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ECOLOGICAL CRITERIA DOCUMENT FOR OCTAHYDRO-1,3,5,7-TETRANITRO-1,3,5,7-TETRAZOCINE (HMX) (CAS No. 2691-41-0)

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DRAFT

This document will be updated as new data are identified.
The addition of new data may result in revision of the
criteria and screening benchmarks found herein.
The document may also be edited following review
by the U.S. Environmental Protection Agency
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EXECUTIVE SUMMARY

HMX is a colorless, crystalline solid that is used as an explosive in rocket fuels and military munitions. Most HMX releases to the environment originate in wastewater from facilities which manufacture, process, or use HMX. HMX has been detected in wastewater, river water, groundwater, and soil at several Army Ammunition Plants. Monitored concentrations as high as 5.4 mg/L in untreated wastestreams, 3.36 mg/L in plant effluents, 0.067 mg/L in river water, 4.2 mg/L in groundwater, and 5700 mg/kg in soil have been noted.

Direct photolysis is the primary pathway for degradation of HMX in the aquatic environment. Hydrolysis and oxidation of HMX are negligible. Volatilization, absorption to suspended material, and biosorption are not expected to significantly remove HMX from the water column. Biotransformation will be an important pathway for degradation of HMX only under organic-rich conditions. Photolysis will not occur in soil, and HMX is persistent upon release to the terrestrial environment.

Acute aquatic toxicity tests were conducted on four invertebrate species, four fish species, and four algal species. 96-hr LC_{50} values for fish and algae and 48-hr EC_{50} values for invertebrates were all >32 mg/L with the exception of a 96-hr LC_{50} of 15 mg/L for fathead minnows at 7 days post-hatch. Reported log K_{ow} values of 0.06 to 0.26 indicate little potential for bioaccumulation in aquatic organisms.

Data were not sufficient for calculation of ambient water quality criteria. Therefore, tier II or secondary values were calculated according to the U.S. EPA guidance for the Great Lakes System. Under these guidelines, the calculated acute criterion (Secondary Maximum Concentration) is 1.9 mg/L and the chronic criterion (Secondary Continuous Criterion) is 0.33 mg/L. Lowest chronic values for fish and daphnids were not available but, based on chronic NOAELs (no-observed-adverse-effect-level), are >3.3 and >3.9 mg/L, respectively. It should be noted that the chronic criterion is an order of magnitude lower than chronic NOAELs; therefore, the lower chronic NOAEL of 3.3 mg/L may be a more representative value of the chronic criterion than the calculated value. The final plant value is >32 mg/L. A preliminary organic carbon-normalized Sediment Quality Criterion of 0.47 mg/kg_{oc} was calculated based on measured K_{ow} values and limited aquatic toxicity data.

In mammals, HMX appears to be poorly absorbed by the gastrointestinal tract. Most HMX ingested is excreted in the feces unchanged, with the small amount of absorbed HMX being excreted in urine within a few days. Based on a 13-week study of HMX-induced mortality in mice, a chronic NOAEL of 3 mg/kg/day was determined. This value was used as the basis for calculation of NOAELs and screening benchmarks for food and water intake for seven selected mammalian wildlife species: the short-tailed shrew, white footed mouse, meadow vole, cottontail rabbit, mink, red fox, and whitetail deer. The NOAEL values for the wildlife species were scaled from the test NOAEL on the basis of body weight. Screening benchmarks for food ranged from 5.6 mg HMX/kg food for the cottontail rabbit to 22 mg HMX/kg food for the meadow vole. Screening benchmarks for water intake ranged from 6.6 mg/L for the whitetail deer to 19 mg/L for the meadow vole. For piscivorous species such as the mink, the water concentration that incorporates dietary intake from both water and food consumption was 5.7 mg/L.

No suitable data were found for derivation of benchmark values for avian species, terrestrial plants, soil invertebrates or soil heterotrophic processes, although a soil concentration of 500 mg/kg was not toxic to the earthworm, *Eisenia foetida*.

1. INTRODUCTION

Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine, commonly known as HMX (High Melting Explosive) is a colorless, crystalline solid that has 130% of the explosive power of TNT. HMX is used in nuclear devices to implode fissionable material and is a component of plastic explosives, solid-fuel rocket propellants, and military munitions (McLellan et al., 1988). HMX is also formed during the manufacture of another explosive, hexahydro-1,3,5-trinitro-1,3,5-triazine or RDX. This ecotoxicological profile of HMX summarizes available data on environmental concentrations, environmental fate and transport, and ecotoxicity and bioaccumulation for aquatic and terrestrial plants and animals. Ecological criteria and screening benchmarks are derived for those groups of organisms for which there are sufficient data.

ENVIRONMENTAL FATE

2.1 SOURCES AND OCCURRENCES

HMX is currently produced only at the Holston Army Ammunition Plant (Holston AAP) in Kingsport, Tennessee. More then 30 million pounds of HMX is believed to be produced each year (ATSDR, 1994). Further processing may occur at facilities where load, assemble and pack (LAP) operations are performed by the U.S. military (Ryon et al., 1984). Environmental releases may also occur as a result of treatment and disposal of HMX- and RDX-containing wastes (i.e., RDX contains about 5% HMX [Kitchens et al., 1979]). HMX has been detected at several sites on the Superfund National Priorities List (ATSDR, 1994).

2.1.1 Air

HMX may be atmospherically released by adsorption to airborne particulates released during munitions manufacturing (McLellan et al., 1988). However, this is not expected to be a major pathway for HMX pollution. Estimates of HMX release to the air are unavailable (ATSDR, 1994).

2.1.2 Surface and Groundwater

Most HMX releases to the environment originate in wastewater from facilities that manufacture, process, or use HMX (ATSDR, 1994). HMX released to soil has been shown to partition to groundwater (Gregory and Elliott, 1987).

Stidham (1979) reported that the Holston AAP was releasing 45 lbs of HMX/day in wastewater to the Holston River, with effluent concentrations ranging from 0.09 to 3.36 mg/L. Levels as high as 67 ug/L occurred one mile downstream of the last plant effluent, as a result of incomplete mixing of the wastestream with river water. Kitchens et al. (1978) estimated that at full mobilization at least 123 lbs of HMX would be released per day. In this study, sampling at various points in the river and Holston AAP wastestreams indicated concentrations of HMX between 0.01 (river) and 2.6 (wastewater) mg/L. Stillwell et al. (1977) also sampled various wastestreams from the Holston AAP and found that untreated wastestreams had HMX levels from 0.14 - 5.4 mg/L (except for one sample below which was the detection limit of 0.1 mg/L), while treated wastestreams had concentrations that ranged from <0.1 - 2.07 mg/L.

As part of a remedial investigation of the Louisiana AAP, Gregory and Elliott (1987) measured HMX levels in groundwater from four areas. Around the Area P leaching pits, HMX was detected in samples from 14 of 32 monitoring wells, with concentrations ranging from 3.12 - 4200 ug/L. At Burning Ground #5, levels ranged from 1.23 - 145 ug/L for 8 of 9 wells. At Inert Landfill Area #3, an HMX level of 2.28 ug/L was noted in one well out of 13. At Inert Landfill Area #8, HMX concentrations of 8.3 - 157 ug/L were detected in samples from 6 of 19 wells.

2.1.3 Soils

Soil contamination occurs as a result of spills at Army facilities and disposal of HMX-contaminated wastes in landfills. Over a 10 year period Walsh and Jenkins (1992) collected dried soil samples from 17 Army sites, and analyzed the samples for a number of chemicals relating to munitions use and production. Levels of HMX in excess of the detection limit (1.1 ppm) were found at 5 of these sites; levels ranged from 1.1-115 ppm at the Nebraska Ordinance Works (median 7.7), 2.5-5700 ppm at Iowa AAP (median 28.7), 56-2435 ppm at Hawthorne AAP (median 142), 3.7-86 ppm at Milan AAP (median 53) and 68-258 ppm at Louisiana AAP (median 163). As part of a study designed to evaluate the toxicity of Joliet AAP soils, Simini et al. (1995) determined HMX concentrations in soil taken from a LAP area. Levels of HMX ranged from less than detection to 3055 ppm and exceeded the detection limit in 13 of 40 samples. Spiker et al. (1992) and Funk et al. (1993) reported that a soil sample from an Army munitions depot near Umatilla, OR, contained 300 ppm of HMX. Four soil samples taken from the Naval Surface Warfare Center, Crane, IN, contained concentrations ranging from 0.10 to 12.2 mg/kg (dry weight) (Grant et al., 1995).

2.2 TRANSPORT AND TRANSFORMATION PROCESSES

2.2.1 Abiotic Processes

Air. HMX has a vapor pressure of 3.3×10^{-14} mm Hg and a Henry's law constant of 2.6×10^{-15} atm-m^3/mole at 25°C (Burrows et al., 1989), thus it will not partition to the air in the vapor phase. However, HMX may be atmospherically transported via adsorption to airborne particulates, especially those from munitions manufacture (ATSDR, 1994). Photolysis will limit the persistence of airborne HMX as it does in the aquatic environment. However, no data are available on the fate of airborne HMX.

Water. Direct photolysis is the primary pathway for degradation of HMX in the aquatic environment. Hydrolysis and oxidation of HMX are negligible (McLellan et. al., 1988). Spanggord et al. (1982) determined photolytic half-lives for HMX of 4-5 days in surface water from the Holston River. In a more detailed follow-up study, photolytic half-lives for HMX were determined to be 1.4, 1.7, and 70 days in pure water, Holston River water, and Louisiana AAP lagoon water, respectively (Spanggord et al., 1983). The rate of photolysis was apparently related to the amount of UV-absorbing material in the water; it was not affected by any co-substrates present. Measured stable end-products of photolysis were nitrate, nitrite, and formaldehyde. Dinitrogen oxide, ammonia, and formaldehyde were proposed as additional end products. Modeling studies on the fate of HMX in the Holston River and Louisiana AAP lagoon under their natural conditions (including such factors as water flow, depth, and seasonal changes) indicated that the half-life of HMX would be 17 days in the Holston River and 7900 days in the Louisiana AAP lagoon (Spanggord et al., 1983). HMX was predicted to persist in the Holston River for distances greater than 20 km downstream.

Volatilization, adsorption to suspended material, and biosorption are not expected to significantly remove HMX from the water column (Spanggord et al., 1982). Volatilization half-lives of 1000-3000 days have been estimated. Partitioning of HMX to Holston River sediment was studied, and upper limit values of 8.7 for the sediment-water partition coefficient (K_p) and 670 for the organic carbon-water partition coefficient (K_{oc}) were determined. These values indicate a low to moderate affinity of HMX in the water column for soil or suspended material; thus, HMX dissolved in the water will not be removed though adsorption to sediment. For the same reason, dissolved HMX will readily migrate to groundwater from soil-contaminated areas. However, the low solubility of HMX (6.6 mg/L at 20°C; McLellan et. al., 1988) limits the partition to, and thus the migration of, HMX in groundwater.

Soil and Sediment. Hale et al. (1979) studied the migration of TNT and RDX in soil by treating the top 10 cm of soil-filled cylinders with 20,000 ppm of contaminant and sampling various soil depths after 6 months. The concentrations of TNT and RDX decreased from 20,000 ppm in the top 10 cm to <10,000 at 10 to 20 cm and <1000 at 20 to 30 cm. Below 60 cm, levels remained below 300 ppm. It was assumed by Ryon et al. (1984) that HMX would behave similarly. This is a result of the low solubility of HMX, limiting its mobility in soil, despite the low affinity of HMX to bind to soil particles. Migration of HMX is greatest in coarse, loamy soil (Ryon et al., 1984). Direct photolysis will not occur in soil, and the chemical should be persistent in the soil environment (Ryon et al., 1984). This was confirmed by DuBois and Baytos (1991) who buried HMX in soil exposed to the environment at Los Alamos, NM and calculated a half-life of 39 years.

Spanggord et al. (1982) calculated an upper limit partition coefficient (K_p) for HMX on Holston River sediment of $\leq$8.7 mL/g (L/kg). The K_p was calculated from a linear least squares plot of HMX water concentration versus HMX sediment concentration (dry weight) from water-sediment samples prepared from HMX-contaminated water and Holston River sediment. The organic content of the sediment was determined to be 1.3%, from which a K_{oc} value of $\leq$670 L/kg was calculated. These values indicate that sediment sorption will not be a major fate for HMX in surface waters.

The transport of pink water compounds including HMX (4 mg/L) through columns of garden soil (6.5% organic matter content) amended with microbes from activated sludge and anaerobic sludge digest was studied by Greene et al. (1984). Pink water solutions were continuously pumped through the columns and effluent samples were collected weekly over a period of 110 days. Flow rates were varied and some columns were amended with glucose. Unmetabolized HMX was rapidly recovered in the leachates, indicating little adsorption to soil.

Checkai et al. (1993) collected intact soil-core columns from an uncontaminated area at the Milan AAP in order to study transport and transformation of munitions chemicals in site-specific soils. The soil was a Lexington silt loam with a 6-in A horizon containing 16 g/kg organic matter and a B horizon extending from 6 to 27 inches and containing 5 g/kg organic matter. A mixture of munitions simulating open burning/open detonation ash was added to the soil surface. Concentrations were 1000 mg/kg RDX, 1000 mg/kg HMX, 1000 mg/kg 2,4-dinitrotoluene, and 400 mg/kg 2,6-dinitrotoluene. The columns were leached with simulated rainfall over a period of 32.5 weeks; controlled tension was applied. HMX was measurable in leachates throughout the study and averaged 0.4 mg/L. Over time, HMX was found at progressively greater depths within the soil column; by 19.5 weeks, it had migrated throughout the full column (detection level, 2.9 mg/kg).

2.2.2 Biotransformation

Biotransformation will be an important pathway for degradation of HMX under organic-rich conditions. Spanggord et al. (1982) discovered that no biodegradation of HMX occurred after 15 weeks when 4 ppm HMX was added to river waters or river waters with HMX contaminated sediment. However, the addition of yeast to river water containing 4 ppm of HMX reduced the concentration to <0.1 ppm in three days. Spanggord et al. (1982) found that anaerobic transformation of HMX followed a similar pattern. No degradation occurred in river water, slow degradation occurred in water over HMX-contaminated sediment (4 ppm to < 0.2 ppm in 91 days), and rapid degradation occurred in the yeast-treated water (same rate as for aerobic conditions). Aerobic and anaerobic biotransformation of HMX ultimately produces 1,1-dimethylhydrazine. Under natural conditions biotransformation may not be a significant loss mechanism for HMX.

In a detailed follow up study, Spanggord et al. (1983) further investigated the biotransformation of HMX in water. HMX biotransformation occurred under both aerobic and anaerobic conditions in wastestream effluent treated with yeast. Under aerobic conditions a first-order half-life of 17 hours was observed; under anaerobic conditions the first-order half-life was 1.6 hours. However, conditions in the Holston River environment are not expected to induce noticeable biotransformation of HMX, thus biotransformation is not expected to contribute to loss of HMX in this environment.

3. AQUATIC TOXICOLOGY

3.1 TOXICITY

3.1.1 Acute effects - Invertebrates and fish

Bentley et al. (1977) studied the acute effects of HMX on four species of freshwater invertebrates: the midge *Chironomus tentans*, the cladoceran *Daphnia magna*, the amphipod *Gammarus fasciatus*, and the isopod *Asellus militaris* (Table 1). After 48 hours under static conditions, even the highest tested nominal concentration (32 mg/L) had no adverse effect on any of the organisms. Thus, the 48 hour-EC_{50} of >32 mg/L based on immobilization is reported for the invertebrates tested in this study.

Bentley et al. (1977) also studied the effects of HMX on fish tested under static conditions. Test species included bluegill sunfish (*Lepomis macrochirus*), channel catfish (*Ictalurus punctatus*), fathead minnow (*Pimephales promelas*), and rainbow trout (*Oncorhynchus mykiss*). Bluegill sunfish were used to test the effect of changes in temperature, pH, and water hardness. None of these factors had a significant effect. Fathead minnows were used to determine how HMX acute toxicity would vary with life stage. Fathead minnows were most susceptible at 7 days post-hatch (96-hour LC_{50} of 15 mg/L). All other life stages for fathead minnows were unaffected by even the highest levels of HMX tested (96-hour LC_{50} values of >32 mg/L). Channel catfish and rainbow trout were similarly unaffected.

TABLE 1. RESULTS OF ACUTE TOXICITY STUDIES WITH AQUATIC INVERTEBRATES AND FISH[a]			
Test Species	Test Duration	LC_{50} (mg/L) for fish; EC_{50} (mg/L) for invertebrates[b]	Reference
Daphnia magna (water flea)	48-hr	>32	Bentley et al., 1977
Asellus militaris (sowbug)	48-hr	>32	Bentley et al., 1977
Gammarus fasciatus (scud)	48-hr	>32	Bentley et al., 1977
Chironomus tentans (midge)	48-hr	>32	Bentley et al., 1977
Lepomis macrochirus (bluegill)	96-hr	>32	Bentley et al., 1977
Ictalurus punctatus (channel catfish)	96-hr	>32	Bentley et al., 1977
Pimephales promelas (fathead minnow)	96-hr	15	Bentley et al., 1977
Oncorhynchus mykiss (rainbow trout)	96-hr	>32	Bentley et al., 1977

[a] Static conditions.
[b] These nominal concentrations exceed the solubility limit of 6.6 mg/L at 20°C.

3.1.2 Chronic effects - Invertebrates and Fish

One study was located that tested the chronic toxicity of HMX to aquatic organisms. Bentley et al. (1984) tested the effects of long term HMX exposure on the water flea (*Daphnia magna*) and fathead minnow (*Pimephales promelas*) under flow-though conditions (6.4 aquarium volume replacements per day for fathead minnows, 4-5 volume replacements per day for water fleas) (Table 2). Fathead minnow embryo-larval stages were exposed to HMX for 32 days. Within 48 hours of fertilization, embryos in cups (120 per treatment concentration) were suspended in HMX-treated aquariums, and percentage hatch was evaluated at five nominal concentrations of HMX (0.31 - 5.0 mg/L) and a control. All live larvae were transported to the aquariums upon hatching, and 32 days later the percentage survival, mean total length, and average wet weight were determined. Water fleas were exposed to five nominal concentrations of HMX (0.69 - 11 mg/L; 80 daphnids per treatment concentration) for a total of 28 days. Weekly survival and offspring production were measured. All toxicity endpoints for both the fathead minnow and water flea were comparable to control levels for all concentrations tested. HMX concentrations were also measured, and were found

to be significantly lower than nominal concentrations, especially at the higher nominal HMX levels. For the nominal concentration of 11 mg/L used in the water flea test, measured concentrations ranged from 2.7 - 6.0 mg/L with a mean of 3.9 mg/L, and at the nominal concentration of 5 mg/L used in the fathead minnow test, measured concentrations ranged from 2.3 - 4.9 mg/L with a mean of 3.3 mg/L. Overall, it was concluded that no adverse effects of exposures were seen to *Daphnia magna* at 3.9 mg/L HMX or to *Pimephales promelas* at 3.3 mg/L HMX.

3.1.3 Plants

The acute toxicity study of Bentley et al. (1977) was the only study found that investigated the effects of HMX on aquatic plants. The cyanophytes (blue-green algae) *Microcystis aeruginosa* and *Anabaena flos-aquae*, the chlorophyte (green alga) *Selenastrum capricornutum*, and the chrysophyte (diatom) *Navicula pelliculosa* were tested by measurement of chlorophyll *a* content and culture cell numbers. HMX had no adverse effect on any of the four species of phytoplankton at the tested concentrations; in fact, cell counts or chlorophyll *a* content increased in each case. EC_{50} values were then assumed to be >32 mg/L (Table 3). Sullivan et al. (1979) summarized and evaluated the HMX aquatic data of Bentley et al. (1977) and considered the statistical analysis to be questionable. Upon re-analysis, considering growth in cell counts or chlorophyll *a* content as the endpoint, HMX concentrations as low as 10 mg/L significantly increased growth in three of the algae species and the chlorophyll *a* content in one species.

3.2 METABOLISM AND BIOCONCENTRATION

No data on HMX metabolism by aquatic organisms was found. Reported log K_{ow} values of 0.26 (Burrows et al., 1989), 0.13 (Tucker et al., 1985), and 0.06 (Jenkins, 1989) indicate little potential for partitioning to body lipids; thus, HMX is not expected to bioaccumulate in fish.

3.3 AQUATIC CRITERIA AND SCREENING BENCHMARKS

3.3.1 Aquatic Organisms

Data were insufficient for calculation of acute and chronic Water Quality Criteria (WQC) according to U.S. EPA guidelines (Stephan et al., 1985). Guideline data are available for only six of the required eight families, representing planktonic crustaceans (*Daphnia magna*), the family Salmonidae (*Oncorhynchus mykiss*), a second family in the class Osteichthyes (*Lepomis macrochirus* or *Ictalurus punctatus*), a third family in the phylum Chordata (*Pimephales promelas*), benthic crustaceans (*Gammarus fasciatus* or *Asellus militaris*), and insects (*Chironomus tentans*). Therefore, Tier II or Secondary Acute and Chronic Values were calculated according to U.S. EPA guidance for the Great Lakes System (U.S. EPA, 1993a). Using this methodology, Secondary Acute and Secondary Chronic Values may be calculated with less than the eight minimum data requirements necessary for calculation of the Tier I or Final Acute and Final Chronic Values. The methodology and calculations are contained in Appendix A. The types of aquatic criteria and benchmarks that can be calculated are summarized below and values are listed in Table 4.

It should be noted that the tests conducted by Bentley et al. (1977) and on which the criteria/benchmarks are based do not follow the methodology outlined by Stephan et al. (1985). The acute experiments performed by Bentley et al. (1977) were under static conditions, and HMX

TABLE 2. RESULTS OF CHRONIC AQUATIC TOXICITY STUDIES[a]

Species	Stage/age	Parameter Measured	Parameter Response	Concentration (mg/L)	Reference
Daphnia magna (water flea)	adult	weekly survival and offspring production; 28 days	No adverse effect	3.9[b]	Bentley et al., 1984
Pimephales promelas (fathead minnow)	embryo-larvae	percentage hatch for embryos, percentage survival, mean total length, and average wet weight for larvae; 32 days	No adverse effect	3.3[b]	Bentley et al., 1984

[a] Flow-through tests
[b] Mean measured concentration

TABLE 3. RESULTS OF TOXICITY TESTS WITH SPECIES OF ALGAE[a]

Test Species	Test Duration	Effect	Concentration (mg/L)[b]	Reference
Microcystis aeruginosa (blue-green alga)	96-hr	No Adverse Effect	32	Bentley et al., 1977
Anabaena flos-aquae (blue-green alga)	96-hr	No Adverse Effect	32	Bentley et al., 1977
Selenastrum capricornutum (green alga)	96-hr	No Adverse Effect	32	Bentley et al., 1977
Navicula pelliculosa (diatom)	96-hr	No Adverse Effect	32	Bentley et al., 1977

[a] Static conditions.
[b] These nominal concentrations exceed the solubility limit of 6.6 mg/L at 20°C.

concentrations were reported as nominal (not measured), being computed based on the dilution of a superstock solution of HMX dissolved in acetone. Due to the stability and slow removal rates of HMX in the water environment, HMX is not expected to be significantly lost in the time frames used. Thus, the static conditions used by this study should not significantly alter the results. However, nominal concentrations reported in this study were in excess of the solubility limits of HMX (6.6 mg/L at 20°C [McLellan et. al., 1988]). Bentley et al. (1984) reported that measured HMX concentrations averaged only 3.9 mg/L in tests in which the nominal concentration was 11 mg/L. The later study utilized flow-though conditions and used HMX concentrations taken from aquarium measurements in determining HMX levels of concern. For analysis of the acute values taken from Bentley et al. (1977), concentrations exceeding the known solubility of HMX were used in deriving water quality criteria in accordance with Stephan et al. (1985). However, it should be noted that these values based on nominal concentrations may very well be over-estimates and thus the confidence in the aquatic criteria/benchmarks is low.

Secondary Acute Value (SAV). The SAV is calculated by dividing the lowest Genus Mean Acute Value (GMAV) by the Secondary Acute Factor (SAF). U.S. EPA (1993a) lists SAFs for use in Tier II calculations; these SAFs are based on the number of satisfied data requirements for Tier I calculations. A lowest GMAV (15 mg/L for *Pimephales promelas*) and six data requirements for Tier I criteria were available to calculate a Tier II SAV.

Secondary Maximum Concentration (SMC). The SMC, analogous to the acute WQC or criterion maximum concentration, is equal to one half of the SAV.

Secondary Chronic Value (SCV). The SCV is equal to the ratio of the SAV and the geometric mean of the acute/chronic ratios.

Secondary Continuous Concentration (SCC). The SCC, analogous to the chronic WQC, is equal to the lowest of three values, the SCV, the Final Plant Value, and the Final Residue Value.

Lowest Chronic Values. In the absence of a Final Chronic Value, the lowest chronic concentrations found which had an effect on fish or daphnids can be used as potential lower benchmarks, i.e., there is a low likelihood of significant effects. Bentley et al. (1984) reported the only chronic HMX toxicity values found for fish and daphnids, and these are used as the Lowest Chronic Values.

Final Plant Value. For algal species, a test of at least 96-hours duration should be used; concentrations of test material should be measured and the endpoint should be biologically significant (Stephan et al., 1985). In considering protection of plant species, increases in growth will not be considered biologically important and the lower-limit EC_{50} value of >32 mg/L for all four algae species tested by Bentley et al. (1977) will be considered applicable to the Final Plant Value.

Final Residue Value. A maximum permissible tissue concentration has not been determined according to Stephan et al. (1985); thus, a Final Residue Value could not be calculated.

3.3.2 Sediment-Associated Organisms

One study was found that tested the toxicity of HMX to benthic organisms: the midge *Chironomus tentans*, the isopod *Asellus militaris*, and the amphipod *Gammarus fasciatus* (Bentley et

al., 1977); however, the tests were conducted in 250 ml beakers without sediment and cannot be used to derive a Sediment Quality Criterion (SQC). From these tests, it is known that a sediment pore water concentration at the limit of HMX solubility would not be toxic to these organisms (Table 1).

There are many different approaches to the development of Sediment Quality Criteria (SQC) of which three - the measurement of interstitial (pore) water, spiked sediment toxicity tests, and Equilibrium Partitioning (EqP) - have been recommended (Hull and Suter, 1994). For nonionic organic chemicals, the EqP approach can be used to develop a SQC (Di Toro et al., 1991; U.S. EPA, 1993a). SQC may be calculated from K_p values and FCV; the result is expressed as mg of chemical/kg soil or sediment (dry weight). However, normalization to a sediment organic carbon content is recommended by U.S. EPA and is more useful than a SQC based on dry weight in that it allows comparison among sites when the mass fraction of organic carbon in sediment at a particular site is known. The methodology and calculations for SQC are contained in Appendix B and the value is listed in Table 4. In order to compare the SQC_{oc} to dry weight conentrations at a particular site, the dry weight concentration and the organic carbon concentration at the site must be known. The conversion is: mg chemical/kg_{oc} = (mg $chemical_{dry\ weight}$ x 100)/f_{oc}. The organic carbon-normalized concentration can then be compared with the SQC_{oc}. Because the SQC_{oc} is based on a SCV rather than a Final Chronic Value, it should be considered preliminary.

TABLE 4. WATER AND SEDIMENT QUALITY CRITERIA/SCREENING BENCHMARKS FOR AQUATIC ORGANISMS[a]

Criterion	Value
Acute Water Quality Criterion	Insufficient data
Chronic Water Quality Criterion	Insufficient data
Secondary Acute Value	3.8 mg/L
Secondary Maximum Concentration	1.9 mg/L
Secondary Chronic Value	0.33 mg/L
Secondary Continuous Concentration	0.33 mg/L
Lowest Chronic Value - fish	>3.3 mg/L[b]
Lowest Chronic Value - daphnids	>3.9 mg/L[b]
Final Plant Value	>32 mg/L
Final Residue Value	Insufficient data
Sediment Quality Benchmark (SQC_{oc})	0.47 mg/kg_{oc}[c]

[a] See Appendix A for calculations.
[b] These chronic NOAELS may be more realistic representations of the Secondary Continuous Concentration than the calculated value of 0.33 mg/L/
[c] mg chemical/kg organic carbon in sediment.

4. TERRESTRIAL TOXICOLOGY

4.1 TOXICITY

4.1.1 Mammals

No information on the toxicity of HMX to wildlife was found. Two studies were located that investigated the subchronic toxicity of HMX to laboratory animals for periods greater than two weeks.

Everett et al. (1985) incorporated HMX into the diets of male and female Fischer 344 rats for 13 weeks. Twenty rats were tested at each dose level for both sexes; males at 0, 50, 150, 450, 1350, and 4000 mg/kg/day and females at 0, 50, 115, 270, 620, and 1500 mg/kg/day. Three deaths were recorded: one male in the 150 mg/kg/day group after 9 weeks, one control group female at week 13, and a female at the highest dose level during the first week. There were no clinical signs of toxicity in the HMX-treated animals, although significant dose-related reductions in mean body weight gain occurred for all animals concurrent with reductions in food consumption early in the study. After week 5, only animals in the two highest dose groups continued to have depressed weight gains.

Histopathological examinations revealed that males receiving 450 mg/kg/day or more of HMX had a significant incidence of toxic liver changes, with some incidences noted as low as 150 mg/kg/day. In females, the three highest dose levels resulted in significant incidences of tubular kidney changes. Thus, the toxicity of HMX on organs was demonstrated to be sex-specific. Hematology, clinical chemistry, urinalysis, and organ weight and pathology examinations were conducted on the males and females receiving the highest dose levels, with comparisons made to the control group. Decreases in hemoglobin and hematocrit were seen in both males and females at week 5 and in females only at week 12. Red blood cell counts were lower for females at week 12. Significant increases in serum alkaline phosphatase occurred for males at week 12, with a marginal increase seen in females. A slight increase in albumin was seen in males, and a slight increase in blood urea nitrogen was seen in females at weeks 5 and 12. In females, there was a decrease in urine volume, a lower urine pH, and lowered urine specific gravity at 12 weeks, with no such changes noted for males. Organ weights were recorded at all dose levels, and a number of organ weight changes were noticed for both males and females at all but the lowest dose levels. However, it is difficult to interpret their toxicological significance since many organ weight changes simply reflected the decreases in body weight.

Everett and Maddock (1985), in a similar study to the one described above, tested the oral toxicity of HMX in $B6C3F_1$ mice. Males were dosed with 0, 5, 12, 30, 75, and 200 mg/kg/day and females with 0, 10, 30, 90, 250, and 750 mg/kg/day, in groups of twenty animals each. Significant mortality resulted: one male in the 30 mg/kg/day group, two males in the 75 mg/kg/day group, and 13 males in the 200 mg/kg/day group expired; one control female, one female in the 30 mg/kg/day group, 12 females in the 250 mg/kg/day group, and all females in the 750 mg/kg/day group died. Surprisingly, no clinical signs of toxicity were noted in any of the treatment groups. Similarly, no significant changes in body weight or clinical chemistry were observed. It was reported that the deaths in the 30 mg/kg/day dose groups were not attributable to HMX exposure as no deaths occurred in females receiving 90 mg/kg/day. Histologic examination of the highest dosed males and females showed few changes in the liver, kidney, spleen and brain. Lungs were observed to be a dark red color in some of the highest dosed mice, but lungs were not histologically examined.

4.1.2 Birds

No studies were found that tested the toxicity of HMX to birds.

4.1.3 Plants

There are no studies that specifically tested the toxicity of HMX to terrestrial plants. However, Simini et al. (1995) evaluated the toxicity of Joliet AAP soil to radishes and cucumbers. The soil samples were contaminated with several munitions, each at a range of concentrations. HMX was found to be very weakly correlated with the effects of munitions-contaminated soil on plant height.

4.1.4 Soil Invertebrates

A study was found that tested the toxicity of munitions-contaminated soil (contaminated in part with HMX) and calculated the correlation of HMX concentration with overall soil toxicity. Simini et al. (1995) evaluated the toxicity of Joliet AAP soil to earthworms by determining survival rates and differences between initial and final live weights. HMX was found to be very weakly correlated with these endpoints.

In a preliminary study, Phillips et al. (1993) tested the toxicity of HMX added to artificial soil (1.4% organic matter) to the earthworm, *Eisenia foetida* over a 14-day period. Concentrations of HMX were 0, 50, 100, 200, 400, and 500 mg/kg. Although survival was 100% for all tested concentrations, there was an increasing weight loss with increasing concentrations. Weight losses ranged from 6% at the 50 mg/kg concentration to 15-18% at the three highest concentrations.

4.1.5 Soil Heterotrophic Processes

No studies were located that tested the toxicity of HMX to soil microorganisms.

4.2 METABOLISM AND BIOACCUMULATION

4.2.1 Animals

Metabolism. HMX appears to be poorly absorbed by the gastrointestinal tract (ATSDR 1994). Cameron (1986) exposed $B6C3F_1$ mice and F344 rats to single oral doses (500 mg/kg) of [^{14}C]HMX to investigate HMX excretion. Four days after exposure to rats, 85% of the radiolabel was excreted in feces (primarily as unchanged HMX), 4% in the urine, and, within 48 hours, 0.5% in exhaled air (as $^{14}CO_2$). Respective values for the mouse were 70, 3, and 1%. Only 0.6-0.7% of the original radioactivity remained in the animals after four days. Cameron (1986) found that peak HMX plasma levels only reached <0.1% of the administered dose. A comparison of urine and plasma levels of radioactivity following oral and intravenous administration to rats suggested that <5% of the oral dose was absorbed. Similarly, Everett et al. (1985) found low, non-dose dependent levels of HMX in plasma of rats following 13 weeks of exposure to 50 - 4000 mg/kg/day and concluded that little of the dose was absorbed. No information was found on the distribution of HMX in the body following an oral dose. Following intravenous administration to the rat, highest concentrations of radioactivity were observed in liver and kidney and the lowest concentration was in the brain (Cameron, 1986). Little data exist on the metabolism of HMX. Following intravenous

administration to the rat, unidentified polar metabolites were present in the tissues, plasma, and urine (Cameron, 1986). Everett and Maddock (1985) analyzed the stomach contents of $B6C3F_1$ mice dosed with 5 - 750 mg/kg/day for nitrite (which would be formed from cleavage of the ring nitrogen-nitrogen bonds) and did not observe any increases above normal dietary concentrations. It was concluded that HMX was not being metabolized in the stomach. In summary, most HMX ingested will be excreted in the feces as unchanged HMX, with the small amount of absorbed HMX being excreted in urine within a few days (ATSDR 1994).

Bioaccumulation. HMX is rapidly excreted in mammals, with passive diffusion thought to be the process by which HMX is distributed and absorbed by the body (ATSDR 1994). Thus, bioaccumulation is not expected to occur. Biomonitoring studies have been conducted at Aberdeen Proving Ground, Maryland (USACHPPM, 1994), and at several other AAPs (see Opresko, 1995 for review). HMX was not present in tissues of terrestrial wildlife (deer and small mammals) at or above a detection limit of 0.1 mg/kg.

4.2.2 Plants

No studies were found regarding HMX uptake and metabolism in plants.

4.3 TERRESTRIAL CRITERIA AND SCREENING BENCHMARKS

4.3.1 Animals

Mammals. In the same manner that safe doses of contaminants for humans are based on studies using laboratory animals, reference doses or screening benchmarks for wildlife may be calculated by extrapolation among mammalian species. Two 13-week studies of the oral toxicity of HMX to laboratory animals were available for calculation of screening benchmarks for mammalian wildlife. Everett et al. (1985) determined renal and hepatic effects of HMX exposure on male and female F344 rats, and Everett and Maddock (1985) measured HMX-induced mortality in male and female $B6C3F_1$ mice. Considering male and female endpoints from both studies, no-observed-adverse-effect-levels (NOAELs) ranged from 30 - 115 mg/kg/day, and lowest-observed-adverse-effect-levels (LOAELs) ranged from 75 - 270 mg/kg/day. Despite using the more severe endpoint of mortality in the mice study, NOAELs and LOAELs were generally lower than those found based on liver and kidney damage to rats. Mortality was considered the more relevant endpoint for population level effects; thus, the NOAEL of 30 mg/kg/day (for both males and females) from the mouse study was chosen for calculation of screening benchmarks for mammalian wildlife. Conversely, for humans, an oral reference dose (RfD) of 0.05 mg/kg/day was calculated by dividing the NOAEL of 50 mg/kg/day for male rats (endpoint - liver lesions) by an overall uncertainty factor of 1000 (U.S. EPA 1993b).

Screening benchmarks are safe exposure levels (NOAEL values) for environmental media (food and water intake) for wildlife and are calculated by extrapolation from the laboratory mouse study by normalizing for body weight. The general methodology for derivation of these benchmarks is presented in Appendix C (see also Opresko et al., 1995). NOAEL values were calculated for the short-tailed shrew, white footed mouse, meadow vole, cottontail rabbit, mink, red fox, and whitetail deer (Table 5). The dietary concentrations in food or water for the wildlife species listed in Table 5 assume that exposure to HMX occurs from either food or water, and does not occur though other

environmental media. If HMX is present in both food and water, or other media, the values in Table 5 must be adjusted to sum to the NOAEL. Confidence in these benchmarks are low due to the limited number of studies and the variability in HMX toxicity found.

Birds. No studies were found that tested the toxicity of HMX to birds, so screening benchmarks cannot be derived.

4.3.2 Plants

No studies were found that directly tested the toxicity of HMX to terrestrial plants. The correlation of HMX concentration in munitions-contaminated soil to toxicity in garden plants calculated by Simini et al. (1995) is not applicable to deriving screening benchmarks.

4.3.3 Soil Invertebrates

According to the method of Will and Suter (1995), a 20% reduction in growth would be a threshold for significant effects. Although concentrations up to 500 mg RDX/kg of artificial soil were not lethal to the earthworm, *Eisenia foetida*, weight losses occurred but were <20% at the highest concentration tested, 500 mg/kg (Phillips et al., 1993). In addition, HMX in other soil types may behave differently than in artificial soils and additional studies are needed before a screening value for earthworms can be determined.

TABLE 5. SCREENING BENCHMARKS FOR SELECTED MAMMALIAN WILDLIFE SPECIES[a]

Wildlife Species	NOAEL[b] (mg/kg/day)	Screening Benchmarks		
		Diet (mg/kg food)	Water (mg/L)	Piscivorous species (mg/L)[c]
Short-tailed shrew	3.3	5.6	15	
White-footed mouse	3.0	20	10	
Meadow vole	2.6	22	19	
Cottontail rabbit	1.1	5.6	12	
Mink	1.2	8.5	12	5.7
Red fox	0.8	8.0	9.6	
Whitetail deer	0.4	14	6.6	

[a]For a detailed description of the methodology, see Appendix B.
[b]NOAELs are for chronic exposures.
[c]Water concentration that incorporates dietary intake from both water and food consumption.

4.3.4 Soil Heterotrophic Processes

No studies were found that tested the toxicity of HMX to soil microorganisms, ther re, screening benchmarks cannot be derived.

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APPENDIX A

DERIVATION OF AQUATIC BENCHMARKS

A.1 GENERAL METHODOLOGY

When toxicity data are insufficient to derive Tier I or acute and chronic Water Quality Criteria (WQC) for protection of aquatic organisms according to U.S. EPA guidelines (Stephan et al., 1985), the U.S. EPA's *Proposed Water Quality Guidance for the Great Lakes System* (U.S. EPA, 1993a) presents a method for derivation of benchmarks (analogous to the Final Acute Criterion and Final Chronic Criterion) with fewer data points than the number required for WQC. These benchmarks are referred to as Secondary or Tier II values.

The Secondary Acute Value (SAV), analogous to the Tier I Final Acute Value, is calculated by dividing the lowest Genus Mean Acute Value (GMAV) in the data base by the Secondary Acute Factor (SAF). In order to calculate a SAV, the data base must contain a genus mean acute value for one of the three genera in the family Daphnidae: *Ceriodaphnia* sp., *Daphnia* sp., or *Simocephalus* sp. U.S. EPA (1993a) lists SAFs for use in Tier II calculations (Table A-1); these SAFs are based on the number of satisfied data requirements for Tier I calculations. The Secondary Maximum Concentration (SMC), analogous to the acute WQC, is equal to one-half of the SAV.

Table A-1. Secondary Acute Factors

Number of Satisfied Minimum Data Requirements	1	2	3	4	5	6	7
Secondary Acute Factor	20	13	8.6	6.5	5.0	4.0	3.6

Following calculation of the SAV, a Secondary Chronic Value (SCV), analogous to the Tier I Final Chronic Value, can be calculated, depending on the availability of acute:chronic ratios (ACR) for the species of interest. If fewer than three acceptable experimentally-determined ACRs are available, enough assumed ACRs of 18 are added so that the total number of ACRs equals three. The geometric mean of the ACRs is designated the Secondary Acute-Chronic Ratio (SACR). The SCV is equal to the SAV divided by the SACR. The Secondary Continuous Concentration (SCC), analogous to the Tier I chronic WQC, is equal to the lowest of three values: the SCV, the Final Plant Value, and the Final Residue Value.

A.2 CALCULATION OF SECONDARY CONCENTRATIONS

1. Calculation of Secondary Acute Value (SAV)

Acceptable acute tests for Tier I data requirements were available for six of the eight families required for derivation of WQC according to Stephan et al. (1985), representing planktonic

crustaceans (*Daphnia magna*), the family Salmonidae (*Oncorhynchus mykiss*), a second family in the class Osteichthyes (*Lepomis macrochirus* or *Ictalurus punctatus*), a third family in the phylum Chordata (*Pimephales promelas*), benthic crustaceans (*Gammarus fasciatus* or *Asellus militaris*), and insects (*Chironomus tentans*). It should be noted that "'greater than' values and those which are above the solubility of the test material should be used because rejection of such acute values would unnecessarily lower the FAV by eliminating acute values for resistant species" (Stephan et al., 1985). Thus, these values were considered. Although acute/chronic ratios were available for two families: Daphnidae and Cyprinidae, the ">" values for both the acute and chronic data for Daphnidae made the ratio unacceptable.

The SAV is calculated by dividing the lowest GMAV in the data base by the SAF. The lowest GMAV was 15 mg/L for *Pimephales promelas*. U.S. EPA (1993a) lists a SAF of 4.0 for use in Tier II calculations when six satisfied data requirements for Tier I calculations are available (Table A-1).

Therefore,

$$SAV = \frac{15}{4.0} \text{ mg/L} = 3.75 \text{ mg/L}$$

2. Secondary Maximum Concentration (SMC)

SMC = ½(SAV) = 1.875 mg/L

3. Secondary Acute-Chronic Ratio (SACR)

Values for acute and chronic tests are taken from Tables 1 and 2. Although acute and chronic tests were available for *Daphnia magna* and *Pimephales promelas*, several of the values are NOAELs rather than LOAELs, making the LOAELs "greater than" (">") values. The ACR of >32 mg/L/>3.9 mg/L for *Daphnia magna* is not quantifiable and therefore was not used. Only the chronic value for *Pimephales promelas* is a NOAEL and the resultant ACR (15 mg/L/>3.3 mg/L) value can be considered conservative (i.e., <4.545). Thus, only one of three required experimentally determined ACRs were available according to guidelines for Tier I. Therefore, two assumed ACRs of 18 were added. The SACR is the geometric mean of the three ACRs. Note that since the chronic value endpoints were not seen at the highest concentrations measured, some additional uncertainty in the SACR will result from the use of lower limit toxicity endpoints. (All decimal places were carried during calculations).

Pimephales promelas	15 mg/L/>3.3 mg/L = <4.545
Assumed ACRs	18, 18

SACR = Geometric mean of 4.545, 18, and 18 = 11.377

4. Secondary Chronic Value (SCV)

The SCV is equal to the SAV divided by the SACR

$$SCV = \frac{SAV}{SACR} = \frac{3.75}{11.377} = 0.3296 \text{ mg/L}$$

5. Secondary Continuous Concentration (SCC)

The SCC is equal to the lowest of three values: the SCV (0.33 mg/L), the Final Plant Value (>32 mg/L), and the Final Residue Value (not available). The SCV is lower than the Final Plant Value, so the SCC is equal to the SCV, 0.33 mg/L.

Although a SCC was calculated according to Tier II guidelines, it is overly conservative compared to measured concentrations that resulted in no adverse effects in chronic tests with *Daphnia magna* and *Pimephales promelas*. In the absence of data for calculation of a CCC or SCC, the U.S. EPA lists lowest-observed-effect levels (LOEC). A LOEC is not available from the chronic studies but would be greater than the chronic NOAELs of 3.3 and 3.9 mg/L. Therefore, the LOEC of >3.3 mg/L would be a more realistic interim value until additional toxicity tests are performed.

APPENDIX B

DERIVATION OF SEDIMENT QUALITY CRITERIA

B.1 General Methodology

For nonionic organic chemicals, the U.S. EPA has chosen the Equilibrium Partitioning (EqP) approach to develop Sediment Quality Criteria (SQC) for protection of benthic organisms. The basic assumptions, methodology, and uncertainties in the calculation are outlined in Di Toro et al. (1991) and U.S. EPA (1993a). The basic calculation uses the chronic WQC or FCV (a concentration deemed safe for aquatic organisms) together with correction factors for the effects of organic carbon, the predominant phase for sorption of hydrophobic chemicals. The procedure is as follows. The SQC is computed using the Water Quality Criterion Final Chronic Value (FCV in mg/L) and the partition coefficient K_p (L/kg sediment) between water and sediment.

$$SQC = K_p \times FCV$$

The partition coefficient is the ratio of the sediment concentration, C_s, to pore water concentration, C_d. The sorption capacity of the sediment is determined by the mass fraction of organic carbon in the sediment, f_{oc}, and is given by

$$K_p = \frac{C_s}{C_d} = f_{oc} \times K_{oc}$$

where K_{oc} is the partition coefficient for sediment organic carbon. The relationship applies to sediments with f_{oc} >0.2% by weight. U.S. EPA (1993a) recommends using a reliably measured octanol/water partition coefficient, K_{ow}, to estimate the K_{oc}. The K_{oc} may be estimated from the K_{ow} for the chemical using the following equation.

$$\log_{10}(K_{oc}) = 0.00028 + 0.983 \log_{10}(K_{ow})$$

Therefore, the SQC becomes

$$SQC = f_{oc} \times K_{oc} \times FCV$$

This equation is linear in the organic carbon fraction, f_{oc}, and the relationship can be expressed as

$$\frac{SQC}{f_{oc}} = K_{oc} \times FCV$$

If the organic carbon-normalized SQC, SQC_{oc}, is defined as SQC/f_{oc}, then

$$SQC_{oc} = K_{oc} \times FCV$$

B.2 Calculation of Sediment Quality Criteria

Although a FCV could not be calculated for HMX, the SCV an be substituted for the FCV, although with a lesser degree of certainty.

Because K_{ow} values are available, a K_{oc} can be calculated and a SQC_{oc} can be determined. Using the mean of the log K_{ow} values cited by Burrows et al. (1989) of 0.16 and the relationship between K_{ow} and K_{oc} above, the log K_{oc} is 0.1576 and the K_{oc} is 1.437. Then, the organic carbon-normalized SQC_{oc}, i.e., mg/kg organic carbon (mg/kg_{oc}) is

$$SQC_{oc} = 1.437 \text{ L/kg} \times 0.3296 \text{ mg/L} = 0.474 \text{ mg/kg}_{oc}$$

For sediment with a 1% organic carbon content, the above SQC_{oc} should be multiplied by the fraction organic carbon, i.e., the SQC for sediment with a 1% organic carbon content is 0.006 mg/kg:

$$SQC_{1\%} = 0.01 \times 1.437 \text{ L/kg} \times 0.3296 \text{ mg/L} = 0.005 \text{ mg/kg}$$

APPENDIX C

DERIVATION OF WILDLIFE BENCHMARKS

C.1 GENERAL METHODOLOGY

The general method used in estimating screening benchmarks for wildlife is based on EPA methodology for deriving human toxicity values (e.g., Reference Values, Reportable Quantities, and unit risks for carcinogenicity) from laboratory animal data (see Opresko et al., 1995). In this approach a NOAEL (no-observed-adverse-effect level) for population-related effects (e.g., survival, reproductive function) is identified from a study conducted with a species of wildlife or laboratory animals, and the equivalent NOAEL for other species of wildlife (wildlife NOAEL) is obtained by scaling the test data (test NOAEL) on the basis of differences in body size according to the following equation:

$$\textit{Wildlife NOAEL} = \textit{test NOAEL} \times \left[\frac{\textit{test bw}}{\textit{wildlife bw}}\right]^{1/4}$$

where: wildlife bw = body weight of wildlife species in kg
test bw = body weight of test species in kg
test NOAEL = experimental dose in mg/kg/day

Body weights for selected wildlife species are given in Table C-1; data sources are listed in Opresko et al. (1995).

In cases where only a LOAEL is available, the NOAEL is estimated as being equivalent to 1/10th of the LOAEL. If the only available data consists of a NOAEL (or a LOAEL) for a subchronic exposure (approximately 3 months to 1 yr), then the equivalent NOAEL or LOAEL for a chronic exposure is estimated as being 1/10th of the value for the subchronic exposure.

The dietary screening benchmark (Cf, the chemical concentration in food in mg/kg) that would result in a dose equivalent to the NOAEL (assuming no other exposure through other environmental media) is calculated from the food factor f, which is the amount of food consumed per unit body weight per day:

$$Cf = \frac{\textit{Wildlife NOAEL}}{f}$$

Food factors for the species of wildlife used in the calculations are shown in Table C-1; data sources are given in Opresko et al. (1995).

The drinking water screening benchmark (Cw, concentration of the chemical in mg/L) that would result in a dose equivalent to the NOAEL (assuming no other exposure through other environmental media) is calculated from the water factor ω, which is the amount of water consumed per unit body weight per day:

Water factors for the species of wildlife used in the calculations are shown in Table C-1.

$$Cw = \frac{Wildlife\ NOAEL}{\omega}$$

Table C-1. Reference Values for Selected Wildlife Species					
Species	Body Weight (kg)	Food Intake (kg/day)	Food Factor f	Water Intake (L/day)	Water Factor ω
short-tailed shrew	0.015	0.009	0.6	0.0033	0.22
white-footed mouse	0.022	0.0034	0.155	0.0066	0.3
meadow vole	0.044	0.005	0.114	0.006	0.136
cottontail rabbit	1.2	0.237	0.198	0.116	0.097
mink	1.0	0.137	0.137	0.099	0.099
red fox	4.5	0.45	0.1	0.38	0.084
whitetail deer	56.5	1.74	0.031	3.7	0.065

If a wildlife species (such as mink) feeds primarily on aquatic organisms, and the concentration of the contaminant in the food is proportional to the concentration in the water, then the wildlife NOAEL and body weight, its food intake rate (F, in kg/day) and water intake rate (W, in L/day), and the aquatic life bioaccumulation factor (BAF) can be used to derive an overall Cw that incorporates both water and food consumption (U.S. EPA 1993a; 1993c):

$$Cw = \frac{NOAEL_w \times bw_w}{W + (F \times BAF)}$$

The BAF is the ratio of the concentration of the contaminant in tissue (mg/kg) to its concentration in water (mg/L), where the organism and the prey are exposed. The BAF can be predicted by multiplying the bioconcentration factor (BCF), by the appropriate food chain multiplying factor (FCM).

$$BAF = BCF \times FCM$$

The FCM is a function of the log K_{ow} of the contaminant and the prey trophic level (see Table C-2). Because mink feed on small fish, the FCM for trophic level 3 was used in the calculation.

The BCF can be estimated from the log K_{ow} for the chemical by the following equation (Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} - 0.23$$

Table C-2. Aquatic Food Chain Multiplying Factors[a]			
	Prey Trophic Level[b]		
Log K_{ow}	2	3	4
≤ 3.9	1.0	1.0	1.0
4.0	1.1	1.0	1.0
4.1	1.1	1.1	1.1
4.2	1.1	1.1	1.1
4.3	1.1	1.1	1.1
4.4	1.2	1.1	1.1
4.5	1.2	1.2	1.2
4.6	1.2	1.3	1.3
4.7	1.3	1.4	1.4
4.8	1.4	.15	1.6
4.9	1.5	.18	2.0
5.0	1.6	2.1	2.6
5.1	1.7	2.5	3.2
5.2	1.9	3.0	4.3
5.3	2.2	3.7	5.8
5.4	2.4	4.6	8.0
5.5	2.8	5.9	11.0
5.6	3.3	7.5	16.0
5.7	3.9	9.8	23.0
5.8	4.6	13.0	33.0
5.9	5.6	17.0	47.0
6.0	6.8	21.0	67.0
6.1	8.2	25.0	75.0
6.2	10.0	29.0	84.0
6.3	13.0	34.0	92.0
6.4	15.0	39.0	98.0
6.5	19.0	45.0	100.0
>6.5	c	c	c

[a] From U.S. EPA 1993a.
[b] Trophic levels: 2 = zooplankton; 3 = small fish; 4 = piscivorous fish
[c] For chemicals with log K_{ow} > 6.5, FCM can range from 0.1-100; without chemical-specific data, an FCM of 1.0 should be used (U.S. EPA 1993a)

C.2 REFERENCE STUDIES FOR USE IN DERIVING WILDLIFE BENCHMARKS

Two studies are available for calculating toxicity benchmarks for wildlife for HMX. These studies are described below, with toxicity endpoint information summarized in Table C-3.

STUDY 1

Reference:	Everett, D.J., I.R. Johnson, P. Hudson and M. Jones. 1985. 13-Week Toxicity Study in Rats by Dietary Administration. Inveresk Research International, Musselburgh, Scotland. Final Report to the U.S. Army, AD A171602.
Test Species:	F344 rats
Exposure Duration:	13 weeks
Endpoint:	Liver (males) and kidney (females)
Exposure Route:	Oral (diet)
Dosage:	0, 50, 150, 450, 1350, or 4000 mg/kg/day in males and 0, 50, 115, 270, 620, or 1500 mg/kg/day in females.
Comments:	Male rats dosed with 150 mg/kg/day or higher exhibited hepatotoxic effects (necrosis, sinusoidal dilation and centrilobular hypertrophy). No adverse effects occurred at 50 mg/kg/day. Female rats dosed with 270 mg/kg/day or higher exhibited renal toxicity (areas of focal atrophy and tubular dilation). The mean body weight (geometric mean of weekly means) for males in the 50 mg/kg dose group was calculated to be 0.216 kg.
Subchronic NOAEL:	50 mg/kg/day (males)
Final Chronic NOAEL:	5 mg/kg/day (The subchronic NOAEL is multiplied by an uncertainty factor of 0.1 to derive the chronic NOAEL).

STUDY 2

Reference: Everett, D.J. and S.M. Maddock. 1985. 13-Week Toxicity Study in Mice by Dietary Administration. Inveresk Research International, Musselburgh, Scotland. Final Report to the U.S. Army, AD A171601.

Test Species: $B6C3F_1$ mice

Exposure Duration: 13 weeks

Endpoint: Survival

Exposure Route: Oral (diet)

Dosage: 0, 5, 12, 30, 75 or 200 mg/kg/day in males, and 0, 10, 30, 90, 250, or 750 mg/kg/day in females.

Comments: Mortality rates were 0/20, 1/20, 2/20, and 13/20 in males dosed with ≤ 12, 30, 75 and 200 mg/kg/day, respectively, and 1/20, 1/20, 12/20/ and 20/20 in females dosed with 0, 30, 250 and 750 mg/kg/day, respectively. It was reported that the deaths in the 30 mg/kg/day group were not related to exposure to HMX. No other adverse effects were reported. In the absence of chronic or multi-generational reproductive toxicity studies, the only available toxicity endpoint is the decrease in survival at dose levels > 30 mg/kg/day; therefore, 30 mg/kg/day can be considered a subchronic NOAEL.

The mean body weight (geometric mean of weekly means) for males in the 30 mg/kg dose group was calculated to be 0.025 kg, and that for females in the 30 mg/kg dose group was 0.023 kg.

Subchronic NOAEL: 30 mg/kg/day

Final Chronic NOAEL: 3.0 mg/kg/day (The subchronic NOAEL is multiplied by an uncertainty factor of 0.1 to derive the chronic NOAEL).

Table C-3. Toxicity Studies on HMX

Species	Route	Expos. Period	NOAEL mg/kg/day	LOAEL mg/kg/day	Effect/ Endpoint	Ref.
rat (males)	diet	13 wk	50	150*	hepatic effects	Everett et al., 1985
rat (females)	diet	13 wk	115	270	renal effects	Everett et al., 1985
mouse	diet	13 wk	30 (m) 30 (f)	75 (m) 250 (f)	mortality	Everett and Maddock, 1985

* No adverse effects on survival.

C.3 CALCULATION OF WILDLIFE BENCHMARKS FOR HMX

Of the two studies available for deriving wildlife benchmarks for HMX (see section C.2), the mouse study which identifies LOAELS and NOAEL's for mortality is more relevant for population level effects than that identifying liver and renal effects in rats. The subchronic NOAEL in the mouse study was 30 mg/kg/day and the estimated chronic NOAEL is 3.0 mg/kg/day.

Wildlife benchmarks for HMX were calculated using the methodology described in Appendix Section C.1. Examples of the methodology are shown below.

To calculate the chronic NOAEL for the meadow vole from the chronic NOAEL of 3.0 mg/kg/day for the laboratory mouse, the following equation was used:

$$\textit{Wildlife NOAEL} = \textit{test NOAEL} \times \left[\frac{\textit{test bw}}{\textit{wildlife bw}}\right]^{1/4}$$

where: wildlife bw = body weight of meadow vole = 0.044 kg
test bw = body weight of female laboratory mouse = 0.023 kg
test NOAEL = experimental dose = 3.0 mg/kg/day

(The mean body weight for the female mice was used because it provides a slightly more conservative set of benchmarks than the mean male body weight).

Therefore:

$$\textit{Meadow vole NOAEL} = 3.0\ \textit{mg/kg/day} \times \left[\frac{0.023\ \textit{kg}}{0.044\ \textit{kg}}\right]^{1/4} = 2.551\ \textit{mg/kg/day}$$

The food factor for meadow voles is 0.114; therefore, the dietary screening benchmark (Cf) is equivalent to:

$$Cf = \frac{\textit{Meadow vole NOAEL}}{f} = \frac{2.551}{0.114} = 22.38\ \textit{mg/kg food}$$

The water factor for meadow voles is 0.136; therefore, the screening benchmark for drinking water (Cw) is equivalent to:

$$Cw = \frac{\textit{Meadow vole NOAEL}}{\omega} = \frac{2.551}{0.136} = 18.76\ \textit{mg/L water}$$

To calculate a screening benchmark for HMX for a piscivorous species such as mink, the following equation is used:

$$Cw = \frac{NOAEL_w \ x \ bw_w}{W + (F \ x \ BCF \ x \ FCM)}$$

The chronic NOAEL for mink is derived from the chronic NOAEL of 3.0 mg/kg for the laboratory mouse:

$$Wildlife\ NOAEL = test\ NOAEL \ x \left[\frac{test\ bw}{wildlife\ bw}\right]^{1/4}$$

where: wildlife bw = body weight of mink = 1.0 kg
test bw = body weight of female laboratory mouse = 0.023 kg
test NOAEL = experimental dose = 3.0 mg/kg/day

Therefore:

$$Mink\ NOAEL = 3.0\ mg/kg/day \ x \left[\frac{0.023\ kg}{1.0\ kg}\right]^{1/4} = 1.168\ mg/kg/day$$

The BCF for HMX can be estimated from the log K_{ow} of 0.15 (see Section 3.2) by the following equation:

$$\log BCF = 0.76 \log K_{ow} - 0.23 = -0.116$$

$$BCF = 0.766$$

The log K_{ow} of 0.26 is also used to estimate the FCM from Table C-2. For this log K_{ow}, the FCM is 1.0. For mink with a body weight of 1.0 kg, a food consumption rate (F) of 0.137 kg/day, and a water ingestion rate (W) of 0.099 L/day, the overall screening benchmark for water (Cw) is:

$$Cw = \frac{1.168 \ x \ 1.0}{[0.099 + (0.137 \ x \ 0.766 \ x \ 1.0)]} = 5.73\ mg/L$$

The screening benchmarks for HMX for selected species of wildlife are shown in Table C-4.

Table C-4. Screening Benchmarks for HMX[a]

	Wildlife		Experimental Values			Extrapolated Values for Chronic Exposures			
							Screening Benchmarks		
Experimental animal (body weight)	Species	Body Wt. (kg)	LOAEL (mg/kg/day)	NOAEL[b] (mg/kg/day)	Endpoint	NOAEL[c] (mg/kg/day)	Diet (mg/kg food)	Water (mg/L)	Piscivorous species (mg/L)[d]
B6C3F$_1$ mice (0.023 kg)				30 (13 wk)	mortality	3.0			
	short-tailed shrew	0.015				3.34	5.56	15.17	
	white-footed mouse	0.022				3.03	19.6	10.11	
	meadow vole	0.044				2.55	22.4	18.76	
	cottontail rabbit	1.2				1.12	5.64	11.5	
	mink	1.0				1.17	8.50	11.8	5.73
	red fox	4.5				0.80	8.0	9.55	
	whitetail deer	56.5				0.43	13.75	6.56	

[a] For a detailed description of the methodology, see Opresko et al., 1995.
[b] Reference: Everett and Maddock, 1985.
[c] Values are for chronic exposures; NOAEL values have been rounded off, but non-rounded values were used to calculate benchmarks.
[d] Water concentration that incorporates dietary intake from both water and food consumption.