REF ID 0470

ECOLOGICAL CRITERIA DOCUMENT FOR HEXAHYDRO-1,3,5-TRINITRO-1,3,5-TRIAZINE (RDX)
(CAS No. 121-82-4)

May 1996

Prepared by

Sylvia S. Talmage, Ph.D., D.A.B.T.
Dennis M. Opresko, Ph.D.
Biomedical and Environmental Information Analysis
F. Michael Cretella, M.S.
Hazardous Waste Group
Risk Analysis Section
Health Sciences Research Division
Oak Ridge National Laboratory
Oak Ridge, Tennessee

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
and U.S. Department of Army.

Oak Ridge National Laboratory, managed by Lockheed Martin Energy Research Corp. for the U.S. Department of Energy under contact number DE-AC05-96OR22464.

9695

TABLE OF CONTENTS

EXECUTIVE SUMMARY iii

1. INTRODUCTION 1
2. ENVIRONMENTAL FATE 1
   2.1. SOURCES AND OCCURRENCES 1
      2.1.1. Air 1
      2.1.2. Surface Water, Groundwater, and Sediment 1
      2.1.3. Soil 3
   2.2. TRANSPORT AND TRANSFORMATION PROCESSES 4
      2.2.1. Abiotic Processes 4
      2.2.2. Biotransformation 5
3. AQUATIC TOXICOLOGY 7
   3.1. TOXICITY 7
      3.1.1. Acute Effects - Invertebrates and Fishes 7
      3.1.2. Chronic Effects - Invertebrates and Fish 7
      3.1.3. Plants 10
   3.2. METABOLISM AND BIOCONCENTRATION 12
   3.3. AQUATIC CRITERIA AND SCREENING BENCHMARKS 12
      3.3.1. Aquatic Organisms 12
      3.3.2. Sediment-Associated Organisms 15
4. TERRESTRIAL TOXICOLOGY 15
   4.1. TOXICITY 15
      4.1.1. Mammals 15
      4.1.2. Birds 17
      4.1.3. Plants 18
      4.1.4. Soil Invertebrates 18
      4.1.5. Soil Heterotrophic Processes 18
   4.2. METABOLISM AND BIOACCUMULATION 18
      4.2.1. Animals 18
      4.2.2. Plants 19
   4.3. TERRESTRIAL CRITERIA AND SCREENING BENCHMARKS 19
      4.3.1. Animals 19
      4.3.2. Plants 21
      4.3.3. Soil Invertebrates 21
      4.3.3. Soil Heterotrophic Processes 21
5. REFERENCES 22

APPENDIX A: DERIVATION OF AQUATIC BENCHMARKS A-1

APPENDIX B: DERIVATION OF AQUATIC BENCHMARKS B-1

APPENDIX C: DERIVATION OF WILDLIFE BENCHMARKS C-1

LIST OF TABLES

1. RESULTS OF ACUTE TOXICITY STUDIES WITH AQUATIC INVERTEBRATES AND FISH 8

2. RESULTS OF CHRONIC AQUATIC TOXICITY STUDIES 9

3. RESULTS OF TOXICITY TESTS WITH ALGAE 11

4. BIOCONCENTRATION FACTORS (BCF) IN AQUATIC SPECIES 13

5. WATER QUALITY CRITERIA/SCREENING BENCHMARKS FOR AQUATIC ORGANISMS 14

6. SCREENING BENCHMARKS FOR SELECTED MAMMALIAN WILDLIFE SPECIES 20

7. SCREENING BENCHMARKS FOR TERRESTRIAL PLANTS AND INVERTEBRATES 21

EXECUTIVE SUMMARY

RDX is a high-explosive, military-unique compound that has been released to the environment at military sites where it is manufactured, processed into weapons, stored, or destroyed through burning or detonation. RDX is a persistent chemical in the environment and can be found in soil, surface water, sediments and groundwater at Army Ammunition Plants (AAPs) and other military sites. Historical releases of RDX have occurred in discharges from the Holston AAP in Kingsport, Tennessee, the primary site of manufacture, and from numerous load and pack operations as well as during destruction and demilitarization operations. Effluent concentrations as high as 109 mg/L have been measured at the junction of effluents with on-site streams at AAPs. Stream sediment concentrations of 290 to 43,000 mg/kg have been measured at the Milan AAP. Concentrations entering off-site surface waters were usually < 1 mg/L. The maximum RDX concentration in an on-site storage lagoon was 29 mg/L; concentrations in lagoon sediments at other sites ranged up to 120,000 mg/kg. Concentrations monitored in groundwater at military sites ranged up to 36 mg/L (Iowa AAP). An off-site groundwater concentration of 100 μg/L was measured at the Cornhusker AAP. Concentrations in soil at open burning areas ranged up to 74,000 mg/kg, with highest concentrations in the residue and surface soil.

Direct photolysis is the primary degradation pathway for RDX in clear waters. Under experimental conditions, photolysis of RDX in water exposed to natural sunlight occurred with a half-live of several hours to <2 weeks. Photolysis products were formaldehyde, nitrite and nitrate ion, as well as nitroso compounds. However, dilution, not photolysis, is expected to be the major process by which RDX concentrations are reduced in opaque surface waters. RDX is resistant to aerobic bacterial degradation under laboratory conditions. Microbial degradation takes place under anaerobic conditions by reduction to nitroso- and hydroxylaminonitramines followed by cleavage reactions. Biodegradation products include hexahydro-1-nitroso-3,5-dinitro-1,3,5-triazine (MNX), hexahydro-1,3-dinitroso-5-nitro-1,3,5-triazine (DNX), hexahydro-1,3,5-trinitroso-1.3,5-triazine (TNX), hydrazine, 1,1-dimethyl-hydrazine, formaldehyde, and methanol. Although degradation occurs under anaerobic conditions in the laboratory, it probably is not an important fate process in the environment. Hydrolysis is not a major fate pathway at pH levels of natural waters.

When released to the environment, RDX shows moderate to high mobility and can be expected to leach into groundwater where it will persist. The environmental fate of RDX in soils varies with pH, texture, and organic content. A soil organic-carbon absorption coefficient (K_{oc}) of 42 to 538 and a soil absorption coefficient (K_p) of 2.17 have been measured or calculated.

Acute toxicity testing was conducted with four species of invertebrates and four species of fish. For invertebrates, 48-hour EC_{50} concentrations were > 100 mg/L under static conditions and > 15 mg/L under flow-through conditions. Fish appeared to be more sensitive than invertebrates. For fish, 96-hour LC_{50} values ranged from 4.1 to 6.4 mg/L under static conditions and 6.6 to 13 mg/L under flow-through conditions. The lowest measured LC_{50} was for bluegill sunfish (*Lepomis macrochirus*): 3.6 mg/L at a slightly acidic pH. Bioconcentration in aquatic organisms is not an important fate process as experimental Bioconcentration Factors in edible tissue of bluegill sunfish, channel catfish (*Ictalurus punctatus*), and fathead minnows (*Pimephales promelas*), were 3.5-9.0. A log K_{ow} of 0.87 also indicates little potential to bioconcentrate.

Data were not sufficient for calculation of aquatic Water Quality Criteria according to U.S. EPA guidelines. Therefore, Tier II or secondary values were calculated according to U.S. EPA guidance for

the Great Lakes System. Under these guidelines, the calculated acute criterion (Secondary Maximum Concentration) is 0.70 mg/L and the chronic criterion (Secondary Continuous Concentration) is 0.19 mg/L. Lowest chronic values for daphnids and fish, based on two-generation reproductive tests were 4.8 and 4.9 mg/L, respectively. The final plant value, based on a 96-hour study with a green alga (*Selenastrum capricornutum*) in which cell density was decreased by 17% was 3.2 mg/L. A preliminary organic carbon-normalized Sediment Quality Criterion (SQC_{oc}) of 1.3 mg RDX/kg organic carbon was calculated based on measured K_{ow} values and limited aquatic toxicity data.

Following ingestion by mammals, RDX is slowly but extensively absorbed, distributed throughout the body, and metabolized in the liver. Metabolism is extensive as indicated by excretion of 80% of a single ^{14}C-radiolabeled dose in the rat within four days: 43% as $^{14}CO_2$, 34% in the feces, and 3% in the urine.. No intermediate metabolites were identified. Extensive metabolism indicates that bioaccumulation will not occur.

Data are available on the toxicity of RDX to terrestrial animals and plants. Three chronic studies and one teratogenicity/reproductive toxicity study utilizing laboratory animals were located. From a two-year oral study with mice, a no-observed-adverse-effect level (NOAEL), based on testicular degeneration, of 7.0 mg/kg/day was identified. This value was used as the basis for calculation of NOAELs or 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. Equivalent NOAELs were calculated by scaling the test data on the basis of differences in body weight. NOAEL values ranged from 1.1 mg/kg/day for the whitetail deer to 8.7 mg/kg/day for the short-tail shrew. Based on food and water intakes for each species, NOAEL values for food ranged from 15 mg RDX/kg food for the short-tail shrew and cottontail rabbit to 58 mg RDX/kg food for the meadow vole. Screening benchmarks for water intake ranged from 17 mg/L for the whitetail deer to 49 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 6.7 mg/L.

Data indicate that RDX is taken up by terrestrial plants and only partially metabolized to unidentified metabolites. Bioaccumulation in the aerial parts of bush beans occurs with highest concentrations in the leaves and seeds. Biomass of cucumber plants was significantly reduced at a soil concentration of 100 mg RDX/kg soil. This value can be used as a toxicity screening value for terrestrial plants. No suitable data were located for deriving benchmark values for avian species, soil invertebrates, or soil heterotrophic processes, although a soil concentration of 500 mg/kg was not toxic to the earthworm, *Eisenia foetida*.

1. INTRODUCTION

Hexahydro-1,3,5-trinitro-1,3,5-triazine (CAS No. 121-82-4) is a crystalline high explosive used extensively by the military in shells, bombs, and demolition charges. It is commonly referred to as RDX (British code name for Research Department Explosive or Royal Demolition Explosive). Other synonyms are hexolite, cyclonite, hexogen, PBX (AF) 108, T4, cyclotrimethylenetrinitramine, trimethylenetrinitramine, and trinitrocyclotrimethylene. Military grades of RDX contain about 10% octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX).

This ecotoxicological profile on RDX summarizes the available data on concentrations in environmental media, environmental fate and transport, and ecotoxicity and bioaccumulation data for aquatic and terrestrial species. Ecotoxicological criteria and screening benchmarks for aquatic and terrestrial biota, based on population growth and survival effects, were derived for those groups of organisms for which there were sufficient data.

2. ENVIRONMENTAL FATE

2.1 SOURCES AND OCCURRENCES

RDX is a military-unique compound that is released to the environment at Army Ammunition Plants (AAPs) where it is manufactured. It is also released to the environment during conversion to munitions through a load, assemble, and pack (LAP) process, at military depot facilities, and through the demilitarization of obsolete munitions. As of 1992, ATSDR reported that RDX had been identified at 16 hazardous waste sites proposed for inclusion on the National Priorities List (ATSDR, 1993). In 1977, four facilities produced RDX, with the primary site of manufacture at Holston AAP, Kingsport, TN (U.S. EPA, 1989). As of 1989, Holston AAP was the only U.S. facility engaged in the manufacture of RDX (Burrows et al., 1989). Manufacture and LAP operations were reported at the Cornhusker AAP during the Korean War (Spalding and Fulton, 1988) and at the Iowa (Middletown, IA), Milan (Milan, TN), Louisiana (Shreveport, LA), and Lone Star (Texarkana, TX) AAPs during the 1970s (Ryon et al., 1984). In the 1970s, LAP operations were carried out in which RDX/HMX formulations were loaded into bombs and projectiles at 11 Army and Navy ammunitions plants (Sullivan et al., 1979). RDX-containing wastewaters are generated at both manufacturing and LAP plants. The major routes of environmental release are discharge in waste streams generated during manufacture and processing, leaching from wastewater storage lagoons or waste burial areas into groundwater, and demilitarization operations. Sampling studies were conducted at various sites over a number of years by different investigators. Following are some of the available data taken from published and unpublished reports.

2.1.1 Air

No data on the presence of RDX in outdoor air were located. A very low vapor pressure, 1.0 x 10^{-9} mm Hg (Layton et al., 1987) to 4.0 x 10^{-9} mm Hg (Burrows et al., 1989), indicates a low potential to enter the atmosphere or to be dispersed by volatilization.

2.1.2 Surface Water, Groundwater, and Sediment

RDX can be released to surface water in wastewater discharges from production and LAP facilities, and through overflow of waste lagoons and storage areas. Although of low to moderate water

solubility, RDX also has a low adsorption coefficient for soils and sediments and thus will migrate into groundwater.

In 1980, the amount of RDX discharged from the Holston AAP was estimated to be 69.5 kg/day (Spanggord et al., 1980b). Concentrations of nitramines (RDX + HMX) in effluents averaged 2-6 mg/L (Burrows et al., 1989). RDX concentrations in untreated wastewaters ranged from < 0.05 (the limit of detection) to 4.75 mg/L, whereas treated wastewaters contained < 0.05 to 0.7 mg/L (Stilwell et al., 1977). Concentrations were measured in the Holston River downstream of the plant by several investigators. Concentrations ranged from 0.7 mg/L immediately below the plant outfall to < 0.005-0.07 mg/L approximately one mile downstream (Sullivan et al., 1979). Stidham (1979) reported a concentration of 0.079 mg/L one mile downstream of the last plant effluent. Concentrations measured at transect lines across the river at 0.1, 4.5, 8, and 33 km were 0.4-224, 3-24, 4-17, and 4-8 μg/L, respectively (Spanggord et al., 1981). The data indicate that at the initial point of discharge, most of the effluent remained close to the river bank (224 μg/L) with mixing occurring downstream resulting in dilution and smaller ranges of concentrations. In the same study, the sediment concentration at 0 km was 358 ng/g (μg/kg); concentrations in sediment were below the limit of detection at the downstream sites.

Concentrations entering a stream on the Milan AAP site ranged from 0.1 to 109 mg/L with a mean of 11.9 mg/L (Spanggord et al., 1978). Stream water concentrations at the same site ranged from < 0.4 to 110 μg/L with sediment concentrations of 290 to 43,000 mg/kg (Envirodyne Engineers, Inc., 1980). Concentrations in a pink water lagoon at the Milan AAP ranged from < 4 to < 1600 μg/L with sediment concentrations of 2600 to 38,000 mg/kg (Envirodyne Engineers, Inc., 1980). Groundwater samples taken below soil containing 0.05 to 83 mg/kg showed concentrations in 4 of 39 samples of < 20 to 780 μg/L (Envirodyne Engineers, Inc., 1980). The maximum concentrations found in groundwater at this site was 30.0 mg/L (Tucker et al., 1985).

Effluent concentrations at the Iowa AAP ranged from 0.1 to 24 mg/L with a mean of 1.5 mg/L (Spanggord et al., 1978) and concentrations in Brush Creek ranged from 0.1 to 15 mg/L (Small and Rosenblatt, 1974). The maximum concentration found in groundwater at the Iowa AAP was 36.0 mg/L (Tucker et al., 1985).

RDX concentrations in water from an inactive lagoon at the Louisiana AAP ranged from 5.6 to 28.9 mg/L; sediment below several lagoons contained 400-120,000 mg/kg, with the highest concentration within the top 0.05 m (Spanggord et al., 1983). Concentrations in groundwater underlying several areas were: < 1.0 to 14,120 μg/L (Area P leaching pits), < 1.0 to 707 μg/L (burning ground #5), 8.3 μg/L (landfill #3), and < 1.0-537 μg/L (landfill area #8) (Gregory and Elliott, 1987). Groundwater below several waste disposal areas was also sampled for RDX by Todd et al. (1989). RDX was detected in 6 of 11 wells below an area of pink water leaching lagoons; concentrations ranged from 13 to 27,000 μg/L. The maximum concentration found in groundwater at the Louisiana AAP was 17.8 mg/L (Tucker et al., 1985).

Spalding and Fulton (1988) summarized the occurrences of several munition residues in the groundwater east of the Cornhusker AAP near Grand Island, NB. In a previous study cited in this paper, on-site groundwater downgradient of the site contained about 300 μg/L RDX; the off-site plume had concentrations > 35 μg/L. In 1988, the plume of RDX-contaminated groundwater was 6.5 km long and 1.6 km wide with off-site concentrations up to 100 μg/L. The estimated transport velocity was 0.5 m/day. Concentrations in sediments taken from leaching pits at the site ranged from 2 to 40 mg/kg (Rosenblatt, 1986).

Concentrations at the surface of a dry lagoon at the Savanna Army Depot ranged from 3000-4000 mg/kg (Rosenblatt, 1981). At the Lone Star AAP, Texas, sludge from below pink water settling ponds contained up to 50,000 mg/kg (Phung and Bulot, 1981).

Concentrations of < 20 to > 700 μg/L were found in groundwater in the vicinity of contaminated sites by Burrows et al. (1989). Concentrations in sediment below a pond-like structure used to trap washwater from a U.S. Navy facility (Kitsap County, WA) where projectiles were cleaned ranged from less than the limit of detection to 5.5 mg/kg; concentrations in the water table below the sediments ranged from less than the limit of detection to 5 mg/L (Goerlitz, 1992). The facility was active from 1966 to 1970 and the survey was taken in 1974. At an unidentified munitions disposal site, RDX was detected in 7 of 7 groundwater wells at concentrations of 1 to 47 μg/L (Richards and Junk, 1986). It was present in groundwater from a water supply well at an unspecified AAP at a concentration of 70 μg/L (Jenkins et al., 1986).

RDX was not detected in ocean waters near ocean dumping sites for waste munitions (Hoffsommer and Rosen, 1972) nor in ocean floor sediments near dumping sites (Hoffsommer et al., 1972).

2.1.3 Soils

Contamination of soil results from spills (mainly at military facilities), internment of RDX contaminated wastes in landfills, and open burning/detonation operations.

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 RDX in excess of the detection limit (0.5 ppm) were found at 11 of these sites; levels ranged from 0.5-1247 mg/kg (median 19.5) at Nebraska Ordinance Works, 0.5-12,203 mg/kg (median 38.6) at Newport, IN, 0.5 mg/kg (1/29 samples) at Weldon Springs Training Area (MO), 97.4-13,900 mg/kg (median 7000) at Iowa AAP, 0.5-4.38 mg/kg (median 2.4) at Raritan Arsenal (NJ), 2.6-8112 mg/kg (median 127) at Hawthorne AAP (NV), 0.5 mg/kg (1/24 samples) at Hastings East Park (NB), 139-616 mg/kg (median 378) at Milan AAP, 185-972 mg/kg (median 578) at Louisiana AAP, 0.5-0.05 mg/kg at Eagle River Flats (AK) explosive ordnance disposal and impact area, and 0.5-3.83 mg/kg (median 2.2) at Camp Shelby (MO) explosive ordnance disposal site and impact area.

As part of a study to evaluate the toxicity of Joliet AAP soils, Simini et al. (1995) determined RDX concentrations in soil taken from LAP and munition burning areas. RDX concentrations ranged from less than the limit of detection (0.1 mg/kg) to 3574 mg/kg, with levels exceeding the detection limit in 13 of 40 samples. Concentrations at open burning sites at the Holston AAP reached 70-80 mg/kg of soil (Bender et al., 1977). Funk et al. (1993) reported that soil samples from an Army munitions depot near Umatilla, OR contained 3000 mg RDX/kg soil. A soil sample taken from an ordnance-burning area at the Milan AAP contained 39 mg RDX/kg soil (Jenkins and Grant, 1987). Four soil samples taken from the Naval Surface Warfare Center, Crane, IN, contained concentrations ranging from below the limit of detection to 3.3 mg/kg (dry weight) (Grant et al., 1995).

Newell (1984) surveyed open burning and open detonation sites at several AAPs and depots. Concentrations in residues and soil (surface to 18 inches in depth) at the Holston, Iowa, Kansas, Louisiana, Ravenna, Fort Wingate, and Milan AAPs and the Picatinny Arsenal ranged from 1200 to 74,000 mg/kg, with highest concentrations in the residue and soil surface.

2.2 TRANSPORT AND TRANSFORMATION PROCESSES

2.2.1 Abiotic Processes

Air. Because of its low vapor pressure, any RDX in the atmosphere would exist in the particulate form (U.S. EPA, 1989). Although RDX was not reported in air samples, any RDX entering the atmosphere would be rapidly degraded. Photolysis of RDX is expected to be an important fate process in the atmosphere since RDX absorbs ultraviolet wavelengths between 240-350 nm (Etnier, 1986) and it undergoes rapid photolysis in water (Sikka et al., 1980; Spanggord et al. 1980b). RDX in the atmosphere may also be degraded by reaction with photochemically generated hydroxyl radicals (Atkinson, 1987).

Water. With a reported solubility of 38.4 mg/L at 20°C (Spanggord et al. 1983), RDX is of low to moderate solubility in water. An estimated Henry's law constant of 1.2×10^{-5} atm-m^3/mole (McKone and Layton, 1986) indicates slow to moderate volatilization from water. At neutral or acidic pH values, RDX does not hydrolyze to an appreciable extent in freshwater or seawater. Reported hydrolysis half-lives of 170 days to several years indicate that hydrolysis is not a significant fate mechanism in natural waters (Sikka et al., 1980; Spanggord et al., 1980a). Approximately 12% of a 56 mg/L solution in seawater (pH approximately 8) degraded in 112 days (Hoffsommer and Rosen, 1973).

Photolysis is the primary abiotic degradation mechanism in translucent waters. Spanggord et al. (1980b) measured half-lives for RDX (1.3 mg/L) of 13, 14, and 9 days in distilled water, Holston River water and pond water, respectively, under natural winter conditions in Menlo Park, CA. During a sunny period in March, the half-life of RDX in distilled water was 1.8 days. The half-life was 12 days for RDX in a 4-cm deep dish of water from a lagoon at the Louisiana AAP (Spanggord et al., 1983). The presence of photoproducts and humic substances did not accelerate the photolysis rate. In another study, RDX in wastewater (23.9 mg/L) exposed to ultraviolet radiation decomposed with a half life of 3.7 minutes (Burrows et al. 1984), while photolysis of an aqueous solution of RDX in natural sunlight decomposed with an experimental half-life of 9-13 hours. Similar experiments by Sikka et al. (1980) confirmed the half-life of less than one day in surface water exposed to sunlight. Consequently, RDX is not expected to persist for a long period of time in surface waters. Photolysis results in the formation of formaldehyde, nitrite and nitrate ions, and nitroso compounds (Kubose and Hoffsommer, 1977; Spanggord et al., 1980b; Burrows et al., 1989).

In a simulation study of the fate of RDX in the Holston River, the major fate process affecting persistence was expected to be dilution (Spanggord et al., 1980b). The photolysis half-life was estimated at 3 days on a sunny day and up to 14 days in winter. Concentrations in the Tennessee River at Knoxville where the Holston River joins the Tennessee River were estimated at between 1 and 3 μg/L.

Soil and Sediment. Sikka et al. (1980) measured the adsorption of RDX on three sediment types and reported partition coefficients (K_p) of 0.80 for sandy loam, 3.06 for clay loam, and 4.15 for organic clay. Calculated K_{oc} values for the respective sediment types were 7.7, 74.6, and 266.7. Even with such low adsorption, steady-state levels of RDX between 30 and 40 ppm were reported for organic and clay sediments in laboratory evaluations.

Tucker et al. (1985) conducted soil sorption experiments with 12 soils of low organic carbon content. K_p values were estimated from regression equations, with independent variables consisting of the organic content of the soil, cation exchange capacity, pH, and silt content. They found that an

organic content of at least 0.25% by weight was necessary for adsorption to be an important process affecting migration. The authors derived a K_{oc} value of 136 from the measurements with the different soils. Using sterilized sediments taken from the Holston River, Spanggord et al. (1980b) calculated adsorption coefficients (K_p) of 4.2 and 1.4 and associated K_{oc} values of 127 and 42. Burrows et al. (1989) reported a K_{oc} of 100 for RDX in soil (type unspecified).

The transport of pink water compounds including RDX (30 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. In columns not amended with glucose, RDX was not bound and rapidly appeared in leachates. Some degradation, as indicated by decreased recovery, took place in columns with slow flow rates and those amended with glucose.

Soil migration was also studied utilizing both 152 cm deep x 90 cm wide cylinders and 5 cm x 61-cm deep lysimeters, the latter containing ^{14}C-labeled RDX, in a controlled greenhouse experiment (Hale et al., 1979; Kayser and Burlinson, 1988). The soils (four types ranging from silty clay to sandy loam with organic carbon content ranging from 0.39 to 2.2%) were irrigated regularly. After 6 months, concentrations in the large cylinders ranged from the treatment level of 20,000 mg/kg in the upper 10 cm to 150 mg/kg at the 60 to 90 cm depth. RDX was also found at the bottom of the columns. Levels of RDX in water leachates from both experiments were <5 mg/L except in the lysimeters containing coarse, loamy soil where they ranged up to 40 mg/L. Measured K_d values for the four soils were 7.8, 6.4, 0.2, and 1.8 (Hale et al., 1979).

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. RDX was measurable in leachates throughout the study and averaged 12 mg/L. RDX was transported so rapidly in the soil column that it was found at all depths by the first (6.5 week) sampling.

The above studies indicate that adsorption to soil is not a significant fate mechanism for RDX; likewise, sediment sorption will not lead to significant loss from the aquatic environment. The geometric mean of the K_d values from the studies of Hale et al. (1979), Spanggord et al. (1980b), Sikka et al. (1980), and Tucker et al. (1985) was calculated by Layton et al. (1987) to be 2.17.

Twenty years after burial in outdoor soil plots at Los Alamos, NM, approximately 70% of the original concentration of 1 g/kg remained (DuBois and Baytos 1991). A half-life of 36 years for RDX buried in soil was determined.

2.2.2 Biotransformation

The biodegradation of RDX has been studied under aerobic and anaerobic conditions. The data suggest that RDX is resistant to aerobic biodegradation as, with the exception of special conditions, it undergoes no or limited aerobic biodegradation when tested using a variety of inoculums, nutrients, and

microbial sources (Osmon and Klausmeier, 1972; McCormick et al., 1981; Spanggord et al., 1980b; Spanggord et al., 1983). Adaptation of microbes to RDX appears to be a necessary precondition for aerobic biodegradation to take place.

Spanggord et al. (1980b) conducted a biotransformation screening test with Holston River water. RDX (10 ppm) was not degraded during 78 days of incubation. The addition of 30 ppm yeast extract did not significantly affect the disappearance. When Holston River sediment was added to the solutions, RDX remained unchanged for 20 days and was then reduced to 4 ppm between days 20 and 36 followed by no change during the next two weeks. Sikka et al. (1980) also found that, following a lag period, mineralization occurred in Holston River water to which sediment had been added. In another trial experiment, no degradation of RDX was observed during a 90-day aerobic experiment in which RDX was placed in lagoon water alone, with added yeast extract, or with 1% of bottom sediment (Spanggord et al., 1983). Concentrations also remained unchanged when cultures were inoculated with aerobic activated sludge and incubated aerobically (McCormick et al. 1981). These data suggest that RDX is resistant to biotransformation under aerobic conditions.

The degradation of pink water compounds in soil columns was studied by Greene et al. (1984). A pink water solution containing 30 mg/L RDX was continuously applied to a series of soil columns at different flow rates, with and without carbon supplementation. The soil columns were filled with garden soil and inoculated with microorganisms from activated sludge, anaerobic sludge digest, and garden soil. There was a significant decrease in RDX recovery in the leachate of the columns with the slowest and fastest flow rates, indicating microbial activity. Some biodegradation took place as indicated by the identification of hexahydro-1-nitroso-3,5-dinitro-1,3,5-triazine (MNX) and hexahydro-1,3-dinitroso-5-nitro-1,3,5-triazine (DNX) in the leachates. When Palouse soil was amended with ^{14}C-labeled-RDX and sampled 60 days later, only RDX was recoverable; $^{14}CO_2$ accounted for only 0.31% of the amended radiolabel (Harvey et al., 1991).

Cataldo et al. (1993) treated soils with different amounts of organic matter with 60 ppm of RDX. After 60 days, >95% of the RDX was extractable as the parent compound with <2% being non-extractable. No significant transformation products were identified.

In specific inoculum testing of aqueous solutions, a mixed population of purple photosynthetic bacteria of the genera *Chromatium*, *Rhodospirillum*, and *Rhodopseudomonas*, and possibly others degraded 97% of a RDX solution in 5 days (Soli, 1973). In specific genera testing, 60% of the biodegradation activity was attributable to *Chromatium* sp. alone. These cultures did not release oxygen, and it was hypothesized that degradation was due to electron transfer rather than metabolism.

Data are available indicating that biodegradation of RDX occurs primarily under anaerobic conditions. RDX (30 mg/L) when added to Holston River water along with yeast extract and incubated in anaerobic flasks was reduced to <0.1 mg/L within 10 days (Spanggord et al, 1980b). No transformation was observed in flasks without added yeast extract. Additional studies determined that yeast extract serves as a cometabolic substrate. Using lagoon water alone or lagoon water with sediment from the Louisiana AAP, anaerobic incubation of RDX with yeast extract resulted in transformation only after an 80-90 day acclimation period (Spanggord et al., 1983).

McCormick et al. (1981, 1984) incubated RDX (50 or 100 ug/mL) anaerobically in nutrient broth cultures inoculated with anaerobic sewage sludge. RDX disappeared rapidly and biodegradation was complete after 4 days. The disappearance of RDX was accompanied by the appearance of several

products identified as the mono-, di-, and trinitroso RDX derivatives formed by sequential reductions of the nitro groups to nitroso groups. The metabolites formed were MNX, DNX, hexahydro-1,3,5-trinitroso-1,3,5-triazine (TNX), hydrazine, 1,1-dimethyl-hydrazine, 1,2-dimethyl-hydrazine, formaldehyde, and methanol.

3. AQUATIC TOXICOLOGY

3.1 TOXICITY

3.1.1 Acute Effects - Invertebrates and Fish

Results of acute tests conducted under static and flow-through conditions with four species of invertebrates and four species of fish are reported in Table 1. These tests were conducted under U.S. EPA (1975) guidelines available at that time. EC_{50} values, based on immobilization, were > 100 mg/L for invertebrates under static conditions, whereas values were lower under flow-through conditions, > 15 mg/L for the water flea, *Daphnia magna*, and the midge, *Chironomus tentans*. For fish, LC_{50} values ranged from 4.1 mg/L to 13 mg/L, with values slightly higher under flow-through conditions. Under static test conditions, the channel catfish (*Ictalurus punctatus*) was the most sensitive species with an LC_{50} value of 4.1 mg/L. Bentley et al. (1977) also explored the variation of acute toxicity with water quality parameters including temperature, pH, and hardness on the LC_{50} for the bluegill sunfish (*Lepomis macrochirus*) and found essentially no difference. The lowest 96-hour LC_{50} for bluegill sunfish was 3.6 mg/L at a temperature of 20°C, pH of 6.0, and water hardness of 35 mg/L $CaCO_3$.

Several different life stages of the fathead minnow (*Pimephales promelas*) were tested for 96 hours in static tests (Bentley et al., 1977). The egg and 1-day post-hatch fry were the least sensitive stages tested and the 7-day post-hatch fry was the most sensitive stage tested, with an LC_{50} of 3.8 mg/L (Table 1).

3.1.2 Chronic Effects - Invertebrates and Fish

Bentley et al. (1977) conducted chronic tests with two species of invertebrates and two species of fish. Results are reported in Table 2. The protocol included both concurrent controls and vehicle controls (dimethylsulfoxide used to dissolve the RDX). RDX had almost no effect on survival of the parent generation of *Daphnia magna* when tested at mean measured concentrations of 1.4, 2.2, 4.8, 9.5, or 20 mg/L. But the mean number of young produced per parthenogenic female exposed to concentrations of 4.8, 9.5, and 20 mg/L was significantly reduced between 7 and 14 days. At the lower concentrations from day 7 to 14 and at all concentrations from days 14 to 21 and days 28 to 42 (second generation), the daphnid production rate was comparable to controls. However, problems with maintaining RDX concentrations during days 7-14 and borderline acceptable survival of controls (78-82%) render the data from this part of the study of limited value.

The midge, *Chironomus tentans*, was tested at concentrations of 1.3, 2.2, 4.0, 10, and 21 mg/L. RDX had no statistically measurable effects on the larvae, pupae, adult survival or adult emergence in the first generation. However, the average number of eggs produced per adult (treated throughout the larval and pupal stage) was greatly reduced compared with controls, with no eggs produced at the 10 mg/L concentration and no fertile eggs at the 1.3 and 4.0 mg/L concentrations. During a second-generation exposure, eggs from the control containers were substituted in the 1.3, 4.0 and 10 mg/L

TABLE 1. RESULTS OF ACUTE TOXICITY STUDIES WITH AQUATIC INVERTEBRATES AND FISH

Test Species	Test Duration (Test Type)[a]	EC_{50} or LC_{50} (mg/L)[b,c]	Reference
Daphnia magna (water flea)	48-hr (s) 48-hr (ft)	> 100 > 15	Bentley et al., 1977
Gammarus fasciatus (scud)	48-hr (s)	> 100	Bentley et al., 1977
Asellus militaris (sowbug)	48-hr (s)	> 100	Bentley et al., 1977
Chironomus tentans (midge)	48-hr (s) 48-hr (ft)	> 100 > 15	Bentley et al., 1977
Ictalurus punctatus (channel catfish)	96-hr (s) 96-hr (ft)	4.1 13	Bentley et al., 1977
Lepomis macrochirus (bluegill sunfish)	96-hr (s) 96-hr (ft)	6.0 7.6	Bentley et al., 1977
Lepomis macrochirus (bluegill sunfish)	96-hr (s)	3.6-8.4[d]	Bentley et al., 1977
Pimephales promelas (fathead minnow)	96-hr (s) 96-hr (ft)	5.8 6.6	Bentley et al., 1977
Pimephales promelas (fathead minnow)			
eggs	96-hr (s)	> 100	Bentley et al., 1977
1-hr post-hatch fry	96-hr (s)	43	Bentley et al., 1977
7-day post-hatch fry	96-hr (s)	3.8	Bentley et al., 1977
30-day post-hatch fry	96-hr (s)	16	Bentley et al., 1977
60-day post-hatch fry	96-hr (s)	11	Bentley et al., 1977
Pimephales promelas (fathead minnow)	96-hr (s)	4.5	Liu et al., 1983
Oncorhynchus mykiss (rainbow trout)	96-hr (s)	6.4	Bentley et al., 1977

[a] Test type: s = static; ft = flow-through.
[b] EC_{50} values are for immobilization of invertebrates; LC_{50} values are for fish mortality.
[c] Concentrations are nominal.
[d] Tested at temperatures of 15-20°C, pH of 6.0-8.0, and water hardness of 35-250 mg/L $CaCO_3$.

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

Species	Stage/Age	Parameter Measured	Parameter Response	Concentration (mg/L)	Reference
Daphnia magna (water flea)	Neonates through generation two	21-day survival (generation one) Reproduction	No effect Significant decrease, days 7-14 of first generation	1.4-20 4.8-20	Bentley et al., 1977
Chironomus tentans (midge)	Neonates through generation two	23-day survival/emergence (generation one) Reproduction (21 days)	No effect Lower larval survival Lower egg production	1.3-21 1.3-21 1.3-10	Bentley et al., 1977
Ictalurus punctatus (channel catfish)	Eggs, fry	Mean hatch 30-day fry survival 30-day fry length	No effect No effect No effect	0.11-2.3 0.11-0.71[c] 0.11-2.3	Bentley et al., 1977
Pimephales promelas (fathead minnow)	Eggs, fry	Mean hatch 30-day fry survival 30-day fry length	No effect No effect Reduced	0.26-5.8 0.26-5.8 5.8	Bentley et al., 1977
Pimephales promelas (fathead minnow)	Eggs, fry, adults	Percent hatch 30-day fry survival, length 60-day fry survival 30, 60-day fry survival Spawning of 1st generation 2nd-generation parameters	No effect No effect Reduced Reduced No effect No effect	0.29-6.3 0.29-4.3 4.9 6.3 6.3 6.3	Bentley et al., 1977

[a] Flow-through conditions.
[b] Mean measured concentrations except 30-day egg/fry tests which utilized measured concentration for 3/5 treatments.
[c] Equipment failure at higher concentrations.

concentrations. No significant differences from controls were observed in percent survival of pupae at all levels and in survival of adults at the 2.2, 10, and 21 mg/L exposures. A significant reduction in second-generation adult emergence at 2.2 mg/L, in larval survival at all concentrations, and in adult survival at 1.3, and 4.0 mg/L was not conclusively related to RDX exposures, as second-generation studies at 1.3, 4.0, and 10 mg/L were initiated with control eggs. Furthermore, no effects occurred at these concentrations in the first generation.

Thirty-day tests were conducted with early life stages of fish by Bentley et al. (1977). Channel catfish were tested at concentrations of 0.11, 0.30, 0.71, 1.2, and 2.3 mg/L and fathead minnows were tested at concentrations of 0.26, 0.76, 1.2, 3.0, and 5.8 mg/L. For channel catfish, the tested concentrations had no effect on hatching or fry length at 30 days; poor fry survival at 30 days at the two highest concentrations was attributed to equipment malfunction. For fathead minnows, mean hatch and 30-day survival were not affected by concentrations of 0.26-5.8 mg/L; fry length at 30 days was significantly reduced at 5.8 mg/L.

Chronic tests with fathead minnows also resulted in minimal effects (Bentley et al., 1977). Fish were tested at concentrations of 0.29, 0.64, 1.1, 2.7, and 4.9 mg/L for 140 days and at concentrations of 0.43, 0.78, 1.5, 3.0, and 6.3 mg/L for 240 days. In the first test, concentrations as high as 4.9 mg/L had no significant effect on the percentage of fry hatching or on fry survival and total length during the initial 30 days of exposure. After 60 days, survival of fry exposed to 4.9 mg/L RDX was statistically significantly lower than survival of controls or lower treatment groups, but total length at 60 days was unaffected. In the second test, percentage hatchability was not affected at any concentration but after 30 and 60 days, survival of fry exposed to 6.3 mg/L was significantly lower than control survival. Fry length was unaffected by treatment after 30 and 60 days. After 240 days, total lengths and wet weights of mature male and female fathead minnows were not affected by exposure to concentrations of RDX as high as 6.3 mg/L. The spawning activity, percentage hatchability, and number of eggs were essentially unchanged by the presence of RDX in concentrations ≤ 6.3 mg/L. The second generation of fathead minnows also exhibited no measurable effects after 30 days of exposure to RDX at concentrations ranging up to 6.3 mg/L.

3.1.3 Plants

Bentley et al. (1977) examined the toxicity of RDX to two species of bluegreen algae, *Microcystis aeruginosa* and *Anabaena flos-aquae*; the green alga, *Selenastrum capricornutum*; and a species of diatom, *Navicula pelliculosa*, in static tests at nominal concentrations of 0.32 to 32 mg/L. Results are presented in Table 3. Parameters measured were chlorophyll *a* content and cell numbers. Although Bentley et al. (1977) reported 96-hr EC_{50} values of >32 mg/L, all four species showed significant decreases in chlorophyll *a* content and cell density, especially at the higher concentrations. Sullivan et al. (1979) re-examined the test with *Selenastrum capricornutum* and reported a significant reduction of chlorophyll *a* content at all concentrations. However, although statistically significant, the biological significance of a 3% decrease (at a concentration of 0.32 mg/L) is questionable. Therefore, effect levels reported in Table 3 are $\geq 10\%$ changes in the reported parameters. *Selenastrum capricornutum* was the most sensitive species with a 16% reduction in chlorophyll *a* content at 1.0 mg/L and a 17% decrease in cell density at 3.2 mg/L.

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

Test Species	Test Duration	Effect	Concentration (mg/L)	Reference
Anabaena flosaquae (blue-green algae)	96-hr	Decrease in cell density (17%) Decrease in chlorophyll a content (17%)	32 32	Bentley et al., 1977
Microcystis aeruginosa (blue-green algae)	96-hr	Decrease in cell density (18%) Decrease in chlorophyll a content (11%)	32 10	Bentley et al., 1977
Navicula pelliculosa (diatom)	96-hr	Decrease in cell density (17%) Decrease in chlorophyll a content (23%)	32 32	Bentley et al., 1977
Selenastrum capricornutum (green algae)	96-hr	Decrease in cell density (17%) Decrease in chlorophyll a content (16%)	3.2 1.0	Bentley et al., 1977

[a] All tests were conducted under static conditions.
[b] Nominal concentrations.

3.2 METABOLISM AND BIOCONCENTRATION

Studies on the metabolism of RDX by aquatic organisms were not located in the available literature. Bentley et al. (1977) calculated the bioconcentration of RDX in bluegill sunfish, channel catfish, and fathead minnows exposed continuously for 28 days to ^{14}C-labelled RDX in an intermittent-flow system (Table 4). Mean measured concentrations were 0.014 and 1.0 mg/L. Tissues were wet-weighed, air-dried, and combusted, with the resulting $^{14}CO_2$ trapped. Bioconcentration of ^{14}C-labelled RDX residues in the edible tissues and viscera of these species reached a steady state after 14 days of exposure. Uptake in viscera was about 2 to 3 times that found in muscle and the ranges of BCFs in muscle and viscera for all fish were 2.9 to 5.9 and 3.3 to 11, respectively. The mean BCF values for muscle at the two tested concentrations for channel catfish, bluegill sunfish, and fathead minnows were 3.4, 4.1, and 5.0, respectively. It was also noted that elimination of all RDX from the tissues at the lower concentration (0.014 mg/L) occurred after 14 days in the bluegill sunfish and catfish, but did not occur totally in the fathead minnow. At the higher dose, minnows and catfish eliminated 70-87% of the accumulated RDX, but virtually no elimination occurred from either muscle or viscera in the bluegill sunfish.

Bioconcentration was also studied by Liu et al. (1983) using four aquatic species. Tests were performed using a 4-day static exposure to ^{14}C-labelled RDX at a concentration of 0.3 mg/L. Bioconcentration factors were estimated using the average amount of radioactivity recovered (disintegrations/min/g) in tissue and water. The reported 4-day BCFs are listed in Table 4. The 4-day BCF calculated for bluegill sunfish muscle (1.9) and viscera (3.1) are somewhat lower than the 3-day BCF reported by Bentley et al. (1977), i.e., 3.5 and 9.5, respectively (data not shown), when tested at a concentration of 0.014 mg/L, and 4.3 and 13, respectively, when tested at a concentration of 1.0 mg/L. Based on log K_{ow} values of 0.81-0.87, Burrows et al. (1989) calculated a BCF of 1.5 for fish.

3.3 AQUATIC CRITERIA AND SCREENING BENCHMARKS

3.3.1 Aquatic Organisms

With one exception, the acute toxicity studies were all conducted by the same investigator. In the case of *Pimephales promelas*, the two static 96-hour toxicity tests conducted by separate investigators yielded similar results. The chronic studies, unfortunately, suffered from reported equipment failures and, at times, inconsistent results. Nevertheless, because most of the available data were consistent, they were used to estimate aquatic criteria. These estimates can be used as screening benchmarks for potential adverse effects on aquatic organisms (Suter and Mabrey, 1994).

Data were insufficient for calculation of acute and chronic Water Quality Criteria (WQC), i.e. a Criterion Maximum Concentration (CMC) and a Criterion Continuous Concentration (CCC), according to U.S. EPA guidelines (Stephan et al., 1985). Data are available for only six of the required eight families: a planktonic crustacean (*Daphnia magna*), the family Salmonidae (*Oncorhynchus mykiss*), a second family in the class Osteichthyes (*Lepomis macrochirus*), a third family in the phylum Chordata (*Pimephales promelas* or *Ictalurus punctatus*), an insect (*Chironomus tentans*), and a benthic amphipod (*Gammarus fasciatus*). Therefore, Tier II or Secondary Acute and Chronic Values were calculated according to the 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 and the results are listed in

TABLE 4. BIOCONCENTRATION FACTORS (BCF) IN AQUATIC SPECIES			
Species	BCF	Comments	Reference
Selenastrum capricornutum (green algae)	123	Four-day BCF, static test, intact organism	Liu et al., 1983
Lumbriculus variegatus (Oligochaete)	3.0	Four-day BCF, static test, intact organism	Liu et al., 1983
Daphnia magna (water flea)	1.6	Four-day BCF, static test, intact organism	Liu et al., 1983
Ictalurus punctatus (channel catfish)	4.0[a], 2.9[b] 5[a], 3.3[b]	28-day BCF, intermittent flow, muscle viscera	Bentley et al., 1977
Lepomis macrochirus (bluegill sunfish)	1.9 3.1	four-day BCF, static test, muscle viscera	Liu et al., 1983
Lepomis macrochirus (bluegill sunfish)	4.7[a], 3.5[b] 9[a], 6[b]	28-day BCF, intermittent flow, muscle viscera	Bentley et al., 1977
Pimephales promelas (fathead minnow)	5.9[a], 4.0[b] 11[a], 8.8[b]	28-day BCF, intermittent flow, muscle viscera	Bentley et al., 1977

[a] Test water mean measured concentration of 0.014 mg/L.
[b] Test water mean measured concentration of 1.0 mg/L.

Table 5. The types of aquatic criteria and benchmarks that can be calculated are summarized below. Prior to publication of the methodology for Tier II values, Bentley (1977) used an application factor of 0.1 for the bluegill sunfish data to derive a WQC of 0.35 mg/L, Sullivan et al. (1979) recommended a 24-hour average concentration of 0.3 mg/L to adequately protect aquatic life, and Etnier (1986) estimated a Final Acute Value of 5.2 mg/L.

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 GMAV and six data requirements for Tier I criteria were available to calculate a Tier II SAV for RDX.

Secondary Maximum Concentration (SMC). The SMC, analogous to the acute WQC or CMC, is equal to one half of the SAV. Aquatic organisms and their uses should not be affected unacceptably if the CMC (and by analogy the SMC), averaged over a period of one hour, is not exceeded more than once every three years.

Table 5. Water 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	1.4 mg/L[b]
Secondary Maximum Concentration	0.7 mg/L
Secondary Chronic Value	0.19 mg/L
Secondary Continuous Concentration	0.19 mg/L
Lowest Chronic Value - fish	4.9 mg/L
Lowest Chronic Value - daphnids	4.8 mg/L
Final Plant Value	3.2 mg/L[b]
Final Residue Value	Insufficient data
Sediment Quality Criterion (SQC_{oc})	1.3 mg/kg_{oc}[c]

[a] See Appendix A for calculations.
[b] Estimated; based on nominal concentrations.
[c] Mg chemical/kg organic carbon in sediment.

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 or CCC, is equal to the lowest of three values: the SCV, the Final Plant Value, and the Final Residue Value. Aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of the CCC (and by analogy the SCC) is not exceeded more than once every three years on the average.

Lowest Chronic Values. In the absence of chronic criteria, U.S. EPA uses the lowest chronic effects values reported in the literature (for daphnids and fish) as equivalent to chronic criteria.

Final Plant Value. For algal species, a test of at least 96-hours duration may be used; concentrations of test material should be measured and the endpoint should be biologically significant.

Final Residue Value. Bioconcentration studies with fish and aquatic vertebrates indicate that RDX is only slightly accumulated in tissues. Bioconcentration factors ranged from 2.9 to 11 in the

muscle and viscera of freshwater fish (See section 3.2). At two exposure concentrations, elimination from the tissues for two of the three species occurred within 2 weeks. No maximum permissible tissue concentration (FDA action level) was located for RDX, but based on the diet of mink, a residue concentration of 17 mg/kg of fish was calculated as a screening benchmark for reproductive effects (See Section 4.3.1).

3.3.2 Sediment-Associated Organisms

No data on the toxicity of spiked sediments or the interstitial water from spiked sediments to sediment-associated organisms were located for this chemical. One study was found that tested the toxicity of RDX 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 SQC. From these tests, it is known that a sediment pore water concentration of 15 mg/L would not be acutely 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, 1993b). SQC may be calculated from K_p values and the FCV; the result is expressed as mg of chemical/kg soil or sediment (dry weight). However, normalization to a sediment organic carbon content (SQC_{oc}) 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 the SQC_{oc} are contained in Appendix B and the value is listed in Table 5. In order to compare the SQC_{oc} to dry weight concentrations 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 a preliminary or screening value.

4. TERRESTRIAL TOXICOLOGY

4.1. TOXICITY

4.1.1. Mammals

No subchronic or chronic studies on the toxicity of RDX utilizing mammalian wildlife species were located. Three chronic toxicity studies and one combined teratogenicity/reproductive study on RDX utilizing laboratory animals were located. RDX is known to have an affect on the central nervous system, with symptoms of convulsions and hyperactivity in both humans and laboratory animals (Ryon et al., 1984; Layton et al., 1987; U.S. EPA, 1989). In the some of the following studies, these symptoms were observed in rats and mice only at the higher doses. In addition to neurotoxicity, effects on the blood, kidney, liver, eyes, and testes have been observed. Most evidence indicates that RDX is not teratogenic or carcinogenic.

In the first study, Hart (1976) administered RDX to groups of 100 male and 100 female Sprague-Dawley rats at doses of 0, 1.0, 3.1, or 10 mg/kg/day for 104 weeks. Body weights were recorded

weekly for the first 26 weeks, biweekly for the next 26 weeks, and every four weeks thereafter. Clinical signs were observed weekly. Hematology and clinical chemistry analyses were performed at intervals and at termination. At 52 weeks, 10 rats of each sex of each test group were sacrificed, organ weights were recorded, and major tissues and organs were removed for histological examination. At 104 weeks, the remaining animals were sacrificed and tissues and organs were examined. At termination, there were no statistically significant differences between control and treatment groups in survival, food consumption, body or organ weight, or histopathological findings. Fluctuations in clinical chemistry parameters and hematocytology occurred, but were not dose-related.

In the second study, groups of 75 male and 75 female Fischer 344 rats were administered RDX (containing 3-10% HMX) in the diet at doses of 0, 0.3, 1.5, 8.0, or 40 mg/kg/day for 24 months (Levine et al., 1983). Interim sacrifices of 10 rats/sex/group were taken at 6 and 12 months. Food consumption and body weights were monitored weekly or biweekly and clinical signs were observed daily. Prior to necropsy, blood samples were taken and hematology and clinical chemistry parameters were assessed. At necropsy, major organs were weighed and organs and tissues were examined histologically. The incidence of mortality in the high-dose group (68% of the males and 36% of the females) was increased throughout the study and was frequently preceded by tremors and convulsions. Other treatment-related findings in this group were anemia with secondary splenic lesions, hypoglycemia, reduced body weight gains, hepatotoxicity, and possible CNS involvement. In this dose group, cataracts were observed in females and urogenital lesions were present in males. Rats of both sexes administered 8 mg/kg/day had body weights that were approximately 5% lower than those of controls and increased kidney weights. Females had hepatomegaly and decreased total serum protein levels and males had an increase in hemosiderin-like pigment in the spleen and suppurative inflammation of the prostrate. The latter effects were also observed in males receiving 1.5 mg/kg/day. The latter effect may have been due to a bacterial infection as bladder distention and cystitis were also noted. No adverse effects were observed in rats administered 0.3 mg/kg/day.

In the third study, RDX (containing 3-10% HMX) was administered in the diet to groups of 85 male and 85 female $B6C3F_1$ mice at doses of 0, 1.5, 7.0, 35, or 100 mg/kg/day for 24 months (Lish et al., 1984). The 100 mg/kg/day dose group was originally administered 175 mg/kg/day, but due to high mortality through test week 10, the dose was lowered to 100 mg/kg/day beginning with test week 11. Interim sacrifices of 10 mice/sex/group occurred at 6 and 12 months. Animals were observed twice daily and food consumption and body weights were recorded weekly early in the test and then biweekly. Blood samples were taken periodically and prior to necropsy and hematology and clinical chemistry parameters were assessed. At necropsy, major organs were weighed and organs and tissues from the 0 and 100 mg/kg/day dose group were examined histologically; organs and tissues from the other dose groups were subjected to a limited histopathologic examination.

Following additional early mortalities after lowering the high dose to 100 mg/kg/day, survival in the remaining animals was similar to controls. Males receiving the 175 mg/kg/day dose had a high incidence of fighting wounds and skin lesions, but incidences of these lesions were similar in all groups by the end of the test. Food consumption was not altered by RDX treatment, but final body weights were lower than controls in the high-dose group. Hematology parameters were not altered in any dose group. Elevated serum cholesterol levels were present in high-dose males and in females receiving 7 and 35 mg/kg/day. Cataracts were present in high-dose males but not in females. Organ weights were altered in some dose groups: increased liver and kidney weight in both sexes receiving 100 mg/kg/day, increased kidney weight in males receiving 35 mg/kg/day, and increased heart weights in both sexes receiving 100 mg/kg/day. Histopathologic examinations revealed no apparent lesions at the 12-month sacrifice. By 24

months the following statistically significantly elevated lesions were observed: testicular degeneration in males administered 35 and 100 mg/kg/day, liver carcinomas in females administered 35 and 100 mg/kg/day, and malignant lymphoma of the liver in males administered 35 mg/kg/day. When combined liver adenoma/carcinoma data were compared to both historical and concurrent control groups, the incidence was also statistically significant in females receiving 7 mg/kg/day.

The teratogenicity of RDX was studied in rats and rabbits (Cholakis et al., 1980). Rats were dosed by gavage on days 6 through 19 of gestation and rabbits were gavaged on days 7 through 29 of gestation with 0, 0.2, 2.0, or 20 mg/kg/day of RDX. Dams were monitored daily for toxic signs. On day 20 of gestation (rats) or day 30 of gestation (rabbits), animals were sacrificed and the uterus of each animal was examined for live fetuses and resorptions. Fetuses were examined grossly and microscopically for malformations.

A dose of 20 mg/kg/day was toxic to rat dams, resulting in mortality (6/25 animals) and reduced body weight, reduced feed consumption, neurotoxic signs, and reduced liver weights of the survivors. These signs were not noted in the other dose groups. There were no effects on reproductive parameters (number of implants and viable fetuses) or increases in malformations of fetuses of surviving dams in the 20 mg/kg/day dose group or the lower dose groups. The dose of 20 mg/kg/day was embryotoxic. No treatment-related adverse effects were noted on pregnant rabbits. For rabbit fetuses, there were no statistically significantly increased incidences of resorptions or deaths or gross or microscopic anomalies at any dose level.

In the two-generation reproduction study, groups of 22 male and 22 female rats were fed diets designed to deliver doses of RDX of 0, 5, 16, or 50 mg/kg/day (Cholakis et al., 1980). After 13 weeks the rats (F_0 generation) were mated. The resultant F_1 pups were weaned and groups of 26 males and 26 females per dose group were administered respective diets for 13 weeks. After this treatment males and females were mated as above and resulting litters (F_2 generation) were sacrificed and examined microscopically.

Effects were observed primarily in the group receiving the 50 mg/kg/day dose during and following the F_0 mating: deaths prior to mating, reduced fertility in males and females (not statistically significant), reduced number of litters (not statistically significant) and reduced viability of pups in the high-dose group (statistically significant). The body weights of pups in both the mid- and high-dose groups was reduced after 25 days and only one litter in the high-dose group survived to day 21. Following the F_1 mating, meaningful comparisons between the high-dose group and the other groups and controls are difficult to make because only four males and two females survived until week 13 in the F_1 high-dose group compared with 26 mating pairs in the other groups. However, pup survival was obviously affected as the number of pups/litter on day 0 was statistically significantly reduced compared with the control group and no pups in this dose group survived more than a few days. Histopathological evaluations revealed a statistically significant increase in renal cortical cysts in females administered 16 mg/kg/day. Body weights of females were also reduced.

4.1.2. Birds

No subchronic or chronic studies on the toxicity of RDX to birds were located.

4.1.3. Plants

Simini et al. (1992) performed soil toxicity testing with the cucumber (*Cucumis sativus*) at RDX concentrations of 0, 50, 100, or 200 mg/kg of soil. Biomass of plants exposed to 100 and 200 mg/kg was significantly reduced. The results were reported in an abstract and no further information was given. Simini et al. (1995) also evaluated the toxicity of Joliet AAP (IL) soil which contained a mixture of munitions compounds to radishes and cucumbers. RDX was found to be very weakly correlated with the effects of munitions contaminated soil on plant height.

4.1.4 Soil Invertebrates

Simini et al. (1995) tested the toxicity of munitions-contaminated soil (contaminated in part with RDX) and calculated the correlation of RDX concentration with overall soil toxicity. Endpoints were survival rates and differences between initial and final live weights. RDX was found to be very weakly correlated with these endpoints.

In a preliminary study, Phillips et al. (1993) tested the toxicity of RDX in artificial soil (1.4% organic matter) to the earthworm, *Eisenia foetida* over a 14-day period. Concentrations of RDX were 0, 50, 100, 200, 400, and 500 mg/kg. Although survival was 80-100% for all concentrations, there was an increasing weight loss with increasing concentrations. Weight losses ranged from 11% at the 50 mg/kg concentration to 18% at the 500 mg/kg concentration.

4.1.5. Soil Heterotrophic Processes

No data on the effect of RDX on microorganisms in soil were located. In biodegradation studies, concentrations up to 50 mg/L were not toxic to microorganisms in anaerobic sewage sludge (McCormick et al., 1984).

4.2. METABOLISM AND BIOACCUMULATION

4.2.1. Animals

Metabolism. RDX is slowly but extensively absorbed following ingestion. Based on a lack of physiological response following dermal application to dogs and rabbits, McNamara et al. (1974) concluded that RDX did not penetrate the skin. No inhalation data were located. Following administration by gavage or in the drinking water to rats for 90 days, RDX was distributed throughout the body with no preferential uptake by any specific organ or tissue including the fat (Schneider et al., 1978). The pattern of distribution was similar in miniature swine administered a single oral dose (Schneider et al., 1977). Metabolism in the rat is extensive as indicated by excretion of 43% of a single ^{14}C-radiolabeled oral dose as $^{14}CO_2$ within four days (Schneider et al., 1977). Thirty-four percent was excreted in the urine and 3% in the feces, while 10% remained in the carcass. A similar pattern was observed following administration to rats for 13 weeks: 27-50.5% as $^{14}CO_2$, 22-35% in the urine, and 4-5% in the feces (Schneider et al., 1978). Urinary metabolites were not identified.

Bioaccumulation. Absorbed RDX is readily metabolized and excreted by mammals and does not accumulate in any tissue (Schneider et al., 1978). Log K_{ow} values of 0.87 (Banerjee et al., 1980), 0.81, and 0.86 (Burrows et al., 1989) also indicate a low potential for bioaccumulation. Biomonitoring studies have been conducted at Aberdeen Proving Ground, Maryland (USACHPPM, 1994), and at several other

AAPs (see Opresko, 1995 for review). RDX 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

Metabolism. In order to investigate the transformation of RDX prior to plant uptake, transformations were studied in hydroponic culture solutions with and without plant roots (unspecified species) (Harvey et al., 1991). No significant transformations took place in the solution. Twenty-one to 26-day old bush bean plants were grown in solutions containing 10 ppm (mg/L) ^{14}C-labeled RDX. Uptake and translocation of the parent compound to the leaves readily occurred, with recovery of primarily RDX. Some metabolism to unidentified polar metabolites took place by day 7. RDX remained sequestered within the plants, with no evolution of $^{14}CO_2$.

In a follow-up study, bean, wheat, or blando brome were planted in soils amended with RDX at a concentration of 10 ppm (mg/kg, air-dried weight) and after 60 days the plants were analyzed by HPLC and radioactivity for total uptake, tissue distribution, and transformations (Cataldo et al., 1993). Three soils were tested, Palouse (1.7% organic matter), Cinebar (7.2% organic matter), and Burbank (0.5% organic matter) (Cataldo et al., 1989). RDX was accumulated by and was mobile in the plants (Cataldo et al., 1993). For all three plants, highest concentrations were found in the leaves before seed formation; following seed formation, highest concentrations were present in the seeds of bush bean. Total uptake and tissue concentration were inversely proportional to soil organic matter. In roots and stems, 20% of the translocated material was present as the parent compound, while in leaves and seed tissues >50% was RDX, suggesting that RDX itself is mobile within the plant. The balance of the accumulated radioactivity was either unidentified polar metabolites (30-50%) or associated with insoluble plant parts.

Bioaccumulation. Data from several studies indicate that RDX can be taken up by plants. When grown in hydroponic nutrient solutions containing 10 ppm RDX, uptake and translocation to the stems and leaves occurred (Harvey et al., 1991). After 7 days, concentrations of RDX in leaves, stems, and roots were 97, 11, and 6 ppm respectively, indicating bioaccumulation in the aerial parts. RDX remained sequestered within the plants, with no evolution of $^{14}CO_2$.

Simini et al. (1992) studied the uptake of RDX by cucumber plants grown in RDX-amended soil. Plants grown in 50, 100, and 200 mg RDX per kg soil had 464, 1320, and 1702 mg RDX per kg leaf tissue. When grown in soil with a low organic carbon content and containing 10 ppm RDX, the compound is taken up by plants with up to 603 μg RDX equivalents (RDX plus metabolites)/g fresh weight accumulated in the seeds of bush beans grown in soil containing 0.5% organic carbon (Cataldo et al., 1993). Lower concentrations were present in the roots, stems, and leaves.

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 utilizing laboratory animals, reference doses or screening benchmarks for wildlife may be calculated by extrapolation among mammalian species. A chronic oral Reference Dose (RfD) for humans of 0.003 mg/kg/day was calculated by extrapolation and multiplication by uncertainty factors from the chronic oral study with rats by Levine et al. (1983) (U.S. EPA, 1993c). The endpoint in that study was inflammation of the prostate. Three chronic toxicity studies and a two-generation reproduction study on the oral

toxicity of RDX utilizing laboratory animals were available for calculating screening benchmarks for mammalian wildlife (Hart, 1976; Levine et al., 1983; Lish et al., 1984; Cholakis et al., 1980). The first three studies involved chronic exposures, whereas the reproduction study by Cholakis et al. (1980) involved subchronic (13 week) exposures during each of two generations. The study by Hart (1976) did not attain a LOAEL (lowest-observed-adverse-effects level) for population-related effects. In the other chronic study with rats (Levine et al., 1983), it is difficult to predict if the dose of 8 mg/kg/day which resulted in inflammation of the prostate is truly a NOAEL (no-observed-adverse-effects level) for reproductive effects and/or whether the effect was treatment related. Thus, the remaining highest NOAEL of 7 mg/kg/day in the study with mice (Lish et al., 1984) was chosen to derive screening benchmarks for wildlife. A clear NOAEL and LOAEL for reproductive effects (testicular degeneration) were established in this study.

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 deriving 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 6). The dietary concentrations in food or water for the wildlife species listed in Table 6 assume exposure through food or water alone and no exposure through other environmental media. If contaminants are present in both food and water, or in other media, or if other routes of exposure can be shown to be important,

TABLE 6. 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	8.7	15	40	
White-footed mouse	7.9	56	29	
Meadow vole	6.7	58	49	
Cottontail rabbit	2.9	15	30	
Mink	3.0	22	31	6.7
Red fox	2.1	21	25	
Whitetail deer	1.1	36	17	

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

the values in Table 6 must be adjusted so that intake from all sources/routes of exposure sum to the NOAEL. Confidence in the benchmarks is high because of the extensive data base covering various toxicological endpoints.

Birds. No subchronic or chronic studies were available for calculation of screening benchmarks for avian species.

4.3.2. Plants

In the absence of criteria for terrestrial plants, lowest-observed-effect concentrations (LOEC) values from the literature can be used to screen chemicals of potential concern for phytotoxicity (Will and Suter, 1995a). Plants were able to grow for 7 days in hydroponic solutions containing 10 ppm RDX (Harvey et al., 1991), but this data is insufficient to calculate a screening benchmark. Two studies on the growth of plants in RDX-amended soil were available. In the first study, bean, wheat, and blando brome plants were grown in soil amended with 10 mg/kg RDX, but effects on growth were not reported (Cataldo et al., 1989). Simini et al. (1992) reported that RDX concentrations of 100 and 200 mg/kg significantly reduced the biomass of cucumber plants. From this study a screening benchmark of 100 mg/kg was determined (Table 7). Confidence in the benchmark is low, as it is based on a single study involving a single species.

4.3.3. Soil Invertebrates

According to the method of Will and Suter (1995b), 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, RDX 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.

4.3.4. Soil Heterotrophic Processes

Insufficient data were located for calculation of a screening benchmark for soil heterotrophic processes. Studies with laboratory cultures were not considered as they utilized acclimated organisms.

TABLE 7. SCREENING BENCHMARKS FOR TERRESTRIAL PLANTS AND INVERTEBRATES

Screening Benchmark	Value
Plants - solution	Insufficient data
Plants - soil	100 mg/kg[a,b]
Soil invertebrates	Insufficient data
Soil microbial processes	Insufficient data

[a] For a detailed description of the methodology, see Will and Suter, 1995a.
[b] Based on a single study.

5. REFERENCES

Atkinson, R. 1987. A structure-activity relationship for the estimation of rate constants for the gas-phase reactions of OH radicals with organic compounds. Intl. J. Chem. Kinet. 19:799-828.

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for RDX* (Draft). U.S. Department of Health and Human Services, Public Health Service.

Banerjee, S., S.H. Yalkowsky, an S.C. Valvant. 1980. Water solubility and octanol/water partition coefficients of organics. Limitations of the solubility-partition coefficient correlation. Environ. Sci. Technol. 14:1227-1230.

Bender, E.S., P.F. Robinson, M.W. Moore, W.D. Thornton and A.E. Asaki. 1977. *Preliminary Environmental Survey of Holston Army Ammunition Plant, Kingsport, TN.* AD-A043 662, U.S. Army Chemical Systems Laboratory, Aberdeen Proving Ground, MD.

Bentley, R.E., J.W. Dean, S.J. Ellis, T.A. Hollister, G.A. LeBlanc, S. Sauter, and B.H. Sleight. 1977. *Laboratory Evaluation of the Toxicity of Cyclotrimethylene Trinitramine (RDX) to Aquatic Organisms.* Final Report, EG&G Bionomics, Wareham, MA for U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick MD, DAMD-17-74-C-4101.

Burrows, W.D., R.H. Chyrek, C.I. Noss, et al. 1984. Treatment for removal of munition chemicals from Army industrial wastewaters. In: Lagrega, M.D., and D.A. Long, eds. *Toxic and Hazardous Wastes: Proceedings of the 16th Mid-Atlantic Industrial Waste Conference*, Bucknell University, 1984. Lancaster, PA: Technomic Publishing Co., Inc., 331-342.

Burrows, E.P., D.H. Rosenblatt, W.R. Mitchell and D.L. Parmer. 1989. *Organic Explosives and Related Compounds: Environmental and Health Considerations.* AD-A210 554, U.S. Army Biomedical Research and Development Laboratory, Fort Detrick, Frederick, MD.

Cataldo, D.A., S.D. Harvey, R.J. Fellows, et al. 1989. *An Evaluation of the Environmental Fate and Behavior of Munitions Materiel (TNT, RDX) in Soil and Plant Systems.* PNL-7370; AD-A223 546, U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Cataldo, D.A., S.D. Harvey, R.J. Fellows. 1993. *The Environmental Behavior and Chemical Fate of Energetic Compounds (TNT, RDX, Tetryl) in Soil and Plant Systems.* PNL-SA-22362, Pacific Northwest Laboratory, Richland WA.

Checkai, R.T., M.A. Major, R.O. Nwanguma, J.C. Amos, et al. 1993. *Transport and Fate of Nitroaromatic and Nitramine Explosives in Soils from Open Burning/Open Detonation Operations: Milan Army Ammunition Plant (MAAP).* AD-A279 145; ERDEC-TR-136, Edgewood Research, Development & Engineering Center, Aberdeen Proving Ground, MD.

Cholakis, J.M., L.C.K. Wong, D.L. Van Goethem, et al. 1980. *Mammalian Toxicological Evaluation of RDX, Final Report.* AD A092531. Midwest Research Institute, Kansas City, MO. DMD17-78-C-8027.

Di Toro, D.M., C.S. Zarba, D.J. Hansen, W.J. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavlou, H.E. Allen, N.A. Thomas, and P.R. Paquin. 1991. Technical Basis for Establishing Sediment Quality Criteria for Nonionic Organic Chemicals using Equilibrium Partitioning. ***Environ. Toxicol. Chem. 10:1541-1583.***

DuBois, F.W. and J.F. Baytos. 1991. *Weathering of Explosives for Twenty Years*. LA-11931, Los Alamos National Laboratory, Los Alamos, NM.

Envirodyne Engineers, Inc. 1980. *Milan Army Ammunition Plant Contamination Survey. Final Report.* AD-BO53362, Envirodyne Engineers, Inc., St. Louis, MO.

Etnier, E.L. 1986. *Water Quality Criteria for Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)*. ORNL-6178, Oak Ridge National Laboratory, Oak Ridge, TN; U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Federal Register. 1994. Sediment Quality Criteria. FR 59:2652, January 18, 1994.

Funk, S.B., D.J. Roberts, D.L. Crawford, and R.L. Crawford. 1993. Initial-phase optimization for bioremediation of munition compound-contaminated soils. Appl. Environ. Microbiol. 59:2171-2177.

Goerlitz, D.F. 1992. A review of studies of contaminated groundwater conducted by the U.S. Geological Survey Organics Project, Menlo Park, California, 1961-1990. Environ. Sci. Pollut. Control Ser. 4:295-355.

Grant, C.L., T.F. Jenkins, K.F. Myers and E.F. McCormick. 1995. Holding-time estimates for soils containing explosives residues: comparison of fortification vs. field contamination. Environ. Toxicol. Chem. 14:1865-1874.

Greene, B., D.L. Kaplan and A.M. Kaplan. 1984. *Degradation of Pink Water Compounds in Soil - TNT, RDX, HMX*. AD-A157 954, U.S. Army Natick Research and Development Center, Natick, MA.

Gregory, R.G. and W.G. Elliott. 1987. *Remedial Investigation at Louisiana Army Ammunition Plant.* AMXTH-IR-CR-87110, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD.

Hale, V.Q., T.B. Stanford, and L.G. Taft. 1979. *Evaluation of the Environmental Fate of Munitions Compounds in Soil.* AD A034226, U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick; Battelle Columbus Laboratories, Columbus, OH.

Hart, E.R. 1976. *Two-year Feeding Study in Rats, Final Report.* AD A040161, Litton Bionetics Inc., Kensington, MD. N00014-73-C-0162, NR202-043.

Harvey, S.D., R.J. Fellows, D.A. Cataldo and R.M. Bean. 1991. Fate of the explosive hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in soil and bioaccumulation in bush bean hydroponic plants. Environ. Toxicol. Chem. 10:845-855.

Hoffsommer, J.C. and J.M. Rosen. 1972. Analysis of explosives in sea water. Bull. Environ. Contam. Toxicol. 7:177-181.

Hoffsommer, J.C. and J.M. Rosen. 1973. Hydrolysis of explosives in seawater. Bull. Environ. Contam. Toxicol. 10:78-79.

Hoffsommer, J.C., et al. 1972. *Analysis of Explosives in Sea Water and in Ocean Floor Sediment and Fauna.* AD 757778; NOLTR-72-215, Naval Ordinance Laboratory, White Oak, Silver Springs, MD.

Hull, R.N. and Suter, G.W. II. 1994. *Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Sediment-Associated Biota: 1994 Revision.* ES/ER/TM-95/R1. Oak Ridge National Laboratory, Oak Ridge, TN.

Jenkins, T.F. and C.L. Grant. 1987. Comparison of extraction techniques for munitions residues in soil. Anal. Chem. 59:1326-1331.

Jenkins , T.F., D.C. Leggett, C.L. Grant and C.F. Bauer. 1986. Reversed-phase high-performance liquid chromatographic determination of nitroorganics in munitions wastewater. Anal. Chem. 58:170-175.

Kayser, E.G. and N.E. Burlinson. 1988. Migration of explosives in soil: Analysis of RDX, TNT, and tetryl from a 14C lysimeter study. J. Energ. Mater. 6:45-71.

Kubose, D.A. and J.C. Hoffsommer. 1977. *Photolysis of RDX in Aqueous Solution. Initial Studies.* AD A-42199/OST. Available from NTIS, Springfield, VA.

Layton, D., et al. 1987. *Conventional Weapons Demilitarization: A Health and Environmental Effects Data Base Assessment: Phase II. Explosives and their Co-contaminants.* AD-A220 588, U.S. Army Medical Research and Development Command, Fort Detrick, MD.

Levine, B.S., E.M. Furedi, D.E. Gordon et al. 1983. *Determination of the Chronic Mammalian Toxicological Effects of RDX: Twenty-Four Month Chronic Toxicity/Carcinogenicity Study of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the Fischer 344 Rat, Phase V Final Report, Volume I.* ADA 160 774, IIT Research Institute, Chicago, IL; U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Lish, P.M., B.S. Levine, E.M. Furedi, E.M. Sagartz and V.S. Rac. 1984. *Determination of the Chronic Mammalian Toxicological Effects of RDX: Twenty-four Month Chronic Toxicity/Carcinogenicity Study of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 Hybrid Mouse.* Phase VI., Vol. 1. AD A160774. IIT Research Institute, Chicago, IL. U.S. Army Medical Research and Development Command. DAMD17-79-C-9161.

Liu, D.H.W., R.J. Spanggord, H.C. Bailey, H.S. Javitz, and D.C.L. Jones. 1983. *Toxicity of TNT Wastewaters to Aquatic Organisms. Final Report. Volume 1. Acute Toxicity of LAP Wastewater and 2,4,6-Trinitrotoluene.* SRI International, Menlo Park, CA. Report No. LSU-4262-Vol-1 (AD-A142144/5).

Lyman, W.J, W.F. ReehL, A. Rhone, et al. 1982. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds*. McGraw-Hill Book Company, New York.

McCormick, N.G., J.H. Cornell, and A.M. Kaplan. 1981. Biodegradation of hexahydro-1,3,5-trinitro-1,3,5-triazine. *Appl. Environ. Microbiol.* 42:817-823.

McCormick, N.G., J.H. Cornell and A.M. Kaplan. 1984. *The Anaerobic Biotransformation of RDX, HMX, and Their Acetylated Derivatives*. AD-A149 464, U.S. Army Natick Research and Development Center, Natick, MA; U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD.

McKone, T.E. and D.W. Layton. 1986. Screening the potential risks of toxic substances using a multimedia compartment model: Estimation of human exposure. Regul. Toxicol. Pharmacol. 6:359-380.

McNamara, B.P., H.P. Averill, E.J. Owens, et al. 1974. *The Toxicology of Cyclotrimethylenetrinitramine (RDX) and Cyclotetra-methylenetetranitramine (HMX) Solutions in Dimethylsulfoxide (DMSO), Cyclohexanone, and Acetone*. AD-780010, U.S. Army, Aberdeen Proving Ground, MD.

Newell, E.L., Jr. 1984. *Phase 3, Hazardous Waste Study No. 37-26-0147-84, Summary of AMC Open Burning/Open Detonation Ground Evaluations, November 1981-September 1983*. Department of the Army, U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD.

Opresko, D.M. 1995. *Review of Biomonitoring Studies and Ecological Surveys Conducted at U.S. Military Installations*. ORNL/M-4097, Oak Ridge National Laboratory, Oak Ridge, TN.

Opresko, D.M., B.E. Sample, and G.W. Suter II. 1995. *Toxicological Benchmarks for Wildlife: 1995 Revision*. ES/ER/TM-86/R2. Oak Ridge National Laboratory, Oak Ridge, TN

Osmon, J.L. and R.E. Klausmeier. 1972. The microbial degradation of explosives. Dev. Indust. Microbiol. 14:321-325.

Phillips, C.T., R.T. Checkai and R.S. Wentsel. 1993. *Toxicity of Selected Munitions and Munition-Contaminated Soil on the Earthworm (Eisenia foetida)*. AD-A264 408, Edgewood Research Development & Engineering Center, Aberdeen Proving Ground, MD.

Phung, H.T. and M.W. Bulot. 1981. Subsurface investigation of metal sludge and explosive disposal pond areas. In: R.A. Conway and D.C. Malloy, eds, *Hazardous Solid Waste Testing*, First Conference, ASTM STP 760, American Society for Testing and Materials, Philadelphia.

Richards, J.J. and G.A. Junk. 1986. Determination of munitions in water using macroreticular resins. Anal. Chem. 58:723-725.

Rosenblatt, D.H. 1981. *Environmental Risk Assessment for Four Munitions-Related Contaminants at Savanna Army Depot Activity*. Technical Report 8110, U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick, MD.

Rosenblatt, D.H. 1986. *Contaminated Soil Cleanup Objectives for Cornhusker Army Ammunition Plant*. Technical Report 8603, U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick, MD.

Ryon, M.G., B.C. Pal, S.S. Talmage, and R.H. Ross. 1984. *Database Assessment of the Health and Environmental Effects of Munition Production Waste Products*. AD-A145417, U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Schneider, N.R., S.L. Bradley, and M.E. Andersen. 1977. Toxicology of Cyclotrimethylene-trinitramine: Distribution and metabolism in miniature swine. Toxicol. Appl. Pharmacol. 39:531-541.

Schneider, N.R., S.L. Bradley, and M.E. Andersen. 1978. The distribution and metabolism of cyclotrimethylenetrinitramine (RDX) in the rat after subchronic administration. Toxicol. Appl. Pharmacol. 46:163-171

Sikka, H.C., S. Banerjee, E.J. Pack, and H.T. Appleton. 1980. *Environmental Fate of RDX and TNT*. U.S. Army Medical Research and Development Command, Fort Detrick, MD. Contract No. DAMD17-77-C-7026, Syracuse Research Corporation, Syracuse, NY.

Simini, M., R. Rowland, E.H. Lee, and R.W. Wentsel. 1992. Detection of stress in *Cucumis sativus* exposed to RDX using chlorophyll fluorescence. Abstracts, Thirteenth Annual Meeting, Society of Environmental Toxicology and Chemistry, Cincinnati, OH, November 8-12, p. 48.

Simini, M., R.S. Wentsel, R.T Checkai, C.T. Phillips, N.A. Chester, M.A. Major, and J.C. Amos. 1995. Evaluation of Soil Toxicity at Joliet Army Ammunition Plant. Env. Tox. Chem. 14:623-630.

Small, M.J. and D.H. Rosenblatt. 1974. *Munitions Production Products of Potential Concern as Waterborne Pollutants - Phase II*. AD-9190311, U.S. Army Bioengineering Research and Development Laboratory, Aberdeen Proving Ground, MD.

Soli, G. 1973. *Microbial Degradation of Cyclonite (RDX)*. AD-762 751, Naval Weapons Center, China Lake, CA. Technical Publication 5525.

Spalding, R.F. and J.W. Fulton. 1988. Groundwater munition residues and nitrate near Grand Island, Nebraska, U.S.A. J. Contam. Hydrol. 2:139-153.

Spanggord, R.J., B.W. Gibson, R.G. Keck, and G.W. Newell. 1978. *Mammalian Toxicological Evaluation of TNT Wastewaters, Volume I. Chemistry Studies*. Annual Report, SRI International, Menlo Park, CA.

Spanggord, R.J., T. Mill, T.-W. Chou, et al. 1980a. *Environmental Fate Studies on Certain Munition Wastewater Constituents. Final Report, Phase I - Literature Review*. SRI International, Menlo Park, CA; AD AO82372, U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Spanggord, R.J., T. Mill, T.W. Chou, et al. 1980b. *Environmental Fate Studies on Certain Munition Wastewater Constituents, Final Report, Phase II - Laboratory Studies*. AD A099256, U.S. Army

Medical Research and Development Command, Fort Detrick, MD; SRI International, Menlo Park, CA.

Spanggord R.J., W.R. Mabey, T. Mill, T.W. Chou, J.H. Smith and S. Lee. 1981. *Environmental Fate Studies on Certain Munition Wastewater Constituents. Phase III, Part I - Model Validation.* AD A129373, U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, MD.

Spanggord, R.J., et al. 1983. *Environmental Fate Studies on Certain Munitions Wastewater Constituents: Phase IV - Lagoon Model Studies.* AD-A138550, U.S. Army Medical Research and Development Command, Fort Detrick, MD.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. *Guidelines for Deriving Numerical Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses.* Final Report, PB85-227049. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C.

Stidham, B.R. 1979. *Analysis of Wastewater for Organic Compounds Unique to RDX/HMX Manufacturing and Processing. Final Report.* AD AO85765, Holston Defense Corporation, Kingsport, TN.

Stilwell, J.M., M.A. Fischer, W.L. Margard, M.C. Matthews, B.E. Sherwood, and T.B. Stanford. 1977. *Toxicological Investigations of Pilot Treatment Plant Wastewaters at Holston Army Ammunition Plant.* Final Report, AD A042601. Battelle Columbus Laboratories, Columbus, OH. DAMD17-74-C-4123.

Sullivan, J.H., Jr., H.D. Putnam, M.A. Keirn, B.C. Pruitt, Jr., J.C. Nichols, and J.T. McClave. 1979. *A Summary and Evaluation of Aquatic Environmental Data in Relation to Establishing Water Quality Criteria for Munitions-Unique Compounds. Part 4: RDX and HMX, Final Report.* AD-A087-683, U.S. Army Medical Research and Development Command, Ft. Detrick, Frederick, MD; Water and Air Research, Inc., Gainesville, FL.

Suter, G.W. II and J.B. Mabrey. 1994. *Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota on Oak Ridge Reservation, Oak Ridge, Tennessee: 1994 Revision.* ES/ER/TM-96/R1, Oak Ridge National Laboratory, Oak Ridge, TN.

Todd, Q., F. Finger, R. Turner, and D. Morley. 1989. *Delivery Order 8, Louisiana Army Ammunition Plant: Updated Remedial Investigation.* Roy F. Weston, West Chester, PA; U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD).

Tucker, W.A., E.V. Dose, G.J. Gensheimer, et al. 1985. *Evaluation of Critical Parameters Affecting Contaminant Migration through Soils, Final Report.* U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD.

USACHPPM (U.S. Army Center for Health Promotion and Preventive Medicine). 1994. *Field Survey No. 75-23-YS50-94, Health Risk Assessment of Consuming Deer from Aberdeen Proving Ground, Maryland (Draft).* Toxicology Division, USACHPPM, Aberdeen Proving Ground, MD.

U.S. EPA. 1975. *Methods for Acute Toxicity Tests with Fish, Macroinvertebrates, and Amphibians.* 660/3-75-009, Ecological Research Series, Washington, DC.

U.S. EPA. 1989. *Health and Environmental Effects Document for RDX Cyclonite.* ECAO-CIN-GO78, Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1982. *Federal Register* 47:49234-49252, October 29, 1982.

U.S. EPA. 1993a. *Water Quality Guidance for the Great Lakes System and Correction: Proposed Rules.* Fed. Regist. 58:20802-21047.

U.S. EPA. 1993b. *Technical Basis for Deriving Sediment Quality Criteria for Nonionic Organic Contaminants for the Protection of Benthic Organisms by Using Equilibrium Partitioning.* EPA-822-R-93-011, Office of Water, Washington, DC.

U.S. EPA. 1993c. *Integrated Risk Information System (IRIS) Online.* Cincinnati, OH: Office of Health and Environmental Assessment.

U.S. EPA. 1993d. *Wildlife Criteria Portions of the Proposed Water Quality Guidance for the Great Lakes System.* EPA/822/R-93/006. Office of Science and Technology, Washington, DC.

Walsh, M.E., and T.F. Jenkins. 1992. *Identification of TNT Transformation Products in Soil.* AD-A255 308. U.S. Army Cold Regions Research and Engineering laboratory, Hanover, NH.

Will, M.E. and G.W. Suter II. 1995a *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1995 Revision.* ES/ER/TM-85/R2, Oak Ridge National Laboratory, Oak Ridge, TN.

Will, M.E. and G.W. Suter II. 1995b. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Process.* ES/ER/TM-126/R1, Oak Ridge National Laboratory, Oak Ridge, TN.

APPENDIX A

DERIVATION OF AQUATIC BENCHMARKS FOR RDX

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; these SAFs, based on the number of satisfied data requirements for Tier I calculations, are listed in Table A-1. 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 (three different families including a fish, invertebrate, and an acutely sensitive freshwater species), 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 criterion, 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): a planktonic crustacean (*Daphnia magna*), the family Salmonidae (*Oncorhynchus mykiss*), a second family in the class Osteichthyes

(*Lepomis macrochirus*), a third family in the phylum Chordata (*Pimephales promelas* or *Ictalurus punctatus*), a benthic crustacean (*Gammarus fasciatus*), and an insect (*Chironomus tentans*). For two of the families, data were available to calculate acute:chronic toxicity ratios: Daphnidae and Salmonidae.

The SAV is calculated by dividing the lowest GMAV in the data base by the SAF. Although flow-through tests with measured concentrations are recommended by Stephan et al. (1985), the static tests were available for all fish species and had lower, more conservative values. Thus, they were included in the calculations. Values for invertebrates were considerably higher. Values used to calculate the GMAV for the fish species or genera were 4.1 and 13 mg/L for *Ictalurus punctatus*, 6.0 and 7.6 mg/L for *Lepomis macrochirus*, 5.8, 6.6, and 4.5 mg/L for *Pimephales promelas*, and 6.4 mg/L for *Oncorhynchus mykiss*. Other values for these species listed in Table 1 were not used as either the fish were not of the appropriate age/size or the tests were not conducted under the appropriate physical/chemical conditions. The lowest GMAV was 5.56 mg/L for *Pimephales promelas*. (Note: all decimal places were carried during calculations). 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,

$$SAV = \frac{5.56}{4.0} = 1.39 \text{ mg/L}$$

2. Secondary Maximum Concentration (SMC)

SMC = ½(SAV) = 0.6955 mg/L

3. Secondary Acute-Chronic Ratio (SACR)

One of three required experimentally determined ACR were available according to guidelines for Tier I (chronic tests on a sensitive species and an invertebrate were not acceptable). Therefore, two assumed ACRs of 18 were added. Although the tests conducted by Bentley et al. (1977) suffered from equipment malfunctions, for fathead minnows, the data appears to be consistent for the two tests reported in Table 1. In addition, it is the only available data. For *Daphnia magna*, the acute/chronic ratio of (>15 mg/L)/(4.8 mg/L) is not acceptable. The SACR is the geometric mean of the three ACRs.

Daphnia magna >15/4.8 = >3.125 (not acceptable)
Pimephales promelas 5.56/4.9 = 1.136
Assumed ACR = 18, 18

SACR = Geometric mean of 1.136, 18, and 18 = 7.17

4. Secondary Chronic Value (SCV)

The SCV is equal to the SAV divided by the SACR

$$SCV = \frac{SAV}{SACR} = \frac{1.39}{7.17} = 0.194 \text{ mg/L}$$

5. Secondary Continuous Concentration (SCC)

The SCC is equal to the lowest of three values: the SCV, the Final Plant Value, and the Final Residue Value. A Final Plant Value can be estimated from the study of Bentley et al. (1977). Although concentrations were nominal rather than measured, the test by in which *Selenastrum capricornutum* showed a decrease in cell density at a concentration of 3.2 mg/L can be used as a screening value for the Final Plant Value. In other tests conducted by Bentley et al. (1977), in which concentrations were measured, nominal values were close to measured values. A Final Residue Value was not available. Because the estimated Final Plant Value of 3.2 mg/L is higher than the SCV, the SCC is equal to the SCV, 0.19 mg/L.

APPENDIX B

DERIVATION OF SEDIMENT QUALITY CRITERIA FOR RDX

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 (1993b). 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 (1993b) 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.

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

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

Because K_{ow} values are available, a K_{oc} can be calculated and a SQC_{oc} can be determined. Log K_{ow} values of 0.87, 0.86, and 0.81 were cited in Burrows et al. (1989); the geometric mean of

these values is 0.846. Using the relationship between K_{ow} and K_{oc} above, the log K_{oc} is 0.832 and the K_{oc} is 6.79. Then, the organic carbon-normalized SQC_{oc}, i.e., mg chemical/kg organic carbon (mg/kg_{oc}) is

$$SQC_{oc} = 6.79 \text{ L/kg} \times 0.194 \text{ mg/L} = 1.32 \text{ mg/kg}_{oc}$$

If one assumes an organic carbon content in the soil of 1%, the $SQC_{1\%}$ is calculated as

$$SQC_{1\%} = 0.01 \times 6.79 \text{ L/kg} \times 0.194 \text{ mg/L} = 0.01 \text{ mg/kg}$$

APPENDIX C

DERIVATION OF WILDLIFE BENCHMARKS FOR RDX

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; 1993d):

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

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.

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)

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

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

C.2 REFERENCE STUDIES FOR USE IN DERIVING WILDLIFE BENCHMARKS

Reference studies available for calculating toxicity benchmarks for wildlife for RDX are summarized in Table C-3. The LOAELs listed in the table are for population-related effects (i.e., survival and reproductive parameters); for other endpoints (i.e., body or organ weight changes) lower LOAELS and NOAELs may have been reported in these studies. For example, in the Levine et al. (1983) study, inflammation of the prostate occurred in rats dosed with 1.5 mg/kg/day and the NOAEL for this effect, which was 0.3 mg/kg/day, was used to derive a human oral RfD for RDX (U.S. EPA, 1993c).

Table C-3. NOAELs and LOAELs for Population-Related Effects for RDX

Species	Route	Exposure Period	NOAEL mg/kg/day	LOAEL mg/kg/day	Effect/ Endpoint	Reference
rat	diet	2 years	10[a]			Hart, 1976
	diet	2 years	8[b]	40	mortality	Levine et al., 1983
	diet	13 wk/generation (2 generations)	16[c,d]	50[e]	mortality	Cholakis et al., 1980
	diet	13 wk/generation (2 generations)	5	16[d]	reduced pup weight	Cholakis et al., 1980
	gavage	gestation days 6-19	2	20[f]	toxic to dams and embryos	Cholakis et al., 1980
mouse	diet	2 years	7	35	testicular degeneration	Lish et al., 1984
rabbit	gavage	gestation days 7-29	2	20[f]	toxic to dams and embryos	Cholakis et al., 1980

[a] Maximum dose tested.
[b] Inflammation of prostate seen at 1.5 and 8.0 mg/kg/day; NOEL 0.3 mg/kg/day.
[c] No adverse reproductive effects seen at this dose.
[d] Nominal dose; calculated doses were 13.8 (males) and 14.5 (females) mg/kg/day in the F_0 generation and 17.6 (males) and 18.8 (females) mg/kg/day in the F_1 generation.
[e] Nominal dose; calculated doses were 41.3 (males) and 43.6 (females) mg/kg/day in the F_0 generation and 61.3 (males) and 69.6 (females) mg/kg/day in the F_1 generation.
[f] No teratogenic effects seen at this dose.

Of the studies listed in Table C-3, the Hart study provides the highest NOAEL (10 mg/kg/day) for a chronic exposure period; however, if this NOAEL is used to derive benchmarks for the other species listed on the table, the calculated benchmarks will be higher than the listed NOAELs; therefore, the more conservative approach would be to use the mouse NOAEL of 7 mg/kg/day derived in the study by Lish et al. (1984). This study is summarized below:

Reference:	Lish, P.M., B.S. Levine, E.M. Furedi, et al. 1984. *Determination of the Chronic Mammalian Toxicological Effects of RDX: Twenty-four Month Chronic Toxicity/Carcinogenicity Study of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 Hybrid Mouse.* Phase VI., Vol. 1. AD A160774. IIT Research Institute, Chicago, IL. U.S. Army Medical Research and Development Command. DAMD17-79-C-9161.
Test Species:	B6C3F$_1$ mouse
Exposure Duration:	2 years
Endpoint:	Reproductive system; testicular degeneration
Exposure Route:	Oral (diet)
Dosage:	0, 1.5, 7.0, 35.0 or 100 mg/kg/day. The last dose was reduced from 175 mg/kg/day in test week 11 due to high mortality.
Comments:	Statistically significant levels of testicular degeneration occurred at 35 and 100 mg/kg/day. At these dose levels, dosed females exhibited a statistically significant increase in hepatocellular carcinomas. Weekly or biweekly mean body weights for the male mice in the 7 mg/kg/day group ranged from 24.1 g before the test began to 38.6 g after 53 weeks and 37.8 g after 104 weeks. The overall mean of weekly and biweekly means was calculated to be 35.9 g.
Final Chronic NOAEL:	7 mg/kg/day

C.3 CALCULATION OF WILDLIFE BENCHMARKS FOR RDX

Wildlife benchmarks for RDX 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 7 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 mouse = 0.036 kg
test NOAEL = experimental dose = 7 mg/kg/day

Therefore:

$$\textit{Meadow vole NOAEL} = 7.0\ \textit{mg/kg/day} \times \left[\frac{0.036\ \textit{kg}}{0.044\ \textit{kg}}\right]^{1/4} = 6.657\ \textit{mg/kg/day}$$

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

$$Cf = \frac{\textit{Meadow vole NOAEL}}{f} = \frac{6.657}{0.114} = 58.40\ \textit{mg/kg food}$$

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

$$Cw = \frac{\textit{Meadow vole NOAEL}}{\omega} = \frac{6.657}{0.136} = 48.95\ \textit{mg/L water}$$

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

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

The chronic NOAEL for mink is derived from the chronic NOAEL of 7 mg/kg for the laboratory mouse:

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

where: wildlife bw = body weight of mink = 1.0 kg
test bw = body weight of mouse = 0.036 kg
test NOAEL = experimental dose = 7 mg/kg/day

Therefore:

$$Mink\ NOAEL = 7.0\ mg/kg/day \times \left[\frac{0.036\ kg}{1.0\ kg}\right]^{1/4} = 3.049\ mg/kg/day$$

The BCF for RDX can be estimated from the mean log K_{ow} of 0.85 (see Section 3.4) by the following equation:

$$\log BCF = 0.76 \log K_{ow} - 0.23 = 0.413$$

$$BCF = 2.59$$

The log K_{ow} of 0.85 is also used to estimate the FCM from Table C-1. 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:

$$Cw = \frac{3.049 \times 1.0}{[0.099 + (0.137 \times 2.59 \times 1.0)]} = 6.72\ mg/L$$

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

Table C-4. Screening Benchmarks for RDX for Selected Mammalian Wildlife Species[a]									
	Wildlife		Experimental Values			Extrapolated Values for Chronic Exposures			
							Screening Benchmarks		
Experimental animal (body weight)	Species	Body Weight (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]
Mouse (0.036 kg)				7.0	testes	7.0			
	short-tailed shrew	0.015				8.7	14.5	39.6	
	white-footed mouse	0.022				7.9	56.2	29.0	
	meadow vole	0.044				6.7	58.4	49.0	
	cottontail rabbit	1.2				2.9	14.7	30.0	
	mink	1.0				3.0	22.3	30.8	6.72
	red fox	4.5				2.1	20.9	24.9	
	whitetail deer	56.5				1.1	35.9	17.1	

[a] For a detailed description of the methodology, see Opresko et al., 1995.
[b] Reference: Lish et al., 1984.
[c] NOAEL values have been rounded off, but non-rounded values were used to calculate benchmarks.
[d] Water concentration that incorporates intake from both water and food consumption.