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Final Report
(Version 3.2)
Volume II

May 1989
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ENVIRONMENTAL SCIENCE AND ENGINEERING, INC.

Prepared for
OFFICE OF THE PROGRAM MANAGER
ROCKY MOUNTAIN ARSENAL CONTAMINATION CLEANUP

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THE PURPOSE OF THE BIOTA REMEDIAL INVESTIGATION REPORT IS TO PRESENT THE CURRENT NATURE AND EXTENT OF CONTAMINATION IN BIOTA ON RMA. THE REPORT INTEGRATES KNOWN HISTORICAL INFORMATION, THE RESULTS OF PREVIOUS INVESTIGATIONS, AND RESULTS OF CURRENT PROGRAMS.

THE REPORT IS DIVIDED INTO THE FOLLOWING SECTIONS:

1. GENERAL BACKGROUND INFORMATION ON RMA CONTAMINATION AND PAST INVESTIGATIONS
2. DESCRIPTION OF THE PHYSICAL ENVIRONMENT, REGIONAL BIOTA, AND BIOTA WITHIN THE ARSENAL; IDENTIFICATION OF IMPORTANT BIOLOGICAL COMPONENTS AS A BASIS FOR EVALUATING CONTAMINATION
3. SUMMARY OF THE METHODS USED IN THIS ASSESSMENT
4. DATA ON THE TYPES AND CONCENTRATIONS OF CONTAMINANTS IN RMA BIOTA
5. ASSESSMENT OF THE DISTRIBUTION AND CONCENTRATION OF CONTAMINANTS IN BIOTA AND ASSOCIATED ENVIRONMENT TO EVALUATE PATHWAYS FOR CONTAMINANT MOVEMENT

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5.0 CONTAMINATION ASSESSMENT

The criteria developed in this section were used to estimate acceptable concentrations of contaminants of concern in the biotic environment to assist in the evaluation of potential adverse ecological effects resulting from RMA contamination. These values are used in this Biota RI document as a basis for evaluating potential harm to species and ecosystems where the effects may be subtle or difficult to detect by direct field observation alone. Further evaluation of these acceptable concentrations during the endangerment assessment portion of the RI/FS process will lead to the development of the cleanup criteria based on the same approach used to calculate these acceptable concentrations.

In the contamination assessment, the 39 contaminants of concern to biota were systematically evaluated to assess direct and indirect adverse effects on biota and to develop criteria for contaminant concentrations in abiotic media (e.g., soil, water, sediment) that would not be hazardous to biota (Figure 5.0-1). Many of the RMA contaminants are of concern because of their environmental persistence and bioaccumulation potential, but other contaminants are of concern because of adverse effects on biota produced as a result of direct environmental exposure. A toxicity assessment approach was used, whereby environmental fate and toxicological information were combined to evaluate the adverse effects of RMA contaminants on biota and to determine contaminant levels in the abiotic environment that would have no adverse effect on biota.

The "no effect" criteria were developed by assessing the toxic properties of each contaminant to provide an evaluation of the effects of the contaminants on wildlife populations. Pertinent regulatory documents and the general literature were used as sources of information in the development and selection of appropriate criteria (e.g. EPA Ambient Water Quality Criteria (AWQC), Health Advisories, and Health Effects Assessment Documents).

The 39 contaminants of concern were divided into seven "contaminants of major concern" and 32 "other contaminants of concern" on the basis of the criteria listed in Section 3.2.2.3. To evaluate the impact on biota the 32

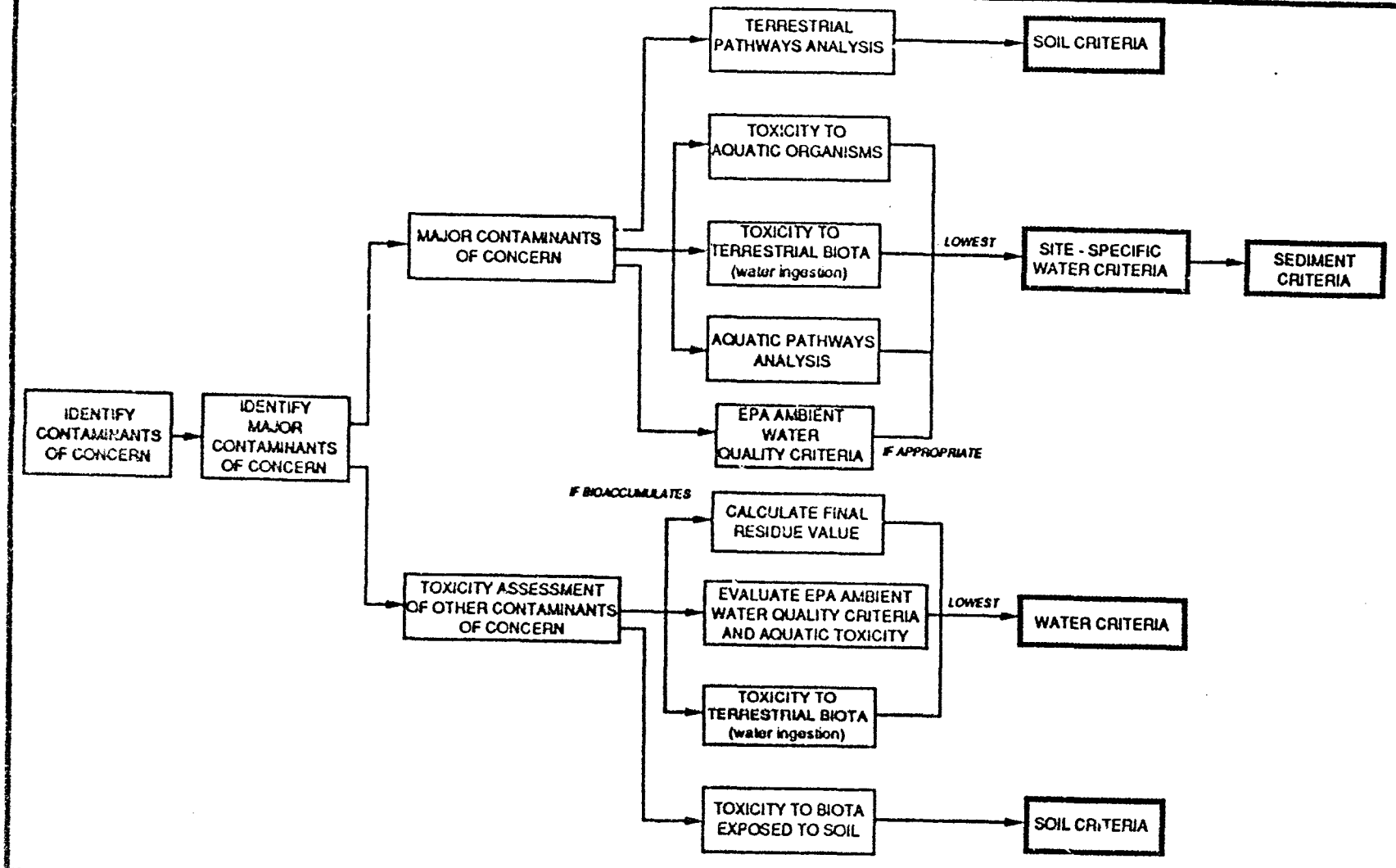


Figure 5.0-1
METHODOLOGY FOR DETERMINING ECOLOGICAL CRITERIA
(Detailed Steps Provided in Figures 5.1-1, 5.1-2, and 5.2-1)

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal

Aberdeen Proving Ground, Maryland

other contaminants of concern were analyzed by the toxicity assessments. The seven contaminants of major concern were subjected to a more detailed examination. The 32 other contaminants of concern were evaluated in the 27 toxicity assessments (Section 5.1). Similar contaminants, such as metabolites and parent compounds, were addressed in the same toxicity assessment. The toxicity assessments were intended to provide brief toxicological profiles centered around health effect information on wildlife populations. The literature review covers the major health effect information available for each contaminant. Data pertaining to wildlife species were emphasized, and information on domestic or laboratory animals was used when wildlife data were unavailable. Data for oral exposure were preferred to data for exposure by injection, as exposure by this route is unrelated to in-situ exposure. The data were compiled primarily for later use in the endangerment assessments, and will be modified as the Phase II data for abiotic media indicate are appropriate.

In the toxicity assessments, toxicity to aquatic organisms was addressed by using EPA Ambient Water Quality Criteria (AWQC) when available, and the water criteria protective of aquatic life are therefore not site-specific as for the seven major contaminants of concern. Toxicity to organisms consuming surface water or exposed to soils was also addressed. Food web contamination was not addressed in depth in the toxicity assessments.

Inhalation toxicity data were provided for background information only. The air pathway was not evaluated because data from air sampling studies indicate low potential for adverse effects on biota via this route of exposure, and because there is little information on the adverse effects on biota in natural ecosystems from exposure to the contaminants of concern by this route.

Dermal exposure values were not calculated for the toxicity assessments although dermal toxicity data were provided when available. Criteria were not estimated because of the uncertainty in correlating dermal toxicity under laboratory conditions (concentrated solutions, shaved skin of test animals) with toxicity under field conditions (generally dilute

concentrations mixed with soil or water, contact with various body surfaces that can be covered with hair or are calloused).

Information from the toxicity assessments was used to correlate observed adverse effects on biota with chemical content of tissues, as well as interpret contaminant data for biotic and abiotic media in the RMA environment. In the evaluation of biological effects, data on contaminant concentrations in biological tissues were related to potential adverse biological effects (e.g., death, diminished reproductive success, reduced population levels, etc.) observed in current biota assessment studies, and to criteria developed for contaminants in abiotic media. Results and discussion of current adverse effects of RMA contamination on biota are provided in Section 5.3.

For the seven major contaminants of concern (aldrin/dieldrin, arsenic, DBCP, endrin/isodrin, and mercury), data were analyzed to determine site-specific criteria (Section 5.2). Toxicity to aquatic organisms and to organisms that consume surface water were addressed for the major contaminants of concern. Accumulation in food chains was addressed by the Pathway Analysis. Pathway Analysis values, water quality criteria for aquatic life or surface water consumption for terrestrial organisms, existing Applicable or Relevant and Appropriate Requirements (ARARs), and biological effect information were evaluated to determine the current effects on biota and to provide a set of criteria to be used in subsequent quantification of biological risk in the forthcoming Onpost and Offpost Endangerment Assessments.

5.1 TOXICITY ASSESSMENTS OF CONTAMINANTS OF CONCERN

Toxicity assessments were performed for the 32 other contaminants of concern. General information on these other contaminants of concern was compiled and systematically evaluated to determine appropriate criteria for water (Figure 5.1-1) and soil (Figure 5.1-2). Three evaluation routes were developed, depending on the availability of information: 1) evaluation of EPA water quality guidelines and aquatic life toxicity information, 2) evaluation of information on toxicity of contaminants to terrestrial organisms through the water ingestion route, and 3) review of toxicity information for organisms directly exposed to contaminants in soil. For each of these evaluation routes, the potential for bioaccumulation was also considered.

Aquatic Life Criteria

For aquatic biota, water quality criteria were developed for the other contaminants of concern as data were available. The information was evaluated by a hierarchical approach. For example, when EPA Ambient Water Quality Criteria for the Protection of Freshwater Aquatic Organisms and their Uses were available, these were used as the appropriate water criteria for a particular chemical. In the instances where the EPA water criterion for the protection of aquatic life was based on a Final Residue Value (FRV) estimated from human guidelines, the Final Chronic Value or Final Acute Value (divided by 10^2) was used in place of the FRV as a criterion for aquatic organisms (see Section 5.1.7). If the EPA water criteria for the protection of aquatic life were unavailable, the EPA chronic Lowest Observed Adverse Effects Level (LOAEL) was divided by an uncertainty factor of 10 to produce a water criterion. If chronic LOAEL data were unavailable, then the Lowest Acute Value (LAV) provided by EPA or in the open literature was divided by an uncertainty factor of 10^2 to estimate a water concentration criterion. These uncertainty factors were also assumed to incorporate uncertainty due to interspecific variation.

In instances where EPA data were lacking, published data regarding toxicity to aquatic organisms were compiled. The approach was again hierarchical, moving from chronic to acute and applying the appropriate uncertainty factors to produce a criterion value (Figure 5.1-1).

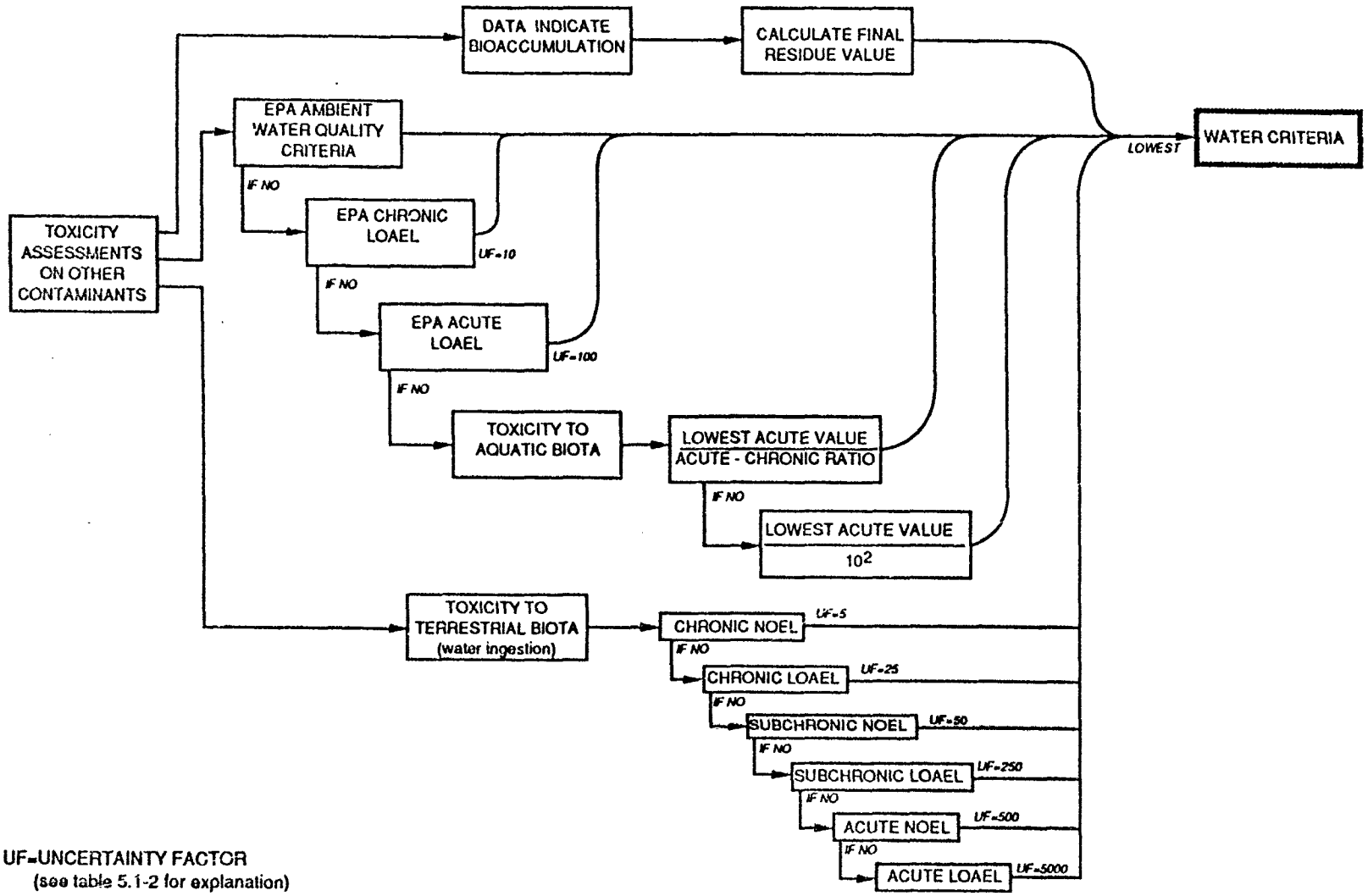
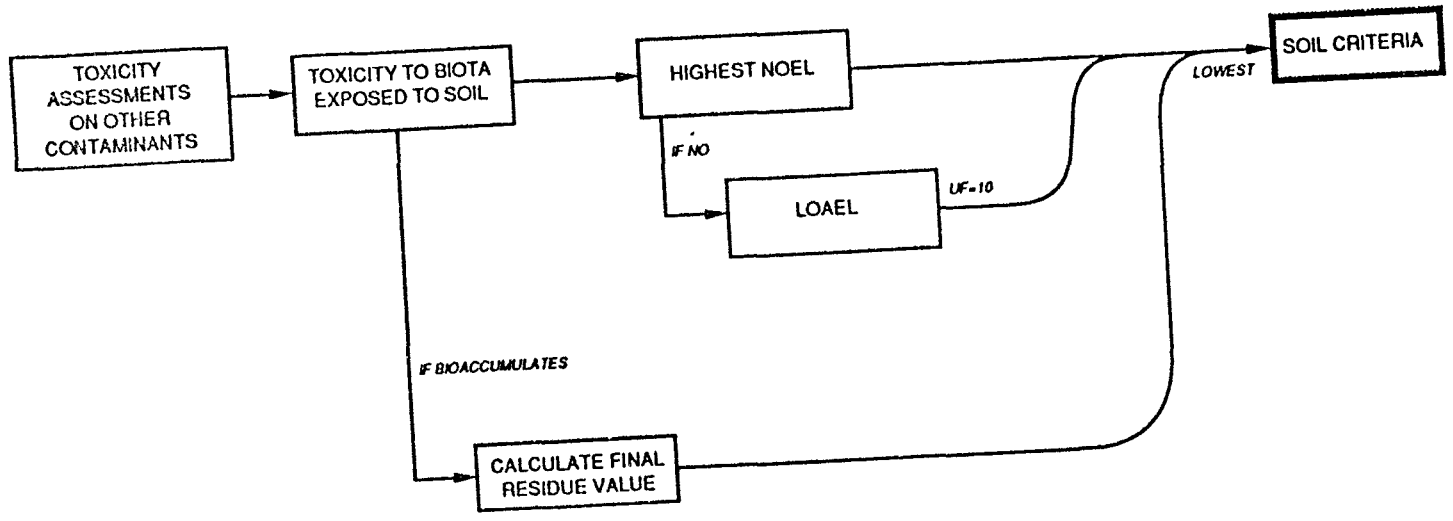


Figure 5.1-1
METHODOLOGY FOR DETERMINING WATER CRITERIA FOR OTHER CONTAMINANTS OF CONCERN

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal

Aberdeen Proving Ground, Maryland



UF=UNCERTAINTY FACTOR

Figure 5.1-2
METHODOLOGY FOR DETERMINING SOIL CRITERIA
FOR OTHER CONTAMINANTS OF CONCERN

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal

Aberdeen Proving Ground, Maryland

The water criterion can be used to produce a corresponding sediment value if it is multiplied by the soil-water partition coefficient normalized for organic carbon (K_{oc}) and the fraction of organic carbon in the sediments at RMA (f_{oc}). When the environmental fate of the contaminant is independent of organic carbon, the soil-water partition coefficient (K_d) is applied instead of K_{oc} and f_{oc}. However, sediment criteria were not developed as part of the toxicity assessments for the contaminants of concern because of the uncertainty involved in the estimate due to the limited data review. Sediment criteria were developed for the major contaminants of concern.

Surface Water Ingestion by Terrestrial Organisms

Information on toxicity of contaminants to terrestrial organisms via oral ingestion was evaluated. By assuming that toxicity via oral ingestion would be similar regardless of the carrier, the most sensitive LOAEL or NOEL divided by both water intake (Table 5.1-1) and the appropriate uncertainty factors (Table 5.1-2) were used to estimate water criteria (Figure 5.1-1).

Where both LOAEL and NOEL values were available, the NOEL was selected as the preferred value. Chronic data were used in preference to subchronic or acute values because there is less uncertainty involved in the estimate.

Inhalation Criteria

Inhalation toxicity data are presented for reference purposes only. Air contamination does not appear to be a significant hazard to wildlife populations (ESE, 1988a); therefore, air criteria were not estimated at this time. Contaminants of major concern occurred in air only in Sections 26 and 36 (ESE, 1986a), and at levels so low that toxic effects are not expected.

Soil Criteria

Soil criteria were developed in each toxicity assessment to the extent that appropriate data were available. Information on toxicity to biota through direct exposure to soil were evaluated to identify the most sensitive LOAEL or NOEL and divided by the appropriate uncertainty factor (Figure 5.1-2). Where both values were available, the NOEL was selected over the LOAEL in the calculation of a criterion.

Table 5.1-1. Water and Food Intake Values for Birds and Mammals Used in Establishing Acceptable Water Concentrations

Species	Daily H_2O Consumption (l/kg bw/day)*	Daily Food Consumption (g/kg bw/day)*
Rat (adult)	0.125	75
Duck (adult)	0.200	100
Mouse	0.2	120
Rabbit (adult)	0.165	30
Chicken (adult)	0.25	175
Dog	0.05	25
Cat	0.05	50
Pig	0.25	--
Mink	0.07	--

* l/kg bw/day = liters/kilogram body weight/day.
g/kg bw/day = grams/kilogram body weight/day.

Source: ESE, 1987; Sax, 1984; Ringer, 1988.

Table 5.1-2. Uncertainty Factors Used in Establishing Acceptable Water Concentrations

Health Effects	Factor Used to Convert Effect to a Chronic NOEL	Factor Applied for Interspecific Variation	Total Uncertainty Factor
Chronic NOEL	--	5	5
Chronic LOAEL	5	5	25
Subchronic NOEL	10	5	50
Subchronic LOAEL	50	5	250
Acute NOEL	100	5	500
Acute LOAEL, LD_{50}	1,000	5	5,000

Source: ESE, 1988.

Criteria for Bioaccumulative Contaminants

In instances where a contaminant in water was known to bioaccumulate or concentrate in organisms, a FRV for water was developed according to EPA methodology (Stephan et al., 1985). The FRV is the maximum permissible tissue concentration (MPTC) for a higher trophic level organism such as a raptor or a mallard, divided by the geometric mean bioconcentration factor (BCF) of the prey. The MPTC can be expressed either as a tissue concentration, such as an FDA action level, or as a dietary concentration for a sensitive wildlife species (Stephan et al., 1985). When the FRV reported by EPA was based on human guidelines, the MPTC was replaced with a value more appropriate for estimating criteria for wildlife populations.

The FRV and water ingestion value for terrestrial organisms were then compared and the lowest value identified. This value was then compared to the lowest value produced through evaluation of toxicity to aquatic biota, and the lower of the two values was selected as the water criterion.

Soil criteria for bioaccumulative contaminants were calculated from an FRV as previously described for the water ingestion route. The calculation was adapted for a terrestrial system by using an ecological magnification factor (EMF) in place of a BCF in the denominator. The EMF relates residue concentration in plants or soil fauna to residue concentrations in soil. The FRV was then compared to soil criteria derived from direct toxicity, and the lower of the two values was then selected as the soil criterion.

The toxicity assessments for each of the 32 other contaminants of concern are presented in Sections 5.1.1 through 5.1.27. Contaminants that were highly similar or metabolites were combined and addressed as a single unit. The estimated "no effect" concentrations in abiotic media derived through the toxicity assessments are summarized in Table 5.1-3.

5.1.1 ALLYL CHLORIDE

EPA water quality criteria for allyl chloride were unavailable in the literature researched.

Table 5.1-3. Acceptable Concentrations in Abiotic Media

Contaminant	Water (ppb)				Soil (ppm)	
	EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life	Toxicity	Final Residue Value
Allyl Chloride	NA	100	NA	100	NA	NA
Atrazine	NA	1,400	NA	NA	0.02	NA
Azodrin	NA	3.5	NA	49	NA	NA
Cadmium	0.66*	0.76	308	NA	NA	13
Chlordane/Oxychlordane	0.17	300	0.64	NA	NA	NA
Chlorobenzene	2.5	11,000	NA	NA	NA	NA
Chloroform	120	4,800	NA	NA	NA	NA
CPMS/CPMSO/CPMSO2	NA	1,800	NA	NA	0.97	NA
Copper	6.5*	42	340	NA	100	NA
DDT/DDE	0.0010	8	0.0010	NA	NA	4
Dicyclopentadiene	NA	200	NA	100	100	NA
DIMP	NA	8,800	NA	2,570	15.8	NA
DMMP	NA	8600	NA	510	NA	NA
Dithiane	NA	3,360	NA	NA	NA	NA
Ethylbenzene	320	130,000	NA	NA	NA	NA
Heptachlor/Heptachlor Epoxide	0.0052	10	6.3	NA	NA	0.005
Malathion	0.1	12,000	NA	NA	0.025	NA
Methyl Parathion	NA	40	NA	0.0014	NA	NA
Methyl Phosphonic Acid	NA	NA	NA	1,000	NA	NA
Mustard	NA	27	NA	NA	NA	NA
Nitrosodimethylamine	58	64	NA	NA	NA	NA
1,4-Oxathiane	NA	4,800	NA	NA	NA	NA
Parathion	0.013	3.2	NA	NA	NA	NA
Polychlorinated Biphenyls	0.014	0.62	0.014	NA	NA	NA
Toluene	127	5,200	NA	NA	NA	NA
Trichloroethylene	2,190	17,900	NA	NA	NA	NA
Xylene	NA	19,200	NA	82	NA	NA

NA = Not Available.
* Hardness dependent criteria

Source: ESE, 1988.

5.1.1.1 Aquatic Ecosystems

Aquatic toxicity data are available as a TLm96 (concentration lethal to 50 percent of the organisms for a 96-hour (h) exposure expressed as a range due to the variety of test methodology and organisms). The TLm96 is 10 to 100 ppm (Sax, 1984). The aqueous solubility of allyl chloride is 1,000 mg/l, and it decays by hydrolysis with a half-life of 6.9 days at 25 degrees centigrade (^{o}C).

5.1.1.2 Terrestrial Ecosystems

The acute oral LD_{50} for rats is 64 mg/kg (NIOSH, 1984). The LC_{Lo} values for rats and mice for inhalation are 290 ppm for an 8-h and 153 g/m^3 for a 10 minute (min) exposure (NIOSH, 1984). Reproductive effects occur in rats exposed by inhalation to 300 ppm for 7 h on days 6 to 15 of pregnancy (NIOSH, 1984). The LD_{50} for rabbits for dermal exposure is 2,066 mg/kg, and skin is irritated by exposure to 10 mg for 24 h (NIOSH, 1984).

5.1.1.3 Quantification of Toxic Effects

No criteria are established; therefore, an acceptable water concentration was estimated by dividing the TLm96 range by a factor of 10^2 to bring the LC_{50} into the range of NOEL. The estimated acceptable water concentration protective of aquatic organisms thus ranges from 0.1 to 1 ppm (100 to 1,000 parts per billion (ppb). To be conservative, the low end of the range, 100 ppb, is used to estimate an acceptable water concentration for aquatic biota.

For terrestrial biota consuming surface water, the lowest health effects level is the LD_{50} for rats. By using the LD_{50} for rats and a water consumption rate for rats of 0.125 liters per kilogram body weight per day (l/kg bw/day), the estimated acceptable water intake concentration becomes:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{64\ \text{mg/kg bw/day}}{0.125\ \text{l/kg bw/day}} = 512\ \text{mg/l}$$

Uncertainty factors of 1,000 to bring the LD_{50} into the range of a NOEL and of 5 for interspecific variation were applied to yield an estimated acceptable water concentration of 0.10 mg/l (100 ppb).

There is no indication that allyl chloride bioaccumulates; therefore, a Final Residue Value was not calculated. A summary of the estimated acceptable water concentrations (ppb) for allyl chloride is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	100	NA	100

The criterion, 100 ppb, is used to estimate an acceptable water concentration of allyl chloride that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Data were insufficient to calculate soil criteria.

5.1.2 ATRAZINE

EPA water quality criteria have not been established for atrazine, and the EPA Health Advisory for atrazine had been withdrawn at the time this Biota RI was in progress. Tolerance levels for various agricultural products have been established by the EPA (1986b). In meat and meat by-products the tolerance level is 0.2 ppm. In various animal fodders the tolerance level is 15 ppm. The half-life of atrazine in soils ranges from 36 to 167 days depending on soil specific parameters such as water holding capacity (Hurle and Kibler, 1976; Warnock and Leary, 1978). A longer soil half-life of three years has been indicated for atrazine in irrigation ditches (Smith et al., 1975). The aqueous solubility of atrazine is 33 mg/l at 25°C, and it decays by hydrolysis with a half-life in water of 2.5 hours at pH 7 and 25°C.

5.1.2.1 Aquatic Ecosystems

Plants

Algal bioassays performed in natural water indicate a 21-day EC_{50} for growth reduction of 410 ppb, and a 96-h EC_{50} for inhibition of photosynthesis of 854 ppb (Turbak et al., 1986). In studies with four species of submerged estuarine macrophytes, the average 2-h EC_{50} was 95 ppb (Jones and Winchell, 1984), although toxicity to freshwater macrophytes might differ.

Invertebrates

Gunkel and Streit (1980) used a mollusc (Ancylus fluviatilis) to study the effects of uptake of atrazine from water and food. One group of mollusks was fed contaminated food, and the other group was starved but placed in contaminated water. Both groups reached equilibrium in 12 to 24 hours and exhibited concentration factors that were not significantly different. The bioconcentration factor for A. fluviatilis was 2.6.

Fish

Fish (Coregonus feral exposed to atrazine equilibrate with the surrounding water within 50.9 minutes (Gunkel and Streit, 1980). The highest accumulation rates occur in organs with high blood circulation such as liver, brain, gills, intestine and gall bladder, with concentration factors for these organs of 9.1, 3.3, 3.8, 5.2-9.3, and 48.5, respectively (Gunkel and Streit, 1980).

5.1.2.2 Terrestrial Ecosystems

Plants

Atrazine is toxic to grassy weeds and annual broadleaf weeds (Sirons et al., 1973), causing inhibition of photosynthesis (Shimabukuro and Swanson, 1969). Twelve months following application of 3 lb/A active ingredient, soil residues were about 0.2 ppm parent compound, and about 0.05 ppm deethylated atrazine (phytotoxic metabolite) (Sirons et al., 1973). Crop growth in these soils was 40 percent that observed in control fields.

Pea plants exposed to 10^{-7} Molar (M) atrazine in nutrient solution exhibited little phytotoxicity at 19 days, whereas plants exposed to 10^{-6}M atrazine were stunted and highly chlorotic (Shimbukuro, 1967). Oat plants were more susceptible than pea plants exposed to a 10^{-5}M solution; oat plants died in 7 days, whereas pea plants died in 21 days (Shimabukuro, 1967).

Invertebrates

A diet containing 0.01 percent atrazine (100 ppm) fed to larvae of Drosophila melanogaster caused a significant increase in dominant and sex-linked recessive lethal mutations (Murnik and Nash, 1977).

Birds

The LD_{50} values for mallard and ring-necked pheasant exceed 2,000 mg/kg bw (Hudson, et al., 1984).

Mammals

Atrazine is re dily absorbed from the mammalian gastrointestinal tract (EPA, 1987a). After 72 hours, 15.8 percent was retained in the tissues of rats from a single dose of 0.53 mg by oral gavage; highest residues were in liver, kidney, and lung, as compared to muscle and fat (Bakke, et al., 1972). Oral LD_{50} values for rats and mice are 3,000 and 1,750 mg/kg bw, respectively (Bashmurin, 1974). Health effects in rats include pulmonary edema, cardiac dilation, and microscopic hemorrhages in liver and spleen (Molnar, 1971). The LD_{50} for a dermal exposure in rabbits is 7,550 mg/kg bw (Frear, 1969).

Two orally administered doses of 250 mg/kg bw were lethal to sheep and dairy cattle, causing degeneration and discoloration of adrenal glands and congestion in lungs, liver, and kidney (Palmer and Radeleff, 1964). Ten oral doses as low as 500 mg/kg bw to pregnant rats on days 6 to 15 of gestation caused an increase in the number of embryonic and fetal deaths, decreased fetal weight, and retarded skeletal growth (Ciba-Geigy, 1971).

Chronic exposures to atrazine at levels as high as 1,000 ppm in diet (estimated to be 75 mg/kg bw/day (Sax, 1984)) caused no observed effects in rats. No effects on maternal health or fetotoxicity were observed for rats or rabbits dosed with 5 mg/kg bw/day (Woodard Research Corporation, 1966), and 1 mg/kg bw/day, respectively (Ciba-Geigy, 1984). In a 2-year study with dogs, a no effect level of 0.35 mg/kg bw/day was estimated (Woodard Research Corporation, 1964). The lowest concentration of atrazine that caused adverse effects in sheep or cows, fed 10 doses, was 5 and 25 mg/kg bw/day, respectively; the NOEL for cows was 10 mg/kg bw/day (Palmer and Radeleff, 1964). The toxic effects in sheep and cows included muscular spasms, stilted gait, and anorexia.

5.1.2.3 Quantification of Toxic Effects

EPA water quality criteria are unavailable, and the toxicity of atrazine to aquatic biota could not be quantified due to lack of appropriate information.

For terrestrial biota consuming surface water, the lowest atrazine concentration correlating with health effects was a chronic NOEL of 0.35 mg/kg bw/day for dogs. By using the NOEL and the estimated water consumption for dogs, an acceptable water concentration is derived as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.35 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 7 \text{ mg/l}$$

This value is divided by an uncertainty factor of 5 for interspecific variation to yield an acceptable water concentration of 1.4 mg/l (1,400 ppb).

Because atrazine does not appear to bioaccumulate to a significant extent, a Final Residue value was not calculated.

A summary of the estimated acceptable water concentrations (ppb) for atrazine is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	1,400	NA	NA

The only estimated criterion, 1,400 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Owing to the lack of data, this estimate is highly uncertain.

Plant growth was reduced to 40% of that observed in controls in soils containing 0.2 ppm atrazine. Applying an uncertainty factor of 10 yields a soil criteria for atrazine of 0.02 ppm.

5.1.3 AZODRIN (MONOCROTOPHOS)

EPA water quality criteria are unavailable for azodrin. Azodrin is a systemic insecticide with an aqueous solubility of 8,100 mg/l at 25°C. Trimethyl phosphate (TMP), a known mutagen, is a minor contaminant in the processing of azodrin (EPA, 1985h). The EPA (1985h) states that azodrin is extremely toxic to wildlife and aquatic invertebrates, with the primary toxicological concern being cholinesterase inhibition. For mature orange trees sprayed at a rate of 1 lb/acre and 10 lb/acre, residue half-life was found to be 13 and 16 days, respectively (Westlake et al., 1970)

5.1.3.1 Aquatic Ecosystems

Plants

No information was available in the literature reviewed on azodrin.

Invertebrates

The 96-hr LC_{50} for Gammarus fasciatus is 0.3 ppm (Johnson and Finley, 1980).

Fish

The 96-hr LC_{50} values for fathead minnow, bluegill, rainbow trout, and channel catfish are >50, 12.1, 5.2, and 4.93 ppm, respectively (Johnson and Finley, 1980).

5.1.3.2 Terrestrial Ecosystems

Plants

No information was available in the literature reviewed on azodrin.

Invertebrates

No information was available in the literature reviewed on azodrin.

Birds

Hudson et al. (1984) reported LD_{50} values of 0.188 mg/kg bw for the golden eagle (Aquila chrysaetos); however, only six birds were used and sex was not specified. The value represents an acute lethal dose, but is not technically an LD_{50}. Signs of intoxication included fluffed feathers, closed eyes, ataxia, lacrimation, salivation, polydipsia, dyspnea, tracheal congestion, defecation, mydriasis, hyperactive nictitating membrane,

tremors, wing-beat convulsions, tetany or opisthotonos. Gross necropsies revealed endocardial and gastrointestinal hemorrhaging. Toxicity studies indicate a 30-day LOAEL in mallards of 0.25 mg/kg bw/day (Hudson et al., 1984).

In a behavioral study by Kreitzer and Fleming (1988), adult male northern bobwhite (Colinus virginianus) fed 0.18 ppm azodrin in diet exhibited significantly more errors than controls in acquisition and reversal of a learned response. Brain AChE levels were decreased below the critical 40 to 60 percent within 7 days of treatment with the azodrin diet.

Mammals

The oral LD_{50} in the rat and mouse ranges from 5.7 to 17 mg/kg bw in a water formulation (Brown et al., 1970; ACGIH, 1986), and 10 to 23 mg/kg bw in an oil formulation (ACGIH, 1986). LD_{50} values for mule deer and domestic goat are 37.5 and 35 mg/kg bw, respectively (Hudson et al., 1984). Signs of intoxication included ataxia, miosis, hyporeactivity, constant quivering, immobility, tracheal congestion, tachypnea, dyspnea, and phonation. Mortalities usually occurred one to six hours after treatment.

In studies by Johnston (1966) and Johnston (1967a), rats given a concentration of 100 ppm azodrin orally for two years were relatively unaffected based on survival and general health. Treated rats did not gain as much weight as controls, but there were no significant findings post mortem. Plasma and erythrocyte cholinesterase were unaffected at 1 ppm, but were significantly decreased at 10 ppm. Brain cholinesterase levels were also decreased at the 10 ppm dose level. In a 2-year study with beagles (Johnston, 1966; Johnston, 1967b), cholinesterase levels were not affected at a dietary concentration of 1.6 ppm azodrin, but were severely reduced at the next higher concentration of 16 ppm. The EPA (1985h) has established a NOEL of 0.03 ppm (an estimated 0.0022 mg/kg bw/day (Sax, 1984)) for cholinesterase inhibition based on a chronic rat feeding study that showed minor depressive trends in AChE at dose levels of 0.09 ppm. Metabolism studies indicate that azodrin is excreted rapidly and does not appear to accumulate in the body (ACGIH, 1986).

5.1.3.3 Quantification of Toxicological Effects

EPA criteria are unavailable for azodrin; therefore, criteria for aquatic biota were estimated using the 96-h LC_{50} for the most sensitive species tested, the channel catfish. The LC_{50} was 4.93 ppm, and an uncertainty factor of 10^2 was applied to yield an acceptable water concentration of 0.049 ppm
(49 ppb).

The acceptable surface water concentration was derived using the chronic NOEL for rats and the water consumption rate for rats of 0.125 l as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.0022 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 0.018 \text{ mg/l}$$

An uncertainty factor of 5 for was applied for interspecific variation to yield an acceptable water concentration of 0.0035 mg/l (3.5 ppb).

Because there was no indication in the available literature that azodrin bioaccumulates, a Final Residue Value was not calculated.

A summary of the acceptable water concentrations (ppb) for azodrin is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	3.5	NA	49

The lower of the estimated criteria, 3.5 ppb, is used to represent the acceptable water concentration that will be protective of all wildlife populations at RMA.

Soil criteria for azodrin could not be established at this time due to lack of data.

5.1.4 CADMIUM

Cadmium toxicity decreases as hardness increases. The formulas for deriving water quality criteria for the protection of aquatic life are $e^{(1.128[\ln(\text{hardness})]-3.828)}$ as a 1-h average (in ppb), and $e^{(0.7852[\ln(\text{hardness})]-3.490)}$ as a 4-day

average (in ppb) (EPA, 1985b). For example, at hardness levels of 50, 100 and 200 ppm $CaCO_3$, acute criteria are 1.8, 3.9 and 8.6 ppb, respectively (EPA, 1986c). Chronic toxicity criteria at the above hardness levels are 0.66, 1.1 and 2.0 ppb, respectively (EPA, 1986c). Levels of cadmium in waters from mixed industrial areas area as high as 0.45 ppb, whereas in remote streams levels can be as high as 0.1 ppb (Moore and Ramamoorthy, 1984). Levels of cadmium in various U.S. soils range from 0.41 to 0.57 ppm (Kabata-Pendias and Pendias, 1984).

5.1.4.1 Aquatic Ecosystems

Plants

The 96-h EC_{50} values for aquatic plants range from 105 ppb in the green alga *Chlorella saccharophila* to 480 ppb in the diatom *Nitzschia costerium* (Rachlin *et al.*, 1984, 1982). A 96-h EC_{50} of 3,700 ppb is observed for *Chlorella vulgaris* at a hardness of 50 ppm $CaCO_3$ (Canton and Sloof, 1982).

Levels as low as 5 ppb cadmium produce a significant reduction in algae at hardness levels of 11.1 ppm $CaCO_3$ (Giesy *et al.*, 1979). At 10 ppb cadmium, growth reduction was observed in a fern (*Salvina natans*) and duckweed (*Lemna valdiviana*) (Hutchinson and Czyrska, 1972).

Bioconcentration factors in *S. natans* and *L. valdiviana* are 960 and 603, respectively, for a 21-day exposure to $Cd(NO_3)_2$ (Hutchinson and Czyrska, 1972). Attached microscopic aquatic plants and animals concentrated cadmium by factors of 580 to 720 in a 365-day exposure to $CdCl_2$ (Giesy *et al.*, 1979).

Invertebrates

The 48-h LC_{50} for a tubificid worm (*Tubifex tubifex*) is 320,000 ppb as $CdCl_2$ at a hardness of 224 ppm $CaCO_3$ (Qureshi *et al.*, 1980). The 72-h LC_{50} for a copepod, *Acanthocyclops viridis*, is 0.5 ppb as $CdSO_4$ (Braginsky and Scherban, 1978). EC_{50} values for *D. magna* range from 5 ppb as $CdCl_2$ (Attar and Maly, 1982) to 160 ppb as $Cd(NO_3)_2$ (Bellavere and Gorbi, 1981).

Reduced survival occurs at levels as low as 0.2 ppb cadmium in the cladoceran *Moina macrocopa* (Hatakeyama and Yasuno, 1981), and reduced reproduction is observed at 0.17 to 1 ppb cadmium in *Daphnia pulex*

(Biesinger and Christensen, 1972; Bertram and Hart, 1979). Levels of cadmium below 5 ppb are toxic to worms and copepods (Giesy et al., 1979), and crayfish (Thorpe et al., 1979).

Bioconcentration factors for $CdCl_2$ range from 164 for the beetle Dytiscidae sp. (Giesy et al., 1979) to 4,190 for the caddisfly, Hydropsyche sp. (Spehar et al., 1978).

Fish

Cadmium levels of 0.2 ppb reduce survival in rainbow trout (Salmo gairdineri) (Birge, et al., 1981). Levels of 0.7 to 1.0 ppb cadmium are lethal to 10 percent of a population (LC_{10}) of rainbow trout (Chapman, 1978); LC_{50} values for rainbow trout for various compounds are less than 7 ppb (Kumada et al., 1980, 1973; Chapman and Stevens, 1978). In fathead minnows (Pimephales promelas), LC_{50} values range from 40.9 ppb (Spehar, 1982) to 2,200 ppb Cd (Sloof et al., 1983).

Whole body concentration factors of 33 to 540 have been observed for rainbow trout (Kumada et al., 1973, 1980). Bioconcentration factors of 1,900 and 2,200 are reported in mosquito fish (Gambusia affinis) (Giesy et al., 1979).

5.1.4.2 Terrestrial Ecosystems

Plants

Plants grown near zinc smelters in soil contaminated with cadmium levels as high as 710 ppm concentrated cadmium by factors of 8.1 in leaves and 1.2 in berries (Beyer et al., 1985). Plants grown on cadmium contaminated soil containing 1.11 ppm and irrigated with wastewater containing 280 ppb accumulated cadmium levels up to 6.4 times higher than controls (Shariatpanahi and Anderson, 1986). Toxic effects on plants were not observed in these studies.

Invertebrates

Cadmium is highly mobile within the invertebrate food web and shows significant accumulation in invertebrates from a wide range of taxonomic groups (Hunter et al., 1987a). Seasonal patterns of accumulation closely follow seasonal trends in metal contamination levels in the indigenous

vegetation (Hunter et al., 1987b). Beyer et al. (1985) determined that detritus feeders and their predators are most likely to have high cadmium concentrations. Van Hook (1974) found concentration factors in earthworms ranging from 11.6 to 22.5 on a dry weight basis. Concentration factors of 33 were found in earthworms from soil treated with sewage sludge (Anderson, 1979). Earthworms exposed to 5 ppm cadmium for 26 days were found to concentrate cadmium by factors of 86.4 (Lumbricus rubellus) and 84.6 (Allolobophora caliginosa) on a dry weight basis (Ireland and Richards, 1981).

Birds

Concentrations of 0.08 (control), 1.6, 15.2, or 210 ppm $CdCl_2$ in the diet of mallard ducks for up to 90 days had no significant effect on body weight, mortality, hematocrit, or hemoglobin levels (White and Finley, 1978). Testes of males in all treated groups weighed less than controls. At the highest dose level, kidney weights and egg production by females were lower than controls. The estimated total daily cadmium intake (based on an average body weight of 1,153 g and daily food intake of 110 g) for the control, 1.6, 15.2, and 210 ppm treatment group was 0.0076, 0.15, 1.4, and 20.0 mg/kg bw/day, respectively.

In another study with mallard ducks, cadmium levels of 50 ppm in the diet enhanced lipid mobilization during food restriction (Di Giulio and Scanlon, 1985), and increased adrenal corticosterone concentrations, which suggested increased gluconeogenesis. Food restricted ducks weighed an average of 1,000 g over a 42 day test period and received 60 g of cadmium contaminated ration daily. Total daily cadmium intake was estimated to be approximately 3 mg/kg bw/day.

Mammals

In immature voles (Microtus pennsylvanicus), diets containing 1.09 to 2.76 ppm resulted in liver concentrations of 0.26 to 2.13 mg/kg, and kidney concentrations of 0.42 to 3.69 mg/kg (Williams et al., 1978). Vole body weight was 14 g at the start of the test, and increased by approximately 0.33 g daily for 40 days, resulting in an estimated final weight of 27 g. Maximum daily cadmium intake from the 1.09 ppm diet was 5.76 ug, and intake

from the 2.76 ppm diet was 16.67 ug (from a final body weight of 27 g, daily intake becomes 0.21 to 0.62 mg/kg bw/day) resulted in kidney concentrations of 3.69 mg/kg. No adverse effects were observed for the 40 day study. The estimated concentration factors (N=8) for liver and kidney were 0.33 and 0.62, respectively.

Insectivorous mammals accumulate higher cadmium concentrations than herbivorous mammals (Andrews et al., 1984). In the common shrew (Sorex araneus), dietary levels of 23.2 ppm resulted in liver and kidney concentrations of 234 and 158 ppm (concentration factors of 10.1 and 6.8), respectively. No adverse health effects were reported. The shrew consumes an amount approximately equivalent to 75 percent of its body weight daily, and 150 percent of its body weight during lactation (Andrews et al., 1984). Shrews weigh approximately 6 g (Palmer and Fowler, 1975); therefore, an estimate of cadmium intake of 17.4 mg/kg bw/day for nonlactating and 34.8 mg/kg bw/day for lactating animals can be obtained. The concentrations observed in kidney (158 ppm) at exposures of 23.2 ppm are less than the 200 ppm critical level for human kidney (Hammond and Beliles, 1980).

5.1.4.3 Quantification of Toxic Effects

The EPA criteria for the protection of aquatic organisms and their uses represent acceptable water concentrations of cadmium for aquatic life.

Subchronic dietary levels of 0.08 ppm (0.0076 mg/kg bw/day) for mallards, or 1.09 to 2.76 ppm (0.21 to 0.62 mg/kg bw/day) for mammals, resulted in no observed effects. Since the subchronic LOAEL for mallard (1.6 ppm or 0.15 mg/kg bw/day) is lower than the NOEL for mammals, the mallard is selected as the most sensitive species. The acceptable water concentration based on surface water ingestion is obtained by using the NOEL and the water intake for mallard ducks as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.0076 \text{ mg/kg bw/day}}{0.200 \text{ l/kg bw/day}} = 0.038 \text{ mg/l}$$

This value is divided by an uncertainty factor of 10 to bring the subchronic NOEL into the range of a chronic NOEL and 5 for interspecific variation, to yield an acceptable water concentration of 0.00076 mg/l (0.76 ppb).

A Final Residue Value has been calculated by EPA based on a dietary level for mallard ducks of 200 ppm and a mean BCF for mallard prey items of 648.6 (EPA, 1985b). A summary of the acceptable water concentrations (ppb) of cadmium is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.66	0.76	308	NA

The lowest of the estimated criteria, 0.66 ppb cadmium, is based on a hardness of 50 ppm $CaCO_3$, and will vary as hardness changes. Because toxicity to aquatic organisms is hardness dependent, the subchronic criterion for surface water ingestion, 0.76 ppb, is used to estimate an acceptable water concentration protective of all wildlife populations at RMA.

Soil criteria were estimated by using a Final Residue Value calculation as described by EPA for bioaccumulative contaminants in aquatic ecosystems. The dietary concentration in small mammals that resulted in no observed effects was 23.2 ppm for shrews. Terrestrial invertebrates, represented by earthworms, concentrate cadmium residues by factors of 11.6 to 86.4 (geometric mean of 36, N = 5) on a dry weight basis. By assuming that earthworms are 95 percent water (Beyer et al., 1987), a geometric mean concentration factor on a wet weight basis is 1.8 (N = 5). The Final Residue Value is calculated as follows:

$$\frac{MPIC}{BAF} = \frac{23.2\ ppm}{1.8} = 13\ ppm$$

The acceptable soil criterion for cadmium based on bioaccumulation in a terrestrial ecosystem is 13 ppm. This level will probably be protective of bird populations as well, as only minor effects on mallard ducks were observed when birds were fed dietary concentrations of 1.6 and 15.2 ppm.

5.1.5 CHLORDANE/OXYCHLORDANE

The criterion for the protection of aquatic life is 0.0043 ppb as a 24-h average, not to exceed 2.4 ppb at any time (EPA, 1986c). The Final Chronic Value is 0.17 ppb (EPA, 1980aa). Chlordane is very persistent in the aquatic environment; in river water in which chlordane was applied, 85 percent remained after 8 weeks (Eichelberger and Lichtenberg, 1971). Another study indicated that the half-life of chlordane in water is 28-33 h (Atlas et al., 1982). The half-life of chlordane in soil is several years (Sanborn et al., 1977), and the half-life in biological tissue is 23 days (Barnett and Dorough, 1974). Oxychlordane is evaluated with chlordane because it is a persistent metabolite, more toxic than the parent compound, that can be formed by metabolism of several of the chlordane compounds (Stickel et al., 1983). The solubility of chlordane is 1.85 mg/l at 25°C.

5.1.5.1 Aquatic Ecosystems

Plants

Little information on the effects of chlordane on aquatic plants was found. A study by Glooschenko and Lott (1977), indicated that a concentration of 0.1 ppb stimulated growth in freshwater algae. A bioconcentration factor of 5,560 (dry weight basis) has been observed for algae (Moore et al., 1977).

The bioconcentration factor can be converted to a wet weight basis of 1,900 by assuming that algae are approximately 65.7 percent water (Isensee et al., 1973). Other data indicate that a conversion factor of 0.1 should be used for plankton (Stephan et al., 1985); thus, reducing the BCF to 556.

Invertebrates

The 96-h LC_{50}s for Gammarus fasciatus and Pteronarcys sp. are 40 and 20 ppb, respectively (Johnson and Finley, 1980). For the invertebrate Simocephalus sp., the 48-h EC_{50} is 20 ppb (Johnson and Finley, 1980). At concentrations of 1.7 ppb in water, chironomid larvae exposed for 25 days exhibited increased mortality (EPA, 1980aa). The chronic value for D. magna is 16 ppb chlordane (Cardwell et al., 1977). A bioconcentration factor of 24,000 (dry weight basis) has been observed for Daphnia (Moore et al., 1977). By

assuming that Daphnia have the same water content as algae, 65.7 percent (Isensee et al., 1973), a BCF of 8,200 on a wet weight basis is estimated. Other data indicate a dry weight conversion factor of 0.1 for plankton, which would result in a BCF of 2,400 (Stephan et al., 1985).

Fish

LC_{50} values for fish range from 3 ppb for carp (Cyprinus carpio) and largemouth bass, to 190 ppb for the guppy (EPA, 1980aa; Johnson and Finley, 1980). Fathead minnow, bluegill, and rainbow trout, have LC_{50} values of 115, 57, and 42 ppb, respectively (Johnson and Finley, 1980).

The chronic value for bluegills is 1.6 ppb (Cardwell et al., 1977). At a concentration of 0.32 ppb in water, reduced embryo viability was observed in brook trout (Salvelinus fontinalis) for a 13-month exposure (Cardwell et al., 1977). Concentration factors of 4,700 have been observed in aquatic organisms with a 1 percent lipid content (EPA, 1980aa). Chronic exposure of a freshwater Indian fish to 17 ppb chlordane resulted in decreased blood triglycerides and increased free fatty acids and magnesium (Bansal et al., 1979).

5.1.5.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of chlordane to plants was available in the literature researched.

Invertebrates

Chlordane residues are toxic to invertebrates 30 years or more after application to the soil (Mampe, 1987). In the American cockroach (Periplaneta americana), chlordane is metabolized to more than 25 products (Feroz and Khan, 1979).

Birds

Half of the starlings (Sturnus vulgaris) dosed with HCS-3260, a mixture of cis-and trans-chlordane, died within 5 days at 500 ppm in diet (Stickel et al., 1983). For technical chlordane (a mixture of cis- and trans-chlordane, heptachlor, and other organochlorines), half the exposed birds for several

species died within 6 to 7 days at 150 ppm in diet (Stickel et al., 1979b). At 14 days, half the starlings dosed with HCS-3260 died at 200 ppm in diet (Stickel et al., 1983). These values do not necessarily reflect LC_{50} values because remaining birds were sacrificed when half of the population died (Stickel et al., 1979b). By using an intake of 175 g/kg bw/day derived from chickens (Sax, 1984), dietary concentrations of 150, 200, and 500 ppm become 26, 35, and 88 mg/kg bw/day.

Chlordane is metabolized in birds to heptachlor epoxide, oxychlordane, trans-nonachlor, cis-chlordane, and other compounds (Stickel et al., 1979b). Heptachlor epoxide and oxychlordane appear to be the metabolites correlated with mortality; brain concentrations diagnostic of poisoning for birds begin near 5.0 ppm oxychlordane on a wet weight basis (Stickel et al., 1979b; Stickel et al., 1983). From data in Stickel et al., 1983, a brain to carcass ratio was estimated to be a geometric mean of 0.16 (N=7); at 5 ppm in brain, the estimated lethal level in carcass is 31 ppm. Residues of heptachlor epoxide diagnostic of poisoning are 8 to 9 ppm in brain (Stickel et al., 1979b). Nonachlor is not highly toxic to birds, although it is metabolized to oxychlordane (Stickel et al., 1983).

Mammals

The acute oral LD_{50} of rats, mice, and hamsters is 350, 390, and 1,720 mg/kg bw respectively (Claude, 1976). The toxicity of chlordane is a function of the configurational purity of the compound; for instance, the LD_{50} in rats from pure cis-chlordane is 83 mg/kg bw (Podowski et al., 1979), while the LD_{50} for chlordane of unspecified purity is 560 mg/kg bw (Ambrose et al., 1953a). Rats stressed by a low protein diet (3.5% protein for 28 days) had an LD_{50} of 137 mg/kg bw, while rats fed commercial rodent chow had an LD_{50} of 311 mg/kg bw (Boyd and Taylor, 1969). Acute symptoms include central nervous system stimulation as evidenced by irritability, tremors, and convulsions (Stohlman et al., 1950).

No effects are reported in rats at dietary levels of 1.2 ppm (approximately 0.09 mg/kg bw/day based on consumption data in Sax (1984)) (DeLong and Ludwig, 1954). At dietary concentrations of 5 to 10 ppm (approximately 0.375 to 0.75 mg/kg bw/day (Sax, 1984), occasional hypertrophy of

hepatocytes and increased liver weight occurs in rats (Ingle, 1952; Ambrose et al., 1953 a,b). In a 2- year study by Ingle (1952), after 80 weeks at concentrations of 30 ppm in diet (2.25 mg/kg bw/day (Sax, 1984)), rats exhibited slight tremors. At 150 ppm (11.25 mg/kg bw/day (Sax, 1984)), decreased growth rate, anorexia, and tremors were observed. Also, liver and kidney hypertrophy, and moderate to marked kidney, lung, myocardial, adrenal and spleen damage were observed. In mice, significantly increased liver weights occur in females at 5 ppm and in males at 25 ppm in diet (0.6 and 3.0 mg/kg bw/day (Sax, 1984)) after 18 months of exposure (Epstein, 1976) at concentrations of 25 ppm diet and greater, benign proliferative lesions occur in the liver of mice (Becker and Sell, 1979). A review panel for WHO/FAO indicate that 3 ppm in diet (estimated as 0.075 mg/kg bw/day (Sax, 1984)) for 2 years is the NOEL for dogs (Wazeter, 1968).

5.1.5.3 Quantification of Toxic Effects

The EPA Final Chronic Value (0.17 ppb) represents acceptable water concentrations for aquatic life. The criteria for the protection of aquatic organisms and their uses are derived from the Final Residue Value (0.0043 ppb), which is based on human consumption, and so are considered inappropriate for this analysis.

The chronic NOEL for dogs was 0.075 mg/kg bw/day. Using the NOEL for dogs and the water intake for dogs, the acceptable water concentration was estimated as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.075 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 1.5 \text{ mg/l}$$

This value is divided by a factor of 5 for interspecific variation to yield an estimated acceptable water concentration of 0.30 mg/l (300 ppb).

Because chlordane appears to bioconcentrate significantly, a Final Residue Value of 0.0043 ppb has been calculated by EPA (1980aa). The value is based on FDA guidelines for human consumption, and is therefore considered inappropriate for this analysis. By using the dietary NOEL for dogs (3 ppm) as a concentration protective of both mammals and birds, and the geometric mean BCF reported in EPA (4,702), a Final Residue Value is calculated as follows:

$$\frac{\text{MPTC}}{\text{BCF}} = \frac{3 \text{ ppm}}{4{,}702} = 0.00064 \text{ ppm}$$

A summary of the acceptable water concentrations (ppb) for chlordane is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.17	300	0.64	NA

The lowest of the estimated criteria, 0.17 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA.

Data were insufficient to calculate soil criteria for chlordane.

5.1.6 CHLOROBENZENE

There are no published criteria concerning chlorobenzene for the protection of freshwater aquatic life (EPA, 1980ab). The available data for chlorinated benzenes, including mono-, di-, tri-, tetra-, penta- and hexachlorobenzene, indicate that acute toxicity occurs at concentrations as low as 250 ppb and would occur at lower concentrations among species that are more sensitive than those tested (EPA, 1980ab).

The half-life of chlorobenzene in air is 3.5 days (Kanno and Nojima, 1979) and in water 0.3 days (Zoetman *et al.*, 1980). The dominant loss mechanism from the soil surface is evaporation with a half-life estimated to be several months (Wilson *et al.*, 1981). Chlorobenzene is expected to partition rapidly to air when released to surface water (EPA, 1984b).

5.1.6.1 Aquatic Ecosystems

Plants

The average 96-h EC_{50} for the alga Selenastrum capricornatum is 228 ppm chlorobenzene; effects were reduction in cell number and in chlorophyll a production (EPA, 1980ab). Research by Calamari *et al.* (1983) on growth inhibition of S. capricornatum indicated a 96-h EC_{50} of 12.5 ppm, while the

NOEL was less than 6.8 ppm. Bringmann and Kuhn (1980) found that concentrations of 120 ppm caused incipient growth inhibition of Microcystis aeruginosa.

A bioconcentration factor of 4,185 is reported for the alga Oedogonium cardiacum (Lu and Metcalf, 1975; EPA, 1977).

Invertebrates

The 48-h EC_{50} and 24-h EC_{50} for Daphnia magna exposed to chlorobenzene are 86 ppm (EPA, 1978) and 140 ppm (Le Blanc, 1980). In toxicity tests by Calamari et al. (1983), D. magna exhibited a 24-h EC_{50} of 4.3 ppm, while a concentration of 2.5 ppm reduced fertility 50 percent in 14 days. The 48-h LC_{50} for Brachydanio rerio is 10.5 ppm (Calamari et al., 1983).

Bioconcentration factors in snails (Physa sp.), D. magna, and mosquito larvae (Culex quinquifasciatus) are 1,313, 2,789 and 1,292, respectively (Lu and Metcalf, 1975; EPA, 1977).

Fish

In goldfish, guppy and bluegill, 96-h LC_{50}s are 51.6, 45.5 and 15.9 to 24 ppm, respectively (Pickering and Henderson, 1966; EPA, 1978). For rainbow trout and largemouth bass LC_{50}s are 0.1 and 0.7 ppm, respectively (Birge et al., 1979a). In another study, the LC_{50} for rainbow trout was 4.1 ppm (Calamari et al., 1983). Hardness does not significantly effect toxicity as evidenced in a study by Pickering and Henderson (1966). In a 7.5 day study, LC_{50}s for embryonic goldfish and largemouth bass ranged from 0.88 to 1.04 ppm and 0.05 to 0.06 ppm, respectively (Birge et al., 1979b). Embryonic trout exposed for 16 days to 0.09 ppm (90 ppb) chlorobenzene exhibited 100 percent mortality (Birge et al., 1979b). A 30-day exposure to 2 and 3 ppm chlorobenzene caused damage to the liver of rainbow trout and largemouth bass (Dalich, 1982).

The bioconcentration factor for chlorobenzene for mosquito fish is 645 (Lu and Metcalf, 1975; EPA, 1977).

5.1.6.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of chlorobenzene was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of chlorobenzene was available in the literature reviewed.

Birds

No information regarding the toxicity of chlorobenzene was available in the literature reviewed.

Mammals

The LD_{50} for chlorobenzene in rats is 3,400 mg/kg bw (Vecerek et al., 1976). Toxic effects include necrosis of the liver and interference with porphyrin metabolism (Rimington and Ziegler, 1963; Khanin, 1969; Knapp et al., 1971). From inhalation studies, LC_{50} for guinea pig and mouse are 0.05 ppm (Rozenbaum, et al., 1947) and 20 ppm (Lecca-Radu, 1959), respectively.

In long-term toxicity studies with rats, a dietary concentration of 50 mg/kg bw/day chlorobenzene for 93 to 99 days caused increased liver and kidney weight (Knapp et al., 1971). In other studies, no effects are reported at 50 mg/kg bw/day for rats (Monsanto Company, 1967), or 60 mg/kg bw/day for mice and rats (NTP, 1983). Studies with mice indicate dietary concentrations of 42.9 mg/kg bw/day for 13 weeks can cause hepatic necrosis (NTP, 1983). A chlorobenzene concentration of 357 mg/kg bw/day is 100 percent lethal to males within 1 week and causes reduced weight gain, polyuria in females, increased liver weights, lesions of the liver, kidney, bone marrow, spleen and thymus (NTP, 1983). Research by Monsanto Company (1967) and Knapp et al. (1971) indicated dogs fed diets containing 27.3, 54.6 and 272.5 mg/kg bw/day chlorobenzene for 90 days had effects at the two highest dose levels. At the 272.5 mg/kg concentration, mortality occurred in 3 to 5 weeks. The 54.6 mg/kg concentration caused diarrhea, vomiting and conjunctivitis; no effects were observed at the lowest concentration.

5.1.6.3 Quantification of Toxic Effects

EPA criteria are unavailable for the protection of aquatic organisms. The EPA acute LOAEL of 250 ppb is divided by an uncertainty factor of 10^2 to yield an acceptable water concentration of 2.5 ppb. The acceptable water concentration is more than an order of magnitude lower than the 7.5-day LC_{50} of 50 ppb for embryonic largemouth bass.

To calculate toxicity due to surface water ingestion, the subchronic NOEL for dogs of 27.3 mg/kg bw/day, and the water intake for dogs, was used. The acceptable water concentration was estimated as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{27.3 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 546 \text{ mg/l}$$

This value is then divided by an uncertainty factor of 10 to bring the subchronic NOEL into the range of a chronic NOEL, and 5 for interspecific variability, to yield an acceptable water concentration of 11 mg/l (11,000 ppb).

Because chlorobenzene appears to bioconcentrate significantly, a Final Residue Value should have been calculated by EPA (1980ab). No value is available; however, probably because data were insufficient to calculate a MPTC.

A summary of the acceptable water concentrations (ppb) for chlorobenzene is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
2.5	11,000	NA	NA

The lowest of the estimated criteria, 2.5 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the limited data available, this estimate is highly uncertain.

Data were insufficient to calculate soil criteria.

5.1.7 CHLOROFORM

Chloroform causes acute and chronic toxicity to aquatic organisms at concentrations of 28.9 and 1.24 ppm, respectively, although toxicity may occur at lower concentrations for more sensitive species than those tested (EPA, 1980b). The available data are inadequate to establish a freshwater aquatic life criterion (EPA, 1985).

There is no appreciable decomposition of chloroform at ambient temperatures in water even in the presence of sunlight (Hardie, 1964). Volatilization into the atmosphere is the major transport process for the removal of chloroform from aquatic systems (EPA, 1979a). The half-life for chloroform in rivers and lakes is 0.3-3 and 3-30 days, respectively (Zoetman et al., 1980). The half-life in soil is not available (EPA, 1984c). The aqueous solubility of chloroform is 870 mg/l.

5.1.7.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of chloroform was available in the literature reviewed.

Invertebrates

In a 48-hr static test, the LC_{50} for Daphnia magna is 28.9 ppm (EPA, 1978).

Fish

In toxicity studies by Bently, et al., (1975), LC_{50}s for rainbow trout were determined to be 66.8 and 43.8 ppm, and for bluegill 115 and 100 ppm. In a 27-day study in hard and soft water, LC_{50} for rainbow trout embryo-larvae were 2.03 and 1.24 ppm, respectively (Birge et al., 1979b). Rainbow trout eggs exposed to 10.6 ppm chloroform 20 minutes after fertilization to 8 days after hatching had a 40 percent incidence of teratogenesis at hatching (Birge et al., 1979b).

The equilibrium bioconcentration factor for the bluegill is 6, with a tissue half-life of less than one day (EPA, 1978). To date there is no evidence for biomagnification in aquatic food chains (EPA, 1985d).

5.1.7.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of chloroform was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of chloroform was available in the literature reviewed.

Birds

No information regarding the toxicity of chloroform was available in the literature reviewed.

Mammals

Chloroform is lipid soluble and passes readily through cell membranes to produce narcosis of the central nervous system (Cornish, 1975), depletion of liver glutathione (Ilett et al., 1973), gonadal and bone marrow abnormalities (Palmer et al., 1979), and carcinomas of several tissues. Gastrointestinal absorption is slower than inhalation but absorption approximates 100 percent (Fry et al., 1972) and lethal tissue level can be reached in minutes to a few hours (VonOettingen, 1955a). Animals on high fat or protein poor diets appear to be more susceptible to hepatotoxicity, while high carbohydrate and protein diets have a protective effect (VonOettingen, 1964).

Intragastric introduction of chloroform to rats caused renal and hepatic tissue pathological changes at a concentration of 250 mg/kg bw; the acute oral LD_{50} was 2,000 mg/kg bw, with death resulting within 2 hours (Torkelson et al., 1976). Oral doses of 126 mg/kg bw/day in pregnant rats caused maternal toxicity but no embryocidal or teratogenic effects; fetal toxicity, hepatitis, and death of dams occurred at 316 mg/kg bw/day (Thompson et al., 1974). A 13- week study with Sprague-Dawley rats given chloroform orally demonstrated that 30 mg/kg bw/day had no effects (Palmer et al., 1979). The next higher dose, 150 mg/kg bw/day, caused increased liver weight with fatty

necrosis, gonadal atrophy, and cellular proliferation in the bone marrow. Oral doses of chloroform greater than 100 mg/kg bw/day in female rabbits were toxic to dam and fetus (Thompson et al., 1974).

Inhalation studies with mice show that at concentrations of 8,000 ppm the mice died within 3 hours and at 12,500 ppm within 2 hours (VonOettingen, 1955a). Pregnant rats exposed to 30 ppm in the ambient air had a significant incidence of fetal abnormalities including delayed skull ossification and rib abnormalities in fetuses (Schwetz et al., 1974). Inhalation of 50 ppm had no effect on male or female rabbits, while the next higher dose of 85 ppm caused pneumonitis, hepatic, and renal pathology (Torkelson et al., 1976).

Dermal applications of 1,000 ppm body weight cause degenerative changes in kidney tubules of exposed rabbits (Torkelson et al., 1976).

5.1.7.3 Quantification of Toxic Effects

EPA criteria for the protection of aquatic organisms and their uses are unavailable for chloroform. Therefore, the chronic LOAEL (1.24 ppm) was divided by an uncertainty factor of 10 to bring the value into the range of NOEL. The resulting acceptable water criterion for the protection of aquatic organisms is 0.12 ppm (120 ppb).

Water criteria based on surface water ingestion were calculated using the health effects data for rats. The subchronic NOEL was 30 mg/kg bw/day for rats during a 13-week study. The acceptable water concentration is derived using the NOEL and the daily water intake for rats as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{30 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 240 \text{ mg/l}$$

By dividing by uncertainty factors of 10 to bring the subchronic NOEL into the range of a chronic NOEL, and 5 for interspecific variation, an acceptable water concentration of 4.8 mg/l (4,800 ppb) is obtained.

There is no indication that chloroform bioaccumulates significantly; therefore, a Final Residue Value was not calculated.

A summary of the acceptable water concentrations (ppb) for chloroform is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
120	4,800	NA	NA

The lower of the estimated criterion, 120 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the limited data available, this estimate is highly uncertain.

Data were insufficient to calculate a soil criterion.

5.1.8 CHLOROPHENYL METHYL SULFIDE, CHLOROPHENYL METHYL SULFOXIDE, AND CHLOROPHENYL METHYL SULFONE (CPMS, CPMSO, $CPMSO_2$)

EPA water quality criteria were unavailable in the literature reviewed. These chemicals behave very differently in the environment. For example, the aqueous solubilities estimated for CPMS, CPMSO, and $CPMSO_2$ are 12, 1,050-1,200, and 1,050-1,170 ppm, respectively (EBASCO, 1987). Persistence data indicate half-life of CPMS ranges from 1 to >5 months (EBASCO, 1987). Half-life estimates for $CPMSO_2$ range from >5 months to 1 year, and for CPMSO the estimate is >5 months (EBASCO, 1987).

5.1.8.1 Aquatic Ecosystems

Plants

No information was available in the literature reviewed.

Invertebrates

No information was available in the literature reviewed.

Fish

No information was available in the literature reviewed.

5.1.8.2 Terrestrial Ecosystems

Plants

Guenzi et al. (1981) examined the effects of the three sulfur compounds on several commercial crops. Toxic effects were determined by measurements of plant height, phytotoxicity symptoms in leaves, and biomass; little

difference was noted in the relative toxicity of the three contaminants. Alfalfa was the most sensitive plant tested and corn the most resistant. Mean soil concentrations for the three chemicals that correlated with a 20 percent growth reduction for alfalfa, fescue, sugar beets, and wheat were 4.7, 6.3, 7.3, and 15.5 ppm (Guenzi et al., 1981). Corn exhibited 20 percent growth reduction at 25 ppm sulfone in soil.

Birds

No information was available in the literature reviewed.

Mammals

The LD_{50} for sulfoxide for male and female rats is 611 and 463 mg/kg bw, respectively (Thake et al., 1979, RIC#81266R06). The LD_{50} for sulfoxide for male and female mice is 328 mg/kg bw and 440 mg/kg bw (Thake et al., 1979, RIC#81266R06). Another study reported a higher LD_{50} value of 933 mg/kg bw (a range of 852 to 1,020 mg/kg bw) for sulfoxide for mice (Miller et al., 1976, RIC#81322R07). Sax (1984) reports acute oral LD_{50} values for the sulfide analog of 400 to 479 mg/kg bw for rats, and 672 mg/kg bw for mice. The sulfone exhibits similar acute LD_{50} values of 400 mg/kg bw for rats and 606 mg/kg bw for mice (Sax, 1984).

Rats and mice subchronically dosed with CPMSO at 750 ppm in diet (estimated to be 56 and 90 mg/kg bw/day, respectively (Sax, 1984)) had increased liver and kidney weights, lesions of the liver, and increased serum mineral and glutamate-oxalate transaminase levels (Thake et al., 1979, RIC#81266R06). Lethal dietary levels for rat and mouse were 3,000 and 5,000 ppm, (approximately 225 and 600 mg/kg bw/day (Sax, 1984)) respectively (Thake et al., 1979, RIC#81266R06). $CPMSO_2$ caused induction of the hepatic microsomal enzyme system in rats (Kimura et al., 1983).

CPMS and $CPMSO_2$ are absorbed through the gastrointestinal tract in cattle, with CPMS oxidizing to $CPMSO_2$ (Oehler and Ivie, 1983). The sulfone did not metabolize further, but distributed in tissues and was slowly excreted by kidneys. In cattle, 1 to 3 percent of the administered dose was excreted into milk in 4 days as the sulfone (Oehler and Ivie, 1983).

5.1.8.3 Quantification of Toxic Effects

No EPA criteria have been established, and aquatic life data were unavailable in the literature reviewed; therefore, criteria for the protection of aquatic biota could not be established.

For terrestrial biota consuming surface water, criteria were based on health effects data for rats. The subchronic LOAEL was 56 mg/kg bw/day in diet for rats, and resulted in sublethal effects. Using a water consumption rate for rats of 0.125 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{56 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 448 \text{ mg/l}$$

By dividing with uncertainty factors of 50 to convert the subchronic LOAEL to a chronic NOEL, and 5 for interspecific variation, an acceptable water concentration of 1.8 mg/l (1,800 ppb) is obtained.

There is no indication that the sulfur compounds bioaccumulate to a significant extent; therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for the sum of the sulfur compounds is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	1,800	NA	NA

The only estimated criterion, 1,800 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain, and may not be protective of aquatic life.

Soil criteria were based on the geometric mean of the mean concentrations of the three sulfur compounds in soil that correlated with 20 percent growth reduction of plants (9.7 ppm). An uncertainty factor of 10 was applied to bring the LOAEL into the range of an NOEL. The acceptable soil concentration is 0.97 ppm.

5.1.9 COPPER

The toxicity of copper to aquatic organisms is due primarily to the cupric (Cu^{2+}) ion, and possibly to some of the hydroxy complexes (EPA, 1985bb). The cupric ion complexes readily with inorganic and organic components in natural water, and adsorbs to suspended solids. The toxicity of copper in water is dependent on parameters such as chemical speciation, seasonal changes in precipitating agents, pH, suspended solids, and alkalinity (EPA, 1985bb; Callahan et al., 1979). In natural waters, copper concentrations range from 0.5 to 1 ppb, and can exceed 2 ppb in urban areas (Moore and Ramamoorthy, 1984). Mean soil levels in the western U.S. are 21 ppm (O'Leary and Meier, 1986).

The criteria for the protection of aquatic freshwater organisms and their uses are estimated by $e^{(0.8545[\ln(hardness)]-1.465)}$ as a four-day average concentration (ppb) not to be exceeded more than once every three years (EPA, 1985bb). The acute criteria are estimated by $e^{(0.9422[\ln(hardness)]-1.464)}$ as a one-h average (ppb) not to be exceeded more than once every three years (EPA, 1985bb). At hardness of 50, 100, and 200 ppm as $CaCO_3$, acute criteria are 9.2, 18, and 34 ppb, respectively, while chronic criteria are 6.5, 12, and 21 ppb, respectively.

5.1.9.1 Aquatic Ecosystems

Plants

Concentrations of copper ranging from 1 to 8,000 ppb inhibit growth of various plant species (EPA, 1985bb). The alga, *Chlorella vulgaris*, exhibits 50 percent growth inhibition when exposed to 100 to 200 ppb (Stokes and Hutchinson, 1976). Depressed growth was observed in *Chorella pyrenoidosa* at concentrations of 1 ppb (Steeman-Nielsen and Wium-Andersen, 1979). Photosynthetic oxygen production was reduced by 50 percent in the macrophyte, *Elodea canadensis*, at concentrations of 150 ppb (Brown and Rattigan, 1979). Using criteria for copper concentrations in water that are protective of aquatic animals will protect aquatic plants (EPA, 1986d).

Bioconcentration of copper residues was examined in two algal species, *Chlorella regularis* and *Chroococcus paris*. Concentration factors were 2,000 for *C. regularis* for a 20-h exposure, and up to 4,000 for *C. paris* for a

10-min exposure on a wet weight basis (EPA, 1985bb). According to Callahan et al. (1979), the bioconcentration factor for the alga Scenedesmus quadricarda is 12.

Invertebrates

At 30 ppm as $CaCO_3$ hardness, copper sulfate was lethal at concentrations of 150 ppb to worms (Lumbriculus variegatus) (Bailey and Liu, 1980), and an acute value of 242.7 ppb was estimated for a hardness level of 50 ppm (EPA, 1985bb). Estimated acute values for other invertebrates at 50 ppm $CaCO_3$ ranged from 9.26 ppb for a cladoceran, Daphnia pulicaria, to 10,240 ppb for the stonefly, Acroneuria lycorias (EPA, 1985bb).

Aquatic invertebrates and fish are equally sensitive to the chronic toxicity of copper (EPA, 1986d). The chronic toxicity of copper to various invertebrates ranges from 6.066 to 29.33 ppb at hardness ranging from 26 to 211 ppm $CaCO_3$ (EPA, 1985bb). The solutions tested were copper chloride and copper sulfate.

Whole body concentration factors for copper range from 203 for the stonefly, P. californica, to 471 for the cladoceran, D. magna (EPA, 1985bb). Exposure duration was 7 days for D. magna and 14 days for P. californica. Molluscs accumulate copper from water by factors of 30,000 (Callahan et al., 1979). Copper concentration increases with trophic level in food chains (Patrick and Loutit, 1970).

Fish

The 96-h LC_{50} values at 50 ppm as $CaCO_3$ for various fish species range from 16.74 ppb for northern squawfish (Ptychocheilus oregonensis) under flow-through, measured conditions, to 5,860 ppb for white perch (Morone americana) under static, measured conditions (EPA, 1985bb). The 96-h LC_{50} values reported by Johnson and Finley (1980) for copper sulfate range from 135 to 3,510 ppb for rainbow trout and green sunfish, respectively. The 96-h LC_{50} values for copper ammonium sulfate with sulfur range from 121 ppb for rainbow trout to 13,700 ppb for bluegill, and for copper ammonium sulfate without sulfur, the 96-h LC_{50} values range from 20.4 ppb for rainbow trout to 3,280 ppb for bluegill (Johnson and Finley, 1980).

The chronic toxicity of copper is greater than the acute toxicity for aquatic organisms. At hardness levels of 45, chronic toxicity values range from 12.86 ppb for brook trout to 60.36 ppb for northern pike (EPA, 1985bb). Brown bullhead (Ictalurus nebulosus) attain equilibrium with water after 30 days of exposure (Brungs et al., 1973). Copper concentrations correlated with exposure concentrations more closely in liver and gill tissues than other tissues examined (Brungs et al., 1973).

5.1.9.2 Terrestrial Ecosystems

Plants

Copper is accumulated by fruiting shrubs as a result of contamination due to smelter activity (Shaw, 1981). Levels were consistently higher in stems than in leaves or fruits.

Invertebrates

High soil concentrations of copper can be toxic to terrestrial invertebrates. Earthworms (Lumbricus rubellus) exposed to 1,000 ppm copper in soil had 50 percent mortality after 6 weeks, and 100 percent mortality after 12 weeks (Ma, 1982). At soil concentrations of 3,000 ppm, mortality was 100 percent at 6 weeks.

Copper is accumulated by earthworms to a lesser extent than cadmium, but to a greater or equal extent as lead (Ma, 1982). Invertebrates that fed on vegetation had lower copper concentrations than invertebrates that fed on prey or detritus (Beyer et al., 1985). Grasshoppers (Chorthippus brunneus) in the vicinity of a copper refinery accumulated a maximum concentration of 1,600 ppm copper (Hunter et al., 1987); 85 percent of total body copper is associated with the integument.

Birds

Songbirds exposed to multiple metals in a field situation accumulated copper at concentrations as high as 10 ppm on a dry weight basis (Beyer et al., 1985). This concentration could not be correlated with health effects.

Mammals

Dietary concentrations of copper of 30 to 50 ppm are toxic to unspecified ruminants, but not non-ruminants (Buck, 1978a). Sheep are sensitive to the effects of copper, and can suffer toxic effects when grazed on pasture where soil concentrations of copper are high (EPA, 1984d).

Levels of copper in diet of 150 and 250 ppm (1.8 and 3.2 mg Cu^{2+}/kg bw/day) resulted in accelerated weight gain in pigs (Kline et al., 1971). At levels of 500 ppm (5.5 mg Cu^{2+}/kg bw/day), reduced growth, reduced hemoglobin levels, and increased liver copper concentrations were observed. Another study with pigs indicated that 250 ppm copper sulfate in diet (2.6 mg Cu^{2+}/kg bw/day) for 79 days resulted in elevated serum AST and jaundice (Suttle and Mills, 1966). Rats fed 5,000 ppm (80 mg Cu^{2+}/kg bw/day) copper acetate in diet for 16 months accumulated copper in liver and kidney; toxic effects were not reported (Howell, 1959).

Mice (P. leucopus) accumulated less copper (6.7 ppm) than shrews (B. brevicauda) collected from the same area (11 ppm) near a zinc smelter (Beyer et al., 1985). Herbivorous mammals, or those feeding on herbivorous insects, have less exposure to metals in the food chain than carnivores (Beyer et al., 1985).

Copper can interact with other trace metals in diet. For example, an antagonistic effect with molybdenum, zinc, and iron is observed such that toxic effects due to copper are mitigated by addition of these metals to diet (EPA, 1984d). Absorption occurs from the upper gastrointestinal tract, and may be influenced by dietary protein and competition with other metals (Evans, 1973). Rats dosed intraperitoneally with copper (2 ppm) had significant differences in dopamine and norepinephrine levels in brain (Malhotra et al., 1982), while rats coexposed to 2 ppm copper (intraperitoneally) and 100 ppm lead (water ingestion) exhibited greater neurotoxic effects than rats exposed to either metal alone.

5.1.9.3 Quantification of Toxic Effects

The EPA criteria for the protection of aquatic organisms and their uses are used to represent acceptable water concentrations for aquatic life.

Water criteria based on surface water ingestion were derived using available health effects data. The subchronic LOAEL for pigs (2.6 mg/kg bw/day) was used to derive the acceptable water concentration. Using an estimated water intake for pigs of 15.14 l/day (McBride, 1987) and an average body weight of 60 kg (Sax, 1984), the acceptable water concentration was calculated as follows:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{2.6 \text{ mg/kg bw/day}}{0.25 \text{ l/kg bw/day}} = 10.4 \text{ mg/l}$$

By using uncertainty factors of 50 to convert the subchronic LOAEL to a chronic NOEL, and 5 for interspecific variation, the acceptable water concentration becomes 0.042 mg/l (42 ppb).

Because copper appears to bioconcentrate significantly, a Final Residue Value was calculated according to EPA Methodology (Stephan et al., 1985). The geometric mean BCF from data presented by EPA (1985bb) is 738.7 (N=8). The subchronic LOAEL for pigs was used as the MPTC. The Final Residue Value is as follows:

$$\frac{\text{MPTC}}{\text{BCF}} = \frac{250 \text{ ppm}}{738.7} = 0.34 \text{ ppm}$$

Since dietary effects data were unavailable for birds, it is not known whether the Final Residue Value is protective of avian species.

A summary of the acceptable water concentrations (ppb) for copper is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
6.5 (50 ppm $CaCO_3$)	42	340	NA

The lowest of the estimated criteria, 6.5 ppb, is a hardness dependent criterion. Therefore, the acceptable water concentration that will be protective of all wildlife populations at RMA is as low as 6.5 ppb at a hardness of 50 ppm $CaCO_3$, but can increase as hardness increases, not to exceed 42 ppb.

Although accumulation studies have been performed, data were insufficient to calculate accumulation factors from soil to plants or invertebrates. However, copper is toxic to invertebrates at concentrations of 1,000 ppm. Using an uncertainty factor of 10 to bring the LOAEL into the range of a NOEL, the acceptable soil criteria for copper is 100 ppm.

5.1.10 DICHLORODIPHENYLTRICHLOROETHANE (DDT)/ 1,1-DICHLORO-2,2-BIS (4-CHLOROPHENYL)-ETHYLENE (DDE)

The freshwater criteria for protection of aquatic ecosystems for DDT is 0.0010 ppb as a 24-h average, and concentrations should not exceed 1.1 ppb at any time (EPA,1986c). The criteria are based on protection of wildlife populations using reduced productivity of brown pelicans as the toxicological endpoint; criteria are 0.0010 ppb in water based on a maximum permissible tissue concentration of 0.15 ppm in pelican (Anderson et al., 1975). The aqueous solubility of DDT is 0.006 mg/l.

DDT has a high assimilation efficiency for exposure by diet or inhalation, is persistent in the environment, and is concentrated in aquatic food chains (EPA, 1980bb). Absorption through skin is minimal (EPA, 1980bb). The half-life in soil is 3 to 10 years (EPA, 1980bb).

Metabolites of DDT are also toxic to freshwater aquatic organisms; acute toxicity for species tested occurs at concentrations as low as 1,050 ppb for DDE (EPA, 1980bb). Toxicity of DDE may be greater for more sensitive species. Data are unavailable for chronic exposures for this metabolite.

5.1.10.1 Aquatic Ecosystems

Plants

DDT has effects on growth, morphology, and photosynthesis in four species of algae (Sodergren, 1968); observed water concentrations resulting in toxic effects ranged from 0.3 to 800 ppb. In another study, no adverse effects were observed at concentrations of 100,000 ppb for 7 days for the alga, *Chlorella pyrenoidosa* (Christie, 1969). Aquatic macrophytes concentrate DDT residues by factors of 495 for *Scirpus validus* to 21,580 for *Cladophora* sp. (Eberhardt *et al.*, 1971).

Invertebrates

In general, invertebrates are more sensitive to DDT than fish (EPA, 1980bb). The 96-h LC_{50} values for several invertebrate species at 15 to 21°C range from 0.18 to 7.4 ppb, while the 48-h EC_{50} values for *Daphnia magna* and *Cypridopsis* are 4.7 and 15 ppb, respectively (Johnson and Finley, 1980). Aquatic invertebrates concentrate DDT residues by factors of 1,947 for crayfish to 12,500 for a composite of 5 clam species (EPA, 1980bb).

Fish

For various fish species, the 96-h LC_{50} values at 18°C range from 1.5 ppb for largemouth bass to 21.5 ppb for channel catfish (Johnson and Finley, 1980). The chronic toxicity of DDT to fathead minnows (0.74 ppb) is 65 times higher than acute values for the same study (48 ppb) (Jarvinen *et al.*, 1977). Toxicity increases slightly with temperature increases (Johnson and Finley, 1980).

Sublethal effects such as enzyme inhibition occur in fathead minnow for exposure duration of 266 days to 0.5 ppb DDT (Desaiah *et al.*, 1975). Other sublethal effects such as behavior changes occur in various species exposed to concentrations of DDT of 0.008 ppb and greater (EPA, 1980bb).

Bioconcentration factors for fish on a whole body basis of DDT range from 17,500 for green sunfish (*Lepomis cyanellus*) for a 15-day exposure, to 363,000 for common shiner (*Notropis cornutus*) (EPA, 1980bb).

Bioaccumulation factors based on field data range from 11,607 for rainbow trout to over 1,000,000 for lake trout and coho salmon (EPA, 1980bb). Dietary contributions are a more important source of DDT residues than water (Johnson and Finley, 1980).

5.1.10.2 Terrestrial Ecosystems

Plants

DDT, DDD, and DDE are found in grain, leafy vegetables, and fruits; DDT is the most commonly occurring residue (EPA, 1980bb). In one study, residues tended to be higher in grasses than forbs (Forsyth et al., 1983).

Invertebrates

In a field investigation where 1 kg/ha was applied, earthworms and slugs accumulated more DDT residues than other invertebrates (Forsyth et al., 1983). Carabid beetles did not accumulate more residues than prey, and may possibly metabolize DDT readily. Isopods were the most sensitive organisms, and were nearly eliminated by DDT application. The ratio of DDT in earthworms as compared to soil was 5 on a dry weight basis. By assuming that earthworms are 95 percent water (Beyer et al., 1987), the concentration factor is 0.25 on a wet weight basis.

Birds

The acute oral LD_{50} for DDT for mallard duck exceeded 2,240 mg/kg bw; ring-necked pheasant LD_{50} was 1,334 mg/kg bw (Hudson et al., 1984). Rock dove was the most resistent avian species tested (LD_{50} values for DDT were greater than 4,000 mg/kg bw) and California quail (Lophortyx californicus) was the most sensitive (LD_{50} values for DDT were 595 mg/kg bw) (Hudson et al., 1984). The 30-day empirical minimum lethal dose, or EMLD (the oral dose resulting in one or two deaths within 30 days) for mallards was 50 mg/kg bw/day (Hudson et al., 1984).

The lower lethal levels of DDE and DDT in brain for osprey are 250 and 86 ppm (wet weight), respectively, while hazardous levels in brain are estimated as 80 percent of the lethal level, or 200 ppm DDE and 69 ppm DDT (Wiemeyer and Cromartie, 1981). Corresponding hazardous carcass levels are

9,200 ppm DDE and 2,800 ppm DDT (Wiemeyer and Cromartie, 1981). For bald eagles, DDE residues correlate with eggshell thinning and reproductive failure more closely than other contaminants examined. Reproductive failure approached 100 percent when egg residues were greater than 15 ppm on a wet weight basis (Wiemeyer et al., 1984). Reproductive potential for bald eagles was nearly normal when DDE residues in eggs were less than or equal to 3 ppm.

DDT at 40 ppm in diet resulted in a 10 percent decrease in eggshell thickness of Japanese quail (Stickel and Rhodes, 1970). Another study indicated no adverse effects on reproduction for Japanese quail at dietary concentrations of 40 ppm DDT or 200 ppm DDE, although two quail on the 40 ppm DDT diet had tremors (Davison et al., 1976). By using dietary intake for chickens of 175 g/kg bw/day (Sax, 1984), the dietary concentrations for Japanese quail are converted to doses of 7 mg/kg bw/day for DDT and 35 mg/kg bw/day for DDE. Japanese quail dosed with 5 or 50 ppm dietary DDE (0.875 or 8.75 mg/kg bw/day (Sax, 1984)) for 12 weeks were more sensitive to subsequent exposure to parathion or paraoxon (Ludke, 1977). For a 90-day exposure, 30 ppm DDT was not lethal to mallard ducks or bobwhite quail; quail exposed to DDT for 60 days at 100 ppm survived, and no signs of intoxication or eggshell thinning were noted (Hudson et al., 1984). Mallard ducks fed 40 ppm DDE (4 mg/kg bw/day (Sax, 1984)) for 96 days laid eggs with shells up to 20 percent thinner than controls (Haegele and Hudson, 1974), and whole body DDE residues were 33.1 ppm (wet weight). DDT in earthworms at concentrations of 32 ppm is hazardous to sensitive bird species (Beyer and Gish, 1980). DDE at levels of 10 ppm may be hazardous to raptors (Beyer and Gish, 1980).

American kestrels concentrate DDE from diet by factors of 12 to 24, while black ducks accumulate DDE from diet by factors of approximately 80 (Szaro, 1978). Concentrations in eggs of kestrels and black ducks are 12 and 25 times higher than in diet (Szaro, 1978). Kestrels do not reach equilibrium with dietary exposure to organochlorines for at least one year, while

domestic species of birds attain equilibrium in shorter time periods (Weimeyer *et al.*, 1986). DDE residue half-life in birds exceeds 200 days, while DDT and DDD residues decline by 50 percent in 28 and 24 days, respectively (Baily *et al.*, 1969).

Mammals

The acute oral LD_{50} values for DDT range from 100 to 400 mg/kg bw for rats (Negherbon, 1959; Hayes, 1963), and from 250 to 400 mg/kg bw for rabbits (Pimentel, 1971). The LD_{50} for mice is 200 mg/kg bw (Pimentel, 1971). Dogs are more sensitive, as indicated by an oral LD_{50} for DDT of 60 to 75 mg/kg bw (Pimentel, 1971). Dietary levels of 5, 10, and 50 ppm DDT (0.25, 0.5, and 2.5 mg/kg bw/day) caused hepatic cell hypertrophy (Laug *et al.*, 1950), while dietary levels of 600 and 800 ppm caused increased mortality and decreased weight gain in rats (Fitzhugh and Nelson, 1947). Reproductive effects in rats were noted in a chronic study at concentrations of DDT as low as 2.5 ppm in diet (Treon and Cleveland, 1955), equivalent to 0.125 mg/kg bw/day (EPA, 1980bb). The subchronic NOEL for rats for hepatic cell hypertrophy was 1 ppm of DDT (0.05 mg/kg bw/day) in diet (Laug *et al.*, 1950).

In a field study, shrews *Blarina sp.* and *Sorex sp.* accumulated whole body DDT residues 6.4 and 3.8 times greater than those in diet, respectively (Forsyth *et al.*, 1983). Observed concentrations were as high as 126.9 ppm in *Blarina* and 9.8 ppm in *Sorex*, correlating with higher residues in *Blarina* diet (earthworms) as opposed to *Sorex* (mixed species of invertebrates). Voles (*Microtus sp.*) feeding primarily on grasses and forbs had lower tissue concentrations (6.8 ppm) than shrews (Forsyth *et al.*, 1983).

Acute toxic effects include CNS symptoms such as hyperexcitability, paralysis, and convulsions (EPA, 1980bb). DDT and its metabolites concentrate in fat, and to a lesser extent in reproductive organs, liver, kidney, and brain (EPA, 1980bb). Brain concentrations correlate more closely with poisoning in mammals than concentrations in other tissues (EPA, 1980bb); brain concentrations of 287 ppm on a lipid basis were correlated with tremors in rats (Dale *et al.*, 1963).

Rats fed purified DDT had DDT, DDD, and DDE in liver at a ratio of 3:5:1 six days following treatment, and in another study 12 to 24 h following treatment, the ratio was 3:3:1 (EPA, 1980bb). Other studies indicate more DDE in tissue than DDT (Fang et al., 1977).

DDT produces increased tumor incidence in rats and mice, and is a strong inducer of the mixed function oxidase system (EPA, 1980bb). DDT can affect reproductive potential in mice dosed with 1 mg/kg bw (McLachlan and Dixon, 1972), rabbits dosed with 50 mg/kg bw, and rats dosed with 200 mg/kg bw (EPA, 1980bb).

5.1.10.3 Quantification of Toxic Effects

The EPA criteria for the protection of aquatic organisms and their uses are used to represent acceptable water concentrations for aquatic life. The EPA chronic criterion for protection of aquatic life is 0.0010 ppb.

Water criteria were also estimated by using surface water ingestion and health effects data. Reproductive effects are observed in rats chronically dosed with DDT concentrations of 0.125 mg/kg bw/day, while the subchronic NOEL for effects on liver is 0.05 mg/kg bw/day. The more sensitive toxicological endpoint is the effect of DDT on liver cells; therefore, the subchronic NOEL for liver cell effects was used to estimate a water criterion in preference to the higher chronic LOAEL for reproductive effects. By using the NOEL for liver effects and the water consumption for rats, an acceptable water concentration is derived as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.05\ \text{mg/kg bw/day}}{0.125\ \text{l/kg bw/day}} = 0.4\ \text{mg/l}$$

Dividing by an uncertainty factor of 10 to bring the subchronic NOEL into the range of a chronic NOEL, and 5 for interspecies variation, an acceptable water concentration of 0.008 mg/L (8 ppb) is estimated.

Due to the tendency of DDT/DDE residues to increase with trophic level, a Final Residue Value was calculated by EPA (1980bb). The Final Residue Value is based on toxicity of DDT to brown pelicans.

A summary of the acceptable water concentrations (ppb) for DDT/DDE is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.0010	8	0.0010	NA

The lowest of the estimated criteria, 0.0010 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA.

Because DDT/DDE residues bioaccumulate, a Final Residue Value was calculated for soil. Soil criteria are based on the toxicity data for small mammals because the dietary NOEL for small mammals (1 ppm) is lower than available dietary NOELs for birds. Soil invertebrates have concentration factors of 0.25 from DDT contaminated soil. Soil criteria are calculated as follows:

$$\frac{MRIC}{BAF} = \frac{1\ ppm}{0.25} = 4\ ppm$$

The acceptable soil concentration of DDT protective of wildlife populations at RMA is 4 ppm.

5.1.11 DICYCLOPENTADIENE (DCPD)

EPA criteria for water were unavailable in the literature researched. Bentley et al. (1976) recommends a water quality criterion of 0.5 ppm based on an application factor of 0.05. DCPD is persistent in both aquatic and terrestrial environments (USAMBRDL, 1985).

5.1.11.1 Aquatic Ecosystems

Plants

DCPD exposure results in a decrease in both cell numbers and chlorophyll a (Bentley et al., 1976). The 96-h EC_{50} for several algal species based on cell number or chlorophyll a reduction ranged from 31 to greater than 1,000 ppm (Bentley et al., 1976).

Invertebrates

The 48-h LC_{50} values for several macroinvertebrates ranged from 10.5 to 120 ppm (Bentley et al., 1976). D. magna was the most sensitive species tested, and the midge, C. tentans, was the least sensitive species tested.

Fish

The 96-h LC_{50} for bluegill, channel catfish, fathead minnow, and rainbow trout were 23.3, 15.7, 31.1, and 15.9 ppm, respectively (Bentley et al., 1976). The NOELs for the above species for a 96-h exposure were 18.0, 14.0, 24.0, and 10.0 ppm, respectively. The maximum BCF was 53 (Bentley et al., 1976). USAMBRDL (1985) estimates BCF values as high as 143.

5.1.11.2 Terrestrial Ecosystems

Plants

DCPD is not accumulated to a great extent by plants grown in 1,000 ppm solutions, as evidenced by concentration factors of less than 0.1, although growth reduction was observed at this concentration (USAMBRDL, 1985).

Birds

The acute oral LD_{50} of DCPD for birds is 1,010 mg/kg bw (NIOSH, 1984); an oral LD_{50} for mallard ducks exceeds 40,000 mg/kg bw (Aulerich et al., 1979).

Mammals

DCPD is toxic to rats by oral routes, but other organisms do not appear as sensitive. The acute oral LD_{50} values for rats and mice are 353 mg/kg bw and 1,041 mg/kg bw, respectively (NIOSH, 1984). The acute oral LD_{50} for cattle is 1,200 mg/kg bw (NIOSH, 1984). No teratological effects were observed in rats following subchronic dietary exposures of 80 or 750 ppm (estimated to be 6 or 56.25 mg/kg bw/day (Sax, 1984)) for three generations (Hart, 1980).

In a 3-month study with dogs fed dietary concentrations of 100, 300 and 1,000 ppm (estimated doses of 2.5, 7.5, and 25 mg/kg bw/day (Sax, 1984)), no toxic effects on clinical chemistry were observed (Hart, 1980). However, intestinal distress occurred in all treated dogs, especially at the 1,000

ppm concentrations. Due to lack of data reported, the intestinal effects on dogs cannot be further quantified. No effects were observed following subchronic dietary exposure of mice to 273 ppm (estimated dose of 32.76 mg/kg bw/day (Sax, 1984)) (USAMBRDL, 1985).

The LC_{Lo} for rats for inhalation is 500 ppm/4-h (NIOSH, 1984). No mortality was observed for rats for 15 daily exposures, 6-h/day, at concentrations of 100 ppm, or for 10 daily exposures to 72 and 146 ppm (ACGIH, 1986). Mortality was observed for 10 daily exposures by inhalation of 332 ppm for rats, and at 72 and 146 ppm for mice (ACGIH, 1986). Toxic effects include eye irritation, loss of coordination, and in fatal cases, convulsions preceded death (ACGIH, 1986). Dogs exposed by inhalation to 9, 23, and 32 ppm had no dose-related changes in internal organs, and minimal biochemical changes (ACGIH, 1986).

For rabbits, DCPD produces irritation on contact with skin at concentrations of 9.3 to 10 mg/24-h (NIOSH, 1984). The LD_{50} for dermal exposure of rabbits is 5,080 mg/kg bw (NIOSH, 1984).

5.1.11.3 Quantification of Toxic Effects

EPA criteria are unavailable for DCPD. The lowest acute value was the LC_{50} for the aquatic organism D. magna (10.5 ppm). Chronic data are unavailable; therefore, an acute-chronic ratio cannot be calculated. An uncertainty factor of 10^2 is applied to yield an acceptable water concentration of 0.105 mg/l (100 ppb).

Water criteria were also estimated for surface water consumption. Subchronic dietary NOELs for dogs are reported as high as 1,000 ppm (25 mg/kg bw/day); however, intestinal distress was evident at all concentrations tested. This indicates that the reported NOEL values may not have considered appropriate toxicological endpoints; therefore, the LOAEL for dogs of 100 ppm (2.5 mg/kg bw/day) was selected to represent toxicity. By using the subchronic LOAEL for dogs and a water consumption rate for dogs of 0.05 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{2.5 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 50 \text{ mg/l}$$

Uncertainty factors of 50 to bring the subchronic LOAEL into the range of a chronic NOEL, and 5 for interspecific variation, yield an acceptable water concentration of 0.2 mg/l (200 ppb).

DCPD does not bioaccumulate to a significant extent as indicated by measured BCF values of 53; therefore, Final Residue Value calculations were not performed. A summary of the acceptable water concentrations (ppb) for DCPD is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	200	NA	100

The lower of the estimated criteria, 100 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria were based on the toxicity to plants of 1,000 ppm. An uncertainty factor of 10 was applied to bring the LOAEL into the range of an NOEL to yield a soil criterion for DCPD of 100 ppm.

5.1.12 DIISOPROPYLMETHYLPHOSPHONATE (DIMP)

EPA water quality criteria for the protection of aquatic organisms are unavailable. A water quality criterion of 12.5 ppm is recommended based on the toxicity of DIMP to bluegill (Bentley et al., 1976). The aqueous solubility of DIMP is 1,500 mg/l, and it decays by hydrolysis with a half-life of 530 years at 10°C.

5.1.12.1 Aquatic Ecosystems

A study by Bentley et al. (1976) indicated acute toxicity to aquatic organisms ranged from 257 to 6,322 mg/l DIMP. Bluegill were the most sensitive organisms, as indicated by a 96-h LC_{50} of 257 mg/l at 25°C.

5.1.12.2 Terrestrial Ecosystems

Plants

Plants exposed to 10 mg/l DIMP in a nutrient solution exhibited a slight amount of leaf burn and leaf chlorosis (O'Donovan and Woodward, 1977).

Plants irrigated for 64 days with a total of 15 liters of water containing 20 mg/l DIMP (300 mg total DIMP applied to soil) exhibited no effects, while foliar damage was observed at water concentrations of 50 mg/l (O'Donovan and Woodward, 1977).

Birds

The effects of DIMP on mallards and bobwhite quail have been investigated (Aulerich et al., 1979). The acute oral LD_{50} values for mallard and quail are 1,490 and 1,000 mg/kg bw, respectively. In 8-day subacute feeding studies, mallard ducks receiving 3,200 ppm or more exhibited decreased feed consumption; mortality was not observed even at the highest dose level of 16,000 ppm (2,062.4 mg/kg bw/day). In 24-week feeding studies ducks receiving 10,000 ppm in diet exhibited decreased egg production. In subacute studies with quail, feed consumption decreased at dietary levels of 16,000 to 36,000 ppm (2,685 to 4,982.9 mg/kg bw/day). In chronic studies (29-week), high mortality occurred at dietary concentrations of 3,800 and 12,000 ppm; egg production decreased at levels of 1,200 ppm as compared to controls.

Tissue residue studies with duck and quail indicated little tissue accumulation of DIMP residues. By day five posttreatment, all tissues except skin were free of residues. Birds dosed at 100 mg/kg bw had tissue residues as high as 756 ppm two hours following dose administration. Residues decreased rapidly with a half-life of 12.7 hours, and at 65 hours tissues were free of DIMP residue.

Mammals

The acute oral LD_{50} for mink is 503 mg/kg bw, about half of that observed for birds (Aulerich et al., 1979). For dietary exposures of 21 days, the LC_{50} was estimated to be greater than 10,000 ppm. For a 21-day ingestion by mink, animals receiving 10,000 ppm (1,851.9 mg/kg bw/day) in diet had decreased hematocrits, lower numbers of lymphocytes, and increased aggressive behavior. Male mink fed diets containing 1,000 ppm (201.2 mg/kg bw/day) had decreased lung weights; at 10,000 ppm, decreased heart, kidney, lung, and liver weights were observed. Chronic ingestion of DIMP for 12 months at concentrations ranging from 50 to 450 ppm (10.98 to 95.06 mg/kg bw/day) caused increased mortality (17 to 29 percent) in female mink.

Inconsistent dose-response effects were observed at the lower dietary concentrations. In the 21-day study, levels of 1 ppm (0.20 mg/kg bw/day) in diet of female mink for 21 days resulted in decreased kidney weights, although higher concentrations did not produce a similar effect. At dose levels of 10 ppm, decreased numbers of lymphocytes were observed in both sexes, while no effect on lymphocytes was noted at 100 ppm (17.159 mg/kg bw/day). In the chronic study, no effects were observed on organ weights or blood chemistry at dose levels ranging from 50 to 450 ppm. Because of the inconsistencies in the results at the 1 and 10 ppm concentrations, 100 ppm is considered the acceptable NOEL for the 21-day study, and 50 ppm the LOAEL for the chronic study.

5.1.12.3 Quantification of Toxic Effects

EPA criteria and LOAEL data are unavailable; therefore, criteria were established using the lowest acute value for bluegill of 257 mg/l and an uncertainty factor of 10^2. The acceptable water concentration protective of aquatic life is 2.57 mg/l (2,570 ppb).

Water criteria are also estimated by using surface water consumption and health effects data. The chronic LOAEL was 10.98 mg/kg bw/day for female mink. Assuming that water consumption for mink is similar to a domestic cat (0.05 ml/kg bw/day; Sax, 1984), and that all ingested DIMP comes from water consumption, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{10.98 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 219.6 \text{ mg/l}$$

Using an uncertainty factor of 5 to bring the chronic LOAEL into the range of a NOEL, and 5 for interspecific variation, an acceptable water concentration of 8.78 mg/l (8,800 ppb) is estimated.

There is no indication that DIMP bioaccumulates to a significant extent; therefore, Final Residue Value calculations were not performed. A summary of the acceptable water concentrations (ppb) for DIMP is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	8,800	NA	2,570

The lower of the estimated criteria, 2,570 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria were calculated using the following formula, and the NOEL for DIMP in irrigation water for plants (O'Donovan and Woodward, 1977, RIC=81335R08).

$$C_s = \frac{(Q)(NVP)}{(W_s)(UF)}$$

where: C_s = concentration in soil (mg/kg),
Q = total amount of DIMP applied to soil (300 mg),
UF = uncertainty factor,
NVP = nonvolatilized percentage (0.78), and
W_s = weight of soil (kg)

Plants were grown in 3-gal containers. Soil weight was estimated by assuming that 3 gal of soil was used, and that the soil in each container weighed 1.3 g/cm^3, for a total soil weight of 14.8 kg. An uncertainty factor was not applied because the Q value represents a NOEL. The NVP was calculated from data presented by O'Donovan and Woodward (1977, RIC=81353R08) where water was applied to DIMP contaminated soils at regular intervals, and soil concentrations tested. The acceptable soil concentration was determined to be 15.8 mg/kg DIMP.

5.1.13 DIMETHYLMETHYL PHOSPHONATE (DMMP)

EPA criteria for the protection of aquatic organisms and their uses for DMMP are unavailable. The half-life of DMMP in water is 13 years at 15°C (Bel'skii et al., 1969). DMMP is soluble in water and has a low vapor pressure. Therefore, leaching into groundwater with no significant volatilization is to be expected (Kuzhikalail, 1974).

5.1.13.1 Aquatic Ecosystems

Plants

No information was available in the literature reviewed.

Invertebrates

No information was available in the literature reviewed.

Fish

The 96-h LC_{50}s for bluegill and fathead minnow were 51 and 63 ppm, respectively (Department of the Army, 1976).

5.1.13.2 Terrestrial Ecosystems

Plants

No information was available in the literature reviewed.

Invertebrates

In a study by Penman and Osborne (1976), bean leaf discs were dipped into DMMP in an ethanol and water mixture then fed to female two-spotted spider mites (*Tetranychus urticae*). At the lowest dose of 1.5 percent DMMP, there was a significant reduction in the number of eggs hatched.

Birds

Intraperitoneal injections of 50 mg/kg bw/day DMMP for 10 days caused no delayed neurotoxicological effects in hens (Hollingshaus *et al.*, 1981).

Mammals

DMMP is not acutely toxic to mammals as indicated by oral LD_{50} values for rats and mice that exceed 6,810 mg/kg bw (Dynamac, 1983). When administered subchronically by oral gavage, DMMP caused 100 percent mortality in rats at dose levels of 4,000 mg/kg bw/day for a 5-day week (Litton Bionetics, 1981). The lethal LOAEL for male rats was 2,000 mg/kg bw, and the LOAEL for female rats was 250 mg/kg bw. Subchronic LOAELs for both male and female mice were 4,000 mg/kg bw/day for a 5-day week (Litton Bionetics, 1979).

Reproductive effects were observed in subchronic tests with male rats at dose levels of 250, 500, 1,000, and 2,000 mg/kg bw/day (Dunnick *et al.*,

1983). Effects included decreased sperm count and sperm motility, as well as increased embryo resorption in females bred to treated males.

DMMP has central nervous system effects in rats and mice: weak cholinesterase inhibition has been observed (Dynamac, 1983).

5.1.13.3 Quantification of Toxic Effects

EPA criteria are unavailable: therefore, the lowest acute value (51 ppm) is divided by an uncertainty factor of 10^2 to yield an acceptable water concentration of 0.51 ppm (510 ppb).

Water criteria are also estimated for toxicity due to consumption of contaminated surface water. The subchronic LOAEL for reproductive effects and lethality in rats is 250 mg/kg bw/day. From a water consumption rate for rats of 0.125 l/kg bw/day and the LOAEL for rats an acceptable water concentration is calculated as follows:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{250 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 2.000 \text{ mg/l}$$

Applying an uncertainty factor of 50 to convert the subchronic LOAEL to a chronic NOEL, and an uncertainty factor of 5 for interspecific variation, an acceptable water concentration of 8 mg/l (8000 ppb) is derived.

There is no indication that DMMP significantly bioaccumulates: therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for DMMP is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	8000	NA	510

The lower of the estimated criterion, 510 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of available data this estimate is highly uncertain. Soil criteria could not be estimated due to insufficient data.

5.1.14 DITHIANE

EPA criteria for the protection of aquatic organisms for dithiane are unavailable in the literature reviewed. Dithiane is an impurity in mustard (Berkowitz and Rosenblatt, 1982, RIC=82042R07) that increases with time, indicating that dithiane is formed from mustard gas rather than being a by-product of mustard preparation (Bell *et al.*, 1927). At ambient temperatures it exists as a white, crystalline solid (Marsh and McCullough, 1951; Kirner, 1946), is soluble in water, and has a low octanol/water partition coefficient of 44 (Berkowitz *et al.*, 1978a, RIC=82166R03). Dithiane is readily oxidized to sulfoxides and sulfones when exposed to the atmosphere (Schroyer and Jackman, 1947). The presence of water facilitates photo-oxidation to the sulfoxide (Foote and Peters, 1971). Dithiane contains nutrient elements carbon and sulfur, and therefore may be subject to biodegradation (Berkowitz *et al.*, 1978a, RIC=81266R03). Little biomagnification is expected based on the estimated octanol/water partition coefficient (Berkowitz *et al.*, 1978a, RIC=81266R03).

5.1.14.1 Aquatic Ecosystems

No information regarding the toxicity of dithiane was available in the literature reviewed.

5.1.14.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of dithiane was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of dithiane was available in the literature reviewed.

Birds

No information regarding the toxicity of dithiane was available in the literature reviewed.

Mammals

In a study by Mayhew and Muni (1986), male and female rats were given dithiane by oral gavage. LD_{50} values for male and females were 3,680 and 2,767 mg/kg bw, respectively. Toxic effects included crusty eyes and muzzle, hyperactivity, muscle tremors, red stained fur around the eyes, emaciation, lethargy, few or no stools, ataxia, squinting, prostration, lacrimation, irregular breathing, damp fur, and stained fur in the perianal region. Necropsy results in rats that died showed discoloration of the lung, small intestine, stomach, and liver. Gastrointestinal contents appeared as a dark, thick, red and/or white fluid. In a 90-day study by Schieferstein (1986), rats were given 0, 105, 210, and 420 mg/kg bw/day dithiane by oral gavage. At the high dose, female liver and male kidney weights were significantly heavier and renal lesions were observed. At 105 mg/kg bw/day, 7 percent of the males and 33 percent of the females exhibited crystalline nasal deposits.

5.1.14.3 Quantification of Toxic Effects

EPA criteria are unavailable, and aquatic life data were unavailable in the literature reviewed; therefore, an acceptable water concentration could not be calculated for the protection of aquatic life.

By using the subchronic LOAEL for rats and the water consumption rate for rats of 0.125 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{105 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 840 \text{ mg/l}$$

Uncertainty factors of 50 to bring the subchronic LOAEL into the range of a chronic NOEL, and 5 for interspecific variation, yield an acceptable water concentration of 3.36 mg/l (3,360 ppb).

There is no indication that dithiane bioaccumulates to a significant extent; therefore, Final Residue Value calculations were not performed. A summary of the acceptable water concentrations (ppb) for dithiane is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	3.360	NA	NA

The only estimated criterion, 3.360 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain, and may not be protective for aquatic organisms.

Soil criteria for dithiane could not be established at this time due to insufficient data.

5.1.15 ETHYLBENZENE

Acute toxicity to freshwater aquatic life occurs at an ethylbenzene concentration in water as low as 32 ppm (EPA, 1980d). No chronic freshwater data are available (EPA, 1980d). The water solubility is 1.52×10^2 (EPA, 1985k).

The half-life of ethylbenzene in air is 35 hours (NAS, 1980). In water the half-life is estimated to range from 1.5 to 7.5 days (EPA, 1984f); half-life has not been determined for soil. Evaporation is expected to be the predominant loss mechanism from the soil surface (EPA, 1980d).

The estimated bioconcentration factor for aquatic organisms containing 7.6 percent lipids is 95 (EPA, 1980d).

5.1.15.1 Aquatic Ecosystems

Plants

For the alga, *Selenastrum capricornatum*, the 96-hr EC_{50} causing a reduction in cell numbers and chlorophyll a production is 438,000 ppb ethylbenzene (EPA, 1980d).

Invertebrates

The LC_{50} for *D. magna* exposed to ethylbenzene is 75,000 ppb (EPA, 1978). A reported bioconcentration factor for the clam, *Tapes semidecussata*, is 4.7 (Nunes and Benville, 1979).

Fish

LC_{50} values for goldfish, fathead minnow, and guppy are 94,440, 42,330 to 48,510, and 97,100 ppb, respectively (Pickering and Henderson, 1966). Two different toxicity studies with bluegills indicate LC_{50} values of 32,000 ppb (Pickering and Henderson, 1966) and 155,000 ppb (EPA, 1978), or geometric mean value of 70,400 ppb.

5.1.15.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of ethylbenzene was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of ethylbenzene was available in the literature reviewed.

Birds

No information regarding the toxicity of ethylbenzene was available in the literature reviewed.

Mammals

Ingestion of ethylbenzene has been reported to cause effects similar to those produced by inhalation (Wolf et al., 1956). Acute toxicity symptoms include coordination disorders, narcosis, convulsions, pulmonary irritation and conjunctivitis (Ivanov, 1962). Target organs for acute exposure are primarily the central nervous system and lungs (Smyth et al., 1962; Faustov, 1958, 1960). In research by Wolf et al. (1956), the acute oral LD_{50} in rats was determined to be 3,500 mg/kg bw. Another study found the LD_{50} for male rats orally exposed to ethylbenzene to be 4,730 mg/kg bw, for female rats exposed through inhalation the LC_{50} was 4,000 ppm in 4 hours, and for male rabbits exposed dermally the LD_{50} was 17,800 mg/kg bw (Smyth et al., 1962).

In research by Wolf et al. (1956), target organs for chronic exposures to ethylbenzene were liver and kidney. Rats exposed orally to 408 mg/kg bw/day for 5 days/week for 6 months exhibited increased liver and kidney weights, and cloudy swelling in hepatocytes and renal tubular epithelium. When rats

were exposed to air concentrations of 400 ppm for 7 hr/day for 5 days/week, a slight increase in liver and kidney weight occurred. Rabbits and rhesus monkeys showed slight testicular degeneration at ambient air concentrations of 600 ppm for a 7 hr day for 186 days. The NOEL for guinea pig, rabbit and rhesus monkey for exposure via inhalation was 200 ppm.

In inhalation studies with rats and rabbits by Hardin et al. (1981), 100 ppm ethylbenzene caused a significant reduction in the number of live kits per litter in rabbits and a high incidence of extra ribs in rats. No maternal toxicity was observed at this level. At 1,000 ppm via inhalation, maternal health effects observed in rats were increased liver, kidney, and spleen weight.

5.1.15.3 Quantification of Toxic Effects

EPA criteria were unavailable; therefore, the lowest acute value reported by EPA (32,000 ppb reported as the LC_{50} for bluegill) was used to calculate a water criterion. An acute-chronic ratio could not be calculated due to lack of chronic toxicity data. An uncertainty factor of 10^2 was applied to yield an acceptable water concentration of 320 ppb.

An acceptable water concentration based on surface water ingestion was calculated by using the chronic LOAEL for rats and the water consumption rate for rats of 0.125 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{408 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 3.264 \text{ mg/l}$$

Uncertainty factors of 5 to bring the chronic LOAEL into the range of an NOEL, and 5 for interspecific variation yield an acceptable water concentration of 130 mg/l (130,000 ppb).

There is no indication that ethylbenzene bioaccumulates to a significant extent; therefore, Final Residue Value calculations were not performed. A summary of the acceptable water concentrations (ppb) for ethylbenzene is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
320	130,000	NA	NA

The lower of the estimated criterion, 320 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria could not be estimated due to lack of data.

5.1.16 HEPTACHLOR/HEPTACHLOR EPOXIDE

Criteria for the protection of aquatic organisms are 0.0038 ppb as a 24-hr average, not to exceed 0.053 ppb at any time (EPA, 1980dd). The Final Acute Value is 0.52 ppb: insufficient data exist to calculate a Final Chronic Value (EPA, 1980dd). WHO has established an acceptable daily intake level from diet of 0.5 mg/kg bw/day (NAS, 1977). Separate criteria are unavailable for heptachlor epoxide.

The half-lives of heptachlor and heptachlor epoxide in soil are 6 months (EPA, 1987h) and approximately 3 years (Beyer and Gish, 1980), respectively. The aqueous solubility of heptachlor is 56 ppb at 25°C. Volatilization is a major route of loss of heptachlor from treated surfaces, plants and soils (Nisbet, 1977).

Heptachlor is metabolized to heptachlor epoxide: epoxidation represents an activation reaction, and heptachlor epoxide is the stored product (Casarett and Doull, 1980). The epoxide is equally or more toxic than the parent compound, and can be further metabolized to a more hydrophilic compound and excreted (Casarett and Doull, 1980). Studies relating the toxicity of heptachlor to heptachlor epoxide indicate heptachlor is 2 to 4 times more toxic than heptachlor epoxide when given intravenously to mice (Radomski and Davidow, 1953) and 10 times more toxic in dairy calves when given orally (Buck et al., 1959).

5.1.16.1 Aquatic Ecosystems

Plants

The mean EC_{50} for heptachlor in the alga Selenastrum capricornatum is 33.1 ppb (Call and Brooke, 1980). Most algal species tested exhibited growth reduction when exposed to 10 ppb heptachlor (O'Kelley and Deason, 1976). In a field study, plankton and algae bioconcentrated heptachlor and heptachlor epoxide residues by a factor of 183 (Hannon et al., 1970).

Invertebrates

The 26-hr LC_{50} values for heptachlor and heptachlor epoxide in Daphnia magna are 52 and 120 ppb, respectively (Frear and Boyd, 1967). Other LC_{50} values in 12 invertebrate species range from 0.9 ppb in Pteronarcella badia to 80 ppb in Simocephalus serrulatus, with a geometric mean value of 13.3 ppb (EPA, 1980dd).

Fish

LC_{50} values for fish range from 10.0 ppb in rainbow trout to 320 ppb in goldfish (EPA, 1980dd). In a 40-wk study with fathead minnows, 1.84 ppb heptachlor was lethal to 100 percent of the population after 60 days. At a concentration of 0.86 ppb and lower, no adverse effects were observed (Macek et al., 1976). Bluegills fed 0 to 25 mg/kg bw/day heptachlor had dose-related growth decreases at concentrations greater than 5 mg/kg bw/day, with increased mortality above 10 mg/kg bw/day (Andrews et al., 1966).

In a 32-day study, the whole body bioconcentration factor for the fathead minnow was determined to be 9,500 (Veith, 1979). The bioconcentration factor normalized for percent lipids for aquatic life (fresh and saltwater) is 5,222 (EPA, 1980dd). Biomagnification was observed in a field study of an aquatic system containing heptachlor and heptachlor epoxide (Hannon et al., 1970). Contaminant concentrations in water and sediment were 0.006 and 0.8 ppb, respectively. Residue concentrations in crayfish, plankton and algae, fish, and aquatic insects were 1.0, 1.1, 8.0, and 312 ppb, respectively (Hannon et al., 1970). Bluegills concentrated heptachlor by a factor of 300 in relation to water concentration (Cope, 1966). Mosquito fish concentrated residues by a factor of 2,258 in a model ecosystem study (Sanborn et al., 1976).

5.1.16.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of heptachlor or heptachlor epoxide was available in the literature reviewed.

Invertebrates

Earthworms metabolize heptachlor epoxide from and concentrate residues 10 times in relation to soil concentration (Beyer and Gish, 1980). The biological half-life of heptachlor epoxide in earthworms is approximately 3 years (Beyer and Gish, 1980).

Birds

The LC_{50} values for bobwhite quail, Japanese quail, ring-necked pheasant, and mallard duck in 5-day feeding tests ranged from 92 to 480 ppm (Hill et al., 1975). Birds fed 50 ppm heptachlor died within 9 to 24 days (Stickel et al., 1979b).

The lethal residues in brain tissue for several species range from 9 to 27 ppm on a wet weight basis (Stickel et al., 1979b), and the lethal hazard level begins at 8 ppm. The half-life of heptachlor epoxide in birds is 29 days (Stickel et al., 1979b).

Heptachlor epoxide was more toxic to woodcock (Scolopax minor) than DDT (Stickel et al., 1965a and b). Woodcock fed earthworms containing 2.86 ppm heptachlor epoxide on a wet weight basis died within 35 days. A lower concentration of 0.65 ppm in earthworms was not lethal to woodcock when the birds were adequately fed; however, when the birds were food deprived, tissue residues mobilized and became lethal.

Heptachlor epoxide has been detected in osprey and bald eagle eggs at concentrations ranging from 0.02 to 0.41 and 0.02 to 0.36 ppm on a wet weight basis, respectively (Wiemeyer et al., 1978; Wiemeyer et al., 1984). Heptachlor epoxide residues did not correlate significantly with average reproductive productivity for bald eagles (Wiemeyer et al., 1984).

Mammals

Heptachlor and/or heptachlor epoxide are both readily absorbed from the gastrointestinal tract (EPA, 1980dd). The acute oral LD_{50} for heptachlor epoxide in rats is 46.5 to 60 mg/kg bw (NAS, 1977; Terling and Ewinike, 1969; Podowski et al., 1979). Work by Gak et al. (1976), determined the oral LD_{50} for heptachlor for mice, rats, and hamsters to be 70, 105, and 100 mg/kg bw, respectively. Gaines (1960) determined the oral LD_{50} for heptachlor to be 100 mg/kg bw in male and 162 mg/kg bw in female rats. The acute oral LD_{50} for heptachlor epoxide in mice and rats is 39 mg/kg bw and 47 mg/kg bw, respectively (NIOSH, 1984). Effects of acute intoxication due to heptachlor or heptachlor epoxide include tremors, convulsions, paralysis, and hypothermia (Hrdina et al., 1974; Yamaguchi et al., 1980).

In rats fed 1 mg/kg bw/day heptachlor for 14 days, liver damage and altered liver function occurred (Enan et al., 1982). At dose levels of 2 mg/kg bw in pigs, depletion of glycogen, morphological changes in the granular endoplasmic reticulum, and increases in the amount of agranular endoplasmic reticulum occur (Dvorak and Halacka, 1975). In dogs fed heptachlor epoxide for 60 weeks, increased liver weights were seen at the lowest dietary level of 0.5 ppm (approximately 0.0125 mg/kg bw/day (Sax, 1984)) (EPA, 1958).

Various studies using rats have shown that the amount of protein in the diet affects the toxicity of heptachlor (EPA, 1980dd). At a protein level of 18 percent, heptachlor is twice as toxic as in a low protein diet (EPA, 1980dd). Low protein diets impaired or slowed the metabolism of heptachlor to the more toxic heptachlor epoxide (EPA, 1980dd).

Oral doses of 1 mg/kg bw heptachlor given to male rats caused dominant lethal changes, as evidenced by statistically significant increases in the number of resorbed fetuses in intact pregnant rats (Cerey et al., 1973). A diet of 6 mg/kg bw in rats caused a marked decrease in litter size and significantly shortened the life span of suckling rats (Mestitzova, 1967).

5.1.16.3 Quantification of Toxic Effects

The EPA criteria for the protection of aquatic organisms and their uses (0.0038 ppb) were derived from the Final Residue Value based on human

health, and so were considered inappropriate for this analysis. Therefore, the EPA Final Acute Value (0.52 ppb) was used to derive an acceptable water concentration for the protection of aquatic organisms. The Final Acute Value was divided by an uncertainty factor of 10^2, to yield an acceptable water concentration of 0.0052 ppb.

Water criteria are also estimated for surface water consumption by terrestrial organisms. Heptachlor was chronically toxic to dogs at concentrations of 0.0125 mg/kg bw/day. Using the chronic LOAEL and the water consumption for dogs, an acceptable water concentration is derived as follows:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{0.0125 \text{ mg/kg bw/day}}{0.05 \text{ l/kg bw/day}} = 0.25 \text{ mg/l}$$

Using an uncertainty factor of 5 for interspecies variation and 5 to bring the chronic LOAEL into the range of a NOEL, a water concentration of 0.01 mg/l (10 ppb) heptachlor is estimated.

The freshwater Final Residue Value reported by EPA is based on the FDA action level of 0.3 ppm (EPA, 1980dd), and was therefore judged inappropriate for this analysis. The lowest dietary level of heptachlor reported for birds or mammals in the literature surveyed, the LOAEL for dogs (0.5 ppm), was substituted for the FDA value as the appropriate MPTC. The BCF reported by EPA (1980dd) was used to represent bioconcentration. The Final Residue Value is as follows:

$$\frac{\text{MPTC}}{\text{BCF}} = \frac{0.5 \text{ ppm}}{78.9} = 0.0063 \text{ ppm}$$

A summary of the acceptable water concentrations (ppb) for heptachlor/heptachlor epoxide is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.0052	10	6.3	NA

The lowest of the estimated criteria, 0.0052 ppb, is used as the acceptable water concentration of heptachlor/heptachlor epoxide that will be protective of all wildlife populations at RMA.

Soil criteria were estimated using the LOAEL for dogs as the MPTC representative of toxicity to wildlife populations, and a BAF of 10 for earthworms as follows:

$$\frac{MPTC}{BAF} = \frac{0.5\ ppm}{10} = 0.05\ ppm$$

The dietary NOEL for birds of 0.65 ppm in earthworms exceeds the LOAEL for mammals of 0.5 ppm in diet: therefore, using the LOAEL for mammals should protect bird populations as well. Since the MPTC is a LOAEL, an uncertainty factor of 10 was applied to yield an acceptable soil level of 0.005 ppm heptachlor/heptachlor epoxide.

5.1.17 MALATHION

The EPA chronic criterion for the protection of freshwater aquatic life is 0.1 ppb (EPA, 1986c). Acute criteria are unavailable. Malathion is moderately soluble in water with a half-life of 5 months at pH 6 and of 1 to 2 weeks at pH 8 (Weiss and Gakstatter, 1964). Variations in water hardness do not appear to alter the toxicity of malathion to fish and aquatic invertebrates (Johnson and Finley, 1980). Malathion in combination with parathion has a synergistic effect in rainbow trout and bluegill (Johnson and Finley, 1980).

5.1.17.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of malathion was available in the literature reviewed.

Invertebrates

The 96-hr LD_{50}s for 12 invertebrate species range from 0.69 ppb in Isoperla sp. to 3,000 ppb in Asellus sp. (Johnson and Finley, 1980) with a geometric mean value of 12.8 ppb. The 48-hr EC_{50}s for four invertebrate species range from 1.0 ppb in Daphnia magna to 47 ppb in Cypridopsis sp. (Johnson and Finley, 1980), with a geometric mean value of 4.1 ppb. Bluegill and channel catfish were not affected in ponds dosed with four semimonthly treatments (during May to July) of up to 0.02 ppm, but the aquatic invertebrate population was significantly reduced (Johnson and Finley, 1980). Temperature fluctuations in water may influence the toxicity of malathion as evidenced by a study in which Simocephalus sp. exhibited an eleven fold decrease in toxic symptoms when the temperature changed from 10 to 21° C (Johnson and Finley, 1980). According to the EPA (1986c), complete life cycle tests to determine safe concentrations for the most sensitive species have not been conducted, nor have tests determined the effects of low concentrations on invertebrate behavior.

Fish

The 96-hr LC_{50} values in fish range from 23 ppb in chinook salmon (Katz, 1961) to 285 ppb in a combination of centrarchids and salmonids (Macek and McAllister, 1970). Static (Macek and McAllister, 1970) and flow through tests (Eaton, 1970) in bluegill indicate similar values of 110 ppb.

Trout exposed to sublethal levels (level not specified) for one hour had severe tissue damage to gill tissues and minor nonspecific liver lesions (Johnson and Finley, 1980). Other sublethal effects include AChE inhibition, reduced activity compared to controls, and reduced frequency of avoidance response (Johnson and Finley, 1980). An increase in temperature from 7 to 29°C causes a 4 fold increase in toxicity to bluegills (Johnson and Finley, 1980).

5.1.17.2 Terrestrial Ecosystems

Plants

There was 15 percent less vegetation biomass increase in a treated plot as compared to the control plot when malathion was applied at a rate of 8 oz/acre (McEwen and Ellis, 1975). By using a conversion factor of

2,000,000 lbs soil/6 inch acre (Korschgen, 1970), the estimated concentration applied was 0.25 ppm.

Invertebrates

In the above study by McEwen and Ellis (1975), malathion was applied at a rate of 8 oz/acre (an estimated value of 0.25 ppm). Immediately post-treatment, the number of invertebrates on treated plots dropped to 11 percent of controls, and five weeks post-treatment invertebrate numbers on treated plots were 18 percent of controls. The total number of invertebrate species present on treated plots dropped by 50 percent compared to controls.

Birds

LD_{50} values for mallard, ring-necked pheasant, and horned lark (*Eremophila alpestris*) are 1,485, 167, and 403 mg/kg bw, respectively (Hudson *et al.*, 1984). The oral LD_{50} for an adult domestic chicken is 150 to 200 mg/kg bw (Radeleff, 1964). In a field study by McEwen and Brown (1966), sharp-tailed grouse (*Pedioecetes phasianellus*) were administered a single oral dose of malathion, and a lethal dose of 200 to 240 mg/kg bw was determined. The reaction of grouse to malathion was rapid, with death or apparent full recovery occurring within 72 hours. Symptoms of acute toxicity included depression and inactivity, increasing slowness and slowed reactions, blinking and head nodding, and finally death from heart or respiratory failure. Cholinesterase levels in grouse that died were 0.00 and 0.03 (pH change/hr) compared to 0.56, 0.56, and 0.54 in control birds. Three known cases of predation were on birds that were given doses they might have survived under pen conditions, suggesting that birds subjected to sublethal doses may be more vulnerable to predation.

Mammals

The oral LD_{50} in rats is 2,100 mg/kg bw (ACGIH, 1986). In a 2-year study with rats, the NOEL was determined to be 100 ppm in diet (approximately 7.5 mg/kg bw/day (Sax, 1984)) based on inhibition of blood or brain cholinesterase and observable injury (Johnson *et al.*, 1952).

5.1.17.3 Quantification of Toxic Effects

The EPA criteria for the protection of aquatic organisms and their uses represent acceptable water concentrations of malathion for aquatic life.

Water criteria are also estimated based on toxicity due to surface water consumption by terrestrial biota. From the chronic NOEL and the water consumption for rats, an acceptable water concentration is derived as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{7.5 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 60 \text{ mg/l}$$

Applying an uncertainty factor of 5 for interspecies variation, a water concentration of 12 mg/l (12,000 ppb) malathion is estimated.

There is no indication in the available literature that malathion bioaccumulates. A summary of the acceptable water concentrations (ppb) for malathion is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.1	12,000	NA	NA

The lowest of the estimated criteria, 0.1 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA.

Soil criteria were established based on toxicity to plants and soil invertebrates. At an application rate of 0.25 ppm, adverse effects were observed on plants and soil fauna in treated areas. By applying an uncertainty factor of 10 to convert the LOAEL to a NOEL, an acceptable level of malathion in soil of 0.025 ppm is derived.

5.1.18 METHYL PARATHION

EPA water quality criteria for methyl parathion for the protection of aquatic life were unavailable. The solubility of methyl parathion in water is 55 to 60 mg/l. Methyl parathion is more rapidly hydrolyzed in alkaline

solutions than acidic. The half-life in water is 1 to 3 days (AgChem, 1983). Residue tolerance levels for raw agricultural commodities are 0.1 to 0.5 ppm (CFR, 1985). Residue tolerence levels for raw agricultural commodities are 0.1 to 0.5 ppm (CFR, 1985). Methyl parathion is relatively immobile in a 30-cm soil column of sandy loam, silty clay loam and silt loam soils (EPA, 1987f). The half-life in soils appears to be dependent on the form in which it is applied. The half-life of the emulsifiable concentrate is 1 to 3 days, and half-life of the microencapsulated form is 3 to 7 days (AgChem, 1983).

5.1.18.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of methyl parathion was available in the literature reviewed.

Invertebrates

Aquatic invertebrates are sensitive to the effects of methyl parathion. The 48-h LC_{50} for *D. magna* is 0.14 ppb (Johnson and Finley, 1980). The 96-h LC_{50} values for three other species of invertebrates ranged from 3.8 to 33 ppb. Water hardness did not alter toxicity of methyl parathion to invertebrates (Johnson and Finley, 1980).

Fish

Fish are not as sensitive to the effects of methyl parathion as aquatic invertebrates. The 96-h EC_{50} for rainbow trout is 2.0 ppm, and the 96-h LC_{50} is 2.8 ppm (Palawski *et al.*, 1983). The mean 96-h LC_{50} values for 13 species of freshwater fish tested ranged from 1.85 to 9.00 ppm (Johnson and Finley, 1980).

5.1.18.2 Terrestrial Ecosystems

Plants

Methyl parathion reduced the rate of photosynthesis in plants at application rates of 0.5 kg/ha, and reduced growth rate occurred as application frequency increased (Johnson *et al.*, 1983).

Invertebrates

The acute lethal dose of methyl parathion to bees was 0.165 and 0.324 ug/bee for European and Africanized bees, respectively (Danka et al., 1986). The LD_{50} for the wasp (Microplitis croceipes) is 2.28 mg/kg bw, calculated from a mean body weight of 5.8 mg for wasps (Powell et al., 1986). Methyl parathion was more toxic than dieldrin, aldrin, or malathion when applied topically to M. croceipes (Bull et al., 1987).

Birds

Hudson et al. (1984) reported LD_{50} values, symptoms of intoxication, and changes in acetylcholinesterase (AChE) levels for several species of birds. LD_{50} values for mallard ranged from 6.60 to 60.5 mg/kg bw. LD_{50} values for bobwhite quail, ring-necked pheasant, and red-wing blackbird were 7.56, 8.21, and 23.7 mg/kg bw, respectively. The median lethal dose for American kestrels was 3.08 mg/kg bw, indicating that kestrels are more sensitive than other species tested (Rattner and Franson, 1984). AChE inhibition was significantly higher in birds that died than those that survived (Hudson et al., 1984). In birds that survived, AChE inhibition was as high as 68.6 percent for bobwhite. Signs of intoxication included polydipsia, regurgitation, ataxia, falling, convulsions, dyspnea, salivation, withdrawal, using wings for pedestrian locomotion, wing-beat convulsions, nutation, lacrimation, asynergy, immobility, and opisthotonos.

Mammals

Acute oral LD_{50} values range from 8.9 mg/kg bw in rats (Sabol, 1985) to 30 mg/kg bw in mice (Isshiki et al., 1983). Symptoms of acute toxicity include sweating, salivation, diarrhea, bradycardia, bronchoconstriction, muscle fasciculations, and coma (Barnes and Denz, 1953). The LOAEL was 0.25 mg/kg bw/day for rats based on hematology, body weight, organ weights, clinical chemistry, retinal degeneration, and cholinergic signs (Daly et al., 1984). The LOAEL was 1.0 mg/kg bw/day for dogs based on cholinesterase depression (Tegeris and Underwood, 1978). The LOAEL for fetotoxic effects in rats was 1.0 mg/kg bw/day based on protein synthesis in maternal and fetal tissues, altered postnatal development of cholinergic neurons, and subtle alterations in selected behaviors (Gupta et al., 1984; 1985). A higher LOAEL of 3.0 mg/kg bw/day was reported for a three generation study

based on fertility, number of litters, number of stillborn, and reduced survival (Lobdell and Johnston, 1964).

The NOEL is reported to be as low as 0.025 mg/kg bw/day for a 2-year study (Daly et al., 1984) and as high as 2 mg/kg bw/day for rats (NCI, 1978). In a one year study using dogs, the NOEL was determined to be 0.3 mg/kg bw/day (Ahmed et al., 1981).

5.1.18.3 Quantification of Toxic Effects

EPA criteria were unavailable: therefore, the lowest acute value reported (0.14 ppb reported as the 48-h LC_{50} for D. magna) was used to calculate a water criteria. An acute-chronic ratio could not be calculated due to lack of chronic toxicity data. An uncertainty factor of 10^2 was applied to yield an acceptable water concentration of 0.0014 ppb.

Water criteria are also estimated for toxicity due to surface water consumption. The chronic NOEL of 0.025 mg/kg bw/day for rats is lower than the NOEL for dogs of 0.3 mg/kg bw/day or the LOAEL for rats of 0.25 mg/kg bw/day. From the chronic NOEL and the water consumption for rats, an acceptable water concentration is derived as follows:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.025 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 0.2 \text{ mg/l}$$

Applying an uncertainty factor of 5 for interspecific variation, a water concentration of 0.04 mg/l (40 ppb) is estimated for methyl parathion.

There is no indication that methyl parathion bioaccumulates in the environment: therefore, a Final Residue value was not calculated. A summary of the acceptable water concentrations (ppb) for methyl parathion is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	40	NA	0.0014

The lowest estimated criteria, 0.0014 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of available data this estimate is highly uncertain.

A soil criterion for methyl parathion could not be established at this time due to lack of data.

5.1.19 METHYLPHOSPHONIC ACID (MPA)

EPA water quality criteria are unavailable for methylphosphonic acid (MPA). MPA is a water soluble hydrolysis product of isopropyl methylphosphonate that moves readily in groundwater (Rosenblatt et al., 1975a).

5.1.19.1 Aquatic Ecosystems

Plants

Schott and Worthley (1974) examined six species of vascular aquatic plants and two species of algae for toxic effects of MPA. In all vascular species, death occurred at a concentration of 1,000 ppm, while decreased growth occurred at 100 ppm in all but two species. For both species of algae, death occurred at 100 ppm, while decreased growth was observed at 10 ppm.

Invertebrates

No information regarding the toxicity of MPA was available in the literature reviewed.

Fish

No information regarding the toxicity of MPA was available in the literature reviewed.

5.1.19.2 Terrestrial Ecosystems

Plants

In a study by COE (1955), MPA aerially sprayed on plants at a rate of 1.0 lb/acre had a slight effect on black valentine bean and soybean and caused chlorosis and necrosis in morning glory. Slight stunting of oats occurred at an application rate of 0.1 lb/acre. Data regarding toxicity to plants of MPA residues in soil were unavailable.

Invertebrates

No information regarding the toxicity of MPA was available in the literature reviewed.

Birds

No information regarding the toxicity of MPA was available in the literature reviewed.

Mammals

No information regarding the toxicity of MPA was available in the literature reviewed.

5.1.19.3 Quantification of Toxic Effects

EPA criteria for the protection of aquatic organisms are unavailable. Water criteria were estimated using the lowest concentration that resulted in growth reduction for algae, and an uncertainty factor of 10 was applied to bring the LOAEL into the range of a NOEL. The acceptable water concentration is 1 ppm. This value may not be protective for other aquatic species such as macroinvertebrates or fish.

Due to the lack of data, acceptable surface water concentrations cannot be established at this time. A summary of the acceptable water concentrations for MPA (ppb) is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	NA	NA	1,000

The only estimated criterion, 1,000 ppb, is used to estimate the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of available data this estimate is highly uncertain.

Due to the lack of data, acceptable soil concentrations cannot be established at this time for MPA.

5.1.20 MUSTARD

EPA water quality criteria were unavailable in the literature reviewed. Based on the soil/water partition coefficient ranging from 27.5 (Lyman and Loreti, 1986) to 132.5 (Kenaga and Goring, 1980), some sorption to soils and sediments may occur. The half-life of mustard in water at 10°C is 55 minutes and at 25°C is 4 minutes (Small, 1984). The persistence in soil or water is 3 to 30 years (Small, 1984). Mustard spilled onto soil was still vesicant after three years (USA, 1974). Mustard is a cytotoxic agent on all tissue surfaces: repeated exposure results in hypersensitivity (USA, 1986).

5.1.20.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of mustard was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of mustard was available in the literature reviewed.

Fish

No information regarding the toxicity of mustard was available in the literature reviewed.

5.1.20.2 Terrestrial Ecosystems

Plants

No information was available in the literature reviewed for the toxicity of mustard in soil to plants. Mustard in gaseous or liquid form contacting plant leaf surfaces caused leaf tissue death and injury (Rosenblatt et al., 1975b).

Invertebrates

No information is available in the literature reviewed on the toxic effects of mustard in soil to invertebrates. Mustard is toxic and mutagenic to invertebrates but the effects could not be quantified with respect to soil concentration (Rosenblatt et al., 1975b).

Birds

No information regarding the toxicity of mustard was available in the literature reviewed.

Mammals

Ingestion of mustard contaminated food or water causes diarrhea, nausea, vomiting, pain, and prostration (USA, 1974). Once in the body, mustard reacts with proteins and nucleic acids of the lung, liver and kidney as seen in a study with mice (IARC, 1975). The LD_{50} for rats given mustard intragastrically was 17.0 mg/kg bw.

Acute dermal LD_{50} values as reported by Rosenblatt et al. (1975b) for dog, goat, guinea pig, and rabbit are 20, 50, 20, and 100 mg/kg, respectively. Dermal LD_{50} values for rat and mouse are 9 and 92 mg/kg, respectively (Sax, 1984). Toxic dermal effects include capillary hyperemia, dermal edema, and blistering (USA, 1986).

In rats and mice, inhalation LC_{50} values are 420 mg/m^3 for a two minute and 189 mg/m^3 for a 10 minute exposure (NIOSH, 1984). Via inhalation, mustard damages laryngeal and tracheobronchial mucosa, causes congestion of the pulmonary parenchyma, edema, and collapse of part or all of the lung (USA, 1986). A 2-hr exposure to concentrations barely detectable by odor produces eye lesions in rats (USA, 1974).

5.1.20.3 Quantification of Toxic Effects

EPA criteria for the protection of aquatic organisms have not been established and data on the toxicity to aquatic life are unavailable; therefore, an acceptable water concentration of mustard for aquatic organisms cannot be established.

Water criteria are also estimated based on toxicity due to consumption of contaminated surface water. The lowest health effects level for exposure by ingestion was an LD_{50} of 17 mg/kg/day for rats. Using water intake for rats of 0.125 l/kg bw/day, the acceptable water concentration is as follows:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{17 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 136 \text{ mg/l}$$

Applying an uncertainty factor of 1,000 to bring the LD_{50} into the range of a chronic NOEL, and 5 for interspecific variation, an acceptable water concentration of 0.027 mg/l (27 ppb) is derived.

There is no indication that mustard bioaccumulates: therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	27	NA	NA

The only estimated criterion, 27 ppb, is used to estimate the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of available data this estimate is highly uncertain.

Soil criteria for mustard are unavailable at this time due to lack of data.

5.1.21 NITROSODIMETHYLAMINE (DMNA)

The available data for nitrosamines indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 5,850 ppb (EPA, 1980f). Chronic data are available only for unsensitive aquatic species (EPA, 1980f). DMNA is very soluble in water and chemically stable (ACGIH, 1986). There is some dispute as to whether or not DMNA will persist in an aquatic environment. According to Tate and Alexander (1976), nitrosamines are rapidly decomposed by photolysis and do not persist in water illuminated by sunlight. Other data indicate little degradation (Tate and Alexander, 1975; Fine et al., 1977).

5.1.21.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of DMNA was available in the literature reviewed.

Invertebrates

DMNA is acutely toxic to D. magna at concentrations of 7,760 ppb (EPA, 1980f). Crayfish (Procambarus clarkii) exposed to 100 ppm DMNA for 6 months

had hyperplasia of tubular cells in the hepatopancreas, while those exposed to 200 ppm had extensive degeneration in all parts of the antennal gland (Harshbarger et al., 1971).

Fish

DMNA is acutely toxic to bluegill at concentrations of 5,850 ppb. After 52 weeks of feeding with 200 ppm DMNA, rainbow trout exhibited dose-related hepatocellular carcinoma. After 78 weeks, a higher incidence of hepatocellular carcinoma was observed even though feeding was discontinued after 52 weeks (Greico et al., 1978). The BCF for bluegill is 217; the biological half-life is less than one day (EPA, 1980f).

5.1.21.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of DMNA was available in the literature reviewed. DMNA may be taken up by some crop types under experimental conditions, but nitrosamines are not commonly found in plants under environmental conditions (EPA, 1980f).

Invertebrates

No information regarding the toxicity of DMNA was available in the literature reviewed.

Birds

No information regarding the toxicity of DMNA was available in the literature reviewed.

Mammals

The acute oral LD_{50} for rats is 40 mg/kg bw (Druckrey et al., 1967). ACGIH (1986) indicates the 4-hr LC_{50} values for inhalation by rats and mice are 78 and 57 ppm, respectively. One of three dogs exposed by inhalation for 4 hr to 16 ppm survived with liver damage (ACGIH, 1986).

Chronic exposure effects include biliary hyperplasia, fibrosis, nodular parenchymal hyperplasia, formation of enlarged hepatic parenchymal cells with large nuclei (Magee et al., 1976), and tumors of the liver and other organs (EPA, 1980f).

5.1.21.3 Quantification of Toxic Effects

No chronic EPA criteria have been established: therefore, an estimate of an acceptable water concentration was made using the EPA lowest acute value (5,850 ppb). An uncertainty factor of 10^2 was applied to yield an acceptable water concentration of DMNA of 58 ppb.

The acute oral LD_{50} was 40 mg/kg bw/day for rats. From a water consumption rate for rats of 0.125 l/kg bw/day and the LD_{50}, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{40 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 320 \text{ mg/l}$$

Application of uncertainty factors of 1,000 to convert the LD_{50} to a NOEL, and 5 for interspecific variation, result in an acceptable water concentration of 0.064 mg/l (64 ppb).

There is no indication that DMNA bioaccumulates: therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for DMNA is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
58	64	NA	NA

The lower of the estimated criteria, 58.5 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is uncertain.

Soil criteria for DMNA were not calculated due to lack of data.

5.1.22 1,4-OXATHIANE

EPA water quality criteria are unavailable for oxathiane. Oxathiane is a mustard decomposition product with a water solubility of 20 g/l.

5.1.22.1 Aquatic Ecosystems

No information regarding the toxicity of oxathiane was available in the literature reviewed.

5.1.22.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of oxathiane was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of oxathiane was available in the literature reviewed.

Birds

No information regarding the toxicity of oxathiane was available in the literature reviewed.

Mammals

In a study by Mayhew and Muni (1986), the LD_{50} for male and female rats was 3,328 mg/kg bw and 3,000 mg/kg bw, respectively. Toxic effects included coma, polypnea, lacrimation, dyspnea, lethargy, ataxia, cyanosis, squinted eyes, epistaxis, wheezing, decreased body temperature, piloerection, hunched posture, and alopecia. Results from necropsy showed discolored intestines, intestinal contents and stomach contents, gaseous stomach or intestines, and distended and discolored urinary bladder.

5.1.22.3 Quantification of Toxic Effects

No water quality criteria have been established, and data were unavailable in the literature reviewed; therefore, criteria for the protection of aquatic life could not be established for oxathiane.

Water criteria are also estimated for surface water consumption. The LD_{50} was 3,000 mg/kg bw/day for female rats. From a water consumption rate for rats of 0.125 l/kg bw/day and the LD_{50}, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{3{,}000 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 24{,}000 \text{ mg/l}$$

Uncertainty factors of 1,000 to convert the LD_{50} to a NOEL, and 5 for interspecific variation, result in an acceptable water concentration of 4.8 mg/l (4,800 ppb).

There is no indication that oxathiane bioaccumulates; therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for oxathiane is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	4,800	NA	NA

The only estimated criterion, 4,800 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria for oxathiane were not calculated due to lack of data.

5.1.23 PARATHION

EPA criteria for the protection of freshwater aquatic organisms for parathion are 0.013 ppb for a four day average concentration with the one hour average not to exceed 0.065 ppb more than once every three years (EPA, 1986a). Toxicity of parathion is the result of metabolic conversion to its oxygen analogue, paraoxon, and its subsequent binding to and inhibition of various enzyme systems, particularly acetylcholinesterase (AChE) (EPA, 1986a). Parathion has a great affinity for organic matter and is quickly adsorbed to sediments and particulate matter (EPA, 1986a). Parathion is highly insoluble in water.

5.1.23.1 Aquatic Ecosystem

Plants

The blue-green alga, Microcystis aeruginosa, exhibits incipient inhibition at a parathion concentration of 30 ppb (Bringmann and Kuhn, 1978a,b). The green alga, Scenedesmus quadricauda, is not adversely effected at concentrations below 390 ppb (Bringmann and Kuhn, 1977; 1978a,b).

Invertebrates

Acute LC_{50} values for aquatic invertebrates range from 0.04 ppb in early instar crayfish (Orconectes nais) to 5,230 ppb in tubificid worms (Tubifex sp. and Limnodrilus sp.) (EPA, 1986a) . Ahmed (1977) observed a range for 24-hr LC_{50}s from 1.8 to 40 ppb in six freshwater coleopteran species.

In a 21-day life-cycle test with Daphnia magna, the LC_{50} was 0.14 ppb; reduced number of young was observed at 0.12 ppb, and no observed effects at 0.0817 ppb (Spacie, 1976; Spacie et al., 1981).

Fish

Acute LC_{50} values for fish range from 56 ppb in guppy (Poecilia reticulata) to 2,650 ppb in channel catfish (Ictalurus punctatus) (EPA, 1986a).

Spacie (1976) and Spacie et al. (1981) conducted life-cycle tests on fathead minnows and bluegill. Fathead minnows were significantly affected by exposure to parathion at 9.0 ppb, but not at 4.4 ppb. Parathion concentrations of 0.34 ppb caused deformities and tumors in adult bluegills, but did not effect reproduction or survival of any life stage. Inhibition of AChE has been reported in brains of several freshwater fish with no effects observed at 0.17 ppb (Weiss, 1961). The results of reduction of brain AChE on normal activities such as feeding, reproduction, and predator-prey relationships are not known (EPA, 1986a).

Studies with various fish species indicated that brook trout concentrated parathion residues to a greater extent than fathead minnow or bluegill (Spacie, 1976; Spacie et al., 1981). Brook trout exposed for 180 and 260

days at varying parathion concentrations had geometric mean BCFs of 155, with a range from 31 to 573. In 260 days at varying concentrations, fathead minnows concentrated parathion residues by a mean factor of 95, with a range from 32.9 to 201.4. After 540 days, the BCF for bluegill was 27.

5.1.23.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of parathion was available in the literature reviewed.

Invertebrates

Parathion was not highly toxic to the mite, *Eriselus hibisci* (Chant) (Tanigoshi and Fargerlund, 1984). The LC_{50} for *E. hibisci* was 443.4 g (AI)/100 l (4.430 mg (AI)/l). Resistant populations of *E. hibisci* have been reported (Tanigoshi and Fargerlund, 1984), and large, species-specific differences in susceptibility to parathion have been observed (Bellows and Morse, 1988). Mortality relating to parathion treatment fell almost to control levels within 10 days following application (Bellows and Morse, 1988).

Birds

Oral LD_{50}s for nine avian species range from 1.6 mg/kg bw in mallard to 24.0 mg/kg bw in chukar (EPA, 1975). The geometric mean value for the nine species represented is 4.2 mg/kg bw.

Mammals

Acute oral LD_{50}s for parathion in rat and mouse are 2 mg/kg bw (Weiss and Orzel, 1967) and 6 mg/kg bw (Agricultural and Biological Chemistry, 1961), respectively. Female rats are more sensitive to oral doses of parathion than males as indicated by average oral LD_{50} values for male and female rats of 7.6 mg/kg bw and 3.5 mg/kg bw, respectively (EPA, 1975). The acute oral toxicity of parathion to dogs has been reported to range from 3.0 to 5.0 mg/kg bw (Council of Europe, 1964).

In long-term studies, 30 percent mortality occurred in rats fed 15.4 mg/kg bw/day for 15 to 16 weeks (Edson and Noakes, 1960). A dietary level of

50 ppm was toxic to 42 percent of a population of rats, with death occurring early in the test period (Barnes and Denz, 1951). Lehman (1952) reported a NOEL of 10 ppm (an estimated value of 0.75 mg/kg bw/day (Sax, 1984)) for rats for 2-year exposures. Based on changes in cholinesterase levels, the NOEL in rats was reported by Edson et al. (1964) as 0.02 mg/kg bw/day when fed over an 84-day period, and the LOAEL was found to range from 0.04 to 0.06 mg/kg bw/day. A dietary level of 50 ppm produced no effect on gestation in rats (Hazelton and Holland, 1959). At dietary levels of 2 ppm, plasma and RBC cholinesterase levels are reduced as much as 70 percent in dogs (Frawley and Fuyat, 1957). In a 90-day study by Hazelton and Holland (1950), dogs were nervous and irritable at concentrations of 1 mg/kg bw/day in the early stages of the study, but resumed normal behavior in the final month. Some degenerative changes in the liver were observed at this concentration. In the species studied, there is no appreciable tissue accumulation of residue, and death generally occurred only in cases where parathion was given as an acute toxic dose (EPA, 1975).

5.1.23.3 Quantification of Toxic Effects

The EPA criteria are used to establish the acceptable water concentration of parathion for aquatic organisms.

Water criteria are also estimated based on surface water consumption and health effects data. Since the subchronic NOEL for rats of 0.02 mg/kg bw/day was lower than the chronic NOEL of 0.75 mg/kg bw/day, the subchronic value was used to derive water criteria. Using a water consumption rate for rats of 0.125 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.02 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 0.16 \text{ mg/l}$$

Applying an uncertainty factor of 10 to convert the subchronic NOEL to a chronic NOEL, and 5 for interspecific variation, result in an acceptable water concentration of 0.0032 mg/l (3.2 ppb).

There is no indication that parathion bioaccumulates to a significant extent: therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for parathion is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.013	3.2	NA	NA

The lower of the estimated criteria, 0.013 ppb, is used as the acceptable water concentration of parathion that will be protective of all wildlife populations at RMA.

Soil criteria for parathion could not be calculated at this time due to lack of data.

5.1.24 POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are a mixture of chlorinated biphenyls with varying numbers of chlorine atoms on the aromatic rings (EPA, 1984g). The acute and chronic criteria for protection of aquatic organisms are 2.0 and 0.014 ppb, respectively (EPA, 1980g). PCBs have high octanol-water partition coefficients (Chiou et al., 1977), accumulate in food chains, are relatively insoluble in water, and have a high affinity for organic matter in sediments (EPA, 1980g).

5.1.24.1 Aquatic Ecosystems

Plants

PCBs reduce growth, motility, and affect cell productivity in various species of algae (EPA, 1980g). Effective concentrations range from 0.1 ppb for Scenedesmus quadricauda exposed to Aroclor 1254 for 24 hr, to 10,000 ppb for the alga, Euglena gracilis, exposed to Aroclor 1242 for 8 days (EPA, 1980g).

Invertebrates

The LC_{50} values for Aroclor 1254 for D. magna for 2 or 3 week exposures ranges from 1.3 to 24 ppb (EPA, 1980g). The 96-hr LC_{50} values for the scud, Gammarus fasciatus, range from 10 ppb for Aroclor 1242 to 2,400 ppb for Aroclor 1254 (EPA, 1980g). For the scud, G. pseudolimnaeus, 96-hr LC_{50} values ranged from 29 ppb for Aroclor 1248 to 210 ppb for 2,4,5,2',5'-pentachloro-biphenyl (EPA, 1980g). The LC_{50} for exposure to Aroclor 1254

for the damselfly, *Ischnura verticalis*, is 200 ppb (EPA, 1980g). The midge, *Tanytarsus dissimilis*, exhibits toxic effects at concentrations of 0.8 ppb, making it the most sensitive aquatic invertebrate reported in EPA, 1980g.

Fish

Under flow-through, measured conditions, PCBs are acutely toxic to fish at concentrations of 2.0 to 300 ppb (EPA, 1980g). Studies by Johnson and Finley (1980) indicated 96-hr LC_{50} values ranging from 3 to 433 ppb. Concentrations resulting in toxic effects for chronic exposures are lower, ranging from 0.1 to 15.0 ppb for fathead minnows exposed to Aroclor 1248, 1260, 1242, and 1254 (EPA, 1980g). The concentrations resulting in toxic effects on brook trout for chronic exposures also fall within this range. Brook trout fry exhibited decreased growth when exposed for 48 days to Aroclor 1254 at concentrations of 1.5 ppb or greater (Johnson and Finley, 1980). The NOEL for fish is 0.43 ppb for Aroclor 1254 using decreased hydroxyproline concentration in brook trout collagen as a toxicological endpoint (Johnson and Finley, 1980).

Bioconcentration factors for PCBs increase with increasing chlorine content (Callahan et al., 1979). BCFs for various species exposed to different PCBs range from 3,000 in brook trout muscle, to 274,000 for fathead minnow (whole body) (EPA, 1980g). Bioconcentration factors for Aroclor 1248 and 1254 by channel catfish were 56,370 to 60,190 after 60 days (Johnson and Finley, 1980).

5.1.24.2 Terrestrial Ecosystems

Plants

No information on the toxicity of PCBs to plants was found in the literature reviewed.

Invertebrates

No information on the toxicity of PCBs to invertebrates was found in the literature reviewed.

Birds

PCBs in diet at levels of 10 to 20 ppm reduced hatchability of eggs of chickens and Japanese quail: the NOEL was 1.0 ppm (0.175 mg/kg bw/day was estimated for quail assuming a food consumption equivalent to a chicken (Sax, 1984)) (Scott, 1977). No effects were observed on eggshell thickness. Reproductive success was decreased and behavior of offspring altered for adult pheasants orally administered 50 mg PCB weekly, whereas pheasants receiving 12.5 mg weekly did not differ significantly from controls (Dahlgren and Linder, 1971). Aroclor 1254 in diet at levels of 200 ppm resulted in behavioral changes in coturnix quail chicks (Kreitzer and Heinz, 1974).

Levels of PCBs are higher in fat than in other tissues of cormorants and pelicans (Greichus et al., 1973). PCBs accumulate in bald eagles: carcass residues on a lipid weight basis in one study were approximately two orders of magnitude higher than brain residues on a wet weight basis (Barbehenn and Reichel, 1981). Residues of 310 ppm or more in brain indicate death from PCB poisoning (Stickel et al., 1984). Residue concentrations in birds that died on dosage were similar in most species tested, although the time to 50 percent mortality and the brain residue levels in sacrificed survivors varied.

Mammals

Acute oral LD_{50} values for rats for various PCBs range from 0.794 to 3.169 g/kg bw (794 to 3,269 mg/kg bw) (EPA, 1980g). Chronic exposure to PCBs results in toxic effects at much lower concentrations. Exposure to PCBs can affect the liver, skin, gastrointestinal tract, and nervous system (EPA, 1980g). ATSDR (1987) presents criteria for minimal risk of noncarcinogenic effects in animals as 0.002 mg/kg/day for exposures less than or equal to 14 days, and as 0.0001 mg/kg/day for exposures exceeding 14 days.

Lethal dietary levels for different PCBs for several mammalian species range from 379 to 2,000 ppm, while nonlethal levels are observed to be 100 ppm and

lower (EPA, 1980g). Diets of 1,000 ppm (50 mg/kg bw) over a period of 3 days were lethal to male rats (EPA, 1980g); mortality occurred in rats dosed at levels of 500 ppm and higher in diet (Kimbrough et al., 1972).

Rats dosed with 100 mg/kg bw for 21 days had liver cell changes, although other toxic symptoms were lacking (Bruckner et al., 1973); litter size was decreased for rats exposed to 100 to 500 ppm Aroclor 1254 (Linder, [illegible]'4). Liver cell changes were noted in rats at doses ranging from 3.5 [illegible] 9 mg/kg bw/day Aroclor 1242 and Aroclor 1016 over a 10-month period (Bu[illegible] et al., 1974). A significant increase in liver weight and mild hepatopathology was observed in mice after dosing with Aroclor 1254 for 6 months at concentrations of 3.75 ppm in diet; increased liver weight and moderate hepatopathology were observed with Aroclor 1242 at dietary levels of 375 ppm; no liver lesions were observed in mice dosed with Aroclor 1221 (Koller, 1977).

Mink are highly sensitive to PCBs; dietary levels of 30 ppm for 6 months were lethal to 100 percent of the animals tested (Aulerich et al., 1985). No toxic effects were observed in mink fed 1 ppm PCB (Araclor 1254) in diet (0.05 mg/kg bw/day was estimated assuming a food consumption equivalent to a domestic cat (Sax, 1984)) for 8 months (Wren et al., 1987); other studies indicate no effects at concentrations in diet of 5 ppm (Byrne, 1974). However, Aulerich et al. (1985) reported increased liver weights, depressed progesterone concentrations, and elevated cytochrome P-450 concentrations following exposure to 2.5 ppm in diet.

PCBs can cross the placental membrane, and fetotoxicity can occur in the absence of maternal toxicity (EPA, 1984g). In rabbits, dosages of 10 mg/kg/day and higher resulted in maternal hepatomegaly, while 12.5 mg/kg bw/day and higher resulted in fetal death and abortion (Villeneuve et al., 1971). Progeny of mice dosed with 1 mg/kg bw/day had an increased incidence of cream colored liver and undersized renal papillae (Marks et al., 1981). Rats dosed with 70 ppm in water (6.4 mg/kg bw/day) exhibited maternal mortality at 7 weeks treatment, and fetal resorption (Orberg and Kihlstrom, 1973).

5.1.24.3 Quantification of Toxic Effects

The EPA chronic criteria (0.014 ppb) are used to establish the acceptable water concentration for aquatic organisms.

The NOEL was 0.0001 mg/kg bw/day for animals exposed to PCBs for durations exceeding 14 days. Using a geometric mean water consumption rate for rats, mice, and rabbits of 0.16 l/kg bw/day to represent an overall water consumption value for mammals, the acceptable water concentration becomes:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{0.0001 \text{ mg/kg bw/day}}{0.16 \text{ l/kg bw/day}} = 0.00062 \text{ mg/l}$$

Uncertainty factors for interspecific variation were not applied because the data were calculated from toxicity estimates and water consumption estimates for different species. The NOEL of 0.0001 mg/kg bw/day is two orders of magnitude lower than the estimated NOEL for mink (0.05 mg/kg bw/day), which are quite sensitive to the effects of PCBs; therefore, adding further uncertainty factors appears to be overly conservative.

Because PCBs bioaccumulate to a significant extent, a Final Residue Value has been calculated by EPA (1980g). The Final Residue Value is based on a mink NOEL as a MPTC. A summary of the acceptable water concentrations (ppb) for PCBs is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
0.014	0.62	0.014	NA

The lowest of the estimated criteria, 0.014 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA.

Soil criteria for PCBs could not be calculated at this time due to lack of data. Soil criteria have been estimated by acute aquatic bioassay with fish and aquatic invertebrates; however, PCBs are not as acutely toxic as they are chronically toxic, and criteria should be based on chronic toxicity or bioaccumulation potential (Hose et al., 1986).

5.1.25 TOLUENE

The EPA lowest acute value for protection of aquatic freshwater life is 17,500 ppb (EPA, 1980i). No chronic data are available. The half-life in water is 4.1 hr (MacKay and Zeum, 1983). Toluene is a volatile compound and a component of gasoline. It undergoes rapid degradation by microorganisms. The aqueous solubility of toluene is 515 mg/l.

5.1.25.1 Aquatic Ecosystems

Plants

The 24-hr EC_{50} for an alga, Chlorella vulgaris, for reduction of cell numbers is 245,000 ppb (Kauss and Hutchinson, 1975). The 96-hr values for another alga, Selenastrum capricornutum, for reduction of cell numbers or for reduction of chlorophyll a production exceed 433,000 ppb (EPA, 1978).

Invertebrates

The 48-hr EC_{50} for Daphnia magna under static test conditions is 60,000 ppb (Bringman and Kuhn, 1959). Chronic data were unavailable.

Fish

The 96-hr LC_{50} for goldfish (Carassius auratus) for static test conditions was 57,680 ppb (Pickering and Henderson, 1966), while the LC_{50} under flow-through test conditions was approximately half that of static, or 22,800 ppb (Brenniman et al., 1976). The 96-hr LC_{50}s for fathead minnow and guppy were 34,270 to 42,330, and 59,300 ppb, respectively (EPA, 1980i). The bluegill was the most sensitive fish species, with LC_{50} values ranging from 12,700 to 24,000 ppb under static test conditions; flow-through test data were unavailable for bluegill. No chronic data were available for freshwater species.

5.1.25.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of toluene was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of toluene was available in the literature reviewed.

Birds

No information regarding the toxicity of toluene was available in the literature reviewed.

Mammals

No data were available in the literature reviewed for toxic effects of toluene on wild mammals; however, data exist for laboratory animal studies. The acute oral LD_{50} for adult rats ranges between 6.4 and 7.53 g/kg bw (Wolf et al., 1956; Smyth et al., 1969; Kimura et al., 1971). Toxic effects are first observed as inhibition of central nervous system functions at dose levels of 2.0 g/kg bw (Kimura et al., 1971). The dermal LD_{50} for rabbits is 12.2 g/kg bw (Smyth et al., 1969).

Rats exposed orally to concentrations as high as 590 mg/kg bw/day for 27.6 weeks had no observed histological effects to kidneys and liver (Wolf et al., 1956). Mice dosed by gavage with 0.3, 0.5, and 1.0 ml/kg/day (260, 430, and 870 mg/kg bw/day) on days 6 through 15 of gestation had an increase of fetal mortality (Nawrot and Staples, 1979).

The LC_{50} for rats for exposure by inhalation is 4,618 ppm (17,400 mg/m^3) for a 6-hr exposure (Bonnet et al., 1982). No effects were observed at concentrations of 670 or 1,100 ppm (2,340 or 4,150 mg/m^3), but at 1,250 ppm (4,710 mg/m^3), mucous membranes became irritated and coordination was affected (Bonnet et al., 1982).

Rats chronically exposed to 30, 100, or 300 ppm in air (113, 377, or 1,130 mg/m^3) for 6 h/day, 5 day/wk, for 24 months had reduced hematocrit values at the 100 and 300 ppm dose levels (CIIT, 1980). Rats exposed to concentrations in air as high as 1,500 ppm for 26 weeks had no observed

effects (API, 1980a). In another study, rats exposed to 265 ppm by inhalation for 3 months had increased levels of cytochrome P-450, decreased body weight, and increased liver weight in females (Ungvary et al., 1980).

Toluene can interact with other contaminants. Toluene potentiates the toxicity of perchloroethylene, competitively inhibits the effects of trichloroethylene, and when administered in conjunction with m-xylene, rate of urinary excretion is depressed (EPA, 1984h).

5.1.25.3 Quantification of Toxic Effects

The EPA Lowest Acute Value (17,500 ppb) is not as low as the LC_{50} for bluegill (12,700 ppb); therefore, the LC_{50} for bluegill was used to estimate the acceptable water concentration for aquatic organisms. An uncertainty factor of 10^2 was applied to bring the acute value into the range of a chronic value. The acceptable water concentration is 127 ppb.
Water criteria are also estimated for consumption of surface water. The subchronic LOAEL for mice (260 mg/kg bw/day) is lower than the chronic NOEL for rats (590 mg/kg bw/day); therefore, the subchronic LOAEL for mice is used to derive water quality criteria. From a water consumption rate for mice of 0.2 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{LOAEL}}{\text{Water Intake}} = \frac{260 \text{ mg/kg bw/day}}{0.2 \text{ l/kg bw/day}} = 1{,}300 \text{ mg/l}$$

Applying an uncertainty factor of 50 to bring the subchronic LOAEL into the range of a chronic NOEL, and 5 for interspecific variation, result in an acceptable water concentration of 5.2 mg/l (5,200 ppb).

There is no indication that toluene bioaccumulates to a significant extent; therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for toluene is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
127	5,200	NA	NA

The lower of the estimated criteria, 127 ppb, is used as the acceptable water concentration of toluene that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria for toluene could not be calculated at this time due to insufficient data.

5.1.26 TRICHLOROETHYLENE

Data were insufficient to calculate acute criteria, but the LOAEL for acute exposure of freshwater life to trichloroethylene is 45,000 ppb (EPA, 1980j). Data were insufficient to calculate chronic criteria of trichloroethylene to freshwater life, but adverse effects were observed at concentrations as low as 21,900 ppb (EPA, 1980j). Trichloroethylene does not tend to persist in the environment due to rapid photo-oxidation in air (EPA, 1980j). Trichloroethylene is highly soluble in lipids (ACGIH, 1986).

5.1.26.1 Aquatic Ecosystems

Plants

No information regarding the toxicity of trichloroethylene was available in the literature reviewed.

Invertebrates

Static tests with D. magna resulted in 48-hr EC_{50} values ranging from 41,000 to 100,000 ppb (EPA, 1980j). Static tests with D. pulex resulted in a lower range of 48-hr EC_{50} values than D. magna, with values ranging from 39,000 to 51,000 ppb (EPA, 1980j).

Fish

In flow-through tests with fathead minnow, the 96-hr LC_{50} was 40,700 ppb (Alexander et al., 1978). Static tests resulted in a higher 96-hr LC_{50} of 66,800 ppb (Alexander et al., 1978). The 96-hr LC_{50} for bluegill was 44,700 ppb (EPA, 1978). At exposures for 96-hr to 21,900 ppb, fathead minnows exhibited loss of equilibrium (Alexander et al., 1978).

Trichloroethylene does not tend to concentrate in biota as indicated by a whole body BCF of 17 for a 14-day exposure period for bluegill (EPA, 1978). BCFs less than 100 indicate little transfer of residues up aquatic food chains (ASTM, 1985). Tissue half-life was less than one day (EPA, 1978).

5.1.26.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of trichloroethylene was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of trichloroethylene was available in the literature reviewed.

Birds

No information regarding the toxicity of trichloroethylene was available in the literature reviewed.

Mammals

Trichloroethylene is readily absorbed by all routes of exposure (Goldstein et al., 1974). In rats, 72 to 85 percent of an oral dose is excreted in expired air, 10 to 20 percent in urine, and less than 0.5 percent in feces (Daniel, 1963). The acute oral LD_{50} values for mammals range from 2,400 mg/kg bw for mice to 7,330 mg/kg bw for rabbit (ATSDR, 1988). At oral dose levels of 660.2 mg/kg bw/day for male mice and 793.3 mg/kg bw/day for female mice, decreased body weight, increased kidney and liver weights, and increased ketones and proteins in urine were observed (Tucker et al., 1982). Increased liver weights and elevated urine ketones and proteins were observed in male mice at a lower dose level of 216.7 and 393.0 mg/kg bw/day, respectively. The NOELs for male and female mice for a 6-month oral exposure are 18.4 and 17.9 mg/kg bw/day, respectively, based on effects such as increased organ weights and urinary protein levels (EPA, 19841).

The LC_{50}s for exposure by inhalation for mice range from 7,480 ppm for a 4-hr period to 49,000 ppm for a 0.5-hr period. The LC_{50}s for rats range from 12,500 ppm for a 4-hr exposure to 26,300 ppm for a 1-hr exposure

(ATSDR, 1988). Trichloroethylene is a central nervous system depressant: the effects are reversible in rats following 4-hr daily exposures by inhalation to 670 mg/m^3 over a 3 to 4 week period (Goldberg et al., 1964). Rabbits exposed by inhalation to 9,530 mg/m^3 over a 20 to 30 day period exhibited damage to the cerebellum, basal ganglia, and brain stem nuclei (Bernardi et al., 1956). Similar effects appeared in dogs exposed to 1,600 to 2,700 mg/m^3 (Baker, 1958). Other toxic effects include liver and kidney failure at high dose levels (Klaasen and Plaa, 1967), where liver failure is marked by binding of trichloroethylene metabolites to proteins and nucleic acids (Bolt and Filser, 1977).

The toxicity of trichloroethylene is enhanced by exposure to PCBs and other contaminants (Carlson, 1974; Moslen et al., 1977b; Reynolds and Moslen, 1977). For example, a synergistic effect is observed with PCBs and trichloroethylene to cause liver damage.

Data for persistence in rats exposed orally to trichloroethylene indicate a half-life of 5 hours (Daniel, 1963). Trichloroethylene was undetectable in expired air of rats 8 hours after treatment with concentrations in air of 330 ppm (Kimmerle and Eben, 1973a).

5.1.26.3 Quantification of Toxic Effects

The chronic LOAEL reported by EPA (21,900 ppb) was used to establish the acceptable water concentration for aquatic organisms. An uncertainty factor of 10 was applied to bring the LOAEL into the range of a NOEL (2,190 ppb).

The chronic NOEL was 17.9 mg/kg bw/day for female mice. Using a water consumption rate for mice of 0.2 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{17.9\ \text{mg/kg bw/day}}{0.2\ \text{l/kg bw/day}} = 89.5\ \text{mg/l}$$

Applying an uncertainty factor of 5 for interspecific variation results in an acceptable water concentration of 17.9 mg/l (17,900 ppb).

There is no indication that trichloroethylene bioaccumulates: therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for trichloroethylene is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
2.190	17.900	NA	NA

The lower of the estimated criterion, 2.190 ppb, is used as the acceptable water concentration of trichloroethylene that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria for trichloroethylene could not be calculated at this time due to insufficient data.

5.1.27 XYLENE

Ambient water quality criteria are unavailable for the protection of freshwater aquatic life. The half-life in water ranges from 2.6 to 11 days for the three forms (Burns et al. 1981). According to the EPA (1987g), xylenes bind to soil and slowly migrate with groundwater. Xylenes are biodegradable in surface water, but not in ground water. The aqueous solubility of xylene is 180 mg/l.

5.1.27.1 Aquatic Ecosystems

Plants

Elodea sp. and Potomogeton nodosus exposed to 100 ppm xylene died within 4 weeks: no effects were observed at an exposure of 5 ppm (Frank et al., 1961). Dunstan et al. (1975) exposed four species of phytoplankton to xylene. Growth was inhibited at 100 ppm for one species and at 10 ppm for the other five.

Invertebrates

No information regarding the toxicity of xylene to aquatic invertebrates was available in the literature reviewed.

Fish

For rainbow trout and bluegill, 96-hr LC_{50} values are 8.2 and 13.5 ppm, respectively (Johnson and Finley, 1980). In four species of fish, 24-hr LC_{50} values were 24 ppm for bluegill, 28.8 ppm for fathead minnow, 30.6 ppm for goldfish under flow-through conditions, and 36.8 ppm for goldfish under static conditions; the 48-hr and 96-hr LC_{50}s were similar to the 24-hr LC_{50}s for the above species (Pickering and Henderson, 1966). The major action of xylene on coho salmon is an increase in permeability of the membranes causing a loss of fatty substances (Morrow et al., 1975). Effects of acute toxicity include rapid, violent and erratic swimming; coughing or backflushing water over the gills; increased irritability; loss of equilibrium; paralysis and death (Liebmann, 1960; Morrow et al., 1975).

5.1.27.2 Terrestrial Ecosystems

Plants

No information regarding the toxicity of xylene was available in the literature reviewed.

Invertebrates

No information regarding the toxicity of xylene was available in the literature reviewed.

Birds

No information regarding the toxicity of xylene was available in the literature reviewed.

Mammals

In rats, acute oral LD_{50} values range from 4,300 to 5,000 mg/kg bw (NIOSH, 1978). Pregnant mice exposed orally to 2,060 mg/kg/day on days 6 through 15 of gestation had increased resorption, fetal malformations, and decreased fetal body weights while at 1,030 mg/kg/day, no apparent effects were observed on fetal or maternal toxicity (Marks et al., 1982).

In a study by Bowers et al. (1982), 20 male rats weighing 0.8 to 0.9 kg were fed o-xylene at a dose of 200 ppm in diet (approximately 12 mg/kg bw/day (Sax, 1984)). Animals were sacrificed at 1, 2, 3, and 6 months. No gross

pathological abnormalities of the liver were observed. This study indicated a NOEL of 200 ppm based on ultrastructural changes in liver morphology. The 4-hour inhalation LC_{50} for rats is 4,700 to 6,700 ppm (Carpenter et al., 1975; Harper et al., 1975). For female mice exposed to 2,000 ppm xylene in the air 6 h/d on days 6 through 12 of gestation, decreased fetal weights and delayed ossification occurred (Shigeta, 1983). In another study, Ungvary et al. (1980) determined an inhalation NOEL for fetotoxicity of 96 ppm.

In a study by Carpenter et al. (1975), male rats and dogs were exposed to mixed xylene vapors 6 hr/day, 5 day/week for 13 weeks. At the highest dose, 810 ppm, rats had increased erythrocyte and monocyte counts after 3 weeks which disappeared during weeks 7 through 13 of the experiment with no adverse effects at 460 ppm. At the highest dose, male dogs showed no effect on blood cell count, clinical chemistry, urinalysis, body weight, liver and kidney weight. In an 18-week inhalation study by Savolainen et al. (1979), male rats were exposed to 300 ppm for 6 hr/day, 5 day/week. Brain enzymes decreased during the study but after 18 weeks were not significantly lower than controls. Behavioral changes such as decreased preening and reduced activity were observed. In a one year toxicity study of inhaled o-xylene, Tatrai et al. (1981) estimated a NOEL in rats to be 1,000 mg/kg.

5.1.27.3 Quantification of Toxic Effects

EPA criteria have not been established for the protection of aquatic organisms for xylene, and chronic toxicity data are unavailable. The lowest acute value, 8.2 ppm for rainbow trout, is divided by an uncertainty factor of 10^2 to yield an acceptable water criterion of 0.082 mg/l (82 ppb).

Water criteria are also estimated based on toxicity due to consumption of surface water. The chronic NOEL was 12 mg/kg bw/day for male rats. Using a water consumption rate for rats of 0.125 l/kg bw/day, the acceptable water concentration becomes:

$$\frac{\text{NOEL}}{\text{Water Intake}} = \frac{12 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 96 \text{ mg/l}$$

An uncertainty factor of 5 for interspecific variation results in an acceptable water concentration of 19.2 mg/l (19,200 ppb) for xylene.

There is no indication that xylene bioaccumulates; therefore, a Final Residue Value was not calculated. A summary of the acceptable water concentrations (ppb) for xylene is as follows:

EPA	Surface Water Ingestion	Final Residue Value	Aquatic Life
NA	19,200	NA	82

The lower of the estimated criterion, 82 ppb, is used as the acceptable water concentration that will be protective of all wildlife populations at RMA. Due to the lack of data, this estimate is highly uncertain.

Soil criteria for xylene could not be calculated at this time due to insufficient data.

5.2 PATHWAYS ANALYSES FOR MAJOR CONTAMINANTS OF CONCERN

The seven major contaminants of concern (aldrin, dieldrin, arsenic, DBCP, endrin/isodrin, mercury) were addressed in greater detail than the 32 other contaminants of concern, and the estimated criteria are site-specific as opposed to more general values. The overall criteria development process is outlined in Figure 5.2-1. The acceptable concentrations in water, sediments, and soil developed by this process for the major contaminants of concern are summarized in Table 5.2-1. Because tissue concentrations in the criteria calculations were on a wet-weight basis, soil and sediment criteria area also on a wet-weight basis.

Water criteria were estimated by using several approaches and choosing the most conservative value. As in the toxicity assessments for other contaminants of concern, direct toxicity to aquatic life and to terrestrial organisms ingesting surface water were addressed: however, an effort was made to include primarily organisms that might be expected to commonly occur on RMA. EPA water quality criteria were reviewed for applicability, but not always used to represent criteria for aquatic life. In addition, food web contamination was addressed with the Pathway Analysis to estimate acceptable surface water, sediment, or soil concentrations by calculating bioaccumulation of residues in a food web developed for RMA.

Surface Water Ingestion

Organisms are potentially exposed to contaminants by ingestion of surface water, soil, and food items. The surface water pathway becomes important for animals such as small mammals, waterfowl, and raptors that might utilize surface water as a drinking water source. Bioconcentration as defined for aquatic organisms is not applicable to nonaquatic organisms, because tissue concentrations are not a direct function of water concentration. However, uptake of contaminants from surface water consumption can occur, with accumulation rates depending on the amount of water ingested daily and the concentration of contaminants in the water supply.

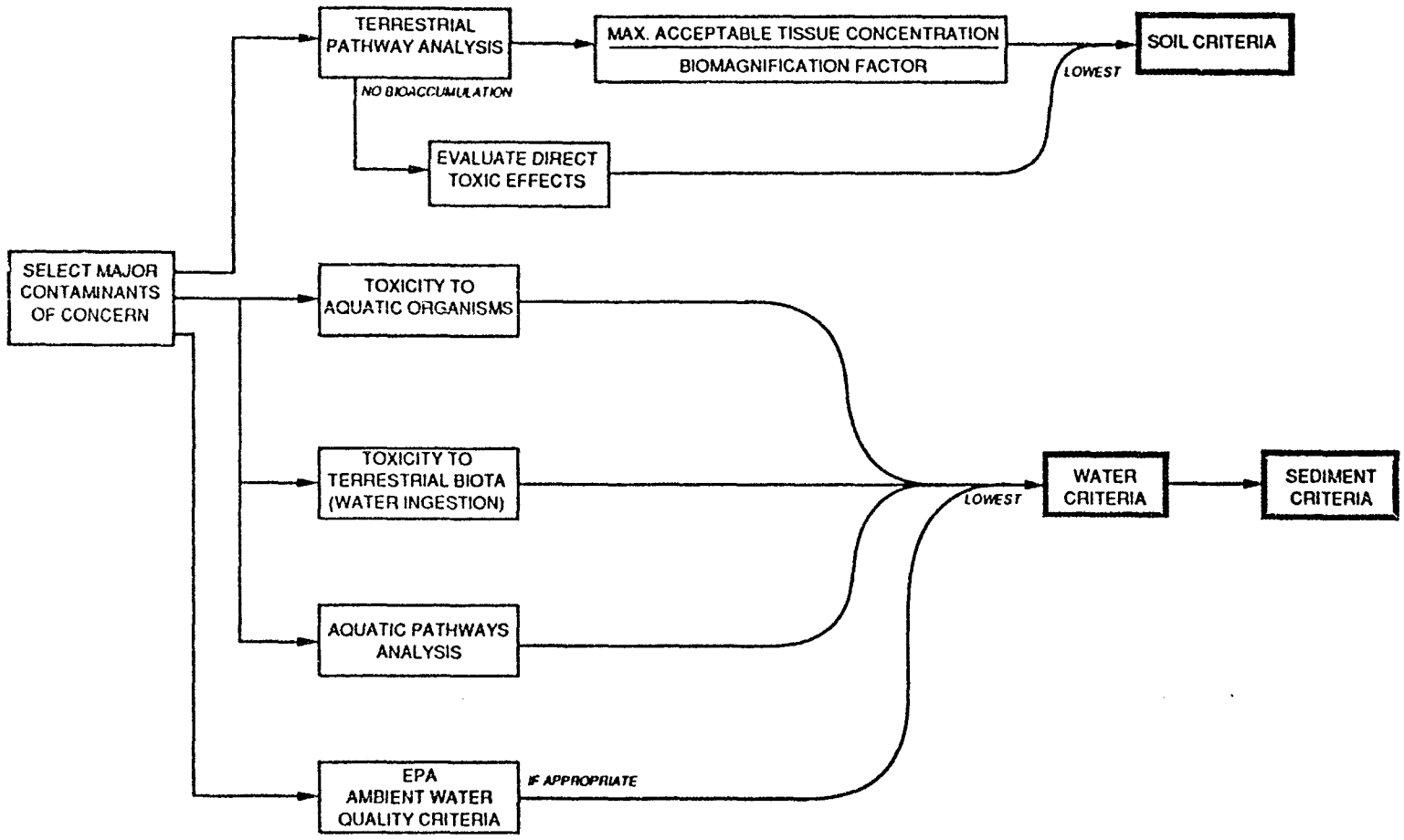


Figure 5.2-1
METHODOLOGY FOR DETERMINING CRITERIA FOR MAJOR CONTAMINANTS OF CONCERN

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal

Aberdeen Proving G nd, Maryland

Table 5.2-1. Acceptable Concentrations of the Major Contaminants of Concern in Abiotic Media

	Water (ppb)	Sediment (ppm)	Soil (ppm)
Aldrin/Dieldrin	0.034	0.0055	0.10
Arsenic	100	15	52
DBCP	60 6.10	0.086	6.10
Endrin/Isodrin	0.032	0.0019	9.2
Mercury	0.004	0.004	1.1

Source: ESE, 1988.

Many small mammals at RMA are adapted for a semi-arid environment and do not consume surface water on a regular basis. For example, black-tailed prairie dogs (Cynomys ludovicianus) and desert cottontails (Sylvilagus audubonii) obtain most of their water needs from metabolic water (Tileston et al., 1966; Turkowski, 1975). Therefore, criteria developed from water ingestion rates for laboratory animals represent a conservative estimate because many small mammals on RMA don't consume surface water on a regular basis, and many animals that consume surface water could have access to uncontaminated water supplies.

Water consumption data for laboratory rats, mice, and rabbits were used to represent small mammals at RMA that may consume surface water (Sax, 1984):

rabbit - 0.165 l/kg bw/day
mouse - 0.2 l/kg bw/day
rat - 0.125 l/kg bw/day

Assuming toxicity from dietary exposure is similar to toxicity due to ingesting contaminated water, an acceptable water concentration is derived from toxicity data for dietary intake (LOAEL or NOEL) and water ingestion rates for a similar species.

Aquatic Life

Toxicity data for aquatic life were examined to determine the most sensitive species that might occur on RMA. EPA criteria were reviewed, and used when the criteria were appropriate.

Pathway Analysis

Pathway Analysis was performed to determine cleanup criteria for the major contaminants of concern in an aquatic based food web (sediment-water-biota) and a terrestrial based food web (soil-biota) system at RMA. The method is based on reasonable estimates of exposure of various organisms to contaminants in the physical environment and the potential for bioconcentration (concentration from water), bioaccumulation (concentration from water and diet), and biomagnification (systematic concentration as chemicals are passed to higher trophic levels) exhibited by aldrin/dieldrin.

Observed data were used when available for the chemical and species specific parameters; when data were unavailable (as in the DBCP Pathway Analysis), regression techniques were utilized to obtain input such as BCFs. For the purposes of the analysis, all organisms are assumed to be in equilibrium with their environment.

The objective of the Pathway Analysis approach was to calculate a "no effect" level for the major contaminants of concern for nonhuman species and ecosystems on and near RMA. The approach arrives at a no effect level in sediments and soils on RMA by assuming that: (1) sediments or soils are the source of contamination on RMA, (2) the contaminants enter the food web from soils or sediments via water, and (3) the contaminants become concentrated in biota by the mechanisms of bioconcentration and bioaccumulation. The no effect level in water, sediment, or soil is determined by the lowest concentration obtained for these compartments based upon health effect levels and estimated concentration ratios between the biotic and abiotic environment.

Two separate food webs were developed for use in the Pathway Analysis: the bald eagle food web and the American kestrel food web. The eagle food web was composed of aquatic and terrestrial food chains, whereas the kestrel food web was strictly terrestrial. The eagle food web was applied first, and if biomagnification was insignificant (factors less than 1) in the single terrestrial food chain, the kestrel food web was not constructed for that chemical, i.e., if a contaminant showed no magnification in the eagle terrestrial food chain, then results were assumed to be similar for the kestrel food web.

The bald eagle is a federally listed endangered species and is a seasonal component of food webs on RMA. The bald eagle was selected as the target species because of its endangered status and because it represents the highest trophic level potentially affected by the bioaccumulation of contaminants through aquatic and terrestrial food chains. Aquatic organisms are considered to be the most important links in the bald eagle food web because they are constantly exposed to the contaminants in their environment

via surface adsorption, absorption, and uptake across respiratory membranes. Concentration factors from the abiotic environment through the aquatic food subweb are therefore large.

Mallards were selected to represent the waterfowl component of the bald eagle diet because they are the most abundant species of waterfowl on RMA. Breeding female mallards were chosen to represent dietary habits because they consume a large percentage of invertebrates in their diet (Swanson et al., 1979; Swanson et al., 1985), and invertebrates were expected to bioaccumulate larger residue levels than plants. Other waterfowl such as blue-winged teal were considered because they consume a diet of up to 90 percent invertebrates. However, they do not form a component of the bald eagle food web as the time each species spends at RMA does not overlap to any extent. The mallard exposure may be reduced due to their tendency to feed on seeds (which may have lower BCF values) as opposed to whole plants. To protect all species of waterfowl, the BCF values for whole plants were used in the plant to duck pathway.

BAF for ducklings was calculated to determine if criteria acceptable for adult waterfowl would be protective of ducklings. Ducklings may be at a greater risk than adult birds because they prey predominantly on insects during the first few weeks of life (Chura, 1961) and they consume a greater quantity of food per unit body weight than do adults (Heinz, 1975; Heinz, 1988). Class I ducklings (1 to 18 days of age) consume large numbers of chironomids and other invertebrates (Chura, 1961).

Dietary percentages for adult ducks in the Pathway Analysis were reported by Swanson et al. (1979, 1985). Actual food habits vary with food item availability. Pondweed, crayfish, and snails are some of the mallard food items that occur in the RMA lakes, whereas earthworms (washed in by storm events) are not expected to add to the contaminant load in mallards. Food habit data for ducks at RMA were unavailable.

Organisms used to represent lower trophic levels differ between the Pathway Analysis for each contaminant due to differences in data availability. Chironomids were separated from other invertebrates for aldrin/dieldrin

because data indicated higher BCFs; similar data were not available for DBCP; therefore, invertebrates were addressed as one group. The food web for each contaminant therefore differs slightly.

Small Mammal Soil Ingestion

Small mammals ingest soil during feeding, grooming, and burrowing activities. A mean soil ingestion rate of 0.000873 g soil/g bw/day was estimated from data reported by Garten (1980) for hispid cotton rat (*Sigmodon hispidus*), white-footed mouse (*Peromyscus leucopus*), and eastern chipmunk (*Tamias striatus*), and body weight data (Hoogland, 1981; King, 1988; Linder, 1988) as follows:

Species	Soil Content of GI Tract (g)	Median Body Weight (g)	Soil Ingestion Rate (g soil/g bw)
Cotton Rat	0.045	160	0.00028
Chipmunk	0.14	90	0.0016
White-footed Mouse	0.013	17.5	0.00074

When soil content of the gastrointestinal tract (GI tract) was reported as a range (Garten, 1980), the median was used in calculating ingestion rates. It is assumed that soil content of the GI tract represents daily intake.

The soil ingestion rate was compared to the soil criteria for each contaminant to determine if the criteria were protective of both food and soil ingestion exposure. Because the method used to estimate soil criteria does not incorporate direct ingestion rates, but relies instead on calculating overall residue magnification, the soil ingestion rate cannot be applied to criteria formulation.

5.2.1 PATHWAY ANALYSIS FOR ALDRIN/DIELDRIN

5.2.1.1 Background Information

The data for water and tissue concentrations used in this analysis were from previously collected and documented RMA samples (Rosenlund et al., 1986). Where applicable, published values are used for BCF and BAF.

The organochlorine pesticides aldrin and dieldrin have been observed in soil, water, and biota on and near RMA. These chemicals tend to be stable in the environment and are known to accumulate in food chains (Stickel, 1973). Dieldrin was selected for analysis because of its known distribution on RMA (ESE, 1987, RIC#88204R02), its toxicity and persistence in the environment, and its high potential for bioaccumulation (Stickel, 1973). Values obtained for dieldrin using the Pathway Analysis approach were assumed to represent aldrin as well because aldrin is generally found in low concentrations, and because it converts rapidly to dieldrin in the environment and in vivo (Hall et al., 1971; Metcalf et al., 1973). Using values for dieldrin to represent behavior of both organochlorines is consistent with current EPA methodology (EPA, 1980a).

Toxicity of Dieldrin

Dieldrin is toxic to all forms of biota in both aquatic and terrestrial ecosystems. The EPA criterion for protection of aquatic life is 0.0019 ppb dieldrin as a 24-hr average, not to exceed 2.5 ppb at any time (EPA, 1980a; EPA, 1986d).

Aquatic Plants--Aquatic plants are more resistant to the toxic effects of dieldrin than animals. The lowest concentration of dieldrin in water that is toxic to plants was 100 ppb for a period of 10 days (EPA, 1980a).

Aquatic Invertebrates--Some aquatic invertebrates are highly sensitive to dieldrin. In a South Carolina river, long term dieldrin exposure reduced numbers of organisms by as much as 100 percent and altered population distributions of aquatic invertebrates in relation to upstream controls (Wallace and Brady, 1971). The groups adversely affected included Ephemeroptera, Megaloptera, Trichoptera, and Plecoptera. Water concentrations ranged from a high of 17 ppb directly downstream of the discharge water to 6 ppb approximately 5 miles downstream. For aquatic invertebrates, the concentration that produced mortality in 50 percent of the population (LC_{50}) for a 30-day chronic exposure was as low as 0.2 ppb (EPA, 1980a). The lowest acute value for invertebrates was the 96-h LC_{50} for isopod of 5 ppb, whereas the chronic value for D. magna in a life cycle test was 57 ppb (EPA, 1980).

Fish--Low levels of dieldrin are highly toxic to fish. Dieldrin is acutely toxic with a 96-hr LC_{50} ranging from 1.1 to 9.9 ppb for rainbow trout (Salmo gairdneri), the most sensitive species considered (EPA, 1980a). Bluegill (Lepomis macrochirus) are less sensitive than trout, with a 96-hr LC_{50} range of 8 to 32 ppb (EPA, 1980a). Rainbow trout were the most sensitive species in chronic as well as acute tests, with average concentrations of 0.22 ppb producing toxic effects in an early life stage study (EPA, 1980a). Sensitivity to dieldrin in aquatic systems does not appear to correlate with trophic level.

Birds--The acute oral toxicity of dieldrin to birds varies. The LD_{50} for sharp-tailed grouse (Pedioecetes phasianellus), bobwhite quail (Colinus virginianus), and ring-necked pheasant (Phasianus colchicus) was 6.9 milligram toxicant per kilogram body weight (mg/kg bw), 12 to 14 mg/kg bw, and 10 mg/kg bw, respectively (McEwen and Brown, 1966). In a study of six avian species, the acute oral toxicity of dieldrin ranged from an LD_{50} of 23.4 mg/kg for chukar (Alectoris chukar) to 79.0 mg/kg for ring-necked pheasant (Tucker and Haegele, 1971). Mourning doves (Zenaida macroura) were observed to have an LD_{50} of 44 to 46 mg/kg (Dahlen and Haugen, 1954).

In birds, diagnosing death by dieldrin poisoning is best done by measuring concentrations in brain tissue, due to the mobilization of fat during later stages of poisoning and the subsequent redistribution of residues to other tissues (Wiemeyer and Cromartie, 1981). Studies indicate that brain levels as low as 5 ppm are hazardous to some bird species, and that 9 ppm is diagnostic for dieldrin poisoning (Ohlendorf et al., 1981). Other studies indicate that the lower lethal level in avian brain tissue is 4.0 ppm, with 80 percent of this level, or 3.2 ppm, considered hazardous (Wiemeyer and Cromartie, 1981). Other evidence indicates that levels as low as 1 ppm in brain have been observed to affect cowbirds (Molothrus ater) adversely (Heinz and Johnson, 1981).

Brain residues representative of dieldrin poisoning in quail range from 7.48 to 11.43 ppm; brain residues in birds that died during treatment with dieldrin were not correlated with treatment level, sex, or reproductive

status (Fergin and Schafer, 1977). Previous observations by Stickel et al. (1969) indicate that treatment level affects time of death, but not brain residues, which apparently attain a lethal threshold regardless of dietary concentration. These observations indicate that as long as uptake rate exceeds loss rate, even low levels of dieldrin exposure, given enough time, could eventually result in lethal brain concentrations.

Snow geese (Chen caerulescens) found dead after feeding on aldrin-treated seed in rice fields had brain levels of dieldrin ranging from 2.1 to 31 ppm, while brain levels in moribund geese ranged from 4.9 to 14.0 ppm (Flickinger, 1979). In a study on kestrels, 69 percent of the birds receiving 3 ppm dieldrin in diet in conjunction with DDT, had brain levels of 1 ppm dieldrin (Wiemeyer et al., 1986).

Increasing dieldrin levels in the diet of mallard ducks (Anas platyrhynchos) (4, 10, and 30 ppm) resulted in a decrease in the biogenic amines serotonin, norepinephrine, and dopamine (Sharma et al., 1976). Additionally, increases in hepatic microsomal enzymes and liver protein, DNA, and RNA were observed.

The ratio of liver and brain weight to body weight increased with increasing dietary levels of dieldrin, and behavioral changes (decreased pecking and increased avoidance action) were seen (Sharma et al., 1976). Depletion of neurotransmitters has been observed in other bird species fed 4 and 16 ppm (Heinz et al., 1980). Dieldrin has been observed to affect whole brain serotonin when 10 mg/kg body weight was given orally to hens (Willhite and Sharma, 1978).

Behavioral changes in birds at low dieldrin exposures have been noted in other studies. Busbee (1977) observed changes in the ontogeny of mouse killing in loggerhead shrikes (Lanius ludovicianus) at dietary levels of 2 ppm. At dietary levels of 5 ppm in quail chicks (Coturnix coturnix), dieldrin suppressed the group avoidance response to a moving silhouette (Kreitzer and Heinz, 1974). Offspring of pheasants dosed with dieldrin were more easily caught by hand and chose the deep side of a visual cliff more often than controls (Dahlgren and Linder, 1974). Both behavioral responses in pheasant chicks could have potentially negative effects on survivability.

Environmental factors can play a critical role in determining levels of dieldrin toxic to birds. Dietary levels of 1 ppm in a high protein diet fed ad libitum resulted in 100 percent mortality in quail when administered during a growth period (DeWitt, 1956). The same amount administered in a winter maintenance diet produced no ill effects. As consumption data were not presented, it is not known whether the rate of intake influenced the mortality rates. Chickens exposed to dieldrin at dietary levels of 10 and 20 ppm died before controls during periods of starvation (Davison et al., 1971). Breeding birds on long photoperiods were more susceptible to dieldrin toxicosis than nonbreeding birds (Fergin and Schafer, 1977).

Dieldrin accumulates in egg yolks, and while not affecting hatchability, may poison chicks (St. Omer, 1970). Dieldrin produced slight but significant eggshell thinning in barn owls (Tyto alba), but did not reduce overall breeding success (Mendenhall et al., 1983). The estimated critical level (lowest concentration at which effects occur) of dieldrin in eggs is greater than 1 ppm (Blus, 1982).

Low levels of dieldrin have been detected in several species of birds collected in eight western states (DeWeese et al., 1986). Arithmetic mean dieldrin concentrations ranged from <0.01 to 0.13 ppm on a wet-weight basis. Detections occurred primarily in migratory insectivores: dieldrin did not occur in migratory ornnivores or herbivores, or in any non migratory species. Dieldrin occurred in only 4 percetn of 124 samples collected (DeWeese et al., 1986).

Mammals--The acute oral LD_{50} values for mammals tend to be higher than those for birds. The LD_{50} values for rats, mice, and rabbits are 43 to 64 mg/kg, 38 to 75 mg/kg, and 45 to 50 mg/kg, respectively (St. Omer, 1970). LD_{50} values ranged from 94 to 229 mg/kg in several species of voles (Cholakis et al., 1981). Chronic exposure to 2.5, 12.5, or 25.0 ppm dieldrin in diet for 2 years had no effect on mortality or longevity of rats (Treon and Cleveland, 1955a). For voles, 30-day LC_{50} values ranged from 43 to 129 mg/kg (Cholakis et al., 1981).

Brain concentrations diagnostic with death range from 2.4 to 9.4 ppm in dogs (Harrison et al., 1963) and 2.1 to 10.8 ppm in rats (Hayes, 1974). Rabbits from a dieldrin-treated area were found dead with 8.4 to 19.1 ppm in brain (Stickel et al., 1969). Gray bats (Myotis grisescens) were recovered from caves with 5.6 to 21 ppm (wet weight basis) in brain tissue (Clark et al., 1983), levels considered diagnostic of dieldrin poisoning. Dieldrin has been observed to affect whole brain serotonin when 10 mg/kg was injected intraperitoneally into hamsters (Willhite and Sharma, 1978).

Bioaccumulation Potential of Dieldrin

Dieldrin tends to accumulate in food chains, with residue levels increasing with trophic level (Chadwick and Brocksen, 1969). Residues correlate not only with feeding habits, but with age and fat content of fish (Frank et al., 1974). Under laboratory conditions, bioconcentration factors in a food chain consisting of algae, Daphnia, and guppies were 1,282, 13,954, and 49,307 (dry weight basis), respectively (Reinert, 1972).

Aquatic Ecosystems--Aquatic animals can accumulate dieldrin by factors many times greater than its concentration in water. The EPA (1980a) gives a range of BCFs for various freshwater fish from 2,385 to 68,286, and factors as high as 100,000 are documented (Reinert, 1970). Sculpins exposed to concentrations of dieldrin ranging from 0.017 to 8.60 ppb in water for 32 days had BCFs approaching 10,000 (Chadwick and Brocksen, 1969). The fish had not attained equilibrium with water at 32 days. Bioconcentration factors of 70,000 have been observed in bottom feeding fish from a contaminated reservoir (Schnoor, 1981). Observed BCFs for dieldrin between aquatic invertebrates and water are as high as 17,000 (Wallace and Brady, 1971). In 7- to 12-day tests, BCFs of 1,160 were observed in freshwater mussels (Lampsilis siliquoidea) (EPA, 1980a). BCFs of 2,000 for a 72-hr test have been observed in an estuarine mollusk (Rangia cuneata) (Petrocelli et al., 1973).

The bulk of residue accumulation in aquatic ecosystems is derived from water, with accumulation due to consumption of contaminated food making up a

small percent (Chadwick and Brocksen, 1969). The amount of dieldrin that fish accumulated through food was approximately 16 percent in one study (Chadwick and Brocksen, 1969).

Terrestrial Ecosystems--In terrestrial ecosystems, the bulk of residue accumulation is a function of uptake from diet as opposed to uptake from water and diet. This residue accumulation is lower in terrestrial ecosystems than in aquatic ecosystems because dosage is not continual as it is in aquatic systems. Earthworms concentrate aldrin-dieldrin residues from 4 to 15 times the level found in field soil (Korschgen, 1971). Residues in earthworms have been observed under field conditions to be eight times higher than in soil (Beyer and Gish, 1980). In laboratory studies, swine and cattle concentrated dieldrin at factors of 0.8 to 2.7 and 1.6 to 3.0 times greater than dietary levels, respectively (Kenaga, 1980).

Simulated terrestrial ecosystem studies indicate that earthworms concentrate dieldrin 7.1 times the soil level; various insect species had concentration factors 11.9 to 58.4 times the soil level; and adult snails had concentration factors 61.4 times the soil level (Gile and Gillett, 1979). Juvenile snails concentrate dieldrin 3 to 4 times more than adult snails. Voles (*Microtus canicaudus*) from the same microcosm had average concentration factors of 59.5.

Fate of Dieldrin in the Environment

Dieldrin is stable and persistent in the environment, with a low volatility (a vapor pressure of 1.78×10^{-7} mm Hg at 20°C) and a low water solubility (186 ug/l at 25 to 29°C) (EPA, 1980a). Dieldrin is apolar and lipophilic, attracted to fats, plant waxes, and organic matter such as in sediments or soils (EPA, 1980a).

In soil, dieldrin has a half-life of 5.1 years, and a half-life in earthworms of 2.6 years (Beyer and Gish, 1980). Persistence is apparently a function of treatment level. Concentrations potentially hazardous to earthworms (8 ppm) remained in soil for up to 3 years in plots treated with

2.2 kilograms/hectare (kg/ha), and up to 11 years in plots treated with 9.0 kg/ha (Beyer and Gish, 1980). Edwards (1966) estimated that 5 to 25 years was necessary for 95 percent of an application to be removed from soil.

Dieldrin does not leach readily; the bulk of depletion is a function of volatilization as opposed to uptake, degradation, or runoff (Beyer and Gish, 1980). Neither mixing soil or adding organic matter appear to influence loss of residues (Guenzi et al., 1971).

Few soil microbes degrade dieldrin, as evidenced by experiments indicating that only 10 of 600 soil cultures were active with respect to dieldrin (Matsumura and Boush, 1967). Microbial attack usually occurs on the nonchlorinated ring, with epoxidation (such as aldrin to dieldrin) and rearrangement (such as intramolecular bridge formation to form photodieldrin from dieldrin) being the most common reactions (Matsumura, 1980). Photodieldrin can be further metabolized to two hydrophilic metabolites by various microorganisms. The major end products of microbial metabolism are ketones (Matsumura, 1980).

In insects, a monohydroxylated dieldrin, 9-hydroxy-dieldrin, and both cis- and trans-aldrindiol have been observed (Matsumura, 1980). Photodieldrin and photoaldrin are metabolites that are approximately as toxic as the parent compound to blue-green algae (Batterton et al., 1971). Trans-aldrindiol and photodieldrin are more toxic than dieldrin to insects (Matsumura, 1980).

In mammals, dieldrin is metabolized by three separate mechanisms to 2-ketodieldrin, trans-aldrindiol, and 9-hydroxy-dieldrin, respectively (Matsumura, 1980). Trans-aldrindiol is less toxic than the parent compound to mammals, and further metabolizes to aldrin diacid.

5.2.1.2 Surface Water Ingestion

Mammals

The chronic NOEL for rats was the lowest observed health effects level for mammals (Table 5.2-2). The NOEL for rats exposed for 2 years was 2.5 ppm in diet (0.19 mg/kg bw/day) (Treon and Cleveland, 1955a). The acceptable water

Table 5.2-2. Toxic Effects Levels of Dieldrin for Mammals and Birds by Ingestion.

Species	Exposure Route	Dose (mg/kg bw/day)	Effect	Acceptable Water Concentration (ppm)	Source
Rat	Diet	8.2	30-d LC_{50}	0.26	Cholakis et al., 1981
Vole*	Diet	9	30-d LC_{50}	0.29	Cholakis et al., 1981
Rat	Diet	0.19	No observed effects in 2 yrs	0.30	Treon and Cleveland, 1955a
Mallard	Diet	0.40	Decrease neurotrans-mitters (75 days)	0.0080	Sharma et al., 1976
Loggerhead Shrike	Oral	1	Behavioral changes (105 days)	0.2	Busbee, 1977.

* Assuming a water consumption rate equivalent to rats.

Source: ESE, 1988.

concentration was derived from the NOEL for rats and the water intake for rats:

$$\frac{\text{NOEL}}{\text{Water Intake/kg bw/day}} = \frac{0.19 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 1.52 \text{ mg/l}$$

Applying an uncertainty factor of 5 for interspecific variation, the acceptable surface water concentration based on toxicity to mammals is 0.30 mg/l (300 ppb). It is assumed that criteria developed from health effects data and water consumption rates for small mammals will be protective of large mammals as well.

Birds

Data were examined to determine the most sensitive toxicological endpoint for avian species. Busbee (1977) observed changes in the ontogeny of mouse killing behavior in loggerhead shrikes (*Lanius ludovicianus*) at dose levels of 1 mg/kg bw/day. At dietary levels of 5 ppm, dieldrin suppressed quail chicks (*Coturnix coturnix*) group avoidance response to a moving silhouette (Kreitzer and Heinz, 1974). Depletion of neurotransmitters has been observed in birds fed 4 and 16 ppm dieldrin (Heinz *et al.*, 1980). Dieldrin levels of 4 ppm in diet for 75 days of mallard ducks (*Anas platyrhynchos*) resulted in a decrease in the biogenic amines serotonin, norepinephrine, and dopamine, as well as other dose related effects (Sharma *et al.*, 1976). Ducks in captivity consume a diet of 100 g/kg bw daily (Sax, 1984). Total intake correlating with 4 ppm in diet is estimated to be 0.40 mg/kg bw/day.

Ducks in captivity consume 200 ml/kg bw water on a daily basis (Sax, 1984). Assuming that wild populations of ducks consume an equivalent amount of water as ducks in captivity, an acceptable water concentration can be derived as follows:

$$\frac{\text{LOAEL or NOEL}}{\text{Water Intake/kg bw/day}} = \text{Acceptable Surface Water Concentration}$$

The acceptable water concentration is estimated as follows:

$$\frac{\text{LOAEL}}{\text{Water Intake/kg bw/day}} = \frac{0.40 \text{ mg/kg bw/day}}{0.200 \text{ l/kg bw/day}} = 2.0 \text{ mg/l}$$

An uncertainty factor of 50 is applied to bring the subchronic LOAEL into the range of chronic NOEL, and an uncertainty factor of 5 is applied for interspecific variation, resulting in an acceptable water concentration of 0.0080 mg/l (8.0 ppb).

The lowest acceptable concentration in surface water for birds or mammals is 0.0080 ppm, based on toxicity to waterfowl (Table 5.2-2). The corresponding sediment criterion, based on a K_d of 24,400 and f_{oc} of 0.0065, is 1.27 ppm. This level is assumed to be protective of all wildlife consuming surface water at RMA.

5.2.1.3 Aquatic_Life

To estimate site-specific criteria for aquatic life in the RMA lakes, data for species that occur at RMA were examined for the lowest acute value or the lowest chronic LOAEL. The lowest acute value for fish or invertebrates that occur at RMA was the 96-h LC_{50} for isopod of 5 ppb, whereas the chronic value for D. magna for a life cycle test was 57 ppb (EPA, 1980). Since the acute value for isopods was lower than the chronic value for the cladoceran, the acute value of 5 ppb, divided by an uncertainty factor of 10^2 (see Section 5.1), represents a "no effects" level in water of 0.05 ppb for aquatic life at RMA. If the chronic value was used to estimate the criterion, the criterion would exceed the acute values for isopods even after application of uncertainty factors to the chronic value. The corresponding sediment criterion is calculated as follows:

$$C_{sed} = C_w \times K_{oc} \times f_{oc}$$

where: K_{oc} = 24,400 (Kadeg et al., 1986)

f_{oc} = 0.0065 (EBASCO, 1988)

$$C_{sed} = 0.05 \text{ ppb} \times 24{,}400 \times 0.0065$$

$$C_{sed} = 7.93 \text{ ppb } (0.0079 \text{ ppm})$$

5.2.1.4 Aquatic_Pathway_Analysis

Introduction_to_Aquatic_Pathway_Analysis

The Pathway Analysis for dieldrin is based on the bald eagle sink food subweb (portion of the comprehensive ecosystem food web leading to a target

species) and includes all major food chains leading to the selected sink species (Cohen, 1978). Because the same organisms/groups appear in more than one food chain throughout the web, percentage contributions for each organism or compartment have been estimated based on existing literature. The subweb has been simplified (e.g., bluegill represent all fish species at that trophic level) because of the limited data available.

Methods for Aquatic Pathway Analysis

The food habits data specific for the dieldrin Pathway Analysis are presented in Table 5.2-3. Bioconcentration factors for the lower trophic level organisms (assumed to be in equilibrium with their environment) were estimated from data collected on RMA (Rosenlund et al., 1986). Because contaminant levels in biota were higher in Lower Derby Lake, data from Lower Derby Lake were used to represent all the lakes. Field data actually represent BAFs as opposed to BCFs, but because residue contribution from diet at the lower trophic levels is less than residue contribution from water, BAF and BCF are therefore considered to be equivalent for the lowest trophic level organisms. Field data were not used to represent BCFs for fish due to the potentially significant contribution dietary residues can make to whole body residues of the consumer organism. BCFs derived from data collected on RMA (Lower Derby Lake) were used to represent bioconcentration in lower trophic level organisms because these data are believed to estimate actual chemical fate at this site more realistically than laboratory derived data.

The observed concentration of dieldrin in surface water at RMA at the time of the Rosenlund et al. (1986) study was below the detection limit of 0.00004 ppm (0.04 ppb) for dieldrin in water. Sediment concentrations in Lake Derby ranged from 1 to 4 ppb near the shore to 220 ppb in the deeper areas (Myers et al., 1983, RIC*84086R01). Based on a K_{oc} of 24,400 and a f_{oc} of 0.0065, water concentrations were estimated to range from a low of 0.0063 ppb to 0.025 ppb near the shore, to 1.39 ppb in deeper areas. Due to dilution, water concentrations as high as 1.29 ppb have not been observed: therefore, a median value obtained from the data for shallow sediments and water (0.016 ppb), was used to represent water concentrations in Lake Derby.

Table 5.2-3. Summary Of Feeding Habits for Dieldrin Pathways Analysis

Species	Food Items	% in Diet	Sources
Mallard	Snails	14	Swanson et al., 1985;
	Other Invertebrates[1]	29	Swanson et al., 1979
	Chironomids	1	
	Plants[2]	30	Swanson et al., 1979 Swanson et al., 1985
	Annelids[3]	26	Swanson et al., 1979
Bald Eagle	Waterfowl	24	Cash et al., 1985; Todd et al., 1982
	Fish	66	Cash et al., 1985
	Mammals	10	Cash et al., 1985
Bluegill	Invertebrates	88	Martin et al., 1961
	Plankton, Algae	12	Martin et al., 1961
Pike	Fish[4]	100	Inskip, 1982

1 Includes Crustacea, Insecta (other than chironomids), and miscellaneous animal food items.

2 "Plants" includes fruits such as barnyard grass (Echinochloa crusgalli) and other miscellaneous seeds (Swanson et al., 1979; Swanson et al., 1985). Fruits were included with other vegetation forming the mallards diet, although data quantifying dieldrin adsorption or absorption by aquatic fruits was unavailable in the literature researched.

3 These food items were not utilized in the pathways analysis. Annelids are apparently washed into aquatic systems (Swanson et al., 1979) and were not included because areas upgradient of the RMA lakes are considered to be uncontaminated.

4 Pike are opportunistic feeders that will utilize other food sources, but are assumed to prey completely on fish for the sake of the analysis.

Source: ESE, 1988.

Bioconcentration factors can also be calculated from various regression equations and chemical data. Using the formula,

$$\log BCF = 0.85 \log K_{OW} - 0.70 \text{ (Veith et al., 1979)} \quad (9)$$

where: K_{OW} = octanol-water partition coefficient

Calculated BCFs for fish can range from 4,853 to 9,078, depending on the value chosen for log K_{OW}, e.g., 5.16 (Garten and Trabalka, 1983) or 5.48 (Kenaga, 1980). Use of K_{OW} to define BCF does not take into consideration biological or species variation unless the regression is obtained by testing with different species. Static laboratory tests have resulted in BCFs (2,385 to 68,286) (EPA, 1980a) exceeding the range given by equation 1 by almost an order of magnitude. Flow-through tests are probably more representative of actual conditions in the field; for this reason, data derived from flow-through tests were used to represent bioconcentration in fish.

Geometric mean BCF values for aquatic invertebrates, aquatic plants, and snails using RMA data for biota and water were 920, 2,200, and 4,600, respectively (Rosenlund et al., 1986); these results fall below the low end of the estimated range using equation (9). If bioconcentration by organisms at RMA is better approximated by using regression equations than by observation, then some values derived using the Rosenlund et al. (1986) data are too low. Observed BCFs possibly underestimate actual BCFs for organisms at specific locations in the lower lakes, since the concentration of dieldrin in the lake water is below the current detection limit.

Published values were used for bioconcentration factors for fish because tissue levels observed in fish from RMA are a function not only of bioconcentration but of biomagnification. Tissue concentrations in higher trophic level organisms; therefore, cannot be related directly back to water concentrations. Table 5.2-4 lists the BCF values used in this study. When tissue concentrations were below the detection limit, a value of one-half the detection limit was used to represent tissue concentration.

Table 5.2-4. Bioconcentration Factors Used in the Pathways Analysis for Dieldrin

Organism/Group	C_b	BCF	Geometric Mean	Source
Snail[1]	0.090	2,250		2
	0.060	1,500	1,800	2
Chironomid[1]	0.210	5,200	NA	2
Invertebrate[1]	0.02	500		2
	0.05	1,250	790	2
Aquatic Plants[1]	0.047	1,175		2
	0.026	650		2
	0.059	1,475		2
	0.083	2,075		2
	0.032	800		2
	0.018	450		2
	0.127	3,175		2
	0.056	1,400		2
	0.052	1,300	1,200	2
Plankton[1]	0.120	3,000		2
	0.140	3,500		2
	0.160	4,000		2
	0.060	1,500		2
	0.320	8,000		2
	0.500	12,500	4,300	2
Bluegill		5,800	NA	3
Northern Pike		5,800	NA	3

1 A C_w of 0.00004 ppm was used in order to calculate BCF.

2 Rosenlund et al., 1986.

3 Kenaga, 1980.

Source: ESE, 1988.

Eight food transfer pathways ultimately terminating with the bald eagle were established for the dieldrin Pathway Analysis as follows:

Pathway	Source	Trophic Level 1	2	3	4
1	H_2O	Snails	Mallard	Bald Eagle	
2	H_2O	Chironomid	Mallard	Bald Eagle	
3	H_2O	Invertebrates	Mallard	Bald Eagle	
4	H_2O	Aquatic Plants	Mallard	Bald Eagle	
5	H_2O	Plankton	Bluegill	Pike	Bald Eagle
6	H_2O	Invertebrates	Bluegill	Pike	Bald Eagle
7	H_2O	Chironomids	Bluegill	Pike	Bald Eagle
8	Soil	Terrestrial Plants	Small Mammals	Bald Eagle	

The bald eagle sink food web (combined food transfer pathways based on an aquatic or terrestrial diet) is presented in Figure 5.2-2.

The mallard and the pike (*Esox lucius*) represent the sum total of birds and fish fed upon by the bald eagle. Chironomids (midges) and snails were treated separately from other invertebrates because their accumulation of dieldrin residues was higher (Table 5.2-4). Pathway Eight originates in soil and is addressed in Section 5.2.2.6.

The lowest step in the food chain is assumed to be in equilibrium with the aquatic environment, which gives equation (1):

$$BCF = C_b/C_w \qquad (1)$$

where: C_b = the concentration of dieldrin in biota

C_w = the concentration of dieldrin in water

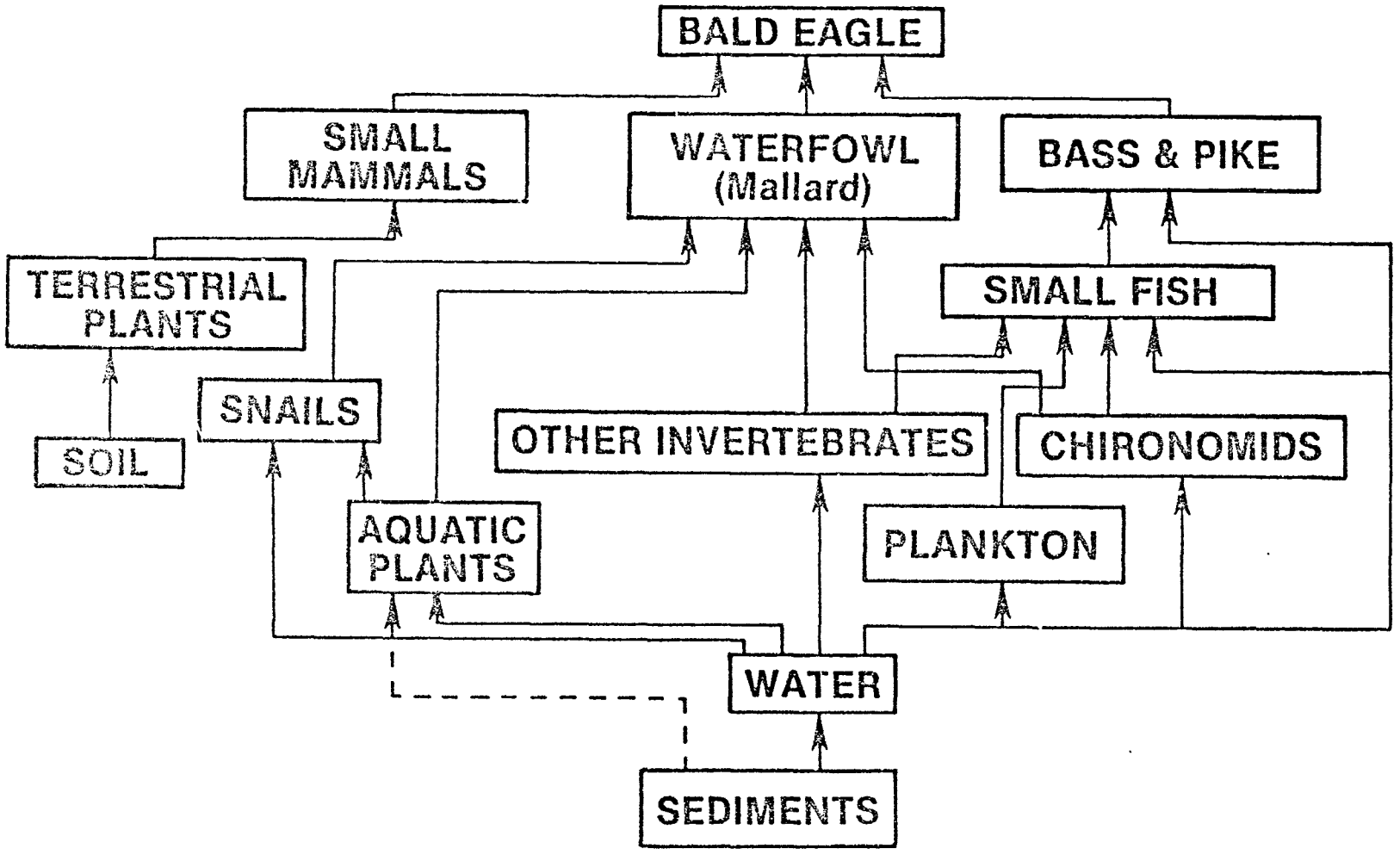


Figure 5.2-2

BALD EAGLE SINK FOOD WEB FOR DIELDRIN

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

This equation is vital to the rest of the analysis. The end result, the total BAF for the bald eagle, can be ultimately traced back through water to the sediment, because all dieldrin is assumed to enter the water compartment from sediment before being taken up by the biological compartment; i.e.,

$$C_w = \frac{C_{sed}}{K_{oc} \times f_{oc}} \qquad (7)$$

or solving for C_{sed} gives equation (8):

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \qquad (8)$$

where: C_{sed} = concentration of dieldrin in the sediment

K_{oc} = soil-water partition coefficient normalized for organic carbon

f_{oc} = fraction of organic carbon

The method used in the aquatic Pathway Analysis to estimate bioaccumulation factors is the Thomann (1981) bioaccumulation model of food chain transfer in aquatic ecosystems where each level is a step in the food chain:

Level #1 $BCF_1 = C_b/C_w$ (1)

Level #2 $BAF_2 = BCF_2 + f_2BCF_1$ (2)

Level #3 $BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1$ (3)

Level #4 $BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1$ (4)

The food term (f_i) is dependent on the trophic level in question and is calculated by the following equation:

$$f_i = \frac{@ \times R \times \%}{k_2} \qquad (5)$$

where: @ = Assimilation efficiency, $\frac{\text{ug absorbed}}{\text{ug ingested}}$

R = Total daily diet, intake (g)/body weight(g)/day

k_2 = Depuration or loss rate, day^{-1}

% = Percent of item in diet

The assimilation efficiency (@) could not be obtained for every animal addressed in this analysis; therefore, it was assumed to be 0.9 for all animals. Spacie and Hamelink (1985) used 0.9 as the assimilation efficiency for PCBs and DDT.

The depuration rate (k_2) includes loss due to growth, excretion, and metabolism. Because rate constants have not been measured for each species in this analysis, k_2 values were taken from the literature or derived by calculation using regression equations in order to represent all species. The following k_2 values were used in the aquatic Pathway Analysis:

k_2 = 0.02/day	This is based on a study of turkeys that lost approximately 2 percent of labeled dieldrin per day (Davison and Sell, 1979).
k_2 = 0.0075/day	Derived from data from pheasant carcass for three dose levels, where k2 ranged from 0.0037 to 0.013/day (Hall et al., 1971).
k_2 = 0.0083/day	An observed value in fish (Schnoor, 1981).

The observed loss rate for birds (a geometric mean of two values of 0.012/day) and fish (0.0083/day) was applied to the pathways wherever avian or fish food terms were required for calculations. Other values can be derived from regression equations (Spacie and Hamelink, 1982). As the k_2 value ultimately has a large influence on the BAF, the most conservative BAF is obtained by use of smaller k_2 values. However, observed values were used because they were assumed to represent behavior of dieldrin residues in tissue better than values derived from regression equations.

Pathway Analysis

The aquatic Pathway Analysis for dieldrin uses BCF, k_2, and f_2 values in the Level #1 through Level #4 equations to derive a BAF for each food chain. When BAFs for each food chain have been calculated, they are summed to give a biomagnification factor (BMF) for the food web. The food chain BAF calculations are presented in the following sections.

Pathway One: H_2O -> Snails -> Mallard -> Bald Eagle--The BCF for snails is calculated using equation (1) and observed concentrations for dieldrin in snails at RMA (Table 5.2-4):

$$BCF_{snails} = C_b/C_w = 4{,}600 \qquad (1)$$

BCFs for snails and other mollusks (Gastropoda) have been recorded in the literature from 2,000 to 115,000 (Petrocelli et al., 1973; Brown, 1978). The RMA value falls within this range.

The food term (f_2) is calculated by assuming that an adult mallard weighs approximately 1,100 g and consumes about 57.4 g total diet each day (Miller, 1975), of which for a breeding female, 16.4 percent of the diet is snails (Swanson et al., 1985). The BAF for a mallard is calculated by assuming that the first term in the Level #2 bioaccumulation equation (2) equals zero, because the amount of bioconcentration by nonaquatic organisms is assumed to be negligible:

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2 BCF_{snail}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (57.4\ g/1{,}100\ g\ bw/day) \times 14\%}{0.012/day} = 0.55 \qquad (5)$$

When the BCF for snails is 4,600, the BAF for mallard is 2,500.

Available data did not indicate Class I mallard ducklings consumed large quantities of snails (Chura, 1961).

An adult eagle weighs approximately 4,500 g (Schafer, 1986) and consumes 255 g daily (Swies, 1986), of which 24 percent of the diet is birds (Cash et al., 1985; Sherrod, 1978). Energy requirements are different for wild birds than birds living in captivity, so these dietary quantities are only approximate (Sherrod, 1986). The following BAF values for an eagle are

calculated by assuming that the first two terms in the Level #3 bioaccumulation equation (3) equal zero (bioconcentration by the eagle, BCF_3, and by the mallard, BCF_2, are negligible):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$
$$BAF_{eagle} = f_3f_2BCF_{snail}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 24\%}{0.012/day} = 1.02 \quad (5)$$

When the BCF for snails is 4,600, BAF for the eagle is 2,600.

Pathway Two: H_2O => Chironomid => Mallard => Bald Eagle--Chironomids were analyzed separately from other invertebrates because their observed tissue concentration was higher by nearly a factor of 10. The BCF for dieldrin in chironomids is calculated using equation (1) and observed concentrations of dieldrin in chironomids (Table 5.2-4):

$$BCF_{chiron} = C_b/C_w = 13{,}000 \quad (1)$$

To calculate the BAF for a mallard, the food term (f_2) remains the same as in Pathway One, except for the percent of food item in the diet. The BAF for a mallard is calculated using the Level #2 bioaccumulation equation (2):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$
$$BAF_{mallard} = f_2BCF_{chiron}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (57.4\ g/1{,}100\ g\ bw/day) \times 1\%}{0.012/day} = 0.039 \quad (5)$$

When the BCF for chironomids is 13,000, the BAF for the mallard is 510.

The R value for ducklings is a geometric mean derived from food consumption data for control ducklings at 1 to 3 weeks of age, or 0.26 g/g bw/day at 7

percent water content (Heinz, 1975). Since natural foods contain approximately 80.5 percent water (Meeks, 1968), ducklings must consume 1.24 g/g bw/day on a wet weight basis to derive the same caloric value. The assimilation efficiency was assumed to be the same as that for adult birds; loss rate for birds was developed from data for birds, and a growth dilution effect of 0.29 (Heinz et al., 1988).

The BAF for duckling is calculated from equations (2) and (5) as follows:

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$
$$BAF_{duckling} = f_2BCF_{chiron}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (1.24\ g/g\ bw/day) \times 52.6\%}{0.302/day} = 1.94 \qquad (5)$$

When the BCF for chironmids is 13,000, the BAF for duckling is 25,000.

To calculate the BAF for an eagle, the food term (f_3) remains the same as in Pathway One:

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$
$$BAF_{eagle} = f_3f_2BCF_{chiron}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 24\%}{0.012/day} = 1.02 \qquad (5)$$

When the BCF for chironomids is 13,000, the BAF for the eagle is 520.

Pathway Three: H_2O => Other Invertebrates => Mallard => Bald Eagle--The BCF for dieldrin in aquatic invertebrates other than snails or chironomids is a geometric mean calculated using equation (1) and observed concentrations of dieldrin in invertebrates other than snails or chironomids (Table 5.2-4):

$$BCF_{invert} = C_b/C_w = 920 \qquad (1)$$

The BCF for aquatic invertebrates exposed in a field situation for a period of 6 months was 4,620 (EPA, 1980); other studies document values as high as 17,000 (Wallace and Brady, 1971) under similar conditions.

To calculate the BAF for a mallard, the food term (f_2) remains the same except for the percent of food item in the diet. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{mallard} = f_2BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (57.4\ g/1{,}100\ g\ bw/day) \times 29\%}{0.012/day} = 1.13 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for mallard is 1,000.

The BAF for duckling is calculated from equations (2) and (5) as follows:

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{duckling} = f_2BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (1.24\ g/g\ bw/day) \times 34.6\%}{0.302/day} = 1.28 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for duckling is 1,200.

To calculate the BAF for an eagle in Pathway Three, the food term (f_3) remains the same. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{invert}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 24\%}{0.012/day} = 1.02 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for eagle is 1,100.

Pathway Four: H_2O -> Aquatic Plants -> Mallard -> Bald Eagle--The BCF for plants in Pathway Four is based on Rosenlund et al.'s (1986) data for dieldrin concentrations in aquatic plants (Table 5.2-4):

$$BCF_{plant} = C_b/C_w = 2{,}200 \qquad (1)$$

Algae have been observed in other studies to concentrate dieldrin by factors of 128 to 5,558 (EPA, 1980a); RMA data for aquatic macrophytes fall within this range.

To calculate the BAF for a mallard, the food term f_2 remains the same except for the percent of the food item in the diet. Plants (including fruits) form 30 to 31 percent of the mallard's diet. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2 BCF_{plant}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (57.4\ g/1{,}100\ g\ bw/day) \times 30\%}{0.012/day} = 1.17 \qquad (5)$$

When the BCF for aquatic plants is 2,200, the BAF for mallard is 2,600. The BAF for duckling is calculated from equations (2) and (5) as follows:

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{duckling} = f_2 BCF_{plant}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.9 \times (1.24\ g/g\ bw/day) \times 12.8\%}{0.302/day} = 0.473 \qquad (5)$$

When the BCF for aquatic plants is 2,200, the BAF for duckling is 1,000.

To calculate the BAF for an eagle in Pathway Four, the food term (f_3) remains the same. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{plant}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 24\%}{0.012/day} = 1.02 \quad (5)$$

When the BCF for aquatic plants is 2,200, the BAF for eagle is 2,600.

Pathway Five: H_2O => Plankton => Bluegill => Pike => Bald Eagle--Pathways leading to the bald eagle via fish are more complex because bioconcentration occurs at each trophic level, not just at the lowest level. This introduces a fourth factor into the BAF equation, and the eagle is at Level #4 instead of Level #3. The BCF for plankton, calculated using equation (1) and the observed concentrations of dieldrin in plankton at RMA (Table 5.2-4), exceeds the range of 128 to 5,558 observed for algae (EPA, 1980a):

$$BCF_{plankton} = C_b/C_w = 11{,}000 \quad (1)$$

The BCF for the bluegill (*Lepomis macrochirus*) (5,800) was derived from flow-through tests for freshwater fish (Kenaga, 1980). Larger BCF values for fish have been observed (EPA, 1980a), but the tests were static (flow-through tests are generally recommended for chronic exposures (ASTM, 1984)) and water concentrations of dieldrin were not always maintained at a constant level.

If a bluegill consumes 3 percent of its body weight daily (Chadwick and Brocksen, 1969), the total daily intake term (R) would be 0.03 regardless of actual body weight. Various algal forms account for approximately 12 percent of the bluegills diet (Martin *et al.*, 1961): this value was used for the percent of plankton in the bluegill diet. The k_2 values used for the bluegill and pike food terms are based on Schnoor's (1981) observation for fish, where k_2 equals 0.0083/day. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{plankton}$$

$$\text{where: } f_2 = \frac{0.9 \times (0.03/day) \times 12\%}{0.0083/day} = 0.39 \quad (5)$$

When the BCF for plankton is 11,000, the BAF for bluegill is 10,000.

The BCF for the pike was also assumed to be 5,800 (Kenaga, 1980), although the actual BCF may differ.

The pike is also estimated to consume 3 percent of its body weight daily (Chadwick and Brocksen, 1969), such that the total daily intake term (R) would be 0.03 regardless of actual body weight. It is assumed that pikes feed entirely on bluegills for the sake of this analysis. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{plankton}$$

$$\text{where: } f_3 = \frac{0.9 \times (0.03/day) \times 100\%}{0.0083/day} = 3.3 \quad (5)$$

When the BCF for plankton is 11,000, the BAF for pike is 39,000.
The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. The k_2 of 0.02/day for birds is used to calculate the f_4. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{plankton}$$

$$\text{where: } BCF_4 = 0$$

$$f_4 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 66\%}{0.012/day} = 2.8 \quad (5)$$

When the BCF for plankton is 11,000, the BAF for eagle is 110,000.

Pathway Six: H_2O => Invertebrates => Bluegill => Pike => Bald Eagle--The BCF for invertebrates is calculated using equation (1) and observed concentrations of dieldrin in invertebrates other than chironomids (Table 5.2-4):

$$BCF_{invert} = C_b/C_w = 920 \quad (1)$$

The BCF for the bluegill (Lepomis macrochirus) is the same as Pathway Five (5,800). The food term differs from Pathway Five only in the percent of the food item in the bluegill diet. A bluegill diet consists of 88 percent total invertebrates; half of these are designated as general invertebrates and the other half are designated as chironomids (Pathway Seven). Specific food habits are an estimate only; bluegill diets would vary seasonally with fluctuations in invertebrate populations. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{invert}$$

$$\text{where: } f_2 = \frac{0.9 \times (0.03/day) \times 44\%}{0.0083/day} = 1.4 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for bluegill is 7,100.

The BCF for the pike was also assumed to be 5,800 (Kenaga, 1980), although the actual BCF may differ. The food term (f_3) for the pike remains the same as Pathway Five. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{invert}$$

$$\text{where: } f_3 = \frac{0.9 \times (0.03/day) \times 100\%}{0.0083/day} = 3.3 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for pike is 29,000.

The food term (f_4) for the eagle also remains the same as Pathway Five. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{invert}$$

$$\text{where: } f_4 = \frac{0.9 \times (255\ g/4{,}500\ g\ bw/day) \times 66\%}{0.012/day} = 2.8 \quad (5)$$

When the BCF for aquatic invertebrates is 920, the BAF for eagle is 82,000.

Pathway Seven: H_2O => Chironomids => Bluegill => Pike => Bald Eagle--The BCF for chironomids is a geometric mean calculated using equation (1) and observed concentrations of dieldrin in chironomids (Table 5.2-4):

$$BCF_{chiron} = C_b/C_w = 13{,}000 \quad (1)$$

The BCF for the bluegill (Lepomis macrochirus) is the same as the previous two pathways (5,800). The food term is the same as Pathway Six, as chironomids are assumed to make up the same percentage as other invertebrates (44 percent) of the bluegill diet. This is a conservative assumption because food habits are an estimate only; actual diet and therefore residue accumulation would vary seasonally with fluctuations in invertebrate populations. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{chiron}$$

$$\text{where: } f_2 = \frac{0.9 \times (0.03/day) \times 44\%}{0.0083/day} = 1.4 \quad (5)$$

When the BCF for chironomids is 13,000, the BAF for bluegill is 24,000.

The BCF for the pike was also assumed to be 5,800 (Kenaga, 1980), although the actual BCF may differ. The food term (f_3) for the pike remains the same as Pathways Five and Six. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{chiron}$$

$$\text{where: } f_3 = \frac{0.9 \times (0.03/day) \times 100\%}{0.0083/day} = 3.3 \quad (5)$$

When the BCF for chironomids is 13,000, the BAF for pike is 85,000.

The eagle food term (f_4) also remains the same as Pathways Five and Six. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{chiron}$$

$$\text{where: } f_4 = \frac{0.9 \times (255\ g/4{,}500\ g/day) \times 66\%}{0.012/day} = 2.8 \quad (5)$$

When the BCF for chironomids is 13,000, the BAF for eagle is 240,000.

Results and Discussion

Biomagnification is the result of bioconcentration and bioaccumulation by which tissue concentrations of chemicals increase as the chemical is transferred up food chains (Rand and Petrocelli, 1985). The term implies systematic transfer between trophic levels and can be used to predict interrelationships between the abiotic environment and selected target species.

BAF values as derived for the individual pathways (Table 5.2-5) represent accumulation in separate single food chains. To derive overall accumulation in the entire food web, variations of the following equation are used:

$$BMF_i = BCF_i + f_iBAF_{i-1}$$

The BAFs cannot be directly summed, because BCF for the higher trophic level organisms would be factored in with every pathway. Total magnification of residues for each of the key organisms in the aquatic Pathway Analysis is presented in Table 5.2-6.

Table 5.2-5. Summary of Bioaccumulation or Biomagnification Factors for Each Species in the Pathways Analysis for Dieldrin

	Bluegill	Pike	Duck	Duckling	Mammal*	Eagle
Pathway 1	--	--	2,500	--	--	2,600
Pathway 2	--	--	510	25,000	--	520
Pathway 3	--	--	1,000	1,200	--	1,100
Pathway 4	--	--	2,600	1,000	--	2,600
Pathway 5	10,000	39,000	--	--	--	110,000
Pathway 6	7,100	29,000	--	--	--	82,000
Pathway 7	24,000	85,000	--	--	--	240,000
Pathway 8	--	--	--	--	4.3	2.0

* See Section 5.2.2.5

Source: ESE, 1988.

Table 5.2-6. Total Biomagnification of Dieldrin Residues for Each of the Key Organisms in the Aquatic Pathways Analysis.

Organism	Level	Equation	BMF
Mallard	#2	$\Sigma f_2 BCF_1$	6,600
Duckling	#2	$\Sigma f_2 BCF_1$	27,000
Bluegill	#2	$BCF_2 + \Sigma f_2 BCF_1$	30,000
Pike	#3	$BCF_3 + f_3(BMF_{bluegill})$	100,000
Eagle	#3, #4	$f_4(BMF_{pike}) + f_3(BMF_{duck}) + BMF_{terrestrial}$*	290,000

* See Section 5.2.2.5.

Source: ESE, 1988.

Total BMF can be used to determine maximum allowable levels of dieldrin in sediments by relating sediment concentration to the Maximum Acceptable Tissue Concentration (MATC) as follows (Tucker, 1986):

$$\frac{\text{MATC}}{\text{Total BMF}} = C_w \qquad (6)$$

and,

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \qquad (8)$$

The MATC is based on the lowest observed effect level obtained from the scientific literature for a species similar to the target organism, and assumes that criteria derived for the protection of the target organism (bald eagle) will protect other species as well. No safety factors have been used in the calculation of MATCs. The MATC for the bald eagle obtained by examining the literature for the lowest tissue concentration correlating with toxic effects:

SPECIES	ORGAN	PPM	EFFECT	REFERENCE
Mallard	brain	0.125	Decrease NE, DOPA, serotonin	Sharma et al., 1976
Bald Eagle	brain	3.6	Decreased body fat, Death	Prouty et al., 1977
Bald Eagle	brain	5.0	Death	Barbehenn and Reichel, 1981
Grouse	assorted	0.6	Decreased activity	McEwen and Brown, 1966
Cowbird	brain	1.0	Adverse effects	Heinz and Johnsen, 1981

The lowest tissue concentration at which toxic effects have been observed is divided by the largest BMF (the BMF for eagle) from Table 5.2-6, then corrected with K_{oc} and f_{oc} to give the sediment concentration at which "no effects" are likely to occur. The lowest observed level at which adverse effects occurred was 0.125 ppm in mallard brain tissue. On a lipid weight basis, brain levels of dieldrin tend to be 5.1 times lower than carcass levels (Barbehenn and Reichel, 1981); most data for birds; however, are presented as carcass residue on a lipid weight basis compared to brain residue on a wet weight basis (Barbehenn and Reichel, 1981; Wiemeyer and

Cromartie, 1981), resulting in a carcass to brain ratio close to 80. Since the Sharma et al. (1976) data for brain were expressed on a wet weight basis, a carcass to brain ratio of 80 was used to obtain the MATC of 10 ppm for carcass. Although the MATC was developed using data from mallard brains, it represents the most conservative estimate of sublethal effects levels in avian species, and is therefore used to represent sublethal effects levels in both mallard and bald eagle.

Using equations (6) and (8):

$$\frac{\text{MATC}}{\text{Total BMF}} = C_w = \frac{10\ \text{ppm}}{290{,}000} = 3.4 \times 10^{-5}\ \text{ppm} \qquad (6)$$

$$C_{sed} = C_w \times K_{oc} \times f_{oc} = 3.4 \times 10^{-5}\ \text{ppm} \times 24{,}400 \times 0.0065 \qquad (8)$$
$$= 0.0055\ \text{ppm}$$

The water and sediment concentrations derived from the bald eagle Pathway Analysis are protective of other wildlife populations as well. For instance, fish die with brain concentrations of dieldrin of 10.31 ppm, and blood concentrations of 5.65 ppm (EPA, 1980a); sublethal effects data were unavailable. If an uncertainty factor of 10^2 is applied, a "no effects" brain concentration for fish of 0.103 ppm is estimated. As this is in the range of the avian LOAEL for brain (0.125 ppm), and total BMF for fish is about half that for eagles, it is assumed that "no effects" water concentrations for bioaccumulation in eagles will be protective of toxicity due to residue concentration in fish as well.

Waterfowl as well as fish are protected from residue accumulation in food chains by acceptable levels estimated with the bald eagle Pathway Analysis. Mallard adults and ducklings have lower total BMFs than eagle (Table 5.2-6), while the MATC would remain the same. Therefore, acceptable water and sediment concentrations would be higher for waterfowl than for eagle.

The water and sediment criteria (0.034 ppb and 0.0055 ppm, respectively) derived from the MATC for birds (10 ppm) and the BMF for eagle (290,000) represent criteria for food web transfer of residues for all wildlife populations.

Protecting high trophic level species will satisfy "no effects" levels in water for surface water consumption by wildlife species (8 ppb), or exposure by aquatic life (0.05 ppb). Therefore, cleanup levels in water and sediments should be based on criteria derived from the bald eagle Pathway Analysis. The EPA chronic criteria for the protection of aquatic organisms and their uses (0.0019 ppb) are based on a Final Residue Value with human guidelines as the MATC, and are therefore not considered applicable to this analysis.

5.2.1.5 Terrestrial Pathway Analysis

Introduction to Terrestrial Pathway Analysis

This analysis was performed to determine cleanup criteria for aldrin and dieldrin in a terrestrial based food web (soil-biota) pathway. There are no aquatic components in this food web as compared to the previous food web where most of the food chains were aquatic based. The method is based on estimates of exposure by various organisms to contaminants in the physical environment and the potential for bioaccumulation (concentration from diet and water) and biomagnification (systematic increase in tissue concentrations of chemicals as they pass to higher trophic levels) exhibited by aldrin and dieldrin.

The approach used for the terrestrial pathway analysis arrives at a "no effects" level in soil of terrestrial ecosystems on RMA by assuming that: (1) soils are a source of aldrin and dieldrin contamination, (2) aldrin and dieldrin enter the food web from soils, and (3) aldrin and dieldrin become concentrated in higher trophic levels by the mechanisms of bioaccumulation and biomagnification.

Data used to estimate BMF values for biota in a terrestrial ecosystem were obtained from either RMA data or available literature on aldrin and dieldrin. In some cases, data used in the pathway analysis were from studies where chemical analysis did not distinguish between aldrin or dieldrin, or exposure was initially to aldrin but residues were expressed as either chemical. In these instances, residues were assumed to be dieldrin. Model ecosystem studies indicate that the behavior of the two chemicals is very similar (Metcalf et al., 1973).

The behavior and fate of dieldrin in a terrestrial ecosystem was used to represent both aldrin and dieldrin because dieldrin has a widespread distribution on RMA and is persistent in the environment. Aldrin was not selected for pathway analysis because it converts rapidly to dieldrin in the environment and *in vivo* (Hall *et al.*, 1971; Metcalf *et al.*, 1973).

Methods for Terrestrial Pathway Analysis--Bald Eagle Food Web

The single food chain (Pathway Eight) of the bald eagle food web originating in soil is evaluated differently than food pathways originating in an aquatic environment. Since bioconcentration is not occurring as in the aquatic pathways, Thomann's (1981) model is not applied. Instead, bioaccumulation is estimated in the food chain by comparing C_b to C_{soil} to obtain uptake in relation to soil, and by comparing C_b directly to C_{diet} to obtain uptake relative to the next lower trophic level. By comparing C_b directly to C_{diet}, factors such as assimilation efficiency and loss rate are adjusted for in the BMF. This was necessary because data regarding terrestrial systems are not as extensive as those regarding aquatic systems.

Pathway Eight: Soil -> Terrestrial Plants -> Small Mammals -> Bald Eagle--

Terrestrial plants accumulate dieldrin residues from soil by factors of approximately 0.5 (Davidson, 1986). Data used to derive the BAF values for the terrestrial pathway are presented in Table 5.2-7.

In a study by Garten and Trabalka (1983) small mammals were observed to accumulate dieldrin residues from their diet by factors of 4.3 (tissue analyzed was not specified, and variability in the data was not presented).

The fraction of dieldrin ingested by eagles is related to the amount of small mammals in their diet. The portion of dieldrin that an eagle could obtain from the terrestrial mammal part of the food chain can be estimated from data given for barn owls (Mendenhall *et al.*, 1983) and kestrels (Wiemeyer *et al.*, 1986) by assuming that (1) concentration from diet under laboratory conditions will resemble concentration under natural conditions, and (2) concentration from diet by eagles will resemble that of other raptors.

Table 5.2-7. Bioaccumulation Factors Used in the Terrestrial Pathway Component of the Aquatic Pathways Analysis for Dieldrin

Organism/Group	BAF	Mean BAF	Sources
Terrestrial Plant	0.5	NA	Davidson, 1986
Small Mammal	4.3	NA	Garten and Trabalka, 1983
Eagle			
(Barn Owl)	16.6		
	15.9		Mendenhall et al., 1983
(Kestrel	5.0		
	5.1		Wiemeyer et al., 1986
Geometric Mean		9.1	

NA = Not Available

Source: ESE, 1988.

Barn owls (12 males and 7 females) concentrated dieldrin from a 0.58 ppm diet by factors of 16.6 for males and 15.9 for females (Mendenhall et al., 1983). Geometric mean tissue concentrations were 9.6 (± 1.48 SD) and 9.2 (± 1.32 SD) ppm, respectively, for male and female barn owls. Data for kestrels that died on a 0.28 or 0.84 ppm diet (wet weight basis) indicate a range of concentration factors from 1.6 to 17.9 (Weimeyer et al., 1986). A geometric mean of 9.1, derived from the barn own and kestrel data, was used to represent concentration from diet by bald eagle.

The terrestrial part of the bald eagle food web is as follows:

$$\text{soil} \xrightarrow{0.5} \text{plants} \xrightarrow{4.3} \text{mammals} \xrightarrow{9.1} \text{eagles}$$

Total magnification in terrestrial ecosystems is 20 times greater than the soil; when corrected for the percent in the eagles diet (10 percent) the total BMF for the terrestrial pathway becomes 2.0.

Results and Discussion

The terrestrial pathway, Pathway Eight, assumes greater significance based on observed winter feeding behavior of eagles at RMA, where eagles apparently subsist primarily on small mammals pirated from other raptors. Observations indicate that approximately 90 percent of the eagle diet is made up of small mammals; the "no effects" level in soils is then based on 90 percent of the diet represented by Pathway Eight. The total BMF is equal to 90 percent of the BMF estimated by Pathway Eight, or 18. Using equation (6):

$$\frac{\text{MATC}}{\text{Total BMF}} = \frac{10\ \text{ppm}}{18} = 0.56\ \text{ppm} \qquad (6)$$

The soil criterion derived from Pathway Eight can also be used to predict toxicity to small mammals exposed to contaminants from ingesting contaminated soil. An exposure rate as a function of the acceptable soil criteria can be estimated from the soil criterion and the soil ingestion rate for small mammals as follows:

Soil Criterion x Soil Ingestion Rate = Daily Exposure

0.56 mg/kg soil x 0.000873 kg soil/kg bw/day = 0.00049 mg/kg bw/day

The exposure rate based on a soil criterion of 0.56 mg/kg soil is five to six orders of magnitude lower than observed LD_{50}s for small mammals, and therefore direct toxic effects are not expected at the criterion level of 0.56 mg/kg in soil. The daily intake of dieldrin from ingesting soil represents a conservative estimate as an assimilation efficiency of 100 percent is assumed.

Aldrin and dieldrin apparently accumulate to a significant extent in terrestrial systems (BMF in the single terrestrial food chain to bald eagle greater than 1.0), as opposed to other contaminants such as arsenic (BMF less than 1.0). BMF values less than 1 indicate that residues are probably not concentrating in terrestrial food chains. Since total potential residue accumulation in a terrestrial food chain leading to bald eagle was 18 (assuming 90 percent dietary intake from terrestrial sources), a separate pathway analysis for kestrel food web was constructed for aldrin and dieldrin.

Method for Terrestrial Pathway Analysis -- Kestrel Food Web

This Pathway Analysis is based on the American kestrel (*Falco sparverius*) sink food subweb (portion of the comprehensive ecosystem food web leading to a target species) and includes the major food chains leading to the selected sink species (Cohen, 1978). Percentage contributions for each group of organisms in the kestrel's diet have been estimated based on existing literature (Table 5.2-8). The subweb has been simplified for the purposes of the analysis (e.g. small birds include all species that the kestrel is assumed to feed on: grasshoppers represent all insects).

The American kestrel was selected as the target species because: (1) it represents a relatively high trophic level in the terrestrial food web, (2) it is common at RMA, (3) its diet includes species which are known to be contaminated, and (4) previous studies on RMA indicate that this species has possibly been affected by pesticide contamination on RMA (DeWeese *et al.*, 1986). The "no effects" level for soil derived from the pathway analysis is based on sublethal effects levels obtained from the literature and assumes that if kestrels are not affected, other species will be protected.

Table 5.2-8. Summary of Feeding Habits for all Organisms in the Terrestrial Pathways Analysis for Dieldrin

Species	Food Items	Percent in Diet	Sources
Kestrel	Insects	51.8	Sherrod, 1978
	Small Birds	16.4	Sherrod, 1978
	Reptiles	4.5	Sherrod, 1978
	Mammals	27.3	Sherrod, 1978
Mammals	Insects	50	Jones et al., 1985;
	Plants	50	Hall, 1981
Small Birds	Plants	50	Assumed
	Insects	25	
	Earthworms	25	
Reptiles/ Amphibians	Insects	100	Assumed
Insects	Plants	100	Assumed

Source: ESE, 1988.

Seven food transfer pathways originating in soil and ultimately terminating with the kestrel were established as follows:

1. Soil -> Plants -> Insects -> Kestrels
2. Soil -> Plants -> Birds -> Kestrels
3. Soil -> Earthworms -> Birds -> Kestrels
4. Soil -> Plants -> Insects -> Birds -> Kestrels
5. Soil -> Plants -> Insects -> Reptiles -> Kestrels
6. Soil -> Plants -> Insects -> Mammals -> Kestrels
7. Soil -> Plants -> Mammals -> Kestrels

The combined food transfer pathways are presented in Figure 5.2-3. BAF values (obtained from RMA data or published sources) are presented in Table 5.2-9.

The information required to perform the terrestrial Pathway Analysis includes health effects levels, food habits for species at each trophic level, and BMF values for species at each trophic level. The analysis was performed by using BMFs for each trophic level in a food chain, and then weighting the importance of each food chain in a food web by utilizing food habits data. The end result is a total estimated BMF for kestrel that can be used to estimate safe soil levels of dieldrin.

Pathway Analysis

The kestrel BAF (9.1) is a geometric mean value estimated from data from studies on two raptors, the kestrel and the barn owl (*Tyto alba*). In a study where kestrels were dosed with dieldrin in combination with DDT, kestrels that died concentrated dieldrin approximately 1.6 to 17.9 times from diet (Wiemeyer, 1986). At treatment levels of 0.28 ppm (wet weight basis), geometric mean accumulation from diet was 5.0; at 0.84 ppm (wet weight basis) mean accumulation from diet was 5.1. In a study with barn owls, geometric mean carcass residues of dieldrin were 15.9 to 16.6 times greater than dietary levels of 0.58 ppm (Mendenhall *et al.*, 1983). The four means were averaged to obtain an overall geometric mean for bioaccumulation.

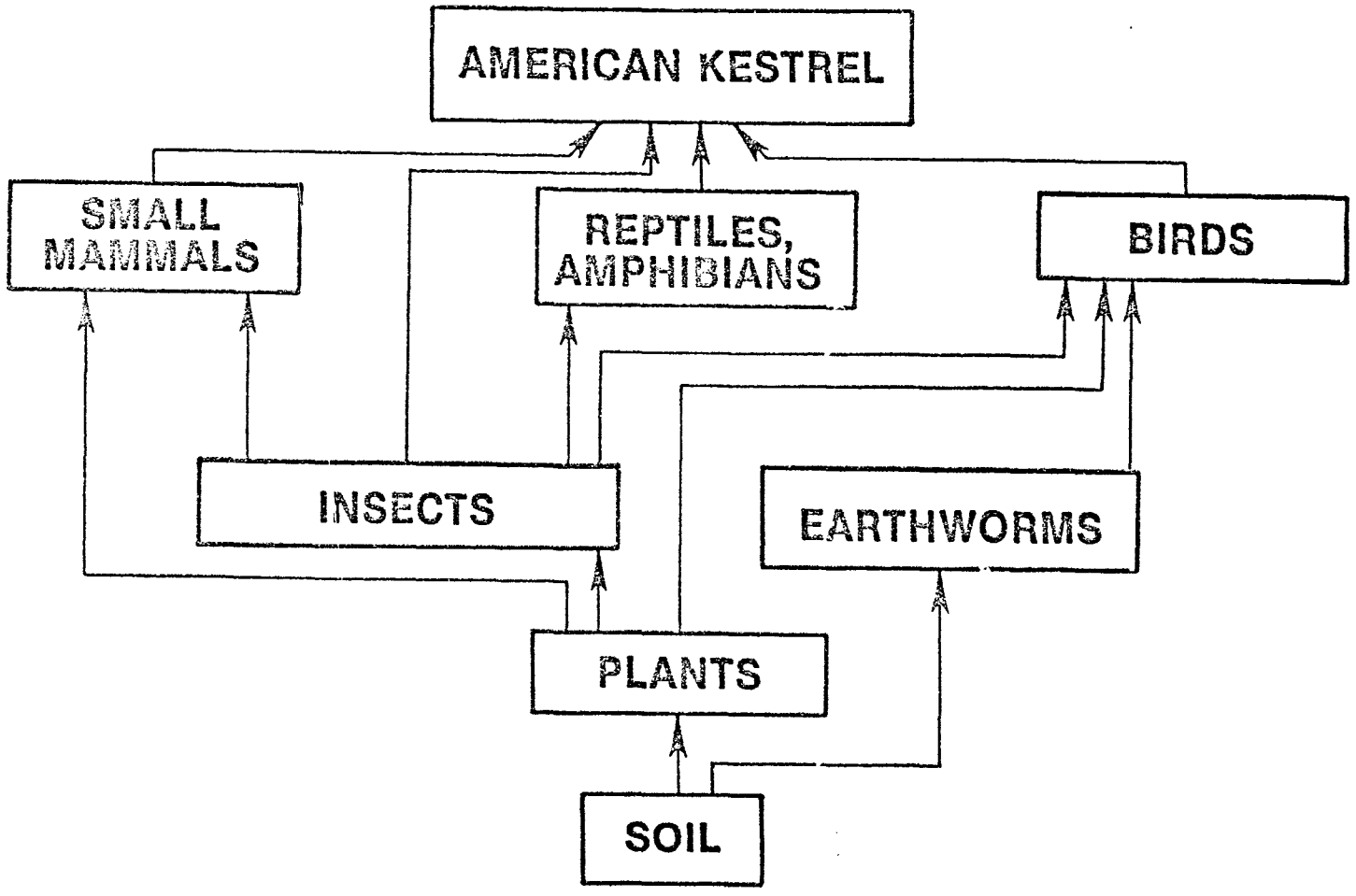


Figure 5.2-3

KESTREL SINK FOOD WEB FOR DIELDRIN

SOURCE: ESE, 1989

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

Table 5.2-9. Bioaccumulation Factors Used in the Pathway Analysis for Dieldrin

Species	BAF/BMF	Mean BAF*	Sources
Kestrel	5.0		Wiemeyer, 1986
	5.1		Wiemeyer, 1986
	16.6		Mendenhall et al.,
	15.9	9.0	Mendenhall et al.,
Mammals	4.3	NA	Garten and Trabalka, 1983
Reptiles	1.1		Korschgen, 1970
	4.4	2.8	
Small Birds	8.2		Jefferies and Davis, 1968
	9.0		Robinson et al., 1967
	15.2		Davison et al., 1971
	8.9		Garten and Trabalka, 1983
	10.7	10	Garten and Trabalka, 1983
Insects	1.83		Thorne et al., 1979
	7.86		Thorne et al., 1979
	7.71		Thorne et al., 1979
	76.9		Thorne et al., 1979
	25.0		Thorne et al., 1979
	4.38	9.9	Thorne et al., 1979
Earthworms	3.56		Korschgen, 1970
	3.96		Korschgen, 1970
	4.16		Korschgen, 1970
	7.40		Korschgen, 1970
	3.48		Korschgen, 1970
	3.88		Korschgen, 1970
	12.4		Korschgen, 1970
	5.64	5.0	Korschgen, 1970
Plants	0.5	NA	Davidson, 1986

* Geometric Mean
NA = Not Available

Source: ESE, 1988.

In a study by Garten and Trabalka (1983) small mammals were observed to accumulate dieldrin residues from their diet by factors of 4.3 (tissue analyzed was not specified).

Both the deer mouse (*Peromyscus maniculatus*) and the grasshopper mouse (*Onychomys leucogaster*) are found at RMA. Mice were assumed to subsist on a diet of insects (as represented by grasshoppers) and plant material. When food habits of the two species were examined, their combined diets were estimated to contain approximately 50 percent plant material and 50 percent animal material (Jones *et al.*, 1985; Hall, 1981).

The BAF for amphibians and for reptiles (2.8) is a geometric mean value based on field data collected by Korschgen (1970). The data indicate that toads, (*Bufo americanus*), concentrate dieldrin 1.1 times from a diet consisting primarily of insects and other invertebrates (concentration in toad/average concentration in prey items), while garter snakes (*Thamnophis sirtalis*) concentrate dieldrin 4.4 times from a diet that includes both invertebrate and vertebrate species (Korschgen, 1970). Kestrels prey on all types of small reptiles and amphibians including small snakes, lizards, and toads: it was assumed for the purposes of the analysis that small reptiles and amphibians (prey size suitable for kestrels) would feed predominantly on insects.

The BAF for small birds (10) is a geometric mean value derived from several studies. Since the dietary habits of this group vary widely, food habits were approximated by assuming that the dietary proportions of plants, insects, and earthworms were 50, 25, and 25 percent, respectively. Chickens have been observed to accumulate dieldrin 14 to 17.5 times from diet, with a geometric mean of 15.2 (Davison *et al.*, 1971) and pigeons accumulate dieldrin by factors of 9 (Robinson *et al.*, 1967). Garten and Trabalka (1983) indicate that small birds bioaccumulate dieldrin 8.9 times, while chickens accumulate by factor of 10.7. Songthrushes (*Turdus ericetorum*) were observed to concentrate dieldrin from a diet consisting of contaminated earthworms by factors of 4 to 12 (Jefferies and Davis, 1968). A geometric

mean was calculated for each avian species in a study where data were sufficient; mean BAFs and single estimate BAFs were combined to obtain an overall geometric mean BAF value for small birds.

The BAF for insects (9.9) is a mean value based on data obtained for grasshoppers at RMA. Grasshoppers form an average of 44 percent of the kestrels diet, and other invertebrates such as crickets and beetles are utilized as well (Sherrod, 1978). The BAF was calculated by dividing the median concentration observed in grasshoppers at RMA by the median concentration observed in plants at RMA (Thorne et al., 1979, RIC#81286R06). Grasshoppers were assumed to feed entirely on plant tissue for the purposes of the analysis. It is possible that using the grasshopper BAF to represent concentration by all insects underestimates the actual residue magnification along food chains containing carnivorous or omnivorous insects. Data indicate low magnification factors for crickets (Korschgen, 1970). However, RMA data were considered preferable and less uncertain than data from other sources. When data were presented for both annual and perennial foliage, the two points were averaged before calculating the BAF. Only data for which both plant and insect residues were available at a sampling location were used in calculating the BAF.

The EMF for earthworms (Lumbricus spp.) (5.0) is a mean value based on both terrestrial microcosm studies and field data. Earthworms have been observed to concentrate residues under field conditions to a level 4 to 12 times greater than soils that contained an average residue content of 0.25 ppm over a 3 year period (Korschgen, 1971).

The EMF for plants (0.5) is a general value intended to represent a wide variety of plant species, and is based on findings by Davidson (1986).

For each pathway, the magnification of residues from soil to kestrel was calculated by multiplying the BMF values for each trophic level (Table 5.2-10). The BMF values must then be adjusted for the proportion of the lower trophic levels in the diet of the higher trophic levels. The kestrel, for example, consumes 16.4 percent small birds, and the BMF for the pathways to the kestrel through birds (Pathways 2, 3, and 4) must be adjusted

Table 5.2-10. Biomagnification Factors for each Food Chain in the Pathway Analysis for Dieldrin*

Pathway	BMF
1. Soil -> Plants -> Insects -> Kestrels (0.5, 9.9, 9.1)	45
2. Soil -> Plants -> Birds -> Kestrels (0.5, 10, 9.1)	46
3. Soil -> Earthworms -> Birds -> Kestrels (5.0, 10, 9.1)	460
4. Soil -> Plants -> Insects -> Birds -> Kestrels (0.5, 9.9, 10, 9.1)	450
5. Soil -> Plants -> Insects -> Reptiles -> Kestrels (0.5, 9.9, 2.8, 9.1)	130
6. Soil -> Plants -> Insects -> Mammals -> Kestrels (0.5, 9.9, 4.3, 9.1)	190
7. Soil -> Plants -> Mammals -> Kestrels (0.5, 4.3, 9.1)	20

* Based on Bioaccumulation Factors for Each Trophic Level.

Source: ESE, 1988.

accordingly. In addition, birds feed on several food items, so the BMF must be adjusted to represent the importance of these items in the small bird diet (Table 5.2-11). The pathways leading to the kestrel via mammals (Pathways Six and Seven) must be adjusted in a similar manner. Because reptiles/amphibians and insects were assumed to have only one food source, Pathways One and Five need only be weighted for kestrel food habits.

Results and Discussion

The final value for the BMF represents biomagnification of dieldrin residues from soil through several trophic levels to the kestrel, and is much lower than the magnification of residues in the aquatic food web. This is expected because, in general, the processes of bioconcentration outweigh accumulation of residues as a result of biomagnification.

The MATC is obtained by examining the literature for the lowest concentration which results in toxic effects:

SPECIES	ORGAN	PPM	EFFECT	REFERENCE
Mallard	brain	0.125	Decrease NE, DOPA, serotonin	Sharma et al., 1976
Bald Eagle	brain	3.6	Decreased body fat, Death	Prouty et al., 1977
Bald Eagle	brain	5	Death	Barbehenn and Reichel, 1981
Grouse	assorted	0.6	Decreased activity	McEwen and Brown, 1966
Cowbirds	brain	1.0	Adverse effects	Heinz and Johnsen, 1981

The lowest tissue concentration at which toxic effects have been observed is divided by the total adjusted BMF from Table 5.2-11 to arrive at a "no effects" soil level. The lowest observed level at which adverse effects occurred was 0.125 ppm in mallard brain tissue. On a lipid weight basis, brain levels tend to be 5.1 times less than carcass levels (Barbehenn and Reichel, 1981); most data for birds; however, is presented as carcass

Table 5.2-11. Biomagnification Factors for Each Pathway Following Adjustment for Dietary Proportions, Dieldrin Pathways Analysis

		Relative Proportion of Pathway in Diet			
Pathway	BMF	Percent Bird	Percent Mammal	Percent Kestrel	Adjusted BMF
1	45	--	--	51.8	23.0
2	46	50	--	16.4	3.8
3	460	25	--	16.4	19.0
4	450	25	--	16.4	18.0
5	130	--	--	4.5	5.9
6	190	--	50	27.3	26.0
7	20	--	50	27.3	2.7
				Total	98.0

Source: ESE, 1988.

residue on a lipid weight basis compared to brain residue on a wet weight basis (Barbehenn and Reichel, 1981; Wiemeyer and Cromartie, 1981), resulting in a carcass to brain ratio close to 80. Since the Sharma et al. (1976) data for brain was expressed on a wet weight basis, a carcass to brain ratio of 80 is used to obtain a maximum permissible tissue concentration or "no effects" level of 10 ppm for carcass.

The MATC for carcass was divided by the total BMF to obtain a probable "no effects" soil concentration as follows:

$$\frac{\text{MATC}}{\text{Total BMF}} = \frac{10\ \text{ppm}}{98} = 0.10\ \text{ppm}$$

At 0.10 ppm in soil, after allowing for concentration up the terrestrial food web, it is likely that no significant adverse effects on the target species will be observed. This "no effects" soil concentration is slightly lower than that derived from the single terrestrial food chain in the bald eagle pathway analysis (0.56 ppm). The kestrel food web thus represents the more conservative soil concentration, and should be used to represent "no effect" soil concentrations for protection of terrestrial species. Because the soil criterion derived from the kestrel pathway analysis is lower than that derived for the single food chain in the bald eagle Pathway Analysis, which was protective of small mammals ingesting soil, the soil criterion from the kestrel pathway analysis is also protective of small mammals ingesting soil.

Although no safety factors have been considered in the analysis, it is likely that this soil concentration will protect all avian predators subsisting on terrestrial organisms because of the broad, general nature of the analysis, i.e. mean values have been used to represent accumulation by whole trophic levels, and seasonal migratory patterns that remove organisms from RMA have not been addressed, thereby assuming a worst case scenario.

5.2.1.6 Uncertainty Analysis

In the uncertainty analysis, all of the intake rates (R values) and percent of items in diet are treated as triangular distributions where the minima and maxima are known and a best estimate within that range has been

determined. Using the triangular distribution as input, the best estimate will be more likely than values near either end of the range. Methodology for the uncertainty analysis is described in detail in the forthcoming Offpost Endangerment Assessment. Diets of each link on the sink food web are summarized in Table 5.2-12.

Several assumptions were made in order to conduct the analysis:

- The diet of the target organism, the bald eagle, is supplied only by the aquatic food chain, with ducks and pike the representative prey organisms; and
- Absorption, or assimilation, of ingested dieldrin is assumed to be 100 percent.

Based on the available data, different uncertainty distributions were developed for depuration rate in birds and fish. Cummings et al., (1967) and Davison and Sell (1978) have observed dieldrin loss in hens and turkeys, respectively. Based on data reported by Cummings et al., (1967), an estimated loss rate of dieldrin in laying hens is 0.0125 day^{-1} based on fatty tissue, and 0.0063 day^{-1} based on breast muscle. The data for fatty tissue and muscle were combined to get an estimate of loss rate for whole bird of 0.009 $\pm$ 0.002 day^{-1}. Davison and Sell (1978) report data from two experiments from which whole bird loss rates can be calculated of 0.027 $\pm$ 0.009 and 0.008 $\pm$ 0.006. The three results for whole body loss rate were equally weighted as each represents a separate experimental estimate of whole body loss. Because of the relatively large uncertainty and the fact that depuration cannot be negative, the uncertainty in this parameter was estimated by a log-normal distribution with a mean of 0.014 and a standard deviation of 0.005.

Depuration in fish appears to be considerably uncertain. The field-observed value of Schnoor (1981), which appears to have been corroborated experimentally by Sudershan and Khan (1980), was weighted more heavily than the value estimated using the Spacie and Hamelink (1982) regression equation that correlates log k_2 with log k_{ow}. This equation has a standard error in prediction of $\log_{10} k_2$ of 0.19 log units, assuming k_{ow} is known precisely.

Table 5.2-12. Dietary Input Factors, Dieldrin Pathways Analysis.
R = Total Dietary Intake $(day)^{-1}$

	Minimum	Best Estimate	Maximum
Eagle	0.51	0.57	0.76
Mallard	0.45	0.52	0.93
Pike	0.01	0.03	0.05
Bluegill	0.01	0.03	0.05
Percent of Item in Diet			
Eagle/Mallard	14	28	42
Eagle/Pike	58	72	86
Mallard/Invertebrates	40	58	75
Mallard/Aquatic Plants	25	42	60
Bluegill/Plankton	6	12	18
Bluegill/Invertebrates	82	88	94

Source: ESE, 1988.

Uncertainty in k_2 for fish is represented by a lognormal distribution with a mean of 0.017 day^{-1} and a standard deviation equivalent to the mean value.

Fish BCF values were estimated based on regression equations from Lyman et al. (1982) and Davies and Dobbs (1984); data were composited to yield a best estimate of 2,050. This value was treated as a single data point along with four measured values:

- 12,590 (Davies and Dobbs, 1984)
- 13,000 (Waller and Lee, 1979)
- 5,800 (Kenaga, 1980)
- 4,420 (Kenaga, 1980)

These data indicate that uncertainty in BCF for fish is best represented by a log normal distribution with a mean of 6,500 and a standard deviation of 2,300.

For the purposes of the uncertainty analysis, BCF data for snails, chironomids, and other invertebrates were combined. Based on data presented in Table 5.2-4, uncertainty distribution for the BCF of invertebrates, aquatic plants, and plankton were defined as lognormal with mean ± standard deviation of 1,900 ± 1,300: 2,400 ± 900; and 11,200 ± 3,500, respectively.

To develop an uncertainty distribution for K_{oc}, measured values by Saha et al. (1971) and Briggs (1981) were weighted more strongly than estimated values. The reported values appear to follow a normal distribution as opposed to lognormal. The mean of these values, plus a composite of several estimates (treated as though the composite was a single data point), was 31,700 ± 7,600.

Organic carbon content of the sediment of the RMA lakes is a measured value (EBASCO, 1988). In the upper 1 foot (ft) of sediment, organic carbon appears to follow a lognormal distribution with a mean of 0.65 percent and a standard deviation of 0.62 percent.

Based on these values, the results of the uncertainty analysis are summarized as follows. The 5th, 50th, and 95th percentile for BMF are 2.23 x 10^6, 2.58 x 10^5, and 1.00 x 10^5, respectively. For the estimate of the acceptable concentration in water, the 5th, 50th, and 95th percentiles are 0.103 ug/l, 0.039 ug/l, and 0.006 ug/l. For the criterion level in sediment, the 5th, 50th, and 95th percentiles are 0.032, 0.0056, and 0.006 ppm. The 50th percentile represents the best estimate, with the 5th and 95th percentiles representing the lower and upper bounds, respectively.

For the terrestrial food chain component of the aquatic Pathway Analysis, a semi-quantitative uncertainty analysis was performed. The soil to plant uptake factor (EMF) was found to be good to a factor of 3. The plant to mammal BAF was more uncertain due to a lack of documentation and problems in the data collected by Garten and Trabalka, and is good only to a factor of 5. The mammal to bird BAF is good to a factor of 2.

Overall error in the terrestrial pathway is approximately a factor of 8 for the BMF or final soil criteria.

5.2.1.7 Summary and Conclusions

Biomagnification of dieldrin residues through a food web applicable to RMA appears to be a problem based on the Pathway Analysis approach. The estimated total BMF for bald eagle was 290,000 from the Pathway Analysis. Cleanup levels based on protection of a high trophic level species (bald eagle) in water, sediments, and soils 0.034 ppb, 0.0055 ppm, and 0.56 ppm, respectively. Criterion levels based on "no effects" levels for aquatic life in water and sediments were 0.05 ppb and 0.0079 ppm, respectively.

The total BMF for kestrel, adjusted for dietary proportions, is 98. The "no effects" soil concentration level calculated for the kestrel food web (0.10 ppm) is lower than that derived from the bald eagle food web (0.56 ppm), and should be used to represent a "no effects" soil concentration for protection of terrestrial species. The lowest acceptable surface water concentration was 8.0 ppb, based on water consumption by and toxicity to waterfowl.

Site specific criteria for dieldrin are summarized as follows:

Method	Water (ppb)	Sediment (ppm)	Soil (ppm)
Water Ingestion	8.0	1.27	NA
Aquatic Pathway Analysis	0.034	0.0055	NA
Aquatic Life	0.05	0.0079	NA
Terrestrial Pathways Analysis--Eagle	NA	NA	0.56
--Kestrel	NA	NA	0.10

5.2.2 PATHWAY ANALYSIS FOR ARSENIC

5.2.2.1 Background Information

The data for water and tissue concentrations used in this analysis were from published values for BCF and BAF. Arsenic was selected for analysis due to its toxicity, widespread distribution on RMA, and persistence in the environment.

Toxicity of Arsenic

The freshwater Final Acute Value for arsenite for protection of aquatic life is 360 ug/l, while the freshwater Final Chronic Value for arsenite is 190 ug/l (EPA, 1986c). For arsenate, insufficient data exist to derive criteria, but the LOAEL for acute and chronic effects are 0.85 and 0.048 ppm, respectively (EPA, 1986c). Water used for irrigation purposes should not exceed 0.1 ppm (EPA, 1981). Background levels of arsenic in western U.S. soils range from <0.10 to 97 ppm (Shacklette and Boerngen, 1984), although not all soil arsenic is in a bioavailable form. Arsenic levels in unpolluted fresh water are usually less than 1 ppb (Moore and Ramamoorthy, 1984).

Aquatic Ecosystems

Aquatic Plants--Several species of algae and a submerged macrophyte (Potamogeton sp.) exposed to concentrations of 2,320 ppb sodium arsenite had 95 to 100 percent mortality within two weeks (Cowell, 1965). For a four day exposure, 31,200 ppb sodium arsenite resulted in 50 percent growth

inhibition for the alga, Selenastrum capricornatum (Richter, 1982). Sodium arsenate decreased growth of various algal species at concentrations of 48 to 202,000 ppb (EPA, 1985a).

Aquatic Invertebrates--For exposure to Arsenic (III) the 96-h LC_{50} for D. magna is 4,340 ppb (EPA, 1985a). The LC_{50} values for invertebrates range from 812 ppb for a cladoceran (Simocephalus serrulatus) to 24,500 ppb for a snail (Aplexa hypnorum). Data for predominately running water genera were not considered appropriate. The chronic value for D. magna exposed to sodium arsenite was 914.1 ppb (EPA, 1985a).

The LC_{50} for cladocerans Bosmina longirostris and D. magna exposed to sodium arsenate were 850 and 7,400 ppb, respectively (EPA, 1985a). The LC_{50} values for D. pulex range from 3,600 to 49,600 ppb (EPA, 1985a).

Fish--The 96-hr lethal concentration for 50 percent of a population (LC_{50}) of rainbow trout (Salmo gairdneri) and bluegill (Lepomis macrochirus) is 25.6 ppm and 34 ppm sodium arsenite, respectively (Gilderhus, 1966). Decreased survival and growth have been observed in bluegill chronically exposed to 4 ppm sodium arsenite in water, and behavioral changes have been observed in goldfish (Carassius auratus) exposed to 0.1 ppm arsenic in water for 48 hr (Gilderhus, 1966: Weir and Hine, 1970). Following an 8-week exposure to 10, 20, or 30 ppm sodium arsenite in the diet, hemoglobin in treated rainbow trout was significantly reduced compared to controls (Oladimeji et al., 1984).

Terrestrial Ecosystems

Plants--Inorganic arsenic is toxic to plants, affecting photosynthesis, respiration, growth, and reproduction (Wauchope, 1983). Symptoms include wilting and tissue necrosis followed by death (Woolson et al., 1971). Arsenic levels higher than 2.1 to 8.2 ppm on a dry weight basis in leaves can result in injury to fruit trees (Kabata-Pendias and Pendias, 1984).

Phytotoxicity is dependent on the concentration of available arsenic in the soil (Kabata-Pendias and Pendias, 1984). Since the amount of available arsenic is only a fraction of the total soil arsenic, toxicity estimates

based on total soil arsenic indicate artificial resistance to soil arsenic (e.g., plants will appear to tolerate 100 ppm when actual exposure is 10 ppm).

Phytotoxicity is a function of the chemical form (Woolson et al., 1971), as water soluble arsenite is up to 10 times more toxic than arsenate (Woolson, 1983). Soil parameters such as soil type, nutrient status, and pH influence the toxicity of arsenic to plants. For example, arsenic in sandy soil is more phytotoxic at a given level than in clay loams and silt loams (Woolson, 1983). Increases in soil phosphorus can also dislodge arsenic from adsorption sites, resulting in increased toxicity (Woolson, 1983). Bioavailability of arsenic is greatest to plants in soil with a neutral pH (Kenyon et al., 1979).

Although some plants (Jasione montana) grown on highly contaminated soil (8,500-26,500 ppm) have been observed to contain as much as 6,640 ppm arsenic (Porter and Peterson, 1975), residues usually remain low as a result of phytotoxicity (Woolson, 1983). The concentration of available soil arsenic that reduced growth by 50 percent for several species of crops ranged between 6.2 and 48.3 ppm, with green beans the most sensitive and cabbage the most resistant. Assuming that only 10 percent of the total arsenic is bioavailable, the concentration of total soil arsenic that results in 50 percent growth reduction becomes 62 to 483 ppm (Woolson, 1983).

Invertebrates--No information on the toxicity of arsenic to terrestrial invertebrates was found in the literature researched.

Birds--The toxicity of arsenic varies with chemical form. The safe level of organic arsenic in diet of young turkeys ranges from 5 to over 3,200 ppm for various organic arsenicals (Sullivan and Al-Timimi, 1972a, 1972b, 1972c).

A selenium-vitamin E deficiency in ducklings resulted from 600 ppm sodium arsenilate added to a commercial diet for 4 weeks, (Van Vleet, 1982). Wiemeyer et al. (1980) found osprey (Pandion haliaetus) containing elevated levels of arsenic; one bird had a potentially lethal level of 16.7 ppm in

liver. Cowbirds (Molothrus ater) fed a diet containing 225 ppm copper acetoarsenite died; there was 38.5 and 42.6 ppm (dry weight basis) in the liver (Stickel, 1980). Those fed a 25 ppm diet had 2.68 and 2.95 ppm in liver.

Mammals--The toxic dose for wild rabbits of copper acetoarsenite, calcium arsenate, and lead arsenate is 10.5, 23.5, and 40.4 mg/kg bw as arsenic, respectively (Chappellier and Raucourt, 1936). Boyce and Verme (1954) observed a toxic dose of 923 mg sodium arsenite in white-tailed deer. The chronic NOEL for rats fed arsenic as arsenite and arsenate was 62.5 and 125 ppm (estimated as 4.68 and 9.38 mg/kg bw (Sax, 1984), respectively (Casarett and Doull, 1980). Chronic exposure of dogs to 125 ppm arsenic as arsenite resulted in 100 percent lethality (estimated as 3.12 mg/kg bw (Sax, 1984)) (Casarett and Doull, 1980).

Toxic effects result primarily from arsenite reacting with sulfhydryl groups and disrupting cellular enzyme systems (Buck, 1978b). Tissues with oxidative functions such as liver, lung, and kidney are the most affected. Because arsenic is rapidly excreted via the kidneys and to a lesser extent the gastrointestinal tract, tissue levels don't always correlate with symptoms of poisoning. Buck (1978b) observed that liver concentrations of 2 to 100 ppm in unspecified species resulted in acute toxicosis and mortality. Background levels of arsenic in tissue are less than 0.5 mg/kg (Goede, 1985).

Organic arsenicals are not as toxic to plants or animals as inorganic forms. Organic arsenicals, primarily the pentavalent phenylarsonic acids and their salts, are used as feed additives for livestock to improve feed efficiency (Ledet and Buck, 1978). Phenylarsonic compounds are poorly absorbed from the digestive tract and rapidly excreted via the kidneys: tissue levels decrease quickly once exposure stops (Ledet and Buck, 1978). Pigs consuming 1,000 ppm arsanilic acid in the diet had a maximum concentration of 10 ppm in tissue (kidney) and exhibited signs of acute toxicosis following 27 days of exposure (Ledet and Buck, 1978). In other mammals (dairy sheep and goats), 90 percent of a single 10 mg/kg dose was excreted within 120 h, and concentration in tissues and milk was low (Shariatpanahi and Anderson,

1984). Arsenocholine, while almost 100 percent absorbed from the digestive tract, is 70 to 80 percent excreted in urine within 3 days (Marafante et al., 1984).

Bioaccumulation Potential of Arsenic

Arsenic is not significantly concentrated by aquatic invertebrates. Whole body concentration factors for various invertebrates ranged from 3 to 17 for exposure to arsenic trioxide (trivalent), and from 0 to 7 for arsenic pentoxide (pentavalent) (EPA, 1985a).

Although arsenic is bioconcentrated by animals at the bottom of aquatic food chains, data do not indicate significant biomagnification (EPA, 1985a; Isensee et al., 1973). The high depuration rate of arsenic in fish and the low bioconcentration factors indicate that higher predators are not at significant risk (EPA, 1985a). When arsenic was analyzed in sediments and bottom feeding fish, bioaccumulation factors were less than one (Hunter et al., 1981). The highest tissue concentrations were observed in planktivores as opposed to omnivores or piscivores.

In general, arsenic concentrations in soil must exceed 200 to 300 ppm for edible crops to accumulate 1 ppm on a wet weight basis (Woolson, 1983). Root crops; however, tend to accumulate higher arsenic residues. In a study on potatoes, 2.2 to 25.7 ppm total arsenic in soil resulted in 0.2 to 2.6 ppm in the skin of the tubers, although the inner tissue did not exceed 0.6 ppm in concentration (Steevens et al., 1972). Due to high phytotoxicity, plants growing in soils with high arsenic concentrations usually die before accumulating concentrations high enough to poison herbivores. Arsenic poisoning from plants to animals is uncommon (Kabata-Pendias and Pendias, 1984), although foraging animals are sometimes poisoned by consuming plants contaminated with arsenical pesticides (Buck, 1978).

Fate of Arsenic in the Environment

Arsenic is ubiquitous in the environment (EPA, 1976). Arsenate (pentavalent) is the predominant form in oxygenated water, whereas arsenite (trivalent) prevails under anaerobic conditions (EPA, 1981). Arsenite is

slowly oxidized to arsenate in aerobic water at neutral pH and more rapidly in alkaline or acidic solutions (EPA, 1985a).

Inorganic arsenic forms relatively insoluble complexes in soil, binding to hydrous oxides on clays or cations in the soil solution (Woolson, 1983). Levels for arsenic in untreated soils in the United States range between less than 0.1 to 69 ppm (Kabata-Pendias and Pendias, 1984; Woolson, 1983).

Inorganic arsenic resembles phosphorus in chemical behavior, and its activity is influenced by iron, aluminum, calcium, phosphorus, and humus in the soil (Woolson et al., 1971). Arsenic binds to iron, and to a lesser extent aluminum and calcium, which greatly reduces its water solubility, leachability, and bioavailability (Woolson, 1983; Kenyon et al., 1979). Soil texture also affects the behavior of arsenic, because reactive iron and aluminum vary with clay content, increasing as clay content increases (Woolson et al., 1971).

In general, the more water soluble forms of arsenic are found in areas receiving little rainfall (Woolson et al., 1971). RMA soil parameters (low clay content of several soil types, low organic carbon, oxidizing environment, a relatively low cation exchange capacity as predicted by bulk mineral analysis (Wullschleger and Schloz, 1981)) and local precipitation patterns suggest that arsenic will be in the more mobile forms in the RMA environment.

Arsenic can be methylated in sediment or complex with organic ligands in water to form organoarsenical compounds (EPA, 1981). Two common organoarsenicals in water are the postemergence herbicides, methanearsonic acid and dimethylarsinic acid (cacodylic acid) (EPA, 1985a). While insufficient data are available to derive water quality criteria for organic arsenicals, data indicate that aquatic organisms are less sensitive to organic arsenic such as monosodium methanearsenate (MSMA) (acutely toxic at 1,900 ug/l) than to inorganic arsenate (acutely toxic at 850 ug/l) (EPA, 1985a).

Arsenic is stable in water in the arsenate, arsenite, organic, and arsine forms (EPA, 1981). In sandy soils, arsenic can leach into the groundwater,

although studies on various soil types and forms of arsenic indicate undetectable leaching below 90 centimeters (cm) (EPA, 1981). There is a greater likelihood that arsenic will enter surface water as a result of surface runoff, with the amount entering surface water dependent on terrain, vegetation, and precipitation (EPA, 1981). Of the arsenical pesticides applied to soil for agricultural purposes, approximately 7 percent migrate into surface water annually (EPA, 1981).

In aerobic soils, arsenic is found mainly as arsenate (Woolson, 1983; EPA 1981), whereas in anaerobic, flooded soils arsenic is reduced to arsenite (EPA, 1981). Under extreme reducing conditions, arsine can be formed as well as organic arsenicals and arsenic (EPA, 1981). Arsenite can be oxidized by microbial action to arsenate in well oxidized soils. Increased mobility or leaching can occur in loam or sandy soils, or under alkaline conditions, clays tend to decrease mobility (EPA, 1981). Significant quantities of arsenic can be lost from soils as a result of volatilization of methylated forms, with the amount lost dependent on soil characteristics and vegetative growth (EPA, 1981).

5.2.2.2 Surface Water Ingestion

In addition to exposure by ingestion of contaminated food items, the key organisms are potentially exposed to contaminants by ingestion of surface water. The key organisms for which a surface water pathway becomes important are the nonaquatic animals such as small mammals, waterfowl, and raptors. Bioconcentration as defined for aquatic organisms is not applicable to nonaquatic organisms, because tissue concentrations are not a direct function of water concentration. However, uptake of contaminants in surface water consumption can occur, with accumulation rates depending on the amount of water ingested daily and the concentration of contaminants in the water supply.

The acute toxic dose of arsenic for wild rabbits is 10.5, 23.5, and 40.4 mg/kg bw for copper acetoarsenite, calcium arsenate, and lead arsenate, respectively (Chappellier and Raucourt, 1936) (Table 5.2-13). Boyce and Verme (1954) reported a toxic dose of 923 mg sodium arsenite in white-tailed deer; however, this was not correlated with body weight, and so was not used

Table 5.2-13. Toxic Effects Levels of Arsenic for Mammals and Birds by Ingestion

Species	Exposure Route	Dose (mg/kg bw/day)	Acceptable Water Concentration (ppm)	Effect
Rabbits	oral	10.5	0.013	Death[1]
Rats	diet	4.68	7.49	NOEL[2]
Dogs	diet	3.12	2.5	Death[2]
Duckling	diet	60	1.2	Selenium-vitamin E deficiency[3]

Sources:
1. Chappellier and Raucourt, 1936.
2. Casarett and Doull, 1980.
3. Van Vleet, 1982.

in calculating water criteria. An acceptable water concentration was calculated from the chronic lethal level for dogs and water consumption data for dogs as follows:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{3.12 \text{ mg/kg bw/day}}{0.05/\text{kg bw/day}} = 62.4 \text{ mg/l}$$

The LOAEL is divided by an uncertainty factor of 5 to convert the chronic LOAEL to a chronic NOEL, and an uncertainty factory of 5 for interspecific variation, resulting in an acceptable water concentration of 2.5 mg/l (2,500 ppb).

Birds--Data were examined to determine the most sensitive toxicological endpoint for avian species. Water consumption rate for birds was based on water consumption data for waterfowl. Ducks in captivity consume 200 ml/kg bw on a daily basis (Sax, 1984). Assuming that wild populations of ducks consume an equivalent amount of water as ducks in captivity, an acceptable water concentration can be derived as follows:

$$\frac{\text{LOAEL or NOEL}}{\text{Intake/kg bw/day}} = \text{Acceptable Surface Water Concentration}$$

A selenium-vitamin E deficiency was observed in ducklings after subchronic exposure to 600 ppm sodium arsenite in commercial diet (Van Vleet, 1982) (Table 5.2-13). Arsenic intake was estimated from food consumption data for adult ducks of 100 g/kg bw/day (Sax, 1984); as food consumption rates would be higher for ducklings, this results in a minimum arsenic intake of 60 mg/kg bw/day. From the following equation:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{60 \text{ mg/kg bw/day}}{0.2 \text{ l/kg bw/day}} = 300 \text{ mg/l}$$

Applying uncertainty factors of 50 to convert the subchronic LOAEL to a chronic NOEL, and 5 for interspecific variation, an acceptable water concentration of 1.2 mg/l (1,200 ppb) is derived.

Acceptable water concentrations are summarized in Table 5.2-13.

The lowest "no effect" concentration for surface water ingestion was 0.013 ppm; however, uncertainty in this estimate is very high because the estimate is derived from an acute lethal dose. The estimate based on subchronic toxicity to mallards (1.2 ppm), although higher, is considered to be a better estimate. The value for duckling was considered to be better than the value for dogs, because it was based on sublethal as opposed to lethal effects. Corresponding sediment criteria, based on a K_d of 148.4 l/kg (see Section 5.2.3.6), were 178.1 ppm. This value is assumed to be protective of all species that may consume water at RMA.

5.2.2.3 Aquatic Life

The EPA chronic criteria for the protection of aquatic organisms and their uses (190 ppb) were considered appropriate as site-specific criteria for arsenic. The toxicity values for all fish species were within the range of values for species that could be found at the RMA lakes. The corresponding sediment criterion is calculated as follows:

$$C_{sed} = C_w \times K_d \qquad (8)$$

where: K_d = 148.4 l/kg (see Section 5.2.3.6)

$$C_{sed} = 190 \text{ ug/l} \times 148.4 \text{ l/kg}$$
$$= 28{,}196 \text{ ug/kg}$$
$$= 28.2 \text{ ppm}$$

5.2.2.4 Aquatic Pathway Analysis

Introduction to Aquatic Pathway Analysis

This Pathway Analysis is based on the bald eagle sink food subweb and includes all major food chains leading to the selected sink species (Cohen, 1978). Because the same organisms/groups appear in more than one food chain throughout the web, percentage contributions for each organism or compartment have been estimated based on existing literature (Table 5.2-14). The subweb has been simplified (e.g., bluegill represent all fish species at that trophic level) because of the limited data available.

Methods for Aquatic Pathway Analysis

Published values were used for BCF values for the aquatic organisms (Table 5.2-15), as RMA data were unavailable.

Table 5.2-14. Summary Of Feeding Habits, Pathways Analysis for Arsenic

Species	Food Items	% in Diet	Reference
Mallard	Invertebrates[1]	44	Swanson et al., 1979; Swanson et al., 1985
	Plants[2]	30	Swanson et al., 1979 Swanson et al., 1985
	Annelids[3]	26	Swanson et al., 1979
Bald Eagle	Waterfowl	24	Cash et al., 1985; Todd et al., 1982
	Fish	66	Cash et al., 1985
	Mammals	10	Cash et al., 1985
Bluegill	Invertebrates	88	Martin et al., 1961
	Plankton, Algae	12	Martin et al., 1961
Pike	Fish[4]	100	Inskip, 1982

1 Includes Crustacea, Insecta, and Mollusca.

2 "Plants" includes fruits and miscellaneous seeds (Swanson et al., 1979; Swanson et al., 1985). Fruits were included with other vegetation forming the mallards diet, although data quantifying dieldrin adsorption or absorption by aquatic fruits was unavailable in the literature researched.

3 These food items were not utilized in the pathways analysis. Annelids are apparently washed into aquatic systems (Swanson et al., 1979) and were not included because areas upgradient of the RMA lakes are considered to be uncontaminated.

4 Pike are opportunistic feeders that will utilize other food sources, but are assumed to prey completely on fish for the sake of the analysis.

Source: ESE, 1988.

Table 5.2-15. Bioconcentration Factors Used In The Pathways Analysis for Arsenic (Page 1 of 2)

Organism/Group	Form*	BCF	Sources
Aquatic Plants			
Submergent	As	286	Reay, 1972**
Submergent		81.6	
Submergent		696	
Submergent		1,981	
Submergent		1,310	
Submergent		457	
Submergent		534	
Water Hyacinth	V	2.6	Chigbo et al., 1982**
Mixed	As	97	Wagemann et al., 1978
Geometric Mean	BCF	240	
Plankton			
Mixed	III	20.9	Dupree, 1960**
		714	
		118	
		366	
		206	
		278	
Zooplankton	As	46	Wagemann et al., 1978
Daphnia magna	As_2O_3	10	Spehar et al., 1980
Daphnia magna	As_2O_5	4	Spehar et al., 1980
Geometric Mean BCF		74	
Invertebrate			
Snail	As_2O_3	3	Spehar et al., 1980
Snail		17	
Stonefly		9	
Snail	As_2O_5	3	
Snail		6	
Stonefly		7	
Pelecypoda	As	140	Wagemann et al., 1978
Gastropoda		80	
Oligochaeta		33.7	
Ephemeroptera		111	
Trichoptera		19	
Diptera		39	
Zygoptera		12	
Coleoptera		24	
Coleoptera		23	
Diptera		5.6	
Diptera		4.2	

Table 5.2-15. Bioconcentration Factors Used In The Pathways Analysis for Arsenic (Page 2 of 2)

Organism/Group	Form*	BCF	Sources
Hydracarina		13.2	
Hirudinea		57	
Amphipoda		104	
Hemiptera		14.4	
Hemiptera		18	
Hemiptera		23	
Hemiptera		4.2	
Anisoptera		26	
Geometric Mean BCF		18	
Fish			
Bluegill	III	4	Barrows et al., 1980
Fathead Minnow	V	3	DeFoe, 1982
Unspecified	As	44	EPA, 1985b
		8.1	

* Form of arsenic to which biota were exposed
- III - arsenite
- V - arsenate
- As - form not specified
- As_2O_3 - arsenic trioxide
- As_2O_5 - arsenic pentaoxide

** Concentrations converted to wet weight from dry weight using a water content of 90% before calculating BCF (EPA, 1985).

Source: ESE, 1988.

The K_d value for arsenic used in this analysis is a geometric mean 148.4 l/kg (see Section 5.2.3.6).

The sink food web (combined food transfer pathways based on an aquatic or terrestrial diet) for arsenic is presented in Figure 5.2-4. Five food transfer pathways ultimately terminating with the bald eagle were established as follows:

		Trophic Level			
Pathway	Source	1	2	3	4
1	H_2O	Invertebrates	Mallard	Bald Eagle	
2	H_2O	Aquatic Plants	Mallard	Bald Eagle	
3	H_2O	Plankton	Bluegill	Pike	Bald Eagle
4	H_2O	Invertebrates	Bluegill	Pike	Bald Eagle
5	Soil	Terrestrial Plants	Small Mammals	Bald Eagle	

The number of pathways varies with each contaminant depending on the quality and quantity of data available for the analysis: therefore, there are only five pathways for arsenic as opposed to eight pathways for dieldrin. The mallard and the pike represent all birds and fish fed upon by the bald eagle.

All pathways (except Pathway Five) originate with water. The lowest step in the food chain is assumed to be in equilibrium with the aquatic environment, which gives equation (1):

$$BCF = C_b/C_w \qquad (1)$$

where: C_b = the concentration of arsenic in biota
C_w = the concentration of arsenic in water

This equation is vital to the rest of the analysis. The end result, the total BMF for the bald eagle, can be ultimately traced back through water to the sediment, because it is assumed that all arsenic enters the water compartment from sediments before being taken up by the biological compartment:

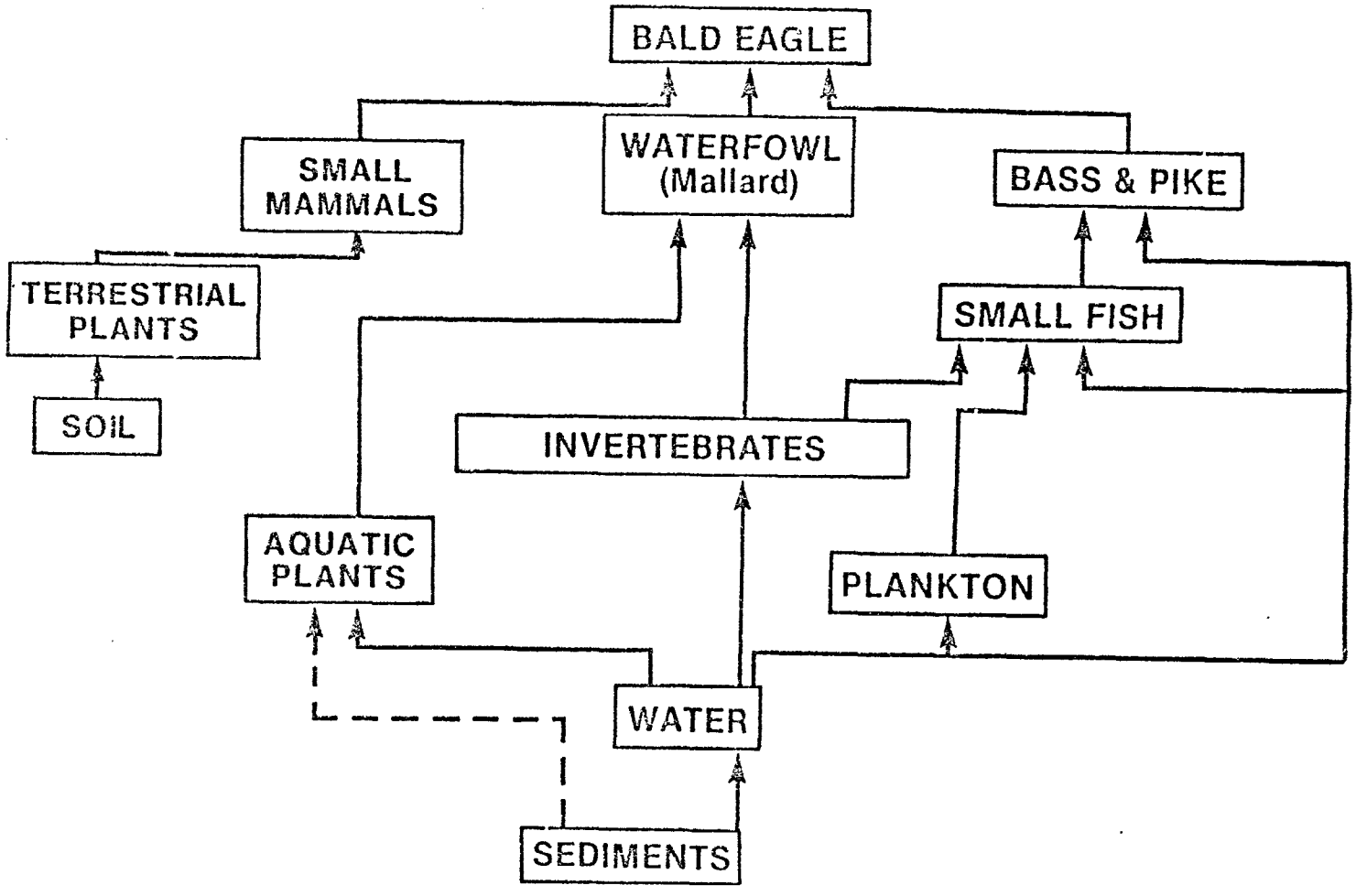


Figure 5.2-4

BALD EAGLE SINK FOOD WEB FOR ARSENIC

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

$$C_w = \frac{C_{sed}}{K_d} \quad (7)$$

or solving for C_{sed} gives equation (8):

$$C_{sed} = C_w \times K_d \quad (8)$$

where: C_{sed} = concentration of arsenic in the sediment
C_w = concentration of arsenic in water
K_d = sediment-water partition coefficient

The method used in the Pathway Analysis is the Thomann (1981) bioaccumulation model of food chain transfer in aquatic ecosystems where each level is a step in the food chain:

Level #1 $BCF_1 = C_b/C_w$ (1)

Level #2 $BAF_2 = BCF_2 + f_2BCF_1$ (2)

Level #3 $BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1$ (3)

Level #4 $BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1$ (4)

The food term (f_i) is a function of the trophic level in question and is calculated by the following equation:

$$f_i = \frac{@ \times R \times \%}{k_2} \quad (5)$$

where: @ = Assimilation efficiency, $\frac{\text{ug absorbed}}{\text{ug ingested}}$
R = Total daily diet, intake (g)/body weight (g)/day
k_2 = Depuration or loss rate, day^{-1}
% = Percent of item in diet

Two studies provided data used to estimate assimilation efficiency. A study by Coulson et al. (1935) observed that rats retained 80 percent of inorganic arsenic ingested in diet. Only four rats were utilized in the excretion study. Another study by Oladimeji et al. (1984) observed arsenic residues in muscle and liver in relation to diet. An estimate of assimilation efficiency can be made by comparing total ingested arsenic for 14 days to tissue residue, thereby obtaining a geometric mean of 0.4. Since excretion

was occurring during the ingestion period, actual assimilation of arsenic by fish may be much higher. Therefore, 0.8 was used to represent all species in the analysis.

The depuration or loss rate (k_2) includes residue loss due to growth dilution, excretion, and metabolism. Because rate constants have not been measured for each species in this analysis, k_2 values taken from the literature were used to represent all species. The following k_2 values were employed in the Pathway Analysis:

k_2 = 0.06/day	Derived from Woolson et al., 1976 study on catfish that lost from 75 to 93 percent of body burden in 14 days, or an average daily rate of 0.06.
k_2 = 0.28/day	Calculated from a study with chickens (Overby and Fredrickson, 1965) that indicated half-life of arsenate in blood was >2.5 days. For the purposes of the analysis, 2.5 days was used as the half-life.
k_2 = 0.46/day	Calculated from a study with chickens (Overby and Fredrickson, 1965) that indicated half-life of arsenilic acid in blood was 1.5 days.

A geometric mean value of 0.36/day calculated from the half-life data in chickens was used to represent loss from avian species. For fish, the loss rate of 0.06/day was used.

A Pathway Analysis was not performed for arsenic accumulation by ducklings because the high loss rate due to growth dilution is expected to outweigh the increased exposure due to feeding rate. Ducklings are at greater risk as a result of direct toxicity from ingestion of contaminated water or food items than from residue bioaccumulation.

Pathway Analysis

The Pathway Analysis uses BCF, k_2, and f_2 values in the Level #1 through Level #4 equations to derive a BAF for key organisms in each food chain. When BAFs for each food chain have been calculated, they are summed to give a BMF for the food web. The food chain BAF calculations are presented in the following sections.

Pathway One: H_2O => Invertebrates => Mallard => Bald Eagle--BCFs for invertebrates range from 3 to 140 for various aquatic invertebrates including gastropods, oligochaetes, and dipterans, and others (Spehar et al., 1980; Wagemann et al., 1978). Data from the Wagemann et al. study were collected in field studies comparing accumulation factors over a several month period for several lakes (a geometric mean of data from four lakes for five months for each taxon was calculated to obtain one data point per taxon); therefore, these data actually represent BAF values. However, for the purposes of the analysis, small aquatic invertebrates are assumed to be in equilibrium with their environment; at this trophic level, the processes of bioconcentration are assumed to outweigh biomagnification to the extent that the BAF is equivalent to the BCF. A geometric mean value was used to represent bioconcentration in the Pathway Analysis:

$$BCF_{invert} = 18 \qquad (1)$$

The food term (f_2) is calculated by assuming that an adult mallard weighs approximately 1,100 g and consumes about 57.4 g total diet each day (Miller, 1975), of which 44 to 56 percent of the diet is invertebrates (Swanson et al., 1979). Sax (1984) indicates adult ducks in captivity consume nearly twice this much daily. For the pathway analysis, 44 percent was used to represent invertebrate intake by mallards. The BAF for a mallard is calculated by assuming that the first term in the Level #2 bioaccumulation equation (2) equals zero, because bioconcentration by nonaquatic organisms is considered to be negligible:

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2 BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.8 \times (57.4\ g/1{,}100\ g/day) \times 44\%}{0.36/day} = 0.051 \qquad (5)$$

When the BCF for invertebrates is 18, the BAF for mallard is 0.92.

An adult eagle weighs approximately 4,500 g (Schafer, 1986) and consumes 255 g daily (Swies, 1986), of which 24 percent of the diet is birds (Cash et al., 1985; Sherrod, 1978). Energy requirements are different for wild birds than birds living in captivity, so these dietary quantities are only approximate (Jehnkins, 1986; Sherrod, 1986). The following BAF values for an eagle are calculated by assuming that the first two terms in the Level #3 bioaccumulation equation (3) equal zero (bioconcentration by the mallard and the eagle are both negligible):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{invert}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f_3 = \frac{0.8 \times (255\ g/4{,}500\ g/day) \times 24\%}{0.36/day} = 0.030 \qquad (5)$$

When the BCF for invertebrates is 18, the BAF for eagle is 0.028.

Pathway Two: H_2O => Aquatic Plants => Mallard => Bald Eagle--The BCF for plants was estimated from observed values for submerged macrophytes of 2.58 to 2,000 (Reay, 1972; Chigbo et al., 1982; Wagemann et al., 1978). Tissue concentrations for Reay (1972) and Chigbo et al. (1982) have been corrected with a factor of 0.1 (Rickett, 1921) to convert values given on a dry weight basis to wet weight prior to calculating the BCF. A geometric mean value was used to represent BCF for aquatic plants:

$$BCF_{plant} = 240 \qquad (1)$$

To calculate the BAF for a mallard, the food term f_2 remains the same as in Pathway One, except that the percentage of the food item in the diet is now 30 percent. The BAF is calculated using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2BCF_{plant}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.8 \times (57.4\ g/1{,}100\ g/day) \times 30\%}{0.36/day} = 0.035 \qquad (5)$$

When the BCF for aquatic plants is 240, the BAF for mallard is 8.4

The food term for the consumption of mallards by the eagle, f_3, remains the same as in the Pathway One equation. The BAF is calculated using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{plant}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.8 \times (255\ g/4{,}500\ g/day) \times 24\%}{0.36/day} = 0.030 \qquad (5)$$

When the BCF for aquatic plants is 240, the BAF for eagle is 0.25.

Pathway Three: $H_2O \Rightarrow$ Plankton $\Rightarrow$ Bluegill $\Rightarrow$ Pike $\Rightarrow$ Bald Eagle--Pathways leading to the bald eagle via fish are more complex because bioconcentration occurs at each trophic level, not just at the lowest trophic level. This introduces a fourth factor into the BAF equation, as the eagle is at Level #4 instead of Level #3. The BCF for plankton ranges from 4 to 714 for various forms of arsenic (Table 5.2-16) after applying a correction factor of 0.1 (Rickett, 1921) to convert dry weight to wet weight. For zooplankton exposed to arsenic in a field study, the BCF was 46 (Wagemann et al., 1978). The geometric mean was used to represent bioconcentration in the Pathway Analysis:

$$BCF_{plankton} = 74 \qquad (1)$$

The BCF for the bluegill (Lepomis macrochirus) is derived from studies indicating BCF values for bluegill of 4 (Barrows et al., 1980), fathead minnow of 3 (DeFoe, 1982), and unspecified fish of 44 (EPA, 1985a). A geometric mean value was selected as the BCF for bluegill:

$$BCF_{bluegill} = 8.1 \qquad (1)$$

Table 5.2-16. Summary of Bioaccumulation Factors for Each Species in the Pathways Analysis for Arsenic.

	Bluegill	Pike	Duck	Mammal	Eagle
Pathway 1	--	--	0.92	--	0.028
Pathway 2	--	--	8.4	--	0.25
Pathway 3	12	13	--	--	1.1
Pathway 4	14	14	--	--	1.2
Pathway 5	--	--	--	3.5	0.00056

Source: ESE, 1988.

If a bluegill consumed 3 percent of its body weight daily (Chadwick and Brocksen, 1969), the total daily intake term (R) would be 0.03 regardless of actual body weight. Various algal forms account for approximately 12 percent of the bluegills diet (Martin et al., 1961); this value was used for the percent of plankton in the bluegill diet. The k_2 value used for the bluegill is based on a loss rate of 0.06/day (Woolson et al., 1976). The BAF is calculated using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$
$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{plankton}$$

$$\text{where: } f_2 = \frac{0.8 \times (0.03/day) \times 12\%}{0.06/day} = 0.048 \qquad (5)$$

The BCF for the pike (8.1) is derived from the same data set as the BCF for bluegill. The pike is also estimated to consume 3 percent of its body weight daily (Chadwick and Brocksen, 1969), such that the total daily intake term (R) would be 0.03 regardless of actual body weight. It is assumed that pikes feed entirely on bluegills for the sake of this analysis. The k_2 value used for the bluegill is based on a loss rate of 0.06/day (Woolson et al., 1976). The BAF is calculated using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$
$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{plankton}$$

$$\text{where: } f_3 = \frac{0.8 \times (0.03/day) \times 100\%}{0.06/day} = 0.40 \qquad (6)$$

The eagle food term (f_4) was calculated by assuming an eagle weighs 4,500 g and consumes 255 g food daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. The BAF was calculated using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \qquad (4)$$
$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{plankton}$$

where: $BCF_4 = 0$

$$f_4 = \frac{0.8 \times (255\ g/4{,}500\ g/day) \times 66\%}{0.36/day} = 0.083 \qquad (5)$$

When the BCF for plankton is 74, the BAF for bluegill is 12. The BAFs for pike and eagle are 13 and 1.1, respectively.

Pathway Four: $H_2O \Rightarrow$ Invertebrates $\Rightarrow$ Bluegill $\Rightarrow$ Pike $\Rightarrow$ Bald Eagle--BCFs for aquatic invertebrates range from 3 to 140 for various aquatic invertebrates including gastropods, oligochaetes, and dipterans, and others (Spehar et al., 1980; Wagemann et al., 1978). Data from the Wagemann et al. study were collected in field studies comparing accumulation factors over a several month period for several lakes (a geometric mean of data from four lakes for five months for each taxon was calculated to obtain one data point per taxon): therefore, these data actually represent BAF values. For the purposes of the analysis, small aquatic invertebrates are assumed to be in equilibrium with their environment; at this trophic level, the processes of bioconcentration outweigh biomagnification to the extent that the BAF is equivalent to the BCF. A geometric mean value was used to represent bioconcentration in the Pathway Analysis:

$$BCF_{invert} = 18 \qquad (1)$$

The BCF for bluegill (8.1) is the same as Pathway Three. Aquatic invertebrates account for approximately 88 percent of the bluegills diet (Martin et al., 1961). The BAF is calculated using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2 BCF_{invert}$$

where: $$f_2 = \frac{0.8 \times (0.03/day) \times 88\%}{0.06/day} = 0.35 \qquad (5)$$

The BCF for pike (8.1) was derived from the same data set as bluegill. The food term is the same as Pathway Three. The BAF is calculated using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{invert}$$

where: $f_3 = \frac{0.8 \times (0.03/day) \times 100\%}{0.06/day} = 0.40$ (5)

The eagle food term (f_4) was calculated by assuming an eagle weighs 4,500 g and consumes 255 g food daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. The BAF is calculated using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{invert}$$

where: $BCF_4 = 0$

$f_4 = \frac{0.8 \times (255\ g/4{,}500\ g/day) \times 66\%}{0.36/day} = 0.083$ (5)

When the BCF for aquatic invertebrates is 18, the BAF for bluegill is 14. The BAF for pike is 14, and the BAF for eagle is 1.2.

Discussion and Conclusions

Biomagnification is the result of bioconcentration and bioaccumulation by which tissue concentrations of chemicals increase as the chemical is transferred up food chains (Rand and Petrocelli, 1985). The term implies systematic transfer between trophic levels and can be used to predict interrelationships between the abiotic environment and selected target species.

BAF values as derived for the individual pathways (Table 5.2-16) represent accumulation in separate single food chains. To derive overall accumulation in the entire food web, variations of the following equation are used:

$$BMF_i = BCF_i + \Sigma f_i BAF_{i-1}$$

For each of the major trophic levels in the aquatic Pathway Analysis, total biomagnification is presented in Table 5.2-17.

Total BMF can be used to determine maximum allowable levels of arsenic in sediment by relating sediment concentration to maximum acceptable tissue concentration as follows (Tucker, 1986):

$$\frac{MATC}{\text{Total BMF}} = C_w \qquad (6)$$

and, $$C_{sed} = C_w \times K_d \qquad (8)$$

where: K_d = 148.4 (See Section 5.2.3.6)

The MATC was based on a published value for the lowest concentration which resulted in sublethal or lethal toxic effects:

SPECIES	ORGAN	PPM	EFFECT	REFERENCE
Cowbird	liver	10.2 (9.6-10.7)	Death	Wiemeyer et al., 1980
Cowbird	liver	0.70 (0.67-0.74)	Survived	Wiemeyer et al., 1980
Animals (species unknown)	kidney, liver	>10 (2-100)	Death	Buck, 1978
Chicken	liver	4-12	Death	Wiemeyer et al., 1980

The lowest tissue concentration at which toxic effects have been observed is divided by the BMF for eagle from Table 5.2-17, then corrected with K_d to give the water or sediment concentration at which "no effects" are likely to occur. Sublethal effects data were unavailable in the literature researched. At liver concentrations less than 0.74 ppm, cowbirds survived treatment: liver concentrations of 4 ppm and greater are correlated with mortality in birds. Buck (1978b) states that levels greater than 10 ppm are diagnostic of arsenic poisoning, while levels of 2 to 10 ppm can be indicative of toxicosis or mortality. The highest concentration in liver of cowbirds that survived was used to represent the MATC. The margin of safety is very narrow, because at less than 1 ppm in tissue birds survived, but at 2 ppm mortality in animals can possibly occur.

Table 5.2-17. Total Biomagnification of Arsenic Residues for each of the Key Organisms in the Aquatic Pathways Analysis.

Organism	Level	Equation	BMF
Mallard	#2	$\Sigma f_2 BCF_1$	9.3
Bluegill	#2	$BCF_2 + \Sigma f_2 BCF_1$	19
Pike	#3	$BCF_3 + f_3 BMF_{bluegill}$	16
Eagle	#3, #4	$f_4 BMF_{pike} + f_3 BMF_{mallard} + BMF_{terrestrial}$	1.6

Source: ESE, 1988.

The "no effects" level in sediment and water for bald eagle is derived as follows:

$$\frac{\text{MATC}}{\text{Total BMF}} = C_w = \frac{0.74\ \text{ppm}}{1.6} = 0.46\ \text{ppm} \qquad (11)$$

$$C_{sed} = C_w \times K_d = 0.46\ \text{ppm} \times 148.4 = 68.26\ \text{ppm} \quad (3)$$

Due to the high loss rate of arsenic from tissue, it may not be appropriate to base water, or sediment criteria on arsenic levels in tissue, because arsenic does not tend to accumulate in tissues. Toxic effects can occur with no significant increase in tissue concentrations. Criteria for the abiotic environment should consider toxicity to organisms at lower trophic levels in the food web. Some phytotoxic effects occur as a result of irrigating crops with groundwater containing levels higher than the EPA criteria for irrigation water of 100 ppb (0.1 ppm) (EPA, 1981). When a K_d of 148.4 l/kg is used to convert water criteria for crop irrigation to sediment concentrations, a "no effects" level of 14.8 ppm in sediments is obtained.

Organic arsenicals are less toxic than inorganic arsenicals (50 to 100 ppm phenylarsonic compounds recommended in poultry feed as a dietary supplement (Ledet and Buck, 1978)) and the excretion rate is higher. A sediment or soil level based on toxicity of inorganic arsenic will thus protect against toxicity of organic arsenic.

5.2.2.5 Terrestrial Pathway Analysis

Introduction to Terrestrial Pathway Analysis

This analysis was performed to determine cleanup criteria for arsenic in a terrestrial based food web (soil-biota) pathway. The approach used for the terrestrial pathway analysis arrives at a "no effects" level in soil of terrestrial ecosystems on RMA by assuming that soils are a source of arsenic contamination.

Methods for Terrestrial Pathway Analysis-- Bald Eagle Food Web

The terrestrial based food chain in the bald eagle food web was analyzed to determine concentration of arsenic from soil to the target organism. The total BMF for the terrestrial food chain was used to derive a soil criterion for arsenic.

Pathway Five: Soil-> Terrestrial Plants-> Small Mammals-> Bald Eagle--

Residue concentration in the pathway leading to the bald eagle through herbivorous mammals becomes insignificant due to the effect of arsenic on plants. Most terrestrial plants are incapable of accumulating arsenic to any extent due to phytotoxic effects. The EMF, the concentration in plants compared to concentration in soil, is therefore usually less than one. Exceptions may occur in plants growing near tailings or smelters (Porter and Peterson, 1975).

Concentration from soil to plants is 0.004 to 0.06 (Steevens et al., 1972), when total soil arsenic was compared to arsenic in potato flesh (N=18). Values below detection were not used. In another study with plants, concentration factors for arsenic in controls were higher than concentration factors in treated plants, while tissue concentrations between controls and treated plants were equal (Kenyon et al., 1979); these data were not used in the Pathway Analysis. A geometric mean value based on data from Steevens et al. (1972) was used to represent the EMF:

$$EMF_{plants} = 0.02$$

The accumulation factor for small mammals was derived from data for rats (Coulson et al., 1935). Animals were fed a stock diet containing 0.2 and 17.9 ppm inorganic As or diets containing naturally occurring arsenic from shrimp, with and without added inorganic arsenic, for a 52 week period. Only inorganic arsenic data were used to calculate accumulation from diet as compared to concentrations in liver: the concentration factors ranged from 2.7 (for the 17.9 ppm diet) to 4.6 (for the 0.2 ppm diet). Since tissue concentrations are only available for liver, these values may be too high, and do not reflect concentration on a whole body basis. The geometric mean value was used to represent the BAF:

$BAF_{mammals} = 3.5$

The amount accumulated from the diet by the eagle was estimated from unpublished data by Stickel, where two cowbirds were dosed with 225 ppm and two with 25 ppm copper aceto-arsenite (Wiemeyer et al., 1980). Data for tissue concentrations were converted from dry weight to wet weight (tissues contained 75 percent water according to Wiemeyer et al., 1980); liver concentrations ranged from 0.67 to 10.7 ppm on a wet weight basis. Dietary concentrations of 25 or 225 ppm copper aceto-arsenite were converted to arsenic concentrations of 11.1 and 100 ppm before calculating BAFs. BAFs ranged from 0.060 to 0.11. A geometric mean value of 0.08 was used to represent concentration by eagle.

The terrestrial pathway is as follows:

$$\text{soil} \xrightarrow{0.02} \text{terrestrial plants} \xrightarrow{3.5} \text{mammals} \xrightarrow{0.08} \text{eagle}$$

The amount accumulated from the diet by the eagle (assuming an accumulation rate equivalent to mammals) is thus 0.0056 times the amount in soil. The terrestrial pathway is 10 percent of the eagles diet; therefore, the total BMF for this pathway is 0.00056.

Results and Discussion

Pathway Five assumes greater significance based on observed winter feeding behavior of eagles at RMA, where eagles subsist primarily on small mammals pirated from other raptors. Observations indicate that approximately 90 percent of the eagle diet is made up of small mammals: the "no effects" level in soil is then based on 90 percent of the diet represented by Pathway Five. The total BMF is equal to 90 percent of the BMF estimated by Pathway Five (0.0056), or 0.0050. For the terrestrial pathway, the MATC is divided by the BMF for the soil pathway to arrive directly at the "no effects" soil level as follows:

$$\frac{\text{MATC}}{\text{Total BMF}} = C_{soil} = \frac{0.74\ \text{ppm}}{0.0050} = 148\ \text{ppm} \qquad (6)$$

Since plants tend to be more susceptible to toxic effects as a result of arsenic exposure, soil criteria should also consider the relative phytotoxicity of arsenic to plants. The soil concentration that results in "no effects" to 50 percent growth reduction for various crop types in different soils ranges from 9 to 1,000 ppm total arsenic (Woolson, 1983), resulting in a geometric mean value of 52 ppm total soil arsenic as the level for protection of plants. When data were presented as a range, a median value was used as the point value to calculate the mean. Only data indicated as statistically significant were used. By applying K_d, concentrations of 52 ppm in soil result in 0.35 ppm in potential runoff water. This is slightly higher than the recommended level of 0.1 ppm in irrigation water.

The soil criterion can also be used to predict toxicity to small mammals exposed to contaminants from ingesting contaminated soil. An exposure rate as a function of the acceptable soil criteria can be estimated from the soil criterion and the soil ingestion rate for small mammals as follows:

Soil Criterion x Soil Ingestion Rate = Daily Exposure

52 mg/kg soil x 0.000873 kg soil/kg bw/day = 0.046 mg/kg bw/day

The exposure rate based on a soil criterion of 52 mg/kg soil is nearly three orders of magnitude lower than the observed toxic dose for small mammals, and therefore direct toxic effects are not expected at the criterion level of 52 mg/kg in soil. The daily intake of arsenic from ingesting soil represents a conservative estimate as an assimilation efficiency of 100 percent is assumed.

Because biomagnification of arsenic in the terrestrial food chain is less than 1, the terrestrial food web, with the American kestrel as the top carnivore, was not evaluated.

5.2.2.6 Uncertainty Analysis

In the uncertainty analysis, all of the intake rates (R values) and percent of items in diet are treated as triangular distributions where the minima

and maxima are known and a best estimate within that range has been determined. Using the triangular distribution as input, the best estimate will be more likely than values near either end of the range. Methodology for the uncertainty analysis is described in detail in the forthcoming Offpost Endangerment Assessment. Diets of each link on the sink food web are summarized in Table 5.2-18.

Several assumptions were made in order to conduct the analysis:

- The diet of the target organism, the bald eagle, is supplied only by the aquatic food chain, with ducks and pike the representative prey organisms; and
- Absorption, or assimilation, of ingested arsenic is assumed to be 100 percent.

Based on the available data, different k_2 values were reported for fish and birds. Woolson et al. (1976) conducted a model aquatic study in which catfish were exposed to a mixture of sodium arsenate and arsenic acid at 0.01, 0.10, and 1.0 parts per trillion (ppt) for 17 days prior to transferring to arsenic-free water for 14 days. Results from tissue analysis indicated that 75, 80, and 93 percent of the sorbed arsenic was flushed out of the catfish at the corresponding exposure levels, resulting in depuration rates of 0 054, 0.057, and 0.060 day^{-1}. Since only one study has been reported on only one species of fish, the spread in this data does not adequately represent the uncertainty in the estimate of depuration rate for several different species indigenous to the RMA aquatic ecosystems. To more realistically represent this uncertainty, reference is made to other indicator chemicals for the biota assessment, specifically DBCP, Dieldrin, and Endrin. For these chemicals, the standard deviation was generally 60 percent of the mean. By analogy it is assumed that the arsenic depuration rate in birds is of similar reliability. This is expressed as a log-normal distribution with a mean of 0.057 and standard deviation of 0.03.

Arsenic depuration rates in birds were estimated from a study with white leghorn hens (Overby and Fredrickson, 1965). Converting half-lives of 60 and 36-hr for arsenate and arsenic acid, respectively, in blood results in

Table 5.2-18. Dietary Input Factors, Pathways Analysis for Arsenic
R = Total Dietary Intake $(day)^{-1}$

	Minimum	Best Estimate	Maximum
Eagle	0.51	0.57	0.76
Mallard	0.45	0.52	0.93
Pike	0.01	0.03	0.05
Bluegill	0.01	0.03	0.05
Percent of Item in Diet			
Eagle/Mallard	14	28	42
Eagle/Pike	58	72	86
Mallard/Invertebrates	40	58	75
Mallard/Aquatic Plants	25	42	60
Bluegill/Plankton	6	12	18
Bluegill/Invertebrates	82	88	94

Source: ESE, 1988.

Table 5.2-20. Toxic Effects Levels of DBCP for Small Mammals and Birds by Ingestion

Species	Exposure Route	Dose	Effect	Acceptable Water Concentration (ppm)	Sources
Rat	Diet	0.5 mg/kg bw/day	No chronic effects on organ weights	0.8	1
	Diet	0.3 mg/kg bw/day	Toxic to kidney and liver; stomach tumors	0.096	2
	Drinking water	2 mg/kg bw/day	No observed subchronic effects	0.32	3
Mouse	Diet	0.3 mg/kg bw/day	Stomach tumors	0.06	2
Rabbit	Drinking water	0.94 mg/kg bw/day	No effects as measured by sperm morphology	0.11	4
Chicken	Oral	60 mg/kg bw	LD_{50}	0.048	1

Sources:

1. Torkelson et al., 1961.
2. Hazelton Laboratories, 1977, 1978.
3. Johnston et al., 1986.
4. Foote et al., 1986a, 1986b.

* Uncertainty factors have been applied (see Section 5.1).

is similar to toxicity due to exposure from water ingestion, an acceptable surface water concentration is derived using the following equation, where LOAEL is the lowest observed adverse effects level:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \text{Acceptable Surface Water Concentration}$$

Using 0.3 mg/kg bw/day as the LOAEL, and dividing it by daily water intake for mice, the following acceptable water concentration is derived:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{0.3\ \text{mg/kg bw/day}}{0.2\ \text{l/kg bw/day}} = 1.5\ \text{mg/l}$$

This value is divided by an uncertainty factor of 5 to bring the chronic LOAEL into the range of an NOEL (EPA, 1985b) and 5 for interspecific variation, to yield an acceptable water concentration of 0.06 mg/l (60 ppb).

For rats, the following water concentration is derived:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{0.3\ \text{mg/kg bw/day}}{0.125\ \text{l/kg bw/day}} = 2.4\ \text{mg/liter}$$

This value is then divided by an uncertainty factor of 5 to bring the chronic LOAEL into the range of an NOEL (EPA, 1985b) and 5 for interspecific variation, to yield an acceptable water concentration of 0.096 mg/l (96 ppb).

Table 5.2-20 lists the water intake concentrations that correlate with the toxic effects levels in diet or drinking water based on daily water intake for each species and the appropriate uncertainty factors (see Section 5.1). The lowest water concentration, 0.06 ppm (60 ppb), is used to represent a "no effects" water concentration for mammals.

Rats exposed to 100 ppm DBCP in water exhibited renal lesions (Heindel et al., 1983). Rats exposed to 20 mg/kg/day in drinking water had lower body weights and fetal birth weights than controls (Johnston et al., 1986); from an average water consumption of 125 ml/kg/day (Sax, 1984), an estimated DBCP concentration of 160

ppm is obtained. These water intake concentrations exceed the recommended level for mammals in water of 0.06 ppm.

Birds--The oral LD_{50} for chickens is the lowest value available for avian species. From the acute value for chickens and a surface water consumption rate for chickens (Sax, 1984), the acceptable water concentration is derived as follows:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{60\ \text{mg/kg bw/day}}{0.25\ \text{l/kg bw/day}} = 240\ \text{mg/l}$$

Applying an uncertainty factor of 1,000 to convert the LD_{50} to a chronic NOEL, and a factor of 5 for interspecific variation, yields an acceptable water concentration of 0.048 (48 ppb).

The value for birds (0.048 ppm) is lower than the value for mammals (0.06 ppm). However, because the value for birds was based on LD_{50}, more uncertainty is involved in the estimate. The acceptable water concentration based on chronic toxicity to mammals is thus considered to be the more appropriate value.

A DBCP concentration in surface water of 0.06 mg/l (60 ppb) is assumed to be protective of all species consuming water at RMA. The corresponding sediment concentration, based on a K_{oc} of 221 and foc of 0.0065, is 0.086 ppm.

5.2.3.3 Aquatic Life

Due to the lack of data regarding aquatic life, water criteria for the protection of aquatic life could not be estimated at this time.

5.2.3.4 Aquatic Pathway Analysis

Introduction to Aquatic Pathway Analysis

This Pathway Analysis is based on the bald eagle sink food subweb and includes all food chains leading to the selected sink species. Because the same organisms and groups of organisms appear in more than one food chain throughout the web, percentage contributions to the food subweb for each organism or compartment have

been estimated based on existing literature. The subweb has been simplified (e.g., bluegill represent all fish species at that trophic level), because of the limited data available.

The bald eagle was selected as the target species because of its federally endangered status and because it represents the highest trophic level affected by the bioaccumulation of contaminants through aquatic and some terrestrial food chains on RMA. Aquatic organisms are considered to be the most important links in the bald eagle food web because they are constantly exposed to the contaminants in their environment via surface adsorption, absorption, and uptake across respiratory membranes; thus, the potential for bioconcentration tends to be large.

The "no effects" level is based on sublethal effects levels obtained from the scientific literature and presumes that if bald eagles are protected, other species will also be protected. No safety factors have been used in the calculation of "no effects" levels.

Methods

There were no documented values for BCFs or BAFs available in the literature. BCFs estimated from regression equations from various sources ranged from 17.6 to 53 (Table 5.2-19). A geometric mean BCF of 31.6 was used in the Pathway Analysis to estimate the range of possible cleanup criteria for water, soil, and sediments.

K_{oc} is based on a measured value by Sabljic (1984) and regressions on K_{ow} and solubility by Lyman et al. (1982), Lyman and Loreti (1986), and Kadeg et al. (1986). K_{oc} follows a lognormal distribution with a mean of 221 and a standard deviation of 110.

Five potential food transfer pathways ultimately terminating with the bald eagle were established from the bald eagle food subweb as follows:

Pathway	Source	Trophic Level 1	2	3	4
1	H_2O	Invertebrates	Mallard	Bald Eagle	
2	H_2O	Aquatic Plants	Mallard	Bald Eagle	
3	H_2O	Plankton	Bluegill	Pike	Bald Eagle
4	H_2O	Invertebrates	Bluegill	Pike	Bald Eagle
5	Soil	Terrestrial Plants	Small Mammals	Bald Eagle	

Pathways are developed based upon biological and chemical specific parameters such as dietary habits, tendency of a species or group of organisms to accumulate a contaminant, and sensitivity of a given species to a particular contaminant. Because the data base for DBCP is limited, there are fewer pathways than for a contaminant with a detailed data base such as dieldrin. For example, for dieldrin, different types of invertebrates are observed to have different BCFs, and so can be differentiated into separate pathways by tendency to accumulate dieldrin. For DBCP, a mean bioconcentration factor is used to represent all organisms due to lack of data, so that separating the organisms into more than the basic pathways does not increase the sensitivity of the analysis.
The mallard (*Anas platyrynchos*) and the pike (*Esox lucius*) represent the waterfowl and fish fed upon directly by the bald eagle. Feeding habits for the consumer organisms are presented in Table 5.2-21. The combined food transfer pathways are presented in Figure 5.2-5.

All pathways (except Pathway Five) originate with water. The lowest step in the food chain is assumed to be in equilibrium with the aquatic environment, which gives equation (1):

$$BCF = C_b/C_w \qquad (1)$$

where: C_b = the concentration of DBCP in biota
C_w = the concentration of DBCP in water

This equation is vital to the rest of the analysis. The end result, the total BMF for the bald eagle, can be ultimately traced back through water to the sediment, because it is assumed that all DBCP enters the water compartment from sediments before being taken up by the biological compartment: i.e.,

$$C_w = \frac{C_{sed}}{K_{oc} \times f_{oc}} \qquad (7)$$

Table 5.2-21. Summary of Feeding Habits for DBCP Pathways Analysis

Species	Food Items	Percent in Diet	Reference
Mallard	Invertebrates[1]	44-56	Swanson et al., 1979 Swanson et al., 1985
	Plants, Fruits[2]	30-31	Swanson et al., 1979 Swanson et al., 1985
	Annelids[3]	26	Swanson et al., 1979
Bald Eagle	Waterfowl	24	Cash et al., 1985 Todd et al., 1982
	Fish	66	Cash et al., 1985
	Mammals	10	Cash et al., 1985
Bluegill	Invertebrates	88	Martin et al., 1961
	Plankton, Algae	12	Martin et al., 1961
Pike	Fish[4]	100	Inskip, 1982

1 Includes Crustacea, Insecta, and Mollusca.

2 Fruits were grouped with aquatic plants for this pathways analysis due to the possibility that DBCP is absorbed by fruit. The term "fruits" includes miscellaneous seeds (Swanson et al., 1979: Swanson et al., 1985).

3 These food items were not utilized in the pathways analysis. Annelids are apparently washed into aquatic systems (Swanson et al., 1979) and were not included, because areas upgradient from the RMA lakes are assumed to be uncontaminated.

4 Pike are opportunistic feeders that will utilize other food sources, but are assumed to prey completely on fish for the sake of the analysis.

Source: ESE, 1988.

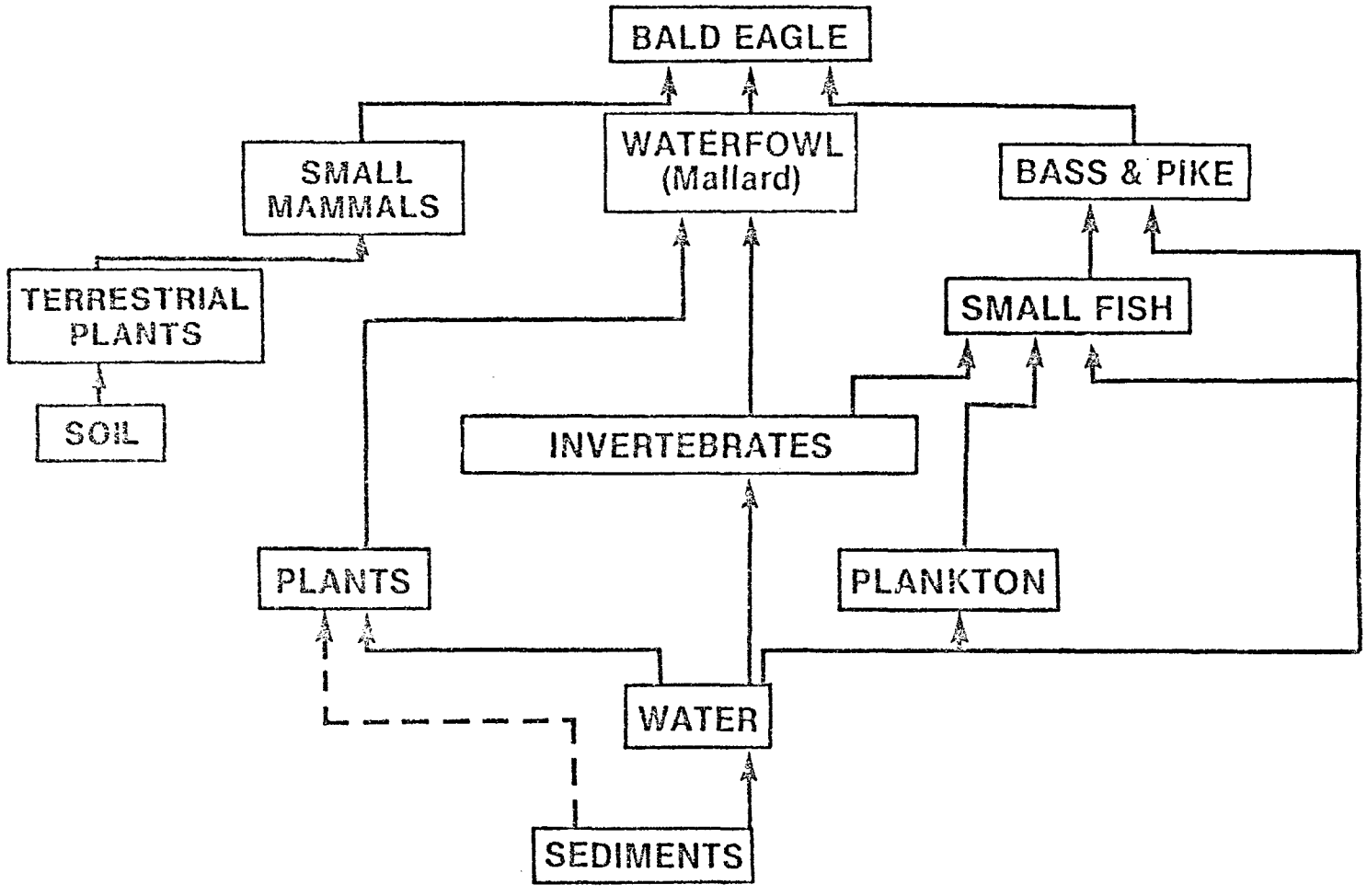


Figure 5.2-5

BALD EAGLE SINK FOOD WEB FOR DBCP

SOURCE: ESE, 1989

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

or solving for C_{sed}:

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \quad (8)$$

where: C_{sed} = concentration of DBCP in the sediment

C_w = concentration of DBCP in water

K_{oc} = soil-water partition coefficient normalized for organic carbon

f_{oc} = fraction of organic carbon

The method used in the Pathway Analysis is based on Thomann's (1981) bioaccumulation model of food chain transfer in aquatic ecosystems where each level is a step in the food chain, modified to address multiple food chains leading to a target organism:

Level #1 $BCF_1 = C_b/C_w$ (1)

Level #2 $BAF_2 = BCF_2 + f_2BCF_1$ (2)

Level #3 $BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1$ (3)

Level #4 $BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1$ (4)

The Level #1 equation represents the bottom of the food chain and is technically a non-feeding level where tissue contaminant concentration is a direct function of water concentration. For the purposes of the analysis, Level #1 was expanded to include low trophic level consumer organisms such as aquatic macroinvertebrates. It was assumed that bioconcentration by small organisms would outweigh concentration from diet to the extent that biomagnification of residues at Level #1 would be insignificant.

The food term (f_i) is a function of the trophic level in question and is calculated by the following equation:

$$f_i = \frac{@ \times R \times \%}{k_2} \quad (5)$$

where: @ = Assimilation efficiency, $\frac{\text{ug absorbed}}{\text{ug ingested}}$

R = Total Daily Intake, intake (g)/body weight (g)/day

k_2 = Depuration or loss rate, day^{-1}

% = Percent of item in diet

The assimilation efficiency was approximated from a study on metabolic fate in rats where over 99 percent of a dose was absorbed (Kato et al., 1979). The assimilation efficiency (@) could not be obtained for every animal addressed in this analysis; thus, it was assumed to be 1 for all animals.

The depuration or loss rate (k_2) includes loss due to growth, excretion, and metabolism. Because rate constants have not been measured for each species in this analysis, k_2 values derived from the literature were used to represent all species.

A k_2 value of 4.8/day was derived by graphical interpolation of data according to the method described in Spacie and Hamelink (1985). The data were provided by a study of pregnant rats (Ruddick and Newsome, 1979) using concentrations of DBCP in two tissues, adipose and lung, following oral administration of 25 mg/kg DBCP for 10 consecutive days. The loss rate was calculated from the time tissue concentrations reached maximum to final concentrations at end of experiment. This loss rate was applied to bird species due to the lack of data pertaining to loss rate in birds.

A duckling pathway was not constructed for DBCP. The lack of loss rates for birds makes the analysis highly uncertain, and applying the loss rate to growing birds may grossly overestimate the potential for DBCP accumulation in tissue.

A k_2 value of 3.4/day was provided using a regression equation correlating k_2 in aquatic organisms with log K_{ow} (Spacie and Hamelink, 1982), where log K_{ow} was 2.29 (Jaber et al., 1984). Since the regression equation was for fish, it was used to represent loss rates for all fish species in the Pathway Analysis.

Pathway Analysis

The Pathway Analysis model is applied in the following section using the input parameters BCF, k_2, and f_2 described in Section 3.2. The species specific dietary habits are described for each of the higher trophic levels.

Pathway One: H_2O => Aquatic Invertebrates => Mallard => Eagle--The BCF ranges from 17.6 to 53 depending on the regression equation used to calculate bioconcentration. Invertebrates are at Level #1 of the food chain:

bioconcentration is represented by the geometric mean BCF derived from regression equations in Table 5.2-19:

$$BCF_{invert} = 31.6 \qquad (1)$$

The food term (f_2) is calculated by assuming that an adult mallard weighs approximately 1,100 g and consumes about 57.4 g total diet each day (Miller, 1975), of which for a laying female, 44 to 56 percent of the diet is invertebrates other than annelids (Swanson et al., 1979). Actual quantities of invertebrates consumed by mallards fluctuate with season, and sex and breeding condition of mallard. Laying females consume more invertebrates, and are considered to be a sensitive subpopulation due to breeding stress. Because the lower end of the range for breeding females (44 percent) corresponded closely to a geometric mean of consumption by all mallards (including males and non-laying females) (Swanson et al., 1985), 44 percent was used to represent invertebrate intake for mallards in the pathway analysis. The BAF for a mallard is calculated by assuming that the first term in the Level #2 bioaccumulation equation (2) equals zero (because bioconcentration by nonaquatic organisms is considered to be negligible):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2 BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{1 \times (57.4\ g/1{,}100\ g/day) \times 44\%}{4.8/day} = 0.0048 \qquad (5)$$

When the BCF for aquatic invertebrates is 31.6, the BAF for the mallard is 0.15.

An adult eagle weighs approximately 4,500 g (Schafer, 1986) and consumes 255 g daily (Swies, 1986), of which 24 percent of the diet is birds (Cash et al., 1985; Sherrod, 1978). Energy requirements are different for wild birds than birds living in captivity, so these dietary quantities are only approximate (Jehnkins, 1986; Sherrod, 1986). The following BAF values for an eagle are calculated by assuming that the first two terms in the Level #3 bioaccumulation equation (3) equal zero (bioconcentration by the mallard and the eagle are both negligible):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$
$$BAF_{eagle} = f_3f_2BCF_{invert}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f_3 = \frac{1 \times (255\ g/4{,}500\ g/day) \times 24\%}{4.8/day} = 0.0028 \quad (5)$$

When the BCF for aquatic invertebrates is 31.6, the BAF for the eagle is 0.00042.

Pathway Two: H_2O => Aquatic Plants => Mallard => Eagle--Because the BCF for all aquatic life is estimated from a regression equation, the BCF for aquatic plants is the same as the BCF for aquatic invertebrates in Pathway One:

$$BCF_{plant} = 31.6 \quad (1)$$

To calculate the BAF for mallards, the food term f_2 remains the same as Pathway One except for the percent of the food item in the diet. Laying female mallards consume 30 to 31 percent plants and fruits or seeds, although actual amounts fluctuate with season. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$
$$BAF_{mallard} = f_2BCF_{plant}$$

where: $BCF_2 = 0$

$$f_2 = \frac{1 \times (57.4\ g/1{,}100\ g/day) \times 30\%}{4.8/day} = 0.0033 \quad (5)$$

When the BCF for aquatic plants is 31.6, the BAF for mallard is 0.10.

The food term for the consumption of mallards by the eagle, f_3, remains the same as the Pathway One equation. The BAF is calculated using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$
$$BAF_{eagle} = f_3f_2BCF_{plant}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{1 \times (255\ g/4{,}500\ g/day) \times 24\%}{4.8/day} = 0.0028 \qquad (5)$$

When the BCF for aquatic plants is 31.6, the BAF for eagle is 0.00029.

Pathway Three: $H_2O \Rightarrow$ Plankton $\Rightarrow$ Bluegill $\Rightarrow$ Pike $\Rightarrow$ Eagle--Pathways leading to the bald eagle via fish are more complex because bioconcentration occurs at each trophic level, not only at the lowest trophic level. This introduces a fourth factor into the BAF equation, and the eagle is at Level #4 instead of Level #3. The geometric mean from the regression equations in Table 5.2-19 was used to represent bioconcentration as in the previous pathways:

$$BCF_{plankton} = 31.6 \qquad (1)$$

The BCF for the bluegill (Lepomis macrochirus) is from the same set of regression equations:

$$BCF_{bluegill} = 31.6 \qquad (1)$$

It is assumed that bluegill consume a dietary intake equal to 3 percent of their body weight daily (Chadwick and Brocksen, 1969): the total daily intake term is then a ratio of 0.03 regardless of bluegill body weight. Various algal forms account for approximately 12 percent of the bluegills' diet (Martin et al., 1961): this value was used for the percent of plankton in the bluegill diet. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{plankton}$$

$$\text{where: } f_2 = \frac{1 \times (0.03/day) \times 12\%}{3.4/day} = 0.0011 \qquad (5)$$

The BCF for the pike is from the same set of regression equations as bluegill (Table 5.2-19):

$$BCF_{pike} = 31.6 \qquad (1)$$

It is assumed that pike also consume a dietary intake equal to 3 percent of their body weight daily (Chadwick and Brocksen, 1969); the total daily intake term is then a ratio of 0.03 regardless of pike body weight, or the same ratio as for bluegill. It is assumed that pikes feed entirely on bluegills for the sake of this analysis. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{plankton}$$

$$\text{where: } f_3 = \frac{1 \times (0.03/day) \times 100\%}{3.4/day} = 0.0088 \quad (5)$$

The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g food daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{plankton}$$

$$\text{where: } BCF_4 = 0$$

$$f_4 = \frac{1 \times (255\ g/4{,}500\ g/day) \times 66\%}{4.8/day} = 0.0078 \quad (5)$$

When the BCF for plankton is 31.6, the BAF values for bluegill and pike are 31.6 and 31.9, respectively. The BAF for eagle is 0.25.

Pathway Four: H_2O => Aquatic Invertebrates => Bluegill => Pike => Eagle--The range of values used to represent bioconcentration in aquatic invertebrates is the same as the previous pathways:

$$BCF_{invert} = 31.6 \quad (1)$$

The range of values used to represent bioconcentration for bluegill is the same as the previous pathways:

$$BCF_{bluegill} = 31.6 \quad (1)$$

It is assumed that bluegill consume a dietary intake equal to 3 percent of their body weight daily (Chadwick and Brocksen, 1969); the total daily intake term is then a ratio of 0.03 regardless of bluegill body weight. Invertebrates are

believed to account for 88 percent of the bluegills diet (Martin et al., 1961). Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{invert}$$

$$\text{where: } f_2 = \frac{1 \times (0.03/day) \times 88\%}{3.4/day} = 0.0078 \quad (5)$$

The BCF for the pike was also assumed to be represented by the geometric mean of the regression equations in Table 5.2-19:

$$BCF_{pike} = 31.6 \quad (1)$$

It is assumed that pike also consume a dietary intake equal to 3 percent of their body weight daily (Chadwick and Brocksen, 1969); the total daily intake term is then a ratio of 0.03 regardless of pike body weight, or the same ratio observed in bluegill. It is assumed that pikes feed entirely on bluegills for the sake of this analysis. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{invert}$$

$$\text{where: } f_3 = \frac{1 \times (0.03/day) \times 100\%}{3.4/day} = 0.0088 \quad (5)$$

The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g food daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{invert}$$

$$\text{where: } BCF_4 = 0$$

$$f_4 = \frac{1 \times (255\ g/4{,}500\ g/day) \times 66\%}{4.8/day} = 0.0078 \quad (5)$$

When the BCF for aquatic invertebrates is 31.6, the BAF values for bluegill and pike are 31.8 and 31.9, respectively. The BAF for eagle is 0.25.

Results and Discussion

BAF values as derived for the individual pathways (Table 5.2-22) represent accumulation in separate single food chains. To derive overall accumulation in the entire food web, variations of the following equation are used:

$$BMF_i = BCF_i + f_i BAF_{i-1}$$

For each of the major trophic levels in the aquatic Pathway Analysis, total biomagnification is presented in Table 5.2-23.

The total BMF was not rounded to the nearest whole number due to the low values observed. Total BMF can be used to determine maximum allowable levels of DBCP in sediments and soils by relating sediment concentration to a MATC as follows (Tucker, 1986):

$$\frac{MATC}{\text{Total BMF}} = C_w \qquad (6)$$

and,

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \qquad (8)$$

where: K_{oc} = 221 (Sablijic, 1984; Lyman et al., 1982; Lyman and Loreti, 1986; Kadeg et al., 1986)

f_{oc} = 0.0065 (EBASCO, 1988)

The MATC is obtained by examining the literature for the lowest tissue concentration which results in sublethal or lethal toxic effects:

SPECIES	TISSUE	PPM	EFFECT	SOURCE
Rat	adipose	0.17±0.071	decreased fetal and maternal weight gain	Ruddick and Newsome, 1979

Because maximum DBCP accumulation occurred in adipose tissue, and residues remained at 24 hr post-treatment (the time of the next dosing), adipose was used as the reference tissue. In the Ruddick and Newsome (1979) study,

Table 5.2-22. Summary of Bioaccumulation Factors for each Species in the DBCP Pathways Analysis

	Bioaccumulation Factors				
	Bluegill	Pike	Mallard	Mammal	Eagle
Pathway 1	--	--	0.15	--	0.00042
Pathway 2	--	--	0.10	--	0.00029
Pathway 3	31.6	31.9	--	--	0.25
Pathway 4	31.8	31.9	--	--	0.25
Pathway 5	--	--	--	0.0032	3.0×10^{-7}

Source: ESE, 1988.

Table 5.2-23. Total Biomagnification of DBCP Residues for each of the Key Organisms in the Aquatic Pathways Analysis.

Organism	Level	Equation	BMF
Mallard	#2	$\Sigma f_2 BCF_1$	0.25
Bluegill	#2	$BCF_2 + \Sigma f_2 BCF_1$	31.88
Pike	#3	$BCF_3 + f_3 BMF_{bluegill}$	31.88
Eagle	#3, #4	$f_4 BMF_{pike} + f_3 BMF_{mallard} + BMF_{terrestrial}$	0.25

Source: ESE, 1988.

toxic effects occurred following 10 consecutive daily doses with 25 mg/kg DBCP, at which time tissues were analyzed for residues. Data regarding tissue residues were unavailable for avian or mammalian wildlife species in the literature surveyed.

The lowest tissue concentration at which toxic effects are observed was divided by the BMF for eagle from Table 5.2-23, then corrected with K_{oc} and f_{oc}; thus, giving the sediment concentration at which no effects to higher trophic levels are likely to occur.

Using equations (6) and (8) to obtain the "no effects" level of DBCP in water and sediments for eagle based on accumulation from the abiotic environment and transfer through the food web:

$$\frac{MATC}{\text{Total BMF}} = C_w = \frac{0.17 \text{ ppm}}{0.25} = 0.68 \text{ ppm} \qquad (6)$$

$$C_{sed} = C_w \times K_{oc} \times f_{oc} = 0.68 \text{ ppm} \times 221 \times 0.0065 = 0.98 \text{ ppm} \qquad (8)$$

The acceptable water concentrations derived by the Pathway Analysis for the bald eagle are higher than those derived based on water consumption only (0.06 ppm). Therefore, the surface water pathway represents the best estimate of an acceptable water concentration for DBCP, with a corresponding sediment concentration, calculated using K_{oc} and f_{oc} as above, of 0.086 ppm.

Due to the high loss rate of DBCP from tissues, accumulation within a food chain does not appear to be a problem to high level predators. BCFs than than 100 indicate concentration of residues (ASTM, 1985), and BMFs less than 1 indicate actual reduction of residues with increasing trophic levels.

Although evidence does not suggest food chain accumulation of DBCP, toxic effects could occur if food items contained high concentrations of DBCP. Rats exposed to 0.3 mg/kg bw/day DBCP in diet exhibited carcinogenic effects (Hazelton Laboratories, 1978). This is roughly equivalent to a total daily dietary dose of 1.35 mg for a 4.5 kg eagle. If the eagle consumes 0.255 kg of diet (255 g diet/day), the dietary concentration becomes 1.35 mg/0.255 kg diet, or 5.3 ppm. Since this is a chronic LOAEL, it should be reduced by an

uncertainty factor of 5 to bring the chronic LOAEL into the range of a chronic NOEL, and an uncertainty factor of 5 for interspecific variation. For the protection of eagles at RMA, food items such as prairie dogs should not contain concentrations of DBCP exceeding 0.212 ppm.

5.2.3.5 Terrestrial Pathway Analysis

Introduction to Terrestrial Pathway Analysis

This analysis was performed to determine cleanup criteria for DBCP in a terrestrial based food web (soil-biota) pathway. The approach used for the terrestrial pathway analysis arrives at a "no effects" level in soil of terrestrial ecosystems on RMA by assuming that soils are a source of DBCP contamination.

Methods for Terrestrial Pathway Analysis--- Bald Eagle Food Web

The terrestrial based food chain in the bald eagle food web was analyzed to determine concentration of DBCP from soil to the target organism. The total BMF for the terrestrial food chain was used to derive a soil criterion for DBCP.

Pathway Five: Soil => Terrestrial Plants => Mammals => Eagle--DBCP has been observed to accumulate in some root crops (Newsome et al., 1977). In other crops, increased inorganic bromide residues were observed, but not residues of the parent compound or organic metabolites (Castro and Schmitt, 1962; Beckman and Bevenue, 1963; Guinn and Potter, 1962).

The ratio of DBCP in plant tissue as compared to soil, the EMF, is estimated from Newsome et al. (1977) where highest residues were observed in carrot tops 7 weeks after seeding (EMF = 1.8), and lowest residues were observed in carrot tops 16 weeks after seeding (EMF = 0.036). Only data for DBCP treatment at time of seeding were used, because it was assumed that this would be more representative of exposure at RMA. Concentration factors in roots and foliage of carrot and radishes were averaged over time for two crop types, to obtain one value each for root and foliage for each crop. These four values were then averaged to obtain a geometric mean representative of concentration in plants over the duration of a growing season (Table 5.2-24):

Table 5.2-24. Concentration Factors Over Time for Carrot and Radish Crops When DBCP Was Applied To Soil at Seeding

CARROT	ROOT	TOP
Week 7	1.43	1.80
8	1.23	0.443
9	0.615	0.183
10	0.178	0.032
11	0.848	0.197
12	0.276	0.099
13	0.278	0.141
14	0.124	0.112
15	0.047	0.019
16	0.076	0.036
Geometric Mean	0.298	0.126

RADISH	ROOT	TOP
Week 4	0.850	0.390
5	0.357	0.053
6	0.818	0.444
7	1.49	0.388
Geometric Mean	0.780	0.244

Concentration Factor = $\frac{C_B}{C_{soil}}$

Source: Newsome et al., 1977.

$$EMF_{plant} = \frac{C_B}{C_{soil}} = 0.29 \qquad (10)$$

where: C_B = the concentration of DBCP in biota
C_{soil} = the concentration of DBCP in soil

No studies were found in the literature surveyed where dietary levels of DBCP were compared to tissue residues in animals. It is assumed for the purposes of the analysis that uptake following exposure in diet will resemble uptake following oral gavage. One study (Ruddick and Newsome, 1979) presented data for various tissue residues in rats following daily dosing by oral gavage. At 6 hr after dosing with 25 mg/kg, residues in adipose tissue reached a maximum of 5.38 ± 0.41 ppm standard error (SE), or a maximum concentration ratio of 0.22. At 24 hr post-treatment, residue levels in fat had decreased to 0.17 ± SE 0.071 ppm, or a concentration ratio of 0.0068. Only the 24-hr value was used in calculating the mean, because residues were decreasing rapidly at the other times, and dosing occurred at 24-hr intervals, so that this point approximates equilibrium with daily exposures.

Kenaga (1980) presents regression equations derived from observations with various organic chemicals correlating magnification from diet with log K_{ow} or log S:

$$\log BMF = (-1.476 - 0.495 \log S);\ r^2 = -0.82$$
$$\log BMF = (-3.457 + 0.500 \log K_{ow}):\ r^2 = 0.79 \qquad (9)$$

where: S = 1,230 mg/L
log K_{ow} = 2.29

Using these equations, the biomagnification estimate ranges from 0.00099 (based on log S) to 0.0049 (based on log K_{ow}). A geometric mean value based on the concentration factors 0.00099, 0.0049, and 0.0068 (N = 3) was used to represent biomagnification in small mammals, or a BMF of 0.0032. It is assumed that this BMF will account for instances when a small mammal is consumed by a predator within a short time of a DBCP exposure. Actual

magnification of residues from diet may differ. A BMF of 0.0032 was also used to represent biomagnification by eagle, as no pertinent data were available in the literature.

The terrestrial pathway is thus:

$$\underset{\text{soil} \rightarrow \text{plants}}{0.29} \times \underset{\text{plants} \rightarrow \text{mammals}}{0.0032} \times \underset{\text{mammals} \rightarrow \text{eagle}}{0.0032} = \text{Total BMF} = 3.0 \times 10^{-6}$$

Multiplying the concentration factors at each trophic level results in a total BMF for the terrestrial food chain. The total BMF for the eagle based on consumption of contaminated plants by small mammals is 3.0×10^{-6}. The consumption of small mammals makes up 10 percent of the eagles diet: therefore, the contribution from the terrestrial pathway is 10 percent of 3.0×10^{-6}, or 3.0×10^{-7}.

Because biomagnification in the terrestrial food chain of the bald eagle food web is less than 1, a terrestrial food web, based on the American kestrel as the top carnivore, will not be constructed for DBCP.

Results and Discussion

For the single terrestrial pathway in the aquatic based food web, the MATC is divided by the BMF to arrive directly at the "no effects" soil level as follows:

$$\frac{\text{MATC}}{\text{Total BMF}} = C_{soil} = \frac{0.17 \text{ ppm}}{3.0 \times 10^{-7}} = 570{,}000 \text{ ppm} \qquad (6)$$

Observed winter feeding behavior by eagles at RMA indicates that Pathway 5 forms more than 10 percent of the diet. Approximately 90 percent of the eagles' diet at RMA is composed of small mammals, such that the total BMF for Pathway 5 is 90 percent of 3.0×10^{-6}, or 2.7×10^{-6}. The "no effects" soil level becomes:

$$\frac{\text{MATC}}{\text{Total BMF}} = \frac{0.17 \text{ ppm}}{2.7 \times 10^{-6}} = 63{,}000 \text{ ppm} \qquad (6)$$

Because DBCP does not tend to bioaccumulate, very high levels in the abiotic environment may not produce adverse biological effects when considering toxicity due to food chain transfer of residues. For this reason, the pathways approach is not the appropriate method for use to establish soil criteria for DBCP. At these levels of DBCP in soil, the soil faunal community would be devastated, thereby resulting in severe food chain disruptions. Because soil toxicity to plants was unavailable, a suggested criteria based on levels applied for invertebrate pest control is recommended as a criterion soil level for DBCP. The lowest level of DBCP applied to soil for pest control in the available literature was 12.26 lb/acre (Newsome et al., 1977). Using a conversion factor of 2 million lb soil/6 inch acre (Korschgen, 1970), a maximum soil concentration of 6.10 ppm is derived.

The soil criterion can also be used to predict toxicity to small mammals exposed to contaminants from ingesting contaminated soil. An exposure rate as a function of the acceptable soil criteria can be estimated from the soil criterion and the soil ingestion rate for small mammals as follows:

Soil Criterion x Soil Ingestion Rate = Daily Exposure

6.13 mg/kg soil x 0.000873 kg soil/kg bw/day = 0.0054 mg/kg bw/day

The exposure rate based on a soil criterion of 6.13 mg/kg soil is two orders of magnitude lower than the observed chronic LOAEL for small mammals, and therefore direct toxic effects are not expected at the criterion level of 6.13 mg/kg in soil. The daily uptake of DBCP from ingesting soil represents a conservative estimate as an assimilation efficiency of 100 percent is assumed.

5.2.3.6 Uncertainty Analysis

In the uncertainty analysis, all of the intake rates (R values) and percent of items in diet are treated as triangular distributions where the minima and maxima are known and a best estimate within that range has been determined. Using the triangular distribution as input, the best estimate will be more likely than values near either end of the range. Methodology

for the uncertainty analysis is described in detail in the forthcoming Offpost Endangerment Assessment. Diets of each link on the sink food web are summarized in Table 5.2-25.

Several assumptions were made in order to conduct the analysis:

- The diet of the target organism, the bald eagle, is supplied only by the aquatic food chain, with ducks and pike the representative prey organisms; and
- Absorption, or assimilation, of ingested DBCP is assumed to be 100 percent.

Uncertainty in depuration rates could be estimated only after additional research and investigation of the literature and reports of other organic chemicals on RMA. An independent statistical analysis of the reliability of the Spacie and Hamelink (1982) correlation of log k_2 with log K_{ow} was performed. The Spacie and Hamelink equation was used to estimate depuration in aquatic organisms, and has a standard error in prediction of $\log_{10} k_2$ of 0.19 log units, assuming log K_{ow} is known precisely. However, log K_{ow} is based on estimates using the fragment constant method, where the most reliable available estimate is 2.29 (Jaber et al., 1984), which is used in the Superfund Public Health Evaluation Manual. According to data presented by Lyman et al. (1982), standard error of the fragment constant method is approximately 0.24 $\log_{10}$ units. Applying standard techniques for propagation of error through a series of calculations, it was determined that k_2 (depuration rate) for DBCP in aquatic biota follows a lognormal distribution with a mean of 3.4 day^{-1} and a standard deviation of 1.2 day^{-1}.

The k_2 for rats (4.8 day^{-1}) is a measured value, and was used to represent the loss rate in birds. No data exist for birds, and it was assumed that loss in mammals more clearly represents loss in birds than would application of the Spacie and Hamelink equation based on fish. Therefore, the best estimate of loss in birds is 4.8 day^{-1}. A triangular distribution is proposed relying on the standard deviation developed above, 1.2 day^{-1}. Using the mean developed for the aquatic pathways (3.4 day^{-1}) to calculate the low end of the range yields a larger uncertainty, and since the loss rate is for mammals as opposed to birds the estimate is considered somewhat

Table 5.2-25. Dietary Input Factors, DBCP Pathways Analysis
R = Total Dietary Intake $(day)^{-1}$

	Minimum	Best Estimate	Maximum
Eagle	0.51	0.57	0.76
Mallard	0.45	0.52	0.93
Pike	0.01	0.03	0.05
Bluegill	0.01	0.03	0.05
Percent of Item in Diet			
Eagle/Mallard	14	28	42
Eagle/Pike	58	72	86
Mallard/Invertebrates	40	58	75
Mallard/Aquatic Plants	25	42	60
Bluegill/Plankton	6	12	18
Bluegill/Invertebrates	82	88	94

Source: ESE, 1988.

uncertain. This yields a range of 2.2 (3.4 - 1.2) to 6.0 (4.8 + 1.2) for the estimate of loss rate in birds.

For BCF, a lognormal distribution with a mean of 25 and a standard deviation of 10 was applied. This is based on the measured solubility of 1,230 mg/l (5,210 umol/l), the previously reported log K_{ow}, and regressions presented by Davies and Dobbs (1984), Lyman et al. (1982), and Oliver and Niimi (1983). Other sources prior to 1982 were considered to be superceded by the Lyman et al. (1982) critical review of the prior literature.

K_{oc} is based on a measured value by Sabljic (1984) and regressions on K_{ow} and solubility by Lyman et al. (1982), Lyman and Loreti (1986), and Kadeg et al. (1986). K_{oc} follows a lognormal distribution with a mean of 221 and a standard deviation of 110.

Organic carbon content of the sediment of the RMA lakes is a measured value (EBASCO, 1988). In the upper 1 foot (ft) of sediment, organic carbon appears to follow a lognormal distribution with a mean of 0.65 percent and a standard deviation of 0.62 percent.

The median estimate of BMF from the uncertainty analysis is 0.253. There is a 5 percent chance that the eagle BMF is as high as 0.658 or as low as 0.097. The best estimate of the water concentration that would not produce adverse biological effects is 583 ppb, with upper and lower bounds of 1,942 and 212 ppb, respectively. The best estimate of the sediment concentration that would not result in tissue levels related to adverse biological effects is 665 ppb, with upper and lower bounds (95 percent confidence interval) of 3,589 and 125 ppb, respectively.

To perform the uncertainty analysis for the terrestrial component of the aquatic Pathway Analysis, the following assumptions were made:

- The diet of the target organism, the bald eagle, is supplied only by the terrestrial food chain, with small mammals the representative prey species; and
- The uptake parameters (BAFs) follow a lognormal distribution.

For plant uptake, a best estimate of the EMF was a lognormal distribution with a mean of 0.137± SD 0.124. For uptake by mammals, the three information sources were weighted according to the precision of the data (Beers, 1953), such that observed values (Ruddick and Newsome, 1979) were weighted higher than values from regression equations with low r^2 values (Kenaga, 1980). For mammal uptake from diet, the best estimate of the BAF was a lognormal distribution with a mean of 0.0062 ± 0.0023 SD.

The best estimate of the total BMF for bald eagle terrestrial food chain, based on the uncertainty analysis, is 3.0×10^{-6}. The 95 percent confidence interval has lower and upper bounds of 6.5×10^{-7} and 1.4×10^{-5}, making the best estimate good to approximately one order of magnitude. The best estimate of the soil criteria is 57,000 ppm, with lower and upper bounds of 10,800 and 300,000, respectively.

5.2.3.7 Summary and Conclusions

DBCP does not bioaccumulate from the soil or water environment to biota to any significant extent. Biomagnification of DBCP within a food chain does not appear to be a problem.

At this time, no relevant criteria exist for DBCP with respect to wildlife, and EPA water criteria have not been developed for protection of aquatic life. The "no effects" water and sediment concentrations based on the Pathway Analysis are 0.68 ppm (680 ppb) and 0.98 ppm, respectively. The "no effects" level in soil derived by the Pathway Analysis is 63,000 ppm based on observed feeding behavior by eagles at RMA; however, a better estimate is derived by using the lowest level applied for invertebrate pest control (6.10 ppm).

Surface water ingestion provides a lower estimate of an acceptable water concentration (0.06 ppm, with corresponding sediment concentrations of 0.086 ppm) than that determined by the Pathway Analysis. Therefore, acceptable water concentrations should be based on estimates of toxicity from surface water consumption.

A summary of the site-specific criteria is as follows:

Method	Water (ppb)	Sediment (ppm)	Soil (ppm)
Aquatic Life	NA	NA	NA
Aquatic Pathways Analysis	680	0.98	NA
Water Ingestion	60	0.086	NA
Terrestrial Pathways Analysis--Eagle	NA	NA	NA
Toxicity to Soil Fauna	NA	NA	6.10

5.2.4 PATHWAY ANALYSIS FOR ENDRIN/ISODRIN

5.2.4.1 Background Information

While isodrin and endrin are less stable in the environment than their isomers aldrin and dieldrin (Matsumura, 1980), they are persistent compounds and are slow to biodegrade EPA (1987c). Endrin has the potential to bioaccumulate (EPA, 1987c). Values obtained for endrin using the Pathway Analysis approach were also assumed to represent its isomer, isodrin, because isodrin converts to endrin under biological conditions, and the toxicity of both chemicals is similar (Matsumura, 1980).

Toxicity of Endrin

Endrin is highly toxic to organisms in both aquatic and terrestrial ecosystems. The ambient water quality criteria for protection of aquatic life for endrin are 0.0023 ppb as a 24-hr average and 0.18 ppb as a maximum concentration (EPA, 1980c; EPA, 1986d). Toxic effects are possible through food chain contamination (Stickel et al., 1979). The EPA water quality criterion of 0.0023 ppb is based on a Final Residue Value calculated with human guidelines and so is not considered appropriate to this analysis.

Aquatic Plants--Toxic effects have been observed in green algae (Scenedesmus quadricauda and Oedogonium spp.) at concentrations exceeding 20 ppm (Vance and Drummond, 1969). The LOAEL for plants is 475 ppb, based on observations

of growth inhibition in an alga, Anacystis nidularas (EPA, 1980c). Other studies indicate growth inhibition in algae from greater than 1,000 ppb to greater than 20,000 ppb (EPA, 1980c).

Aquatic Invertebrates--Endrin resulted in behavior changes ' caddis-fly (Brachycentrus americanus) and stonefly (Pteronarcys dorsata) larva within 4 days at concentrations of 0.07 ppb for B. americanus and 0.15 ppb for P. dorsata (Anderson and DeFoe, 1980). Behavioral effects included spontaneous twisting and altered swimming patterns by stoneflies, and evacuation of cases by caddisflies. The 28-d LC_{50}s were less than 0.03 ppb for B. americanus and 0.07 ppb for P. dorsata.

Aquatic Vertebrates--The 96 hour (96-hr) LC_{50} for endrin is less than 1 ppb for many species of freshwater fish (EPA, 1979b). Surface waters in the lower Mississippi River basin during a period when numerous fish kills were reported contained a recorded maximum of 0.214 ppb (EPA, 1979b). The 24-hr and 48-hr LC_{50} values for bass fingerlings were 0.49 and 0.27 ppb, respectively (Fabacher, 1976). The 28-d LC_{50} for bullhead (Ictalurus melas) was 0.10 ppb (Anderson and DeFoe, 1980).

Endrin was acutely toxic to channel catfish (Ictalurus punctatus) at concentrations of 0.25 to 0.30 ppb for a 10-d exposure period (Mount et al, 1966). Blood levels diagnostic of endrin poisoning in catfish were 0.3 ppm. Catfish exposed to 0.1 or 0.2 ppb for 44 days showed CNS effects, with corresponding mean blood levels of 0.18 ppm (range 0.11 to 0.25 ppm) and 0.25 ppm (range 0.20 to 0.28 ppm), respectively.

Sharma et al. (1979) observed a 96-hr LC_{50} of 33 ppb for Ophiocephalus punctatus (a type of Indian fish) in static tests. The NOEL was 5 ppb for 96-hr. At 80 ppb, mortality was 100 percent. Effects of exposure resulted in decreased activity of several enzymes (succinic, pyruvic, and lactic dehydrogenases) that function in cellular metabolic activity or energy release. The LC_{50} values derived by Sharma et al. (1979) are higher than other values derived for fish, possibly because static tests can underestimate the acute toxicity of endrin to fish (EPA, 1980c).

In male goldfish (Carassius auratus) dietary endrin at low doses of 0.99, 3.3, and 9.9 ppm (4.3, 14.3, and 43 ug/kg bw/day) for 157 days caused no adverse effects; stimulatory effects such as increased growth rate were observed (Grant and Mehrle, 1970). High doses of 33 and 99 ppm (143 and 430 ug/kg bw/day) resulted in decreased growth rates, decreased amounts of body fat, decreased gametogenesis, and increased mortality. Thyroid activity was decreased in the 33 and 99 ppm groups, and serum sodium (Na) levels were elevated at all dose levels except the highest. CNS effects such as altered respiration rate, hypersensitivity to stimuli, and convulsions were observed in the high dose groups.

Frogs are not as sensitive to the effects of endrin as are many fish (Hall and Swineford, 1980). The 96-hr LC_{50} values by static test methods for Pseudacris triseriata and Bufo woodhousei tadpoles were 120 and 180 ppb, respectively (Sanders, 1970). Observed LC_{50} values by flow-through bioassay for Rana sphenocephala resulted in lower LC_{50} values than those obtained by static test methods (Hall and Swineford, 1980); LC_{50} values by flow-through bioassay for larvae and transformed frogs ranged from a measured concentration of endrin in water of 0.002 to 0.011 ppm (2 to 11 ppb).

Terrestrial Plants--Terrestrial plants are relatively insensitive to the toxic effects of endrin, but can absorb, translocate, and metabolize endrin (EPA, 1979b). Absorption varies with species, soil type, and endrin concentration. Uptake of plant macro- and micronutrients is altered by exposure to high soil concentrations of endrin. Endrin exposure resulted in a decreased rate of germination and variations in tissue amino acid composition. A 500 ppm concentration of endrin for a 24-hr exposure period resulted in 50 percent growth inhibition (GR_{50}) of barley seed (Hordeum vulgare) (EPA, 1979b).

Birds--The acute LD_{50}s for endrin in female mallard ducks (Anas platyrhynchos), sharp-tailed grouse (Tympanuchus cupido), and California quail (Lophortyx californicus), are 5.64, 1.06, and 1.19 mg/kg bw, respectively (Hudson et al., 1984). For male ring-necked pheasant (Phasianus colchicus), the acute oral LD_{50} is 1.78 mg/kg bw (Hudson et al.,

1984). For male and female rock doves (*Columba livia*) the acute oral LD_{50} is 2.0 to 5.0 mg/kg bw (Hudson *et al.*, 1984). The 5-day dietary LC_{50} values for bobwhite quail (*Colinus virginianus*), coturnix quail (*Coturnix coturnix*), ring-necked pheasant (*Phasianus colchicus*), and mallard ducks were 14, 18, 14, and 22 ppm, respectively (Heinz and Johnson, 1979).

Screech owls (*Otus asio*) fed 0.75 ppm endrin in diet for 83 days laid fewer eggs per day and had fewer eggs hatch per clutch than controls (Fleming *et al.*, 1982). There were fewer fledglings per total number of pairs and 43 percent fewer fledged owlets than controls. Although residue concentration did not correlate significantly with hatching success, a trend was observed that clutches with higher residues had lower hatching success. A concentration of 0.3 ppm in eggs was indicated as a threshold level for toxic effects (Fleming *et al.*, 1982). However, PCB and DDE residues were found in higher concentrations in treated as compared to controls, possibly affecting study results.

Mallard ducks were chronically exposed to endrin over a period of approximately 7 months by feeding 0, 1, and 3 ppm endrin in diet (Spann *et al.*, 1986). Birds receiving 1 ppm in diet had higher reproductive success than controls, while birds receiving 3 ppm in diet had reduced reproductive success, although the difference was not statistically significant. In another study with mallard ducks, endrin fed to breeding birds at 0.5 and 3.0 ppm in diet did not affect egg production, fertility, or hatchability, although a 9.6 percent decrease in embryo survivability was observed at the 3.0 ppm dose level (Roylance *et al.*, 1985). Males on the 3.0 ppm diet lost weight.

Brain residues diagnostic of endrin poisoning in birds are concentrations exceeding 0.8 ppm, while concentrations less than or equal to 0.6 ppm indicate survival (Stickel *et al.*, 1979). In a study by Spann *et al.* (1986), a male mallard that died while receiving 3 ppm endrin in diet had 1 ppm endrin on a wet weight basis in brain tissue. A male and a female bald eagle that survived for 13 and 20 days at 20 ppm endrin in diet, died

with brain residues of 1.2 and 0.92 ppm (wet weight basis), respectively (Stickel *et al.*, 1979). Lipid levels decreased although the eagles continued to feed regularly until death.

The dermal LD_{50} of endrin to 10 month old male mallards is greater than 140 mg/kg bw after a 24-hr foot exposure to a 97 percent solution (Hudson *et al.*, 1984). CNS symptoms such as hyperexcitability and ataxia were observed within 3-hr of treatment. Exposure resulted in mild dermal irritation.

Mammals--The acute oral LD_{50} values for endrin for several mammalian species range from 1.37 to 50 mg/kg bw (EPA, 1980c). Mice and monkeys are the most sensitive mammals, and guinea pigs and goats are the most resistant. The LD_{50} for female mule deer (*Odocoileus hemionus*) (N = 3) is 6.25 to 12.5 mg/kg bw (Hudson *et al.*, 1984). The acute oral LD_{50} values for isodrin for rats and mice are 8.8 and 7 mg/kg bw, respectively (NIOSH, 1982).

Endrin was fatal to dogs exposed to dietary concentrations of 0.49 to 0.81 ppm (0.012 to 0.020 mg/kg bw/day (Sax, 1984)) for 5 to 6 months (EPA, 1980c), making endrin more chronically toxic than the other organochlorine pesticides. Chronic exposure to low concentrations can produce damage to major organs such as the liver, kidney, and heart; nervous system disorders have also been reported (EPA, 1980c). Sublethal effects are observed in wildlife populations such as altered behavioral and reproductive function (EPA, 1980c).

After one week, rats dosed daily with 3.5 mg/kg bw exhibited electroencephalograph (EEG) changes and convulsions (Speck and Maaske, 1958), whereas the NOEL was less than or equal to 1.7 mg/kg bw/day. At 0.75 mg/kg/day administered to female hamsters at days 5 through 14 of gestation, endrin resulted in increased fetal mortality, decreased fetal weight, and decreased skeletal ossification (Chernoff *et al.*, 1979).

Age and sex can influence the toxicity of endrin to mammals (Blus, 1978). The LD_{50} values for 30-day old female and male rats were 16.8 and 28.8 mg/kg bw, respectively (Treon and Cleveland, 1955). For 6-month old rats, there was a greater sex-related difference in LD_{50} values; LD_{50} values

were 7.3 mg/kg bw for females and 43.4 mg/kg bw for males. In a chronic (2 year) study with rats, mortality was significantly higher for female rats exposed to 25, 50, and 100 ppm than for those exposed to 0, 1, or 5 ppm (Treon and Cleveland, 1955). Male rats were not as sensitive to the effects of endrin as females; however, mortality increased in the 50 ppm and higher treatment groups. Male shrews (Blarina brevicauda) were more sensitive to endrin in the diet than were females, and younger females were more sensitive than older females (Blus, 1978). The LC_{50} for shrews of both sexes and of varying ages ranged from 87 to 174 ppm. All shrews that died lost body weight. Shrews that were severely intoxicated with endrin tended to stay under cover as opposed to DDT intoxicated shrews that came to the surface (Blus, 1978).

Rabbits dermally exposed to 0.25 to 3.6 grams per kilogram body weight (g/kg bw) died within 24-hr of application (Treon and Cleveland, 1955). The NOEL was 0.06 g/kg bw.

Bioaccumulation Potential Of Endrin

Aquatic Ecosystems--Compared to the other organochlorine pesticides, accumulation of endrin residues is not high (Kan, 1978). Due to the dynamic nature of endrin residues in tissue, residue concentration of the parent compound decreases rapidly once exposure ceases, although metabolites may be lost more slowly (EPA, 1980c). The half-life of endrin in channel catfish tissues was 12 days (Jackson, 1976), in bluegill half-life was 4 weeks (Sudershan and Khan, 1980),

BCFs observed in algae following exposure to endrin for 7 days were 140 to 222 (EPA, 1980c). BCFs for various freshwater invertebrates ranged from 7 to 2,600 for exposure durations of 1 to 24 days (EPA, 1979b): for various species of fish, BCFs ranged from 1,600 to 15,000 for exposure durations of 21 to 300 days (EPA, 1980c). BCFs in clams were approximately 3,000 (Jarvinen and Tyo, 1978). BCFs in frogs were approximately 100 (range 34 to 94) (Hall and Swineford, 1980). Bluegill exposed for 24-hr to 0.2 and 2 ug/l concentrated endrin by factors of 250 and 150, respectively (Bennett and Day, 1970). Maximum BCFs were approximately 10,000 after 56 days for fathead minnows (Jarvinen and Tyo, 1978).

BAFs in snails (*Physa sp.*) were 49,000; observed tissue concentrations reached 492 ppm (EPA, 1979b). In stoneflies, *P. dorsata*, BAFs were 600 to 1,000 in laboratory tests (the ratio is not a BCF because the insects were fed while in the test chamber) (Anderson and DeFoe, 1980). In fish, the major route of uptake is through the gills; uptake is approximately 2,000 times greater from water than from food in channel catfish (EPA, 1979b). For other studies, observed BMFs for fish were less than 1, indicating little uptake from food as compared to water (Jackson, 1976; Argyle *et al*, 1973). Maximum observed BAFs for fathead minnows (*Pimephales promelas*) were 13,000, while BMFs were 0.8; the residues were additive (Jarvinen and Tyo, 1978). For 4 and 7 day exposures, bullhead BAFs were 3,700 and 6,200, respectively (Anderson and DeFoe, 1980).

Under static conditions, bluegill (*Lepomis macrochirus*) absorb 85 percent of a 1 ppb dose from water in 48-hr (Sudershan and Khan, 1980). A 24-hr exposure to 0.2 and 2 ug/l resulted in BCFs of 250 and 150, respectively (Bennett and Day, 1970). At equilibrium, BCF values range from 1,640 to 15,000 for various species of freshwater fish (EPA, 1980c).

Terrestrial Ecosystems--Small amounts of endrin (approximately 10 percent compared to soil residues) are taken up into soybeans from clay loam soil with 1 to 1.5 percent organic matter (Barrentine and Cain, 1969). Magnification factors between turnips or peanuts and soil were also less than 1 (Wheeler *et al.*, 1969; Dorough and Randolph, 1969).

Soil invertebrates concentrate endrin more than do plants. Gish (1970) measured concentrations in soils and soil invertebrates (earthworms and others) from various locations with various soil types. A mean concentration factor ± standard deviations (SD) of 29 ± 32 on a dry weight basis was estimated from data points with both soil and invertebrate concentrations of endrin. Compared to the other organochlorine pesticides, accumulation of endrin residues is not high (Kan, 1978). The concentration ratio between poultry eggs and feed was 0.6 (Kan, 1978), and between poultry fat and feed was 7 to 10 (Kan, 1978). Mallard eggs contained approximately the same amount as the dietary concentration consumed by females (Roylance *et al.*, 1985). Similar results were observed by Spann *et*

al. (1986), where 1 and 3 ppm endrin in diet resulted in 1.1 or 2.9 ppm in mallard eggs, respectively, while fat residues were 4 to 8 times higher.

The half-life of endrin in biota ranges from days to weeks depending on the species and tissue being measured. Endrin is metabolized rapidly by mammals (EPA, 1985f) but stored in the fat of birds (Reichel et al., 1969). The half-life in poultry eggs and fat was 4 to 5 weeks (Kan, 1978). Elimination in mallards indicated a half-life in whole body of 3 days (Heinz and Johnson, 1979), and in rats 2 to 4 days (Korte, 1970).

Fate Of Endrin In The Environment

Endrin is subject to microbial degradation to ketoendrin, endrin aldehyde, and endrin alcohol (EPA, 1979b). Microbial degradation is favored under anaerobic conditions such as flooded soil.

In plants, the major metabolite is an endrin alcohol, with some ketoendrin occurring as well (EPA, 1979b). In bluegill, 12-anti-hydroxyendrin and 12-syn-hydroxyendrin were the major metabolites identified in tissue, with 73 percent of the tissue residues being composed of parent compound (Sudershan and Khan, 1980). The primary metabolic pathway in bluegill is hydroxylation followed by conjugation: elimination products were the conjugated metabolites and not the parent compound (Sudershan and Khan, 1980).

The primary metabolic pathway in mammals is similar to that in fish, with hydroxylation producing three monohydroxylated products (Sudershan and Khan, 1980). Endrin is excreted as a mixture of polar metabolites in rabbit urine, while 50 percent of an oral dose is excreted as the unchanged parent compound in feces (Bedford et al., 1975). Metabolism occurs by hydroxylation at the C-12 methylene bridge to form anti- and syn-12-hydroxyendrin, which are then excreted as sulfate and glucuronide conjugates. 12-Hydroxyendrin can be further oxidized to 12-ketoendrin, which is the major toxic metabolite (EPA, 1987c). The route of excretion of endrin is different in the rat, where the liver is the major excretory organ (Cole et al., 1970), and only 2 percent of an oral dose is excreted in urine (Bedford et al., 1975). In rabbits, fecal excretion is almost complete in

24-hr, while for rats fecal excretion takes several days (Bedford et al., 1975). The metabolites 12-ketoendrin and anti- and syn-12-hydroxyendrin are 2 to 5 times more toxic than the parent compound (Bedford et al., 1975).

5.2.4.2 Surface Water Ingestion

In addition to exposure by ingestion of contaminated food items, the key organisms are potentially exposed to contaminants by ingestion of surface water. The key organisms for which a surface water pathway becomes important are the nonaquatic animals such as small mammals, waterfowl, and raptors. Bioconcentration as defined for aquatic organisms is not applicable to nonaquatic organisms, because tissue concentrations are not a direct function of water concentration. However, uptake of contaminants from ingestion of surface water can occur, with accumulation rates depending on the amount of water ingested daily and the concentration of contaminants in the water supply.

Small Mammals--The adverse effects levels for laboratory mammals are listed in Table 5.2-26. All data for mammals are from subchronic studies. The lowest ingestion rate that produced adverse effects for rats was 0.15 mg/kg/day for oral gavage for days 7 through 15 of pregnancy (Gray et al., 1981). At this dose, behavior was altered in offspring. No toxic effects were noted at the next lower dose, 0.075 mg/kg bw/day. Assuming toxicity from oral exposure is similar to toxicity due to exposure from ingestion of surface water, an acceptable water concentration is derived as from health effects and water consumption data for rats as follows:

$$\frac{\text{NOEL}}{\text{Intake/kg bw/day}} = \frac{0.075 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 0.6 \text{ mg/l}$$

Applying an uncertainty factor of 10 to convert the subchronic NOEL to a chronic NOEL, and 5 for interspecific variation, yields an acceptable surface water concentration of 0.012 mg/l (12 ppb).

Table 5.2-26 lists the surface water concentrations that correlate with the toxic effects levels in diet or by oral ingestion based on daily water intake for each species. Uncertainty factors were applied to all values in

Table 5.2-26. Toxic Effects Levels of Endrin for Small Mammals and Birds by Ingestion

Species	Route	Dose (mg/kg bw/day)	Effect	Corresponding Water Concentration (ppm)	Source
Rat	oral	3.5	Convulsions EEG changes	0.112	Speck & Maaske, 1958
Rat	oral	1.7	No adverse effects	0.272	Speck & Maaske, 1958
Rat	oral	0.15	Altered behavior of offspring	0.048	Gray et al., 1981
Rat	oral	0.075	No adverse effects	0.012	Gray et al., 1981
Mallard	3 ppm (diet)	0.16*	Slight mortality, Weight loss, Reproductive effects	0.032	Spann et al., 1986
Mallard	1 ppm (diet)	0.05*	No adverse effects	0.05	Spann et al.,
Bald Eagle	20 ppm (diet)	1.1*	Death	0.022	Stickel et al., 1979

* Daily dose was calculated from dietary concentrations using the food intake factor, R, described previously.

Source: ESE, 1988.

Table 5.2-26 as described in Section 5.1. The lowest water concentration for mammals, 0.048 ppm, is based on a subchronic LOAEL for rats with behavior of offspring as the toxicological endpoint. Values obtained using LOAELs are considered more uncertain than the water concentration derived from a NOEL. Therefore, the value based on a subchronic NOEL (0.012 ppm) is used to represent a "no effects" water concentration for mammals.

Birds--The lowest acceptable concentration in surface water is derived using the lowest toxic effects levels for birds from Table 5.2-26. Data for mallard are chronic, whereas data for eagle are subchronic. The chronic NOEL was 0.05 mg/kg bw/day for mallard. From the NOEL and the water intake for mallard, the acceptable water concentration is as follows:

$$\frac{\text{NOEL}}{\text{Intake/kg bw/day}} = \frac{0.05\ \text{mg/kg bw/day}}{0.2\ \text{l/kg bw/day}} = 0.25\ \text{mg/l}$$

An uncertainty factor of 5 was used for interspecific variation to yield an acceptable water concentration of 0.05 ppm (50 ppb). Lower values of 0.032 ppm based on a chronic LOAEL for mallards or 0.022 ppm based on a subchronic LOAEL for eagles (and a water intake of 0.2 l/kg bw/day) were considered more uncertain than a water concentration based on a chronic NOEL for mallards. Therefore, 0.05 ppm was used to represent an acceptable surface water concentration for birds.

The "no effects" level derived for birds is slightly higher than the value derived for mammals, although the difference is probably not significant. Because the "no effects" level in birds was based on a chronic NOEL, there is less uncertainty in the estimate. Therefore, an endrin concentration in surface water of 0.05 mg/l (50 ppb) as derived from data for mallards is assumed to be protective of all species consuming water at RMA. The corresponding sediment concentration, based on a K_{oc} of 9,100 l/kg (see Section 5.2.5.6) and f_{oc} of 0.0065 (EBASCO, 1988), is 2.96 ppm.

5.2.4.3 Aquatic Life

To estimate site-specific criteria for aquatic life in the RMA lakes, data for species that occur at RMA were examined for the lowest acute value or

the lowest chronic LOAEL. Data for cold water fish species such as rainbow trout were not considered appropriate. The lowest acute and chronic values were for fathead minnow (0.41 and 0.19 ppb, respectively) (EPA, 1980c). When the lowest acute value is divided by the acute-chronic ratio of 4 (EPA, 1980c), a water concentration of 0.10 is obtained. More stringent criteria are derived from an MPTC of 0.63 ppm for fathead minnow and a geometric mean BCF of 1,324 adjusted for 15 percent liquid. The acceptable water concentration is 0.032 ppb.

The corresponding sediment criterion is calculated as follows:

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \quad (8)$$

where: K_{oc} = 9,100 (see Section 5.2.5.6)
f_{oc} = 0.0065 (EBASCO, 1988)

C_{sed} = 0.032 ppb x 9,100 x 0.0065
C_{sed} = 1.90 ppb (0.0019 ppm)

5.2.4.4 Aquatic Pathway Analysis

Introduction To Aquatic Pathway Analysis

This Pathway Analysis is based on the bald eagle sink food subweb and includes all food chains leading to the selected sink species (Cohen, 1978). Because the same organisms/groups appear in more than one food chain throughout the web, percentage contributions for each organism or compartment have been estimated based on existing literature (Table 5.2-27). The subweb has been simplified (e.g., bluegill represent all fish species at that trophic level) because of the limited data available.

The bald eagle is a federally listed endangered species and is a component of food webs on RMA. The bald eagle was selected as the target species because of its endangered status and because it represents the highest trophic level affected by the bioaccumulation of contaminants through aquatic food chains. Aquatic organisms are considered to be the most important links in the bald eagle food web because they are constantly exposed to the contaminants in their environment via surface adsorption, absorption, and uptake across respiratory membranes; thus, bioconcentration

Table 5.2-27. Summary of Feeding Habits for the Pathway Analysis for Endrin

Species	Food Items	% in Diet	Reference
Mallard	Invertebrates[1]	44	Swanson et al., 1979; Swanson et al., 1985
	Plants[2]	30	Swanson et al., 1979
	Annelids[3]	26	Swanson et al., 1979
Bald Eagle	Waterfowl	24	Cash et al., 1985; Todd et al., 1982
	Fish	66	Cash et al., 1985
	Mammals	10	Cash et al., 1985
Bluegill	Invertebrates	88	Martin et al., 1961
	Plankton, Algae	12	Martin et al., 1961
Pike	Fish[4]	100	Inskip, 1982

1 Includes Crustacea, Insecta, and Mollusca.

2 Plants includes fruits of aquatic and terrestrial plant species as well as vegetation.

3 These food items were not utilized in the pathways analysis. Annelids are apparently washed into aquatic systems (Swanson et al., 1979) and were not included due to the temporary nature of availability.

4 Pike are opportunistic feeders that will utilize other food sources, but are assumed to prey completely on fish for the sake of the analysis.

Source: ESE, 1988.

factors tend to be large. The "no effects" level is based on health effects levels obtained from the scientific literature and presumes that if bald eagles will not be affected, other species will be protected. No safety factors have been used in the calculation of acceptable "no effects" levels.

Methods

Published values were used to represent BCFs and BAFs. Table 5.2-28 lists the BCF values utilized in this study. For invertebrates, data indicate equilibrium with endrin in water is attained after 2 to 2.5 days of exposure (EPA, 1980). Another source indicates equilibrium is attained in 5 days (Hamelink, 1971). Therefore, only studies with exposure durations of 5 days or more were used in calculating the invertebrate BCF.

Five food transfer pathways ultimately terminating with the bald eagle were established as follows:

		Trophic Level			
Pathway	Source	1	2	3	4
1	H_2O	Invertebrates	Mallard	Bald Eagle	
2	H_2O	Aquatic Plants	Mallard	Bald Eagle	
3	H_2O	Plankton	Bluegill	Pike	Bald Eagle
4	H_2O	Invertebrates	Bluegill	Pike	Bald Eagle
5	Soil	Terrestrial Plants	Small Mammals	Bald Eagle	

The mallard and the pike represent the sum total of birds and fish fed upon by the bald eagle. The combined food transfer pathways are presented in Figure 5.2-6.

All pathways (except Pathway Five) originate with water. The lowest step in the food chain is assumed to be in equilibrium with the aquatic environment, which gives equation (1):

$$BCF = C_b/C_w \qquad (1)$$

where: C_b = the concentration of endrin in biota

C_w = the concentration of endrin in water

Table 5.2-28. Bioconcentration Factors Used in the Endrin Pathways Analysis

Species	BCF	Mean BCF	Source
Plants			
Microcystis aeruginosa	200		EPA, 1980c
Anabaena cylindrica	222		EPA, 1980c
Scenedesmus quadricauda	156		EPA, 1980c
Oedogonium sp.	140		EPA, 1980c
Geometric Mean		180	
Plankton[1]			
Geometric Mean		180	
Invertebrates			
Mussels (mixed species)	3,000		Jarvinen and Tyo, 1978
Mussel (*Hyridella australis*)	38		EPA, 1979b
Geometric Mean		340	
Bluegill			
Fathead minnow	10,000		EPA, 1980c
Fathead minnow	7,000		EPA, 1980c
Channel catfish	1,640 - 2,000		EPA, 1980c
Flagfish (*Jordanella floridae*)	15,000		EPA 1980c
Geometric Mean		5,098	
Pike[2]		5,098	

1 BCFs used to calculate the mean for plankton were the same as those used to calculate the mean for plants.

2 BCFs used to calculate the mean were the same as bluegill. BCFs for fish are for whole body.

Source: ESE, 1988.

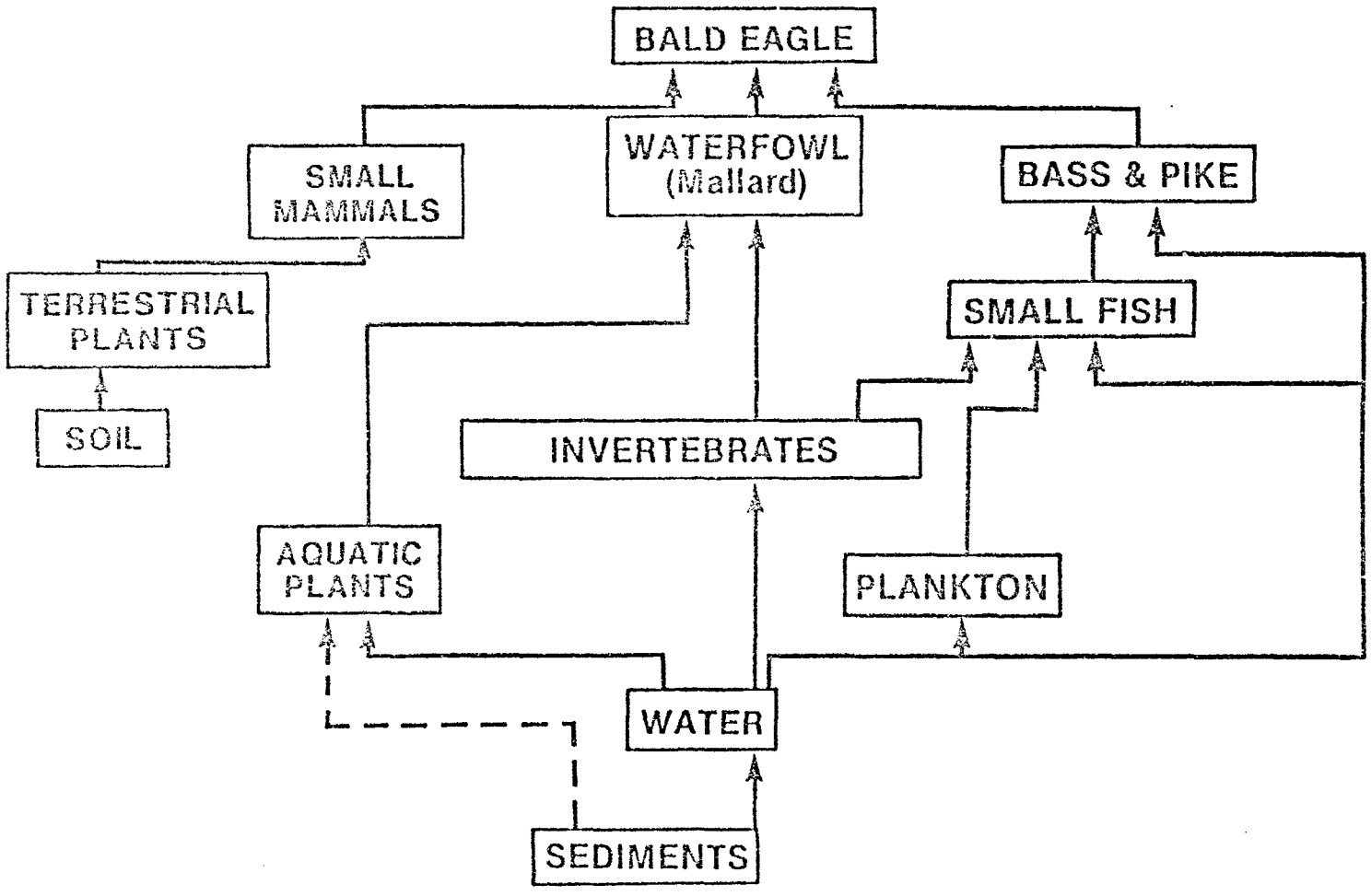


Figure 5.2-8

BALD EAGLE SINK FOOD WEB FOR ENDRIN

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

This equation is vital to the rest of the analysis. The end result, the total BMF for the bald eagle, can be ultimately traced back through water to the sediment, because it is assumed that all endrin enters the water compartment from sediments before being taken up by the biological compartment; i.e.,

$$C_w = \frac{C_{sed}}{K_{oc} \times f_{oc}} \qquad (7)$$

or solving for C_{sed}:

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \qquad (8)$$

where: C_{sed} = concentration of endrin in the sediment
C_w = concentration of endrin in water
K_{oc} = soil-water partition coefficient normalized for organic carbon
f_{oc} = fraction of organic carbon

There is a great deal of uncertainty in the estimate of K_{oc}. Kenaga (1980) estimated K_{oc} for endrin to be 34,000. K_{oc} can be estimated to range from 19,000 to 43,000 from regression equations reported in Kenaga and Goring (1980), Lyman and Loreti (1986), and Kadeg et al. (1986). K_{oc} is 10,000 based on measured water solubility (Richardson and Miller, 1959) and a regression equation from Lyman (1982). The best estimate of K_{oc} is 9,100 l/kg (see Section 5.2.5.6).

The f_{oc} is a measured value of 0.0065 (EBASCO, 1988), obtained from data from the RMA lakes for the surficial sediments.

The method used in the Pathway Analysis is the Thomann (1981) bioaccumulation model of food chain transfer in aquatic ecosystems where each level is a step in the food chain:

Level #1 $BCF_1 = C_b/C_w$ (1)

Level #2 $BAF_2 = BCF_2 + f_2BCF_1$ (2)

Level #3 $BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1$ (3)

Level #4 $BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1$ (4)

The food term (f_i) is a function of the trophic level in question and is calculated by the following equation:

$$f_i = \frac{@ \times R \times \%}{k_2} \qquad (5)$$

where: @ = Assimilation efficiency, $\frac{\text{ug absorbed}}{\text{ug absorbed}}$

R = Total Daily Diet, intake (g)/body weight (g)/day

k_2 = Depuration rate, day^{-1}

% = Percent of item in diet

The assimilation efficiency was approximated from a study using fish, where 85 percent of a dose was assimilated in 48 hr under static conditions (Sudershan and Khan, 1980). Because data regarding assimilation of endrin were unavailable for other species in the literature surveyed, 0.85 was used to represent the assimilation efficiency of all species in the Pathway Analysis.

The depuration rate (k_2) includes loss due to growth, excretion, and metabolism. Because rate constants have not been measured for each species in this analysis, k_2 values taken from the literature were used to represent all species. The loss rate can be calculated using a standard decay equation:

$$C_t = C_o e^{-kt} \qquad (11)$$

where:

C_o = initial concentration

t = time

C_t = concentration at time t, using biological half-life or $0.5C_0$

k = loss rate constant

Rearranging equation 11 to solve for k gives equation 12:

$$\frac{0.693}{t} = k \qquad (12)$$

The following k_2 values were utilized in the Pathway Analysis:

k_2 = 0.025/day Based on an observed half-life of endrin in bluegill of 4 weeks (Sudershan and Khan, 1980).

k_2 = 0.058/day Based on an observed half-life of endrin in channel catfish of 12 days (Jackson, 1976).

k_2 = 0.020/day Based on an observed half-life of endrin in poultry fat and eggs of 4 to 5 weeks (Kan, 1978).

k_2 = 0.035/day Based on an observed half-life of endrin in mallard duck whole body of 19.8 days. Data were recalculated to yield a first order rate constant (Heinz and Johnson, 1979).

A geometric mean was used to represent the loss rate in each of the different classes in the pathway analysis. For fish, a geometric mean value of 0.038/day was used to represent k_2 for bluegill and pike. For birds, a geometric mean value of 0.026/day was used to represent k_2 for mallard and bald eagle. Because BAF values for the mammalian pathway were calculated in a different manner, k_2 values were not required to estimate accumulation for Pathway 5 (soil -> plants -> mammals -> eagle).

Pathway Analysis

Pathway One: H_2O => Invertebrates => Mallard => Bald Eagle--The BCF for invertebrates ranges from 7 to 2,600 for various freshwater invertebrates for exposure durations ranging from 1 to 24 days (EPA, 1979b). However, it appears that at least 5 days are required for aquatic invertebrates to reach equilibrium conditions with environmental endrin concentrations (Hamelink, 1971). Therefore, only values where exposure duration was equal to or exceeded 5 days were used to calculate BCF. For freshwater mussels (mixed species) exposed for 21 days, the BCF was 3,000 (Jarvinen and Tyo, 1978), and for another freshwater mussel (*Hyridella australis*) exposed for 24 days, the BCF was 38 (EPA, 1979b). A geometric mean was calculated to represent bioconcentration by aquatic invertebrates.

$$BCF_{invert} = 340 \quad (1)$$

Small aquatic invertebrates are assumed to be in equilibrium with their environment; because of the large surface area to volume ratio, the processes of bioconcentration outweigh biomagnification to the extent that uptake from diet is insignificant to uptake from water. Therefore, BCFs are equivalent to BAFs, and these organisms can be considered to be Level #1 or non-feeding organisms.

The food term (f_2) is calculated by assuming that an adult mallard weighs approximately 1,100 g and consumes about 57.4 g total diet each day (Miller, 1975), of which 44 to 56 percent of the diet is invertebrates (Swanson et al., 1979). For the pathway analysis the value of 44 percent was selected although seasonal fluctuations in invertebrate populations will cause variation in consumption of this food type. The BAF for a mallard is calculated by assuming that the first term in the Level #2 bioaccumulation equation (2) equals zero (because bioconcentration by nonaquatic organisms is considered to be negligible):

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.85 \times (57.4\ g/1{,}100\ g/day) \times 44\%}{0.026/day} = 0.75 \qquad (5)$$

An adult eagle weighs approximately 4,500 g (Schafer, 1986) and consumes 255 g daily (Swies, 1986), of which 24 percent of the diet is birds (Cash et al., 1985; Sherrod, 1978). Energy requirements are different for wild birds than for birds living in captivity, so these dietary quantities are only approximate (Sherrod, 1986). The following BAF values for an eagle are calculated by assuming that the first two terms in the Level #3 bioaccumulation equation (3) equal zero (bioconcentration by the mallard and the eagle are both negligible):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{invert}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f_3 = \frac{0.85 \times (255\ g/4{,}500\ g/day) \times 24\%}{0.026/day} = 0.44 \qquad (5)$$

When the BCF for aquatic invertebrates is 340, the BAF for the mallard is 260, and the BAF for the eagle is 110.

Pathway Two: H_2O => Aquatic Plants => Mallard => Bald Eagle--The BCF for plants is based on observed values for algae (EPA, 1980c). The geometric mean represents the BCF for aquatic plants:

$$BCF_{plant} = 180 \qquad (1)$$

To calculate the BAF for mallards, the food term f_2 remains the same as Pathway One except for the percent of the food item in the diet. Using equation (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2BCF_{plant}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.85 \times (57.4\ g/1{,}100\ g/day) \times 30\%}{0.026/day} = 0.51 \qquad (5)$$

The food term for the consumption of mallards by the eagle, f_3, remains the same as the Pathway One equation. The BAF is calculated using equations (2) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{plant}$$

where: BCF_3 and $f_3BCF_2 = 0$

$$f_3 = \frac{0.85 \times (255\ g/4{,}500\ g/day) \times 24\%}{0.026/day} = 0.44 \qquad (5)$$

When the BCF for aquatic plants is 180, the BAF for duck is 92, and the BAF for eagle is 40.

Pathway Three: H_2O => Plankton, Algae => Bluegill => Pike => Bald Eagle--
Pathways leading to the bald eagle via fish are more complex because bioconcentration occurs at each trophic level, not just at the lowest trophic level. This introduces a fourth factor into the BAF equation, and the eagle is at Level #4 instead of Level #3. The BCF for plankton is derived from the data for algae (EPA, 1980c):

$$BCF_{plankton} = 130 \qquad (1)$$

The BCF for the bluegill (*Lepomis macrochirus*) is derived from studies indicating values for various fresh water fish species ranging from 1,640 to 15,000 (EPA, 1980c). A geometric mean value was used to represent the BCF for bluegill:

$$BCF_{bluegill} = 5,098 \qquad (1)$$

If a bluegill consumed 3 percent of its body weight daily (Chadwick and Brocksen, 1969), total daily intake would be a factor of 0.03 regardless of body weight or length. Various algal forms are known to account for 12 percent of the bluegill's diet (Martin *et al.*, 1961); this value was used for the percent of plankton in the bluegills diet. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2 BCF_{plankton}$$

$$\text{where: } f_2 = \frac{0.85 \times (0.03/day) \times 12\%}{0.038/day} = 0.081 \qquad (5)$$

The geometric mean BCF derived for freshwater fish and applied to bluegill was also applied to pike (5,098). If pike are also assumed to consume 3 percent of their body weight daily, the daily intake term is 0.03 regardless of individual weight or size. For the purposes of the analysis, pike are assumed to feed entirely on small fish, represented by bluegill. Using equations (3) and (5),

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{plankton}$$

$$\text{where: } f_3 = \frac{0.85 \times (0.03/day) \times 100\%}{0.038/day} = 0.67 \quad (5)$$

The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g food daily, of which 66 percent of the diet is fish (Cash *et al.*, 1985). The first term of the Level #4 equation equals zero. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{plankton}$$

$$\text{where: } BCF_4 = 0$$

$$f_4 = \frac{0.85 \times (255\ g/4{,}500\ g/day) \times 66\%}{0.026/day} = 1.2 \quad (5)$$

When the BCF for plankton is 180, the BAF values for bluegill and pike are 5,100 and 8,500, respectively. The BAF value for the bald eagle is 10,000.

Pathway Four: H_2O => Invertebrates => Bluegill => Pike => Bald Eagle--
For freshwater mussels (mixed species) exposed for 21 days, the BCF was 3,000 (Jarvinen and Tyo, 1978), and for a freshwater mussel (*Hyridella australis*) exposed for 24 days, the BCF was 38 (EPA, 1979b). A geometric mean was calculated to represent bioconcentration by aquatic invertebrates:

$$BCF_{invert} = 340 \quad (1)$$

The BCF for the bluegill is:

$$BCF_{bluegill} = 5{,}098 \quad (1)$$

If a bluegill consumed 3 percent of its body weight daily (Chadwick and Brocksen, 1969), total daily intake would be 0.03 regardless of body weight or size. Invertebrates are believed to account for 88 percent of the bluegills diet (Martin *et al.*, 1961). Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2 BCF_{invert}$$

$$\text{where: } f_2 = \frac{0.85 \times (0.03/day) \times 88\%}{0.038/day} = 0.59 \qquad (5)$$

The geometric mean derived for freshwater fish and applied to bluegill was also applied to pike (5,098). If pike are also assumed to consume 3 percent of their body weight daily, the daily intake term is 0.03 regardless of individual weight or size. For the purposes of the analysis, pike are assumed to feed entirely on small fish, represented by bluegill. Using equations (3) and (5),

$$BAF_3 = BCF_3 + f_3 BCF_2 + f_3 f_2 BCF_1 \qquad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3 BCF_{bluegill} + f_3 f_2 BCF_{invert}$$

$$\text{where: } f_3 = \frac{0.85 \times (0.03/day) \times 100\%}{0.038/day} = 0.67 \qquad (5)$$

The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g food daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4 f_3 BCF_2 + f_4 f_3 f_2 BCF_1 \qquad (4)$$

$$BAF_{eagle} = f_4 BCF_{pike} + f_4 f_3 BCF_{bluegill} + f_4 f_3 f_2 BCF_{invert}$$

$$\text{where: } BCF_4 = 0$$

$$f_4 = \frac{0.85 \times (255\ g/4{,}500\ g/day) \times 66\%}{0.026/day} = 1.2 \qquad (5)$$

When the BCF for aquatic invertebrates is 340, the BAF for bluegill and pike are 5,300 and 8,600, respectively. The BAF for eagle is 10,000.

Results and Discussion

BAF values as derived for the individual pathways (Table 5.2-.) represent accumulation in separate single food chains. To derive overall accumulation in the entire food web, variations of the following equation are used:

Table 5.2-29. Summary of Bioaccumulation Factors for each Pathway for Species in the Endrin Pathways Analysis.

	Bluegill	Pike	Duck	Mammal	Eagle
Pathway 1	--	--	260	--	110
Pathway 2	--	--	92	--	40
Pathway 3	5,100	8,500	--	--	10,000
Pathway 4	5,300	8,600	--	--	10,000
Pathway 5	--	--	--	0.49	0.010

Source: ESE, 1988.

$$BMF_i = BCF_i + \Sigma f_i BAF_{i-1}$$

For each of the major trophic levels in the aquatic Pathway Analysis, total biomagnification is presented in Table 5.2-30. Total BMF represents accumulation of residues originating in sediments, soil, and water by lower organisms directly: and accumulation of residues by higher organisms via food chain exposure. Because dietary percentage contributions have been considered, net residue accumulation is a function of the accumulation of residues by the lower trophic levels.

Total BMF can be used to determine maximum allowable levels of endrin in water by relating water concentration to a MATC as follows (Tucker, 1986):

$$\frac{MATC}{\text{Total BMF}} = C_w \qquad (6)$$

When the MATC is divided by total accumulation from water up all food chains in the food web, a "no effects" water concentration is obtained. This is related to sediment concentration by equation (8):

$$C_{sed} = C_w \times K_{oc} \times f_{oc} \qquad (8)$$

where: K_{oc} = 9,100 (see Section 5.2.5.6)
f_{oc} = 0.0065 (EBASCO, 1988)

The MATC is obtained by examining the literature for the lowest concentration which results in sublethal or lethal toxic effects:

SPECIES	ORGAN	PPM	EFFECT	SOURCE
Mallard	brain	2	death	Spann et al., 1986
Bald Eagle	brain	0.92	death	Stickel et al., 1979
Bald Eagle	carcass	1.5	death	Stickel et al., 1979
Mallard	brain	0.62	death	Stickel et al., 1979

Table 5.2-30. Total Biomagnification of Endrin Residues for each of the Key Organisms in the Aquatic Pathways Analysis.

Organism	Level	Equation	BMF
Mallard	#2	$\Sigma f_2 BCF_1$	350
Bluegill	#2	$BCF_2 + \Sigma f_2 BCF_1$	5,300
Pike	#3	$BCF_3 + f_3 BMF_{bluegill}$	8,700
Eagle	#3, #4	$f_4 BMF_{pike} + f_3 BMF_{mallard} + BMF_{terrestrial}$	11,000

Source: ESE, 1988.

The lowest tissue concentration at which toxic effects were observed was 0.62 ppm in mallard brain (Stickel et al., 1979). In this study, various species (including mallards and serveral types of passerines) with brain concentrations at or below 0.6 ppm survived testing, while birds with brain concentrations of 0.8 ppm and greater died during testing. Brain concentrations between 0.6 ppm and 0.8 ppm resulted in variable mortality. Because the food term, f_i, relates to whole body, health effects data for brain tissue must be related to carcass.

Brain concentrations can be approximately correlated with carcass concentrations from data presented by Stickel et al. (1979). For mallard, the geometric mean of residue data for two females indicates a brain to carcass ratio of 0.39. For bald eagle, the geometric mean of residue data for three eagles indicates a brain to carcass ratio of 0.75.

Using 0.62 ppm in brain to represent the lowest adverse health effects level in birds, the corresponding carcass concentration for mallard is 1.59 ppm, and for eagle the corresponding carcass concentration is 0.83 ppm. Mallards fed 3 ppm endrin accumulated up to 2.5 ± 0.49 (Mean ± SE) ppm in carcass: one male died and reproductive effects were observed. Mallards on a 1 ppm endrin diet accumulated up to 1.1 ppm in carcass with no adverse effects. Therefore, levels below 1.1 ppm probably are sublethal for mallard.

The lowest tissue concentration (0.83 ppm) at which toxic effects are estimated is divided by the Total BMF for the bald eagle from Table 5.2-30, then corrected with K_{oc}: thus, giving the sediment concentration at which "no effects" to bald eagle through food chain contamination are likely to occur:

$$\frac{MATC}{\text{Total BMF}} = C_w = \frac{0.83 \text{ ppm}}{11{,}000} = 7.5 \times 10^{-5} \text{ ppm} \qquad (6)$$

and,

$$C_{sed} = C_w \times K_{oc} \times f_{oc} = 7.5 \times 10^{-5} \times 9{,}100 \times 0.0065 = 0.0045 \text{ ppm} \qquad (8)$$

Since the total BMF is lower for mallard than bald eagle, corresponding criteria would be less stringent. Therefore, using the water and sediment criteria based on the bald eagle should be protective for mallards and other avian species lower in the food web as well. The criteria developed using the Pathway Analysis does not satisfy the acceptable levels established for surface water ingestion of 0.05 ppm. Due to the chronic toxicity of endrin to aquatic life, predicted "no effects" levels based on food chain accumulation are not protective for aquatic life. Therefore, water criteria should be based on the EPA (1980c) water quality criteria of 0.0023 ppb, with corresponding sediment criteria calculated with f_{oc} and K_{oc} of 0.00015 ppm.

5.2.4.5 Terrestrial Ecosystems

Methods

The terrestrial pathways must be addressed differently than the aquatic pathways, because data such as k_2 and assimilation efficiency are lacking for terrestrial organisms. BAFs are calculated by comparing C_b to C_{diet} or C_{soil}, and loss and uptake are therefore accounted for.

Pathway Five: Soil => Terrestrial Plants => Small Mammals => Bald Eagle--
Although endrin is accumulated from soil by plants, the EMFs (concentration in soil compared to plants) are usually less than one. From data summarized in EPA (1979b), EMF values were found to range from 0.0045 for radish (whole plants) exposed for 5 weeks to 8.91 ppm endrin in soil, to 1.11 for carrot roots exposed to 3.6 to 3.8 ppm in soil for 142 days (Table 5.2-31). Only data for which soil and plant values were given were used to calculate the mean; only data expressed on a fresh weight basis were used, in order to represent concentrations to which wild mammal populations might be exposed. A geometric mean (N=32) was used to represent the EMF:

$$EMF_{plants} = 0.031 \qquad (10)$$

The partition coefficient between small mammals and their diet is used to define bioaccumulation in the soil-based food chain (C_b/C_{diet}). Mammals have been observed to accumulate endrin from the diet, but because endrin is rapidly metabolized by mammals, partition coefficients between diet and fat

Table 5.2-31. Bioaccumulation Factors for the Terrestrial Food Chain, Pathways Analysis for Endrin

Species	N	BAF	Mean	Source
Plants	32		0.031	EPA, 1979
Mammals				
Cattle		0.3		Kenaga, 1980
		0.5		
Swine		0.3		
		1.28		
Geometric Mean for Mammals			0.49	
Birds				
Mallard		7.0		Spann et al., 1986
		4.4	5.55	Spann et al., 1986
Poultry		9		Kan, 1978
		7-10		Kan, 1978
		7		Kan, 1978
		10	8.55	Kan, 1978
Geometric Mean for Birds			6.9	

N = Number of samples.

Source: ESE, 1988.

are not high. Cattle and swine concentrate endrin from diet into body fat by factors of 0.3 to 1.28 for dietary concentrations of 2.5 to 5 ppm in diet (Kenaga, 1980). Data for small mammals were unavailable in the literature surveyed. A geometric mean was used to represent the BAF:

$$BAF_{mammals} = 0.49 \qquad (13)$$

The concentration ratio between poultry fat and feed ranges from 7 to 10 (Kan, 1978). Similiar results are observed in mallards, where fat residues are from 4 to 7 times higher than residues found in diet (Spann et al., 1986). A geometric mean was used to represent the BAF:

$$BAF_{birds} = 6.9 \qquad (13)$$

The terrestrial pathway is thus:

$$\text{soil} \xrightarrow{0.031} \text{plants} \xrightarrow{0.49} \text{mammals} \xrightarrow{6.9} \text{eagle}$$

The total BAF is calculated by multiplying the BAF values for each step in the soil based food chain. The amount accumulated from the diet by the eagle (assuming an accumulation rate equivalent to poultry or waterfowl) is 0.10 times the amount in soil. The terrestrial pathway is 10 percent of the eagles diet: therefore, the total BMF for this pathway is 0.010.

Results and Discussion

Pathway Five, the terrestrial based food chain, forms 10 percent of the eagle diet. "No effects" soil criteria can be estimated as follows:

$$\frac{MATC}{\text{Total BMF}} = C_{soil} = \frac{0.83}{0.01} = 83 \text{ ppm} \qquad (6)$$

Based upon observed winter feeding behavior of bald eagles at RMA, Pathway 5 forms approximately 90 percent of the eagle diet. This reduces the soil criteria by a corresponding amount:

$$\frac{MATC}{\text{Total BMF}} = C_{soil} = \frac{0.83}{0.09} = 9.2 \text{ ppm} \qquad (6)$$

The soil criterion derived from Pathway Five can also be used to predict toxicity to small mammals exposed to contaminants from ingesting contaminated soil. An exposure rate as a function of the acceptable soil criteria can be estimated from the soil criterion and the soil ingestion rate for small mammals as follows:

Soil Criterion x Soil Ingestion Rate = Daily Exposure

9.2 mg/kg soil x 0.000873 kg soil/kg bw/day = 0.0080 mg/kg bw/day

The exposure rate based on a soil criterion of 9.2 mg/kg soil is one order of magnitude lower than observed NOELs for small mammals (0.075 mg/kg bw), and therefore direct toxic effects are not expected at the criterion level of 9.2 mg/kg in soil. The daily intake of endrin from ingesting soil represents a conservative estimate as an assimilation efficiency of 100 percent is assumed.

Because biomagnification in the terrestrial food chain is less than 1, the terrestrial food web, with the American kestrel as the top carnivore, will not be constructed for endrin.

5.2.4.6 Uncertainty Analysis

In the uncertainty analysis, all of the intake rates (R values) and percent of items in diet are treated as triangular distributions where the minima and maxima are known and a best estimate within that range has been determined. Using the triangular distribution as input, the best estimate will be more likely than values near either end of the range. Methodology for the uncertainty analysis is described in detail in the forthcoming Offpost Endangerment Assessment. Diets of each link on the sink food web are summarized in Table 5.2-32.

Several assumptions were made in order to conduct the analysis:

- The diet of the target organism, the bald eagle, is supplied only by the aquatic food chain, with ducks and pike the representative prey organisms; and

Table 5.2-32. Dietary Input Factors, Endrin Pathways Analysis.
R = Total Dietary Intake (day^{-1})

	Minimum	Best Estimate	Maximum
Eagle	0.51	0.57	0.76
Mallard	0.45	0.52	0.93
Pike	0.01	0.03	0.05
Bluegill	0.01	0.03	0.05
Percent of Item in Diet			
Eagle/Mallard	14	28	42
Eagle/Pike	58	72	86
Mallard/Invertebrates	40	58	75
Mallard/Aquatic Plants	25	42	60
Bluegill/Plankton	6	12	18
Bluegill/Invertebrates	82	88	94

Source: ESE, 1988.

- Absorption, or assimilation, of ingested endrin is assumed to be 100 percent.

BCFs for endrin have been measured for several species. Based on four measured values, the BCF for plants and plankton was input as normal with a mean of 180 and a standard error of 19. Two reported values for aquatic invertebrates were quite divergent at 38 and 3,000. To reflect this uncertainty, a log triangular distribution was used with a minimum of 38, a mode of 340 (best estimate) and a maximum of 3,000. In reviewing the Ambient Water Quality Criteria Document (EPA, 1980c) for endrin and several of the primary references cited by that report, nine independent sources of information regarding the endrin BCF in fish were identified. These are summarized in Table 5.2-33. These values appear to follow a lognormal distribution with a mean of 4,800 ± 1,500.

Endrin depuration rates in fish have been measured by Sudershan and Khan (1980: k_2 = 0.02 day $^{-1}$ in bluegill), Jackson (1976: 0.06 ± 0.01 day $^{-1}$ in channel catfish), and Argyle et al., (1973: 0.12 day $^{-1}$ in channel catfish). Based on these data, an input uncertainty distribution for k_2 in fish was represented as a lognormal distribution with a mean of 0.07 day $^{-1}$ and a standard error of 0.03.

Endrin depuration in birds has been studied by Cummings, et al., (1967) and Heinz and Johnson (1979). Heinz and Johnson's data were weighted more heavily because they studied mallard ducks, a component of the aquatic food web for eagles, while Cummings et al., (1967) studied hens. Heinz and Johnson (1979) reported a first order rate constant of 0.23 day^{-1}, but this result was based on scaling the data by the square root of time, which is not the standard definition of a first order rate constant. A reanalysis of their data, using only results beginning four days after endrin dosing was discontinued, indicates a depuration rate from carcass of 0.035 day^{-1} and 0.045 day^{-1} from blood data. The carcass data was weighted higher than the blood data. These data were evaluated along with Cummings' data from fatty tissue (0.0223 day^{-1}) and muscle (0.011 day^{-1}) in hens. Based on all these data, an uncertainty distribution for k_2 was established as a lognormal distribution with a mean of 0.031 day^{-1} and a standard error of 0.007.

Table 5.2-33. BCFs Used to Perform the Uncertainty Analysis, Endrin Pathways Analysis

BCF	Species	Source	Comments
21,000	Medaka	Johnson (1967)	Geometric mean of range reported by Jarvinen and Tyo (1978)
10,000	Fathead Minnow	Mount & Putnicki (1966)	Reported by Jarvinen and Tyo (1978)
8,300	Flagfish	Hermanutz (1978)	Geometric mean of 12 values reported
7,000	Fathead Minnow	Jarvinen & Tyo (1978)	
5,012	Fathead Minnow	Davies & Dobbs (1978)	Reinterpretation of Jarvinen & Tyo's data
4,050	not specified	Schimmel, et al. (1975)	Reported by Kenaga and Goring (1978)
1,810	Channel Catfish	Argyle, et al. (1973)	Reported by EPA (1980)
1,480	not specified	Neely, et al. (1974)	
1,360	not specified	Metcalf (1974)	Reported by Kenaga and Goring (1978)

Source: ESE, 1988.

K_{OC} has not been measured experimentally for endrin, so it is necessary to rely on estimates based on either solubility or K_{OW}. Widely divergent values of endrin's log K_{OW} have been reported, including a purported measured value of 3.2 (Rao and Dividson, 1983), and estimated values as high as 5.6 (Mabey et al., 1984; Kenaga, 1980; Neely et al., 1974; Kadeg et al., 1986). Based on the variability in reported values, high uncertainty is attributed to a "best estimate" of 4.0. This estimate was applied to estimate log K_{OC} from Lyman et al. (1982) equation 4-8, Lyman and Loretti's (1987) equation I, and the prediction equation developed by Kadeg et al., 1986. To each of these estimates a standard error of 1.0 $\log_{10}$ units is attributed. The results: however, of these predictions were consistent, ranging from 3.6 to 3.7.

K_{OC} may also be estimated from water solubility. Using equation 4-5 of Lyman et al. (1982), and the measured solubility of 230 mg/l (Richardson and Miller, 1959), an estimated log K_{OC} of 4.0 is calculated. Because it is based on a measured solubility, this value was weighted more heavily, resulting in a "best estimate" of 3.8. Although each individual estimate is relatively uncertain, the estimates are consistent, and the value is probably reliable to a factor of 5. As an input, a lognormal with a mean of 9,100 and a standard error of 6,800 was used.

Organic carbon content of the sediment of the RMA lakes is a measured value (EBASCO, 1988). In the upper 1 foot (ft) of sediment, organic carbon appears to follow a lognormal distribution with a mean of 0.65 percent and a standard deviation of 0.62 percent.

Results for endrin are summarized as follows: the best estimate of BMF is 27,700 with 95 percent confidence, with upper and lower bounds of 118,000 and 7,115, respectively. The median estimate of the ambient water concentration that will not result in unacceptable tissue concentrations in bald eagles is 0.030 ppb, with upper and lower bounds of 0.119 and 0.007 ppb, respectively. The corresponding sediment bounds are 0.00797 ppm and 0.00146 ppm, for a best estimate of 0.00129 ppm.

For the terrestrial food chain, it was assumed that all the factors were log normal. The 50th percentile is the best estimate with lower and upper bounds of 5th and 95th percentiles. It was assumed for purposes of the analysis that the terrestrial food chain comprised 100 percent of the eagle's diet.

The best estimate of BMF is 0.10: with lower and upper bounds of 0.048 and 0.215, respectively. The best estimate of a "no effects" soil concentration is 8.1 ppm, with lower and upper bounds of 3.9 and 17.3 ppm, respectively.

5.2.4.7 Summary and Conclusions

Endrin bioaccumulates in aquatic ecosystems, and to a lesser extent in terrestrial ecosystems. Total residue magnification in the terrestrial food chain is 0.10 times the amount in soil. Residue magnification in the bald eagle aquatic food web is by a factor of 11,000.

Based on the Pathway Analysis, "no effects" levels in water, sediments, and soil on RMA are 0.075 ppb, 0.0045 ppm, and 9.2 ppm, respectively. Acceptable levels in surface water are 0.05 ppm based on toxicity to avian species: therefore, the Pathway Analysis approach does not provide a level in water stringent enough to protect biota consuming surface water. However, due to the bioaccumulation potential of endrin in aquatic organisms, criteria based on aquatic life are lower than criteria established based on surface water ingestion or by using the pathways approach. For this reason, the acceptable levels in water are the EPA criteria of 0.032 ppb, with corresponding sediment criteria of 0.0019 ppm, calculated using K_{oc} and f_{oc}. Because endrin is chronically toxic at low levels, soil criteria were adjusted to protect small mammals from chronic effects due to soil ingestion.

The site-specific criteria for abiotic media at RMA are summarized as follows:

Method	Water (ppb)	Sediment (ppm)	Soil (ppm)
Water Ingestion	50	2.96	NA
Aquatic Pathways Analysis	0.075	0.0045	NA
Aquatic Life	0.032	0.0019	NA
Terrestrial Pathway Analysis	NA	NA	9.2

5.2.5 PATHWAY ANALYSIS FOR MERCURY

5.2.5.1 Background Information

Mercury compounds tend to be persistent in the environment and are known to accumulate in food chains (Bothner et al., 1980: Gough et al., 1979). Mercury was selected for Pathway Analysis because of its known distribution on RMA (ESE, 1987, RIC#88204R02), its toxicity and persistence in the environment, and its high potential for bioaccumulation. Methylmercury is more chronically toxic than inorganic mercury: therefore, criteria based on toxicity of methylmercury are protective of inorganic mercury exposure as well. Mercury levels in western soils range from <0.01 to 4.6 ppm (Shacklette and Boerngen, 1984). Mercury concentrations in unpolluted natural water ranges from 0.02 to 0.1 ppb (Moore and Ramamoorthy, 1984).

Concentration factors obtained for methylmercury were assumed to represent inorganic mercury as well because methylation occurs readily in the environment (Gough et al., 1979). In addition, methylmercury is generally more toxic than inorganic mercury, and methylmercury is more readily accumulated by biological receptors. The chronic criteria for the protection of aquatic organisms and their uses are based on bioconcentration and toxicity of methylmercury (EPA, 1985c). Therefore, using values for methylmercury to represent chronic toxicity and accumulation of all forms of mercury is consistent with EPA methodology. For organisms at the bottom of the food web, concentration factors for total mercury and methylmercury were considered because the data were derived from RMA samples.

Toxicity Of Mercury

Mercury is toxic to all forms of biota in both aquatic and terrestrial ecosystems. The freshwater acute criterion for mercury is 2.4 ppb, and the chronic freshwater criterion is 0.012 ppb (EPA, 1985c). Many factors influence toxicity of mercury such as alkalinity, pH, and temperature (EPA, 1985). Organic mercury is 4 to 31 times more toxic to several aquatic species than inorganic forms (EPA, 198[illegible]).

Plants--Inorganic mercury produces toxic effects in plants at concentrations ranging from 5 to 3,400 ppb (EPA, 1985c). These toxic effects include the lethal concentration for 50 percent of the population (LC_{50}) as well as the effective concentration for 50 percent of the population (EC_{50}) for effects such as inhibition of growth or cell division. Other studies indicate toxic effects at 80 to 2,600 ppb inorganic mercury, whereas methylmercury results in toxic effects at concentrations as low as 4.8 ppb (EPA, 1980e).

The uptake rates of inorganic and organic mercury compounds are similar in aquatic plants, although methylmercury compounds are more toxic (Mortimer et al., 1975; EPA, 1985c). Uptake is a result of both absorption and adsorption, and is proportional to water concentration (Mortimer et al., 1975). In rooted macrophytes, uptake also occurs from absorption of mercury from interstitial water by roots (Huckabee et al., 1979; Forstner and Wittman, 1979).

Terrestrial vascular plants are relatively insensitive to the toxic effects of mercury, and may accumulate high levels into tissues before effects occur (Gough et al., 1979). Seeds incubated for 3 to 7 days in mercury solutions exceeding 100 ppm failed to germinate, and growth of cucumber roots was inhibited at 1 ppm mercuric chloride solution (Siegel et al., 1971). Mercury accumulates from the soil through the roots, and from air by retention on the above ground parts of herbaceous plants and absorption by stomata (Shaw and Panigrahi, 1986). Little accumulation is expected in plants grown on normal soils (Gough et al., 1979). In a study of mercury accumulation in different plant tissues, approximately twice as much mercury is found in leaves as in roots, stems, or fruits (Shaw and Panigrahi, 1986). Magnification factors for uptake from soil determined from seven common

garden vegetables and herbs yield an estimated range of values from 0.19 to 0.41 on a wet weight basis (Shariatpanahi and Anderson, 1986), assuming a dry weight to wet weight correction factor of 0.5 (Bies et al., 1984).

Aquatic Invertebrates--The LC_{50} values for aquatic invertebrates acutely exposed to inorganic mercury range from 1.5 to 2,100 ppb (EPA, 1985c). Snail embryos (Amnicola sp.) were the most resistant of the invertebrates tested, and Daphnia magna were the most sensitive. Chronic toxicity values for exposure of D. magna to inorganic mercury in life cycle tests ranged from 0.72 to 1.82 ppb (EPA, 1985c). Daphnia were more sensitive in chronic exposure to methylmercury compounds than to inorganic, with effects occurring at 0.04 ppb, the lowest level tested (EPA, 1985c).

Aquatic macroinvertebrates are not as sensitive as protozoans and algae. The aquatic invertebrates Artemia spp. and Oniscus spp. survived 200 times greater dosages for exposure durations 2 to 10 times longer than protozoans and algae (Siegel et al., 1971). At dimethylmercury levels of 7 ppm for a 5-h exposure, planaria are immobilized and disintegrated; Turbatrix spp. are immobilized after approximately 7 days at exposures of 10 ppm dimethylmercury (Siegel et al., 1971). Other aquatic macroinvertebrates are affected at levels of 100 ppm and greater, at exposure times ranging from 1 hour to 50 hours; observed toxic effects included behavioral abnormalities, immobilization, and death (Siegel et al., 1971).

The half-life of mercury in zooplankton and Daphnia is approximately 3 days (Huckabee et al., 1979). The half-life in a freshwater mussel was reported to be 194 days for exposure to inorganic mercury and 860 days for methylmercury (Huckabee et al., 1979).

Fish--LC_{50}s for fish exposed to inorganic mercury under flow-through conditions range from 150 to 420 ppb (EPA, 1985c). The LC_{50} values for fish exposed to organic mercury compounds under flow-through conditions ranged from 24 to 84 ppb (EPA, 1985c). Fathead minnows (Pimephales promelas) exposed to inorganic mercury had adverse effects at the lowest concentration tested, 0.23 ppb, in an early life stage test and a life cycle test (EPA, 1985c).

The symptoms of acute mercury poisoning in fish are flaring of gill covers, increased frequency of respiratory movements, loss of equilibrium, and sluggishness (Armstrong, 1979). Chronic mercury poisoning symptoms in fish are emaciation, brain lesions, cataracts, and CNS effects such as abnormal motor coordination, erratic behaviors, and inability to capture food (Armstrong, 1979; Hawryshyn et al., 1982).

At concentrations of 10, 50, and 100 ppb, the ability of mosquito fish (Gambusia affinis) to avoid predation by bass was impaired (Kania et al., 1974). Concentrations of 0.3 to 4 ppb did not produce adverse effects as measured by increased oxygen consumption by rainbow trout (Salmo gairdneri) (Kania and O'Hara, 1974). Mercury in water at concentrations of 10 ppb over an exposure period of 21 days altered the opercular rhythm in largemouth bass (Micropterus salmoides) (Morgan, 1979). Phenylmercuric acetate at concentrations of 0.11 to 1.1 ppb caused growth inhibition in rainbow trout (Matida et al., 1971).

The half-life of mercury in fish can be as long as 2 to 3 years, and decreased tissue concentration is primarily due to dilution from growth as opposed to excretion (EPA, 1985c). The half-life of mercury in fish ranges from 20 days for guppies to 1,000 days for mosquito fish, brook trout (Salvelinus fontinalis), and rainbow trout (Huckabee et al., 1979). In bluegill (Lepomis macrochirus) exposed to methylmercury and guppies (Poecilia reticulata) exposed to inorganic mercury, elimination has been observed to be a two step process consisting of an initial fast stage and a second slow stage (Burrows and Krenkel, 1973; Kramer and Neidhart, 1975). In pike (Esox lucius), the half-life of methylmercury was 640 days (Jarvenpaa et al., 1970).

Birds--The LD_{50}s for quail, Coturnix japonica, for inorganic and organic mercury are 42 and 18 mg/kg bw, respectively (Hill, 1984). The 5-day dietary LC_{50}s for quail for inorganic and organic mercury are 5,086 and 47 ppm, respectively (Hill, 1984). The LD_{50} values for five different organic mercurials for 10 bird species range from 11.5 mg/kg bw to greater than 80 mg/kg bw (McEwen, 1968).

Concentrations of inorganic mercury of 250 ppm and higher in ingested water for 35 days caused death and decreased growth of chickens, whereas no effects were observed at concentrations of 5, 25, and 125 ppm (Parkhurst and Thaxton, 1973). From a water ingestion rate for chickens of 0.25 l/kg bw/day (Sax, 1984) and the highest NOEL in ingested water of 125 ppm, a NOEL is estimated of 31.25 mg/kg bw/day.

The subchronic lethal dietary concentration of methylmercury for young chickens is 5.09 ppm (0.21 mg/kg bw) (Soares et al., 1973). In raptors, dietary levels of 7 to 10 ppm can result in lethal effects (Fimreite and Karstad, 1971). Ring-necked pheasants subchronically dosed by oral capsule with an ethylmercury-containing fungicide had decreased reproduction at mercury dose levels of 0.64 mg/kg bw/day (McEwen et al., 1973).

The responses of birds exposed to methylmercury include mortality, decreased survivability of young, loss of body weight, behavior abnormalities, and physical malformations in offspring (Heinz, 1975; Heinz, 1979; Fimreite and Karstad, 1971; Hoffman and Moore, 1979; Borg, 1970; Soares et al., 1973). Responses of birds exposed to inorganic mercury include mortality, abnormal sexual maturity, and depressed growth (Parkhurst et al., 1973; Hill et al., 1984). Studies conducted on mallard ducks (Anas platyrynchos) and ring-necked pheasants (Phasianus colchicus) indicated that methylmercury in the diets of females increased embryo mortality, decreased egg production, and reduced the hatchability of eggs (Heinz, 1979; Prince, 1981; Spann, 1972; Fimreite and Karstad, 1971; Birge and Roberts, 1976). Because of the sensitivity of the avian embryo, concentrations of mercury that are not lethal to adults may prove lethal to chicks (Birge and Roberts, 1976). When laying females are fed methylmercury, embryonic mortality is greatest during late stages of incubation and in offspring within the first four days after hatching (Prince, 1981).

Brain concentrations of 10 ppm are diagnostic for poisoning for birds (Braune, 1987). Lethal brain levels in the goshawk (Accipiter g. gentilis)) were observed to be 30 to 40 ppm (Borg et al., 1970). In pheasants, 30 to 130 ppm in liver and kidney correlated with lethal effects (Borg et al.,

1969). Lethal levels for leghorn cockerels were 18 ppm in diet, with observed concentrations of 10 ppm in liver (Fimreite, 1970). Lethal levels in liver of pheasants, magpies, and jackdaws ranged from as low as 30 to as high as 200 ppm (Borg et al., 1969a). Lethal levels in liver of American kestrels (Falco sparverius) fed a diet of contaminated mice were 49 to 122 ppm (Koeman et al., 1971).

Exposure to mercury at sublethal concentrations produces a wide range of reproductive effects for birds. Wiemeyer et al. (1984) observed that 0.5 ppm in diet resulted in fewer eggs laid by mallards, and decreased the number of young produced: the residue levels in eggs were 0.79 to 0.86 ppm. Pheasants exposed to mercury in diet also produced significantly fewer eggs and had higher embryo mortality than controls (Spann et al., 1972). Residue levels in pheasant eggs that correlated with decreased hatchability were between 0.5 and 1.5 ppm in a study by Fimreite and Karstad (1971), and between 1.3 to 2.0 ppm in another study (Borg et al., 1969b).

Behavioral effects such as hypersensitivity to frightening stimuli were observed for mallard ducklings when parents were fed 0.1 and 0.6 ppm mercury in diet (Heinz, 1975): hens fed 0.1 ppm in diet had 1 ppm in eggs. Methylmercury externally applied to eggs reduced hatchability at concentrations as low as 1 ppb, and decreased chick survival after treatment with 0.9 ug Hg per egg (Hoffman and Moore, 1979).

Mammals--The LD_{50} for mule deer is 17.86 mg/kg bw (Hudson et al., 1984). The LC_{50} for mammals is approximately 1 to 5 ppm in diet (Eisler, 1987). In mammals, more than 90 percent of methylmercury in diet is absorbed (Berglund and Berlin, 1969). The toxic effects of mercury result from affinity for sulfhydryl groups, enzyme inhibition, and precipitation of proteins (Clarkson, 1971).

Mercury can have a synergistic action with temperature stress, as indicated by a toxic level to mink of 1.0 ppm methylmercury (estimated as approximately 0.05 mg/kg bw/day from a food intake for cats (Sax, 1984)) in

diet when mink were maintained outdoors in winter (Wren et al., 1987), and increased toxicity of methylmercury when rats were maintained at high or low temperatures (Yamaguchi et al., 1984 in Wren et al., 1987).

Rats exposed to 800 ppm inorganic mercury died (Gough et al., 1979). Pigs exposed to methylmercury intravenously by a 2 mg/kg bw dose had no toxic effects (Gyrd-Hansen, 1981). Rats exposed to 0.2, 1, and 5 ppm methylmercury in diet (estimated doses of 0.015, 0.075, and 0.375 mg/kg bw/day (Sax, 1984)) reached equilibrium with diet in 6 months (Berglund and Berlin, 1969). Toxic effects were not observed, and concentrations in brain tissue were 8 ppm (Berglund and Berlin, 1969). The loss rate from tissue was 0.02 day^{-1} (Berglund and Berlin, 1969). Subchronic dietary exposure of rats to methylmercury resulted in decreased body weights at dose levels of 0.13 mg/kg bw/day (Soares et al., 1981).

Methylmercury administered to rats in a single dose of 10 mg/kg bw caused changes in cerebellar neurons, swelling of the granular cells in the cerebellar hemispheres, and changes in the granulated endoplasmic reticulum (Syversen et al., 1981). Methylmercury administered daily to rabbits at 7.5 mg/kg bw for 1 to 4 days produced degenerative changes in cerebellar and cerebral neurons (Jacobs et al., 1977).

Kidney and liver accumulate the highest amounts of methylmercury (Gyrd-Hansen, 1981: Berglund and Berlin, 1969). Concentrations of 8 ppm brain tissue or higher correlate with neurological symptoms in cats and dogs, while in mice and rats concentrations of 10 ppm or higher and 49 ppm, respectively, correlate with neurological symptoms (Berglund and Berlin, 1969). A single dose of methylmercury of 10 mg/kg bw administered to rats resulted in brain tissue concentrations of 1.4 to 2.2 ppm (Syversen et al., 1981).

The whole body half-life of methylmercury varies from 7 days in mice to 29 days in sheep, while primates have whole body half-lives exceeding 50 days (Gyrd-Hansen, 1981). The half-life of methylmercury in blood was 25 days for pigs (Gyrd-Hansen, 1981). The half-life in rats is 15 to 20 days (Swensson and Ulfvarson, 1968b).

Bioaccumulation Of Mercury

Aquatic Ecosystems--Methylmercury is soluble in water, and can be readily accumulated into biological tissue (Gough et al., 1979). Observations indicate that mercury is magnified within aquatic food chains but that concentrations in terrestrial animals are low unless diets are highly contaminated (NAS, 1978). Aquatic invertebrates such as mussels (Margaritifera margaritifera) have BCFs for inorganic and organic mercury of 302 and 2,463, respectively (Mellinger, 1973). Freshwater clams (mixed species) exposed to mercury at levels below the detection limit of 0.03 ppb in water concentrated mercury over 4,000 times as compared to the detection limit (Wren and MacCrimmon, 1986).

The levels of organic mercurials in aquatic invertebrates and plankton are extremely variable due in part to variations in food habits (Moore and Ramamoorthy, 1984), and physical location in the water column (Hamelink et al., 1977). Zooplankton near the bottom had higher levels of mercury than those found in the upper portions of the water column (Hamelink et al., 1977).

In fish, uptake has been observed to be proportional to water concentration, and can be predicted by correlating water concentration with rate of oxygen consumption (Rogers and Beamish, 1981). However, biota mercury levels exhibit great variability between adjacent bodies of water due to differing environmental conditions (Wren and MacCrimmon, 1986). Methylmercury uptake in fish increases with increasing temperature and water concentration of mercury (Rodgers and Beamish, 1981), and increases in lakes with lower pH (Wren and MacCrimmon, 1986). Other factors influencing uptake are size, or age of fish, breeding status, food ingestion rate, species, and metabolic differences (Huckabee et al., 1979).

Fish BCFs are documented as high as 10^8 (Johnels et al., 1967). Johnels et al. (1967) observed concentration factors of 3,000 in pike muscle, while Hannerz (1968) found concentration factors in pike muscle to be 2,000. Whole body mercury content in fish does not differ significantly from muscle (Phillips, 1980), so that values for muscle can be compared to whole body

values. Methylmercury makes up the larger proportion of tissue mercury in fish, in general >90 percent (Huckabee et al., 1979).

For rainbow trout exposed for 3 weeks to methylmercury in water at approximately 15°C, the BCF was 1,800 (Phillips and Buhler, 1978). These fish were probably not at equilibrium, as it can take as long as several months for fish to equilibrate with mercury in the environment. For example, Snarski and Olson (1982) observed that whole body residues at 41-weeks were double those observed at 60 days. In addition, BCFs for rainbow trout have been observed as high as 127,000 for the same temperature (Reinert et al., 1974); in another study BCFs for rainbow trout following exposure to methylmercury for 90 days were 8,000 (Willford and Reinert, 1973). Concentration factors in brook trout muscle were 30,000 for a 144 week exposure to methylmercury (McKim et al., 1976).

Fish tissue mercury levels increase with trophic level, and are higher in predatory fish than prey species of a similar age (Wren and MacCrimmon, 1986). BAFs for bluntnose minnow, smelt, and white sucker were below 10,000, while the BAF for pike was 32,000 (Wren and MacCrimmon, 1986). It appears that about 50 percent of this is due to bioconcentration while 50 percent is due to biomagnification (Burrows and Krenkel, 1973; Huckabee et. al., 1979). Other authors indicate that uptake from food may outweigh uptake from water as the primary source of mercury accumulation by fish (Phillips and Buhler, 1978; MacCrimmon et al., 1983). The actual amount of mercury derived from food or water is probably dependent on trophic level, as indicated by data from Jernelov (1972) where prey fish obtained 10 percent of their mercury residues from food, while pike obtained 50 percent of their mercury residues from food. Mercury accumulation is probably less from a natural diet than an artificial one (Snarski and Olson, 1982), so that laboratory studies using a commercial fish food may give artificially high accumulation rates.

Terrestrial Ecosystems--Birds feeding primarily on vegetation or terrestrial food sources have lower mercury contents than birds that feed on aquatic food sources (Nriagu, 1979). Piscivorous birds have mercury levels approximately 10 times higher than levels found in diet (Greichus et. al.,

1973). Elevated mercury levels are found in ospreys (Nriagu, 1979), with concentrations reaching 18 and 45 ppm in liver and kidneys, respectively. A nationwide survey of 5,200 adult mallards and black ducks (*Anas rubripes*) revealed muscle tissue levels of 0.08 to 0.33 ppm (Heath and Hill, 1974).

Ducks, pheasants, and chickens fed three different concentrations of mercury in diet magnified mercury by factors of 1.05 to 2.82 in kidney, 0.655 to 2.12 in liver, and 0.909 to 1.33 in muscle (Gardiner, 1972). Quail fed 4 ppm methylmercury for 18 weeks accumulated 21 ppm in liver and 8.4 ppm in carcass (Dieter and Ludke, 1975). Mallards fed 0.1 and 0.6 ppm in diet accumulated 6 to 9 ppm in eggs (Heinz, 1975). Goshawks consuming mercury contaminated chickens had magnification factors ranging from 3.23 in muscle to 14.4 in liver (Borg *et al.*, 1970). Greichus (1973) observed BMFs of 6 and 14 in white pelicans (*Pelecanus erythrorhynchos*) and double-crested cormorants (*Phalacrocorax auritus*) consuming freshwater fish.

Fate Of Mercury In The Environment--Mercury in sediments tends to be in the inorganic form (Snarski and Olson, 1982), and is methylated in the top layer (Fagerstrom and Jernelov, 1972). Because microorganisms are capable of converting inorganic and organic mercury compounds into highly toxic methylmercury and dimethylmercury, any form of mercury in the environment should be considered hazardous (EPA, 1980e: EPA, 1985c). The synthesis of methylmercury from other forms of mercury by bacteria in sediment or water is the major source of methylmercury in the aquatic environment (Boudou and Ribeyre, 1983). Methylation in the water column has also been indicated by Furutani and Rudd (1980).

Mercury is methylated in the intestines of fish (Jernelov, 1972: Rudd *et al.*, 1980). Methylation also occurs in the mucous layer of fish, and by enzymatic processes, although these sources of methylmercury are not as significant as dietary intake (Huckabee *et al.*, 1979: Boudou and Ribeyre, 1983). Demethylation also occurs in the environment (Eisler, 1987), the gastrointestinal tract of mammals (Clarkson *et al.*, 1984), and in the liver and kidneys of fish (Burrows and Krenkel, 1973). Since methylmercury is the

more toxic and readily accumulated form of mercury, it should be assumed that any mercury in the environment has the potential to be in the form of methylmercury.

5.2.5.2 Surface Water Ingestion

Small Mammals--

Table 5.2-34 lists the water concentrations that correlate with the toxic effects levels in diet or water based on daily water intake for each species. A chronic dietary NOEL for rats of 0.075 mg/kg bw/day was estimated from data reported by Berglund and Berlin (1969). The chronic NOEL was lower than the subchronic LOAEL of 0.13 mg/kg bw/day, or the acute values reported for mammals that resulted in CNS effects (Table 5.2-34). Although using the chronic dietary NOEL results in higher water concentrations than using the subchronic LOAEL would, there is less uncertainty in the estimate (see Section 5.1). From the NOEL and the daily water intake for rats, the following water concentration is derived:

$$\frac{\text{NOEL}}{\text{Intake/kg bw/day}} = \frac{0.075 \text{ mg/kg bw/day}}{0.125 \text{ l/kg bw/day}} = 0.6 \text{ mg/l}$$

An uncertainty factor of 5 for interspecific variation was used in calculating the acceptable water concentration. The water concentration 0.015 ppm, represents an acceptable surface water concentration for mammals.

Birds--All data for birds were based on subchronic studies. The lowest LOAEL was 0.21 mg/kg bw/day in diet for chickens. The LOAEL and the water ingestion for chickens was used to calculate an acceptable water concentration of:

$$\frac{\text{LOAEL}}{\text{Intake/kg bw/day}} = \frac{0.21 \text{ mg/kg bw/day}}{0.2 \text{ l/kg bw/day}} = 1.05 \text{ mg/l}$$

Applying an uncertainty factor of 50 to convert the subchronic LOAEL to a chronic NOEL, and a factor of 5 for interspecific variability, yields an acceptable water concentration of 0.0034 mg/l (3.4 ppb).

Table 5.7-34. Toxic Effects Levels of Mercury in Mammals and Birds by Ingestion

Species	Hg Type	Exposure Route	Dose	Effect	Acceptable Water Concentration (ppm)	Source
Rat	O	diet	0.075 mg/kg bw/day	No observed effects	0.015	Berglund and Berlin, 1969
Rat	O	oral	10 mg/kg bw/day	Affect CNS	0.016	Syversen et al., 1981
Rat (male)	O	diet	0.13 mg/kg bw/day	Decrease body weight	0.0042	Soares et al., 1973
Rabbit	O	oral	7.5 mg/kg bw/day	Affect CNS	0.0091	Jacobs et al., 1977
Chicken	I	water ingestion	125 ppm (31.25 mg/kg bw/day)	No observed effects	0.63	Parkhurst and Thaxton, 1973
Chicken	[illegible]	diet	0.21 mg/kg bw/day	Lethal	0.0034	Soares et al., 1973
Pheasant*	O	oral	0.64 mg/kg bw/day	Decrease reproduction	0.01	McEwen et al., 1973

I = inorganic, O = organic.
* = Calculated using water intake for chickens of 0.25 l/kg bw/day.

Source: ESE, 1988.

The acceptable water concentration for birds for organic mercury is 2 orders of magnitude lower than acceptable water concentrations for inorganic mercury for chickens (Table 5.2-34). Acceptable concentrations for organic mercury should therefore be protective for organic mercury.

The water concentration derived from toxicity to birds is lower than the concentration dervived from chronic toxicity to mammals. A mercury concentration in surface water of 0.0034 mg/liter (3.4 ppb) derived from toxicity to birds is assumed to be protective of all wildlife species consuming water at RMA. The corresponding sediment criterion is 3.4 ppm.

5.2.5.3 Aquatic Life

The EPA water quality criteria for the protection of aquatic organisms and their uses are based on a Final Residue Value derived using human guidelines, and so were considered inappropriate for this analysis. Site-specific water criteria for the protection of aquatic life were derived from the lowest chronic value or the Final Residue Value. Adverse effects were observed in D. magna for chronic exposure to 0.04 ppb methylmercury. Applying an uncertainty factor of 10 to convert the chronic LOAEL to a NOEL, an acceptable water concentration of 0.004 ppb is derived.

Due to the tendency of methylmercury to bioaccumulate, a Final Residue Value was calculated from a dietary intake that resulted in behavioral effects in mallard ducklings of 0.1 ppm (Heinz, 1975). The BCF reported by EPA is for fathead minnows only; however, ducks feed on a variety of aquatic life, and the BCF values for plants and invertebrates are much less than 81,700. The maximum BCF for a plant or an invertebrate was 13,000 for Scenedesmus obliquus exposed to phenylmercuric chloride (EPA, 1985c). BCFs for fish species that would occur at RMA are also less than 13,000. The Final Residue Value calculated using the BCF of 13,000 and an MPTC of 0.1 ppm is 0.0077 ppb.

The water concentration of [illegible] ppb, derived from chronic toxicity to Daphnia, is used to represent toxicity to [illegible] life because it is lower than the water criteria esitimated using the Final Residue Value approach.

The corresponding sediment criterion is calculated as follows:

$$C_{sed} = C_w \times K_d \quad (8)$$

where: K_d = 1,000 (see Section 5.2.5.6)

$$C_{sed} = 0.004 \text{ ppb} \times 1{,}000$$

$$C_{sed} = 0.004 \text{ ppm}$$

5.2.5.4 Aquatic Pathway Analysis

Introduction To Aquatic Pathway Analysis

This Pathway Analysis is based on the bald eagle sink food subweb and includes all major food chains leading to the selected sink species (Cohen, 1978). Because the same organisms or groups of organisms appear in more than one food chain throughout the web, percentage contributions to the food subweb for each organism or compartment have been estimated based on existing literature. The subweb has been simplified (e.g., bluegill represent all fish species at that trophic level), because of the limited data available.

The bald eagle is a federally listed endangered species and is a component of food webs on RMA. The bald eagle was selected as the target species because of its endangered status and because it represents the highest trophic level affected by the bioaccumulation of contaminants through aquatic food chains. Aquatic organisms are considered to be the most important links in the bald eagle food web because they are constantly exposed to the contaminants in their environment via surface adsorption, absorption, and uptake across respiratory membranes; thus, the potential for bioconcentration tends to be large. The "no effects" level is based on sublethal effects levels obtained from the scientific literature and presumes that if bald eagles are protected, other species will also be protected.

Methods

Six food transfer pathways ultimately terminating with the bald eagle were established as follows:

Pathway	Source	Trophic Level 1	2	3	4
1	H_2O	Snails	Mallard	Bald Eagle	
2	H_2O	Invertebrates	Mallard	Bald Eagle	
3	H_2O	Aquatic Plants	Mallard	Bald Eagle	
4	H_2O	Plankton	Bluegill	Pike	Bald Eagle
5	H_2O	Invertebrates	Bluegill	Pike	Bald Eagle
6	Soil	Terrestrial Plants	Small Mammals	Bald Eagle	

The combined food transfer pathways are presented in Figure 5.2-7. Pathways are developed based upon chemical parameters such as concentration factors, and biological parameters such as dietary habits. There are fewer pathways for mercury than for dieldrin because chironomids were not treated as a separate pathway: data were limited for RMA (Rosenlund et al., 1986), and unavailable in the literature researched. Although data were limited, snails were maintained as a separate pathway because (1) data indicated slightly lower BCFs, and (2) snails form a significant part of the mallards' invertebrate consumption.

All pathways (except Pathway Four) originate with water. The lowest step in the food chain is assumed to be in equilibrium with the aquatic environment, which gives equation (1):

$$BCF = C_b/C_w \qquad (1)$$

where: C_b = the concentration of mercury in the biota
C_w = the concentration of mercury in water

This equation is vital to the rest of the analysis. The end result, the total biomagnification factor (BMF) for the bald eagle, can be ultimately traced back through water to the sediment, because it is assumed that all mercury enters the water compartment from sediments before being taken up by the biological compartment: i.e.,

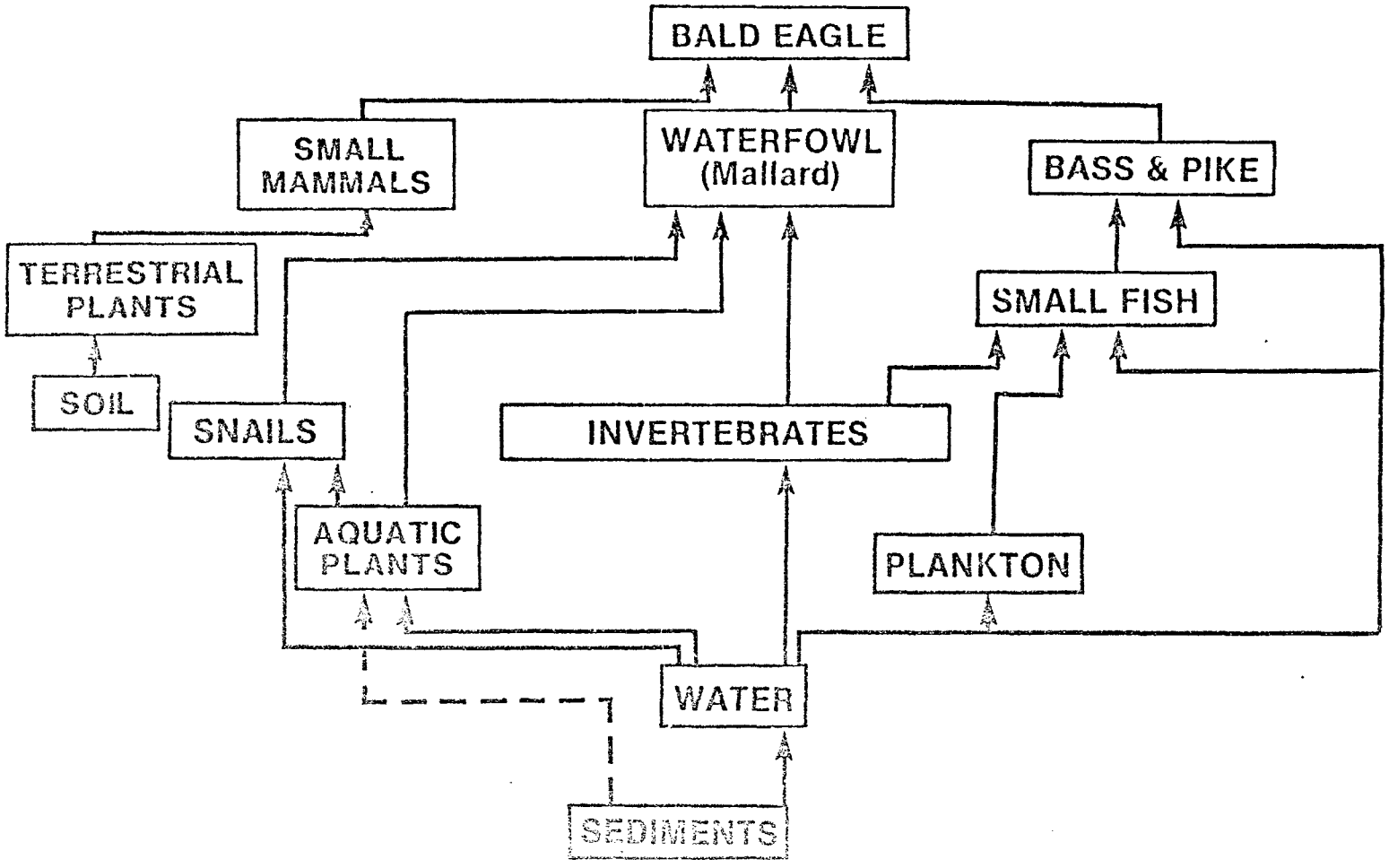


Figure 5.2-7

BALD EAGLE SINK FOOD WEB FOR MERCURY

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

$$C_w = \frac{C_{sed}}{K_d} \quad (7)$$

or solving for C_{sed}:

$$C_{sed} = K_d \times C_w \quad (8)$$

where: C_{sed} = concentration of mercury in the sediment
K_d = sediment-water partition coefficient

The K_d or sediment-water partition coefficient was calculated to be approximately 1,000 (Tucker, 1988). This is based on two lines of reasoning:

- Based on U.S. Army Medical Bioengineering Research and Development Laboratory (USAMBRDL) preliminary pollutant limit value (PPLV) documentation, soil K_d for Hg is 170. If soils contain 0.5% organic matter and sediments contain 5%, the estimated K_d would be 5 to 10 times higher in sediments than soils, or 850 to 1,700: and
- An EPA report gives a K_d for lakes of 600-900 (EPA, 1979a).

The method used in this aquatic Pathway Analysis is an adaptation of the Thomann (1981) bioaccumulation model of food chain transfer in aquatic ecosystems where each level is a step in the food chain:

Level #1 $BCF_1 = C_b/C_w$ (1)

Level #2 $BAF_2 = BCF_2 + f_2BCF_1$ (2)

Level #3 $BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1$ (3)

Level #4 $BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1$ (4)

The data for BCF values used in this analysis were from previously collected and documented RMA samples (Rosenlund et al., 1986) or from the available literature. Because mercury in the RMA lakes was below detection limits at the time of the Rosenlund et al. (1986) study, tissue concentrations from Rosenlund et al. (1986) were compared to the detection limit in water used at ESE (0.24 ppb). Published values as well as the RMA data were used for BCFs and BAFs, and geometric mean values were calculated to represent

bioconcentration by exposed organisms. Table 5.2-35 lists the BCF values utilized in this study. Only BCFs with exposure times of seven days or more were considered.

The mallard and the pike represent the waterfowl and fish fed upon directly by the bald eagle. Feeding habits for the consumer organisms are presented in Table 5.2-36.

The accumulation of mercury from water by lower trophic level organisms is due not only to bioconcentration, but to bioaccumulation as well. However, mercury residues resulting from water as opposed to diet cannot be effectively separated for animals at the lowest trophic levels. The BCF for the lowest trophic level is actually a BAF for small aquatic organisms with large surface area to volume ratios, such that bioconcentration from water tends to outweigh concentration from diet (Huckabee et. al., 1975).

Since the concentration of mercury in RMA lake water and sediments at Rosenlund et al.'s (1986) sampling locations is below current detection limits, concentration factors derived from RMA data potentially underestimate actual bioconcentration for organisms at specific locations in the RMA lakes. Because methylmercury BCFs derived from the literature were an order of magnitude higher for several groups of organisms, methylmercury BCFs from the literature were used to increase the range of BCF values that may be expected in lower trophic level organisms from RMA lakes. Literature values only for methylmercury BCFs were used to represent concentration by fish because the RMA field data represent BAFs, and using these field data would weight dietary contributions twice.

The model has been modified to be used for an entire food web as opposed to a single food chain by use of dietary percentage coefficients. The food term (f_1) is dependent on the trophic level in question and is calculated by the following equation:

$$f_1 = \frac{@ \times B \times \%}{k_2} \qquad (5)$$

Table 5.2-35. Bioconcentration Factors for Mercury Used in the Pathways Analysis

Species	Form	BCF	Tissue	Mean	Source
Plants					
Algae	MeHg	2,100	--		Havlik et al., 1979
Algae	MeHg	990	--		Havlik et al., 1979
Macrophytes	Total	983*	--		Rosenlund et al., 1986
				2,000	
Plankton					
Algae	MeHg	2.100	--		Havlik et al., 1979
Algae	MeHg	990	--		Havlik et al., 1979
Mixed	Total	820*	--		Rosenlund et al., 1986
				2,000	
Snails					
Mussel	MeHg	2,463	--		Mellinger, 1973
Snails	Total	750**	--		Rosenlund et al., 1986
				1,900	
Other Invertebrates					
Amphipod	MeHg	8,000	--		Zubarik and O'Connor, 1978
Mixed	Total	618*+	--		Rosenlund et al., 1986
				6,800	
Bluegill					
Mosquito fish	MeHg	2,500	whole		Boudou et al., 1979
Mosquito fish	MeHg	4,300	whole		Boudou et al., 1979
Bluegill	MeHg	373	whole		Cember et al., 1978
Bluegill	MeHg	921	whole		Cember et al., 1978
Bluegill	MeHg	2,400	whole		Cember et al., 1978
				1,500	
Pike					
Rainbow trout	MeHg	4,530	whole		Reinert et al., 1974
Rainbow trout	MeHg	6,620	whole		Reinert et al., 1974
Rainbow trout	MeHg	8,049	whole		Reinert et al., 1974
Pike	MeHg	3,000	muscle		Johnels et al., 1967
Pike	MeHg	2,000	muscle		Hannerz, 1968
				4,300	

* - Geometric mean, N = 50, C_w = 0.05 ppb
\+ - Geometric mean, N = 16, C_w = 0.05 ppb
** - Geometric mean, N = 2, C_w = 0.05 ppb
*+ - Geometric mean, N = 19, C_w = 0.05 ppb

Source: ESE, 1988.

Table 5.2-36. Summary of Feeding Habits, Pathways Analysis for Mercury

Species	Food Items	Percent in Diet	Source
Mallard	Snails	14	Swanson et al., 1979 Swanson et al., 1985
	Other Invertebrates[1]	30	
	Plants, Fruits[2]	30	Swanson et al., 1979 Swanson et al., 1985
	Annelids[3]	26	Swanson et al., 1979
Bald Eagle	Waterfowl	24	Cash et al., 1985 Todd et al., 1982
	Fish	66	Cash et al., 1985
	Mammals	10	Cash et al., 1985
Bluegill	Invertebrates	88	Martin et al., 1961
	Plankton, Algae	12	Martin et al., 1961
Pike	Fish[4]	100	Inskip, 1982

1 Includes Crustacea and Insecta.

2 Fruits were grouped with aquatic plants for this pathways analysis due to the possibility that mercury is absorbed by fruit. The term "fruits" includes miscellaneous seeds (Swanson et al., 1979; Swanson et al., 1985).

3 These food items were not utilized in the pathways analysis. Annelids are apparently washed into aquatic systems (Swanson et al., 1979) and were not included, because areas upgradient from the RMA lakes are assumed to be uncontaminated.

4 Pike are opportunistic feeders that will utilize other food sources, but are assumed to prey completely on fish for the sake of the analysis.

Source: ESE, 1988.

where: @ = Assimilation efficiency = $\frac{\text{ug absorbed}}{\text{ug ingested}}$

R = Total daily diet grams(g)/body weight(g)/day

k_2 = Depuration rate, day^{-1}

% = Percent of item in diet

The assimilation efficiency (@) could not be obtained for every animal addressed in this analysis. Various assimilation efficiencies ranging from 0.1 to 0.9 have been documented for fish (Phillips and Gregory, 1979). The assimilation efficiency can be related to the type of diet, the species, and metabolic rate. A geometric mean was calculated from data for fish; when a range was presented, the median of the range was used as the data point used to calculate the geometric mean (Table 5.2-37). A mean value of 0.40 was obtained for fish to represent uptake from a variety of diets.

An assimilation efficiency based on uptake of methylmercury in chickens was used to represent uptake by wild avian species. A geometric mean was calculated from data on the percentage retention of mercury compared to amount of mercury ingested for five concentrations at 4 and 7 weeks (Soares et al., 1973). The geometric mean was 0.49 for both 4 and 7 weeks (Table 5.2-37).

The depuration rate (k_2) includes loss due to growth, excretion, and metabolism (Table 5.2-38). Because rate constants have not been measured for each species in this analysis, a range of k_2 values was taken from the literature or derived by calculation using regression equations. The depuration of mercury in birds is influenced by feather growth and the rate will fluctuate with seasonal molting (Stickel, et. al., 1977). Half-life, T_b, can be used to calculate k_2 (Huckabee, et. al., 1975) as follows:

$$\frac{\text{Ln } 0.5}{T_b} = \text{Depuration rate}$$

A geometric mean k_2 of 0.0025/day for fish was calculated from several studies. Mercury is lost more slowly from fish than from mammals, and the rate of loss may be biphasic (Burrows and Krenkel, 1973). A half-life of approximately 60 days

Table 5.2-37. Assimilation Efficiencies for Fish and Birds Fed Methylmercury

Species	Assimilation Efficiency	Diet	Source
Fish			
Pike	0.19	natural	Phillips and Gregory, 1979
Predator	0.15	natural	Jernelov, 1968
Predator	0.40 - 0.45	natural	Jernelov, 1968
Pike	0.38	cow liver	Miettinen et al., 1970
Rainbow trout	0.52 - 0.71	artificial	Lock, 1975
Rainbow trout	0.68	artificial	Phillips and Buhler, 1978
Goldfish	0.71 - 0.89	artificial	Sharp et al., 1977
Geometric Mean	0.40		
Birds			
Chicken	0.429	artificial	Soares et al., 1973
	0.617		
	0.615		
	0.409		
	0.414		
	0.365		
	0.634		
	0.617		
	0.473		
	0.405		
Geometric mean	0.49		

Source: ESE, 1988.

Table 5.2-38. Methylmercury Loss Rates for Various Species

Species	Half-life (days)	k_2 day^{-1}	Source
Fish			
Bluegill	60	0.012	Burrows and Krenkel, 1973
Gambusia	1,000	0.00069	Huckabee et al., 1975
Pike	100	0.0069	Miettinen et al.,1970
Brook trout	1,000	0.0006	McKim et al., 1976
Geometric Mean For Fish		0.0025	
Birds			
Mallards	84	0.008	Stickel et. al., 1977
Fowl	35	0.020	Swensson and Ulfvarson, 1968a
Fowl	7 - 14	0.066	Gardiner, 1972
Geometric Mean For Birds		0.022	

Source: ESE. 1988.

was estimated from data presented by Burrows and Krenkel (1973) using a combination of both fast and slow depuration curves.

All k_2 values for fish were estimated from half-life data using equation (8).

A geometric mean of 0.022/day for birds was calculated from several studies (Table 5.2-38). In a study with mallards, birds lost approximately 50 percent of their methylmercury residues over an 84 day period (Stickel, et. al., 1977). A Values for k_2 of 0.066 and 0.020/day were derived from Gardiner (1972) and Swensson and Ulfvarson (1968a), using different types of domestic fowl. The half-life formula (8) was used to obtain k_2 for each of the above studies.

Inorganic mercury is converted to methylmercury (MeHg) by bacteria in sediments (Phillips and Buhler, 1978: Ramamoorthy and Blumhagen, 1984) and in the intestines of fish (Jernelov, 1972). This conversion affects the toxicity, bioaccumulation, and depuration rate of mercury in food chains leading to higher trophic levels. Using toxicity, uptake, and loss rate information for methylmercury will provide a more conservative and more defensible estimate of the behavior of mercury for the Pathway Analysis.

Pathway Analysis

The Pathway Analysis model is applied in the following section using the input parameters BCF, k_2, and f_2 described in Methods section. The species specific dietary habits are described for each of the higher trophic levels.

Pathway One: H_2O => Snails => Mallards => Bald Eagle--The BCF used for snails is derived from Rosenlund et al. (1986) data for mercury in snails from the RMA lakes, and other molluscs such as freshwater mussels that bioconcentrate methylmercury by factors of 2,463 (Mellinger, 1973). Snails were separated from the other invertebrates because they form a significant part of the mallards' invertebrate diet. The geometric mean was used to represent bioconcentration:

$$BCF_{snails} = 600 \qquad (i)$$

Freshwater clams bioconcentrate mercury by factors estimated to exceed 4,000 when compared to water data from a previous study on the same lake (MacCrimmon et al., 1983: Wren and MacCrimmon, 1986), but these data were not used in calculating the

mean because of the uncertainty in the estimate. Since data for snails were unavailable in the literature researched, the RMA data were used despite the size of the data set (N = 2).

The food term (f_2) is calculated by assuming that an adult mallard weighs approximately 1,100 g and consumes about 57.4 g total diet each day (Miller, 1975), of which for a breeding female 16.4 percent is snails (Swanson et al., 1985; Swanson et al., 1979). The BAF for a mallard is calculated by assuming that the first term in the Level #2 bioaccumulation equation (2) equals zero, because the amount of bioconcentration of mercury by nonaquatic organisms is considered to be negligible: i.e.,

$$BAF_2 = BCF_2 + f_2BCF_1 \qquad (2)$$

$$BAF_{mallard} = f_2BCF_{snail}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.49 \text{ x } (57.4 \text{ g}/1{,}100 \text{ g bw/day}) \text{ x } 14\%}{0.022/\text{day}} = 0.16 \qquad (5)$$

An adult eagle weighs approximately 4,500 g (Shafer, 1986) and consumes 255 g daily (Swies, 1986), of which 24 percent of the diet is birds (Cash et al., 1985; Sherrod, 1978). Energy requirements are different for wild birds than birds living in captivity, so these dietary quantities are only approximate (Sherrod, 1986). The following BAF values for an eagle are calculated by assuming that the first two terms in the Level #3 bioaccumulation equation (3) equal zero (bioconcentration by the eagle, and the mallard are negligible):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{snail}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f_3 = \frac{0.49 \text{ x } (255 \text{ g}/4{,}500 \text{ g bw/day}) \text{ x } 24\%}{0.022/\text{day}} = 0.30 \qquad (5)$$

When the BCF for snails is 600, the BAF for mallard is 96 and the BAF for eagle is 29.

Pathway Two: H_2O => Invertebrates => Mallards => Bald Eagle--The BCF for aquatic invertebrates other than molluscs was a geometric mean of one value for methylmercury concentration by amphipods and one value for total mercury based on RMA data (Zubarik and O'Connor, 1978; Rosenlund et al., 1986):

$$BCF_{invert} = C_b/C_w = 2{,}200 \quad (1)$$

To calculate the BAF for a mallard, the food term (f_2) remains the same as Pathway One except for the percent of food item in the diet. Invertebrates comprise approximately 30 percent of a mallards diet (Swanson et al., 1985; Swanson et al., 1979). Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{mallard} = f_2BCF_{invert}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.49 \times (57.4\ g/1{,}100\ g\ bw/day) \times 30\%}{0.022/day} = 0.35 \quad (5)$$

To calculate the BAF for an eagle in Pathway Two, the food term (f_3) remains the same as Pathway One. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{invert}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f3 = \frac{0.49 \times (255\ g/4{,}500\ g\ bw/day) \times 24\%}{0.022/day} = 0.30 \quad (5)$$

When the BCF for aquatic invertebrates is 2,200, the BAF for mallard is 770 and the BAF for eagle is 230.

Pathway Three: H_2O => Aquatic Plants => Mallard => Bald Eagle--The BCF for plants in Pathway Three is based on data for mercury concentrations in aquatic macrophytes (Rosenlund et al., 1986), and on methylmercury concentration values for algae (Havlik et al., 1979):

$$BCF_{plant} = C_b/C_w = 1{,}300 \quad (1)$$

The food term (f_2) for mallard remains the same as previous pathways except for the contribution plants and fruits make to the mallard diet (30 to 31 percent).

Bioaccumulation is calculated using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$

$$BAF_{mallard} = f_2BCF_{plants}$$

where: $BCF_2 = 0$

$$f_2 = \frac{0.49 \text{ x } (57.4 \text{ g}/1{,}100 \text{ g bw/day}) \text{ x } 30\%}{0.022/\text{day}} = 0.73 \quad (5)$$

To calculate the BAF for an eagle in Pathway Three, the food term (f_3) remains the same as previous pathways. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$

$$BAF_{eagle} = f_3f_2BCF_{plant}$$

where: $BCF_3 + f_3BCF_2 = 0$

$$f_3 = \frac{0.49 \text{ x } (255 \text{ g}/4{,}500 \text{ g bw/day}) \text{ x } 24\%}{0.022/\text{day}} = 0.30 \quad (5)$$

When the BCF for aquatic plants is 1,300, the BAF for mallard is 455 and the BAF for eagle is 140.

Pathway Four: H_2O => Plankton => Bluegill => Pike => Bald Eagle--Pathways leading to the bald eagle via fish are more complex because bioconcentration occurs at each trophic level. This introduces a fourth factor into the BAF equation, and the eagle is at Level #4 instead of Level #3.

The BCF for plankton is based on data for algae (which can be attached or planktonic) and for mixed planktonic species. From data presented by Havlik et al. (1979), bioconcentration of methylmercury in two species of algae were 990 and 2,100. Data from Rosenlund et al. (1986) indicate a mean BCF of 820 for plankton from RMA lakes. The BCF used to represent plankton in the Pathway Analysis is a geometric mean:

$$BCF_{plankton} = C_b/C_w = 1{,}200 \quad (1)$$

Methylmercury uptake in fish increases with increasing temperature and mercury concentration in water (Rodgers and Beamish, 1981). The bioavailability of mercury as a function of water quality and sediment conditions will affect uptake as well (Wren and MacCrimmon, 1986). BCF values for whole fish are preferable for the Pathway Analysis, because higher level predators have the opportunity to consume all or part of the prey. Concentration factors for various fish tissues are similar according to some studies, and dissimilar according to others (EPA, 1985c): for consistency, whole body concentrations were used in calculating a mean BCF unless data for muscle were for the species in question or the data base was limited. The BCF for bluegill is derived from studies on methylmercury uptake by small fish:

$$BCF_{bluegill} = C_b/C_w = 1{,}500 \qquad (1)$$

Based on data from Chadwick and Brocksen (1969), fish consume approximately an amount equal to 3 percent of their body weight daily. It is assumed for the purposes of the analysis that regardless of interspecific variability and differences in metabolic rate that the total daily intake term (R) is 0.03 regardless of bluegill or pike body weight. Various algal forms account for approximately 12 percent of the bluegills diet (Martin et al., 1961): this value was used for the percent of plankton in the bluegill diet. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2 BCF_1 \qquad (2)$$

$$BAF_{bluegill} = BCF_{bluegill} + f_2 BCF_{plankton}$$

$$\text{where: } f_2 = \frac{0.40 \times 0.03/\text{day} \times 12\%}{0.0025/\text{day}} = 0.58 \qquad (5)$$

The BCF for the pike is derived from several studies on large fish. Values used to calculate the mean were on a whole body basis, or were for muscle tissue from pike:

$$BCF_{pike} = C_b/C_w = 4{,}300 \qquad (1)$$

It is assumed that pikes feed entirely on bluegills for the sake of this analysis. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \quad (3)$$
$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{plankton}$$

where: $f3 = \frac{0.40 \times 0.03/day \times 100\%}{0.0025/day} = 4.8$ (5)

The eagle food term (f_4) was based on a 4,500 g eagle consuming 255 g daily, of which 66 percent of the diet is fish (Cash et al., 1985). The first term of the Level #4 equation equals zero, because bioconcentration by nonaquatic organisms is considered to be negligible. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \quad (4)$$
$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{plankton}$$

where: $BCF_4 = 0$

$f_4 = \frac{0.49 \times 255\ g/4{,}500\ g\ bw/day \times 66\%}{0.022} = 0.83$ (5)

When the BCF for plankton is 1,200, the BAF values for bluegill and pike are 2,200 and 15,000, respectively. The BAF for bald eagle is 12,000.

Pathway Five: H_2O => Invertebrates => Bluegill => Pike => Bald Eagle--The BCF for aquatic invertebrates other than molluscs was a geometric mean of one value for methylmercury and one value for total mercury based on RMA data (Zubarik and O'Connor, 1978; Rosenlund et al., 1986):

$$BCF_{invert} = C_b/C_w = 2{,}200 \quad (1)$$

The BCF for bluegill is 1,500, or the same as Pathway Four. The bluegill food term (f_2) remains the same as Pathway Four except for the percentage of the food item in the diet. The bluegill diet consists of approximately 88% invertebrates. Using equations (2) and (5):

$$BAF_2 = BCF_2 + f_2BCF_1 \quad (2)$$
$$BAF_{bluegill} = BCF_{bluegill} + f_2BCF_{invert}$$

$$\text{where: } f_2 = \frac{0.40 \times (0.03/\text{day}) \times 88\%}{0.0025/\text{day}} = 4.2 \qquad (5)$$

The BCF for pike (4,300) is the same as in Pathway Four. The food term (f_3) for the pike also remains the same as Pathway Four. Using equations (3) and (5):

$$BAF_3 = BCF_3 + f_3BCF_2 + f_3f_2BCF_1 \qquad (3)$$

$$BAF_{pike} = BCF_{pike} + f_3BCF_{bluegill} + f_3f_2BCF_{invert}$$

$$\text{where: } f_3 = \frac{0.40 \times (0.03/\text{day}) \times 100\%}{0.0025/\text{day}} = 4.8 \qquad (5)$$

The first term of the Level #4 equation equals zero, because bioconcentration by nonaquatic organisms is considered to be negligible.
The food term (f_4) for the eagle remains the same as Pathway Four. Using equations (4) and (5):

$$BAF_4 = BCF_4 + f_4BCF_3 + f_4f_3BCF_2 + f_4f_3f_2BCF_1 \qquad (4)$$

$$BAF_{eagle} = f_4BCF_{pike} + f_4f_3BCF_{bluegill} + f_4f_3f_2BCF_{invert}$$

$$\text{where: } f_4 = \frac{0.49 \times (255\ g/4{,}500\ g\ bw/\text{day}) \times 66\%}{0.022} = 0.83 \qquad (5)$$

When the BCF for aquatic invertebrates is 2,200, the BAF values for bluegill and pike are 11,000 and 56,000, respectively. The BAF for eagle is 46,000.

Results and Discussion

BAF values as derived for the individual pathways (Table 5.2-39) represent accumulation in separate single food chains. To derive overall accumulation in the entire food web, variations of the following equation are used:

$$BMF_i = BCF_i + \Sigma f_i BAF_{i-1}$$

For each of the major trophic levels in the aquatic Pathway Analysis, total biomagnification is presented in Table 5.2-40. Total BMF represents accumulation of residues originating in sediments, soil, and water by lowerorganisms directly;

Table 5.2-39. Summary of Bioaccumulation Factors for each Species in the Pathways Analysis for Mercury.

	Bioaccumulation Factors				
	Bluegill	Pike	Mallard	Mammal	Eagle
Pathway 1	--	--	96	--	29
Pathway 2	--	--	770	--	230
Pathway 3	--	--	460	--	140
Pathway 4	2,200	15,000	--	--	12,000
Pathway 5	11,000	56,000	--	--	46,000
Pathway 6	--	--	--	4.3	0.085

Source: ESE, 1988

Table 5.2-40. Total Biomagnification of Mercury Residues for each of the Key Organisms in the Aquatic Pathways Analysis.

Organism	Level	Equation	BMF
Mallard	#2	$\Sigma f_2 BCF_1$	1,300
Bluegill	#2	$BCF_2 + \Sigma f_2 BCF_1$	11,000
Pike	#3	$BCF_3 + f_3 BMF_{bluegill}$	59,000
Eagle	#3, #4	$f_4 BMF_{pike} + f_3 BMF_{mallard} + BMF_{terrestrial}$	50,000

Source: ESE, 1988.

and accumulation of residues by higher organisms via food chain exposure. Because dietary percentage contributions have been considered, net residue accumulation is a function of the accumulation of residues by the lower trophic levels.

Total BMF can be used to determine maximum allowable or "no effects" levels of mercury in sediments and soils by relating sediment or soil concentration to a MATC as follows (Tucker, 1986):

$$\frac{MATC}{\text{Total BMF}} = C_w \qquad (6)$$

and,

$$C_{sed} = C_w \times K_d \qquad (8)$$

where: $K_d = 1{,}000$

The lowest tissue concentration at which health effects are observed in an avian species (Table 5.2-41) is divided by the BMF for the eagle: thus, giving the sediment concentration at which "no effects" are likely to occur to key organisms at the top of the food web. The most sensitive avian species is used as opposed to one most closely related to the target organism because no other safety factors have been considered in the analysis. The goal of the "no effects" level is to protect populations as opposed to individuals, with the exception of members of an endangered species.

From regression equations presented by Heinz (1980), egg concentrations can be correlated with blood, muscle, and liver concentrations for mallards. The egg:liver concentration ratio is 1:2.52, and the egg:muscle concentration ratio is 1:1.02. Using these concentration ratios to relate egg concentration to tissue concentrations for the Heinz (1975) data yields corresponding liver and muscle concentrations of 2.52 and 1.02 ppm, respectively. The corresponding liver and muscle concentrations for the Heinz (1976) egg concentration (5.46 ppm) are 13.76 and 5.57 ppm, respectively. The mallard muscle concentration of 0.8 ppm appears to be the lowest tissue concentration that can be correlated with toxic effects.

Table 5.2-41. Toxic Effects of Methylmercury on Birds

Species	Organ	ppm	Effect	Source
Mallard	egg	1	Alter behavior	Heinz, 1975
Mallard	egg	5.46	Alter behavior Decrease survival	Heinz, 1976
Mallard	muscle	0.8	Alter nesting behavior, reduce number of off-spring	Heinz, 1979
Redtail Hawk	liver	20	Lethal	Fimreite and Karstad, 1971
	muscle	4.3	Lethal	
Pheasant	liver	1.8	Decreased egg hatchability	Hesse et al, 1975

Based on a muscle concentration 0.8 as the MATC, the "no effects" levels in water and sediments are:

$$\frac{\text{MATC}}{\text{Total BMF}} = C_w = \frac{0.8\ \text{ppm}}{50{,}000} = 1.6 \times 10^{-5}\ \text{ppm} \qquad (6)$$

$$C_{sed} = C_w \times K_d = (1.6 \times 10^{-5}\ \text{ppm}) \times 1{,}000 = 0.016\ \text{ppm} \qquad (8)$$

Thus, the "no effects" concentration in water is 1.6×10^{-5} ppm or 0.016 ppb, and the "no effects" level in sediments is 0.014 ppm. Since the bulk of the values in the Pathway Analysis are based on toxicity and accumulation values for methylmercury, which is more toxic and more accumulative than inorganic mercury, criteria derived using the Pathway Analysis should protect against inorganic mercury contamination as well.

The EPA chronic criteria for the protection of aquatic organisms are based upon food chain contamination with respect to humans. The FDA action level for mercury was divided by a BCF for fish, to arrive at criteria in water where fish would not accumulate more mercury than the FDA action level. The Pathway Analysis provides similar information, but uses a multiple food chain approach and tissue concentrations correlating with health effects levels for wildlife species. The Pathway Analysis provides a more comprehensive estimate of water and sediment criteria than the Final Residue Value, because the Pathway Analysis incorporates food habits information and can weight the importance of different dietary inputs.

5.2.5.5 Terrestrial Pathway Analysis

Methods

The terrestrial pathways must be addressed differently than the aquatic pathways, because data such as k_2 and assimilation efficiency are lacking for terrestrial organisms. BAFs are calculated by comparing C_b to C_{diet} or C_{soil}, and loss and uptake are therefore accounted for.

Pathway Six: Soil => Terrestrial Plants => Mammals => Bald Eagle

Available data on mercury in terrestrial syystems was limited in comparison to information on aquatic ecosystems; therefore information regarding uptake

of total mercury was considered as well as uptake of methylmercury. The ratio of mercury in soil to plant tissue, the EMF, can be estimated from results from a study by Shaw et al. (1986). Observed geometric mean magnification factors in different parts of various plant species on a wet weight basis ranged from 0.013 for fruit to 0.029 for leaves (Table 5.2-42). Each plant tissue type represents a separate data point, since the data were relatively extensive.

$$EMF = C_b/C_{soil} = 0.018 \quad (10)$$

The concentration of mercury in terrestrial mammals is usually low and is directly related to the concentration in the diet (NAS, 1978). Significant concentrations (exceeding 0.5 ppm) have been observed in animals grazing near a chlor-alkali factory (Shaw and Panigrahi, 1986): however, the plants in the area were highly contaminated, with some concentrations exceeding 5 ppm.

Sheep grazing on contaminated fields concentrated mercury from vegetation (Eisler, 1987). Highest tissue concentrations were observed in lung, kidney, and liver, while brain and muscle concentrations were lowest. For dietary concentrations ranging from 1.9 to 6.5 ppm in forage, tissue concentrations in sheep after 23 months were <1.0 ppm in muscle to 4.0 ppm in lung. After applying a correction factor to forage concentrations of 0.5 (based on values reported by Baes et al., 1984) to convert dry weight to wet weight, a geometric mean BAF of 1.14 was calculated (Table 5.2-42). Values below the detection were not used to calculate the mean.

For mink simultaneously exposed to methylmercury and PCBs, the geometric mean magnification from a 0.5 ppm commercial diet to internal organs was 23.2 (Wren et al., 1987). Whole body BAFs would probably be lower. In a field study that compared tissue concentrations in mink and otter with concentrations in fish, BAFs were 3.93 and 3.39, respectively (Foley et al., 1988). Accumulation factors for different organs were averaged to obtain one data point each for the Eisler (1987) and the Wren et al. (1987)

Table 5.2-42. Bioaccumulation Factors for Mercury for Terrestrial Species in the Pathways Analysis (Page 1 of 2)

Species	Form	Organ	N**	BAF	Mean	Source
Plants*						
	Total	root	18	0.019		Shaw and Panigrahi, 1986
		stem	18	0.014		
		leaf	18	0.029		
		fruit	8	0.013		
Geometric Mean for Plants					**0.018**	
Birds						
Mallard	MeHg	egg	11	9.38		Heinz, 1976; Heinz, 1979
		liver	5	14.5		
		kidney	5	18.2		
		muscle	5	7.92		
		brain	5	5.48		
		ovary	5	7.0		
Geometric Mean for Mallard					9.5	
Black duck	MeHg	kidney	--	5.3		Finley and Stendell, 1978
Black duck	MeHg	liver	--	7.7		Finley and Stendall, 1978
Geometric Mean for Black Duck					6.4	
Chicken	MeHg	liver, kidney	--	40		March et al., 1983
Chicken	MeHg	organs	--	8.7 - 24.7		March et al., 1983
Chicken	MeHg	muscle	--	7.3 - 18.2		March et al., 1983
Geometric Mean for Chicken					20.4	
Geometric Mean for Birds					11	

Table 5.2-42. Bioaccumulation Factors for Mercury for Terrestrial Species in the Pathways Analysis (Page 2 of 2)

Species	Form	Organ	N**	BAF	Mean	Source
Mammals						
Sheep	Total	brain	--	0.52		Eisler, 1987
		liver	--	1.14		
		kidney	--	1.48		
		lung	--	1.90		
Geometric Mean for Sheep					1.14	
Mink	MeHg	brain	3	7.09		Wren et al., 1987
		liver	3	36.9		
		kidney	2	47.6		
Geometric Mean for MeHg in Mink					23.2	
Mink	Total	liver	--	3.93		Foley et al., 1988
Otter	Total	liver	--	3.39		Foley et al., 1988
Geometric Mean for Mammals					4.3	

* A median value of mercury levels in summer and winter vegetation was used to calculate the BAF.

** Number of samples.

studies. The data for mink and otter collected by Foley et al. (1988) were treated as separate data points since the data were extensive (Table 5.2-42). The concentration factor for mammals is:

$$BAF_{mammal} = C_b/C_{diet} = 4.3 \quad (13)$$

For birds, mean BAF was derived from several studies. Data from two studies by Heinz were combined by organ to derive a geometric mean accumulation factor for mallard of 9.5. Data from March et al. (1983) were used to derive a data point for black duck of 6.4, and data from Finley and Stendell (1978) were used to derive a data point for chickens of 20.4. When the data are reported as a range, the midpoint of the range was used to calculate the geometric mean. To calculate the overall geometric mean, the three data points derived for each species were used.

The BAF for birds is:

$$BAF_{bird} = C_b/C_{diet} = 11 \quad (13)$$

Thus, the terrestrial part of the food web becomes:

$$\text{soil} \xrightarrow{0.018} \text{plants} \xrightarrow{4.3} \text{mammals} \xrightarrow{11} \text{eagles}$$

The fraction of mercury ingested by eagles is related to the amount of small mammals in their diet, which is 10 percent. The amount of bioaccumulation and transfer of mercury from one trophic level to the next is negligible compared to the amount from the aquatic sections of the food web. Total biomagnification in terrestrial systems from soil to bald eagle is 0.85, and correcting for the fraction of mammals in the eagle diet, biomagnification through the terrestrial food chain becomes 0.085.

Results and Discussion

Pathway Six, the terrestrial based food chain, forms 10 percent of the eagle diet. Soil criteria can be estimated using MATC and the accumulation in the terrestrial food chain as follows:

$$\frac{MATC}{\text{Total BMF}} = C_{soil} = \frac{0.8}{0.085} = 9.4 \text{ ppm} \qquad (6)$$

Based upon observed winter feeding behavior of bald eagles at RMA, Pathway Four forms approximately 90 percent of the eagle diet. This means that bioaccumulation in the terrestrial based food chain is 90 percent of total possible accumulation in the terrestrial food chain (0.85), or 0.76. This reduces the soil criterion by a corresponding amount:

$$\frac{MATC}{\text{Total BMF}} = C_{soil} = \frac{0.8}{0.76} = 1.1 \text{ ppm} \qquad (6)$$

Because eagles depend on terrestrial prey at RMA, the lower soil criterion should be used to represent the "no effects" level in soil on RMA.

The soil criterion derived from Pathway Six can also be used to predict toxicity to small mammals exposed to contaminants from ingesting contaminated soil. An exposure rate as a function of the acceptable soil criteria can be estimated from the soil criterion and the soil ingestion rate for small mammals as follows:

Soil Criterion x Soil Ingestion Rate = Daily Exposure

1.1 mg/kg soil x 0.000873 kg soil/kg bw/day = 0.00096 mg/kg bw/day

The exposure rate based on a soil criterion of 1.1 mg/kg soil is about two orders of magnitude lower than estimated chronic NOEL for rats (0.01 to 0.38 mg/kg bw), and the chronic LOAEL for mink (0.05 mg/kg bw/day), and therefore direct toxic effects are not expected at the criterion level of 1.1 mg/kg in soil. The daily intake of mercury from ingesting soil represents a conservative estimate as an assimilation efficiency of 100 percent is assumed.

Because biomagnification in the terrestrial food chain is less than 1, a terrestrial food web, based on the American kestrel as the top carnivore, will not be constructed for mercury.

5.2.5.6 Uncertainty Analysis

In the uncertainty analysis, all of the intake rates (R values) and percent of items in diet are treated as triangular distributions where the minima and maxima are known and a best estimate within that range has been determined. Using the triangular distribution as input, the best estimate will be more likely than values near either end of the range. Methodology for the uncertainty analysis is described in detail in the forthcoming Offpost Endangerment Assessment. Diets of each link on the sink food web are summarized in Table 5.2-43. Organic carbon content of the sediment of the RMA lakes is a measured value (EBASCO, 1988). In the upper 1 foot (ft) of sediment, organic carbon appears to follow a lognormal distribution with a mean of 0.65 percent and a standard deviation of 0.62 percent.

In this analysis, the diet of eagles is assumed to be supplied only by the aquatic food chain with mallards and pike as the prey. Therefore, the pathway for mercury in terrestrial systems was excluded in the uncertainty analysis. Assimilation, or absorption of ingested mercury in birds and fish was determined to follow a log-normal distribution with the mean and standard deviation of 0.488± 0.023 and 0.408± 0.100, respectively. The uncertainty analysis is applied to the best estimation of k_2, BCFs, K_d, and their effects on the resulting BMF, C_w, and C_{sed}.

The rate of depuration of absorbed mercury is apparently faster in birds than in fish (Stickel et al., 1977; Swenson and Ulfvarson, 1968a). Based on the two k_2 values from mallard drakes and fowl (Stickel et al., 1977; Swensson and Ulfvarson, 1968a, respectively) and the three averaged k_2 values from the breast of chicken, pheasant, and duck fed with 0.33 and 3.3 ppm mercury diets (as methylmercury dicyandiamide), a log-normal distribution with a mean of 0.042 day^{-1} and a standard deviation of 0.021 day^{-1} was chosen to be the most representative one. Mercury depuration rates in fish were estimated from four different fish. Applying these values to the distribution analysis results in a log-normal distribution with a mean of 0.003 day^{-1} and a standard deviation of 0.003 day^{-1}.

Table 5.2-43. Dietary Input Factors, Mercury Pathways Analysis.
R = Total Dietary Intake $(day)^{-1}$

	Minimum	Best Estimate	Maximum
Eagle	0.51	0.57	0.76
Mallard	0.45	0.52	0.93
Pike	0.01	0.03	0.05
Bluegill	0.01	0.03	0.05
Percent of Item in Diet			
Eagle/Mallard	14	28	42
Eagle/Pike	58	72	86
Mallard/Invertebrates	40	58	75
Mallard/Aquatic Plants	25	42	60
Bluegill/Plankton	6	12	18
Bluegill/Invertebrates	82	88	94

Source: ESE, 1988.

All available BCF data for aquatic plants and plankton were combined to yield a database with four values. Analyzing this database results in a log-normal distribution with a mean standard deviation of 1,163±246. Similarly, BCFs for snails and invertebrates were composited and weighted equally to yield a log-normal distribution with a mean of 1,311 and a standard deviation of 1,040. It was also determined that BCFs for mercury in bluegills and pike follow the same log-normal distribution with the mean standard deviation of 1,700±444 and 4,427±1147, respectively.

Considering the two K_d values determined directly from the lake sediments (Bonner and Bustamente, 1976) and the two sediment K_d values derived from the geometric mean of the lowest and highest K_d values in soils (Andersson, 1967: Aomine and Inoue, 1967), a log-normal distribution was chosen to represent the K_d distribution in sediments (mean=963 ml/g: standard deviation=211 ml/g).

Based on the input values determined above regarding mercury uncertainty analysis for bald eagles, the best estimate BMF is 4.30×10^4 with a 5 percent chance that the eagle BMF will be equal or less than 7.76×10^3 (or equal or greater than 3.56×10^5). The medium estimate of the water concentration that will not result in unacceptable tissue concentrations in bald eagles is 1.89×10^{-5} ppm, with lower and upper bounds of 3.01×10^{-6} and 1.12×10^{-4} ppm, respectively. The best estimate of the sediment concentration that will not result in unacceptable tissue levels is 1.80×10^{-2} ppm. There is a 5 percent chance that sediment concentrations of 2.09×10^{-3} ppm or less could result in unacceptable tissue concentrations: there is also a 5 percent chance that sediment concentrations up to 0.112×10^{-2} ppm are acceptable in the aquatic food chains for bald eagle.

5.2.5.7 Summary and Conclusions

Mercury is a highly toxic contaminant of aquatic ecosystems on RMA, and large BCFs are observed in aquatic organisms. The BCFs increase with increasing trophic level: thus, threatening animals at the top of the food web. BCF values for lower trophic level organisms were based on methylmercury and on RMA data, which measured total mercury. BCF values for fish were based strictly on methylmercury, because BCF values were higher

for methylmercury than for inorganic mercury. Toxcity data for avian species were also based on values for methylmercury, because in general methylmercury is more toxic.

"No Effects" levels in water based on the Pathwyas Analysis (0.016 ppb) are essentially equivalent to EPA chronic criteria for water (0.012 ppb). The "no effects" level in sediment based on the Pathways Analysis is 0.016 ppm, and in soil the "no effects" level is 1.1 ppm. For terrestrial biota consuming surface water, an acceptable level is 0.01 ppm. The lowest value in water (0.004 ppb) is based on toxicity to aquatic life, with a corresponding sediment criteria of 0.004 ppm.

The site-specific criteria for water, sediments, and soils are as follows:

Method	Water (ppb)	Sediment (ppm)	Soil (ppm)
Water Ingestion	3.4	3.4	NA
Aquatic Pathways Analysis	0.016	0.016	NA
Aquatic Life	0.004	0.004	NA
Terrestrial Pathway Analysis	NA	NA	1.1

5.3 CONTAMINANT EFFECTS

A variety of adverse effects are known to occur in biota as a result of exposure to many of the contaminants that are found on RMA. Lethal effects (e.g., effects resulting in death) and sublethal effects (e.g., behavioral effects, physiological effects such as eggshell thinning and reduced acetylcholinesterase levels in the brain) on individuals can result, and these can impact populations through decreased reproductive success. Other adverse effects (e.g., carcinogenic and teratogenic effects) may also occur but are more difficult to detect.

In order to establish a relationship between a contaminant and an observed effect on an organism, the contaminant must be present in the environment of the organism, a pathway must exist between the environment and the organism (e.g., direct exposure in the aquatic environment or food chain pathways), and the observed effect must be demonstrably related to the particular contaminant(s) being evaluated. For some chemicals, it is additionally appropriate to document the occurrence and concentration of particular contaminants (e.g., organochlorine pesticides) in the environment to correlate these values with specific effects.

This section synthesizes information on various specific contaminant effects in biota. These data are discussed in combination with data on contaminants in abiotic media and in biota from onpost and offpost sites. Where appropriate, the results of effect studies and contaminant concentrations were statistically analyzed in order to evaluate contamination effects. Statistical analyses of contaminant data in biota are discussed in Section 4.0. Detailed descriptions of statistical analyses used for contaminant and effects investigations are provided in Appendix B. Contaminant effects on vegetation, invertebrates, and aquatic ecosystems are discussed in turn in the sections that follow.

5.3.1 TERRESTRIAL VEGETATION

5.3.1.1 Community Ecology

Contaminant effects on community ecology, their variation among species, and food chain implications of the levels detected are discussed.

The distribution and species composition of terrestrial vegetation on RMA is the result of the existing natural vegetation, past land use practices (e.g., grazing, cropland development, RMA facility development), and current RMA land use management practices. These factors provide a background against which possible contaminant effects are evaluated.

Vegetation studies conducted by Shell/MKE (MKE, 1988) compared aspects of the community ecology of RMA with offpost control sites at Buckley ANG and the PCC. Observed differences in total vegetation cover between crested wheatgrass at RMA and Buckley ANG were statistically significant, while differences in total productivity were not. Species richness in native grassland on RMA was higher than at either of the offpost sites. The greater number of species recorded at RMA probably relates to the greater areal extent of native grasslands onsite, and the greater numer of samples taken. Comparisons of phenology revealed no detectable differences between RMA and the offpost control areas.

Plant communities in proximity to Basins A and F on RMA were compared with those from other portions of RMA. These areas were dominated by weedy communities, but were not found to be significantly different from the communities in other parts of RMA with respect to cover, production, or species composition. Comparisons with vegetation communities within major sites of contamination such as Basin A were not possible because the potential effects of chemical contamination could not be separated from the extensive surface disturbance and soil compaction that existed at the time of field studies.

5.3.1.2 Effects of Contaminant Levels in Terrestrial Plants

Arsenic levels observed in the leaves of sunflower samples from Basin A are within a range that is toxic to some species of plants (Section 4.3), but that is tolerated by others. Sunflowers within Basin A did not show signs of obvious phytotoxicity. Arsenic levels in the soils of Basin A may have contributed to the low diversity of plant species within the area, but the high level of physical disturbance and soil compaction in much of the area made this hypothesis difficult to evaluate.

Dieldrin was detected in sunflowers collected in Sections 26 and 36 (Basin C and Basin A areas, respectively), and morning glory collected in the vicinity of Basin A (no morning glory was found in the Basin C area of Section 26). Dieldrin is not a phytotoxic chemical, and no direct adverse effects would be expected from the levels detected.

The endrin level documented for sunflower leaves from Basin C (0.188 ppm) is lower than any documented hazardous level for the diet of birds or mammals. Pathways analysis for endrin (Section 5.2.5) also indicates that these levels are probably not a problem for higher taxa in the terrestrial food chain.

Lipid-soluble chemical contaminants (e.g., organochlorine pesticides) that might enter a plant through the roots would be expected to be translocated within the plant and concentrated in the oil-rich seed heads. The presence of contaminants in leaf samples but not in seed heads of sunflowers suggests that these contaminants may have been present in surface soil and deposited on the waxy cuticle of leaves during showers or surface disturbances.

5.3.2 INVERTEBRATES

Three invertebrate groups (aquatic snails, grasshoppers, and earthworms) were selected for population studies as a means of evaluating potential contaminant effects.

5.3.2.1 Aquatic Snails Populations

Snail samples were collected in five onpost lakes and two offpost control lakes. Data on snail weight and snail numbers were collected.

Sampling results indicated significant differences in snail population density between RMA lakes and offpost control lakes ($p > 0.001$) for 1986 and 1987, and for snail weights per unit area between onpost and control lakes in 1987. No significant differences were detected between control and onpost snail weights per unit area during 1986. Significant differences iin weight were detected between controls for 1986 and 1987 ($p > 0.001$), and among onpost lakes ($p > 0.01$) for 1986 but not in 1987.

The results of statistical analyses indicate that a very high degree of variability exists among sites and between years. Multiple regression analyses of snail results with the covariates of vegetation (substrate) weight, temperature, and pH indicated that these factors affected results. Interpretation of these analyses suggests that differences between onpost (contaminated) sites and offpost (control) areas are attributable to a number of environmental factors, some of which vary with time (e.g., temperature, amount of substrate, etc.). The lack of contaminant analyses for aquatic snails and the lack of pattern in variability do not allow any conclusions with respect to the possible effects of RMA contaminants on aquatic snail populations at RMA.

5.3.2.2 Grasshopper Populations

Grasshopper populations were surveyed at onpost and offpost control sites, in Section 26 (Basins C-F area) and Section 36 (Basin A area). Sample results were highly variable, and statistical analyses indicated no significant differences among sites. Field observations and vegetation data from sample sites indicated that grasshoppers were abundant in sample areas with forb cover, especially in areas such as Basin C where sunflowers dominated.

None of the seven target analytes were detected in samples from either the offpost or onpost control areas. Samples from Section 26 (Basin C-F area) contained organochlorine pesticides: aldrin (4 of 4 samples), dieldrin (4 of 4 samples), and endrin (3 of 4 samples) but no DDE, DDT, mercury, or arsenic. Samples from Section 36 (Basin A area) contained only one organochlorine pesticide, dieldrin (4 of 4 samples), but also contained mercury (2 of 4 samples) and arsenic (4 of 4 samples).

The highest level of mercury (0.103 ppm) detected in grasshoppers could pose a potential hazard to birds at upper trophic levels in the terrestrial food web because it exceeds the recommended bird dietary levels of 0.05 to 0.1 ppm (Eisler, 1987). Although this was a single sample, it was a composite of more than 50 individuals and represents an average value for the sample location. Levels of 0.5 ppm of mercury in the diet of birds can adversely affect reproduction (Section 5.2.6).

The maximum detected level of arsenic was 6.60 ppm in composite samples of grasshoppers from Section 36 on RMA. Arsenic does not tend to bioaccumulate in the terrestrial food chain, and no adverse effect levels are documented for invertebrates. However, levels this high may be hazardous to insectivorous animals. Eisler (1987) establishes a criterion of <2 mg/kg total arsenic in the diet of domestic livestock. Adverse effects on plants and acceptable levels in soils are discussed in Section 5.2.3.

The organochlorine pesticides aldrin, dieldrin, and endrin have high bioaccumulation factors. Grasshoppers provide a pathway component for the biomagnification of organochlorine pesticides such as aldrin, dieldrin, and endrin in terrestrial food chains on RMA. Levels in the range of those found in grasshoppers from Section 26 on RMA may produce sublethal effects in birds such as the American kestrel that consume grasshoppers (see discussion in Section 5.3.3.4, Avian Reproductive Success). Many bird and mammal species that inhabit RMA feed on grasshoppers, particularly at seasons when they are abundant. They are a major source of food for kestrels, pheasants, and other species important in RMA food webs.

Aldrin and dieldrin are treated together because of their similarity in structure and effect (see Section 5.2.2), and their effective concentrations are considered additive in terms of effect because aldrin is converted to dieldrin. The highest composite sample level of dieldrin detected in Section 36 was 0.446 ppm: no aldrin was detected from this area. In Section 26, dieldrin levels reached a high of 7.2 ppm, and aldrin levels reached 5.8 ppm. The concentrations of dieldrin found in bird species collected near sites of contamination are probably due in part to the consumption of grasshoppers and other invertebrates contaminated with aldrin/dieldrin. The role of grasshoppers in terrestrial food webs containing aldrin/dieldrin is discussed in Section 5.2.1.

Endrin was detected in grasshoppers at levels reaching 1.65 ppm in Section 26. Endrin is several times more toxic to wildlife than either aldrin or dieldrin. Dietary levels of 0.5 ppm are lethal to dogs, and levels of 3.0 ppm in the diet of birds are correlated with decreased embryo

survivability and weight loss. Although levels of 3.44 and 3.47 ppm of endrin were found in mourning dove tissue (Section 4.3), toxic levels of endrin were not detected in samples of species from RMA that prey on grasshoppers. It is possible that endrin levels found in grasshoppers could, through biomagnification, produce sublethal and/or lethal effects on insectivores and higher order consumers in RMA biota. Endrin effects through food chain pathways are discussed in Section 5.2.5.

Only one grasshopper species, Melanoplus sanguinipes, was represented in each of the 6 samples collected in contamination sites on RMA (in or near Basins A, C, and F). Four and six grasshopper species were found in the two onpost control area samples, respectively, and six species were found in each of the two offpost controls. M. sanguinipes was not found in any of the control samples. The differences in species richness (numbers) between control and contaminated sites is probably the result of the reduced diversity of vegetation in sites of contamination, which were dominated by sunflowers, and the corresponding food preferences of the grasshopper species involved. Melanoplus sanguinipes is a widespread omnivorous species known to prefer forbs (Capinera and Sechrist, 1982) and hence would be expected to forage in all areas sampled.

5.3.2.3 Earthworms Populations

Statistical differences were determined using a hierarchical set of orthogonal comparisons first to test for differences between population numbers at the offpost and onpost control sites, then compare controls as a group with the South Plants site. Results of population comparisons indicated that onpost and offpost controls were significantly different, and that controls as a group were significantly different from the South Plants site.

All earthworms identified in composite samples from all sites on and off RMA were of the genus Apporectodea, and those that could be identified to species were A. trapezoides. This species is the most common earthworm throughout much of the arid portions of the United States (Fender, 1988).

Four of the seven target analytes (arsenic, dieldrin, endrin, and mercury) were found in earthworms from the three sites surveyed. Contaminant levels in worm samples are presented in Table 4.3-1. Only arsenic showed significant differences among sites; the offpost control differed significantly from the onpost control, but no differences were detected between controls and the South Plants site. Arsenic levels were highest in the onpost control site, which also had the highest population levels: thus, it appears that population levels were not adversely affected by levels of RMA contaminants.

Dietary levels of both mercury and organochlorine pesticides could pose a hazard to animals that consume large quantities of earthworms (see preceding discussion for effects on grasshoppers). Pocket gophers and other species that feed on and around the roots of plants, where worms are usually found, may also be exposed. The relatively low density and patchy distribution of earthworms found during surveys suggest that this may not be a significant ecological problem at RMA.

5.3.3 VERTEBRATES

For vertebrates, AChE inhibition, impacts on prairie dog populations, eagle, and other birds of prey populations, and avian reproductive success were considered in the evaluation of contaminant effects.

5.3.3.1 Brain Acetylcholinesterase Inhibition

Birds

AChE assays were run on mallards from RMA (n = 9, mean = 14.84) and offpost control sites (n = 6, mean = 13.51) and pheasants from RMA (n = 6, mean = 21.77) and offpost control sites (n = 7, mean = 23.08). Neither of the RMA samples differed significantly from controls (Appendix B). Values for both of the RMA groups differed from the offpost control values by less than 20%, the level generally accepted as indicative of exposure to AChE-inhibiting toxicants (Robinson et al., 1988).

Assays were also run on one mourning dove, two golden eagles, and three red-tailed hawks found dead on RMA. The AChE level in the mourning dove (39.33) was higher than the mean of 16 for apparently normal mourning doves, as were

the values for golden eagles (23.89 and 28.99 on RMA compared to 14) and for red-tailed hawks (27.43, 23.89, and 28.99 on RMA compared to 19). Values for apparently normal birds are from Hill (1988).

Knittle and Tucker (1974) found no significant differences in AChE activity in avian species due to age or sex within species or the process of freezing and thawing of samples, but did detect reductions in AChE due to postmortem decomposition and differences among different areas of the brain. Studies conducted at Patuxent Wildlife Research Laboratories have shown age differences in avian brain AChE activity, particularly in altricial birds. Brains analyzed in this study were homogenized whole prior to taking a subsample in order to reduce any possible differences due to subsampling prior to homogenization.

Mammals

Black-tailed Prairie Dog--AChE levels were highest in samples from offpost control (mean = 16.45), followed by the onpost control (mean = 14.06), Section 36 (mean = 13.68), and TSY (mean = 10.69). Statistical analysis of all groups indicated that the combined onpost and offpost controls differed significantly from the combined onpost contaminated groups ($p < 0.01$), that the two control groups differed significantly from each other ($p < 0.05$), and that the two onpost contaminated sites differed significantly from each other ($p < 0.05$) (see Appendix B for detailed discussion).

Brain AChE inhibition exceeding 20 percent is generally considered indicative of exposure to AChE-inhibiting chemicals (Robinson et al., 1988). Neither the onpost control or Section 36 groups differed from the offpost control by 20 percent, but the TSY group was 35 percent lower then offpost controls. Several chemicals found in the area could contribute to the observed AChE inhibition. Arsenic compounds (arsenite ion and to a lesser extent the arsenate ion) are known to inhibit AChE (Olson and Christensen, 1980), as do some metal ions including cadmium, copper, and lead (Tomlinson et al., 1980). Some of these toxicants can have an accumulative effect in combination with the enzyme AChE (Olson and Christensen, 1981), but the mechanism of action is not known. These metal ions were found in the near

surface soils (0 to 1 ft) during soil assessment studies, but were not above the background levels for these soils.

Cottontail Rabbits--No significant differences were detected in brain AChE among cottontails from the three locations sampled. Differences in diet and level of fossorial activity may account for differneces in AChE respones between cottontails and prairie dogs.

5.3.3.2 Black-tailed Prairie Dog Populations

A number of wildlife species depend either directly or indirectly on the existence of prairie dogs. Rattlesnakes, desert cottontails, and burrowing owls use the burrows on prairie dog towns for cover and nesting, while many other birds utilize prairie dog towns as feeding and resting locations (Butts and Lewis, 1982; Clark et al., 1982). Badgers, coyotes, rattlesnakes, bald eagles, golden eagles, ferruginous and a variety of other hawks all prey upon prairie dogs at RMA. Black-tailed prairie dogs obviously hold an important position as a key species and as developer of their unique ecosystem on approximately 30 percent of RMA acreage (1,961 ha, 4,840 acres of prairie dog colonies).

The summer 1987 prairie dog minimum population estimates (prairie dogs per one hectare plot), the plot numbers, and dates of observation have been compiled and are shown in Table 5.3-1. An analysis of the mean and confidence limits of the prairie dog population is presented in Table 5.3-2. A mean of 19.9 black-tailed prairie dogs per ha was found at RMA in the summer. The extent of prairie dog colonies and the plot locations on RMA are displayed in Figure 5.3-1. The relationship between prairie dog colonies and shallow ground-water is shown on Figure 5.3-2.

Numbers of prairie dogs observed above ground in January 1988 on RMA plots are listed in Table 5.3-3. The mean number of prairie dogs on all plots counted in both summer and winter studies was 21.1 in the summer, and 21.0 in the winter. In general, the relative populations of prairie dogs counted in both winter and summer on the same plots were not correlated.

Table 5.3-1. Summer 1987 Population Estimates for Black-tailed Prairie Dogs on RMA.

PLOT	POPULATION	DATES_OF_OBSERVATION
RMA 1	28	6/4 - 6/6/87
RMA 2	10	6/4 - 6/6/87
RMA 3	33	6/4 - 6/6/87
RMA 4	10	6/10 - 6/12/87
RMA 5	10	6/10 - 6/12/87
RMA 6	35	6/10 - 6/12/87
RMA 7	18	6/10 - 6/12/87
RMA 8	7	6/10 - 6/12/87
RMA 9	37	6/16 - 6/18/87
RMA 10	10	6/16 - 6/18/87
RMA 11	14	6/16 - 6/18/87
RMA 12	26	6/16 - 6/18/87
RMA 13	6	7/16 - 7/18/87
RMA 14	40	7/16 - 7/18/87
RMA 15	9	7/13 - 7/15/87
RMA 16	18	7/13 - 7/15/87
RMA 17	28	7/16 - 7/18/87
RMA 18	12	7/13 - 7/15/87
RMA 19	20	7/13 - 7/15/87
RMA 20	27	7/16 - 7/18/87

Source: Clippinger, 1987

Table 5.3-2. Prairie Dog Population Mean (per hectare) and Confidence Limits for Summer 1987.

Mean: 19.9 prairie dogs per hectare.

Standard Deviation = s = 10.98

Variance = s^2 = 120.56

Standard Error (SE) = 2.455

At 95 % Confidence: 19.9 / ha ± 5.1 (± 25%)

At 90% Confidence: 19.9 / ha ± 4.2 (± 20%)

Source: Clippinger, 1987.

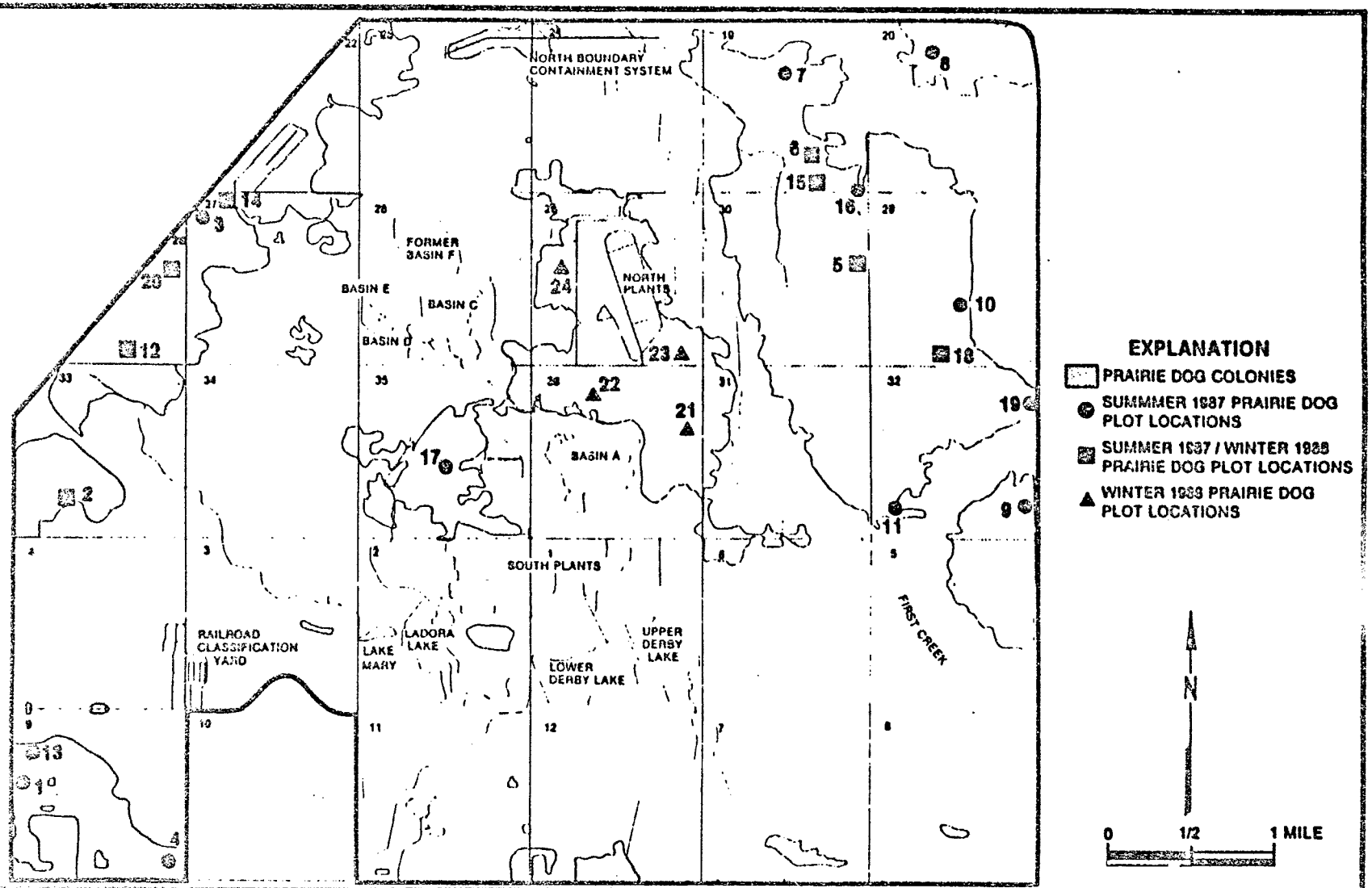
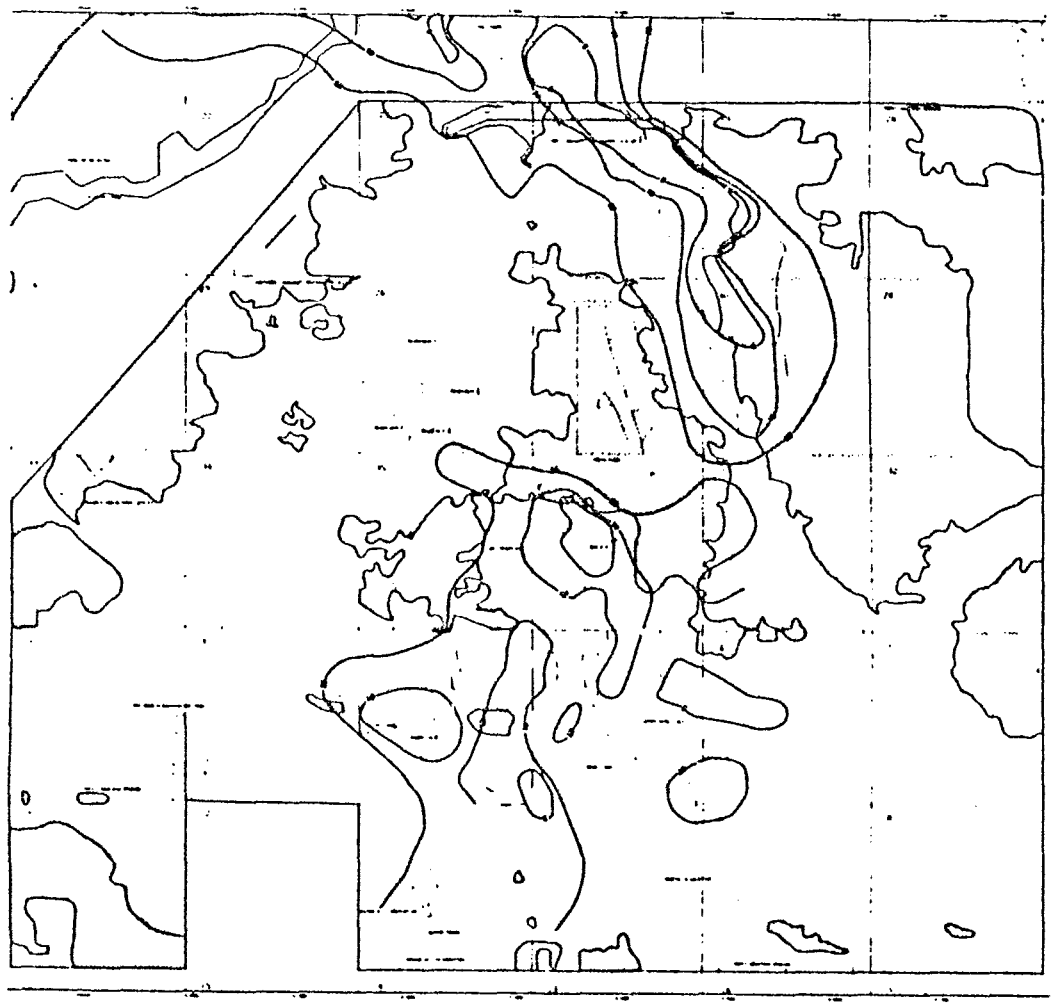


Figure 5.3-1

RMA PRAIRIE DOG COLONIES

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland



EXPLANATION

PRAIRIE DOG COLONIES

DEPTH TO GROUND WATER CONTOURS (C.I. 10 Feet)

N

0 1/2 1 MILE

Figure 5.3-2
RMA PRAIRIE DOG COLONIES AND SHALLOW GROUND WATER CONTOURS

SOURCE: ESE, 1987

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

Table 5.3-3. January 1988 Survey: Active Prairie Dogs per Hectare.

Plot	Maximum Prairie Dog Count	Dates Observed
RMA 2	7	1/14, 1/25-1/26/88
RMA 5	13	1/26 - 1/28/88
RMA 6	38	1/26 - 1/28/88
RMA 12	13	1/14, 1/25-1/26/88
RMA 14	46	1/11, 1/13-1/14/88
RMA 15	11	1/25 - 1/27/88
RMA 18	12	1/26 - 1/28/88
RMA 20	28	1/11, 1/13-1/14/88
RMA 21	28	1/25 - 1/27/88
RMA 22	23	1/25 - 1/27/88
RMA 23	24	1/11, 1/13-1/14/88
RMA 24	40	1/11, 1/13-1/14/88

Overall Mean: Winter 1988 Counts, All Plots: 23.6 per hectare

Mean of Plots Counted Summer 1987 (and also counted Winter 1988): 21.1

Mean of Plots Counted Winter 1988 (and also counted Summer 1987): 21.0

Source: ESE, 1988.

Prairie dog densities found in ESE studies for summer 1987 and winter 1988 were compared by a one way ANOVA with a priori contrasts designed to take into account season and contamination status. There were three major contiguous prairie dog colonies on RMA: the eastern, central and western colonies (see Figure 5.3-1). The central colony included portions of Sections 36 and 25, which are possible sources of contamination. The first level of the hierarchical division of groups was between the summer and winter census data. Within the winter study, combined controls (the eastern and western colony plots) were compared to the central colony plots, and the eastern colony plots were compared to the western colony plots. Within the summer data, plots in Sections 9, 20, and 35 ("Others") were compared to the combined control plots, and eastern plots were compared to western plots. No significant differences were found between the winter prairie dog densities of the central RMA colonies and the control groups, nor were any significant differences detected between the eastern and western colonies in any season. The statistical analysis for the comparison of plot groups is presented in Appendix B.

Surveys for adult to juvenile prairie dog ratios were completed by MKE in June 1986 and May 1987 (MKE, 1988). At locations along roads on RMA, at Buckley ANG, and the PCC, age-class estimates were made at non-random intervals. The ratios of adults to juveniles were then compared for RMA versus the offpost control areas.

Juvenile prairie dog percentages at locations on RMA, Buckley ANG, and the PCC are presented in Table 5.3-4 (MKE, 1988). Estimates of adult-young prairie dog ratios for both 1986 and 1987 show significantly higher percentages of young prairie dogs offpost than on RMA. Differences in the percentage of juveniles averaged 23 percent higher offpost ($t = 2.31$, $df = 24$, $P < 0.05$) in 1986 and 20 percent higher offpost ($t = 5.3$, $df = 38$, $P < 0.001$) in 1987 (MKE, 1988).

The absolute minimum density for a sustained population of black-tailed prairie dogs is about 10 per ha (Lewis et al., 1979). Most prairie dog densities reported in the literature range from 22 (King, 1955) to 32 per ha

Table 5.3-4. Percent of Young Prairie Dogs at Each Sampling Location

RMA			OFFSITE			
Location No.	Percent 1986	Percent 1987		Location No.	Percent 1986	Percent 1987
1	43	60	Buckley	1	62	83
2	30	67		2	63	70
3	47	47		3	63	81
4	49	59		4	77	81
5	61	70		5		75
6	44	61		6		73
7	34	58		7		84
8	49	47		8		81
9	29	55		9		72
10	61	63		10		66
11	57	41				
12	50	78	PCC	1		67
13	41	76		2		70
14	56	63		3		87
15	40	53		4		83
16	16	77		5	61	81
17	65	62		6	38	74
18	69	63		7		87
19	41	66		8		76
20	48	81		9		73
				10		
Mean	47	62		Mean	61	77

Source: MKE, 1988.

(Tileston and Lechleitner, 1966). Thus, the mean of 20 prairie dogs per ha reported at RMA in summer is on the lower end of expected mean population densities for the species.

The fact that prairie dog densities and juvenile to adult ratios are relatively low at RMA could be due to a variety of causes including normal cyclic population fluctuations on each site, the temporal ecology of prairie dog colonies, overall habitat suitability, past management practices, rainfall and other environmental factors, predation, and contamination effects. Of the above possibilities, the most likely causes are the temporal distribution of prairie dogs, the cyclic population fluctuations in colonies, and the variation of habitat suitability at RMA. New, expanding black-tail colonies typically have higher litter sizes, lower juvenile mortality, and twice the density (40 per ha vs. 18 per ha) of colonies that are 5 or more years old (Garrett et al. 1982). This observed effect has been attributed both to limited food supplies as density increases, and a lack of preferred sites for territories. In short, prairie dogs reach carrying capacity for a specific site, and normal selective pressures lead to fewer young and a lower overall density at the older colonies. Since the RMA colonies sampled in this study are relatively stable overall and are at least 5 years old, the densities and smaller percentage of young at RMA may be colonies with similar age and structure.

Areas considered optimal habitat for black-tailed prairie dogs must be at least 0.25 ha in size with low vegetation height, a diverse composition of native forbs and grasses, slopes of less than 10 percent, and about 30 to 80 percent herbaceous cover (Clippinger, 1987). RMA prairie dog colonies fit the above criteria, except they have relatively low diversity of vegetation. This is reflected in the high percent of cover of cheatgrass on many of the prairie dog plots. There was a fairly wide variation in the summer populations (from 40 per ha to 6 per ha) across the plots on RMA, indicating there is wide variation in the habitat suitability across RMA. The low diversity of vegetation in some areas may have contributed to a lower mean population density of prairie dogs on RMA.

Direct effects of arsenic and dieldrin contamination on prairie dog health are possible, given the concentrations found in our samples. Arsenic levels in prairie dog carcass samples from the TSY and Section 36 exceeded the reported background tissue concentration of 0.5 ppm (Goede 1985). A prairie dog carcass from Section 36 was found to contain 13.4 ppm of dieldrin; an estimated brain concentration from this carcass level is 2.67 ppm (Clark et al., 1978). Brain concentrations of dieldrin above 2.0 ppm are considered potentially hazardous to mammalian health (Harrison et al. 1963; Hays, 1974).

Secondary effects of contamination in prairie dogs (e.g., the adverse effect on predators consuming prairie dogs) from sites of contamination on RMA are likely more important than any direct effects on prairie dog population. Dieldrin levels in the tissues of prairie dogs from contaminated sites (Sections 36, 26, TSY) on RMA were significantly higher than those of prairie dogs offpost, and were highest in Section 36 and the TSY (see Section 4.3.1.4). Dieldrin levels in the tissues of prairie dogs from onpost controls (Sections 19, 20, and 9) were not significantly different from offpost controls. Dieldrin from sites of contamination may be accumulating in food chains including black-tailed prairie dogs and their predators: eagles, hawks, badgers, and coyotes. The role of prairie dogs in contaminant pathways is discussed in Section 5.1.2.

5.3.3.3 Eagles and Other Birds of Prey

American kestrels and bald eagles were extensively studied on RMA as part of the biota assessment investigations conducted by ESE. Other raptors studied during the course of other biota investigations or analyzed as species of chance include red-tailed hawks, ferruginous hawks, great horned owls, and golden eagles.

Eagles

Bald Eagles--ESE biologists discovered a communal roost of wintering bald eagles on RMA in December, 1986. A study was initiated to determine the population status of bald eagles occupying RMA, the extent of eagle use of RMA, and the food habits of the eagles (for a complete account of the bald eagle study see: ESE, 1988, Bald Eagle Study, Winters 1986-1987, 1987-

1988). Bald eagles arrive on RMA in November, increase in numbers through December and early January, decline in February, and depart RMA in late February through March. Maximum numbers of bald eagles roosting on RMA were 21 individuals in January 1987 and 28 individuals in January 1988. Simultaneous roost counts at RMA and Barr Lake indicated that two additional resident adult bald eagles and an occasional wintering bald eagle roosted at Barr Lake. The summer population of bald eagles in the area is limited to the resident pair at Barr Lake. This pair attempted to nest in 1987 and 1988 at Barr Lake. In 1987 the nest was abandoned and a single egg was collected for analysis.

Contaminant analysis of the bald eagle egg collected from the abandoned nest at Barr Lake in 1987 revealed mercury, dieldrin, and DDE contamination at levels of 0.099, 0.808, and 6.93 ppm respectively (Table 4.3-2). This concentration of DDE could potentially affect bald eagle reproductive success. Wiemeyer et al (1984) found that reproductive failure in bald eagles approached 100 percent when egg residues were greater than 15 ppm on a wet weight basis, and that reproductive potential was nearly normal when DDE residues in eggs were less than or equal to 3 ppm. Based on this study it would be expected that 6.93 ppm DDE in bald eagle eggs could reduce reproductive success to some extent.

The level of mercury (0.099) detected in the egg would probably have little effect on hatchability. Mallards have been found to lay fewer eggs and produce fewer young when egg residues of mercury were 0.79 and 0.86 ppm (Spann et al, 1972). Residue levels in pheasant eggs that correlated with decreased hatchability were between 0.5 and 1.5 ppm (Fimreite, 1971). Toxic levels are approximately an order of magnitude higher than the 0.099 ppm found in the Barr Lake bald eagle egg.

Little is known about the effects levels of dieldrin on bald eagle eggs, but Lockie et al. (1969) in a study of golden eagles in West Scotland, found that the proportion of eagles successfully rearing young doubled following the ban of dieldrin use in sheep dips: the average dieldrin residues in eagle eggs dropped significantly (from 0.87 ppm to 0.38 ppm) during the same period. Lockie and Ratcliff (1964) earlier correlated reproductive failure

with amounts of dieldrin exceeding 1.0 ppm in eggs of golden eagles, a level just slightly higher than the 0.808 ppm found in the bald eagle egg from Barr Lake.

The bald eagle egg was measured for shell thickness by Dr. James Enderson of Colorado College. The results of the analysis revealed that the thickness of the shell with membrane was 0.541 millimeters at the equator. This is eight percent thinner than pre DDT eggs, but within the normal expected range (Hickey and Anderson, 1968).

Contamination in bald eagle eggs is a widespread problem, making determination of the origin of the contamination in the Barr Lake egg difficult. Wiemeyer et al., (1972) in a study of bald eagle eggs from 5 states (Alaska, Maine, Minnesota, Michigan, and Florida) found residues of DDE, dieldrin, and mercury in 100 percent of the eggs analyzed.

The Barr Lake eagles are permanent residents, and do not migrate: thus, avoiding potential contamination during migration. Current evidence indicates that this pair feeds primarily in the immediate region, it is reasonable to assume that the primary source of contamination is also in the immediate region. Winter studies of the bald eagles from RMA and Barr Lake (ESE 1988) and extensive observations of the Barr Lake birds in the spring and summer of 1988 (Carter, 1988, Personal Communication) have indicated very little if any use of RMA by the Barr Lake pair. Preliminary observations suggest that this particular pair feeds occasionally on migrating waterfowl in fall, winter, spring: this prey source may contribute to the contaminant levels found.

Other possible sources of contamination may be from residues transported to Barr Lake via the O'Brian Canal from upstream sources, including RMA, and transferred to the eagles through fish eaten as prey. Offpost sediment samples collected by ESE reveal little or no organochlorine pesticide consistently in the sediment of Barr Lake (ESE, 1988). Dieldrin was detected in sediment samples taken just upstream from Barr Lake on the O'Brian Canal (OBC3S, 0.003 ppm), and from Figure 5.3-3 locations on O'Brian Canal (OBC1S, 0.003 ppm) and First Creek (FC1S, 0.006 ppm) near RMA, but not

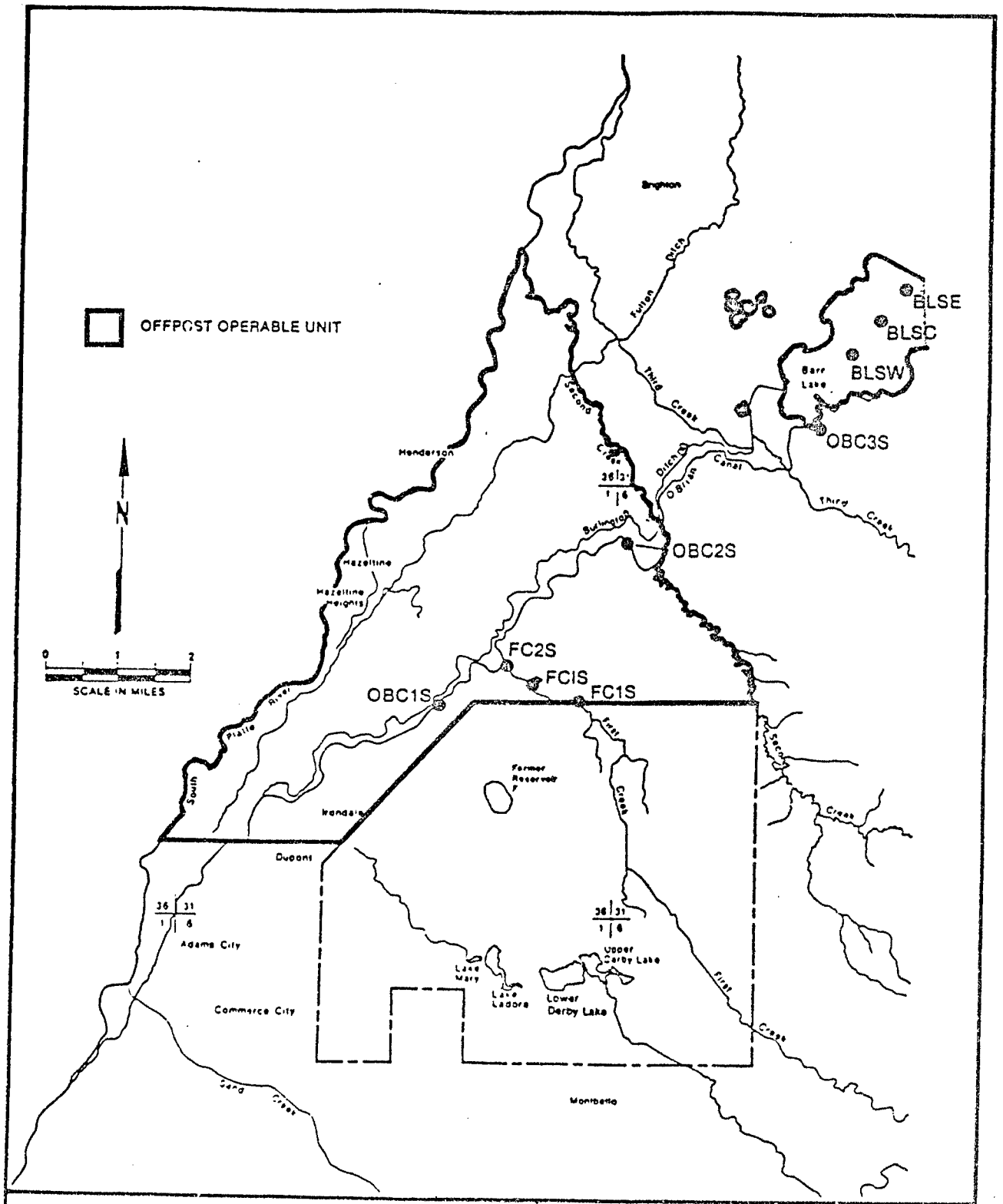


Figure 5.3-3
LOCATIONS FOR SEDIMENT SAMPLING IN OFFPOST OPERABLE UNIT

SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

from any of three sample locations between RMA and Barr Lake (FCI, FC2S, and OBC2S on Figure 5.3-3). DDE and DDT was detected in one sediment sample collected on the O'Brian Canal near RMA (OBC1S) at concentrations of 0.004 ppm and 0.008 ppm, respectively, but not from any other sediment sample locations on First Creek, O'Brian Canal, or Barr Lake. These data suggest that organochlorine pesticides may be migrating off RMA, but apparently not reaching Barr Lake, and that other sources may have contributed to the contamination of O'Brian Canal near Barr Lake.

Barr Lake sediment samples contained heavy metals, including mercury (range <0.05 to 0.252 ppm, N = 3), and this may account for the level of mercury found in the bald eagle egg. A second, deeper sediment sample was collected from the center of Barr Lake (BSLC) and revealed higher concentrations of all heavy metals including mercury (1.84 ppm). This suggests historical heavy metal contamination of Barr Lake, probably from sewage sludge that was historically dumped into the lake. Additionally, the eagles may occasionally feed on potentially contaminated fish taken from the South Platte River downstream from Denver.

Although analyses of bald eagle blood samples collected from individuals frequenting RMA during the winters of 1986-1987 and 1987-1988 indicated no significant contamination of heavy metals, organochlorines, or organophosphates (USFWS, June 1988, personal communication), the possibility exists that the eagles are accumulating contaminants from prey taken on RMA. Feeding observations of the bald eagles wintering on RMA and analysis of their castings revealed that prairie dogs, jackrabbits, and cottontail rabbits comprised the bulk of the prey consumed on RMA (ESE, 1988). Contaminant analysis of prairie dog tissue (Section 4.3) indicated that prairie dogs from contaminated areas of RMA exhibit high levels and high incidence of contamination with dieldrin. Furthermore, feeding observations of bald eagles on RMA indicate that while a large percentage of feedings occur in areas of little contamination, eagles do capture prey in areas of known contamination (Basin A, Basin C, Basin F, South Plants, and the Lower Lakes) (Figure 5.3-4, 5.3-5). The potential that bald eagles may be contaminated through exposure pathways is discussed in Section 5.1.2.

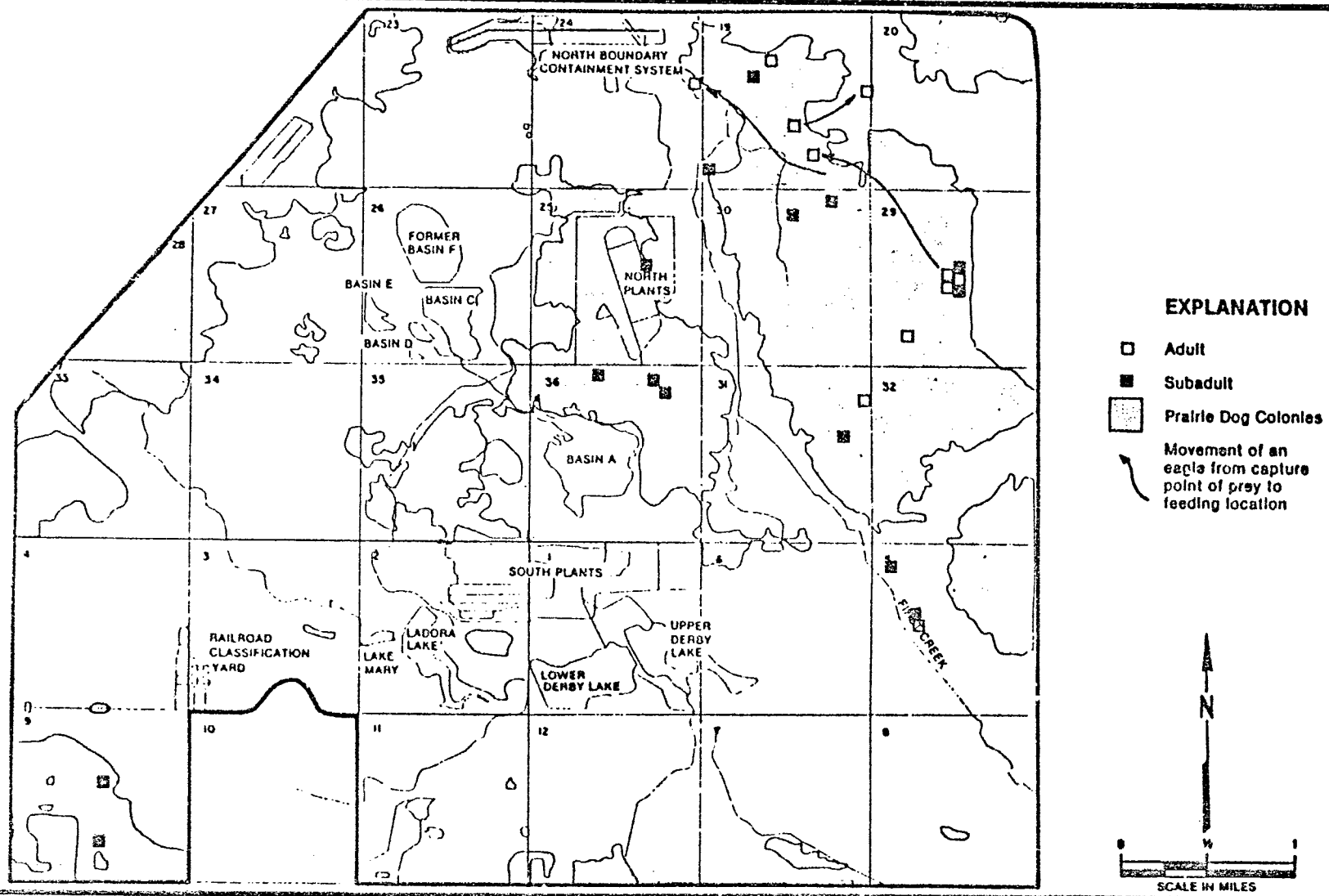
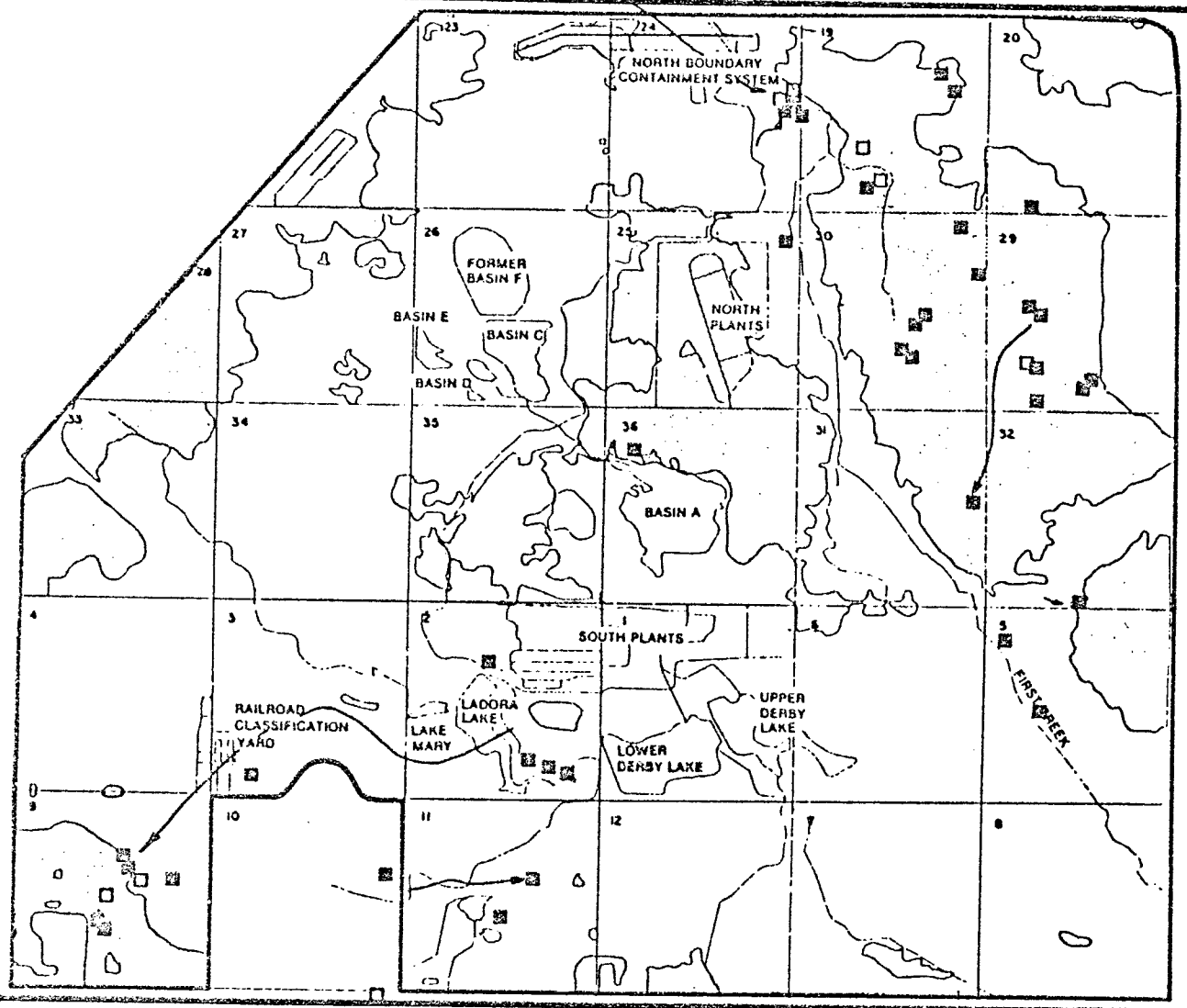


Figure 5.3-4
ROCKY MOUNTAIN ARSENAL BALD EAGLE FEEDING OBSERVATIONS, WINTER 1986-1987
SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office
For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland



EXPLANATION

- Adult
- Subadult
- Prairie Dog Colonies
- Movement of an eagle from capture point of prey to feeding location

N

0 ½ 1
SCALE IN MILES

Figure 5.3-5
ROCKY MOUNTAIN ARSENAL BALD EAGLE FEEDING OBSERVATIONS, WINTER 1987-1988
SOURCE: ESE, 1988

Prepared for:
U.S. Army Program Manager's Office For Rocky Mountain Arsenal
Aberdeen Proving Ground, Maryland

Golden_Eagles--Golden eagles are a common winter resident and an occasional summer visitor of RMA. As many as five individuals have been recorded during winter census counts (ESE,1988). The golden eagle is approximately the same size as the bald eagle and has similar winter feeding habits on RMA. Therefore it is possible to predict that contamination levels in bald eagles will be similar to levels found in golden eagles.

Liver and brain tissue from one of the two golden eagles found dead on RMA were analyzed and found to contain no detectable contaminant concentrations (Table 5.3-5). Dr. Leroy Eggleston (DVM) determined the cause of its death was probable respiratory failure. The other golden eagle analyzed contained mercury in brain tissue, and mercury and dieldrin in liver tissue, at concentrations well below toxic effects criteria for birds as described in Section 5.1. These two samples are inadequate to draw definitive conclusions, but no individuals of either eagle species have apparently died from environmental contamination on RMA.

Other_Raptors

Dieldrin appears to be the primary contaminant accumulating in tissues of raptors (Table 5.3-5). Most hawks and owls found dead on RMA and analyzed for contaminants in brain and liver tissue were found to contain residues of dieldrin. Lethal dieldrin levels in brain tissue of birds have been reported to range between 4 and 20 ppm (Robinson et al., 1967; Coon et al., 1968; Belisle et al., 1972; Mulhern et al., 1970), and most raptors found dead due to unknown causes on RMA fall within this range. Dieldrin levels in brain tissue from four RMA raptors documented to be in amaciated condition were 0.678, 9.98, 9.44, and 9.32 ppm (Table 5.3-5).

Often higher levels of dieldrin were found in brain than in liver tissue. This may indicate mobilization of organochlorine pesticides to the brain of individuals experiencing dieldrin poisoning. Heinz and Johnson (1980) concluded that brain levels of dieldrin well below the lethal level, perhaps as low as 1 ppm in highly sensitive individuals, may prove hazardous to birds by triggering irreversible starvation. Once starvation has begun, mobilization of dieldrin to the brain eventually leads to death. Raptors

Table 5.3-5. Brain and Liver Contaminant Concentrations and Necropsy Results for Rocky Mountain Arsenal Raptors.

Species	Age*	Physical Condition	Contaminant Levels of Brain/Liver			Cause of Death
			Mercury	Dieldrin	DDE	
Ferruginous Hawk	A	Emaciated	BDL/BDL	0.678/0.527	BDL/BDL	Unknown
Ferruginous Hawk	A	Good	0.152/0.293	7.73/4.79	BDL/BDL	Unknown
Ferruginous Hawk	I	Emaciated/ Convulsions	BDL/BDL	9.98/3.45	BDL/BDL	Unknown
Ferruginous Hawk	A	Good	BDL/BDL	BDL/0.263	BDL/BDL	Electrocution***
Ferruginous Hawk	I	No body fat	BDL/BDL	6.85/4.26	BDL/BDL	Unknown***
Red-tailed Hawk	I	Emaciated	BDL/BDL	9.44/5.19	BDL/0.529	Unknown***
Red-tailed Hawk	A	Unknown	0.093/0.345	9.2/6.59	BDL/0.759	Unknown
Red-tailed Hawk	I	Unknown	BDL/BDL	BDL/0.52	BDL/BDL	Electrocution
Great-horned Owl	A	Unknown	BDL/0.086	15.6/10.8	10.3/15.5	Unknown
Great-horned Owl	A	Emaciated	BDL/BDL	9.32/27.7	0.475/2.47	Unknown
Great-horned Owl	A	Good	BDL/BDL	BDL/0.143	BDL/BDL	Unknown
Great-horned Owl	A	Unknown	BDL/ 0.051	10.2/8.89	2.24/5.49	Enterotoxemia***
Golden Eagle		Unknown	0.257/0.216	BDL/0.221	BDL/BDL	Unknown***
Golden Eagle	I	Good	BDL/BDL	BDL/BDL	BDL/BDL	Respiratory Failure***

* A = Adult
I = Immature

** In wet weight basis

BDL = Below Detection Limit

*** Determined by Dr. Leroy Eggleston, DVM, or Dr. Terry Spraker, DVM.

Source: ESE, 1988.

from RMA with the highest brain levels were emaciated with empty stomachs and crops, suggesting irreversible starvation caused by dieldrin contamination. Necropsy results failed to produce any evidence to parasites, pathagens, lead poisoning or other causes of death.

In contrast, two hawks determined to have died from electrocution had very low levels of all contaminants in brain and liver tissue, often below detection limits, and were in good physical condition. Additionally a ferruginous hawk was found just south of RMA boundaries suffering from convulsions and panting profusely before death: symptoms of acute OCP poisoning. Dieldrin residues in brain and liver tissues from this hawk were 9.98, and 3.45 ppm, respectively, strongly suggesting that cause of death was dieldrin poisoning. Three of four great-horned owls and two of three red-tailed hawks found dead on RMA contained dieldrin levels in the range characteristics of lethal effects (Table 5.3-5).

Other analytes detected in raptor tissues were mercury and DDE. No arsenic, aldrin, endrin, or DDT were detected. None of the 14 raptor samples contained brain residues of mercury above 0.257 ppm or DDE above 10.3 ppm, both levels are well below lethal levels reported in the literature. Braune (1987) reported 10 ppm as a lethal concentration of mercury in the brain, and lethal brain levels of 30 to 40 ppm were reported by Borg et al (1979) for goshawk. Wiemeyer and Cromartie (1981) reported a lethal level of 250 ppm DDE in the brain of osprey.

5.3.3.4 Avian Reproductive Success

Overview

Toxic chemical effects on ducks and several other avian species inhabiting RMA have been observed since 1951 (Jensen, 1955). Avian mortality at RMA has continued up to the present time although at a lower level in recent years (McEwen and DeWeese, 1984). Before measures were taken to reduce exposure to toxicants, mortality was estimated minimally at 20,000 waterfowl over a 10-year period (Finley, 1959). Many other species of wildlife including other bird species, mammals, and amphibians died (U.S. Fish and Wildlife Service, 1961).

In response to the concern about chemical contaminants in wildlife at RMA, Patuxent Wildlife Research Center of the USFWS initiated a 2-year study in 1982 of American kestrels (Falco sparverius) as indicators of terrestrial contamination. This project was undertaken at the invitation of the Department of the Army and the USFWS provided most of the funding for the work. Results of the kestrel study indicated that some RMA kestrels were unable to successfully reproduce and fledge young, probably because of dieldrin toxicity.

Wildlife can serve as bioindicators of the presence and concentrations of toxic chemicals in the environment and can provide information for decisions on a decontamination action program for RMA. The 1984 USFWS survey determined that Dieldrin was the main contaminant found although mercury and endrin were also detected in most eggs. Canada goose and coot eggs had the lowest dieldrin concentrations ranging from trace amounts to 1.6 ppm with means of 0.17 and 0.76 ppm, respectively. Mallard eggs had much higher dieldrin concentrations with a mean of 2.8 and maximum of 5.7 ppm (all residues on a whole egg, wet weight basis). The results of both the kestrel terrestrial and waterfowl aquatic investigations signified continuing environmental contamination at the RMA.

This present study was a follow-up of the 1982-83 American kestrel investigations and was expanded to include nesting success of ring-necked pheasants and mallards. The major objectives of the study were to determine current tissue organochlorine concentrations and nesting success of American kestrels, and to measure concentrations of xenobiotic chemicals in eggs and young of mallards and pheasants and examine the relationship to their reproductive success. Methods and study area locations are described in Section 3.2.2.3 and discussed in detail in the Final Biota Assessment Technical Plan (ESE, 1988).

Results of the 1986 studies are as follows:

- Nesting Success

  Productivity of kestrels on RMA was much higher in 1986 than in earlier studies in 1982 and 1983 and were not statistically different from controls (Table 5.3-6 and Table B.2-13 in

Table 5.3-6. Nesting Productivity of American Kestrels on RMA and off (Control) During 1986 and in Earlier Studies (1982, 1983).

Reproductive Parameters	Rocky Mountain Arsenal (RMA)			Control		
	1982	1983	1986	1982	1983	1986
Nest Attempts	17[a]	26[b]	21	19	10[b]	9
Clutch Size	4.59	4.75	4.81	4.74	4.80	4.78
Percent of Nests Hatched[c]	65	54	81	58	90	89
Hatchlings/Nest	3.09	2.85	3.65	3.45	3.11	3.25
Percent of Nests Fledged	38	50	71	47	89	89
Number Fledged/Successful Nest[d]	2.83	2.67	3.13	3.33	3.12	3.12
Number Fledged/Nest Attempt[d]	1.06	1.33	2.24	1.58	2.78	2.78

[a]Includes one nest in natural activity.
[b]An extra egg was collected from one nest.
[c]Hatched nest - > = 1 egg hatched: fledged nest - > = 1 fledged.
[d]Nestlings collected for pesticide analysis are included.

Source: ESE, 1988.

Appendix B). All of the reproductive parameters measured, such as percent nests hatched and fledged and mean number of young hatched per nest, were higher in 1986. The mean number of young fledged/nest attempt on RMA in 1986 was 2.24.

This was below the mean number of 2.88 considered necessary to maintain the population (Henry, 1972). Productivity of control kestrel nests averaged 2.78 young fledged/nest attempt, the same as in 1983 (Table 5.3-6). Kestrel nest box locations offpost are shown in Figure 3.2-4.

Active kestrel nests on RMA in 1986 are shown in Figure 3.2-5: failed nests are indicated by the letter F. The pattern of nest failures differed from those in the previous study in 1982-83. In the earlier study, failed nests (those that fledged no young), were concentrated around the lower lakes northward to Basin F in the central part of the RMA. In 1986, most of the kestrel nest failures were along First Creek in the eastern part of RMA (Figure 3.2-5).

- Egg Measurements

  Collected eggs of kestrels, pheasants, and mallards were measured for weight, volume, dimensions, and shell thickness. There were few differences between eggs from RMA and control sites (Table 5.3-7). Kestrel eggs from RMA were slightly larger than controls and pheasant eggs from RMA averaged smaller and lighter than controls. RMA kestrel mean shell thickness did not differ from controls and was in the normal range for the species.

- Organochlorine and Mercury Concentrations

  Eggs of kestrels, pheasants, and mallards from RMA and control areas were analyzed for aldrin, dieldrin, endrin, and mercury concentrations.

  As in previous studies, dieldrin was the primary contaminant in all bird species (Table 5.3-8). Eggs from 22 kestrel nests contained an arithmetic mean of 0.504 ppm dieldrin and a maximum

Table 5.3-7. Mean Measurements of eggs collected on (RMA) and at Control Sites During 1986.

	(mm) Egg			(g) Whole	(cc) Egg
	Shell Thickness	Length	Breadth	egg weight	Volume
Kestrel Eggs					
RMA (22)[a]	0.220	34.8	28.5	14.36	14.25
Control (10)	0.216	34.9	28.2	13.82	14.11
Ring-Necked Pheasant Eggs					
RMA (7)	0.307	42.6	34.2	25.15	24.94
Control (8)	0.306	44.4	34.8	27.79	26.69
Mallard Eggs					
RMA (1)	0.328	53.7	39.3	45.09	42.40
Control (9)	0.340	56.4	40.3	45.72	46.72

[a] Number of eggs.

Source: ESE, 1988.

Table 5.3-8. Concentrations (Arithmentric Means, ugg^{-1}, Wet-weight Basis) of Predominant Organochlorine (OC) Chemicals and Metals Detected During 1986 in Eggs From the Colorado Rocky Mountain Arsenal (RMA) and Control Sites

Tissue and Study Area	Mercury	Aldrin	Dieldrin	Endrin
Kestrel Eggs				
RMA	(23)[a] 0.031[b] [BDL - 0.235][c]	(22) BDL [BDL]	(22) 0.504 [BDL - 2.820]	(22) BDL [BDL]
Control	(10) 0.006) [BDL - 0.057]	(10) BDL [BDL]	(10) BDL [BDL]	(10) BDL [BDL]
Ring-necked Pheasant Eggs				
RMA	(10) BDL [BDL]	(10) [BDL]	(10) 0.667 [BDL - 2.930]	(10) 0.014 [BDL - 0.143]
Control	(10) BDL [BDL]	(10) BDL [BDL]	(10) BDL [BDL]	(10) BDL [BDL]
Mallard Eggs				
RMA	(2) 0.179 [0.173 - 0.185]	(2) BDL [BDL]	(2) 3.945 [3.00 - 4.890]	(2) BDL [BDL]
Control	(10) 0.068 [BDL - 0.186]	(10) BDL [BDL]	(10) BDL [BDL]	(10) BDL [BDL]

[a]Sample size.
[b]Arithmetic mean (ugg^{-1})
[c]Range
BDL - Below Detection Limit

Source: ESE, 1988.

of 2.82 ppm dieldrin. This was in contrast to the control kestrel eggs which had no detectable dieldrin. Only one mallard nest with eggs was located on RMA. One dead newly hatched duckling from a different nest was grouped with the eggs for analysis. One egg from the nest and the duckling contained 4.89 and 3.0 ppm dieldrin, respectively, lethal concentrations. Nest locations for mallards and pheasants are shown on Figure 3.2-8.

The geometric mean dieldrin concentration in the 1986 RMA kestrel eggs was 0.005 ppm. This was less than half the geometric mean of 0.115 ppm dieldrin in the 1982 and 1983 kestrel eggs.

Carcasses of juvenile kestrels and juvenile and adult pheasants and mallards were analyzed for aldrin, dieldrin, endrin, and mercury. As with the eggs, dieldrin was the only biologically important contaminant in RMA specimens (Table 5.3-9). Adult pheasants from RMA had the highest mean whole body concentrations -- 0.497 ppm dieldrin (maximum of 2.92 ppm). Young RMA kestrels averaged 0.309 ppm dieldrin, slightly lower than the RMA kestrel egg arithmetic mean of 0.504 ppm. All control specimens of the three species, young and adults, were negative for dieldrin.

Ring-necked pheasant brood counts were conducted on RMA and at offpost control areas. All routes except one on RMA had a minimum of one brood observed during these counts; mean broods per count ranged from 0 to 1.5 (Table 5.3-10). Averages for the control routes (0.9 broods/run) were greater than for RMA (0.4 broods/run). Total hens and clutch sizes were also smaller on RMA. A total of 20 hens were observed on control routes with an average clutch size of 3.6 young. while on RMA, 14 hens were seen with an average clutch size of 1.8 young.

- Waterfowl Counts

  Numbers of dabblers and geese were much higher on the control lakes and divers and coots were higher at RMA (Table 5.3-11). The

Table 5.3-9. Concentrations (Arithmentric Means, ugg^{-1}, Wet-weight Basis) of Predominant Organochlorine (OC) Chemicals and Metals Detected During 1986 in Carcasses From the Colorado Rocky Mountain Arsenal (RMA) and Control Sites

Tissue and Study Area	Mercury	Aldrin	Dieldrin	Endrin
Juvenile Kestrels				
RMA	(10)[a] BDL[b] [BDL][c]	(10) BDL [BDL]	(10) 0.316 [BDL - 1.010]	(10) BDL [BDL]
Control	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL]
Juvenile Ring-necked Pheasant				
RMA	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL - 1.330]	(8) BDL [BDL]
Control	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL]
Adult Ring-necked Pheasant				
RMA	(4) BDL [BDL]	(4) BDL [BDL]	(4) 0.767 [BDL - 2.920]	(4) BDL [BDL]
Control	(2) BDL [BDL]	(2) BDL [BDL]	(3) BDL [BDL]	(3) BDL [BDL]
Juvenile Mallard				
RMA	(3) 0.051 [BDL]	(3) BDL [BDL]	(3) 0.201 [BDL - 0.522]	(3) BDL [BDL]
Control	(6) BDL [BDL]	(6) BDL [BDL]	(6) BDL [BDL]	(6) BDL [BDL]
Adult Mallard				
RMA	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL - 4.53]	(8) BDL [BDL]
Control	(8) BDL [BDL - 0.061]	(8) BDL [BDL]	(8) BDL [BDL]	(8) BDL [BDL]

[a] Sample size.
[b] Arithmetic mean (ugg^{-1}).
[c] Range
BDL - Below Detection Limit

Source: ESE, 1988.

Table 5.3-10. Ring-necked Pheasant Brood Counts Conducted on 6-mile Long Routes on the Colorado Rocky Mountain Arsenal (RMA) and in Northeastern Larimer County (Control) During 1986

Parameters	Rocky Mountain Arsenal (RMA)					Control				
	Route 1	2	3	4	Ave.	Route 1	2	3	4	Ave.
Amount of Time to Traverse (min)	28	30	30	28	29	26	42	25	29	30
Broods Per Run	0	1.2	0.2	0.3	0.4	0.3	1.5	0.2	1.3	0.9
Number of Adult Males Seen Per Run	0.3	0.3	5	0	0.3	0.3	0	0	0.2	0.1
Number of Adult Females Seen Per Run	0	1.5	0.2	0.7	0.4	0.3	1.7	0.2	1.2	0.8
Number of Young Seen Per Run	0	3.7	0.2	0.5	1.1	1.0	5.8	0.7	4.5	3.0

Source: ESE, 1988.

Table 5.3-11. Mean Number of Waterfowl Counted at the Colorado Rocky Mountain Arsenal (RMA) and in Northeastern Larimer County (Control) During 1986.

Waterfowl Groups	Rocky Mountain Arsenal (RMA)						Control					
	Bog A-1	L.Derby A-2	Ladora A-3	Mary A-4	Gun Club A-5	Sec.11 A-6	RD72 C-1	RD70 C-2	Smith C-3	Res.#3 C-4	N.WWA C-5	S.WWA C-6
Dabblers	12.8	4.8	2.4	3.0	15.8	26.0	145.8	118.2	11.0	4.7	28.0	4.0
Divers	0	3.5	4.0	6.0	0	8.0	2.0	0	0	0	1.2	0
Canadian Geese	0	0	9.0	6.0	5.0	0.4	26.4	3.5	0	11.7	7.4	4.2
American Coots	9.8	5.0	74.8	4.0	0	0.4	0	0	0	10.7	31.4	8.5

Source: ESE, 1988

most striking difference was the complete absence of mallard broods at RMA.

Summary of the 1986 Studies

The American kestrel nesting studies demonstrate the value and utility of this species as a bioindicator of terrestrial contamination. Results of the 1986 work provide evidence that, overall, toxic contamination of RMA terrestrial habitat is having less adverse effect than in the past, but that some local areas still may remain too contaminated for kestrel survival and reproduction. Aldrin and dieldrin were the primary contaminants implicated. DDE and DDT residues were relatively low. Reproductive effects generally do not appear in waterfowl and raptors at concentrations below 4 to 5 ppm of DDE (Wiemeyer et al., 1984).

Mallard reproduction appears to be completely inhibited. Dieldrin in conjunction with aldrin are again the primary contaminants implicated in the adverse effects on mallard reproductive success on RMA. Endrin levels were generally low, and its toxic effects, if any, were difficult to evaluate.

Pheasant populations may be adversely affected on RMA, but results are less conclusive than for kestrels or mallards. Habitat differences and total population densities between control and onpost areas may account for some of the differences. Contaminant concentrations in tissues may have direct adverse effects on the pheasants in contaminated areas (see Section 5.2.1). Data collected as part of this investigation (Section 4.3.1.3) were supplemented with contaminant analysis data on 20 pheasants collected by MKE from locations onpost.

Combined results indicate that pheasants collected from locations in and near major sites of contamination on RMA contain tissue levels of dieldrin above CRL, but that tissue from pheasants collected on RMA but away from major contamination sites did not contain dieldrin levels above the CRL (Figure 4.3-8). Potential adverse effects to humans via consumption of pheasants are addressed in the onpost and offpost endangerment assessments for RMA.

One of the chief goals of the 1986 study was to compare kestrel and mallard productivity with earlier investigations of those species on RMA. Comparison of the contaminant concentrations between 1986 and earlier studies correlate with the field observations of upward trends in kestrel productivity, but continuing adverse toxic effects on mallard reproduction and survival.

5.3.3.5 Other Species and Contaminant Effects

Deer

Both mule deer and white-tailed deer populations were counted in roadside surveys by MKE in the winter of 1986-1987. The maximum number of mule deer found by MKE (1988) in their roadside counts was 207, while the maximum count of white-tailed deer was 56. Based on these findings, MKE estimated mule deer density at eight per square mile. Open plains habitat rarely exceeds five deer per square mile (Mackie et al. 1982).

The relatively high density of deer and occurrence of sympatric deer species are probably due to the absence of hunting and to the abundance and interspersion of suitable habitat on RMA. For these reasons, it was not possible to detect any effects of contamination on RMA deer populations. Since no significant concentration differences were detected between RMA deer and control areas, and because only 1 of 14 deer from RMA contained detectable levels of contamination, we conclude that deer populations on RMA appear to be unaffected by contamination.

Lagomorphs

Nighttime roadside counts of jackrabbits and cottontails were completed by MKE in the spring of 1986 in RMA-wide surveys, and offpost at Buckley and the PCC. No significant differences were detected between cottontail populations on and offpost. A significantly higher population index for jackrabbits was found at the offpost sites (MKE, 1988). However, the roadside counts on RMA did not separate counts taken near sites of contamination from those taken in uncontaminated areas.

Statistical comparison was made between the onpost control sites and the contaminated site in Section 36. The cottontails from Section 36 were significantly more contaminated than those from the onpost control areas.

It is likely that the bioaccumulation of contaminants in predators of rabbits is more important than the direct effect of contamination on this prey species. Low but significant levels of dieldrin were found in Section 36 cottontails (See Section 4.3.1). The contaminant biaccumulation pathways are discussed in Section 5.1, Contamination Evaluation.

Carnivores

Badgers and coyotes have been found to contain concentrations of dieldrin in their tissues (see Section 4.3.1). Dieldrin residues have been found throughout the prey of these mammals (prairie dogs and cottontails) as well. It is assumed that these predators obtain the bulk of their contaminants through food chain sources, rather than directly from the contamination sources themselves. Possible pathways and bioaccumulation in carnivores on RMA are discussed in Section 5.2.

Dieldrin levels in liver tissue were 1.64 ppm for badger and 7.6 ppm for coyote (Section 4.3). Walker et al. (1969) observed a relationship between dietary concentrations and concentrations of dieldrin in liver and brain tissue for rats and dogs. Using a brain to liver ratio of 0.47 derived from the Walker data, calculated brain levels are 0.77 ppm for badger and 3.6 ppm for coyote. The brain to liver ratio was calculated for dogs at each dose level (x=0.21). For rats, a mean brain to liver ration was calculated for each group sacrificed after a given time on dosage. For both dogs and rats, data for sex and dose were combined to get the mean. To obtain the overall mean of 0.47, rat and dog means were averaged. The rat data were weighted more heavily because the number of rats used in the test was much higher than the number of dogs. Harrison et al. (1963) determined that brain levels of 2.4 to 9.4 ppm were strongly correlated with death of dogs. Although toxicity levels are known to vary among species, coyote and dog are both in the same genus and probably have similar toxic effect levels. Dieldrin levels found in the coyote may have been responsible for the death of the coyote, inasmuch as no outward signs of death were noted.

Birds

The effects of RMA contamination on mallards, ring-necked pheasants, and American kestrel reproductive success are discussed in Section 5.3.3.4. The information in this section includes concentrations of contaminants contained in adult mallards, pheasants, doves, and waterfowl (collected by ESE and USFWS).

Maximum mercury concentrations found in muscle samples of blue-winged teal (0.559 ppm) and coots (0.339 ppm) were below those reported in mallards (0.8 ppm in muscle) with altered nesting behavior, and decreased number of offspring from mercury contamination (Heinz, 1979). The means for teal muscle samples on RMA (0.391 ppm) and coot samples on RMA (0.179 ppm) exceeded the average concentrations found in 5,200 waterfowl muscle samples nationwide (0.08 ppm in mallards and 0.033 ppm in black ducks) (Heath and Hill, 1974). But blue-winged teal, coot, and redhead carcasses had far below the lethal levels reported in muscle (4.3 ppm) and liver (20 ppm) in hawks (Fimreite and Karsted, 1971).

Adult mallard carcasses from RMA contained up to 4.53 ppm dieldrin: an estimated brain concentration from this carcass is 1.57 ppm. Concentrations above 1 ppm in bird brains can have adverse effects on health and behavior (Barbehenn and Reichel, 1981), and concentrations above 3.2 ppm are considered hazardous (Wiemeyer and Cromartie, 1981).

An adult pheasant carcass from RMA contained 2.92 ppm of dieldrin, from which an estimate of brain concentration was 1.01 ppm (from Barbehenn and Reichell, 1981). With the parameters outlined above for bird health effects, this level could have adverse effects on health and behavior, and is possibly hazardous to pheasant life. One juvenile pheasant collected from a country club in Larimer County contained contaminant levels in the carcass of 18.6 ppm dieldrin and 1.34 ppm DDE. All other offpost juvenile pheasant samples (n = 13) had contaminant levels below detection limits. Contamination of this individual apparently came from a non-RMA source.

Dietary levels of over 4 ppm dieldrin correlated with health effects in birds (Sharma et al., 1976). RMA grasshoppers in Sections 26 and 36 have

been found to contain up to 7.2 ppm dieldrin, and up to 4.8 ppm aldrin. Since aldrin concentrations can be additive to dieldrin concentrations since aldrin is converted to dieldrin in biotic systems), the risk of health effects in birds (such as burrowing owls, pheasants, and kestrels) which consume grasshoppers and other insects could be substantial. Dieldrin concentrations found in pheasant and kestrel carcasses on RMA may be due in part to consumption of contaminated insects. Pathways implications are discussed in Section 5.2.1.

Insectivorous birds feeding in contaminated areas of RMA may consume hazardous concentrations of endrin. Screech owls with 0.75 ppm endrin in their diet suffered adverse health and reproductive success (Fleming et al., 1982). While only one hit of endrin was found in a pheasant egg, and no concentrations of endrin were found in kestrel samples, birds consuming grasshoppers (maximum levels of 1.65 ppm) and other insects in and near Section 26 could be at risk from endrin.

Two mourning dove carcasses found near Building 111 on RMA contained aldrin concentrations up to 1.83 ppm, dieldrin concentrations up to 56.3 ppm, and one bird contained endrin at a concentration of 3.44 ppm. The levels in avian brain that are indicative of dieldrin poisoning range from 4 to 9 ppm (Ohlendorf et al, 1981; Wiemeyer and Comartie, 1981). If a maximum rate of mobilization of 20 percent from carcass to brain is assumed, a potentially hazardous carcass concentration of 5 times the lethal brain level is calculated (DeWeese et al., 1986). Thus, the lethal carcass level corresponding to 9 ppm in brain is 46 ppm; this level was exceeded by the mourning dove carcass. Another mourning dove found on the southern border of Section 36 was dissected and the liver analyzed, which contained 7.37 ppm dieldrin, and 3.74 ppm endrin. Considering the unusually high levels of contaminants in the carcasses, the doves found dead near Building 111 probably did not accumulate contaminant levels from food chain sources, but more likely obtained a high dose in water from a nearby contaminated water source (e.g., Basin F).

Analytical results indicate that RMA contamination, particularly dieldrin, is still a problem for avian species. Bioaccumulation through food web and

other pathways are presented in Section 5.2 and indicate that relatively low levels of organochlorine pesticides (e.g., dieldrin) present in soil and sediments can lead levels and effects documented by the tissue analyses described herein. The spatial extent of these contaminants will be delineated in the forthcoming Study Area Reports (SARs) for the appropriate abiotic media as data from Phase II investigations becomes available.

5.3.4 AQUATIC ECOSYSTEMS

Contamination of sediments in RMA lakes has been documented since the 1950s (Rosenlund et al., 1986). Attempts have been made to eliminate these contaminants, particularly mercury and various chlorinated hydrocarbon pesticides, through partial sediment removal. However, these contaminants persist as they are bound to sediments and are retained in fish and macrophyte tissues. Current soil contamination levels (ESE, 1989) additionally indicate that soils provide a source of chemicals that may be carried into surface waters at the RMA facility.

Contaminants reaching surface water bodies either remain suspended/dissolved in the water column, or settle and become associated with the bottom sediments and sediment interstitial water. Settling rates depend on such factors as natural buoyancy, Vander Waals forces, turbidity, and velocity of the surface water. Changes in surface water velocity or sources of turbulence (e.g., spring and fall turnover in lakes) may scour and resuspend or dissolve chemicals from the sediments back into the water column. Those chemicals suspended/dissolved in the water column may be taken up by aquatic biota or they may undergo such processes as biological degradation, and chemical transformation (i.e., interaction with other chemicals and ultraviolet (solar) degradation). Contaminants that accumulate in the sediments may become sorbed to particulate organic materials (detritus) or remain in sediment interstitial water.

Freshwater species of benthic invertebrates generally do not ingest sediment, although oligochaetes and some species of chironomids occasionally ingest sediment. Rather, the primary source of food, and consequently an important route of exposure, for many benthic invertebrates is the particulate organic material associated with sediments (Adams, 1987).

Particulate organic matter may either be bound to sediments, settled in the sediment layer, or suspended in the water column. Contaminants sorbed to particulate organic material may; therefore, be inadvertently taken up by lower trophic levels of organisms such as plankton and macroinvertebrates through food ingestion or absorption.

Lower trophic level organisms can also inadvertently take up contaminants in sediment interstitial (pore) water while feeding. This water can contain high concentrations of dissolved or suspended contaminants. Other incidental routes of exposure include absorption of suspended or dissolved contaminants in the water column, and ingestion of sediment bound contaminants (Adams, 1988).

Various forms of bacteria, plankton (i.e, phytoplankton, micro-, and macrozooplankton) as well as macrophytes take in dissolved or suspended substances in the water column as well as in inorganic detritus. Higher aquatic plants (aquatic macrophytes) may also take in substances via their root systems.

Lower trophic levels are then preyed upon by higher trophic levels (i.e., small fish), which are then preyed upon by the top level aquatic consumers (i.e., large predatory fish). Contaminants are: therefore, transferred about the ecosystem food web via complex feeding interrelationships (Figure 5.3-1). Consequently, contaminants from many levels can be transferred to and bioaccumulated in the top level consumer via multiple exposure pathways including:

- Bacteria that have absorbed or ingested contaminants:
- Plankton that have absorbed or ingested contaminants:
- Aquatic plants that have absorbed contaminants:
- Fish that have absorbed contaminants through exposed tissues (i.e., gills):
- Benthic macroinvertebrates that have absorbed or ingested contaminants, or ingested contaminated organisms:
- Herbivorous fish that have ingested contaminated plants or have absorbed contaminants through exposed tissues (i.e., gills):
- Aquatic insectivorous fish that have ingested contaminated aquatic

insects or have absorbed contaminants through exposed tissues (i.e., gills): and

- Carnivorous fish that have ingested contaminated fish or have absorbed contaminants through exposed tissues (i.e., gills).

The following sections address the evidence of bioaccumulation in RMA aquatic ecosystems, and the potential impacts of contamination on the communities of various organisms in RMA surface waters.

5.3.4.1 Bioaccumulation in Aquatic Ecosystems

Bioaccumulation of contaminants such as chlorinated hydrocarbons (e.g., DDT and dieldrin) are well documented by Edwards (1970). Mercury in various forms has been shown to not only bioaccumulate, but can also be biotransformed by lower tropic levels to more toxic forms (see Section 5.2). An example of this process is the methylation of mercuric chloride to highly toxic methyl mercury by bacteria, zooplankton, and phytoplankton (Environmental Studies Board, 1978). The processes involved in bioaccumulation are quite complex due to population fluctuations, food web interrelationships, metabolic capabilities of various species, and other ecological considerations. The amount of a contaminant that ultimately accumulates at the higher trophic levels is: therefore, a function of the level of contamination, the availability of sediments and abiotic organic materials, the affinity that the contaminant has for the sediments, and the structure of the aquatic community at each trophic level.

Analysis of tissues from biota collected in RMA aquatic ecosystems by MKE (1988) indicates organochlorine pesticides and mercury are still present in the aquatic community and generally supports Rosenlund's earlier conclusions regarding the bioaccumulation of these contaminants in RMA lakes (Section 4.3).

Bioaccumulation of Organochlorine Pesticides in Aquatic Ecosystems

Organochlorine pesticides (OCPs) entering the environment are highly fat soluble and generally not easily dissolved in water. The data from Rosenlund et al. (1986), summarized in Section 4.3, provide the best evidence of the bioaccumulation of organochlorine pesticides in the aquatic

ecosystems on RMA, as discussed below. The aquatic data collected by MKE (1988) generally support these findings. Rosenlund's data show the greatest concentrations of dieldrin, aldrin, and endrin in the viscera of fish from the Lower Lakes on RMA. Of these three contaminants, dieldrin in tissues from Lower Derby Lake most clearly demonstrates the bioaccumulation of a persistent organochlorine pesticide in an aquatic ecosystem. As shown by the Rosenlund et al. (1986) data from Section 4.3, mean concentrations of dieldrin were greatest in bass viscera (5.397 ppm), and less in other tissues, listed here in order of decending concentration: pike viscera (1.942 ppm), bluegill fillets (0.264 ppm), plankton (0.216 ppm), chironomids (0.2 ppm), bass fillets (0.156 ppm), bullhead fillets (0.133 ppm), young-of-year bullheads (0.123 ppm), leafy pondweed (0.059 ppm), pike fillets (0.056 ppm), and American pondweed (0.044 ppm). Concentrations in biota samples collecte by MKE in their study are presented in Section 4.3.3. It can be seen that dieldrin concentrations in fish fillets were much lower than in viscera, because fillets have a lower lipid content than viscera.

The correlation of fat content with tissue type and with dieldrin concentrations is shown in data that compare mean percent lipid content and dieldrin concentrations of fish viscera and fillets (Roselund et al., 1986). Bass viscera possessed the highest mean percent lipid content of 16.06, with mean percent values for the viscera of other species being: channel catfish (13.8), pike (12.02), bluegill (2.73), and bullhead (2.23). Mean percent lipid content in fillets was generally less than 1.0 with the exception of fillets from ictalurid fish (e.g., bullhead, catfish), which typically have a high lipid content compared to other fishes: mean percent lipid content in bullhead fillets was 1.05 ppm, and in catfish fillets was 5.75 ppm (Rosenlund et al., 1986).

Bioaccumulation of Mercury in Aquatic Ecosystems

Mercury can exist in many forms including inorganic free mercury, Hg^0: ionic mercury in salts and complexes, Hg^{2+}: or organic mercury compounds such as phenylmercuric salts, and alkylmercury compounds such as methyl mercury (Casarett and Doull, 1980). Each form has its own physical, chemical and toxicological properties.

Methyl mercury, is absorbed faster in fish than inorganic mercury, and is cleared from the body at a much slower rate. This is true whether it was released directly into the environment or formed the product of a complex biotransformation of mercury by microorganisms, which occurs under specific enviromental conditions at a rate of less than 1.5 percent per month (Jensen and Jernelov, 1969). The net result is a high methyl mercury concentration in muscle tissue (Casarett and Doull, 1980). High levels of mercury in fish muscle are; therefore, an indicator of direct methyl mercury contamination of the fish, rather than absorption or methylation of environmental sources of inorganic mercury. Fish in RMA lakes tend to show high levels of mercury in their fillets (see Section 4.3.3): concentrations in some fillets from Lower Derby Lake and Lake Ladora exceeding the FDA guideline of 1.0 ppm.

Mercury food web accumulation in part depends upon the diets of the top level carnivores in each lake. Rosenlund et al. (1986) commented that mercury in Lower Derby Lake "appears to quickly accumulate in young fish at the bottom of the food chain and be concentrated by predators in their fillets". Mean mercury concentrations were similar within species at each trophic level, with the greatest concentration in pike fillets (mean = 1.9 ppm). Mean mercury concentrations in other species (Rosenlund et al., 1986) listed in decending order were: adult bullheads (1.735 ppm), bass fillets (1.510 ppm), fillets from 24 to 32 cm carp (0.62 ppm), young-of-year bullheads (0.58 ppm), bluegill fillets (0.505 ppm), dragonflies (0.50 ppm), amphibians (0.385 ppm), crayfish (0.308 ppm), American pondweed (0.247 ppm), leafy pondweed (0.238 ppm), damselflies (0.23 ppm), and plankton (0.198 ppm).

Mercury contamination in Lake Ladora (Rosenlund et al., 1986) was also concentrated in fish fillets, with the greatest mean concentrations in pike fillets (2.940 ppm). Concentrations in other species in decending order were: bass fillets (2.445 ppm), bluegill (0.873 ppm), bullhead (0.420), plankton (0.39 ppm), and aquatic plants (0.227 ppm). Bass and pike in Lake Ladora appear to feed mainly on bluegill, which rely heavily on abundant plankton resources for their diet (Rosenlund et al., 1986).

Similarly, Lake Mary also demonstrated mercury contamination in fish fillets

(Rosenlund et al., 1986), with bass (0.495 ppm) and bluegills (0.505 ppm) generally reflecting equal levels of contamination because of their similar diets of invertebrates. Contamination levels in other species analyzed were similar to mercury levels in similar species in other RMA lakes.

Preferential deposition of mercury to fillet muscle rather than to viscera is well documented in RMA fish analyzed for mercury content. Mean mercury concentrations are higher in fillets than viscera of all species where both tissues were analyzed (Rosenlund et al., 1986), with mean values greatest in pike fillets (2.25 ppm), followed by bass fillets (1.48 ppm), bullhead fillets (0.885 ppm), bluegll fillets (0.66ppm), carp fillets (0.62 ppm), and catfish fillets (0.275 ppm). Mean mercury concentrations in viscera were substantially lower, with the greatest concentrations in bass (0.626 ppm), followed by values for pike (0.47 ppm), bluegill (0.32 ppm), catfish (0.12 ppm), and bullhead (0.11 ppm).

Potential for Transfer of Aquatic Contaminants to Terrestrial Ecosystem

Contaminants that have entered aquatic ecosystems and have bioaccumulated in aquatic organisms can eventually reach terrestrial organisms through aquatic-terrestrial food web interrelationships. Terrestrial organisms such as birds and mammals feed on aquatic organisms such as fish, insects, and plants that may have acquired contaminants through absorption or ingestion of contaminants or contaminated organisms. A more extensive review of this pathway is presented in Section 5.2 of this document.

5.3.4.2 Effects of Contamination in RMA Aquatic Communities

Data collected on the aquatic communities in three of the Lower Lakes (Lower Derby, Ladora, and Mary) on RMA were compared to similar data collected at the McKay Lake control area (MKE, 1988). A brief summary of these comparisons between contaminated and control areas is presented below by taxonomic grouping. In interpreting these data it should be kept in mind that a multitude of interacting causes could be responsible for the differences detected among the contaminated lakes and between the contaminated and control areas.

Phytoplankton

The density and diversity of the phytoplankton community of McKay Lake were generally within the range of values found in the RMA lakes, but the community at McKay Lake was somewhat different in terms of composition. Mean density in McKay was 2,623 per ml, values that resembled those in Lake Mary (1,918 per ml). Both these lakes had a greater diversity than Ladora (1,269 per ml) and a lower diversity than Lower Derby (13,854 per ml).

Phytoplankton diversity in McKay averaged 25 taxa, compared with 24 for Mary, 28 for Lower Derby, and 41 for Ladora.

Community composition of McKay Lake was similar to the Arsenal lakes in the overall dominance of green algae. However, there were differences in the relative abundance patterns of taxa between McKay Lake and the Arsenal lakes: the mean relative abundance of green algae in McKay was greater, euglenophytes and diatoms had a much lower mean relative abundance in McKay, pyrrhophytes were more important, and chrysophytes and cyanophytes were less important in McKay than in the RMA lakes.

Microzooplankton

Microzooplankton density was markedly lower in McKay Lake during the four sampling periods than in the three RMA lakes. This may have resulted from differences in the abundance or availability of food, factors that typically limit populations of rotifers (Pennak, 1978), the primary microzooplankton present in all of these lakes. The numbers of rotifer taxa varied among the lakes, with McKay Lake (8 taxa) most similar to Lower Derby (8 taxa), but with lower diversity than in Lake Ladora (11 taxa) and Lake Mary (17 taxa). These differences may have been related to differences in habitat among the lakes.

Macrozooplankton

The mean density of macrozooplankton in McKay Lake (368 per liter) was below the range for the three Lower Lakes (408 per liter in Lake Mary to 602 per liter in Lower Derby Lake). This range in density could be related to variations in food availability.

The dominant taxa in all four lakes were cladocerans and copepods, which varied among lakes and seasons. The total number of taxa collected at McKay Lake (17) was similar to the numbers collected in Lower Derby Lake (16) and in Lakes Ladora and Mary (19 each). Two cladoceran species found in McKay Lake were absent from the RMA lakes; otherwise, the taxa were the same.

Benthic Macroinvertebrates

The mean density of benthic macroinvertebrates in McKay Lake (2,004 per m^2) was within the range of mean densities in the RMA lakes (1,590 per m^2 in Lower Derby to 2,669 per m^2 in Lake Mary). Overall abundance patterns were similar among the lakes.

The mean and total number of macroinvertebrate taxa collected by Ponar dredge at McKay Lake were within the range of values for the RMA lakes, and the dominant groups of benthic organisms, tubificid worms and chironomid flies, were the same. Seasonal trends for the abundance of these dominant groups varied between the groups and among the lakes with no apparent pattern.

Aquatic Plants

Six taxa of submergent aquatic plants were identified from the RMA lakes; one of these taxa was missing at McKay Lake. The areal coverage of submergent aquatic plants at McKay Lake (5 percent) was closest to that in Lower Derby Lake (<1 percent), with both these lakes being more turbid that Lakes Ladora and Mary, which had areal coverage of 57 percent and 65 percent, respectively.

Broadleaf and narrowleaf cattails were the predominant emergent aquatic plants, covering 3.8 hectares (ha) in McKay Lake, 3.4 ha in Lower Derby Lake, 7.4 ha in Lake Ladora, and 1.0 ha in Lake Mary.

Fish

Twelve species of fish were identified in McKay Lake, compared to eight species in Lower Derby Lake, seven in Lake Ladora, and five in Lake Mary. McKay Lake has been more actively and recently managed as a fishery. Bluegill and bass were two species in common between RMA lakes and McKay

Lake and that were abundant in both. MKE (1988) calculated condition factors for bass and bluegill (Section 4.3.3) which indicated that fish condition in RMA lakes was generally better than in the offpost control lake. These results are not readily related to the characterization of contamination effects on RMA because additional factors (e.g., diet, population density, etc.) could effect fish condition and because the data used to calculate condition factors (weight and length) are not typically evaluated as known effects of the contaminants present in RMA lakes.

The data on chemical contaminants in fish species provided in Section 4.3.3 (MKE, 1988) provide means of evaluating contaminant effects on aquatic communities. A statistical analysis of the 1988 data (MKE, 1988) showed that each of four analytes (dieldrin, aldrin, DDE, and mercury) exhibited highly significant differences between Lower Derby Lake and McKay Lake for bass, despite small sample sizes, but that none of these analytes exhibited significant differences between Lower Derby Lake and McKay Lake for bluegill.

Results of MKE analyses indicate that organochlorine pesticides and mercury are still present in the aquatic ecosystems on RMA. Pathways analyses (Section 5.2) further suggest that the concentrations present in some biota may pose a hazard to animals within the aquatic food web. Concentrations are somewhat lower than those reported by Rosenlund et al. (1986), suggesting that the contaminants are less available to biota than in the recent past.

6.0 GLOSSARY

@ - assimilation efficiency.

Acetylcholinesterase inhibitor - a chemical that causes accumulation of endogenous acetylcholine in nerve tissue and effector organs with consequent signs and symptoms that mimic the muscarinic, nicotinic, and central nervous system actions of acetylcholine. Acetylcholine is the chemical transmitter of nerve impulses at endings of postganglionic parasympathetic nerve fibers, somatic motor nerves to skeletal muscle, preganglionic fibers of both parasympathetic and sympathetic nerves, and certain synapses in the central nervous system (Casarett and Doull, 1986).

ACGIH - American Conference of Governmental Industrial Hygienists.

AChE - acetylcholinesterase.

Acute exposure - a single exposure or multiple exposure occurring within 24 hours or less (Casarett and Doull, 1980).

Ad libitum - in biological studies, feed or food provided without restraint or limit.

AEP - Aurora Environmental Park.

Alopecia - loss of hair, wool or feathers.

Anorexia - loss of appetite, not eating feed (Hudson et al., 1984).

Anurans - frogs, toads, or tree toads, all of which lack a tail in the adult stage.

Apnea - cessation of breathing (Hudson et al., 1984).

ARAR - applicable or relevant and appropriate requirement.

Army - Department of the Army

Assimilation efficiency - ug of contaminant absorbed per ug ingested.

Asthenia - weakness, debility (Hudson et al., 1984).

ASTM - American Society for Testing and Materials

Asynergy - lack of coordination between muscle groups: movements are in serial order instead of being made together (Hudson et al., 1984).

Ataraxia - imperturbability, calmness (Hudson et al., 1984).

Ataxia - muscular incoordination, especially when voluntary muscular movements are attempted (Hudson et al., 1984).

BAF - bioaccumulation factor (C_b/C_{medium}).

BCF - bioconcentration factor (C_b/C_w).

BMF - biomagnification factor (C_b/C_d).

Benthic organisms or benthos - organisms living on or in the bottom of oceans, lakes or streams.

Bioaccumulation - concentration effect of a chemical expressed as a ratio of the concentration of a chemical in the organism to that in the medium (usually water). Bioaccumulation refers to both uptake of dissolved chemicals from water and uptake from ingested food and sediment residues (Casarett and Doull, 1986).

Bioassay - the determination of the strength of a drug or other substance by comparing its effects on an organism with those of a standard substance.

Bioconcentration - a process by which there is a net accumulation of a chemical directly from water into aquatic organisms resulting from simultaneous uptake (e.g., by gill or epithelial tissue) and elimination (Rand and Petrocelli, 1985).

Biomagnification - result of the processes of bioconcentration and bioaccumulation by which tissue concentrations of bioaccumulated chemicals increase as the chemical passes up through two or more trophic levels. The term implies an efficient transfer of chemical from food to consumer, so that residue concentrations increase systematically from one trophic level to the next (Rand and Petrocelli, 1985).

Biome - all plants, animals, and other organisms that make up a distinct natural community in any climatic region.

Bradychardia - slow heart beat (Hudson et al., 1984).

Bradypnea - slow breathing (Hudson et al., 1984).

C_b - concentration in biota.

C_d - concentration in diet.

C_{sed} - concentration in sediment.

C_w - concentration in water.

CAR - Contamination Assessment Report.

Carcinogenic - A substance or agent producing or inciting cancer.

CDH - Colorado Department of Health.

CDOW - Colorado Division of Wildlife.

CERCLA - Comprehensive Environmental Response, Compensation, and Liability Act.

CF&I - Colorado Fuel and Iron Corporation

ChE - see AChE.

Chronic exposure - usually refers to length of experimental exposure extended over the average lifetime of the species. Thus, for a rat, exposure is normally two years. Dosages used are selected that at least 50 percent of the animals will survive for the entire duration of the study (Casarett and Doull, 1986).

Climax - the final or stable community in a successional series. It is self-perpetuating and in equilibrium with the physical and biotic environment (Krebs, 1978).

CG - Phosgene

CK - Cyanogen chloride

COE - U.S. Army Corps of Engineers

CPMS - chlorophenylmethyl sulfide

CPMSO - chlorophenylmethyl sulfoxide

$CPMSO_2$ - chlorophenylmethyl sulfone

DBCP - dibromochloropropane.

DCPD - dicyclopentadiene

DDE - 1,1-dichloro-2,2-bis(4-chlorophenyl)-ethylene.

DDT - Dichlorodiphenyltrichloroethane.

DIMP - diisopropyl methyphosphonate.

DMMP - dimethyl methylphosphonate.

DNA - deoxyribonucleic acid.

DNMA - nitrosodimethylamine

Depuration - a process that results in elimination of a chemical from an organism by desorption, diffusion, excretion, egestion, biotransformation, or another route (Rand and Petrocelli, 1985).

Detritus - decaying organic matter.

Detritivore - a detritus consuming organism.

Disclimax - the community replacing the climax after a disturbance of the climax community (Krebs, 1978).

Dyspnea - shortness of breath, labored breathing (Hudson et al., 1984).

EA - Endangerment Assessment

EC_{50} - concentration affecting 50 percent of a population.

Ecological magnification - soil to organism uptake.

EMF - ecological magnification factor.

EMLD - empirical minimum lethal dose. The oral dose resulting in one or two deaths within 30 days.

EPA - Environmental Protection Agency.

Ephemeral - transitory or of short duration, such as a plant or animal that grows, reproduces and dies all in one day or a few days.

Epistaxis - nose bleed (Hudson et al., 1984).

Erythema - redness of the skin due to dilation of blood vessels (Hudson et al., 1984).

ESE - Environmental Science and Engineering, Inc.

^{o}F - degrees Farenheit

Fasciculation - skin or superficial tremors (Hudson et al., 1984).

FDA - Food and Drug Administration

f_i - food term.

f_{oc} - fraction of organic carbon.

Floristics - the study of the geographical distribution of plants.

Flow-through system - an exposure system for aquatic toxicity tests in which the test material solutions and control water flow into and out of test chambers on a once-through basis either intermittently or continuously (Rand and Petrocelli, 1985).

Food chain - a group of organisms so interrelated that each member of the group feeds upon organisms in the trophic level below it and is in turn eaten by organisms in the higher trophic levels.

Food web - a group of interrelated food chains in a particular community.

ft - foot

Gavage - introduction of material into the stomach by a tube.

GB - Sarin (nerve agent)

GR_{50} - 50 percent growth inhibition.

H - Levenstein mustard

Heteroscedastic - showing unequal variability; not showing the same standard deviation.

Homogeneity - being made up of similar parts or elements: of uniform nature throughout.

Hydrophyte - a plant growing in water or in soil too waterlogged for most plants to survive.

Hyperemia - congestion, an unusual amount of blood in a part of the body (Hudson et al., 1984).

In situ - in the natural or original position: in an organism's natural environment or habitat.

Instar - growth stage or period of growth occurring between any two successive molts, as in insects and crustaceans (Johnson and Finley, 1980).

k_2 - depuration or loss rate.

K_d - sediment-water partition coefficient.

K_{oc} - soil-water partition coefficient normalized for organic carbon.

K_{ow} - octanol-water partition coefficient.

Lacrimation - production of tears (Hudson et al., 1984).

Lagomorph - any of the order of gnawing mammals having two pairs of incisors in the upper jaw one behind the other. Includes rabbits, hares, and pikas.

Littoral - of, belonging to, or found on or near the shore of a lake: or a region along the shore or coast.

LC_{10} - concentration lethal to 10 percent of the exposed population.

LC_{50} - lethal concentration in 50 percent of a population.

LD_{50} - lethal dose in 50 percent of a population.

LOAEL - lowest observed adverse effects level.

MATC - maximum acceptable tissue concentration.

Mesic - moist.

Micromho (umho) - unit of electrical conductivity.

Microohms (uohm) - unit of resistance to electrical current.

Miosis - constriction of the pupil (Hudson et al., 1984).

mg/l - milligrams per liter

MKE - Morrison-Knudsen Engineers, Inc.

mm - millimeter

MPA - methylphosphonic acid.

MPTC - maximum permissible tissue concentration

msl - mean sea level

Mutagenicity - the ability of a chemical to cause changes in the nucleus of cells in ways that can be transmitted during cell division.

Mydriasis - excessive dilation of the pupil of the eye.

NCP - National Oil and Hazardous Substances Pollution Contingency Plan

NOEL - no observed effects level.

NTU - nethelometric turbidity unit. Relative unit measuring scattered light.

Nutation - nodding of the head.

Nonparametric - statistical techniques that are distribution free (Siegal, 1956).

OCP - organochlorine pesticide.

Opercular rhythm - opening and closing of the gill covering.

Opisthotonos - arching of the back and arching of the neck over the back (Hudson et al., 1984).

Orthogonal - comparisons which are independent of each other.

OSHA - Occupational Safety and Health Administration.