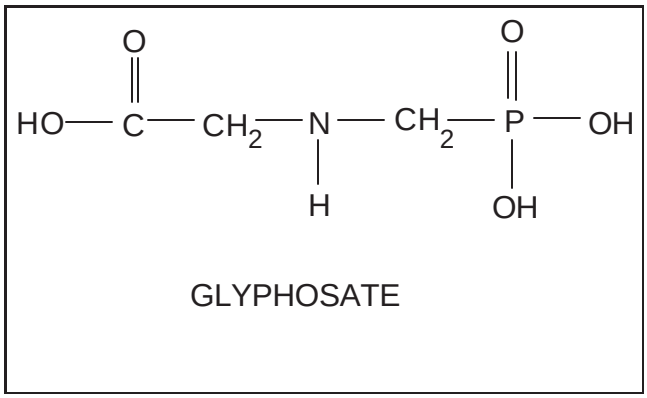
III.4 GLYPHOSATE



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.state.ma.us/dfa/pesticides/water/Aquatic/Herbicides.htm 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	*Quercus 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)

Glyphosate References

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