Hexazinone controls some grasses, many annual and perennial broadleaf herbs, and some woody species, by inhibiting photosynthesis. It is water-soluble and does not bind strongly with soils, and so is of particular concern for groundwater contamination. Hexazinone can enter aquatic systems through surface and subsurface runoff following application and drift during application. It is degraded by microbial metabolism, but not readily decomposed chemically or by sunlight, and can therefore persist in aquatic systems. The average half-life of hexazinone in soils is 90 days, but it can sometimes be found in runoff up to six months after application. Although it is of relatively low toxicity to birds and mammals, legal application rates can leave residues that exceed EPA’s Level of Concern for aquatic and terrestrial plants and small mammals. It is of relatively low toxicity to fish and aquatic invertebrates but can be highly toxic to some species of algae. Hexazinone contamination has been detected in small water-bodies in episodic, low-level pulses that were rapidly diluted in mainstream flows. High concentrations of hexazinone, however, could lead to significant losses of algae and macrophytic biomass, which could produce a ripple effect in the food chain that ultimately could impact fish and wildlife species. Although hexazinone can accumulate in treated crops, concentrations in vegetation are not likely to reach toxic levels for foraging animals when hexazinone is applied properly. Care should be taken in preparing and applying hexazinone as it can cause severe eye damage.
**Herbicide Details**

**Chemical Formula:** 3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4 (1H,3H)-dione

**Trade Names:** Pronone® and Velpar®

**Manufacturers:** Du Pont and Pro-Serve

**Use Against Natural Area Weeds:** Hexazinone is a broad-spectrum herbicide that can control annual and perennial herbaceous broadleaf weeds, some grasses, and some woody species. It is often used to control brush in reforested areas, in tree plantations, and in rangeland and pasturelands.

Hexazinone is absorbed through the roots and foliage of plants, and best results are obtained for herbaceous species when applied in moist soil conditions, as either a foliage spray or basal soil treatment. Larger woody species are best controlled by injection or hack-and-squirt techniques. Species that have been controlled by hexazinone include: tansy-mustard (*Descurainia pinnata*), cheatgrass (*Bromus tectorum*), filaree (*Erodium* spp.), shepards-purse (*Capsella bursa-pastoris*), false dandelion (*Hypochaeris radicata*), privet (*Ligustrum* spp.), and Chinese tallowtree (*Sapium sebiferum*) (Du Pont 1993).

Hexazinone is water-soluble and does not bind strongly with soils, and so is of particular concern for groundwater contamination. It can persist in soils and aquatic systems for some time (average half-life in soil is 90 days), increasing the likelihood of contamination. No use of hexazinone was reported by TNC preserves in the 1998-99 TNC Weed Survey.

**Mode of Action:** Hexazinone is a systemic herbicide that inhibits photosynthesis in susceptible plants, diverting highly reactive molecules into a chain reaction that destroys chloroplast and cell membranes, and other vital compounds. It is usually applied as a pre-emergent herbicide, and soils must be moist (by rain or irrigation) to activate hexazinone. Hexazinone works by binding to a protein component of the photosystem II complex, which blocks electron transport. The result is a chain reaction in which triplet-state chlorophyll reacts with oxygen (O₂) to form singlet oxygen (O), and both the chlorophyll and singlet oxygen strip hydrogen ions (H⁺) from unsaturated lipids in cell and organelle membranes, producing lipid radicals. The lipid radicals in turn attack and oxidize other lipids and proteins, resulting in the loss of cell and organelle membrane integrity, loss of chlorophyll and carotenoids, leakage of cellular contents, cell death, and ultimately death of the plant (WSSA 1994).

**Dissipation Mechanisms:**

*Summary:* Hexazinone is primarily degraded through microbial metabolism in soils and sediments. It is not significantly affected by photo or chemical degradation. It is not readily adsorbed by sediments and can remain mobile in the environment until metabolized by microbes. Hexazinone is not highly volatile (T. Lanini, pers. obs.).
Hexazinone does not volatilize readily when applied in the field (T. Lanini, pers. obs). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

**Photodegradation**
Hexazinone resists photodegradation (Neary et al. 1983). When exposed to artificial sunlight in distilled water, hexazinone degrades slowly (approx. 10% in five weeks) (Rhodes 1980b). Photodegradation can be three to seven times greater, however, in natural river water and/or in water containing a photoinitiator (a compound that catalyzes photodegradation) (Rhodes 1980b). Water pH and temperature do not affect hexazinone photodegradation rates significantly.

**Microbial Degradation**
Hexazinone is degraded primarily by microorganisms in soils (Rhodes 1980a, Jensen & Kimball 1987). Rhodes (1980a) found that no herbicidal degradation or loss occurred in soils kept in anaerobic conditions for 60 days. Conversely, in aerobic soils, 45-75% of the applied hexazinone was released as CO₂ within 80 days of application, likely as a result of microbial degradation (Rhodes 1980a).

**Adsorption**
Hexazinone has a comparatively low adsorption capacity. Adsorption of hexazinone to soil particles increases with increasing soil pH, organic content, and clay cation exchange capacity (Neary et al. 1983; Koskinen et al. 1996). Soil temperature does not alter the adsorption capacity significantly (Koskinen et al. 1996).

**Chemical Decomposition**
Hexazinone has been shown to degrade to eight or more different metabolites, identified as metabolites ‘A’ through ‘H’ (Rhodes 1980b). Only metabolite ‘B’ is believed to be toxic to plants, and even so, it has only 1% of the toxicity of hexazinone. The ratio metabolites formed during degradation varies with environmental conditions (i.e. climate and soil conditions control the predominance of a particular metabolites in soils) (Roy et al. 1989).

Jensen and Kimball (1987) found that under warm, moist conditions, hexazinone breaks down by chemical means to metabolite ‘D’. In general, however, hexazinone has been found to be stable in aqueous solutions without the presence of sunlight or microbes (Rhodes 1980b), suggesting that independent of a catalyst, hexazinone does not readily degrade.

**Behavior in the Environment**
*Summary:* Hexazinone does not bind strongly with soils and can be highly mobile in the environment. It is degraded primarily through microbial metabolism with an average half-life of 90 days in soils and water. Its relative persistence and mobility make it a potential threat to off-site movement and contamination of non-target plants. As a result of its relative persistence and high mobility, it has a high potential to move off-site and contaminate water or kill desirable plants. Hexazinone residues can persist in leaf litter, releasing hexazinone into the environment long after application. Hexazinone metabolites are also persistent and mobile.

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**Soils**

Hexazinone is relatively persistent in soils. Reported half-lives vary between one and six months with a mean of 90 days. Half-lives reported by Rhodes (1980) were one month in Delaware Keyport silt loam, two months in Illinois Flanagan silt loam, and six months in Mississippi Dundee silt loam. Neary et al. (1983) reported a half-life in mineral soils of NE Georgia of 10-30 days. In both clay and sand soils of a boreal forest in Ontario, Canada, the half-life of hexazinone was 43 days (Roy et al. 1989). Prasad and Feng (1990) found 1% of applied hexazinone and 0.4% of its metabolites remained in soils after one year.

Hexazinone does not bind strongly with soil particles and, theoretically, could be highly mobile (Rhodes 1980). Observations of vertical movement in soils, however, have been conflicting. Lavy et al. (1989) hypothesized that because adsorption increases with increasing organic content, adsorption should be highest in the soil’s surface layers and decrease with soil depth. In support of this hypothesis, Roy et al. (1989) found that in sand and clay soils of boreal forests in Ontario, Canada, 98% of the hexazinone remained in the top 15 cm of soil, and leaching appeared to be retarded by mineral layers. Conversely, Zandvoort (1989) concluded that the slow degradation of hexazinone in soils could result in contamination of deep soil layers. In support of this conclusion, Feng et al. (1992) reported hexazinone and its metabolite residues leached to 130 cm and were still detectable two years after application in northern Alberta. Degradation is usually slowest in cold, dry climates, like that of northern Alberta.

The granular formulation of hexazinone has greater lateral mobility than the liquid formulation (Prasad & Feng 1990). Although the granular formulation initially provides spotty coverage, horizontal movement over time redistributes granular formulations to give coverage comparable to liquid formulations (Feng et al. 1992).

Because of the hexazinone’s mobility in soils, it has the potential to move off-site and affect non-target species up to 100 meters away (Allender 1991). Sidhu and Feng (1993) reported that granular formulations applied during fall, contaminated nearby marsh reed grass in surface runoff during spring melt the following year. These residues persisted in the marsh reed grass up to two years later.

**Water**

Hexazinone is water-soluble and does not bind strongly with soils (EXTOXNET 1996). It can be highly mobile in surface and sub-surface water runoff and has the potential to contaminate groundwater and surface water bodies (Schneider et al. 1995; Peterson et al. 1997). Hexazinone has been found in groundwater in four states. The reported half-life of hexazinone in water varies between several days (Solomon et al. 1988) to more than nine months (Thompson et al. 1992). Thompson et al. (1992) reported that in treated enclosures on a boreal lake in Ontario, hexazinone concentrations of 1-10 mg/L persisted for 35-49 days. Neary et al. (1986) concluded that use of hexazinone would result in smaller water quality changes than do commonly used, intensive mechanical weed control techniques, particularly on steep forest slopes with fragile soils.
Hexazinone

Mayack et al. (1982) and Neary et al. (1983; 1984) report that subsurface runoff of small amounts of hexazinone are episodic and unpredictable, and are diluted in the mainstream flow to very low concentrations. In a study funded by DuPont, Neary et al. (1986) found low concentrations of hexazinone in storm water runoff for seven months following application to the upper Piedmont in North Georgia, with a loss of 0.53% of the total volume applied. Lavy et al. (1989) evaluated the fate of hexazinone in a steep watershed in north-central West Virginia and found that 4.7% of the total hexazinone applied to the watershed leached into the local streams, but none of the compound was found in stream sediments.

Vegetation
Hexazinone is absorbed primarily through the root system, but also can be taken up from the foliage. In non-susceptible species, the herbicide is broken down into non-toxic or less toxic forms (e.g. metabolite ‘A’), while in target species, the parent compound, hexazinone, and its phytotoxic metabolite ‘B’ persist and can inhibit photosynthesis and cause chloroplast damage (Sidhu & Feng 1993). Some studies have found that sublethal concentrations of hexazinone (<10^{-6} M) can enhance photosynthesis rates in some species, but the mechanism for this is unknown (Sung et al. 1985; Johnson & Stelzer 1991).

A number of authors hypothesize that the leaves and other litter dropped from treated vegetation may serve as a reservoir for hexazinone, and can release it into the environment up to three years following application (Mayack et al. 1982; Neary et al. 1984; Lavy et al. 1989). This reservoir, however, may also function to prevent excessive runoff immediately following application in steep watersheds (Lavy et al. 1989).

Environmental Toxicity
Birds and Mammals
Hexazinone is of relatively low toxicity to birds and mammals. The oral LD50 for rats is 1,690 mg/kg and 2,258 mg/kg for bobwhite quail (WSSA 1994). Higher application rates can produce residues that exceed EPA’s Level of Concern for small mammals. In a study of rat metabolism of hexazinone, at least 93.3% of the radio-labelled hexazinone was excreted by the animals and none of the compound was detected in the rats after 72 hours (Rhodes & Jewell 1980).

Treated plants can sequester herbicides in their foliate, which could be ingested by foraging wildlife. Sidhu and Feng (1993) found that at application rates of 4 kg a.i./ha, as much as 16 mg of hexazinone could be present in each kg of dry vegetation. They concluded, however, that even at this rate, the maximum level of hexazinone and its metabolites in foliage would be below the levels known to cause toxic effects in animals. Peterson et al. (1997) concluded that detrimental effects of hexazinone on wildlife would most likely be indirect, resulting from declines in food resources and habitat quality due to losses of primary productivity in treated areas.
Aquatic Species
Hexazinone is only slightly toxic to most aquatic animals, but can be extremely toxic to some algae and aquatic macrophytes (EPA 1994). Hexazinone may also cause indirect effects to aquatic communities through destruction of riparian vegetation (Mayack et al. 1982).

Fish
Hexazinone is only slightly toxic to fish (EXTOXNET 1996). The LC50 for rainbow trout and bluegill sunfish are 320 and 370 mg/L, respectively; well above the residue levels found in the streams of treated watersheds. When bluegill sunfish were exposed to concentrations ≤1 ppm for 28 days (more than twice the maximum runoff reported by Mayack et al. (1982) in Georgia), no mortalities or changes in behavior or physical condition were observed (Rhodes 1980b). Tissue residue levels were found to peak after one to two weeks of exposure and to be completely eliminated after two weeks of withdrawal.

Hexazinone has been found to be slightly toxic to juvenile Pacific salmonids, with LC50 (96-hour) values of 236-317 mg/L (Wan et al. 1988). The formulations of Pronone 10G® and Velpar® were found to be significantly less toxic, suggesting that the additives in these formulation were not only less toxic than hexazinone itself, but somehow reduced the toxic effect of hexazinone to salmonids (Wan et al. 1988). No hexazinone formulations are registered for aquatic use.

Phytoplankton
Thompson et al. (1992, 1993a) found that the biomass of phytoplankton in boreal forest lakes in Ontario, Canada was depressed at hexazinone concentrations as low as 0.01 mg/L, and that chronic exposure to concentrations > 0.1 mg/L caused irreversible damage to phytoplankton communities. A corresponding decrease in zooplankton populations occurred as well, likely as a response to food resources lost with the decline of phytoplankton. Field studies have reported temporary contamination levels as high as 0.04 mg/L (Lavy et al. 1982), but chronic exposure to higher rates following proper application of hexazinone is unlikely.

Aquatic Invertebrates
Kreutzweiser et al. (1992) and Schneider et al. (1995) both found that in simulated stream channels, the addition of hexazinone did not affect the survival of stream insects, and concluded that there was little risk of toxic effects to macroinvertebrates (Kreutzweiser et al. 1992, 1995; Schneider et al. 1995). Similarly, Mayack et al. (1982) found no differences in diversity or species composition of aquatic invertebrate communities between treated and untreated sites.

Periphyton
Several studies in labs, simulated streams, and lake enclosures have shown that hexazinone is toxic to algae and can slow growth rates after one day of exposure. Concentrations reported to cause detrimental effects (0.01-0.60 mg/L) are well above the monthly average levels (0.00025-0.0031 mg/L) reported by Lavy et al. (1989) in streams of West Virginia following proper application of hexazinone in the watershed. Although one-time concentrations following significant storm events may exceed the tolerance threshold for some algal species, chronic exposure to lethal doses does not seem likely.

Weed Control Methods Handbook, The Nature Conservancy, Tu et al.
If chronic exposure did occur, it could cause significant losses in biomass of some algae (About-Waly et al. 1991b; Kreutzweiser et al. 1995; Schneider et al. 1995). Schneider et al. (1995) hypothesized that exposure of algae to hexazinone for time periods equivalent to the algae population’s doubling time could have significant consequences for the productivity and recovery of the community. A decline in green algae and diatoms following low-level contamination by hexazinone could ripple through the food chain and impact fish and wildlife productivity (Peterson et al. 1997). In addition, because impacts on cyanobacteria are relatively minimal, these organisms could proliferate where other algae are suppressed, altering the aquatic habitat and possibly contaminating drinking water supplies (Peterson et al. 1997).

Other Non-Target Organisms
Mayack et al. (1982) found that terrestrial macroinvertebrates accumulate hexazinone and its metabolites at levels one to two times the concentration in forest litter. It is not known whether hexazinone “magnifies” up the food chain, with organisms that feed on macroinvertebrates accumulating even higher concentrations. A study using rats suggests that mammals that ingest hexazinone can eliminate it from their systems (Rhodes & Jewell 1980).

Hexazinone has not been shown to be toxic to soil bacteria or fungi (Chakravarty & Chatarpaul 1990; Maynard 1993). Rhodes et al. (1980) found that slight increases in fungal and bacterial populations occurred with the addition of hexazinone. Fungal community structure was not altered, nor were populations of soil microbes reduced at hexazinone concentrations ≤ 10 ppm.

Soil Nutrient Cycling
Hexazinone has little if any effect on the cycling of nutrients in soils (Rhodes et al. 1980; Maynard 1993, 1997). Maynard (1993) found no effect on CO₂ respiration, ammonification, and nitrification or sulfur mineralization in incubated forest soils, and concluded that hexazinone would have little impact on nutrient-cycling processes when applied at the recommended field rates. Rhodes et al. (1980) additionally, found hexazinone had no effect on the soil-nitrifying process in three agricultural soils at hexazinone concentrations of five and 20 ppm over five weeks. Changes in vegetation coverage and the input of litter from plants killed due to the application, however, could lead to indirect effects on soil nutrient and carbon cycles (Maynard 1996). Nonetheless, Maynard (1996) found no changes in the total nutrient pool over six years in a treated boreal mixed-wood forest in Alberta.

Safety Measures:
Hexazinone can cause severe eye damage. Care should be taken to prevent accidental splashing or other exposure to eyes.

Application Considerations:
- Because hexazinone is absorbed by the roots, it is most effective in soils that do not readily bind it, such as those low in organic content, clay, silt, and cation exchange capacity, but high in sand (Minogue et al. 1988; Wilkins et al. 1993).
- Application of the liquid formulation reduces lateral movement of the herbicide, which may reduce impacts on non-target plants (Prasad & Feng 1990).
- Where granular formulations are applied in late autumn or early winter, hexazinone may be released during spring snow melt (Sidhu & Feng 1993).
- Most formulations require water to become activated, thus, best results occur when the soil is moist at the time of application and when 1/4-1/2 inch of rain falls within two weeks of application.

**Human Toxicology:**
Hexazinone is of relatively low toxicity to mammals, but can cause severe eye damage. The U.S. EPA to classifies hexazinone as a “Group D” carcinogen, or a chemical that is not classifiable as a human carcinogen (EPA 1994).

**References**


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