1 - 10 mg/l	MT (Moderately Toxic)
10-100 mg/l	ST (Slightly Toxic)
100-1,000 mg/l	PN (Practically Nontoxic)
>1,000 mg/l	IH (Insignificant Hazard)

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APPENDIX IV -- CLEAN LAKES PROGRAM PROJECTS

Table IV-1a. PROJECTS FUNDED BY THE CLEAN LAKES PROGRAM
Additional information in Table IV-1b by No.

No.	YEAR	LAKE	LOCATION	TYPE
1	1983	LAKE LASHAWAY	EAST BROOKFIELD	II
2	1983	WILLOW POND	NORTHAMPTON	II
3	1983	MARTINS POND	NORTH READING	I
4	1983	MARTINS POND	NORTH READING	M
5	1983	LAKE QUANNAPOWITT	WAKEFIELD	I
6	1983	ELL POND	MELROSE	I
7	1983	WALKER POND	STURBRIDGE	I
8	1983	BIG ALUM LAKE	STURBRIDGE	I
9	1983	LAKE MASSASOIT	SPRINGFIELD	I
10	1983	PUFFER'S POND	AMHERST	I
11	1983	METACOMET AND ARCADIA PONDS	BELCHERTOWN	I
12	1983	LONG POND	YARMOUTH	I
13	1983	CHEBACCO LAKE	HAMILTON/ESSEX	I
14	1983	PORTER LAKE	SPRINGFIELD	II
15	1983	PEQUOT POND	WESTFIELD/SOUTHAMPTON	I
16	1983	DUNN POND	GARDNER	II
17	1983	FLOATING BRIDGE POND	LYNN	I
18	1983	SLUICE AND FLAX PONDS	LYNN	I
19	1983	DUDLEY POND	WAYLAND	IID
20	1983	NORTH POND	HOPKINTON/UPTON/MILFORD	I
21	1983	LAKE WINTHROP	HOLLISTON	I
22	1983	LAKE WINTHROP	HOLLISTON	M
23	1983	CEDAR SWAMP POND	MILFORD	I
24	1983	LAKE RIPPLE	GRAFTON	I
25	1983	CHAUNCY LAKE	WESTBOROUGH	I
26	1983	BARTLETT POND	NORTHBOROUGH	I
27	1983	JENNINGS POND	NATICK	I
28	1983	HARDY POND	WALTHAM	I
29	1983	CONGAMOND LAKES	SOUTHWICK/SUFFIELD, CT	II
30	1983	LAKE BUEL	MONTEREY/NEW MARLBOROUGH	IID
31	1983	QUABOAG AND QUACUMQUASIT PONDS	BROOKFIELD/E. BROOKFIELD	I
32	1983	FORGE POND	WESTFORD/LITTLETON	I
33	1984	LAKE COCHITUATE	NATICK/FRAMINGHAM	II
34	1984	WEBSTER LAKE	WEBSTER	II
35	1984	LAKE QUINSIGAMOND	SHREWSBURY	II
36	1984	FIVE MILE PD./LOON L./L. LORRAINE	SPRINGFIELD	I
37	1984	GREAT POND	EASTHAM	I
38	1984	LAKE ELLIS	ATHOL	I
39	1984	LAKE MASSAPOAG	SHARON	I
40	1984	EAST LAKE WAUSHACUM	STERLING	II
41	1984	EAST AND WEST MONPONSETT PONDS	HALIFAX/HANSON	I
42	1984	ASHUMET POND	FALMOUTH	I
43	1984	HORN POND	WOBURN	I
44	1984	WHITMANS POND	WEYMOUTH	II
45	1984	LAKE LASHAWAY	EAST BROOKFIELD	II
46	1984	FLINT POND	SHREWSBURY/GRAFTON	M
47	1984	WYMAN POND	WESTMINSTER	II
48	1984	LAKE BOON	HUDSON/STOW	I
49	1984	CEDAR POND	STURBRIDGE	II
50	1984	ROCK POND	GEORGETOWN	I
51	1984	WEQUAQUET AND LONG PONDS	BARNSTABLE	I

52	1984	BARE HILL POND	HARVARD	I
53	1984	SPY POND	ARLINGTON	II
54	1984	POPULATIC POND	FRANKLIN/	I
55	1984	CEDAR POND	STURBRIDGE	M
56	1984	HAMILTON RESERVOIR	HOLLAND	M
57	1984	SHALLOW POND	BARNSTABLE	I
58	1984	BROWN'S POND	PEABODY	I
59	1984	PRINDLE POND	CHARLTON	M
60	1984	RED LILY POND	BARNSTABLE	I
61	1984	BROWN'S POND	PEABODY	M
62	1984	FORGE POND	GRANBY	I
63	1984	HILLS POND	ARLINGTON	I
64	1984	HALLS POND	BROOKLINE	I
65	1984	RED LILY POND	BARNSTABLE	M
66	1984	SALISBURY POND	WORCESTER	I
67	1984	BLACK'S NOOK	CAMBRIDGE	I
68	1984	LAGOON POND	OAK BLUFFS	I
69	1985	PONTOOSUC LAKE	PITTSFIELD/LANESBOROUGH	II
70	1985	PONTOOSUC LAKE	PITTSFIELD/LANESBOROUGH	II
71	1985	PONTOOSUC LAKE	PITTSFIELD/LANESBOROUGH	II
72	1985	LAKE MASSAPOAG	SHARON	II
73	1985	ONOTA LAKE	PITTSFIELD	I
74	1985	LAKE QUINSIGAMOND	WORCESTER	II
75	1985	DUDLEY POND	WAYLAND	II
76	1985	RICHMOND POND	RICHMOND/PITTSFIELD	I
77	1985	HARBOR POND	TOWNSEND	I
78	1985	HARDWICK POND	HARDWICK	M
79	1985	LAKE QUINSIGAMOND	WORCESTER	I
80	1985	UPPER MYSTIC LAKE	WINCHESTER	M
81	1985	FORT MEADOW RESERVOIR	MARLBORO/HUDSON	I
82	1985	LONG POND	DRACUT/ TYNGSBOROUGH, MA	I
83	1985	MILL POND	WEST NEWBURY	I
84	1985	WAUSHAKUM POND	FRAMINGHAM/ASHLAND	I
85	1985	WYMAN POND	WESTMINSTER	M
86	1985	BUTTONWOOD POND	NEW BEDFORD	I
87	1985	DOROTHY POND	MILLBURY	II
88	1985	LAKE SHIRLEY/SHIRLEY RESERVIOR	LUNENBERG	I
89	1985	LAKE HOLBROOK	HOLBROOK	I
90	1985	BULLOUGH'S POND	NEWTON	I
91	1986	INDIAN LAKE	WORCESTER	I
92	1986	PUFFER'S POND	AMHERST	II
93	1986	WALKER POND	STURBRIDGE	IIC
94	1986	LOST LAKE/KNOPS POND	GROTON	I
95	1986	INDIAN LAKE	WORCESTER	M
96	1986	PEQUOT POND	WESTFIELD/SOUTHAMPTON	II
97	1986	PEQUOT POND	WESTFIELD/SOUTHAMPTON	II
98	1986	QUABOAG/QUACUMQUASIT PONDS	BROOKFIELD/ E. BROOKFIELD	II
99	1986	LONG POND	YARMOUTH	II
100	1986	SLUICE/FLAX/FLOATING BR PDS	LYNN	II
101	1986	BILLINGTON SEA	PLYMOUTH	I
102	1986	MARTINS POND	NORTH READING	II
103	1986	FURNACE POND	PEMBROKE	I
104	1986	ELL POND	MELROSE	II
105	1986	LAKE METACOMET/LAKE ARCADIA	BELCHERTOWN	II
106	1986	LOST LAKE/KNOPS POND	GROTON	M
107	1986	WYMAN POND	WESTMINSTER	II
108	1986	LAKE BUEL	MONTEREY/NEW MARLBOROUGH	II
109	1986	NASHAWANNUCK POND	EASTHAMPTON	I

110	1986	EAGLE LAKE	HOLDEN	II
111	1986	BIG ALUM POND	STURBRIDGE	IIC
112	1986	LAKE CHAUNCY	WESTBOROUGH	II
113	1986	LAKE ARCADIA	BELCHERTOWN	II
114	1986	FAWN LAKE	BEDFORD	I
115	1986	SILVER LAKE	WILMINGTON	I
116	1986	PRINDLE POND	CHARLTON	I
117	1986	L. SANDY BOTTOM PD.	PEMBROKE	I
118	1986	OLDHAM POND	PEMBROKE	I
119	1986	STETSON POND	PEMBROKE	I
120	1986	HARDY POND	WALTHAM	II
121	1986	WEDGE POND	WINCHESTER	I
122	1986	CHEBACCO LAKE	HAMILTON/ESSEX	M
123	1986	LAKE QUANNAPOWITT	WAKEFIELD	II
124	1986	LITTLE HARBOR POND	COHASSET	I
125	1986	FOUNDRY POND	HINGHAM	I
126	1986	DIMMOCK POND	SPRINGFIELD	I
127	1987	KENDALL POND	GARDNER	I
128	1987	VAN HORN POND	SPRINGFIELD	I
129	1987	HARDWICK POND	HARDWICK	I
130	1987	ASHFIELD POND	ASHFIELD	I
131	1987	SHEEP POND	BREWSTER	I
132	1987	RED LILY POND	BARNSTABLE/(CRAIGVILLE)	II
133	1987	STOCKBRIDGE BOWL	STOCKBRIDGE	I
134	1987	FOREST LAKE	METHUEN	I
135	1987	LONG POND	LITTLETON	I
136	1987	GREAT POND	EASTHAM	II
137	1987	HERRING POND	EASTHAM	I
138	1987	PROSPECT LAKE	EGREMONT	I
139	1987	LAKE MASCUPPIC	TYNGSBOROUGH/DRACUT	I
140	1987	MANSFIELD LAKE	GREAT BARRINGTON	I
141	1987	PORTER LAKE	SPRINGFIELD	II
142	1987	FORGE POND	WESTFORD/LITTLETON	II
143	1987	HILL'S POND	ARLINGTON	IIC
144	1988	CONGAMOND LAKES	SOUTHWICK	II
145	1988	DUDLEY POND	WAYLAND	II

Table IV-1b PROJECTS FUNDED BY THE CLEAN LAKES PROGRAM
Additional information in Table IV-1a by No.

No.	DESCRIPTION	TOTALCOST
1	ADD SECONDARY OUTLET FOR DRAWDOWN,BUILD CHECKDAM AT INLET	397600
2	DREDGING, BANK STABILIZATION, INLET/OUTLET RECONSTRUCTION	194273
3	DIAGNOSTIC/FEASIBILITY STUDY	40000
4	ALUM TREATMENT	10000
5	DIAGNOSTIC/FEASIBILITY STUDY	75000
6	DIAGNOSTIC/FEASIBILITY STUDY	50000
7	DIAGNOSTIC/FEASIBILITY STUDY	60000
8	FEASIBILITY STUDY	15000
9	DIAGNOSTIC/FEASIBILITY STUDY	75000
10	DIAGNOSTIC/FEASIBILITY STUDY	60000
11	DIAGNOSTIC/FEASIBILITY STUDY	85000
12	DIAGNOSTIC/FEASIBILITY STUDY	75000
13	DIAGNOSTIC/FEASIBILITY STUDY	60000
14	DESIGN & CONSTRUCTION--EROSION CONTROL/DREDGING/SEDIMENT BERMS	846340
15	DIAGNOSTIC/FEASIBILITY STUDY	60000
16	RESTORATION: DREDGING, STORMWATER REROUTING TO FILTER DIKE	1250000
17	DIAGNOSTIC/FEASIBILITY STUDY	50000
18	DIAGNOSTIC/FEASIBILITY STUDY	130000
19	DESIGN OF STORMWATER CONTROL SYSTEM/WEED HARVESTING	47500
20	DIAGNOSTIC/FEASIBILITY STUDY	71140
21	DIAGNOSTIC/FEASIBILITY STUDY	22500
22	BOTTOM WEED BARRIER	30000
23	DIAGNOSTIC/FEASIBILITY STUDY	40000
24	DIAGNOSTIC/FEASIBILITY STUDY	60000
25	DIAGNOSTIC/FEASIBILITY STUDY	45000
26	DIAGNOSTIC/FEASIBILITY STUDY	75000
27	DIAGNOSTIC/FEASIBILITY STUDY	50000
28	DIAGNOSTIC/FEASIBILITY STUDY	75000
29	HARVESTER PURCHASE/O + M/ALUM TREATMENT DESIGN	94000
30	HARVESTER SYSTEM PURCHASE/PRELIM DESIGN OUTLET CONTROL STRUCT	94000
31	DIAGNOSTIC/FEASIBILITY STUDY	70000
32	DIAGNOSTIC/FEASIBILITY STUDY	60000
33	PHASE II DESIGN & CONST OF FILTERBERM &2 RE/DETENTION BASINS	2140000
34	CONSTRUCT 2 SEDIMENT. PONDS;SEPTIC TANK INSPEC./MAINTEN.PROG.	160000
35	SEWER INSPECTION STUDY	22500
36	DIAGNOSTIC/FEASIBILITY STUDY	200000
37	DIAGNOSTIC/FEASIBILITY STUDY	75000
38	DIAGNOSTIC/FEASIBILITY STUDY	60000
39	PARTIAL DIAGNOSTIC/FEASIBILITY STUDY	33000
40	SEPTIC LEACHATE DETECTOR STUDY/ PUBLIC AWARENESS PROGRAM	14600
41	DIAGNOSTIC/FEASIBILITY STUDY	96036
42	DIAGNOSTIC/FEASIBILITY STUDY	90000
43	DIAGNOSTIC/FEASIBILITY STUDY	75000
44	DESIGN/CONSTRUCTION OF NUTRIENT UPTAKE POND	156200
45	LIMITED DREDGING PROJECT	50000
46	HYDRORAKING AND WEED HARVESTING	107200
47	HARVESTER PURCHASE AND MAINTENANCE	118000
48	DIAGNOSTIC/FEASIBILITY STUDY	48000
49	DREDGING OF BEACH AREA	17500
50	DIAGNOSTIC/FEASIBILITY STUDY	60000
51	DIAGNOSTIC/FEASIBILITY STUDY	150000

52	DIAGNOSTIC/FEASIBILITY STUDY	50000
53	ALUM, INLET DIVERSION STRUCTURE, GRNDWATER IMPORT FEAS STUDY	394448
54	DIAGNOSTIC/FEASIBILITY STUDY	60000
55	CHEMICAL TREATMENT	22500
56	HYDRORAKING	40000
57	DIAGNOSTIC/FEASIBILITY STUDY	70000
58	DIAGNOSTIC/FEASIBILITY STUDY	75000
59	HERBICIDE TREATMENT	2500
60	DIAGNOSTIC/FEASIBILITY STUDY	50000
61	CHEMICAL TREATMENT OF NUISANCE ALGAE	2000
62	DIAGNOSTIC/FEASIBILITY STUDY	60000
63	DIAGNOSTIC/FEASIBILITY STUDY	45000
64	DIAGNOSTIC/FEASIBILITY STUDY	60000
65	HYDRORAKING	45000
66	DIAGNOSTIC/FEASIBILITY STUDY	61250
67	DIAGNOSTIC/FEASIBILITY STUDY	40000
68	DIAGNOSTIC/FEASIBILITY STUDY	45000
69	TECHNICAL AND ENVIRON EVALUATION, WSHED MGMT. ACTION PLAN	35000
70	PURCHASE HARVESTING EQUIPMENT	290500
71	AGRICULTURAL WASTE MANAGEMENT	140000
72	FEASIBILITY/FINAL DESIGN/CONST STORMWATER DETENTION BASIN	325000
73	DIAGNOSTIC/FEASIBILITY STUDY	90000
74	BELMONT STREET DRAIN INSPECTION STUDY	75000
75	DESIGN/CONST OF PHASE 2 STORMWATER CONTROLS	720000
76	DIAGNOSTIC/FEASIBILITY STUDY	50000
77	DIAGNOSTIC/FEASIBILITY STUDY	80000
78	HARVESTING 25 ACRES OF FANWORT	15000
79	BELMONT STREET DRAIN FEASIBILITY STUDY	30000
80	PHASE II MAINTENANCE, HARVESTING AND RAKING	41000
81	DIAGNOSTIC/FEASIBILITY STUDY	85000
82	DIAGNOSTIC/FEASIBILITY STUDY	70000
83	DIAGNOSTIC/FEASIBILITY STUDY	45000
84	DIAGNOSTIC/FEASIBILITY STUDY	75000
85	HYDRO-RAKING CONTRACT	26000
86	DIAGNOSTIC/FEASIBILITY STUDY	70000
87	INLET WETLANDS FILTRATION/SEDIMENTATION BASIN	270000
88	DIAGNOSTIC/FEASIBILITY STUDY	90000
89	DIAGNOSTIC/FEASIBILITY STUDY	55000
90	DIAGNOSTIC/FEASIBILITY STUDY	58000
91	DIAGNOSTIC/FEASIBILITY STUDY	32000
92	DESIGN, DREDGING, AND CONSTRUCTION OF A SEDIMENTATION BASIN	338800
93	DREDGING - MACROPHYTE CONTROL VIA SEDIMENT REMOVAL	1740600
94	DIAGNOSTIC/FEASIBILITY STUDY	88000
95	MAINTENANCE PROJECT - HERBICIDE APPLICATION	10000
96	TWO SEDIMENT TRAPS AND HYDRORAKING	41600
97	TWO SEDIMENT TRAPS AND HYDRORAKING	28400
98	CONST FLOW BARRIER BETWEEN PONDS, AERATION, WSHED MGT.	107000
99	OUTLET-INLET MODIF.,STORMWATER BASINS,PUBLIC ED,LOCAL DREDG.	200000
100	STORM SEWER DIVERSION AND NEW OUTLETS	1500000
101	DIAGNOSTIC/FEASIBILITY STUDY	80000
102	ALUM TREATMENT, PUBLIC EDUCATION PROGRAM	15000
103	DIAGNOSTIC/FEASIBILITY STUDY	75000
104	SEWER INSPECTION STUDY	200000
105	SEPTIC SYSTEM INSPECTION PROGRAM	25000
106	WEED HARVESTING	25000
107	TRUCK PURCHASE	25000
108	FINAL DESIGN AND CONSTRUCTION OF A LAKE DRAWDOWN STRUCTURE	538000
109	DIAGNOSTIC/FEASIBILITY STUDY	80000

110	DRAWDOWN, REPAIRS TO DAM, AND DREDGING DURING DRAWDOWN	204000
111	DAM MODIFICATIONS - HYPOLIMNETIC WITHDRAWAL	153030
112	FINAL DESIGN, CONSTRUCTION OVERSIGHT, CONSTRUCTION	304000
113	OUTLET RECONSTRUCTION	15000
114	DIAGNOSTIC/FEASIBILITY STUDY	35000
115	DIAGNOSTIC/FEASIBILITY STUDY	72000
116	DIAGNOSTIC/FEASIBILITY STUDY	45000
117	DIAGNOSTIC/FEASIBILITY STUDY	70000
118	DIAGNOSTIC/FEASIBILITY STUDY	75000
119	DIAGNOSTIC/FEASIBILITY STUDY	80000
120	DREDGING,MODIF TO OUTLET, REROUTE STORM DRAINS,PUBLIC EDUC.	1600000
121	DIAGNOSTIC/FEASIBILITY STUDY	80000
122	MAINTENANCE PROJECT - MECHANICAL RAKING AND BENTHIC BARRIERS	60000
123	FILTER BERM/OUTLET MODIFICATION/DREDGING/HARVERSTER PURCHASE	660000
124	DIAGNOSTIC/FEASIBILITY STUDY	44157
125	DIAGNOSTIC/FEASIBILITY STUDY	55000
126	DIAGNOSTIC/FEASIBILITY STUDY	65000
127	DIAGNOSTIC/FEASIBILITY STUDY	75000
128	DIAGNOSTIC/ FEASIBILITY STUDY	100000
129	DIAGNOSTIC/FEASIBILITY STUDY	30000
130	DIAGNOSTIC/FEASIBILITY STUDY	76000
131	DIAGNOSTIC/FEASIBILITY STUDY	95000
132	COMMUNITY SEPTIC SYSTEM/MOSQUITO DITCH DIVERSION	495460
133	DIAGNOSTIC/FEASIBILITY STUDY	66000
134	DIAGNOSTIC/FEASIBILITY STUDY	72000
135	DIAGNOSTIC/FEASIBILITY STUDY	90000
136	GROUNDWATER MONITORING WELLS/WSHED MANAGEMENT/BENTHIC BARRIER	114000
137	DIAGNOSTIC/FEASIBILITY STUDY	86000
138	DIAGNOSTIC/FEASIBILITY STUDY	65000
139	LIMITED DIAGNOSTIC/FEASIBILITY STUDY	45000
140	DIAGNOSTIC/FEASIBILITY STUDY	60000
141	EROSION CONTROL	1480000
142	WINTER DRAWDOWN/SUMMER HYPOLIMNETIC WITHDRAWAL/WSHED MAN.	149000
143	PHASE II PROJECT - DREDGING, SEDIMENT BASINS, GWATER IMPORT	155000
144	ALUM TREATMENT	150000
145	STORMWATER RENOVATIONS	480000

APPENDIX V -- RARE AND ENDANGERED SPECIES LIST

Massachusetts List of Endangered, Threatened and Special Concern Species

Definitions

"Endangered" (E) species are native species which are in danger of extinction throughout all or part of their range, or which are in danger of extirpation from Massachusetts, as documented by biological research and inventory.

"Threatened" (T) species are native species which are likely to become endangered in the forseeable future, or which are declining or rare as determined by biological research and inventory.

"Special concern" (SC) species are native species which have been documented by biological research or inventory to have suffered a decline that could threaten the species if allowed to continue unchecked, or which occur in such small numbers or with such restricted distribution or specialized habitat requirements that they could easily become threatened within Massachusetts.

Any native species listed as endangered or threatened by the U.S. Fish and Wildlife Service is also included on the state list. The rules and regulations and precise definitions relative to the establishment of the Commonwealth's list of endangered, threatened, and special concern species are set forth in 321 CMR 10.00 et seq. Species in BOLD TYPE may occur in Massachusetts wetlands. Click here for a key to the Federal Status abbreviations appearing on the list.

1. Introduction - The list in 321 CMR 10.60 contains the names of all species of plants and animals which have been determined to be Endangered, Threatened, or of Special Concern pursuant to M.G.L. c. 131A and 321 CMR 10.03.

2. List Format - The columns entitled "Common Name" and "Scientific Name" define the species listed. In the "Status" columns the following symbols are used: "E" for Endangered, "T" for Threatened, and "SC" for Special Concern. The status defined under the "MA" column denotes the official status of the species in Massachusetts pursuant to M.G.L. c. 131A and 321 CMR 10.00. The status under the "US" column is the status of the species under the federal Endangered Species Act at the time of the latest revision of 321 CMR 10.00 and is given for informational purposes only. Recent changes in the federal list might not be reflected on this list. The U.S. Fish and Wildlife Service should be consulted for official and up to date information on the federal status of any species. Inquiries may be made by writing to U.S. Fish and Wildlife Service, 400 Ralph Pill Marketplace, 22 Bridge Street, Concord, NH 03301-4901. The "Taxonomic Family/Taxonomic Group" column of the list is included for the purpose of organization. The "Notes" column directs the reader to footnotes which further define or clarify the status of a species or alternative names of species.

3. Organization of the List - The list is generally organized according to the relationship of the listed species as determined by the science of taxonomy, which groups and categorizes species that are similar on the basis of shared evolutionary descent. The most basic division in the list is between animals and plants. Within animals the list is divided between vertebrates, (animals with backbones) and invertebrates (animals without backbones). Within vertebrates, invertebrates, and plants, the list is further divided into categories which are generally recognized, such as fish, mammals, dragonflies, and violets. All such information has no regulatory effect and is provided only for the

purpose of organizing the list. The following outline shows the taxonomic categories used and their order. A species name index is provided after the list at 321 CMR 10.61 to assist the reader in finding species on the list.

Outline of State List:

ANIMALS

Vertebrates
Fish
Amphibians
Reptiles
Birds
Mammals
Invertebrates
Sponges
Flatworms
Moss Animals
Segmented Worms
Snails
Mussels
Crustaceans
Dragonflies
Damselflies
Beetles
Butterflies and Moths

PLANTS

Aceraceae (Maples)
Adiantaceae (Cliff Ferns)...
through...(alphabetically by scientific family name)
Verbenaceae (Vervains)
Violaceae (Violets)

4. The List - The Massachusetts List of Endangered, Threatened, and Special Concern species follows:

(Some of the species names have links: by clicking on those links, you will be directed to that species' fact sheet)

List of Rare Species in Massachusetts

VERTEBRATES:

Common Name	Scientific Name	MA Status	Fed Status	Notes
Fish				
American Brook Lamprey	*Lampetra appendix*	T		

Shortnose Sturgeon	*Acipenser brevirostrum*	E	E	
Atlantic Sturgeon	*Acipenser oxyrinchus*	E		
Lake Chub	*Couesius plumbeus*	E		
Eastern Silvery Minnow	*Hybognathus regius*	SC		
Bridle Shiner	*Notropis bifrenatus*	SC		
Northern Redbelly Dace	*Phoxinus eos*	E		
Longnose Sucker	*Catostomus catostomus*	SC		
Burbot	*Lota lota*	SC		
Threespine Stickleback	*Gasterosteus aculeatus*	T		1
Amphibians				
Jefferson Salamander	*Ambystoma jeffersonianum*	SC		2
Blue-Spotted Salamander	*Ambystoma laterale*	SC		3
Marbled Salamander	*Ambystoma opacum*	T		
Spring Salamander	*Gyrinophilus porphyriticus*	SC		
Four-Toed Salamander	*Hemidactylium scutatum*	SC		
Eastern Spadefoot	*Scaphiopus holbrookii*	T		
Reptiles				
Loggerhead Seaturtle	*Caretta caretta*	T	T	
Green Seaturtle	*Chelonia mydas*	T	T	
Hawksbill Seaturtle	*Eretmochelys imbricata*	E	E	
Kemp's Ridley Seaturtle	*Lepidochelys kempii*	E	E	
Leatherback Seaturtle	*Dermochelys coriacea*	E	E	
Spotted Turtle	*Clemmys guttata*	SC		
Wood Turtle	*Clemmys insculpta*	SC		
Bog Turtle	*Clemmys muhlenbergii*	E		
Blanding's Turtle	*Emydoidea blandingii*	T		
Diamondback Terrapin	*Malaclemys terrapin*	T		
Eastern Red-bellied Cooter	*Pseudemys rubriventris*	E	E	4
Eastern Box Turtle	*Terrapene carolina*	SC		
Eastern Wormsnake	*Carphophis amoenus*	T		
Eastern Ratsnake	*Elaphe obsoleta*	E		
Copperhead	*Agkistrodon contortrix*	E		
Timber Rattlesnake	*Crotalus horridus*	E		
Birds				
Common Loon	*Gavia immer*	SC		
Pied-Billed Grebe	*Podilymbus podiceps*	E		
Leach's Storm-Petrel	*Oceanodroma leucorhoa*	E		
American Bittern	*Botaurus lentiginosus*	E		
Least Bittern	*Ixobrychus exilis*	E		
Bald Eagle	*Haliaeetus leucocephalus*	E	T	

Northern Harrier	*Circus cyaneus*	T	
Sharp-Shinned Hawk	*Accipiter striatus*	SC	
Peregrine Falcon	*Falco peregrinus*	E	
King Rail	*Rallus elegans*	T	
Common Moorhen	*Gallinula chloropus*	SC	
Piping Plover	*Charadrius melodus*	T	T
Upland Sandpiper	*Bartramia longicauda*	E	
Roseate Tern	*Sterna dougallii*	E	E
Common Tern	*Sterna hirundo*	SC	
Arctic Tern	*Sterna paradisaea*	SC	
Least Tern	*Sterna antillarum*	SC	
Barn Owl	*Tyto alba*	SC	
Long-Eared Owl	*Asio otus*	SC	
Short-Eared Owl	*Asio flammeus*	E	
Sedge Wren	*Cistothorus platensis*	E	
Golden-Winged Warbler	*Vermivora chrysoptera*	E	
Northern Parula	*Parula americana*	T	
Blackpoll Warbler	*Dendroica striata*	SC	
Mourning Warbler	*Oporornis philadelphia*	SC	
Vesper Sparrow	*Pooecetes gramineus*	T	
Grasshopper Sparrow	*Ammodramus savannarum*	T	
Henslow's Sparrow	*Ammodramus henslowii*	E	
Mammals			
Water Shrew	*Sorex palustris*	SC	
Rock Shrew	*Sorex dispar*	SC	
Indiana Myotis	*Myotis sodalis*	E	E
Small-Footed Myotis	*Myotis leibii*	SC	
Southern Bog Lemming	*Synaptomys cooperi*	SC	
Sperm Whale	*Physeter catodon*	E	E
Fin Whale	*Balaenoptera physalus*	E	E
Sei Whale	*Balaenoptera borealis*	E	E
Blue Whale	*Balaenoptera musculus*	E	E
Humpback Whale	*Megaptera novaeangliae*	E	E
Northern Right Whale	*Eubalaena glacialis*	E	E

INVERTEBRATES:

Common Name	Scientific Name	MA Status	Fed Status	Notes
Sponges				
Smooth Branched Sponge	*Spongilla aspinosa*	SC		
Flatworms				
Sunderland Spring Planarian	*Polycelis remota*	E		

Moss Animals			
Carter's Moss Animal	*Lophopodella carteri*	SC	
Segmented Worms			
New England Medicinal Leech	*Macrobdella sestertia*	SC	
Snails			
New England Siltsnail	*Cincinnatia winkleyi*	SC	
Walker's Limpet	*Ferrissia walkeri*	SC	
Coastal Marsh Snail	*Littoridinops tenuipes*	SC	
Slender Walker	*Pomatiopsis lapidaria*	E	
Pilsbry's Spire Snail	*Pyrgulopsis lustrica*	E	
Boreal Turret Snail	*Valvata sincera*	E	
Olive Vertigo	*Vertigo perryi*	SC	
Mussels			
Dwarf Wedgemussel	*Alasmidonta heterodon*	E	E
Triangle Floater	*Alasmidonta undulata*	SC	
Swollen Wedgemussel	*Alasmidonta varicosa*	E	
Yellow Lampmussel	*Lampsilis cariosa*	E	
Tidewater Mucket	*Leptodea ochracea*	SC	
Eastern Pondmussel	*Ligumia nasuta*	SC	
Creeper	*Strophitus undulatus*	SC	
Crustaceans			
Appalachian Brook Crayfish	*Cambarus bartonii*	SC	
Mystic Valley Amphipod	*Crangonyx aberrans*	SC	
Intricate Fairy Shrimp	*Eubranchipus intricatus*	SC	
Agassiz's Clam Shrimp	*Eulimnadia agassizii*	E	
Northern Spring Amphipod	*Gammarus pseudolimnaeus*	SC	
American Clam Shrimp	*Limnadia lenticularis*	SC	
Taconic Cave Amphipod	*Stygobromus borealis*	E	
Piedmont Groundwater Amphipod	*Stygobromus tenuis tenuis*	SC	
Coastal Swamp Amphipod	*Synurella chamberlaini*	SC	
Dragonflies			
Spatterdock Darner	*Aeshna mutata*	E	
Subarctic Darner	*Aeshna subarctica*	T	
Comet Darner	*Anax longipes*	SC	
Ocellated Darner	*Boyeria grafiana*	SC	
Spine-Crowned Clubtail	*Gomphus abbreviatus*	E	
Beaver Pond Clubtail	*Gomphus borealis*	SC	
Harpoon Clubtail	*Gomphus descriptus*	E	
Midland Clubtail	*Gomphus fraternus*	E	

Rapids Clubtail	*Gomphus quadricolor*	T	
Cobra Clubtail	*Gomphus vastus*	SC	
Skillet Clubtail	*Gomphus ventricosus*	SC	
Umber Shadowdragon	*Neurocordulia obsoleta*	SC	
Stygian Shadowdragon	*Neurocordulia yamaskanensis*	SC	
Brook Snaketail	*Ophiogomphus aspersus*	SC	
Riffle Snaketail	*Ophiogomphus carolus*	T	
Lake Emerald	*Somatochlora cingulata*	SC	
Ski-tailed Emerald	*Somatochlora elongata*	SC	
Forcipate Emerald	*Somatochlora forcipata*	SC	
Coppery Emerald	*Somatochlora georgiana*	E	
Incurvate Emerald	*Somatochlora incurvata*	T	
Kennedy's Emerald	*Somatochlora kennedyi*	E	
Mocha Emerald	*Somatochlora linearis*	SC	
Riverine Clubtail	*Stylurus amnicola*	E	
Zebra Clubtail	*Stylurus scudderi*	E	
Arrow Clubtail	*Stylurus spiniceps*	T	
Ebony Boghaunter	*Williamsonia fletcheri*	E	
Ringed Boghaunter	*Williamsonia lintneri*	E	
Damselflies			
Tule Bluet	*Enallagma carunculatum*	SC	
Attenuated Bluet	*Enallagma daeckii*	SC	
New England Bluet	*Enallagma laterale*	SC	
Scarlet Bluet	*Enallagma pictum*	T	
Pine Barrens Bluet	*Enallagma recurvatum*	T	
Beetles			
Northeastern Beach Tiger Beetle	*Cicindela dorsalis dorsalis*	E	T
Twelve-Spotted Tiger Beetle	*Cicindela duodecimguttata*	SC	
Bank Tiger Beetle	*Cicindela limbalis*	SC	
Cobblestone Tiger Beetle	*Cicindela marginipennis*	E	
Barrens Tiger Beetle	*Cicindela patruela*	SC	
Puritan Tiger Beetle	*Cicindela puritana*	E	T
Purple Tiger Beetle	*Cicindela purpurea*	SC	
Hentz's Redbelly Tiger Beetle	*Cicindela rufiventris hentzii*	T	
Elderberry Long-Horned Beetle	*Desmocerus palliatus*	SC	
American Burying Beetle	*Nicrophorus americanus*	E	E
Butterflies and Moths			
Coastal Heathland Cutworm	*Abagrotis nefascia*	SC	
Barrens Daggermoth	*Acronicta albarufa*	T	
Spiny Oakworm	*Anisota stigma*	SC	
Coastal Plain Apamea Moth	*Apamea mixta*	SC	
New Jersey Tea Inchworm	*Apodrepanulatrix liberaria*	E	

Straight Lined Mallow Moth	*Bagisara rectifascia*	SC	
Hessel's Hairstreak	*Callophrys hesseli*	SC	
Gerhard's Underwing	*Catocala herodias gerhardi*	SC	
Melsheimer's Sack Bearer	*Cicinnus melsheimeri*	T	
Chain Dot Geometer	*Cingilia catenaria*	SC	
Unexpected Cycnia	*Cycnia inopinatus*	T	
Imperial Moth	*Eacles imperialis*	T	
Early Hairstreak	*Erora laeta*	T	
Persius Duskywing	*Erynnis persius persius*	E	
Sandplain Euchlaena	*Euchlaena madusaria*	SC	
The Pink Streak	*Faronta rubripennis*	T	
Oak Hairstreak	*Satyrium favonius*	SC	
Oithona Tiger Moth	*Grammia oithona*	E	
Phyllira Tiger Moth	*Grammia phyllira*	E	
Williams' Tigermoth	*Grammia williamsii*	T	
Slender Clearwing Sphinx Moth	*Hemaris gracilis*	SC	
Barrens Buckmoth	*Hemileuca maia*	SC	
Buchholz's Gray	*Hypomecis buchholzaria*	E	
Pine Barrens Itame	*Itame* sp. 1	SC	5
Pale Green Pinion Moth	*Lithophane viridipallens*	SC	
Pine Barrens Lycia	*Lycia ypsilon*	T	
Barrens Metarranthis	*Metarranthis apiciaria*	E	
Coastal Swamp Metarranthis	*Metarranthis pilosaria*	SC	
Northern Brocade Moth	*Oligia hausta*	SC	
Dune Noctuid Moth	*Oncocnemis riparia*	SC	
Pitcher Plant Borer	*Papaipema appassionata*	T	
Ostrich Fern Borer	*Papaipema* sp. 2	.SC	6
Chain Fern Borer	*Papaipema stenocelis*	T	
Water-Willow Stem Borer	*Papaipema sulphurata*	T	
Eastern Veined White	*Pieris oleracea*	T	
Pink Sallow Moth	*Psectraglaea carnosa*	SC	
Southern Ptichodis	*Ptichodis bistrigata*	T	
Orange Sallow Moth	*Rhodoecia aurantiago*	T	
Three-Lined Angle Moth	*Semiothisa eremiata*	T	
Spartina Borer	*Spartiniphaga inops*	SC	
Faded Gray Geometer	*Stenoporpia polygrammaria*	T	
Pine Barrens Zale	*Zale* sp. 1	SC	7
Pine Barrens Zanclognatha	*Zanclognatha martha*	T	

PLANTS

Common Name	Scientific Name	MA Status	Fed Status	Notes
Aceraceae (Maples)				
Black Maple	*Acer nigrum*	SC		

Adiantaceae (Cliff Ferns)		
Fragile Rock-Brake	*Cryptogramma stelleri*	E
Alismataceae (Arrowheads)		
Estuary Arrowhead	*Sagittaria calycina* var *spongiosa*	E
Wapato	*Sagittaria cuneata*	T
River Arrowhead	*Sagittaria subulata* var. *subulata*	E
Terete Arrowhead	*Sagittaria teres*	SC
Apiaceae (Parsleys, Angelicas)		
Hemlock Parsley	*Conioselinum chinense*	SC
Saltpond Pennywort	*Hydrocotyle verticillata*	T
Canadian Sanicle	*Sanicula canadensis*	T
Long-Styled Sanicle	*Sanicula odorata*	T
Aquifoliaceae (Hollies)		
Mountain Winterberry	*Ilex montana*	E
Araceae (Arums)		
Green Dragon	*Arisaema dracontium*	T
Golden Club	*Orontium aquaticum*	E
Araliaceae (Ginsengs)		
Ginseng	*Panax quinquefolius*	SC
Asclepiadaceae (Milkweeds)		
Purple Milkweed	*Asclepias purpurascens*	E
Linear-Leaved Milkweed	*Asclepias verticillata*	T
Aspleniaceae (Spleenworts)		
Mountain Spleenwort	*Asplenium montanum*	E
Wall-Rue Spleenwort	*Asplenium ruta-muraria*	T
Asteraceae (Asters, Composites)		
Boreal Wormwood	*Artemisia campestris* ssp. *borealis*	E
Eaton's Beggar-ticks	*Bidens eatonii*	E
Estuary Beggar-ticks	*Bidens hyperborea* var. *colpophila*	E
Cornel-Leaved Aster	*Doellingeria infirma*	E
Lesser Snakeroot	*Eupatorium aromaticum*	E
New England Boneset	*Eupatorium leucolepis* var. *novae-angliae*	E
Purple Cudweed	*Gamochaeta purpurea*	E
New England Blazing Star	*Liatris scariosa* var. *novae-angliae*	SC
Sweet Coltsfoot	*Petasites frigidus* var. *palmatus*	E

Lion's Foot	*Prenanthes serpentaria*	E
Sclerolepis	*Sclerolepis uniflora*	E
Large-Leaved Goldenrod	*Solidago macrophylla*	T
Upland White Aster	*Solidago ptarmicoides*	E
Rand's Goldenrod	*Solidago simplex* ssp. *randii*	E
Eastern Silvery Aster	*Symphyotrichum concolor*	E
Crooked-Stem Aster	*Symphyotrichum prenanthoides*	T
Tradescant's Aster	*Symphyotrichum tradescantii*	T
Betulaceae (Birches, Alders)		
Mountain Alder	*Alnus viridis* ssp. *crispa*	T
Swamp Birch	*Betula pumila*	E
Boraginaceae (Borages)		
Oysterleaf	*Mertensia maritima*	E
Brassicaceae (Mustards)		
Smooth Rock-cress	*Arabis laevigata*	T
Lyre-Leaved Rock-cress	*Arabis lyrata*	E
Green Rock-cress	*Arabis missouriensis*	T
Purple Cress	*Cardamine douglassii*	E
Long's Bitter-cress	*Cardamine longii*	E
Fen Cuckoo Flower	*Cardamine pratensis* var. *palustris*	T
Cactaceae (Cacti)		
Prickly Pear	*Opuntia humifusa*	E
Campanulaceae (Bluebells, Lobelias)		
Great Blue Lobelia	*Lobelia siphilitica*	E
Caprifoliaceae (Honeysuckles)		
Hairy Honeysuckle	*Lonicera hirsuta*	E
Snowberry	*Symphoricarpos albus* var. *albus*	E
Broad Tinker's-weed	*Triosteum perfoliatum*	E
Downy Arrowwood	*Viburnum rafinesquianum*	E
Caryophyllaceae (Pinks, Sandworts)		
Nodding Chickweed	*Cerastium nutans*	E
Michaux's Sandwort	*Minuartia michauxii*	T
Large-leaved Sandwort	*Moehringia macrophylla*	E
Silverling	*Paronychia argyrocoma*	E
Knotted Pearlwort	*Sagina nodosa* ssp. *nodosa*	T
Chenopodiaceae (Saltworts)		

American Sea-blite	*Suaeda americana*	SC
Cistaceae (Rockroses, Pinweeds)		
Bushy Rockrose	*Helianthemum dumosum*	SC
Beaded Pinweed	*Lechea pulchella* var. *monoliformis*	E
Clusiaceae (St. John's-worts)		
Creeping St. John's-wort	*Hypericum adpressum*	T
Giant St. John's-wort	*Hypericum ascyron*	E
St. Andrew's Cross	*Hypericum hypericoides* ssp. *multicaule*	E
Convolvulaceae (Morning Glories)		
Low Bindweed	*Calystegia spithamaea*	E
Crassulaceae (Sedums)		
Pygmyweed	*Crassula aquatica*	T
Cupressaceae (Cedars, Junipers)		
Arborvitae	*Thuja occidentalis*	E
Cyperaceae (Sedges)		
River Bulrush	*Bolboschoenus fluviatilis*	SC
Foxtail Sedge	*Carex alopecoidea*	T
Back's Sedge	*Carex backii*	E
Bailey's Sedge	*Carex baileyi*	E
Bush's Sedge	*Carex bushii*	E
Chestnut-colored Sedge	*Carex castanea*	E
Creeping Sedge	*Carex chordorrhiza*	E
Davis's Sedge	*Carex davisii*	E
Glaucescent Sedge	*Carex glaucodea*	E
Handsome Sedge	*Carex formosa*	T
Slender Woodland Sedge	*Carex gracilescens*	E
Gray's Sedge	*Carex grayi*	T
Hitchcock's Sedge	*Carex hitchcockiana*	SC
Shore Sedge	*Carex lenticularis*	T
Glaucous Sedge	*Carex livida* var. *radicaulis*	E
False Hop-sedge	*Carex lupuliformis*	E
Midland Sedge	*Carex mesochorea*	E
Michaux's Sedge	*Carex michauxiana*	E
Few-fruited Sedge	*Carex oligosperma*	E
Few-flowered Sedge	*Carex pauciflora*	E
Variable Sedge	*Carex polymorpha*	E
Eastern Saline Sedge	*Carex recta*	E
Schweinitz's Sedge	*Carex schweinitzii*	E

Dioecious Sedge	*Carex sterilis*	T		
Walter's Sedge	*Carex striata* var. *brevis*	E		
Fen Sedge	*Carex tetanica*	SC		
Hairy-fruited Sedge	*Carex trichocarpa*	T		
Tuckerman's Sedge	*Carex tuckermanii*	E		
Cat-tail Sedge	*Carex typhina*	T		
Wiegand's Sedge	*Carex wiegandii*	E		
Engelmann's Umbrella-sedge	*Cyperus engelmannii*	T		
Houghton's Flatsedge	*Cyperus houghtonii*	E		
Wright's Spike-rush	*Eleocharis diandra*	E		
Intermediate Spike-sedge	*Eleocharis intermedia*	T		
Tiny-fruited Spike-sedge	*Eleocharis microcarpa*	E		
Ovate Spike-sedge	*Eleocharis obtusa* var. *ovata*	E		
Few-flowered Spike-sedge	*Eleocharis pauciflora* var. *fernaldii*	E		
Three-angled Spike-sedge	*Eleocharis tricostata*	E		
Dwarf Bulrush	*Lipocarpha micrantha*	T		
Slender Cottongrass	*Eriophorum gracile*	T		
Capillary Beak-sedge	*Rhynchospora capillacea*	E		
Inundated Horned-sedge	*Rhynchospora inundata*	T		
Short-beaked Bald-sedge	*Rhynchospora nitens*	T		
Long-beaked Bald-sedge	*Rhynchospora scirpoides*	SC		
Torrey's Beak-sedge	*Rhynchospora torreyana*	E		
Northeastern Bulrush	*Scirpus ancistrochaetus*	E	E	
Long's Bulrush	*Scirpus longii*	T		
Papillose Nut-sedge	*Scleria pauciflora*	E		8
Tall Nut-sedge	*Scleria triglomerata*	E		

Dryopteridaceae (Wood Ferns)

Braun's Holly-fern	*Polystichum braunii*	E
Smooth Woodsia	*Woodsia glabella*	E

Elatinaceae (Waterworts)

American Waterwort	*Elatine americana*	E

Empetraceae (Crowberries)

Broom Crowberry	*Corema conradii*	SC

Equisetaceae (Horsetails)

Dwarf Scouring-rush	*Equisetum scirpoides*	SC

Ericaceae (Laurels, Blueberries)

Great Laurel	*Rhododendron maximum*	T
Mountain Cranberry	*Vaccinium vitis-idaea* ssp. *minus*	E

Eriocaulaceae (Pipeworts)		
Parker's Pipewort	*Eriocaulon parkeri*	E
Fabaceae (Beans, Peas, Clovers)		
Spreading Tick-trefoil	*Desmodium humifusum*	E
Wild Senna	*Senna hebecarpa*	E
Fagaceae (Oaks, Beeches)		
Bur Oak	*Quercus macrocarpa*	SC
Yellow Oak	*Quercus muehlenbergii*	T
Fumariaceae (Fumitories)		
Climbing Fumitory	*Adlumia fungosa*	T
Gentianaceae (Gentians)		
Andrews's Bottle Gentian	*Gentiana andrewsii*	T
Spurred Gentian	*Halenia deflexa*	E
Slender Marsh Pink	*Sabatia campanulata*	E
Plymouth Gentian	*Sabatia kennedyana*	SC
Sea Pink	*Sabatia stellaris*	E
Grossulariaceae (Currants)		
Bristly Black Currant	*Ribes lacustre*	SC
Haemodoraceae (Redroots)		
Redroot	*Lachnanthes caroliana*	SC
Haloragaceae (Water-milfoils)		
Alternate-flowered Water-milfoil	*Myriophyllum alterniflorum*	T
Farwell's Water-milfoil	*Myriophyllum farwellii*	E
Pinnate Water-milfoil	*Myriophyllum pinnatum*	SC
Comb Water-milfoil	*Myriophyllum verticillatum*	E
Hydrophyllaceae (Waterleaves)		
Broad Waterleaf	*Hydrophyllum canadense*	E
Hymenophyllaceae (Filmy-ferns)		
Weft Bristle-fern	*Trichomanes intricatum*	E
Iridaceae (Irises)		
Sandplain Blue-eyed Grass	*Sisyrinchium arenicola*	SC
Slender Blue-eyed Grass	*Sisyrinchium mucronatum*	E

Isoetaceae (Quillworts)		
Acadian Quillwort	*Isoetes acadiensis*	E
Lake Quillwort	*Isoetes lacustris*	E
Juncaceae (Rushes)		
Weak Rush	*Juncus debilis*	E
Thread Rush	*Juncus filiformis*	E
Black-fruited Woodrush	*Luzula parviflora* ssp. *melanocarpa*	E
Lamiaceae (Mints)		
Downy Wood-mint	*Blephilia ciliata*	E
Hairy Wood-mint	*Blephilia hirsuta*	E
Gypsywort	*Lycopus rubellus*	E
Basil Mountain-mint	*Pycnanthemum clinopodioides*	E
False Pennyroyal	*Trichostema brachiatum*	E
Lentibulariaceae (Bladderworts)		
Fibrous Bladderwort	*Utricularia striata*	T
Subulate Bladderwort	*Utricularia subulata*	SC
Liliaceae (Lilies)		
Devil's-bit	*Chamaelirium luteum*	E
Linaceae (Flaxes)		
Sandplain Flax	*Linum intercursum*	SC
Rigid Flax	*Linum medium* var. *texanum*	T
Lycopodiaceae (Clubmosses)		
Mountain Firmoss	*Huperzia selago*	E
Foxtail Clubmoss	*Lycopodiella alopecuroides*	E
Lythraceae (Loosestrifes)		
Toothcup	*Rotala ramosior*	E
Magnoliaceae (Magnolias)		
Sweetbay Magnolia	*Magnolia virginiana*	E
Melastomataceae (Meadow Beauties)		
Maryland Meadow Beauty	*Rhexia mariana*	E
Moraceae (Mulberries)		
Red Mulberry	*Morus rubra*	E

Nymphaeaceae (Water Lilies)			
Tiny Cow-lily	*Nuphar microphylla*	E	
Onagraceae (Evening Primroses)			
Many-fruited False-loosestrife	*Ludwigia polycarpa*	E	
Round-fruited False-loosestrife	*Ludwigia sphaerocarpa*	E	
Ophioglossaceae (Grape Ferns)			
Adder's-tongue Fern	*Ophioglossum pusillum*	T	
Orchidaceae (Orchids)			
Putty-root	*Aplectrum hyemale*	E	
Arethusa	*Arethusa bulbosa*	T	
Autumn Coralroot	*Corallorrhiza odontorhiza*	SC	
Ram's-head Lady's-slipper	*Cypripedium arietinum*	E	
Small Yellow Lady's-slipper	*Cypripedium parviflorum* var. *makasin*	E	
Showy Lady's-slipper	*Cypripedium reginae*	SC	
Dwarf Rattlesnake-plantain	*Goodyera repens*	E	
Small Whorled Pogonia	*Isotria medeoloides*	E	T
Heartleaf Twayblade	*Listera cordata*	E	
Bayard's Green Adder's-mouth	*Malaxis bayardii*	E	
White Adder's-mouth	*Malaxis brachypoda*	E	
Crested Fringed Orchis	*Platanthera cristata*	E	
Leafy White Orchis	*Platanthera dilatata*	T	
Pale Green Orchis	*Platanthera flava* var. *herbiola*	T	
Hooded Ladies'-tresses	*Spiranthes romanzoffiana*	E	
Grass-leaved Ladies'-tresses	*Spiranthes vernalis*	T	
Cranefly Orchid	*Tipularia discolor*	E	
Nodding Pogonia	*Triphora trianthophora*	E	
Oxalidaceae (Wood-sorrels)			
Violet Wood-sorrel	*Oxalis violacea*	E	
Poaceae (Grasses)			
Annual Peanutgrass	*Amphicarpum purshii*	E	
Purple Needlegrass	*Aristida purpurascens*	T	
Seabeach Needlegrass	*Aristida tuberculosa*	T	
Reed Bentgrass	*Calamagrostis pickeringii*	E	
Tufted Hairgrass	*Deschampsia cespitosa* ssp. *glauca*	E	
Commons's Panic-grass	*Dichanthelium commonsianum*	SC	
Mattamuskeet Panic-grass	*Dichanthelium mattamuskeetense*	E	
Rough Panic-grass	*Dichanthelium scabriusculum*	T	
Wright's Panic-grass	*Dichanthelium wrightianum*	SC	

Hairy Wild Rye	*Elymus villosus*	E
Frank's Lovegrass	*Eragrostis frankii*	SC
Saltpond Grass	*Leptochloa fascicularis* var. *maritima*	T
Sea Lyme-grass	*Leymus mollis*	E
Woodland Millet	*Milium effusum*	T
Gattinger's Panic-grass	*Panicum gattingeri*	SC
Philadelphia Panic-grass	*Panicum philadelphicum*	SC
Long-Leaved Panic-grass	*Panicum rigidulum* var. *pubescens*	T
Drooping Speargrass	*Poa languida*	E
Bristly Foxtail	*Setaria geniculata*	SC
Salt Reedgrass	*Spartina cynosuroides*	T
Shining Wedgegrass	*Sphenopholis nitida*	T
Swamp Oats	*Sphenopholis pensylvanica*	T
Small Dropseed	*Sporobolus neglectus*	E
Northern Gama-grass	*Tripsacum dactyloides*	E
Spiked False-oats	*Trisetum triflorum* ssp. *molle*	E
Podostemaceae (Threadfeet)		
Threadfoot	*Podostemum ceratophyllum*	SC
Polygonaceae (Docks, Knotweeds)		
Sea-beach Knotweed	*Polygonum glaucum*	SC
Pondshore Knotweed	*Polygonum puritanorum*	SC
Strigose Knotweed	*Polygonum setaceum* var. *interjectum*	T
Seabeach Dock	*Rumex pallidus*	T
Swamp Dock	*Rumex verticillatus*	T
Portulacaceae (Spring Beauties)		
Narrow-leaved Spring Beauty	*Claytonia virginica*	E
Potamogetonaceae (Pondweeds)		
Fries's Pondweed	*Potamogeton friesii*	T
Hill's Pondweed	*Potamogeton hillii*	SC
Ogden's Pondweed	*Potamogeton ogdenii*	E
Vasey's Pondweed	*Potamogeton vaseyi*	E
Pyrolaceae (Shinleaf)		
Pink Pyrola	*Pyrola asarifolia* var. *purpurea*	E
Ranunculaceae (Buttercups)		
Black Cohosh	*Cimicifuga racemosa*	E
Purple Clematis	*Clematis occidentalis*	SC
Golden Seal	*Hydrastis canadensis*	E
Tiny-flowered Buttercup	*Ranunculus micranthus*	E

Bristly Buttercup	*Ranunculus pensylvanicus*	T	
Rosaceae (Roses, Shadbushes)			
Small-fowered Agrimony	*Agrimonia parviflora*	E	
Hairy Agrimony	*Agrimonia pubescens*	T	
Bartram's Shadbush	*Amelanchier bartramiana*	T	
Nantucket Shadbush	*Amelanchier nantucketensis*	SC	
Roundleaf Shadbush	*Amelanchier sanguinea*	SC	
Bicknell's Hawthorn	*Crataegus bicknellii*	E	
Sandbar Cherry	*Prunus pumila* var. *depressa*	T	
Northern Prickly Rose	*Rosa acicularis*	E	
Northern Mountain-ash	*Sorbus decora*	E	
Barren Strawberry	*Waldsteinia fragarioides*	SC	
Rubiaceae (Bedstraws, Bluets)			
Northern Bedstraw	*Galium boreale*	E	
Labrador Bedstraw	*Galium labradoricum*	T	
Long-leaved Bluet	*Houstonia longifolia*	E	
Salicaceae (Willows)			
Swamp Cottonwood	*Populus heterophylla*	E	
Sandbar Willow	*Salix exigua*	T	
Scheuchzeriaceae (Pod-grasses)			
Pod-grass	*Scheuchzeria palustris*	E	
Schizaeaceae (Climbing Ferns)			
Climbing Fern	*Lygodium palmatum*	SC	
Scrophulariaceae (Figworts)			
Sandplain Gerardia	*Agalinis acuta*	E	E
Winged Monkey-flower	*Mimulus alatus*	E	
Muskflower	*Mimulus moschatus*	E	
Swamp Lousewort	*Pedicularis lanceolata*	E	
Hairy Beardtongue	*Penstemon hirsutus*	E	
Sessile Water-speedwell	*Veronica catenata*	E	
Culver's-root	*Veronicastrum virginicum*	T	
Sparganiaceae (Bur-reeds)			
Small Bur-reed	*Sparganium natans*	E	
Verbenaceae (Vervains)			
Narrow-leaved Vervain	*Verbena simplex*	E	

Violaceae (Violets)		
Sand Violet	*Viola adunca*	E
Britton's Violet	*Viola brittoniana*	T
Northern Bog Violet	*Viola nephrophylla*	E
Viscaceae (Christmas-mistletoes)		
Dwarf Mistletoe	*Arceuthobium pusillum*	SC

1. Trimorphic freshwater population only.

2. Including triploid and other polyploid forms within the *Ambystoma jeffersonianum/Ambystoma laterale* complex.

3. Ditto

4. This species is listed by the U. S. Fish and Wildlife Service as *P. r. bangsi* (Plymouth Redbelly Turtle) in 50 CFR 17.11.

5. Undescribed species near *I. inextricata*

6. Undescribed species near *P. pterisii*

7. Undescribed species near *Z. lunifera*

8. Includes the two varieties of this species that occur in Massachusetts: *s.p.* var. *pauciflora* and *s.p.* var. *caroliniana.*

Last Updated 1/03/2003

APPENDIX VII – POLICY ON LAKE AND POND MANAGEMENT

THE COMMONWEALTH OF MASSACHUSETTS
WATER RESOURCES COMMISSION

Policy on Lake and Pond Management for The Commonwealth of Massachusetts

Effective Date: June 13, 1994

I. DISCUSSION

Massachusetts has approximately 3,000 named lakes and ponds (for simplicity, the word "lake" is used throughout this document to mean an open water body: lake, pond, impoundment or reservoir). Many of the state's lakes are of natural origin, but most have been artificially created or enhanced to achieve one or more specific uses. Lakes are a valuable resource, providing wildlife and fisheries habitat, flood control, water supply, water power, and recreational activities such as fishing, swimming, and boating. Massachusetts places high value on its lakes, recognizing their inherent contribution to the overall quality of life in the Commonwealth, in both environmental and economic terms.

Geologically, lakes are short-lived phenomena that undergo a natural enrichment (aging) process called eutrophication. Generally, the natural life span of most lakes is measured in tens of thousands of years. Over that time period, a lake will gradually fill with sediments and organic materials, with the length of its life depending on its individual characteristics and those of the watershed. In Massachusetts, many lakes are prematurely aging due to stresses caused by human activities.

Watershed activities that disturb soils, increase erosion, or increase stormwater runoff from paved surfaces can lead to increased sediment discharges and accelerate the filling of a lake basin. Nutrient runoff from fertilizers, septic systems, and other nonpoint sources in the watershed can cause undesirable algal blooms and increased growth of aquatic plants. The flow of nutrients and other substances into a lake can degrade overall water quality, altering the ecosystem. Eutrophication may also be accelerated by pre-existing enriched sediments in impoundments. Additionally, ground and surface water withdrawals can affect water quality and accelerate the eutrophication process by concentrating nutrients, increasing water temperature, destabilizing the littoral zone and shoreline habitat, etc.

In addition to eutrophication, another problem plaguing many Massachusetts' lakes has been the introduction (both intentional and unintentional) and proliferation of non-native and/or invasive aquatic plants. Activities such as boating can inadvertently introduce aquatic plants form one lake to another, leading to rapid spread and domination of the ecosystem. Also, non-native/invasive animals such as the grass carp (white amur) and zebra mussel, although not currently found within the state's borders, pose a potential serious and costly threat to the health of all lake ecosystems in the Commonwealth.

Different lake uses may conflict with one another. For example, some recreational uses such as swimming are incompatible with the primary function of a public water supply reservoir. The desire for public assess to a lake may conflict with private land ownership. The use of motorized watercraft may be incompatible with swimming and other passive activities. Management directed at improving active recreational uses can impact fish and wildlife species and their habitats. It is also important to note that not all lakes have the physical characteristics needed to support all desired uses. Because not all lake uses are compatible, lake management programs must be designed according to the ability of a given lake to sustain desired uses.

Premature aging, the spread of non-native/invasive species, and lake-use conflicts are serious problems for many Massachusetts lakes. In many cases, these problems can be minimized through planning, education, and watershed management. Lake management in Massachusetts should combine: (1) inventory and evaluation of the lake/watershed to determine if the lake is attaining its designated uses[1]; (2) assessment of each lake in terms of water quality, biota, and ability to sustain desired uses; (3) development of a comprehensive plan incorporating public participation, public education, and watershed management, and (4) when appropriate, implementation of in-lake and watershed management techniques to address specific problems.

There are a variety of ways to manage a lake, including in-lake management techniques, watershed management, or simply leaving the lake alone. For the reasons discussed above, the absence of active management does not mean that a lake will be "natural", or free from human impact. Active management is not always desirable or feasible. Decisions on lake management must consider the long-term, as well as immediate, costs, benefits and impacts of available management options.

In-lake management techniques are often implemented to enhance particular uses and/or areas of a lake. These techniques include, among other, water level drawdown, sediment removal (dredging), vegetation harvesting, biological treatment, and chemical treatment. These techniques are used to manage eutrophication, restore lake depth, enhance sport fisheries habitat, or increase the amount of pond area available for recreation. In-lake management may also be used to control the spread of non-native and/or invasive plant and animal species. In-lake management can often effectively address one or more specific symptoms, such as poor water quality, algal blooms, oxygen depletion, or excessive plant growth. However, for in-lake management techniques to achieve long-term goals they must be coupled with watershed protection actions.

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[1] "Designated Uses" are those uses specified in 314 CMR 4.05 for each water class, whether or not they are being attained. Uses for which a water body may be designated include Public Water Supply, Recreation, Aquatic Life, Warm Water Fishery, Cold Water Fishery, Marine Fishery, and Shellfishing.

A comprehensive lake management program must identify and address the source of problems, many of which are land-based. Watershed management programs must address problems such as failing and substandard on-site sewage disposal systems, agricultural and residential runoff, stormwater runoff, and erosion. Watershed Management, including careful land-use planning, zoning, erosion control, and other practices, is necessary to prevent lake problems from continually reoccurring. Watershed management must be an integral part of a holistic management program designed to promote lake ecosystem health and the quality of lake uses.

II. POLICY

Recognizing the importance of Massachusetts' lake and pond resources, the rapid deterioration and loss of many of these resources in recent history, and the complexity of issues surrounding their management, the Massachusetts Water Resources Commission establishes the following Policy on Lake and Pond Management[2] [1]:

Massachusetts advocates a holistic approach to lake and pond management and planning which integrates watershed management, in-lake management, pollution prevention and education. Lake management in Massachusetts will be designed with consideration of the quality of the lake's ecosystem, its designated uses and other desired uses, the ability of the ecosystem to sustain those uses, and the long-term costs, benefits and impacts of available management options.

III. GOALS AND OBJECTIVES

The overall goal of this policy is:

To maintain, improve and protect the quality of lake ecosystems in Massachusetts.

Specific goals and objectives designed to achieve the overall goal include:

<u>GOAL #1:</u> To promote a holistic approach to lake management which is based on sound scientific principles and emphasizes the integrated use of watershed management, in-lake management, pollution prevention and education.

<u>Objectives:</u>

[2] This policy recognizes all existing state and federal statutes and regulations and does not diminish the legal authorities of any state, federal, and municipal governmental bodies relative to management and protection of lakes, watersheds, fisheries and wildlife.

[1] This policy and all related Goals and Objectives shall require a review and re-approval by the Water Resources Commission at intervals of no greater than five (5) years.

1. To promote the development of comprehensive, integrated individual lake and watershed management plans in accordance with the policy.

2. To achieve and/or maintain designated lake uses and balance other competing uses with regard to the ecosystem's ability to sustain such uses.

GOAL #2: To promote sound planning and management of lakes and their surrounding watersheds by providing guidance to municipal agencies, local organizations, and the public.

Objectives:

1. To provide municipal agencies, local organizations, and the public with a comprehensive and consistent policy on the management of Massachusetts' lake as well as the guidance, reference materials, and technical support necessary to implement the policy.

2. To develop an education program that provide municipal agencies, local organizations, and the public with the knowledge and guidance necessary to effectively plan and manage lakes and their surrounding watersheds.

GOAL #3: To streamline the permitting process for in-lake management projects.

Objectives:

1. To promote lake management as a joint public and private responsibility, to be pursued in partnership with lake stakeholders.

2. To establish criteria and procedures to review and approve lake and watershed management plans

3. To allow lake management projects to be classified as "Limited Projects" under the Wetlands Protection Act, upon completion and approval of a comprehensive lake management plan.

4. To review the current permitting process to identify opportunities for greater efficiency, consistency, and timeliness, and implement the necessary changes.

5. To have state agencies proactively implement this policy in their planning, regulations, and programs.

6. To adopt a standard set of lake definitions for the Commonwealth of Massachusetts.

GOAL #4: To promote the importance of lake ecosystems and all associated wetland natural and biological resources, including open water.

Objectives:

1. To recognize the importance of open water bodies for their unique ecological, recreational and aesthetic role as well as the tourism opportunities they provide in the Commonwealth.

2. To protect and promote the health of species that are state and/or federally listed as endangered, threatened, or species of special concern.
3. To develop guidance on protecting lake ecosystems from the introduction and proliferation of non-native and/or invasive species that will be harmful to existing flora and/or fauna.

GOAL #5: To assure that decisions on the use of lake and watershed management techniques to remediate the impacts of eutrophication and non-native/invasive species consider the long term, as well as immediate, costs, benefits and impacts of available management options.

Objectives:

1. To develop a standard methodology for assessing, on a case by case basis, the costs and potential effects of lake management techniques with remediate the impacts of eutrophication and non-native/invasive species.

2. To develop and utilize the findings of a Generic Environmental Impact Report (GEIR) for in-lake and watershed management projects.

3. To allow the use of short-term in-lake management techniques for one year, consistent with the GEIR and federal, state and municipal environmental and public health regulations. For short-term in-lake management techniques to continue in subsequent years, long-term watershed based management plans must be developed and implemented.

4. To require completion of an appropriate lake/watershed management plan as a condition for the use of state funds for lake management projects.

5. To assure that public funds are used for lake projects in waters that are open to the public.

Note to reader: The original distribution of the Policy was prefaced by a letter from DEM Commissioner Peter C. Webber, Chairman of the Commission. In the letter, Commissioner Webber noted that the WRC had formed the Lakes Management Policy Committee and charged it to develop this first statewide, comprehensive policy on the management of Massachusetts' lakes and ponds. The Committee represented a broad spectrum of lake stakeholders, including state and federal agencies, non-profit environmental groups,

municipalities, the Massachusetts Congress of Lakes and Ponds Associations (COLAP), and lake management consultants. After a year-long process of policy drafting and reviews, the Commission unanimously approved the Policy on June 13, 1994.

For additional information about this policy, please contact:

Lakes and Ponds Program
Department of Conservation and Recreation
251 Causeway Street, Suite 700
Boston MA 02114-2104

or *www.state.ma.us/dcr* **or** *www.state.ma.us/envir*

APPENDIX VIII -- GLOSSARY

Abiotic - Pertaining to any non-biological factor or influence, such as geological or meteorological characteristics.

Acid precipitation - Atmospheric deposition (rain, snow, dryfall) of free or combined acidic ions, especially the nitrates, sulfates and oxides of nitrogen and sulfur fumes from industrial smoke stacks.

Adsorption - External attachment to particles, the process by which a molecule becomes attached to the surface of particle.

Algae - Aquatic single-celled, colonial, or multi-celled plants, containing chlorophyll and lacking roots, stems, and leaves.

Alkalinity - A reference to the carbonate and bicarbonate concentration in water. Its relative concentration is indicative of the nature of the rocks within a drainage basin. Lakes in sedimentary carbonate rocks are high in dissolved carbonates (hard-water lakes) whereas lakes in granite or igneous rocks are low in dissolved carbonate (soft-water lakes).

Ammonium - A form of nitrogen present in sewage and is also generated from the decomposition of organic nitrogen. It can also be formed when nitrites and nitrates are reduced. Ammonium is particularly important since it has high oxygen and chemical demands, is toxic to fish in un-ionized form and is an important aquatic plant nutrient because it is readily available.

Anadromous - An adjective used to describe types of fish which breed in freshwater rivers but spend most of their adult lives in the ocean. Before breeding, anadromous adult fish ascend the rivers from the sea.

Anoxic - Without oxygen.

Aphotic Zone - Dark zone, below the depth to which light penetrates. Generally equated with the zone in which most photosynthetic algae cannot survive, due to light deficiency.

Aquifer - Any geological formation that contains water, especially one that supplies wells and springs; can be a sand and gravel aquifer or a bedrock aquifer.

Artesian - The occurrence of groundwater under sufficient pressure to rise above the upper surface of the aquifer.

Assimilative Capacity - Ability to incorporate inputs into the system. With lakes, the ability to absorb nutrients or other potential pollutants without showing extremely averse effects.

Attenuation - The process whereby the magnitude of an event is reduced, as the reduction and spreading out of the impact of storm effects or the removal of certain contaminations as water moves through soil.

Background Value - Value for a parameter that represents the conditions in a system prior to a given influence in space or time.

Bathymetry - The measurement of depths of water in oceans, seas, or lakes or the information derived from such measurements.

Benthic Deposits - Bottom accumulations which may contain bottom-dwelling organisms and/or contaminants in a lake, harbor, or stream bed.

Benthos - Bottom-dwelling organisms living on, within or attached to the sediment. The phytobenthos includes the aquatic macrophytes and bottom-dwelling algae. The zoobenthos (benthic fauna) includes a variety of invertebrate animals, particularly larval forms and molluses.

Best Management Practices - (BMP's) State-of-the-art techniques and procedures used in an operation such as farming or waste disposal in order to minimize pollution or waste.

Biological Oxygen Demand - The BOD is an indirect measure of the organic content of water. Water high in organic content will consume more oxygen due to the decomposition activity of bacteria in the water than water low in organic content. It is routinely measured for wastewater effluents. Oxygen consumption is proportional to the organic matter in the sample.

Biota - Plant (flora) and animal (fauna) life.

Biotic - Pertaining to biological factors or influences, concerning biological activity.

Bloom - Excessively large standing crop of algae, usually visible to the naked eye.

Bulk Sediment Analysis - Analysis of soil material or surface deposits to determine the size and relative amounts of particles composing the material.

CFS - Cubic feet per second, a measure of flow.

Chlorophyll - Major light gathering pigment of all photosynthetic organisms imparting the characteristic color or green plants. Its relative measurement in natural waters is indicative of the concentration of algae in the water.

Chlorophyte - Green algae, algae of the division Chlorophyta.

Chrysophyte - Golden or yellow-green algae, algae of the division Chrysophyta.

Coliforms - Generally refers to bacterial species normally present in the large intestine (colon) and feces of all warm-blooded animals.

Color - Color is determined by visual comparison of a sample with known concentrations of colored solutions and is expressed in standard units of color. Certain waste discharges may turn water to colors which cannot be defined by this method; in such cases, the color is expressed qualitatively rather than numerically. Color in lake waters is related to solids, including algal cell concentration and dissolved substances.

Combined Sewer - A sewer intended to serve as both a santiary sewer and a storm sewer. It receives both sewage and surface runoff.

Composite Sample - A number of individual samples collected over time or space and composited into one representative sample.

Concentration - The quantity of a given constituent in a unit of volume or weight of water.

Conductivity - The measure of the total ionic concentration of water. Water with high total dissolved solids (TDS) level would have a high conductance. A conductivity meter tests the flow of electrons through the water which is heightened in the presence of electrolytes (TDS).

Confluence - Meeting point of two rivers or streams.

Conservative Substance - Non-interacting substance, undergoing no kinetic reaction; chlorides and sodium are approximate examples.

Cosmetic - Acting upon symptoms or given conditions without correcting the actual cause of the symptoms or conditions.

Cryptophyte - Algae of variable pigment concentrations, with various other unusual features. Algae of the division Cryptophyta, which if often placed under other taxonomic divisions.

Cyanophyte - Bluegreen algae, algae of the division Cyanophyta, actually a set of pigmented bacteria.

Decomposition - The metabolic breakdown of organic matter, releasing energy and simple organic and inorganic compounds which may be utilized by the decomposers themselves (the bacteria and fungi).

Deoxygenation - Depletion of oxygen in an area, used often to describe possible hypolimnetic conditions, process leading to anoxia.

Diatom - Specific type of chrysophyte, having a siliceous frustule (shell) and often elaborate ornamentation, commonly found in great variety in fresh or saltwaters. Often placed in its own division, the Bacillariophyta.

Dinoflagellate - Unicellular algae, usually motile, having pigments similar to diatoms and certain unique features. More commonly found in saltwater. Algae of the division Pyrrhophyta.

Discharge Measurement - The volume of water which passes a given location in a given time period, usually measured in cubic feet per second (cfs) or cubic meters per minute (m3/min).

Dissolved Oxygen (D.O.) - Refers to the uncombined oxygen in water which is available to aquatic life. Temperature affects the amount of oxygen which water can contain. Biological activity also controls the oxygen level. D.O. levels are generally highest during the afternoon and lowest just before sunrise.

Diurnal - Varying over the day, from day time to night.

Domestic Wastewater - Water and dissolved or particulate substances after use in any of a variety of household tasks, including sanitary systems and washing operations.

Drainage Basin - A geographical area or region which is so sloped and contoured that surface runoff from streams and other natural watercourses is carried away by a single drainage system by gravity to a common outlet. Also referred to as a watershed or drainage area. The definition can also be applied ot subsurface flow in groundwater.

Dystrophic - Trophic state of a lake in which large quantities of nutrients may be present, but are generally unavailable (due to organic binding or other causes) for primary production. Often associated with acid bogs.

Ecosystem - A dynamic association or interaction between communities of living organisms and their physical environment. Boundaries are arbitrary and must be stated or implied.

Elutriate - Elutriate refers to the washings of a sample of material.

Epilimnion - Upper layer of stratified lake. Layer that is mixed by wind and has a higher average temperature than the hypolimnion. Roughly approximates the euphotic zone.

Erosion - The removal of soil from the land surface, typically by runoff water.

Euglenoid - Algae similar to green algae in pigment composition, but with certain unique features related to food storage and cell wall structure. Algae of the division Euglenophyta.

Eutrophic - High nutrient, high productivity trophic state generally associated with unbalanced ecological conditions and poor water quality.

Eutrophication - Process by which a body of water ages, most often passing from a low nutrient concentration, low productivity state to a high nutrient concentration, high productivity stage. Eutrophication is a long-term natural process, but it can be greatly accelerated by man's activities. Eutrophication as a result of man's activities is termed cultural eutrophication.

Fauna - A general term referring to all animals.

Fecal Coliform Bacteria - Bacteria of the coli group that are present in the intestines or feces of warm-blooded animals. They are often used as indicators of the sanitary quality of the water. In the laboratory they are defined as all organisms which produce blue colonies within 24 hours when incubated at $44.5^{o}C \pm 0.2^{o}C$ on M-FC medium (nutrient medium for bacterial growth). Their concentrations are expressed as number of colonies per 100 ml of sample.

Fecal Streptococci Bacteria - Bacteria of the Streptococci group found in intestines of warm-blooded animals. Their presence in water is considered to verify fecal pollution. They are characterized as gram positive, cocciod bacteria which are capable of growth in brain-heart infusion broth. In the laboratory they are defined as all the organisms which produce red or pink colonies within 48 hours at $35^{o}C \pm 1.0^{o}C$ on KF medium (nutrient medium for bacterial growth). Their concentrations are expressed as number of colonies per 100 ml of sample.

Flora - general term referring to all plants.

Food Chain - A linear characterization of energy and chemical flow through organisms such that the biota can be separated into functional units with nutritional interdependence. Can be expanded to a more detailed characterization with multiple linkage, called a food web.

Grain Size Analysis - A soil or sediment sorting procedure which divides the particles into groups depending on size so that their relative amounts may be determined. Data from grain size analyses are useful in determining the origin of sediments and their behavior in suspension.

Groundwater - Water in the soil or underlying strata, subsurface water.

Hardness - A physical-chemical characteristic of water that is commonly recognized by the increased quantity of soap required to produce lather. It is attributable to the presence of alkaline earths (principally calcium and magnesium) and is expressed as equivalent calcium carbonate ($CaCO_3$).

Humus - Humic substances form much of the organic matter of sediments and water. They consist of amorphous brown or black colored organic complexes.

Hydraulic Detention Time - Lake water retention time, amount of time that a random water molecule spends in a water body; time that it takes for water to pass from an inlet to an outlet of a water body.

Hydraulic Dredging - Process of sediment removal using a floating dredge to draw mud or saturated sand through a pipe to be deposited elsewhere.

Hydrologic Cycle - The circuit of water movement from the atmosphere to the earth and return to the atmosphere through various stages or processes such as precipitation, interception, runoff, infiltration, percolation, storage, evaporation, and transpiration.

Hypolimnion - Lower layer of a stratified lake. Layer that is mainly without light, generally equated with the aphotic zone, and has a lower average temperature than the epilimnion.

Impervious - Not permitting penetration or percolation of water.

Intermittent - Non-continuous, generally referring to the occasional flow through a set drainage path. Flow of a discontinuous nature.

Kjeldahl Nitrogen - The total amount of organic nitrogen and ammonia in a sample, as determined by the Kjeldahl method, which involves digesting the sample with sulfuric acid, transforming the nitrogen into ammonia, and measuring it.

Leachate - Water and dissolved or particulate substances moving out of a specified area, usually a landfill, by a completely or partially subsurface route.

Leaching - Process whereby nutrients and other substances are removed from matter (usually soil or vegetation) by water. Most often this is a chemical replacement action, prompted by the qualities' of the water.

Lentic - Standing, having low net directional motion. Refers to lakes and impoundments.

Limiting Nutrient - That nutrient of which there is the least quantity, in relation to its importance to plants. The limiting nutrient will be the first essential compound to disappear from a productivity system, and will cause cessation of productivity at that time. The chemcial form in which the nutrient occurs and the nutritional requirements of the plants involved are important here.

Limnology - The comprehensive study of lakes, encompassing physical, chemcal and biological lake conditons.

Littoral Zone - Shallow zone occurring at the edge of aquatic ecosystems, extending from the shoreline outward to a point where rooted aquatic plants are no longer found.

Loading - Inputs into a receiving water that may exert a detrimental effect on some subsequent use of that water.

Lotic - Flowing, moving. Refers to streams or rivers.

Macrofauna - A general term which refers to animals which can be seen with the naked eye.

Macrophyte - Higher plant, macroscopic plant, plant of higher taxonomic position than algae, usually a vascular plant. Aquatic macrophytes are those macrophytes that live completely or partially in water. May also include algal mats under some definitions.

Mesotrophic - An intermediate trophic state, with variable but moderate nutrient concentrations and productivity.

MGD - Million gallons per day, a measure of flow.

Micrograms per Liter (ug/1) - A unit expressing the concentration of chemical constituents in solution as mass (micrograms) of solute per unit volume (liter) of water. One thousand micrograms per liter is equivalent to one milligram per liter.

Nitrate - A form of nitrogen that is important since it is the end product in the aerobic decomposition of nitrogenous matter. Nitrogen in this form is stable and readily available to plants.

Nitrite - A form of nitrogen that is the oxidation product of ammonia. It has a fairly low oxygen demand and is rapidly converted to nitrate. The presence of nitrite nitrogen usually indicates that active decomposition is taking place (i.e., fresh contamination).

Nitrogen - A macronutrient which occurs in the forms of organic nitrogen, ammonia nitrogen, nitrite nitrogen and nitrate nitrogen. Form of nitrogen is related to a successive decomposition reaction, each dependent on the preceding one, and the progress and decompositon can be determined in terms of the relative amounts of these four forms of nitrogen.

Nitrogen-fixation - The process by which certain bacteria and bluegreen algae make organic nitrogen compounds (initially $NH4^{+}$) from elemental nitrogen (N_2) taken from the atmosphere or dissolved in the water.

Non-point Source - A diffuse source of loading, possibly localized but not distinctly definable in terms of location. Includes runoff from all land types.

Nutrients - Are compounds which act as fertilizers for aquatic organisms. Small amounts are necessary to the ecological balance of a waterbody, but excessive amounts can upset the balance by causing excessive growths of algae and other aquatic plants. Sewage discharged to a waterbody usually contains large amounts of carbon, nitrogen, and phosphorus. The concentration of carbonaceous matter is reflected in the B.O.D. test. Additional tests are run to determine the concentrations of nitrogen and phosphorus. Storm water runoff often contributes substantial nutrient loadings to receiving waters.

Oligotrophic - Low nutrient concentration, low productivity trophic state, often associated with very good water quality, but not necessarily the most desirable stage, since often only minimal aquatic life can be supported.

Organic - Containing a substantial percentage of carbon derived from previously living organisms; of a living organism.

Overturn - The vertical mixing of layers of water in the spring and fall caused by seasonal changes in temperature in temperature climate zones.

Oxygen Deficit - A situation in lakes where respiratory demands for oxygen become greater than its production via photosynthesis or its input fromthe drainage basins, leading to a decline in oxygen content.

Periphyton - Attached forms of plants and animals, growing on a substrate.

pH - A hydrogen concentration scale from 0 (acidic) to 14 (basic) used to characterize water solutions. Pure water is neutral at pH 7.0.

Phosphorus - A macronutrient which appears in waterbodies in combined forms known as ortho- and poly-phosphates and organic phosphorus. Phosphorus may enter a waterbody in agricultural runoff where fertilizers are used. Storm water runoff from highly urbanized areas, septic system leachate, and lake bottom sediments also contribute phosphorus. A critical plant nutrient which is often targeted for control in eutrophication prevention plans.

Photic Zone - Illuminated zone, surface to depth beyond which light no longer penetrates. Generally equated with the zone in which photosynthetic algae can survive and grow, due to adequate light supply.

Photosynthesis - Process by which primary producers make organic molecules (generally glucose) from inorganic ingredients, using light as an energy source. Oxygen is evolved by the process as a byproduct.

Phytoplankton - Algae suspended, floating or moving only slightly under their own power in the water column. Often the dominant algae form in standing waters.

Plankton - The community of suspended, floating, or weakly swimming organisms that live in the open water of lakes and rivers.

Point Source - A specific source of loading, accurately definable in terms of location. Includes effluents or channeled discharges that enter natural waters at a specific point.

Pollution - Undiserable alteration of the physical, chemical or biological properties of water, addition of any substance into water by human activity that adversely affects its quality. Prevalent examples are thermal, heavy metal and nutrient pollution.

Potable - Usuable for drinking purposes, fit for human consumption.

Primary Productivity (Production) - Conversion of inorganic matter to organic matter to organic matter by photosynthesizing organisms. The creation of biomass by plants.

Riffle Zone - Stretch of a stream or river along which morphological and flow conditions are such that rough motion of the water surface results. Usually a shallow rocky area with rapid flow and little sediment accumulation.

Riparian - Of, or related to, or bordering a watercourse.

Runoff - Water and its various dissolved substances or particulates that flows at or near the surface of land in an unchanneled path toward channeled and usually recognized waterways (such as a stream or river).

Secchi Disk Transparency - An approximate evaluation of the transparency of water to light. It is the point at which a black and white disk lowered into the water is no longer visible.

Secondary Productivity - The growth and reproduction (creation of biomass) by herbivorous (plant-eating) organisms. The second level of the trophic system.

Sedimentation - The process of settling and deposition of suspended matter carried by water, sewage, or other liquids, by gravity. It is usually accomplished by reducing the velocity of the liquid below the point at which it can transport the suspended material.

Sewage (Wastewater) - The water borne, human and animal wastes from residences, industrial/commercial establishments or other places, together with such ground or surface water as may be present.

Specific Conductance - Yields a measure of a water sample's capacity to convey an electric current. It is dependent on temperature and the concentration of ionized substances in the water. Distilled water exhibits specific conductance of 0.5 to 2.0 micromhos per centimeter, while natural waters show values from 50 to 500 micromhos per centimeter. In

typical New Engalnd lakes, Specific Conductance usually ranges from 100-300 micromhos per cm. The specific conductance yields a generalized measure of the inorganic dissolved load of the water.

Stagnant - Motionless, having minimal circulation or flow.

Standing Crop - Current quantity of organisms, biomass on hand. The amount of live organic matter in a given area at any point in time.

Storm Sewer - A pipe or ditch which carries storm water and surface water, street wash and other wash waters or drainage, but excludes sewage and industrial wastes.

Stratification - Process whereby a lake becomes separated into two relatively distinct layers as the result of temperature and density differences. Further differentiation of the layers usually occurs as the result of chemical and biological processes. In most lakes, seasonal changes in temperature will reverse this process after some time, resulting in the mixing of the two layers.

Substrate - The base of material on which an organism lives, such as cobble, gravel, sand, muck, etc.

Succession - The natural process by which land and vegetation patterns change, proceeding in a direction determined by the forces acting on the system.

Surface Water - Refers to lakes, bays, sounds, ponds, reservoirs, springs, rivers, streams, creeks, estuaries, marshes, inlets, canals, oceans and all other natural or artificial, inland or coastal, fresh or salt, public or private waters at ground level.

Suspended Solids - Those which can be removed by passing the water through a filter. The remaining solids are called dissolved solids. Suspended solids loadings are generally high in stream systems which are actively eroding a watershed. Excessive storm water runoff often results in high suspended solids loads to lakes. Many other pollutants such as phosphorus are often associated with suspended solids loadings.

Taxon (Taxa) - Any hierarchical division of a recognized classification system, such as a genus or species.

Taxonomy - The division of biology concerned with the classification and naming of organisms. The classification of organisms is based upon a hierarchical scheme beginning with Kingdom and progressing to the Species level or even lower.

Tertiary Productivity - The growth and reproduction (creation of biomass) by organisms that eat herbivorous (plant-eating) organisms. The third level of the trophic system.

Thermocline - Boundary level between the epilimnion and hypolimnion of a stratified lake, variable in thickness, and generally approximating the maximum depth of light penetration and mixing by wind.

Total Coliform Bacteria - A particular group of bacteria that is used as an indicator of possible sewage pollution. They are characterized as aerobic or faculative anaerobic, gramnegative, nonspore-forming, rod-shaped bacteria which ferment lactose with gas formation within 48 hours at $35^{o}C$. In the laboratory these bacteria are defined as the organisms which produce colonies within 24 hours when incubated at $35^{o}C \pm 1.0^{o}C$ on M-Endo medium (nutrient medium for bacterial growth). Their concentrations are expressed as number of colonies per 100 ml of sample.

Trophic Level - The position in the food chain determined by the number of energy transfer steps to that level; 1 = producer; 2 = herbivore; 3, 4, 5 = carnivore.

Trophic State - The stage or condition of an aquatic system, characterized by biological, chemical and physical parameters.

Turbidity - The measure of the clarity of a water sample. It is expressed in Nephelometric Turbidity Units which are related to the scattering and absorption of light by the water sample.

Volatile Solids - That portion of a sample which can be burned off, consisting of organic matter, including oils and grease.

Water Quality - A term used to describe the chemical, physical, and biological characteristics of water, usually with respect to its suitability for a particular purpose or use.

Watershed - Drainage basin, the area from which an aquatic system receives water.

Zooplankton - Microscopic animals suspended in the water; protozoa, rotifers, cladocera, copepods and other small invertebrates.