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Ecotoxicity Literature Review of Selected Hanford Site Contaminants

C. J. Driver

March 1994

Prepared for the U.S. Department of Energy
under Contract DE-AC06-76RLO 1830

Pacific Northwest Laboratory
Operated for the U.S. Department of Energy
by Battelle Memorial Institute



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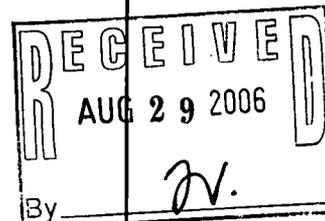
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C. J. Driver

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Pacific Northwest Laboratory
Richland, Washington 99352

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Executive Summary

Available information on the toxicity, food chain transport, and bioconcentration of several Hanford Site contaminants were reviewed. The contaminants included cesium-137, cobalt-60, europium, nitrate, plutonium, strontium-90, technetium, tritium, uranium, and chromium (III and VI). Toxicity and mobility in both aquatic and terrestrial systems were considered. For aquatic systems, considerable information was available on the chemical and/or radiological toxicity of most of the contaminants in invertebrate animals and fish. Little information was available on aquatic macrophyte response to the contaminants. Terrestrial animals such as waterfowl and amphibians that have high exposure potential in aquatic systems were also largely unrepresented in the toxicity literature. The preponderance of toxicity data for terrestrial biota was for laboratory mammals. Bioconcentration factors and transfer coefficients were obtained for primary producers and consumers in representative aquatic and terrestrial systems; however, little data were available for upper trophic level transfer, particularly for terrestrial predators. Food chain transport and toxicity information for the contaminants were generally lacking for desert or sage brush-steppe organisms, particularly plants and reptiles.

Food chain mobility and biotic response data for the contaminants were highly site-specific, reflecting the significant effect that numerous biological and physicochemical factors have on contaminant bioavailability and toxicity. Of the reported factors, soil and sediment conditions and presence of nutrient analogs particularly affected exposure and food chain transport.

In general, the heavy metals--chromium, cobalt, and technetium--are highly toxic to both aquatic and terrestrial biota. They tend to be retained in soils and sediments (except technetium, which rapidly moves into plants) and to accumulate in high concentrations at the lowest trophic levels. However, trophic transfer is low and no biomagnification of the metals has been reported for either aquatic or terrestrial food chains. High food chain mobility is exhibited by tritium, and the light metals, cesium and strontium. Although biomagnification does not usually occur with the light metals, in some instances cesium concentrations can increase with trophic level. Also, cesium mobility can be greatly diminished by high clay and high humic content of soil. The environmental presence of potassium and calcium affect the uptake of cesium and strontium, respectively. The toxicities of the light metals appear to be low or moderate chemically in aquatic and terrestrial organisms. The actinides, uranium and plutonium, exhibit relatively low biological mobility and are largely restricted to root systems of plants in terrestrial ecosystems. The rare-earth element, europium, exhibits low toxicity, poor assimilation and low food chain transport. Although nitrate toxicity is low for most animals, nitrate can accumulate in certain plant species to toxic levels for mammals.

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1.0 Introduction

This document summarizes the literature available on the environmental effects of several Hanford Site contaminants. The contaminants were selected using information from ongoing Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) investigations for operable units 200-BP-1, 300-FF-5, 100-BC-1, 100-DR-1, 100-HR-1 and 100-BC-5. Westinghouse Hanford Company requested Pacific Northwest Laboratory^(a) to prepare this document in response to requirements set forth in the Tri-Party Agreement milestone document "Columbia River Impact Evaluation Plan" (U.S. DOE/RL 1993). Specifically, the plan identifies the need for several tasks to provide information for human health and ecological risk assessments, including the development of an ecotoxicology literature review (Activity 4-1).

The information compiled in this report can be used both to 1) provide background information to operable unit managers in the U.S. Department of Energy, U.S. Environmental Protection Agency, and State of Washington Department of Ecology, and 2) provide ecotoxicological profiles for the ecological risk assessments.

Available information was reviewed on the sensitivity of plants and animals to cesium-137, cobalt-60, europium, nitrate, plutonium, strontium-90, technetium, tritium, uranium, and chromium (III and VI). As the CERCLA investigations and ecological risk assessments proceed, the document may be revised to include additional contaminants. Particular emphasis was given to transfer coefficients of the contaminants from water and soil to biota.

Information on each of the contaminants was obtained from open literature reports dating from the early 1900s to the present. Ten commercial online bibliographic data bases were used to obtain citations for each of the contaminants. The bibliographic source files searched were BIOSIS, AGRICOLA, National Technical Information Service, Science Citation, Environmental Bibliography, Enviroline, Pollution Abstracts, Toxline, Energy Science and Technology, and Nuclear Science Abstracts. Pre-1948 citations were obtained from hand searches of bibliographic indices and from references cited in later publications. Except where indicated, data were obtained from primary sources.

Data for species relevant to the Hanford Site are emphasized in this report. However, uptake and toxicity data for local species are augmented, where necessary, with data from other North American species. The response and sensitivity to heavy metals and radionuclides for tropical fish are within ranges reported for Northern Hemisphere species (Skidmore and Firth 1983; Bywater et al. 1991); therefore, information obtained from tropical fish studies are also included.

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In addition, available data on the toxicokinetics of the contaminants are reported because the relative rates of uptake and loss of a chemical and its distribution within the organism determine its exposure. Also identified are those factors that influence the uptake and toxicity of the particular contaminants in organisms from both aquatic and terrestrial systems. Information on contaminant-specific lesions or changes that provide biomarkers of exposure or indices of injury are also provided.

Bioconcentration factors and transfer coefficients were reported for representative species of each trophic (feeding) level within freshwater and terrestrial ecosystems, when available. A bioconcentration factor is defined as the concentration of a given chemical in an organism divided by the concentration of the same chemical in the environment of that organism. For terrestrial organisms, the bioconcentration factor for organisms in each trophic level is based on the concentration found in the soil. In aquatic systems, the bioconcentration factor can be based on the water concentration or the level of the contaminant found in the sediment. Bioaccumulation factor, concentration ratio, discrimination factor, uptake coefficient, and concentration factor are terms synonymous with bioconcentration factor in the scientific literature. The term "transfer coefficient" may either refer to the bioconcentration factor or describe the ratio between the concentration of a chemical in an organism to the concentration of the same chemical in the biota that constitute that organism's food source. When used in the latter manner in this report, the food source is identified from which the chemical is transferred.

Comparing transfer coefficients and bioconcentration factors for plants reported by different researchers is difficult because of the inconsistency of reporting values based on dry weight or wet weight of the plant tissue. Although concentration values based on wet weights of plants have a more direct application to food chain uptake predictions, they are much less accurate than those based on dry weights (DOE 1974). In 1974, the Department of Energy standardized reporting of transfer coefficients and bioconcentration factors to a dry weight basis to alleviate the problem of data comparability. However, data prior to this time and data from non-DOE sponsored programs are often reported as wet weight values. The dry or wet weight basis is indicated for the bioconcentration factors and transfer coefficients listed in this document.

The radiation toxicity and general trophic level accumulation and transfer of radionuclides are summarized separately from the specific radiobiological effects and chemical toxicity reported for each specific radionuclide.

An explanation of the various dose and exposure units used in radiation toxicity research is provided in Appendix A.

2.0 Ionizing Radiation

2.1 Ionizing Radiation Toxicity

Ionizing radiation can cause damage to all biological systems; however, the sensitivity of organisms varies greatly. In general, larger herbivorous mammals are most vulnerable to ionizing radiation. The next sensitive group of organisms include the smaller mammals, birds, herbivorous insects, filter-feeding aquatic invertebrates, higher plants, and lower plants. Simpler life forms including unicellular plants and animals, bacteria, and viruses are more resistant to the lethal effects of radiation. Also, organisms in rapidly growing stages of their life cycle are more radiosensitive than mature organisms (Krumholz et al. 1957; Whicker and Schultz 1982; Bond et al. 1965; Gleiser 1953; Rust et al. 1954). The relative sensitivities of various organisms to ionizing radiation are shown in Table 2.1.

Table 2.1. Typical LD 50/30^(a) Values for Total Body Exposure of Animals to Gamma-Radiation

<u>Organism</u>	<u>LD50/30 (rads)</u>
Sheep	200-300
Swine	200-300
Dog	350
Guinea Pig	400
Man	250-400
Mouse	550
Monkey	600
Rat	750
Rabbit	800
Chicken	600
Song Sparrow	800
Goldfish	2,300
Frog	700
Tortoise	1,500
Fruit Fly	100,000
Bacteria	500,000
Virus	1,800,000

(a) LD50/30 is the observed dosage of radionuclide that is required to kill 50% of a group of animals in 30 days.

2.1.1 Ionizing Radiation Toxicity in Aquatic Biota

The ionizing radiation toxicity in aquatic invertebrates and fish is reviewed below.

Invertebrates

An LD50 of 500 R has been reported for crustaceans (King 1964). The reproductive ability of a hermaphroditic snail was greatly reduced by radiation exposures between 4000 and 9000 R (Perlowagora-Szumlewicz 1964). Permanent sterility was caused by an accumulated dose of 10,500 R in the cladoceran, *Daphnia magna* (King 1964), and depressed reproduction was observed in *Daphnia magna* populations exposed to 10^{-5} Ci/L for 80 days (Klechkovskii et al. 1973).

Fish

King (1964) reported a general LD50 value for fish of 90,000 R. The 50% survival dose for male germ cells of medaka, a small teleost fish, at hatching was 390 to 500 rad for beta radiation and 500 to 520 rad for gamma emissions (Etoh and Hyodo-Taguchi 1983a, Hyodo-Taguchi and Etoh 1986). For beta radiation, the 50% survival dose for female germ cells in fish at hatching was 140 rad. The 50% survival dose for female germ cells exposed to gamma-radiation was 305 rad (Etoh and Hyodo-Taguchi 1983b). Population growth of white crappie (*Pomoxis sparoides*), largemouth black bass (*Micropterus salmoides*) and the redbreast (*Moxostoma erythrurum*), declined by 25% when exposed to 57 R/yr external radiation (and unknown internal radiation) in a contaminated lake and creek.

2.1.2 Ionizing Radiation Toxicity in Terrestrial Biota

The following sections provide data on the ionizing radiation toxicity in terrestrial biota.

Plants

Plants are relatively resistant to ionizing radiation. The effects of chronic irradiation (6 months) of a late successional oak-pine forest were studied at Brookhaven National Laboratory (BNL) in New York. Changes in ecosystem structure, diversity, primary production, total respiration, and nutrient inventory occurred. The most resistant species were the ones commonly found in disturbed places, i.e., generalists capable of surviving a wide range of conditions. Mosses and lichens survived exposures greater than 1000 R/d. No higher plants survived greater than 200 R/d. Sedge (*Carex pennsylvanica*) survived 150 to 200 R/d. Shrubs (*Vaccinium* and *Quercus ilicifolia*) survived 40 to 150 R/d. Oak trees survived up to 40 R/d, whereas pine trees were killed by 16 R/d. No change was noted in the number of species in an oak-pine forest up to 2 R/d, but changes in growth rates were detected at exposures as low as 1 R/d (Woodwell 1970). Severe defects were observed in *Tradescatia* at an exposure rate of 40 R/d. However, an exposure of 6000 R/d was required to produce the same

effect in a hybrid gladiolus (Odum 1956). The sensitivity of various plant species appears to be related to the cross-sectional area of the nucleus in relation to cell size: the larger the nucleus and chromosome volume, the more sensitive the plant (Underbrink and Sparrow 1968, 1974). The radiosensitivity of plants to gamma-radiation is listed in Table 2.2.

Invertebrates

Although viability and reproduction are reduced in insects exposed to ionizing radiation, the level of exposure required to induce these effects is quite high. Sterilization of the screw worm fly (*Callitroga*) occurred at 500 R, whereas the fruit fly (*Drosophila*) required an exposure of 16,000 R to induce sterilization, and the powder post beetle (*Lyctus*) required 32,000 R. The LD50 for adult fruit flies was about 10^5 R. The LD50 for fly eggs was about 190 R (Packard 1936). Reduction of egg viability was observed in the European corn borer after exposure to 2500 R (Walker and Brindley 1963).

Mammals

Lethal effects are observed in most mammals at acute radiation doses in excess of 200 rad (Myers 1989). Mortality results from failure of the hematopoietic system. Radiation injuries most significant to animal populations are those affecting life span and reproduction. In laboratory rodents, survival time was shortened by 10% when the radiation dose was more than half the LD50/30 dose for that species (Bacq and Alexander 1961). Reproductive cells are the most radiosensitive cells of the mammalian system, and fertility in laboratory animals has been reduced by ionizing radiation. Acute exposures of a few hundred roentgens rendered male mice temporarily sterile. Smaller doses (100 R) resulted in permanent sterility in females (Oakenberg and Clark 1964). The difference in reproductive sensitivity to radiation is due to the presence of spermatogonia in males and the lack of

Table 2.2. Radiosensitivity of Various Plants to Chronic Gamma-Radiation

Plant	Length of Exposure (weeks)	Exposure to Produce Effect (R/d)		
		None	Mild	Severe
<i>Lilim</i> (hybrid) h.v. Tangel (Lily)	8	10	20	40
<i>Tadescantia paludosa</i> (Spiderwort)	12	15	20	40
<i>Wicia faba</i> (Broad Bean)	15	30	60	90
<i>Nicotian rustic</i> (Tobacco)	15	50	100	400
<i>Sedum spurium</i> (Stonecrop)	14	255	528	760
<i>Sedum sieboldi</i> (Stonecrop)	13	1000	2500	4100
<i>Galdolus</i> (Gladiola)	12	800	1500	5000
<i>Luzula Acuminata</i> (Wood Rush)	12	1720	2600	6000

a comparable regenerative stem cell in the ovary (i.e., oocytes cannot be replaced once they are destroyed). Differences in reproductive sensitivity to ionizing radiation among species were largely found in the responses of the females and were probably a result of the different rates of ova development among species, particularly the stage at which the oocyte remains until follicle maturation. In contrast, the radiation response of the male was typical for all species (Oakenberg and Clark 1964, Roderick 1964). Male organisms as diverse as the grasshopper, fruit fly, silkworm, and guinea pig have shown very similar responses to ionizing radiation because of the similarity of spermatogenesis (French 1965). In addition to causing sterility, acute exposure can decrease the number of young produced by the irradiated parent. An acute dose of 30 R in young female mice significantly reduced the number of offspring produced. Exposures of about 400 R in males decreased production of young (Rugh 1964). The reduction in young is attributable to dominant lethal mutations. Somatic effects on the female parent also contribute to loss of young. The cost in number of offspring per litter was 0.009 mice/R if the male parent was irradiated and 0.027 mice/R if the female parent received the dose (Touchberry and Verley 1964). Chronic exposure to radiation for more than 2 R/d resulted in sterility in male rats (Brown et al. 1964). In mice, sterility resulted from exposure to 3 R/day (Stadler and Gowen 1964). Although wild rodents were more resistant to radiation exposure in relation to lethality (Gambino and Linberg 1964), it is not known if the reproductive system of the wild rodents is correspondingly resistant as well.

Chronic exposure to 0.83 R/day of gamma radiation has been reported to decrease survival of the desert rodent (*Perognathus formosus*). The observed population decline was calculated to cause a reduction in the multiplication rate per generation of 40% (French et al. 1974). However, the test design did not include replicates of the irradiated plot, thus making causes of population differences between control and irradiated plots unclear. Indeed, for life expectancy, the only population parameter subjected to statistical testing, the estimates for the three plots (one irradiated plot and two control plots) were all statistically different ($p < 0.001$) indicating that the differences the authors ascribe to radiation could be caused by any number of other factors.

At relatively high doses delivered at high dose rates, ionization radiation is carcinogenic and mutagenic in laboratory animals. However, the dose-response curve at low doses is ambiguous. As a conservative position, ionizing radiation is considered carcinogenic at dose rates that extend down to doses that could be received from environmental exposures. Estimates of cancer risk are based on the absorbed dose of radiation in an organ or tissue, and the cancer risk at a particular dose is the same regardless of the source of the radiation. However, the chemical properties of the radionuclide influence the distribution, biological half-life, and retention of the radionuclide within a target organ. Genetic damage, such as gene mutation or chromosomal aberrations, has been demonstrated in experimental animals (CBEIR 1980, 1988, 1990; UNSCEAR 1982, 1986, 1988). High-energy beta-radiation (0.61 MeV strontium, 0.31 MeV cobalt-60, and 0.55 MeV cesium-137) causes deep injury to the dermis layer of the skin with subsequent development of chronic radiation dermatitis. Chronic radiation dermatitis is characterized by persistent exfoliation and, in some instances, progressive pre-cancerous lesions in the skin (Jones and Hunt 1983).

Biomarkers of Radiation Exposure. Lethal doses of ionizing radiation induce pervasive hemorrhages throughout the body, particularly in the gastrointestinal tract. Generally, microscopic lesions are not unique. However, a characteristic "severance," or separation, of the epiphyseal cartilage from the spongy bone on the diaphyseal side has been described (Bloom 1948).

Birds

As a group, birds appear to be at greater risk of beta-gamma radiation exposure than other wild animals. About 33% of birds collected from a contaminated area had radiation counts above the background level, whereas only 7% of the mammals collected, and 5% of the reptiles collected had higher-than-background counts. The higher rate of contamination was attributed to the grit-use behavior of birds (Bellamy et al. 1949).

The LD50/30s for wild bird species exposed to ionizing radiation range from 485 to 2500 rad (average 790) and are listed in Table 2.3. No gross effects were observed in birds at a waste disposal site with body burdens greater than 5 μ Ci of radioactivity (Willard 1960). Twenty-three species of birds from areas containing 10 to 100 times normal background radiation (1.0 to 4.0 mR/h) were monitored for health effects (Maslov et al. 1967). Increased mitotic abnormalities and inhibition of cell division of the cornea in the birds were observed. However, no other changes were induced, even in radiosensitive organs (bone marrow, spleen, and gonads). The stress of the ionizing radiation added to the existing stresses of predation, weather, and diminished food quantity or quality were noted but unquantifiable (Willard 1960). Reproductive populations of tree swallows (*Iridoprocne bicolor*), rufous-sided towhees (*Pipilo erythrophthalmus*), brown thrasher (*Toxostoma rufum*), Baltimore oriole (*Icterus galbula*), and eastern blue bird (*Sialia sialis*) nesting in the vicinity of the radiation source in the oak-pine forest at BNL were adversely affected by the radiation. The dose found to be lethal to 100% of exposed eggs of wild passerines was between 500 and 1000 R at a dose rate up to 50 R/d. The adult mortality rate approached 100% at about 2000 R at a dose rate of up to 150 R/d (Wagner and Marples 1966). External exposure to gamma-radiation of up to 600 R did not result in the mortality of embryos or nestlings of passerine species (Zach and Mayoh 1984, 1986a, 1986b). Chronic irradiation with 960 rad over 20 days reduced hatchability in domestic chicken (*Gallus domesticus*) and black-headed gull (*Larus ridibundus*) eggs (Phillips and Coggle 1988). A decrease in hatchability of greater than 40% was observed in tree swallow embryos exposed to 1.0 Gy/d (Zach and Mayoh 1986b). Mraz (1971) found that exposure of chicken eggs to 0.4 Gy/d resulted in a significant decrease in hatchability.

High sublethal doses of ionizing radiation did not affect pair-formation in green-winged teal or territorial behavior of the shoveler (Tester et al. 1968). In pheasants (*Phasianus colchincus*), single exposures to the ovaries of 500 to 2025 R and cumulative exposures from 500 to 5316 R did not affect egg production, plumage coloration, or ovarian tissue structure (Greb 1955; Greb and Morgan 1961). Irradiation of female eastern bluebirds and their eggs at 23.5 R/min for accumulated doses of 200 to 600 R did not alter clutch size, hatching success, nestling period, or fledgling success (Norris

Table 2.3. LD50/30s of Avian Species Exposed to Ionizing Radiation

Species	Age	LD50/30 (R)	Reference
Blue-Winged Teal (<i>Anas discors</i>)	Adult	715	Tester et al. 1968a
Green-Winged Teal (<i>Anas crecca</i>)	Adult	485	Tester et al. 1968a
Shoveler (<i>Anas clypeata</i>)	Adult	894	Tester et al. 1968a
Mallard (<i>Anas platyrhynchos</i>)	4 months	704	Abraham 1972
	12 months	630	Cumow et al. 1970
Bluebird (<i>Sialia sialis</i>)	Adult	2500	Willard 1963
	Nestling to Fledgling	500-600 ^(a)	Willard 1963
Greenfinch (<i>Chloris chloris</i>)	Adult	600	Kushniruk 1964
European Goldfinch (<i>Carduelis carduelis</i>)	Adult	600	Kushniruk 1964
Linnet (<i>Acantis cannabina</i>)	Adult	400	Kushniruk 1964
House Sparrow (<i>Passer domesticus</i>)	Adult	625	Kushniruk 1964
Serin (<i>Serinus canarius</i>)	Adult	500	Kushniruk 1964
Weaver Finch (<i>Quelea quelea</i>)	Adult	1060	Lofts and Rotblat 1962
Starling (<i>Sturnis vulgaris</i>)	Adult	800	Garg et al. 1964

(a) LD50 for duration of nesting exposure to time of fledgling, i.e., 15 to 20 days.

1958). Testicular damage and arrested germ cell maturation were induced by radiation doses equal to, or in excess of, 420 R in weaver finches (Lofts and Rotblat 1962). Feather development was inhibited in nestling bluebirds irradiated at 2 days of age with 43 R/min for an accumulated dose of 300 to 500 R. Nestling growth was reduced by 50% when exposures reached 1500 to 2000 R. When birds receiving 800 to 900 R fledged, they were weak and unable to sustain flight, rendering them more vulnerable to predation (Willard 1960). No observable adverse effects to avian reproduction occurred from a chronic dose of 330 R/30-day nesting period (Wagner and Marples 1966). In the bird communities near a 10-megawatt, air-shielded nuclear reactor close to Marietta, Georgia, the number of singing (territorial) birds declined significantly compared to controls (Schnell 1964). Doses associated with the bird declines ranged from 310 rads for bobwhite (*Colinus virginianus*) to 27,700 rads in the white-eyed vireo (*Vireo griseus*). Radiation doses of 160 rad or more to tree swallows' (*Iridoprocne bicolor*) eggs prolonged incubation, depressed subsequent growth (body mass and foot and primary-feather lengths), and delayed primary-feather emergence (Zach and Mayoh 1986a). Data from chick embryo studies indicate that this radiation-induced stunting is likely to be permanent (Muller and Morenz 1966; Tyler et al. 1967). Chronic exposure to 100 rad resulted in far more severe growth depression of nestling passerines than single doses of 320 rad (Zach and Mayoh 1982; Zach and Mayoh 1986a; Guthrie and Dugle 1983). Gross congenital abnormalities induced by radiation are relatively uncommon in birds. Mraz (1971) found no increase in abnormalities in chicken embryos or resulting chicks from exposure to 15.5 Gy. Phillips and Coggle (1988) reported increased foot and limb deformities in gull chicks only after embryonic exposure to 9.6 Gy.

2.2 Bioconcentration Factors and Transfer Coefficients

There appears to be a discrimination against the movement of radionuclides of high atomic number from lower to higher trophic levels.

2.2.1 Radionuclide Transfer in Aquatic Systems

Aquatic invertebrates lose up to half of their body burden of adsorbed and absorbed radionuclides at each molt. Therefore, the total accumulation over their entire life-cycle is reduced. (Wilhm 1970).

2.2.2 Radionuclide Transfer in Terrestrial Systems

The radionuclide transfer in terrestrial systems is described below.

Birds

Food source, behavior, and habitat influence the accumulation of radionuclides in bird tissues. For example, a survey of birds for gross beta activity from a high-mountain watershed (world-wide fallout monitoring) showed that birds that spent a large amount of time feeding and probing for food on the ground had a higher (217 pCi/g) radioactivity level for skin and feathers than raptors (30 pCi/g) (Osburn 1968). Piscivorous (fish-eating) birds had low body burdens of radioactivity compared to those species dependent on insect larvae and vegetation (Krumholz and Rust 1954; Silker 1958). Migratory waterfowl feeding in a radioactive waste impoundment had body burdens of greater than 5 μ Ci of radioactivity, whereas tissues of herons and kingfishers accumulated very little radioactivity (Krumholz 1964). After the impoundment was drained, carnivorous and omnivorous birds were contaminated throughout the food web via insects and herbivorous species were contaminated by ingestion of soil while searching for seed (Willard 1960). Silker (1958) found that shorebirds and dabbling ducks contained 13 times the amount of strontium-90 found in fish-eating birds associated with Hanford Site operations. Concentration factors for beta-emitters were determined for ducks, shorebirds, grebes, and gulls inhabiting the Columbia River adjacent to the Hanford Site from 1956 to 1959 (Hanson and Watson 1960). These values are listed in Table 2.4. Note that the shorebirds and grebes (larvae consumers), and canvasbacks (herbivores) have much higher concentration factors than the fish-eating or omnivorous birds.

Table 2.4. Concentration Factors of Beta Emitters in Birds Using the Columbia River in the Vicinity of the Hanford Site, 1956-1959 (Hanson and Watson 1960)

<u>Bird Group</u>	<u>Consumer Type</u>	<u>Concentration Factor [bird weight (g)/water (mL)]</u>
Shorebirds	Larvae feeding	45
Diving Ducks (Canvasbacks)	Herbivore	30
Grebes	Larvae feeding	20
Gulls	Omnivore	7
Mergansers	Fish-eating	6
River Ducks	Fish-eating	1

3.0 Uranium

3.1 Uranium Toxicity

Uranium emits alpha particles and, as such, does not constitute an external radiation hazard. However, the health effects of internal alpha emission in biota can be significant. In addition, uranium has a chemical toxicity unrelated to radioactivity.

3.1.1 Uranium Toxicity in Aquatic Biota

Generally, uranium is more toxic to aquatic biota in soft water than in hard water.

Aquatic Plants

Uranium inhibits growth of aquatic microflora at about 1.0 mg/L in freshwater systems and appears to be bactericidal at 100 mg/L (Gus'Kova et al. 1966). These effects are attributed to the chemical rather than radiation toxicity of the uranyl ion. Although severe reductions in diatom survival have been observed for waters containing 1.0 mg U/L (Gross and Koczy 1946), other studies have reported abundant diatom populations in waters on uranium mill tailings containing up to 17 mg U/L (Ruggles et al. 1979). Cell division was inhibited at 22 mg/L in the alga, *Scenedesmus* (Bringman and Kuhn 1959).

The threshold-effect level (inhibition of food intake) of uranium as uranyl acetate has been reported to be 28 mg/L for the protozoan, *Microregma* (Bringman and Kuhn 1959).

Invertebrates

Freshwater hydrae (*Hydra viridissima*) are highly sensitive to uranium contamination. In studies conducted at the retention ponds for the Ranger Uranium Mines in tropical Northern Australia, rapid lysis of the hydrae was observed within 48 hr of exposure to greater than 1 mg/L uranium (Hyne et al. 1991). Lower concentrations (150 µg/L) significantly inhibited growth of asexually-reproducing hydrae after 3 to 4 days of culture (Hyne et al. 1991). Hyne et al. (1992) demonstrated that populations of hydrae exposed to greater than 200 µg/L uranium for 3 days suffered a reduction in population growth of about 50%. Concomitant with the population decrease was a reduced ability to capture live prey. Transmission electron microscopy and energy dispersive X-ray microanalysis (EDAX) indicated that the feeding dysfunction and reduced population growth were correlated with a pathological accumulation of uranium in nematocysts. Because of its sensitive, uranium-specific endpoint, this assay could be used to distinguish between environmental effects that are caused by uranium and those that result from other pollutants or environmental conditions. The acute toxicity of

uranium to cladocerans varies with water quality, particularly total hardness and alkalinity (Poston et al. 1974). The concentrations killing 50% of exposed organisms (LC50s) of uranium in cladoceran species are listed in Table 3.1.

Table 3.1. Acute Toxicity of Uranium in Aquatic Invertebrates (Cladoceran Species)

<u>Species</u>	<u>Water Hardness (mg CaCO₃/L)</u>	<u>LC50 (mg/L)</u>	<u>Duration of Test</u>	<u>Reference</u>
Northern Hemisphere				
<i>Daphnia magna</i>	70	6.5	48 h	Poston et al. 1984
<i>Daphnia magna</i>	133	37.5	48 h	Poston et al. 1984
<i>Daphnia magna</i>	197	52.5	48 h	Poston et al. 1984
Tropical				
<i>Diaphanosoma excisum</i>	5	1.0	24 h	Bywater et al. 1991
<i>Latonopsis fasciculata</i>	5	0.4	24 h	Bywater et al. 1991
<i>Daeaya macrops</i>	5	1.1	24 h	Bywater et al. 1991
<i>Moinodaphnia macleayi</i>	5	1.3	24 h	Bywater et al. 1991

Fish

As with the cladocerans, the toxicity of uranium to fish varies markedly with water conditions. The 96-h LC50 of uranium (as UO₂²⁺) in fathead minnows is 3 mg/L in waters with pH 7.4 and hardness of 210 mg/L. In waters of pH 8.2 and a hardness of 400 mg/L, the 96-h LC50 is 135 mg/L (McKee and Wolf 1963). However, when water conditions are similar, differences in both intra-specific and interspecific toxicities are negligible (Bywater 1991). LC50 data for various tropical and Northern Hemisphere freshwater fish are compiled in Table 3.2. To provide a predictive tool of uranium toxicity for freshwater fish in various waterbodies, least-squares linear regression was used to test the relationship between the 48-h LC50 values compiled from the literature and the total hardness (expressed as mg-equivalent CaCO₃/L) of the test water used to establish the LC50. As shown in Figure 3.1, the relationship between the 96-h LC50 and water hardness is linear.

Little information is available on sublethal- or threshold-effect levels of uranium in fish species. Laboratory tests have shown that the hatchability of carp eggs (*Cyprinus carpio*) is not affected by 60 mg/L uranium in areas of high water hardness (Till and Blaylock 1976). Parkhurst et al. (1984) reported a no observable effect concentration (NOEC) of greater than 9.0 mg/L for uranium in brook

Table 3.2. Acute Toxicity of Uranium to Freshwater Fish. LC50 values are for 96-H exposures.

<u>Species</u>	<u>Water Hardness^(a) (mg CaCO₃/L)</u>	<u>LC50 (mg/L)</u>	<u>Reference</u>
Northern Hemisphere			
Fathead Minnow (<i>Pimephales promelas</i>)	20	3.1	Tazwell and Henderson 1960
	20	3.7	Tazwell and Henderson 1960
	20	2.8	Tazwell and Henderson 1960
	400	135.0	Tazwell and Henderson 1960
	70	16.7	Posten et al. 1984
Rainbow Trout (<i>Oncorhynchus mykiss</i>)	31	6.2	Davies 1980
	208	23.0	Parkhurst et al. 1984
Brook Trout (<i>Salvelinus fontinalis</i>)	31	8.0	Davies 1980
	35	5.5	Parkhurst et al. 1984
Tropical			
<i>Hypseleotris compressa</i>	10	6.6	Skidmore and Firth 1983
<i>Melanotaenia nigras</i>	5	2.1	Bywater et al. 1991
	5	2.4	Bywater et al. 1991
	10	4.5	Skidmore and Firth 1983
<i>Melanotaenia splendida</i>	5	2.8	Bywater et al. 1991
	5	3.8	Bywater et al. 1991
<i>Melanotaenia s. inornata</i>	10	6.0	Skidmore and Firth 1983
		1.4	Holdway 1992
<i>Cratérocephalus marianae</i>	5	1.8	Bywater et al. 1991
	10	4.3	Giles 1964
<i>Amniataba percoides</i>	10	25.0	Giles 1964
<i>Leiopotherapon unicolor</i>	10	4.1	Giles 1964
<i>Pseudomugil tenellus</i>	5	0.8	Bywater et al. 1991
<i>Ambassis macleayi</i>	5	0.8	Bywater et al. 1991
<i>Mogurnda mogurnda</i>	5	2.1	Bywater et al. 1991
	5	2.2	Bywater et al. 1991
		1.6	Holdway 1992
		3.3	Holdway 1992
		3.3	Holdway 1992

(a) When not reported directly, hardness was calculated from the concentration of calcium and magnesium in the water by the equation: Hardness = 2.497 [calcium] + 4.118 [magnesium] as mg equivalents of CaCO₃ (APHA 1980).

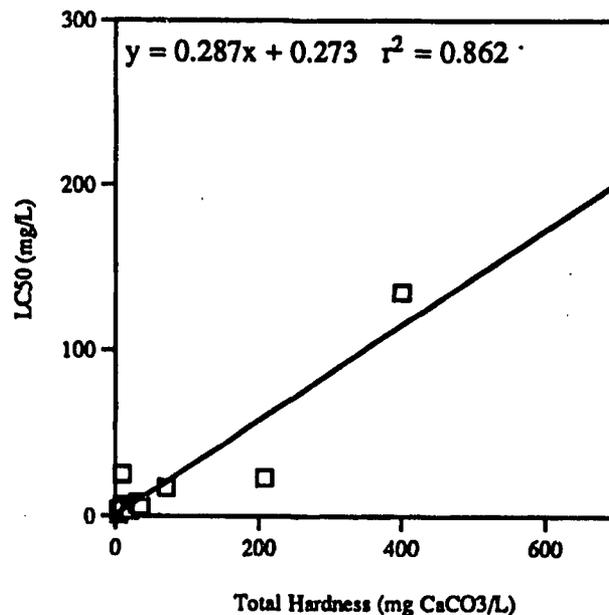


Figure 3.1. Relationship Between Acute Toxicity of Uranium to Freshwater Fish and the Total Hardness of the Test Water. Toxicity is expressed as the 96-h LC50. Data are obtained from references listed in Table 3.2.

trout embryos and larvae. An NOEC of less than 404 $\mu\text{g/L}$ was determined for gudgeon larvae (*Mogurnda mogurnda*) above which body length and weight were affected (Holdway 1992). The calculated threshold response for the most sensitive response (growth) in tropical fish was 200 $\mu\text{g/L}$ (Holdway 1992). At 5.8 mg U/L (total hardness of 5 mg CaCO₃/L), immediate respiratory distress was observed in adults of eight fish species (Bywater et al. 1991). Chronic exposure to elevated radionuclide levels from uranium mine tailings in Langley Bay, Lake Athabasca, Saskatchewan, Canada, did not affect hematocrit, histological characteristics of radiosensitive tissues, rate of parasitism, or growth of whitefish (*Coregonus clupeaformis*) and northern pike (*Esox lucius*). Exposure levels to uranium-series radionuclides in this study were 27 $\mu\text{g/g}$ uranium, 453 pCi/g radium-226, 700 pCi/g lead-210, and 6 pCi/g thorium-228 in the sediment (dry weight).

3.1.2 Uranium Toxicity in Terrestrial Biota

The uranium toxicity in terrestrial biota, including plants, invertebrates, and mammals is described in the following section.

Plants

Four-week-old soybean plants (*Glycine max* [L.] Merr.) grown hydroponically were adversely affected by uranium at concentrations of 0.42 $\mu\text{g/mL}$ and above (Murthy et al. 1984). Chlorosis, early leaf abscission, and reduction in root growth were the toxic symptoms induced by uranium. At 42 mg/L of uranium, widespread tissue necrosis was observed, and total leaf chlorophyll content was reduced by 30% to 40%. These symptoms were probably caused by reduced root absorption capacity and dysfunction of xylem and phloem tissue from uranium precipitation (Cannon 1960). Uranium concentrations in the roots of the affected soybean plants were 57 $\mu\text{g/g}$ dry weight (± 2.90) and 938 $\mu\text{g/g}$ dry weight (± 22.6) in the 0.42- and 42- $\mu\text{g/mL}$ -treated plants, respectively. Shoot concentrations were 1.37 $\mu\text{g/g}$ for the 0.42- $\mu\text{g/mL}$ -treated plants and 91.5 $\mu\text{g/g}$ for plants grown in the 42- $\mu\text{g/mL}$ uranium solutions.

In soil-grown plants, overt effects on growth and survival were not seen below 1000 mg U/kg of soil (Sheppard et al. 1992). However, Sheppard and Evenden (1992) have suggested sublethal toxicity may occur in plants grown in soils containing between 10 to 100 mg U/kg . In this exposure range, the ability to restrict uranium uptake appears to become impaired.

Uranium inhibited root growth in mature Swiss chard plants at soil concentrations of 10 $\mu\text{g U/g}$ in both sand and peat soils. Shoot yields were not affected (Sheppard et al. 1988).

Invertebrates

Earthworm survival was decreased at concentrations of 1000 mg U/kg dry soil and greater (Sheppard and Evenden 1992). Stewart et al. (1992) evaluated the downstream effects of drifted aquatic pond weeds and filamentous algae that originated in an impoundment near the U.S. Department of Energy's (DOE's) Y-12 Plant in Oak Ridge, Tennessee, on aquatic snails (*Elimia claviformis*) and amphipods (*Grammarus* sp.). The vegetation was contaminated with uranium, several heavy metals including chromium and cobalt, and poly-chlorinated biphenyls. In laboratory and in-stream feeding trials, the snails and amphipods distinguished between contaminated and noncontaminated pond weeds, generally avoiding the contaminated vegetation. The snails had lower growth rates on the contaminated plants. However, because of the presence of other contaminating chemicals in the vegetation, it cannot be concluded that uranium was responsible for the avoidance behavior and lowered growth rates of associated invertebrates.

Amphibians/Reptiles

No studies were found on the chemical or radiation toxicity of uranium in amphibians or reptiles.

Mammals

Sensitivity to uranium varies among mammal species. Rabbits, dogs, and guinea pigs are more sensitive to uranium exposure than rats by factors of 2 to 10, with rabbits being the most susceptible to uranium toxicity (Leach et al. 1984; Morrow et al. 1982; Morrow et al. 1981). Solubility of the uranium compounds governs their toxicity. The soluble hexavalent compounds are considerably more toxic than the less soluble tetravalent uranium compounds (Venugopal and Luckey 1978; Haven and Hodge 1949). The impact of uranium exposure on the animals is dependent not only on the dose and chemical form of uranium, but also on the route of exposure.

Toxic Response from Oral Exposure. The lowest oral LD50 value reported for mammals is 5.7 mg/kg in dogs exposed to uranium as uranyl nitrate hexahydrate [UO₂(NO₃)₂·6H₂O]. The oral LD50s for rats and mice are 115 mg/kg and 136 mg/kg [uranium as UO₂AC·2(H₂O)], respectively (Domingo et al. 1987). Threshold concentrations causing slight malaise in domestic livestock are 50 mg/d in sheep and 400 mg/d in dairy cattle (Garner 1963). The levels NOAELs for rodents are 11 mg/kg in rats and 25 mg/kg in mice. The lowest observable adverse effect level (LOAEL) for systemic, sublethal effects in rats is 118 mg U/kg. Adverse effects include hepatic lesions, proteinuria, and weight loss (Domingo et al. 1987). Based on a long-term feeding study, an NOAEL for uranium in dogs of 1 mg/kg body weight/d has been estimated (Bosshard et al. 1992).

In prolonged feeding studies (Maynard and Hodge 1949; Maynard et al. 1953; Tannenbaum and Silverstone 1951), soluble compounds such as uranyl nitrate hexahydrate, uranium peroxide, uranyl acetate dihydrate, and uranyl fluoride were lethal at lower levels than insoluble uranium compounds such as uranium dioxide and uranium tetrafluoride. For example, when uranyl nitrate hexahydrate [UO₂(NO₃)₂·6H₂O] was incorporated in the food, the NOAEL was 24 mg/kg/d (1 year) for rats. Renal necrosis occurred at 118 mg/kg/d and testicular pathology was observed at 474 mg U/kg/d (Maynard et al. 1953). However, no effects were observed in rats fed over 7500 mg U/kg/d in the form of uranium dioxide or uranium tetrafluoride for 2 years (Maynard and Hodge 1949; Maynard et al. 1953). In rabbits, no adverse effects from 30 days of uranyl nitrate hexahydrate at 4.6 mg/kg/d were observed, but levels of 469 ppm uranium in food resulted in death (Maynard and Hodge 1949). A summary of LOAELs for chronic oral exposures to uranium is presented in Table 3.3.

Toxic Response from Inhalation Exposure. Deaths from acute and chronic exposures to uranium compounds result from the chemical, not the radiological, effects of uranium and are attributed to renal toxicity (Leach et al 1984). Acute LC50s in small mammals range from 12,000 to 120,000 mg/m³ for exposures of less than 10 minutes in duration. A 60-minute exposure to uranium hexafluoride resulted in 100% mortality in rats exposed to 2160 mg/m³ of the compound (Leach et al. 1984). Chronic exposures (6.5 to 13 months) of rats, rabbits, guinea pigs, and dogs to aerosols of various uranium compounds resulted in kidney damage, blood abnormalities, and death. These data are summarized in Table 3.4.

Table 3.3. Lowest Concentrations of Uranium in Food and Drinking Water Causing Adverse Health Effects in Mammals (ATSDR 1990a)

<u>Concentration (ppm)</u>	<u>Exposure Duration</u>	<u>Effect</u>
Food		
9480	1 dose	Rats had fewer pups
94	30 days	Kidney damage in rabbits
469	30 days	Death in rabbits
1940	2 years	Death in rats
2315	48 weeks	Death in mice
Water		
16	Days 6-15 of gestation	Weight loss in mothers Deformities in pups
16	8-14 weeks during gestation and after pregnancy	Decreased pup weight in mice
21	Day 13 of gestation to day 21 of nursing	Maternal death in mice
64	4 weeks	Kidney, liver, blood effects in rats
471	4 months	Damage to testes in rats

Table 3.4. Lowest Concentrations of Airborne Uranium Causing Adverse Health Effects in Mammals (Stokinger et al. 1953)

<u>Airborne Uranium (mg/m³)</u>	<u>Exposure Duration</u>	<u>Effect</u>
0.05	1 year	Slight kidney injury in rats
0.20	7.5 months	Kidney damage in guinea pigs
0.25	6.5 months	Kidney damage and death in rabbits
0.25	1 year	Kidney damage and death in dogs

Toxic Response from Dermal Exposure. Orcutt (1949) reported dermal LC50s for uranyl nitrate applied to various mammal species. These values were 59 mg U/kg in rabbits, 2110 mg/kg in guinea pigs, 490 mg/kg in rats, and 7600 mg/kg in mice. Soluble uranium compounds (uranyl nitrate hexahydrate, uranyl fluoride, uranium pentachloride) were toxic when applied dermally to rabbits. The slightly soluble compounds (uranium trioxide, sodium diuranate, ammonium diuranate) were much less toxic, and the insoluble compounds (uranium dioxide, uranium tetrafluoride) were

nontoxic to rabbits (Orcutt 1949). Table 3.5 summarizes the dermal LOAEL observed for soluble and slightly soluble uranium compounds in rabbits.

Immunological Effects of Uranium Exposure. No studies have been reported on immunological effects of uranium exposure through the dermal route. Chronic oral (Stokinger et al. 1953) and inhalation (Maynard and Hodge 1949; Maynard et al. 1953; Tannenbaum and Silverstone 1951) exposures of rats, rabbits, guinea pigs, and dogs to various uranium compounds did not result in any significant histological changes in the lymph nodes, bone marrow, or spleen. An indication of an immune response to uranium exposure was noted in rats exposed to ammonium diuranate (Galibin et al. 1966). The exposed rats showed an increase in mouth microflora and in the phagocytic activity of neutrophils. Although the tracheobronchial lymph nodes of dogs exposed to uranium dioxide for greater than 3 years had areas of necrosis and fibrosis and were high in alpha radiation activity, there was no loss of circulating lymphocytes. This finding indicated that the immunological system was not functionally damaged (Leach et al. 1970). Rabbits given greater than 0.05 to 5 mg U/L drinking water for 6 months showed decreased antibody production and impaired resistance to infection (Novikov and Yudina 1970).

Reproductive Effects of Uranium Exposure. Pregnant mice exposed to 3 mg U/kg in their water (i.e., 16 mg/L) during days 6 through 15 of gestation showed decreased body weight and produced stunted fetuses with skeletal malformations (Domingo et al. 1989a). The reported NOAEL for pregnant mice is 0.3 mg/kg/d (Domingo et al. 1989b). Exposure to 6 mg U/kg/d for 4 to 8 weeks resulted in a serious decrease in pup viability (Paternain et al. 1989). A significant increase in total and late resorptions was observed in mice orally gavaged with 14 mg uranium (as uranyl acetate dihydrate)/kg/d for 4 to 8 weeks (Paternain et al. 1989).

Degenerative changes in the testes of rats have resulted from chronic oral exposure to uranium compounds. Administration of uranyl nitrate hexahydrate in the diet for 1 year produced testicular lesions at 474 mg U/kg/d (Maynard et al. 1953); administration of this compound via water for

Table 3.5. Lowest Concentrations of Uranium that Caused Adverse Health Effects when Applied to the Skin of Rabbits (Orcutt 1949).

<u>LOAEL</u> (mg U/kg/d)	<u>Compound</u>	<u>Exposure Duration</u>	<u>Effect</u>
267	Uranium trioxide	1 day (4 h/d)	Proteinuria
267	Sodium diuranate	1 day (4 h/d)	Proteinuria
6.7	Uranyl nitrate hexahydrate	1 day (4 h/d)	Proteinuria
64	Uranyl nitrate hexahydrate	1 day (4 h/d)	Weight loss
5	Uranyl nitrate hexahydrate	5 weeks (5 d/wk)	Death

4 months resulted in testicular histopathology at 66 mg U/kg/d (Malenchenko et al. 1978). Testicular atrophy was produced in rats after 2 years of exposure to 97 mg uranium (as uranyl fluoride)/kg/d (Maynard and Hodge 1949; Maynard et al. 1953). Exposure to 80 mg/kg/d uranyl acetate dihydrate via drinking water for 64 days resulted in significant histopathological changes in the testes of rats (Llobet et al. 1991). Although testicular function and spermatogenesis were not affected at this or lower concentrations, the uranium exposure produced a significant decrease in the pregnancy rate at 10, 20, 40, and 80 mg/kg/d, probably as a consequence of reduced spermatozoa counts.

Carcinogenic Effects of Uranium Exposure. No animal tests have been conducted to study cancer incidence following oral exposure to uranium. Although non-neoplastic kidney damage has been observed in numerous feeding studies, no tumors in any organs have been observed during these tests. Inhalation exposures with a calculated radiation dose to the lungs of dogs of 600 rad resulted in neoplastic changes in the lung tissue (Leach et al. 1973). It should be noted that exposure to any radioactive substance will potentially cause cancer, and enriched uranium would be expected to present a higher risk for cancer than natural uranium. Bone-seeking, alpha-emitting radionuclides such as radium-226 may give rise to tumors, particularly bone sarcomas (Rowland et al. 1978). Fatal cancer risk in humans from uranium exposure has been inferred from data on skeletal cancer induction by radium isotopes (Mays et al. 1985).

Genotoxicity. No studies have been conducted on the genotoxic effects of uranium in animals following oral or inhalation exposure. Chromosome aberrations have been reported for cultured lymphocytes of uranium miners (Brandom et al. 1978).

Biomarkers of Uranium Exposure/Effect. Kidneys, livers, and bones are uranium accumulator organs, although the cardiovascular system and central nervous system may also accumulate uranium. Kidney and bone tissues are the main targets of both the radiation and chemical toxicity of uranium in vertebrate organisms. Of these two tissues, kidney tissue is the most sensitive and is considered to be the key target organ for hazard assessment (Diamond 1989). The characteristic lesion of uranium poisoning in all mammal species studied is injury and necrosis of the terminal segments of the renal proximal tubule. Injury of the glomerulus is also reported for most species (Avasthi et al. 1980; Haley 1982, Haley et al. 1982). In dogs, the acute renal injury threshold appears to be less than 1 µg U/g kidney with histopathological changes evident in kidney tissue at organ concentrations greater than or equal to 0.5 µg/g kidney (Hodge et al. 1953). This concentration is also the injury threshold level observed in rats; however, nephrotoxicity resulting from less than or equal to 5 µg U/g kidney is reversible in this species (Morrow et al. 1982; Diamond et al. 1987).

Recently, sensitive biochemical parameters have been used to monitor uranium-induced kidney injury. Measures of B-2-microglobulin, amino acids, glucose, aspartate amino-transferase, and alanine amino-transferase activities in blood have been used to document glucosuria, proteinuria, and

tubular effects (osmotic diuresis, amino aciduria, and enzymuria) of uranium damage (Leach et al. 1984; Domingo et al. 1989a; Diamond 1989). No one biochemical biomarker has been identified for use in uranium hazard monitoring.

The critical target organ for chronic exposure to uranium is the skeletal system (Adams and Spoor 1974; Guglielmotti et al. 1984). However, skeletal burden/exposure relationships have not been determined for laboratory or wild mammal species.

Fecal and urinary samples may be used to identify or quantify exposure to uranium (ATSDR 1990a). Fecal sampling can provide information on current uptake levels, but gives no information on body burden (Schieferdecker et al. 1985). It is possible, in humans, to use urinary excretion rates to determine body burden (Lippman et al. 1964). However, this approach has not been applied to animals thus far.

Uranium Toxicokinetics: Metabolism and Distribution. Gastrointestinal absorption of soluble uranium compounds in humans appears to range between 0.5% and 30% (Hursh and Spoor 1973; Hursh et al. 1969; ICRP 1979; DeRay et al. 1983). An average absorption rate of 5% is commonly used for uranium risk assessments in humans (ICRP 1979). In animal studies, dogs, rabbits, and hamsters absorbed about 0.5% to 2% of the administered uranium dose (Wrenn et al. 1985; Harrison and Stather 1981). Rats absorbed less than 0.1% (Wrenn et al. 1985; Sullivan 1980). However, if the dose was administered to fasted rats, gastrointestinal absorption increased to 0.6% to 2.8% (LaTouche et al. 1987). For the less soluble compounds such as uranium dioxide, gastrointestinal absorption approached 0.2% (ICRP 1979). Seventy percent to 85% of the absorbed uranium was rapidly excreted in the urine (Hursh and Spoor 1973; Priest et al. 1982) and less than 1% in feces (NRCC 1982). About 12% to 20% of the absorbed uranium was assumed to be retained in the kidney with a retention half-life of 6 days (Friberg 1977; Adams and Spoor 1974; ICRP 1979). Another 0.5% to 20% of the absorbed dose was deposited in the skeleton with a retention half-life of about 1500 days (ICRP 1979; NRCC 1982).

Birds

No studies were found on the chemical or radiation toxicity of uranium in avian species.

3.2. Bioconcentration Factors and Trophic Transfer Coefficients

Uranium, which forms relatively insoluble compounds in the environment and has no known essential biological function, is not biologically mobile. It attaches to surfaces and accumulates in soils and sediments (Schultz and Whicker 1982). Uranium enters the food chain via adsorption on surfaces of plants and small animals. Because of membrane discrimination against uranium, little uranium is accumulated internally in biota. Consequently, concentration factors for uranium decline substantially with trophic level.

3.2.1 Uranium Transfer in Aquatic Food Chains

Reported water-based bioconcentration factors for uranium in fresh water algae include 1576 (Mahon 1982) and 2096 (Stegnar and Kobal 1982). Water bacteria reportedly concentrate uranium by factors of 2794 to 354,200. It has been suggested that the apparently high bioaccumulation of uranium by algae and bacteria may be due to adsorption of the radionuclide onto cell surfaces rather than actual uptake by the organisms (Atkins 1977; Horikoshi et al. 1981). Maximum accumulation of cell-bound uranium in algae occurs at pH 5.9 to 6.8 (Marvan 1976) and in waters containing low phosphate and carbonate levels (Nakajima et al. 1979). Cell-bound uranium can reach up to 10% to 15% of dry cell weight of algae and other micro-organisms (Strandberg et al. 1981).

Thompson et al. (1972) reported a coefficient of 0.55 for uranium transfer from water to aquatic macrophytes. A transfer coefficient from sediment to pond weeds (*Potamogeton foliosus*) growing in an impoundment near the DOE's Y-12 Plant in Oak Ridge, Tennessee, was 0.0225 (calculated from data presented in Stewart et al. 1992). However, according to the authors, the analytical methods used underestimated uranium concentrations. Therefore, the bioconcentration factor reported for the pond weed was likely underestimated. Using the reported water concentration and the levels of uranium found in pond weeds from a lake receiving uranium mine tailings in Saskatchewan, Canada, a bioconcentration factor of 1.13 can be calculated for *Potamogeton* species and a factor of 1.5 determined for *Myriophyllum* species. Sediment-to-plant transfer coefficients for the pond weeds were 0.16 and 0.20 for *Potamogeton* and *Myriophyllum* species, respectively (Waite et al. 1988).

Accumulation of uranium in plankton was 459 times that of the water concentration, and bioconcentration factors of 306 for mollusca (*Pisidium*) and 14.7 for fish (*Oncorhynchus mykiss* and *Catostomus catostomus*) were reported for the aquatic food chain described by Mahon (1982). Thompson et al. (1972) reported a bioconcentration value of 60 for both mollusca and crustacea and a value of 2 for fish in aquatic systems exposed to uranium. In general, water-to-invertebrate transfer coefficients for uranium are more variable than those reported for fish, probably because of the greater variation in trophic and spatial niches among invertebrates (Swanson 1985). Reported water-to-invertebrate transfer coefficients ranged from 1 to 10,000. However, the majority of the coefficients fall between 100 and 1000 (Anderson et al. 1963; EPS 1978, Gulf Minerals Canada 1980; Mahon 1982; OWRC 1971; Reichle et al. 1970a, 1970b; Thompson et al. 1972; Van der Borgh 1963; Swanson 1985). The highest reported bioconcentration factors for uranium in rainbow trout (*Oncorhynchus mykiss*), suckers (*Catostomus catostomus*), and lake whitefish (*C. clupeaformis*) did not exceed 38 (Mahon 1982; Poston 1982; Swanson 1985). Eyed carp eggs accumulated uranium in the yolk material and concentrated the radionuclide by a factor of 3.3 from water (Till and Blaylock 1976). Bioconcentration factors for uranium in brook trout eggs and fry ranged from 1.9 to 4.3 in hard water (210 mg/L as CaCO₃) (Parkhurst et al. 1984). Although transfer coefficients from water to fish ranged from 1 to 650 (Anderson et al. 1963; EPS 1978; Gulf Minerals Canada 1980; OWRC 1971; Thompson et al. 1972; NRC 1977; Key Lake Mining 1979; Cluff Mining 1979; Parkhurst et al. 1984; Waite et al. 1988; Swanson 1985), the assimilation efficiency for uranium in fish in most

studies was low, with transfer coefficients less than 50. In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for uranium in the flesh of freshwater fish are 10 (NRCC 1983), 20 for fish-eating and plankton feeding fish in water of low mineral content and 2 for fish in water of high mineral content (CSA 1987). A conservative default bioconcentration factor for bottom-feeding fish is 50 (Poston and Klopfer 1986; Myers 1989).

Sediments act as a sink for uranium with the concentration of uranium in the sediments and suspended solids several orders of magnitude higher than the concentration in water. Transport of uranium from water to organisms occurs primarily through the sediment (Brunskill and Wilkinson 1987; Swanson 1985). However, few data are available on sediment-to-organism transfer of radionuclides. In a study of the transfer pathways and effects of uranium-series radionuclides in a stream and a lake receiving contaminated drainage from uranium mill tailings, organisms feeding on or near sediments were found to contain higher levels of uranium than pelagic or predatory species (Swanson 1985). Bottom feeders such as midge larvae (*Chironomus sp.*) and caddisfly larvae (*Nemotaulius sp.*) had uranium concentrations of 15 $\mu\text{g/g}$ and 26 $\mu\text{g/g}$, respectively, compared to 5 $\mu\text{g/g}$ for the predatory dragonfly nymphs. Sediment-to-insect transfer coefficients ranged from 0.1 to 0.3 in this study. Concentration of uranium from insects to forage fish varied between 0.08 for caddisfly larvae transfer to lake chub (*Couesius plumbeus*) and 1.3 for blackfly uranium transfer to small white sucker (*Catostomus commersoni*). Transfer coefficients from forage fish to large white fish were 0.04 (flesh) and 0.98 (skin). Overall sediment-to-fish transfer coefficients ranged from 0.02 to 0.05. Water-to-fish coefficients were 5.7 to 11.0 (Swanson 1985).

In general, there is a decline of about one order of magnitude in the bioconcentration factor at each step in the aquatic food chain (Mahon 1982; Blaylock and Witherspoon 1978; Kovalsky et al. 1967; Thompson et al. 1972; Swanson 1985). Thus, no biomagnification of uranium from the aquatic or semi-aquatic (amphibian, waterfowl, and mammal) food chain is expected.

3.2.2 Uranium Transfer Through Terrestrial Food Chains

Transport of uranium from soil to biota has been documented (Dreesen et al. 1982; Moffett and Tellier 1977; Mahon 1982). It has been assumed that the nature of the soil determines the amount of bioavailable uranium. For example, soil conditions that favor decreased sorption or formation of soluble complexes with uranium will enhance uptake. Swiss chard grown in sandy soils contained 80 times higher concentrations of uranium than chard grown in peat (Sheppard et al. 1983). However, in a study of the effect of 11 different soil types on bioavailability indices for uranium (Sheppard and Evenden 1992), no correlation between plant or invertebrate uptake and soil parameters was observed. The soils were treated with up to 10,000 mg U/kg soil and varied with regard to texture, clay, organic content, pH, background uranium content, and cation exchange capacity. Uranium concentrations in plants and earthworms were not linearly related to uranium concentrations in the soil. Thus, a single value for use as a conservative concentration ratio for a soil type could not be determined, and the implication is that other reported concentration ratios for uranium in plants

should not be applied to soil concentrations outside those for which the concentration ratio was determined. Concentration ratios in plants and earthworms associated with the soil types are summarized in Table 3.6.

Uranium appears to be restricted to the root system of plants and may be precipitated on the outer root membrane rather than accumulated in the interior of the root (Sheppard 1985). Little uranium enters the root sap system (Robards and Robb 1972), and virtually no uranium is translocated from the soil to the above-ground plant tissue (Sheppard 1983; Van Netten and Morley 1983). Several concentration ratios have been reported for shoots, leaves, fruits and seeds (the "edible") portions of plants, but all of these ratios were less than 1. Ng et al. (1982) reported uranium concentration ratios for edible portions of food crops (wet plant/dry soil) from 1.7×10^{-7} to 2.0×10^{-2} . The range of concentration ratios for the edible portions of pasture plants was 1.6×10^{-6} to 8.5×10^{-1} (Ng et al. 1982). Garten et al. (1987) reported concentration factors for plants grown in a contaminated flood plain as 9.3×10^{-1} for leaves and stems of standing crops, 9.2×10^{-2} for fruit and vegetables, 6×10^{-2} for fescue, and 7×10^{-2} for tree leaves. Concentration ratios greater than one appear to be associated with dusty conditions (Garten et al. 1987).

Mahon (1982) studied the trophic transfer of uranium in several wildlife food chains. For the terrestrial food chains studied, there was a drop in body burden of uranium by one order of magnitude for each trophic level (Mahon 1982). The transfer coefficient from vegetation [grouseberry forb (*Vaccinium scoparium*), lichens (*Bryoria freemontia* and *Alectoria sarmentosa*), and grass (*Calamagrostis rubescens*)] to deer (*Odocoileus hemionus*) was 0.7. Bioconcentration ratios for chipmunks (*Eutamias amoenus*) and herbivorous mice feeding on fireweed seed heads (*Epilobium angustifolium*) and grouseberry were 0.5 and 0.26, respectively. The top predator in this food chain was an avian predator, the raven (*Corvus corvus*), which was found to have less than 5 ppb uranium in its tissues. Similar uranium food chain transfers were seen in domestic animal foragers. Transfer coefficients from soil surface layer (0 to 30 cm) to forage grass ranged from 2.67×10^{-5} to 2.98×10^{-4} . Forage grass-to-sheep transfer coefficients were 2.5×10^{-5} to 2.4×10^{-4} in meat.

It should be noted that uranium uptake from water consumption was not addressed in these studies, nor were root-consuming organisms even though roots appear to be a significant source of exposure (Van Netten and Morley 1983).

Table 3.6. Accumulation of Uranium in Plant and Soil Invertebrates as Related to Soil Type (Sheppard and Evenden 1992)

Feature	Texture	Soil Type				Soil Uranium (mg/kg)	Concentration Ratio	
		Clay (%)	pH	Organic (%)	CEC ^(a)		Earthworm	Radish
Heavy	Clay, loam	33	7.0	3.1	24.1	2.1	0.37	0.39
Heavy	Loam	24	7.5	2.2	14.4	2.1	0.38	0.04
Garden	Fine sand, loam	18	7.5	18.4	50.5	3.1	0.089	0.013
Medium	Fine sand, loam	15	7.3	2.6	13.0	2.1	0.34	0.076
Medium	Fine sand, loam	13	6.6	5.7	19.9	1.8	0.31	0.028
Carbonated	Fine sand, loam	12	7.8	4.2	17.8	2.1	0.46	0.063
Acid sand	Loam, sand	6	5.5	3.5	17.0	4.1	0.66	0.025
Neutral sand	Loam, fine sand	4	7.8	0.8	4.4	1.5	0.97	0.047
Limed sand	Fine sand	2	6.2	1.0	11.0	0.6	1.27	0.094
Acid sand	Fine sand	2	4.9	0.7	5.4	0.6	1.53	0.237
Organic		<1	5.1	41.5	81.4	4.9	0.082	0.014

(a) Cation Exchange Capacity (cmol/kg).

4.0 Plutonium

4.1. Plutonium Toxicity

The toxicity of plutonium is related to the radioactive properties of the radionuclide rather than its chemical properties. Its chemical properties affect the distribution, biological half-life, and the retention of plutonium in target organs. Plutonium emits alpha particles that are highly ionizing and, therefore, damaging. However, tissue penetration is slight, and biological damage is limited to cells in the vicinity of the alpha-emitting substance. Plutonium isotopes generally exist as complexes with other elements and compounds. These complexes vary in their solubility in living tissues and, thus, vary also in their uptake, transport, and retention in an organism.

4.1.1 Plutonium Toxicity in Aquatic Biota

A plutonium activity concentration of 7.5 $\mu\text{Ci/mL}$ (0.4 ppm) reduced hatching success of carp eggs (*Cyprinus carpio*). All larvae that hatched were abnormal and died within a few hr (Till 1978). The lowest concentration that produced a significant effect in the carp eggs was 1.6 $\mu\text{Ci/mL}$. Fathead minnows were more sensitive to plutonium. A concentration of 1.0 $\mu\text{Ci/mL}$ decreased the hatching of minnow eggs. The lowest concentration that increased the frequency of abnormal larvae was 0.076 $\mu\text{Ci/mL}$ (Till and Baylock 1976). A concentration of 1.0 $\mu\text{Ci/mL}$ (0.06 ppm) affected the hatching of fathead minnow eggs.

Information on the effects of ionizing radiation is summarized in Section 2.1.

4.1.2 Plutonium Toxicity in Terrestrial Biota

The toxicity of plutonium in terrestrial biota is described below.

Plants

Information on the effects of ionizing radiation is summarized in Section 2.1.

Invertebrates

In long-term field experiments with plutonium-239/241 in chernozem soils (1780 Ci/m², plowed to a depth of 25 to 30 cm and sown in wheat), the radionuclide was shown to decrease the population density of earthworms and insect larvae by 50% over a period of 3 years. Microarthropod populations were decreased by a factor of 7.5. Plutonium was particularly radiotoxic to those microrarthropod species that have a fast rate of development such as gamasid and throglyphoid mites. The density of small acariform mites decreased by a factor of 18 in plutonium-contaminated plots. After

18 years, macrofaunal populations were comparable to those in control plots when the major portion of plutonium-241 had been transformed to americium-241 (Krivolutsky et al. 1992).

Information on the effects of ionizing radiation is summarized in Section 2.1.

Mammals

High-radiation doses of plutonium have resulted in decreases in life span, injury of the respiratory tract, and cancer. Target tissues are the lungs and associated lymph nodes, bone, and liver.

Toxic Response from Oral Exposure. Little information is available on the toxic response of mammals to oral ingestion of plutonium compounds, probably because of the very limited absorption of plutonium from the gastrointestinal tract. (See Section 4.2.4., Plutonium Toxicokinetics: Metabolism and Distribution.)

The NOAEL related to mortality from acute oral exposure to plutonium (as plutonium-238 citrate) is 1×10^5 pCi plutonium-238/kg body weight in neonatal rats. Exposure to 3.3×10^8 pCi/kg resulted in 45% mortality and growth inhibition in the survivors (Fritsch et al. 1987).

Histological changes were observed in the large intestine of adult rats given 160 mCi plutonium-239 dioxide/kg body weight. However, the changes were resolved by 6 days post-exposure (Sullivan et al. 1960). Histological changes in the gastrointestinal tract (e.g., hypertrophy of the crypts of the small intestine) of neonatal rats were produced when the rats were exposed to 1.0×10^5 pCi plutonium-239 dioxide/kg body weight. Exposure to 3.3 mCi/kg resulted in intestinal hemorrhaging and disappearance of the crypts (Fritsch et al. 1987). However, as Fritsch et al. (1987) points out, immature development of the crypts of the small intestine is characteristic of neonatal rats, suggesting that young rats may be more sensitive to the radiological effects of plutonium than adult rats or other neonatal mammals.

Toxic Response from Inhalation Exposure. Significant decreases in longevity have been reported in rats, mice, hamsters, and dogs exposed to aerosols of plutonium-239 or plutonium-238. Early death (within 1 to 3 years after exposure) was usually caused by radiation pneumonitis and related respiratory damage. Single exposures resulting in lung depositions of 2.3×10^4 to 7.2×10^6 pCi/kg body weight decreased survival time in these species in a dose-related manner (Metivier et al. 1986; Sanders 1977, 1978; Sanders et al. 1986; Lundgren et al. 1987; Dagle et al. 1988; Park et al. 1988). Dogs receiving about 1.0×10^6 pCi plutonium/kg body weight died within 600 days of exposure, whereas dogs receiving 2.1×10^5 pCi/kg survived 1000 to 2000 days (Mewhinney et al. 1987). Longer exposures (i.e., once every other month for a total of 6 doses over 10 months) at somewhat lower doses (deposited levels of 1.8×10^4 pCi/kg) also resulted in decreased survival time in mice (Lundgren et al. 1987). However, hamsters receiving similar exposures had

survival times similar to controls (Lundgren et al. 1987). Chronic exposure to plutonium at levels below those causing radiation pneumonitis can result in fibrosis and associated pulmonary dysfunction (Muggenburg et al. 1986).

The earliest observed biological effect in mammals chronically exposed to plutonium was lymphopenia. Lymphopenia occurred in dogs at deposited levels of about 6.1×10^3 pCi/kg for plutonium dioxide (Park et al. 1988) and at 1.3×10^5 pCi/kg for plutonium-239 nitrate (Ragan et al. 1986; Dagle et al. 1988). Deposited lung tissue levels of 2.4×10^4 pCi/kg body weight and higher in dogs (Park et al. 1988) and 3.5×10^5 pCi in hamsters (Lundgren et al. 1983) resulted in degenerative liver lesions. Levels as high as 7.1×10^4 did not cause liver lesions in hamsters (Lundgren et al. 1983).

Toxic Response from Dermal Exposure. No studies are available on the effects of plutonium on the health of wild or domestic mammals following dermal exposure to plutonium.

Immunological Effects of Plutonium Exposure. Inhaled plutonium is transported to and concentrated in lymph nodes, reaching higher concentration in the lymph nodes than in the lungs (Bair et al. 1989). Lymphadenopathy in dogs was associated with plutonium levels in lung tissue as low as 1.7×10^3 pCi/kg body weight of plutonium-239 dioxide (Park et al. 1988). Other immune system effects in mammals included development of fibrosis of the tracheobronchial lymph nodes in dogs (Gillett et al. 1988), decreased numbers of antibody-forming cells in hamsters (Bice et al. 1979), reduced numbers of pulmonary alveolar macrophages in mice (Moores et al. 1986), and depressed primary antibody responses in dogs (Morris and Winn 1978). The LOAEL for depressed antibody production was 7.1×10^4 pCi/kg body weight (Bice et al. 1979), and the lowest plutonium level causing decreased macrophage production was 4.5×10^4 pCi/kg (Moores et al. 1986).

Reproductive Effects of Plutonium Exposure. No studies were found on the reproductive effects of inhaled or ingested plutonium. Plutonium exposure of male mice via intravenous injection (1.6×10^6 to 1.6×10^7 pCi/kg) resulted in fetal intrauterine deaths in female mice mated with male mice treated 4 weeks prior to mating (Lüning et al. 1976a, 1976b). Exposure to higher concentrations resulted in male sterility at 12 weeks post-exposure (Lüning et al. 1976a, 1976b). The dominant lethal mutations caused by the plutonium were also expressed in the F1 males and affected the F2 generation (Lüning et al. 1976a, 1976b).

Carcinogenic Effects of Plutonium Exposure. Depending on the route of exposure, chemical form, and mammalian species, plutonium is a lung, skeletal, and liver carcinogen. Lung tumors were the most frequently observed cancer in dogs exposed to plutonium-239. The LOAEL related to lung tumor development in dogs for plutonium-239 was as low as 6.2×10^3 (Park et al. 1988) to 2.1×10^4 pCi/kg body weight (Muggenburg et al. 1987). Intermittent exposure to plutonium-239 dioxide with lung deposition levels totaling 8.6×10^4 pCi plutonium-239/kg body weight resulted in a high incidence of lung tumors in rats (Sanders and Mahaffey 1981).

Tumors have also been reported in the bone and liver, which are organs that accumulate transported soluble plutonium (e.g., plutonium-238 and the more soluble plutonium-239 forms such as nitrate) from the lung. The primary cause of cancer deaths in dogs exposed to aerosols of plutonium-238 was osteosarcomas. The lowest level producing bone cancer was 1.4×10^3 (Park et al. 1988) to 2.3×10^4 (Dagle et al. 1988).

Dogs receiving a single inhalation dose of 1400 pCi/kg plutonium via inhalation developed bone cancer after 4 years. Mice exposed to plutonium-239 once every other month for a total of six doses over 10 months developed bronchial hyperplasia at deposition levels of 8.1×10^4 and above (Lundgren et al. 1987).

Note that Syrian hamsters appear to be resistant to lung tumor induction by acute, intermittent, or chronic exposure to plutonium or other alpha-emitting radionuclides (Sanders 1977; Lundgren et al. 1983; ATSDR 1990b).

Genotoxicity. In hamsters, inhaled plutonium at deposited levels of 1×10^7 to 2.6×10^8 pCi/g lung tissue resulted in a dose-related increase in the frequency of chromosomal aberrations in blood cells 30 days after exposure (Brooks et al. 1976).

Biomarkers of Plutonium Exposure/Effect. Alpha activity of the urine is a well-established biomarker of exposure to plutonium. Models have been developed to estimate body burden of radiation workers from radioactivity in the urine. It should be noted, however, that body burdens of plutonium determined from tissue analysis at autopsy have been lower than those generated from urinalysis data (Voelz et al. 1979). No data are available on the relationship between exposure and the levels of radioactivity in the urine.

No plutonium-specific biomarkers of effect have been reported. Lymphopenia is the earliest observed biological effect in dogs and can be defined by a dose-response relationship related to inhaled plutonium (Park et al. 1988; Ragan et al. 1986). The degree to which this is a universal marker in mammals is unknown. Chromosome aberrations are also produced by plutonium exposure, but a large number of other chemicals also cause this effect, limiting the usefulness of this effect as a biomarker.

Plutonium Toxicokinetics: Absorption and Distribution. Absorption of plutonium from the gastrointestinal tract is minimal. Plutonium citrate and nitrate are absorbed more readily than other plutonium compounds. In adult rats and hamsters, absorption of these compounds ranges between 0.003% and 0.01% (Carrit et al. 1947; David and Harrison 1984; Stather et al. 1981). Absorption is age-related, and the ability of hamsters to absorb plutonium diminished from 3.5% to 0.003% from 1 to 30 days of age (David and Harrison 1984). Neonatal rats, hamsters, guinea pigs, and dogs absorbed 3% to 6% of administered plutonium (Cristy and Leggett 1986).

Absorption of inhaled plutonium is dependent on the mass deposited, the chemical compound and the particle size (Bair et al. 1962; Guilmette et al. 1984). Less soluble plutonium compounds, such as plutonium-239 dioxide, will be retained longer in lung tissue following inhalation than the more soluble forms, such as plutonium-239 nitrate and the plutonium-238 compounds. Particle size determines the deposition pattern in the lung and the clearance of the radionuclide from the lung. Thus, retention and radiological dose are directly related to particle size as is distribution to target organs. In mammals, particle sizes less than or equal to 10 μm Activity Median Aerodynamic Diameter (AMAD) are able to penetrate to the deep lung and be available for absorption (NEA 1981).

Dermal uptake of plutonium on intact palmar skin of a human is less than or equal to 0.0002%/h in acid solution (Langham 1959). Hair follicles (Weeks and Oakley 1955) and sweat and sebaceous glands (Buldakov et al. 1972) have been found to be portals for plutonium uptake through the skin.

Soluble forms of plutonium are accumulated largely in bone and liver (Dagle et al. 1985; Morin et al. 1972), whereas the less soluble forms are distributed to the lymph nodes and liver (Bair et al. 1966; Park et al. 1972). An hepatic uptake of 45% has been adopted by ICRP (1975a, 1975b, 1979, 1986) for setting annual limits of intake for plutonium. However, partitioning between the liver and skeleton varies widely among individuals (ICRP 1986), and hepatic uptake of 55% and 68% have been reported (Talbot et al. 1992).

In tests on dogs, ingested plutonium was excreted in the feces. About 98% of the dose was excreted after 5 to 6 weeks (Toohey et al. 1984). Total retention of plutonium in mice and rats ranged from 0.17% to 0.24% (Larsen et al. 1981). Loss of deposited plutonium from the liver was rapid (Taylor et al. 1981, 1983), and liver retention in laboratory mice and rats was 0.036% and 0.54%, respectively (Larsen et al. 1981). However, deer mice (*Peromyscus maniculatus*) and grasshopper mice (*Onychomys leucogaster*) retained plutonium in the liver for prolonged periods (Taylor et al. 1981, 1993). Neonates retained 100 times more plutonium from oral uptake than did adults (Sullivan et al. 1984). Inhaled plutonium was excreted in two phases. In the first phase (20 to 30 days in rats), about 70% to 76% of the plutonium was removed. The remainder was removed during the second phase (180 to 250 days) (Sanders et al. 1986, 1977). However, translocated plutonium may be retained in the body for many years. About 85% of the plutonium-239 dioxide dose inhaled by dogs was retained up to 10 years post-exposure (Park et al. 1972).

Birds

No information is available on plutonium effects in wild birds. Ionizing radiation effects are summarized in Section 2.1.1.

4.2 Bioconcentration Factors and Trophic Transfer Coefficients

Solubility of plutonium depends on the chemical form in which it enters the soil or sediment environment, the properties of the soil/sediment, the presence of complexing agents, and the soil/sediment microbe content (Bell and Bates 1988; Kabata-Pendias and Pendias 1984; WHO 1983; Wildung and Garland 1980). Once plutonium enters the soluble phase, it becomes available for uptake by plants. Availability of plutonium in water is dependent upon the oxidation state and the nature of the sediment and suspended solids.

4.2.1 Plutonium Transfer in Aquatic Food Chains

Freshwater studies indicate that plutonium is concentrated in algae but decreases by about a factor of 10 at each trophic level in the food chain (Noshkin et al. 1973; Hanson 1975). Concentration ratios for algae range between about 2.8×10^2 to 5×10^6 on a wet weight basis (Noshkin et al. 1973; Hanson 1975). Transfer of plutonium from water to seston was very high (1.7×10^4 to 1×10^6) in freshwater systems at the Rocky Flats plutonium fabrication plant, Golden, Colorado. The bioconcentration of plutonium in the zooplankton of the ponds was about 1.6×10^3 relative to plutonium content of the water. The plutonium transfer coefficient from phytoplankton to zooplankton was about 0.1. A concentration factor of 320 to 1290 was found in crayfish in the contaminated ponds. Over 75% of the plutonium in the crayfish was associated with the exoskeleton. Fish (minnows, carp, and bass) accumulated very little of the plutonium. Concentration factors relative to water ranged from 0 to 34 for whole fish. Plutonium was not detected in any fish flesh samples (Paine 1980). Plutonium concentration ratios of 5×10^6 (water-based) have been reported for aquatic snails. Aquatic beetles (*Coleoptera* sp.) concentrate plutonium by a factor of 3×10^5 (water) (Eyman and Trabalka 1980). Fish eggs have been shown to concentrate plutonium by a factor of 4 over the concentration of the water (Till 1978). In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for plutonium in the flesh of freshwater fish are 350 (NRCC 1982), 50 for fish in water of low mineral content and 10 for fish in water of high mineral content (CSA 1987), 250 (Myers 1989), and 5 for piscivores fish, 25 for planktivores, and 250 for bottom-feeding fish 5 (Poston and Klopfer 1985).

Bottom sediments are a major reservoir for plutonium in the aquatic environment. Trabalka and Eyman (1976) determined the sediment-based transfer factors for plutonium-237 in the biota of an aquatic microcosm. Plant uptake of plutonium was much greater in submerged vegetation and algae (0.2 to 27.0) than in emergent plants (0.03 to 0.11) and may, in part, be due to adsorption of the radionuclide to the submerged plant tissue. Transfer coefficients in whole animals ranged from 1.2 to 9.9. Trophic transfer coefficients for the biota in the microcosm are listed in Table 4.1. Another microcosm study (Trabalka and Frank 1978) produced similar trophic transfer coefficients, but added a factor for the larvae of the dipteran family, specifically *Chironomus riparus*. Dipteran larvae are important components of the diet of freshwater fish. They inhabit and feed on bottom sediments where plutonium contamination is greatest. The reported transfer factor for the dipteran larvae in the microcosm was 0.4 to 0.79. However, this value was for larvae from which the gut contents had been

Table 4.1. Sediment-Based Trophic Transfer Coefficients for Plutonium-237 in Biota of an Aquatic Microcosm (Trabalka and Eyman 1976)

<u>Organism</u>	<u>Sample Type</u>	<u>Trophic Transfer Coefficient</u>
Emergent Plants		
Grass (<i>Panicum</i>)	Leaves and shoots	≤0.045
Cattail (<i>Typha</i>)	Leaves and shoots	≤0.035
Watercress (<i>Nasturtium</i>)	Leaves and shoots	≤0.089
Submerged Plants		
Algae (<i>Oedogonium</i>)	Clumps	9.1
Moss (<i>Hygrohypnum</i>)	Branches	27.0
Stonewort (<i>Chara</i>)	Branches	0.19
Pondweed (<i>Potamogeton</i>)	Leaves and shoots	2.5
Invertebrates		
Snails (<i>Physa</i>)	Whole body	1.5-2.4
	Carcass	1.2
(<i>Gyraulus</i>)	Whole body	6.4
(<i>Goniobasis</i>)	Whole body	5.0-9.9
	Carcass	3.5
Amphipod (<i>Hyaella</i>)	Whole body	3.6
Vertebrates		
Goldfish (<i>Carassius</i>)	Whole body	2.3
	Flesh	0.47

removed. A more realistic transfer coefficient also would incorporate the gut content exposure (Trabalka and Frank 1978). With the gut contents included, the trophic transfer coefficient for dipteran larvae is 7.1.

Aquatic birds do not appear to bioconcentrate plutonium above levels found in their diet. Plutonium accumulation (3.2 pCi/kg) in the viscera of the thick-billed murre and black guillemot (*Ceppus grylle*) was similar to that of the zooplankton in birds' diet (Lowman et al. 1970). Eider

duck (*Somatiera* sp.) feeding in an area where plutonium had been released from nuclear weapons in an aircraft accident near Greenland had tissue concentrations equal to background levels (Aarkrog 1971).

Concentration ratios for plutonium in aquatic macrophytes are 10^{-2} to 10^{-1} relative to sediment plutonium concentrations (Emery et al. 1974; Emery and Farland 1974; Emery et al. 1975a, 1975b; Emery et al. 1980; Paine 1980).

Plutonium Transfer through Terrestrial Food Chains

Most (96% to 98%) of the plutonium entering soils is initially immobilized (Garland and Wildung 1977) and only a small portion is soluble in soil solution (Jacobson and Overstreet 1948; Price 1972). Because of the small fraction of soluble plutonium in soils, accumulation of plutonium in soil-dwelling organisms is low. The bioconcentration factor for earthworms living in soils containing 3.5 MBq plutonium-239/Kg was 0.0034 (Krivolutsky et al. 1992).

In addition to hydrolysis of plutonium in soil to insoluble forms that are unavailable to the plant, discrimination by plants against plutonium at the root membrane level also occurs (Garland et al. 1987). Soil-to-plant concentration ratios ranging from 1×10^{-6} to 2.5×10^{-4} plutonium in wet vegetation/plutonium in dry soil are typically measured in laboratory studies and crop plants. The findings indicate that plutonium is relatively unavailable for incorporation into plants (Jacobson and Overstreet 1948; Price 1972; Romney and Davis 1972; Wilson and Cline 1966). However, soil microbes have been found that are resistant to plutonium and increase the solubility of the radionuclides in soil. The uptake of plutonium on successive cropping of plutonium-contaminated soil increased to 0.01% of the plutonium present in the soil (Robinson et al. 1977). It should be noted that concentration factors for native plants range from 0.02 to 0.7. These values are 0.1 to 10,000 times greater than the concentration factors observed in laboratory studies (Hakonson et al. 1973; Hakonson and Johnson 1973; Larson et al. 1951; Leitch 1951; Olafson et al. 1957; Whitner et al. 1973). A general concentration factor of 2×10^{-3} has been commonly used for plutonium uptake in plants (Garten et al. 1987). A concentration factor of 10^{-4} was reported for plutonium in plants from the Nevada Test Site (Olafson and Larson 1963).

Once plutonium is absorbed by plants, natural ligands or metabolites effectively stabilize the plutonium. Thus, plutonium is not very mobile in plants as evidenced by low stem, leaf, and seed concentrations (Cataldo et al. 1987). Indeed, the roots contain the highest concentration of plutonium in the plant. The plutonium may be present in the root as a stabilized complex, a soluble complex, or a surface-adsorbed complex (Garland et al. 1981). About 1% to 3% of the plutonium in an ecosystem is associated with the root.

Because little plutonium is associated with edible vegetation, organisms feeding on the above-ground portions of the plants accumulate very little of the radionuclide. Therefore, plutonium is not concentrated along terrestrial food webs. Inhalation of plutonium particles and ingestion from

grooming may be important sources of plutonium contamination in fossorial animals (Hanson 1975). Garten et al. (1987) reported a concentration ratio of 4×10^{-5} in terrestrial mammals. Concentration factors for plutonium in desert mammals of the Nevada Test Site are listed in Table 4.2.

Table 4.2. Concentration Ratios for Plutonium in Desert Mammals (Romney et al. 1970, 1979)

<u>Trophic Level</u>	<u>Carcass/Soil</u>	<u>Carcass/Vegetation</u>
Granivore	0.0067-0.17	0.02-0.51
Omnivore	0.0067-0.053	0.020-0.157
Insectivore	0.02-0.080	0.0588-0.235

5.0 Cesium

5.1 Cesium Toxicity

Cesium is a chemical analog of potassium and exhibits relatively low toxicity in most organisms. However, caustic compounds of cesium can be highly toxic. As a photon emitter, cesium's radiation toxicity can be substantial. See Section 2.1 for a review of radiation toxicity in aquatic and terrestrial organisms.

5.1.1 Toxicity of Cesium in Aquatic Biota

Aquatic Plants

See Section 2.1 for effects of ionizing radiation on biota.

Invertebrates

Little information is available on cesium toxicity to aquatic invertebrates. However, the presence of cesium-137 in an industrial pond (sediment concentrations of about 28,000 pCi/g dry weight 0.29 ng/g) did not prevent the colonization of the pond by numerous invertebrate species including annelids, cladocerans, copepods, amphipods, and several species of aquatic insects and gastropods (Rickard et al. 1981). The toxicity of cesium varied greatly among the invertebrates. For example, the 48-h LC50 for the copepods, *Cyclops abyssorium* and *Eudiaptomus padanus*, was 400 mg/L and 135 mg/L, respectively. *Daphnia hyalina* was much more sensitive to cesium with a 48-h LC50 of 7.4 mg/L (Baudouin and Scoppa 1974).

See Section 2.1 for effects of ionizing radiation on biota.

Fish

Allergic effects of cesium-137 have recently been reported in fish exposed to 2000 Bq/L or more. Hyperemia and focal fatty degeneration of hepatic cells were observed in poisoned fish. Damage was also seen in brain and epithelial cells of renal tubules (Vosniakos et al. 1991). A self-sustaining, apparently healthy population of carp has been monitored for 2 decades in an industrial pond containing sediment levels of cesium-137 of about 28,000 pCi/g dry weight (Rickard et al. 1981). Fish embryos are also tolerant of cesium exposure. Exposure to up to 10 μ Ci/L of cesium-137 for 20 days did not increase the mortality rate of rainbow trout embryos (Kimura and Honda 1977a, 1977b).

5.1.2 Toxicity in Terrestrial Biota

Information regarding cesium toxicity in terrestrial biota is summarized below.

Plants

Bulrushes (*Scirpus acutua*), cattails (*Typha latifolia*), and pondweeds (*Potamogeton* sp. and *Elodea*) were not inhibited from colonizing an industrial pond containing cesium concentrations in the sediment of 28,000 pCi/g dry weight (Rickard et al. 1981).

See Section 2.1 for effects of ionizing radiation on biota.

Invertebrates

See Section 2.1 for effects of ionizing radiation on biota.

Amphibians/Reptiles

No information available.

Mammals

Cesium can replace potassium to some extent in mammals (Relman 1957) and, therefore, cesium is distributed by the blood stream throughout all the active tissues resulting in, essentially, a dose to the whole body (Boecker 1972). Acute toxicity and death are related primarily to bone marrow destruction. Shortening of life has also been observed in mammals exposed to cesium and appears to be related to delayed development of neoplasia (Norris et al. 1966). These effects are the same as those associated with gamma or x-irradiation and are summarized in Section 2.1 (Toxic Effects of Ionizing Radiation).

The toxic response of mammals to cesium resembles rubidium and potassium toxicity. Liver injury, neuroendocrine and neuromuscular disturbance leading to irritability and convulsions are clinical signs of cesium toxicity (Venugopal and Luckey 1978). The oral LD50 of cesium (without regard to its radioactive toxicity) is 84.6 mg/kg body weight as cesium hydroxide. The parenteral LD50s of cesium nitrate, cesium carbonate, and cesium halide compounds range from 716 to 1330 mg/kg (Venugopal and Luckey 1978). The greater toxicity of cesium hydroxide is probably due to its caustic action (Cochran et al. 1950). The lowest oral LD50 of non-caustic forms of cesium was 710 mg/kg in mice (Lewis and Tarken 1979-1980). Irradiation of female cotton rats in enclosed areas of a natural habitat showed that LD50/15 for cesium-137 was 1130 R and that survival time and dose were directly related. At 500 R, a 91% survival rate was observed, whereas only 25% survived 1200 R (Pelton and Provost 1969).

Recent epidemiological studies have indicated that exposure of a human fetus at 8 to 15 weeks conceptus results in mental retardation at a rate of 30 IQ points/Sv (Harley 1991). No behavioral studies are available to assess if any comparable behavioral deficits occur in other mammals exposed to cesium.

Immunological Effects of Cesium Exposure. Immune system dysfunction has been recently described in fish (Section 5.1.1 Fish).

Reproductive, Carcinogenic, and Genotoxic Effects of Cesium Exposure. See Section 2.1 on effects of ionizing radiation.

Biomarkers of Cesium Exposure/Effect. Whole body levels of cesium in humans have been estimated from blood concentrations (Salo et al. 1963). Chronic exposures to cesium in mice and dogs have shown that the major accumulator organ is muscle. Other organs (fat, blood, skin, and bone) accumulate cesium, but at much lower concentrations. In fish, accumulator organs include muscle, gills, liver, and kidneys (Vosniakos et al. 1991). However, no direct relationship between tissue levels and effects has been reported. In mule deer, about 80% to 90% of total body cesium is concentrated in muscle tissue (Hakonson 1975).

Cesium Toxicokinetics: Metabolism and Distribution. Cesium uptake from the gastrointestinal tract is rapid and nearly complete (70% to 100%) (Reichle et al. 1970a, 1970b). Generally, absorption of cesium from the digestive tract of monogastric animals is greater than or equal to 90% and about 80% in ruminants (Stara et al. 1971). In rats, about 98% of ingested cesium is absorbed within 30 minutes. Excretion of cesium is rapid and mainly urinary, although 25% of the absorbed dose can be excreted in the feces (Salo et al. 1963). Distribution of the radionuclide in the body is broad, but mostly to the soft tissues. Like other alkali metals, cesium occurs mainly as a free ion in tissues and fluids. Little binding occurs of cesium to biologically active macromolecules (Venugopal and Luckey 1978). Humans do not accumulate cesium with age, which suggests a poorly defined homeostasis for cesium (Venugopal and Luckey 1978).

The biological half-life of cesium for many wild mammal species has been assumed to be 33 days, a value determined for reindeer by Ekman (1967). However, the biological half-life of radiocesium in pocket mice has been determined to be 5.3 days (Winsor and O'Farrell 1970). The half-life of cesium-137 in mule deer is about 14 days. The biological half-life of cesium-137 in small mammals is listed in Table 5.1.

Table 5.1. Biological Half-Lives of Cesium-137 in Small Mammals Native to the Hanford Site (Rickard et al. 1974; Winsor and O'Farrell 1970)

Species	Half-Life (days)
Sagebrush Vole (<i>Lagurus curtatus</i>)	4.4
Montane Meadow Mouse (<i>Microtus montanus</i>)	4.5
Great Basin Pocket Mouse (<i>Perognathus parvus</i>)	5.3
Western Harvest Mouse (<i>Reithrodontomys megalotis</i>)	5.4
Northern Grasshopper Mouse (<i>Onychomys leucogaster</i>)	5.6
Deer Mouse (<i>Peromyscus maniculatus</i>)	6.3
House Mouse (<i>Mus musculus</i>)	9.4

Biological half-lives for mammals not listed in Table 5.1 may be estimated by the equation:

$$\text{Half-life (days)} = 3.458 (\text{body weight})^{0.2061} \quad (5.1)$$

as reported by Reichle et al. (1970a, 1970b).

Birds

Levels in birds exposed to high levels of radiocesium in the environment have been reported to be in excess of the maximum permissible concentrations for man. However, it was not determined if these levels (average body burden of 5 μCi) were harmful to the birds (Krumholz 1954). Red blood cell abnormalities in mallards that accumulated cesium-137 from an abandoned nuclear reactor cooling tower were observed after 8 months of exposure. Aneuploidy in the blood cells was observed after 9 months of exposure. Such changes only occurred with maximum body burdens of cesium-137 (George et al. 1991). Willard (1963) calculated that a chronic dose LD50 of 21,700 mGy would be needed to kill 50% of bluebird (*Sialia sialis*) nestlings over a 16-day period of irradiation with cesium-137. Growth of tree swallows was significantly affected by acute doses of 2700 to 4500 mGy (Zach and Mayoh 1984). Hatching success was reduced by chronic doses of 100 mGy/d (Zach and Mayoh 1984). Birds environmentally exposed to cesium-137 during breeding season received total dose equivalent rates to the whole body of 9.8×10^{-7} Sv/h or 2.8 mSv for the whole period of 120 days (breeding season). No reproductive or population effects were observed in even the most contaminated individuals and species (Lowe 1991). The number of eggs and chicks produced by American coot (*Fulica americana*) colonizing a cooling pond that received low levels of cesium-137 were similar to the number produced on uncontaminated ponds (Rickard et al. 1981). The coots consumed aquatic plants containing about 11,000 pCi of cesium/g dry weight and, inadvertently, sediments containing about 28,000 pCi of cesium/g dry weight (Rickard et al. 1981).

5.2 Bioconcentration Factors and Trophic Transfer Coefficients

Soil properties greatly influence cesium availability and ecosystem cycling. High clay content effectively immobilizes cesium by chemical binding, thus removing cesium from food chains. However, biological incorporation is substantial in systems containing sandy soils and sediments and low cation exchange capacity (Whicker and Shultz 1982). Biotic accumulation also appears to be dependent on potassium abundance in the environment. In general, cesium concentration factors decrease with each trophic level. However, biomagnification occurs in specific food chains.

5.2.1 Cesium Transfer in Aquatic Food Chains

Physical absorption is a major mode of uptake of cesium for algae and zooplankton (Cushing and Watson 1966). Assimilation of ingested cesium varies with the type of food consumed. For example, carp assimilate 80% of the cesium on algae but only 7% of the cesium in the organic detritus of the sediment (Kevern 1966). Assimilation from water is about 73%. The general aquatic concentration factors for cesium are listed in Table 5.2. Duckweed (*Spirodela punctata*)

Table 5.2. Water-Based Concentration Factors for Cesium in the Aquatic Food-Chain (Reichle et al. 1970a, 1970b; Dunford et al. 1985; Voshell et al. 1985; Cushing and Watson 1974; and Rickard et al. 1981)

<u>Trophic Level</u>	<u>Consumer Type</u>	<u>Concentration Factor</u>
Water		1.0
Algae		500-4,000
Higher Plants		50-25,000
Invertebrates	Saprovore (detritus-feeder)	60-11,000
	Herbivore	600
	Carnivore	800
Frog Muscle	Carnivore	8,000
Fish	Omnivore	125-6,000
	Carnivore	640-9,500
Waterfowl Flesh	Carnivore	2,000

concentrates cesium by a factor of 1000 to 5500 (Polar and Bayulgen 1991). Lower concentration factors (90 to 180) are reported for duckweed in waters containing high concentrations of potassium (Bergamini et al. 1979).

Body size appears to influence cesium-137 uptake in fish (Spigarelli and Edwards 1975). About 34% of the cesium in chironomid larvae is assimilated by bluegills weighing 0.5 to 1.2 g. Fish weighing 9 to 10 g assimilated 71% of the cesium, and 80- to 120-g bluegills absorbed 92% of the ingested cesium (Reichle et al. 1970a, 1970b). Koulikov and Ryabov (1992) described a first-order kinetic model of cesium-137 uptake and excretion in fish that interprets the weight dependence of cesium concentrations in fish flesh and liver.

Additional references on bioaccumulation of cesium in freshwater fish suggest a wider range of concentration factors from 19 to 22,000. Water-based bioconcentration factors for fish species that are locally relevant or are used extensively in aquatic toxicity studies are listed in Table 5.3. The wide range is related to differences in growth, feeding habits, and temperature and water quality parameters (Srivastava et al. 1990). A parameter that greatly affects accumulation of cesium in fish is the potassium concentration in the water. For example, the concentration factor in fish from water with a potassium concentration of 0.074 mmol/L was 15.7 ± 3.4 , whereas the concentration factor from water with potassium levels of 10 mmol/L was 2 ± 0.4 (Srivastava et al. 1990). In zebra fish, the concentration factor for cesium in whole fish is inversely dependent on the potassium content of the water: $CF = 5.2 [K^+]^{-0.44}$. In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for cesium in the flesh of freshwater fish are 400 (NRCC 1982), with 10^4 for fish in water of low mineral content and 100 for fish in water of high mineral content (CSA 1987). Myers et al. (1989) also recommended a default bioconcentration factor of 10^4 . Default values of 0.5 and 15,000 were recommended by Poston and Klopfer (1985) for nonpiscivorous and piscivorous fish, respectively, in water containing 1 mg/L potassium. At 100 mg/L potassium, the recommended values were 0.5 and 500 for nonpiscivores and piscivores, respectively (Poston and Klopfer 1985). For zebra fish, the elimination rate for cesium in freshwater is $0.014 \pm 0.003/d$, and the biological half-life is 51 ± 10 days (Srivastava et al. 1990). A bioconcentration factor of 0.4 has been reported for cesium in rainbow trout eggs (Kimura and Honda 1976b).

Results from a study in which cesium-134 and 5 other radiotracers were added to the epilimnion of a whole lake indicate that direct accumulation of cesium from water is not a major route of uptake in fish. Despite an epilimnetic half-life of 28.1 days, a continuous increase in cesium-134 activity in fish gut contents over a 247-day period was observed (Klaverkamp et al. 1983). Food appeared to be the major exposure pathway. The main source of cesium for cycling in food webs among the biotic and abiotic components of aquatic systems is the sediment. Sediment-based transfer coefficients for cesium in an aquatic food chain can be estimated from studies by Rickard et al. (1981) and Cushing and Watson (1974). These values are presented in Table 5.4.

Table 5.3. Bioconcentration Factors for Cesium in Fish Based on Cesium Levels in Water

<u>Species</u>	<u>Bioconcentration Factor</u>	<u>Reference</u>
Brown Trout (<i>Salmo trutta</i>)	5	Hewett and Jefferies 1976
Perch (<i>Perca fluviatilis</i>)	122-1,000 (eutrophic)	Kolehmainen et al. 1966
	4,867-15,022 (oligotrophic)	Kolehmainen et al. 1966
Walleye (<i>Stizostedion vitreum vitreum</i>)	640-2,500	Dunford et al. 1985
Fathead Minnow (<i>Pimephales promelas</i>)	3,170	Harrison et al. 1990
Lake Trout (<i>Salvelinus namaycush</i>)	7,040	Harrison et al. 1990
Rainbow Trout (<i>Oncorhynchus mykiss</i>)		
Fry (10 days old)	0.4	Kimura and Honda 1977a
Fingerlings (5 months old)	1.3	Kimura and Honda 1977a
Carp (<i>Cyprinus carpio</i>)	12	Horsic et al. 1982
Chub	27	Horsic et al. 1982

Voshell et al. (1985) determined the uptake of cesium at the various trophic levels of an aquatic insect community. Using this information in conjunction with the reported water and sediment concentrations of cesium, the transfer coefficients at each trophic level were calculated. The algae and plankton concentrated cesium from water with bioaccumulation factors of 5200 and 11,700, respectively. Herbivorous insects (adult *Coleoptera*) feeding on the filamentous algae had an algae-to-insect transfer coefficient of 0.009. The transfer coefficient of saprovores (mayflies and chironomid midges) was 0.03. Saprovore-to-carnivore (damselflies and dragonflies) transfer was 0.93. The transfer coefficient for predators (*Notonectidae*) that consumed only the body fluids of their prey was 0.16. (Note: Voshell et al. (1985) suggest that emergent insects that leave the water as adults could be used as a biomarker of cesium exposure.)

American coots (*Fulica americana*) utilizing a cooling pond at the Savannah River Plant accumulated 2 to 3 pCi ¹³⁷Cs/g bird/m (Brisbin et al. 1973). Migratory waterfowl using the pond removed about 3.75 x 10⁻⁵ Ci of radiocesium from the pond each year and redistributed it along their migratory route (Brisbin et al. 1973). Cesium-137 was found to be the major radionuclide in birds using

Table 5.4. Sediment-Based Bioconcentration Factors for Cesium in a Biotic Community (Rickard et al. 1981 and Cushing et al. 1974)

<u>Species</u>	<u>Bioconcentration Factor</u>
Periphyton (<i>Chadophora</i> sp.)	0.88-1.83
Pond weeds (<i>Potamogeton</i> sp.)	0.09-0.10
(<i>Elodea</i>)	0.04-0.07
Mollusks	0.07
Insects (<i>Coleoptera</i> sp.)	0.009
Carp	0.01-0.011
Coots (<i>Fulica americana</i>)	0.01-0.03
Ducks (Fish-Eating)	0.004

the Hanford 200-Area waste swamps in 1969. The range of cesium found in the muscle of the bird was 70 to 420 pCi/g (300 pCi/g average) (Wilson and Essig 1970). Concentration ratios [cesium concentration in the bird (g)/ cesium concentration in water (mL)] for aquatic birds have been derived experimentally (Pendleton and Hanson 1958) and range between 800 and 900 as measured in bone tissue, 1800 to 2200 for muscle tissue, and 2200 to 2800 for liver (Table 5.5)

From the above data, it is apparent that no biomagnification of cesium occurs in aquatic food webs and that cesium concentrations tend to decrease sequentially with successive trophic levels (Cushing et al. 1974; Nelson et al. 1967; Rickard et al. 1981).

5.2.2 Cesium Transfer through Terrestrial Food Chains

Cesium progressively decreases in concentration through invertebrate food chains, generally averaging one-half the concentration of plants after two trophic exchanges (Reichle and Crossley 1969). However, cesium concentrations increase at the higher trophic levels in mammals. A ninefold increase of cesium-137 has been reported in plant-mule deer-cougar food chains (Pendleton et al. 1964). In the lichen-caribou-wolf chain, cesium-137 increased twofold at each successive link in the food chain (Hanson et al. 1967). The general terrestrial food-chain concentration factors for cesium are listed in Table 5.6. Some species-specific concentration factors are listed in Table 5.7.

Table 5.5. Cesium Concentration Factors in Birds (Mellinger and Schultz 1975)

<u>Species</u>	<u>Tissue</u>	<u>Concentration Factor (bird (g)/water (mL))</u>
Coot (<i>Fulica americana</i>)	Muscle	1800
	Liver	2200
	Bone	800
Mallard (<i>Anas platyrhynchos</i>)	Muscle	2000
	Liver	2500
	Bone	700
Ruddy Duck (<i>Oxyural jamaicensis rubida</i>)	Muscle	2200
	Liver	2800
	Bone	900

Table 5.6. Concentration Factors for Cesium in the Terrestrial Food-Chain (Reichle et al. 1970a, 1970b)

<u>Trophic Level</u>	<u>Consumer Type</u>	<u>Concentration Factor</u>
Plants		1.0
Invertebrates	Saprovore ^(a)	0.2
	Herbivore	0.3-0.5
	Carnivore	0.1-0.5
Mammals	Herbivore	0.3-2.0
	Omnivore	1.2-2.0
	Carnivore	3.8-7.0

(a) Detritus-Feeder

Table 5.7. Species-Specific Concentration Factors for Cesium in Terrestrial Ecosystems (Lowe and Horril 1991; Pendleton et al. 1964)

<u>Trophic Level</u>	<u>Consumer</u>	<u>Concentration Factor</u>
Herbivore	Red grouse	1.7
	Black grouse	1.4
	Blue hare	2.0 (male), 2.5 (female)
	Brown hare	3.3
	Rabbit	1.9 (males), 1.5 (females)
	Red deer	1.3 (hinds), 3.8 (stags)
Carnivore	Cougar	3.4
	Wolf	2.0
	Fox	11.2 (males), 7.2 (females)

As seen in Table 5.7, sex and breeding conditions influence the accumulation ratio of cesium-137 in both herbivores and carnivores. It should be noted that the concentration ratios for the carnivores are based on a single, albeit dominant, prey species instead of the variety upon which they normally feed and, therefore, they may be biased.

Cesium uptake from soil by a single crop is less than 0.1% of the soil's content (Menzel 1963). Prairie grasses concentrate cesium by factors of 0.02 to 5.0, depending on soil conditions and grass species (Schuller et al. 1993). On the basis of Menzel's classification of concentration factors of elements in plants, cesium is considered "slightly excluded" (Menzel 1963). Concentration factors for emergent seed plants range from 50 to 600. On the other hand, Voight et al. (1991) reported root transfer factors for cesium-137 of 0.002 for grains, 0.002 for potatoes, 0.0047 for lettuce, and 0.003 for bush beans. Garland et al. (1983) found concentration factors of 3×10^{-4} for tumble mustard and 0.5 for cottonwood and willow leaves.

Assimilation of cesium-137 from detritus is low (53% to 65%) because it is incorporated into poorly digested tissue structures (Reichle et al. 1970a, 1970b). Cesium in herbaceous foliage is more readily available (73% to 94%), especially in sap-sucking animals such as aphids (about 100%). Flesh-eating predators also show high assimilation efficiencies for cesium (i.e., 79% to 94%). Transfer of cesium from forage to milk in ruminants is about 0.25% (Voors and VanWeers 1991). Fielitz (1991) reports a feed-to-meat transfer of 0.045 in fallow deer.

6.0 Strontium

6.1 Strontium Toxicity

Strontium has a relatively low chemical toxicity in those aquatic and terrestrial biota that have been tested. However, because of its similarity to calcium, strontium is deposited in bone of vertebrate animals, where irradiation by beta particles can cause neoplasia and adversely affect blood cell formation.

See Section 2.1 for a review of the radiation toxicity of strontium.

6.1.1 Toxicity of Strontium in Aquatic Biota

See Section 2.1 for a review of the radiation toxicity of strontium.

Strontium toxicity to copepods is low. The 48-h LC50 of strontium in the copepods (*Cyclops abyssorum* and *Eudiaptomus padanus*) is 300 mg/L and 180 mg/L respectively. Cladoceran sensitivity to strontium is also moderate (75 mg/L, 48-h LC50) (Baudouin and Scoppa 1974).

6.1.2 Toxicity of Strontium in Terrestrial Biota

Plants

See Section 2.1 for a review of the radiation toxicity of strontium.

Invertebrates

See Section 2.1 for a review of the radiation toxicity of strontium.

Amphibians/Reptiles

See Section 2.1 for a review of the radiation toxicity of strontium.

Mammals

The strontium ion has a low order of toxicity (Venugopal and Luckey 1978). Moderate to large doses are required to cause nausea, diarrhea, electrocardiographic changes, and death due to respiratory paralysis (Venugopal and Luckey 1978). Oxides and hydroxides of strontium are moderately caustic compounds. The oral LOAEL of strontium dichloride hexahydrate in rats is 405 mg/kg. Oral ingestion of strontium fluoride results in an oral LD50 in rats of 10,600 mg/kg. However, the

radioisotope strontium-90 is highly dangerous, and the radiation hazard of strontium-90 is well established (see Section 2.1 for a summary of ionizing radiation effects). Because strontium is a metabolic analog of calcium, strontium-90 is readily absorbed from the lung, gastrointestinal tract, or bloodstream (dermal exposure). The strontium that is retained in the body, in large part, is deposited in the bone. Therefore, exposure to strontium-90 via any exposure route results in a high incidence of neoplasia on bone and related tissues (Harley 1991). Chronic intake of strontium-90 in dogs produced a high incidence of tumors also, but tumor production was low in miniature swine receiving similar doses. Tumorigenicity has also been observed in wild rodents. A muskrat from White Oak Lake that had more than 1 μC of strontium per gram of bone, a total body burden of nearly 100 μCi (Krumholz and Rust 1954), displayed advanced osteogenic sarcoma with metastasized cells to both kidneys and lungs. Bone sarcoma generation does not fit a linear dose-response relationship over a wide dose range. Low levels of exposure are better fit by sigmoid dose-response relationships (Mays and Lloyd 1972).

Exposure of humans to strontium salts has also been shown to cause a reduction in the activity of the neurotransmitter, acetylcholine, and the enzyme cholinesterase. This reduction in activity has not been measured in other mammals. Impaired tooth development in growing animals has been documented at exposures of about 0.4 mg strontium/kg (Lewis 1992).

Immunological Effects of Strontium Exposure. See Section 2.1 for a review of the radiation toxicity of strontium.

Reproductive Effects of Strontium Exposure. In addition to the radiation effects of radiostrontium on reproduction, strontium can have a chemical toxicity related to the reproductive system of mammals. Strontium salts have been shown to cause changes in the prostate, seminal vesicle, Cowper's gland, and accessory glands of rats. The LOAEL was 400 mg/kg as strontium chloride hexahydrate (NIOSH 1987).

Carcinogenic and Genotoxic Effects of Strontium Exposure. See Section 2.1 for information on the radiation hazard of strontium in mammals.

Biomarkers of Strontium Exposure/Effect. The strontium/calcium ratio is relatively constant and can be used to estimate dietary intakes and body burdens of strontium. Dietary intake can be estimated from the strontium/calcium ratio in urine (Comar et al. 1964). The observed ratio urine/diet can be somewhat variable, but generally it is accepted to be 0.84 (Comar 1965). The strontium/calcium ratio may be a better parameter for estimating uptake because it is less variable than that for urine; however, the urine value reflects the strontium actually absorbed from the diet of the animal. Teeth and hair have also been used to estimate body burden (Rosenthal et al. 1963). Because a large amount of strontium is transferred from the diet of a male deer to the developing antlers, the antlers have been considered for use as indicators of strontium-90 in forage consumed by deer (Rickard et al. 1974).

Strontium Toxicokinetics: Metabolism and Distribution. After radiostrontium is ingested, about 18% (it ranges from 5% to 25%) is absorbed into the body from the diet into the gastrointestinal tract; the rest is excreted unabsorbed in the feces. Excretion of the absorbed strontium in feces is 16%, with up to 96% excreted in the urine. The absorbed strontium is deposited in bone; distributed in an exchangeable pool comprised of soft tissues, bone surface, plasma, etc.; or excreted in feces (up to 16%) and urine (up to 96%) (Comar et al. 1964; Dolphin and Eve 1963). Accumulator organs include the bone, aorta, trachea, and lower gastrointestinal tract (Shacklette et al. 1978). The biological half-life of strontium-90 in mammals can be estimated using the equation:

$$\text{Half-Life} = 107.4 (\text{Body Weight})^{0.2612} \quad (6.1)$$

as reported by Reichle et al. (1970).

Birds

Radiostrontium levels of up to 1700 and 560 pCi/kg ash of the eggshells and inner egg contents, respectively, have been found in Canada goose eggs on the Hanford Site (Rickard and Sweany 1977). No impacts on clutch size, hatching success, viability of the young, or population parameters have been associated with these levels of contamination when compared to uncontaminated goose populations.

6.2 Bioconcentration Factors and Trophic Transfer Coefficients

As an alkaline earth metal, strontium is chemically reactive, commonly forming soluble salts of carbonates, sulfates, and chlorides. It thus is mobile in ecosystems, readily enters food chains, and deposits in calcium-containing tissues. However, tissue concentrations of strontium do not appear to increase with trophic level. This is probably related to metabolic control of strontium uptake by calcium.

6.2.1 Strontium Transfer in Aquatic Food Chains

The uptake of strontium-90 from sediment or soil to plants and from plants to animals is affected by the presence of calcium in the systems. The "observed ratio" described by Comar et al. (1956) relates the amount of strontium-90 and calcium in a sample to the amount of the radionuclide and competing element in the precursor. This empirically determined relationship has proven to be consistent and has been successfully applied to modeling the passage of strontium-90 through food webs

(Comar and Wasserman 1960; Comar 1965). Most of the reported observed ratios have been determined for food chains leading to human consumers. Those ratios potentially applicable to wildlife exposure are listed in Table 6.1.

The concentration of strontium in the bone and muscle of brown trout was inversely related to calcium concentration of the water (Templeton and Brown 1964). The observed ratio for muscle/water varied from 0.53 to 1.00 as the calcium level in the water varied from 0.3 to 100 mg/L (Templeton and Brown 1963). In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for strontium in the flesh of freshwater fish are 5 (NRCC 1983), 800 for fish in water of low mineral content, and 2 for fish in water of high mineral content (CSA 1987). Myers et al. (1989) recommended default values of 180 for water containing 1 mg/L calcium and 0.7 for water containing 100 mg/L calcium. Poston and Klopfer (1986) recommended a default value of 100. The general aquatic concentration factors for strontium are listed in Table 6.2.

Higher concentration ratios than those compiled by Reichle et al. (1970a, 1970b) have been observed in black crappies (*Pomoxis nigro-maculatus*) and bluegills (*Lepomis m. macrochirus*). These species concentrated radiostrontium in amounts 20,000 to 30,000 times that found in the water (Buchsbaum 1958). In contrast, Carraca et al. (1990) found no significant biomagnification of strontium concentrations between predator and prey. In fish, the concentration of strontium was proportional to the size of the body, probably because most of the strontium was found in scales and bones. Concentration factors for strontium in the trophic chains of several streams studied by Carraca et al. (1990) are listed in Table 6.3.

Table 6.1. Observed Ratios Reported for Strontium/Calcium Transport in Food Webs (CRC 1982)

<u>System</u>	<u>Observed Ratio</u>	<u>Comments</u>
Plant tissue/soil	0.9-1.0	Dependent on plant part
Fish muscle/water	0.5-1.0	Dependent on [Ca ²⁺] in water
Fish bone/water	0.5	
Poultry bone/diet	0.6	
Egg yolk/diet	0.6	
Egg white/diet	1.5	
Mammalian bone/diet	0.14-0.57	Various species

Table 6.2. Concentration Factors for Strontium in the Aquatic Food Chain (Reichle et al. 1970a, 1970b; Horsic et al. 1982)

<u>Trophic Level</u>	<u>Consumer Type</u>	<u>Concentration Factor</u>
Algae and higher plants		10-3000
Invertebrates	Saprovore (detritus-feeder)	10-4000
	Herbivore	1
	Camivore	
Fish	Omnivore	1
	Camivore	1-150

6.2.2 Strontium Transfer through Terrestrial Food Chains

Absorption of strontium from soil is influenced by the clay content, organic content, pH, moisture level, concentration of electrolytes and, in particular, the calcium content of the soil. In general, conditions that cause shallow root development tend to increase strontium-90 uptake (Comar 1965). Strontium uptake is greatest from soils of low calcium content. Plant crops assimilate from 0.2% to 3% of the strontium in the soil (Comar 1965; Menzel 1963). As a general rule, if 1 mCi of strontium-90/km² is present in the soil, plants will assimilate about 1.1 pCi of strontium-90/g of calcium (Evans and Dekker 1962).

Strontium is greatly reduced, relative to plant levels, in whole-body concentration in insects and other invertebrates. A concentration factor of about 0.1 has been observed for second-order consumers and predators (excluding species with calcified exoskeletons) (Reichle et al. 1970a, 1970b). "Calcium sink" invertebrates (e.g., millipedes, isopods, snails) concentrate strontium by factors greater than 150 (Reichle et al. 1969). Assimilation of strontium-90 from detritus is low (77%) because it is incorporated into poorly digestible tissue structures (Reichle et al. 1970a, 1970b). The general terrestrial concentration factors for strontium are listed in Table 6.4.

An experimentally determined concentration factor [strontium concentration in the bird (g)/strontium concentration in water (ml)] of 1500 (bone) for coot (*Fulica* sp.) was reported by Hanson and Kronberg (1956). Feeding habits influence the uptake of strontium into aquatic bird tissues. The highest concentrations of strontium were found in shorebirds feeding on insects and larvae. The dabbling ducks had intermediate levels in their bone tissue, and fish-eating birds had the lowest strontium burden of feeding habits of aquatic birds (Silker 1958). The transfer coefficient for strontium from the diet into milk of cows, goats, and pigs as related to the calcium content of the diet

Table 6.3. Strontium Bioconcentration and Transfer Factors for Stream-Dwelling Organisms (Carraca et al. 1990)

<u>Trophic Level</u>	<u>Concentration Factor (dry wt)^(a)</u>	<u>Transfer Factor</u>
Water to benthic animals	26 - 53	
Benthic animals to fish		
Boce		7-11 ^(c)
Carp		21
Barbel		7-10
Goldfish		12
Water to fish ^(b)		
Boce	293 - 390	
Carp	541	
Barbel	248 - 365	
Goldfish	631	
Water to seston	227 - 345	
Seston to fish		
Boce		0.9 1.7
Carp		1.6
Goldfish		2.8
Barbel		0.7-1.6
Water to zooplankton	259-278	
Seston to zooplankton	0.8-1.2	
Zooplankton to fish		
Boce		1.1-1.4
Carp		2.1
Goldfish		2.3
Barbel		1.0-1.3

(a) Calculated concentration factors on dry wt. basis. To convert to wet weight, the dry wt/wet weight ratio is: benthic animals = 0.150, phytoplankton (seston) = 0.138; zooplankton = 0.100; boce = 0.257; barbel = 0.26; carp = 0.231, goldfish = 0.281.

(b) *Chondrostoma polypis* (boce); *Cyprinus carpio* (carp); *Carassius auratus* (goldfish); *Barbus bocagei* (barbel).

(c) transfer factor close to 1 when only edible tissue is considered.

Table 6.4. Concentration Factors for Strontium in the Terrestrial Food Chain (Reichle et al. 1970a, 1970b)

<u>Trophic Level</u>	<u>Consumer Type</u>	<u>Concentration Factor</u>
Plants		1.0
Invertebrates	Saprovore ^(a)	<0.1
	Herbivore	0.1
	Carnivore	0.1
Mammals	Herbivore	0.5-4.5
	Omnivore	
	Carnivore	

(a) Detritus-Feeder.

is 0.1 (range 0.08-0.16) (Comar 1965). The observed ratio value for body/diet of 0.2-0.5 has been reported for cattle, goats, sheep, and pigs. Observed ratio values for egg yolk, eggshell, and femur in chickens was 0.6 (Comar 1965).

7.0 Cobalt-60

7.1 Cobalt-60 Toxicity

The hazard of cobalt-60 exposure in aquatic systems is largely related to the radiation toxicity of the radionuclide. Section 2.1 reviews the impacts of ionizing radiation on biota. Available information on the chemical toxicity of cobalt to various trophic levels is summarized below.

7.1.1 Cobalt-60 Toxicity in Aquatic Biota

Divalent cobalt is highly toxic to zooplanktonic species. Acute toxicity (48-h LC50) of cobalt (II) to 2 copepods was 15.5 mg/L for *Cyclops abyssorum* and 4.0 mg/L for *Eudiaptomus padamus*. The 48-h LC50 for *Daphnia hyalina* was 1.32 mg/L (Baudouin and Scoppa 1974).

See Section 2.1 for a review of the radiation toxicity of cobalt-60.

7.1.2 Cobalt-60 Toxicity in Terrestrial Biota

The toxicity of cobalt in terrestrial biota is given below.

Plants

The threshold toxicity for lettuce seedlings grown in a hydroponic solution was 13 $\mu\text{eq/L}$ (0.38 mg/L). Growth was inhibited by 50% at 62 $\mu\text{eq/L}$ (1.83 mg/L), and cobalt was lethal to the seedlings at 1,500 $\mu\text{eq/L}$ (44.25 mg/L) (Berry 1978). Morphological abnormalities of floral parts occurred at radiation levels of about 2 R/d from cobalt-60 and included multiplication and reduction or malformation of floral parts (Platt 1965).

Invertebrates

See Section 2.1 for a review of the radiation toxicity of cobalt-60.

Amphibians/Reptiles

See Section 2.1 for a review of the radiation toxicity of cobalt-60.

Mammals

Cobalt is an essential element and is found in vitamin B12 (0.0434 μg of cobalt/ μg vitamin B12). Ingestion of excessive amounts of cobalt results in polycythemia (excess formation of red blood cells) in most mammals. This response, in part, is caused by the creation of intracellular hypoxia and

results in an overexertion of the heart and elevated blood pressure (Waldbott 1973). The cardiotoxic effects of cobalt are also related to its antagonistic action toward Ca^{+2} and the complex-forming ability of cobalt with cellular macromolecules (Goyer 1986). Oral uptake of 26 mg/kg for 8 weeks following an initial dose of 100 mg/kg resulted in cardiomyopathy in rats. Vomiting, diarrhea, and a sensation of warmth are sublethal responses to cobalt ingestion (Beliles et al. 1978) and are induced in mammals by chronic exposure to 150 ppm cobalt in the diet (Waldbott 1973). The LOAEL for cobalt nitrate was 250 mg/kg in rabbits; the LD50 in rats was reported to be 434 mg/kg. Oral toxicity of cobalt chloride was 80 mg/kg in rats and 55 mg/kg in guinea pigs. The LOAEL for cobalt chloride in rabbits was 1272 mg/kg (NIOSH 1987).

Inhalation of powdered cobalt produces chronic lung changes that evolve into pulmonary fibrosis (Waldbott 1973). Miniswine exposed to 0.1 mg/m³ cobalt metal dust by inhalation for 3 months showed marked decrease in lung compliance and an increase in collagen in the pulmonary alveolar septa (Kerfoot et al. 1975).

Irradiation of wild rodents with cobalt-60 resulted in LD50/30 values ranging from 525 to 1069 rad. These values are elevated over those for laboratory rodents that have LD50/30 values of 330 to 900 rad (Dunaway et al. 1969). Weights of irradiated rodents receiving greater than or equal to 1000 rad decreased. The radiation toxicity threshold (i.e., 95% survival rate) was an exposure above 450 rad for all species of wild rodents (O'Farrell 1969).

Immunological Effects of Cobalt Exposure. Cobalt can cause an allergic dermatitis in animals (Schwartz 1947). Sensitization of the respiratory tract also occurs in mammals (Kerfoot et al. 1975).

Reproductive Effects of Cobalt Exposure. Pregnant mice exposed to cobalt chloride at a LOAEL of 25 mg/kg produced young with craniofacial abnormalities. In rats, an LOAEL exposure of 30 g/kg resulted in post-implantation mortality and fetotoxicity (NIOSH 1987).

Carcinogenic Effects of Cobalt Exposure. In addition to radiation-induced carcinogenicity, there is a chemical carcinogenicity associated with cobalt as well. Depending on the route of exposure and the form of cobalt, the tumorigenic exposures observed in rats ranged from 75 mg/kg to 4530 mg/kg (NIOSH 1987).

Genotoxicity. See Section 2.1 for a review of the radiation toxicity of cobalt-60.

Biomarkers of Cobalt Exposure/Effect. Fat tissue contains the highest concentration of cobalt, and heart, liver, and hair/fur also concentrate cobalt, but to a much smaller degree (Beliles 1978). However, no data are available on the relationship between cobalt concentrations in these tissues and the exposure of the organism or any toxic effects. Blood and urine levels may be used to estimate "above normal levels."

Cobalt Toxicokinetics: Metabolism and Distribution. Cobalt salts are well absorbed after oral ingestion. However, increased uptake above 0.004 mg/kg does not result in accumulation in humans (Beliles 1978). Significant species differences in excretion rates have been observed in mammals. In man and dogs, about 80% of the absorbed cobalt is excreted in the urine, and 15% of the remaining cobalt is excreted by an entero-hepatic pathway into the feces. In contrast, 80% of the absorbed cobalt is eliminated in the feces of rats and cattle (Beliles 1978). About 10% of the cobalt radioactivity is found in the urine of rats, but less than 0.5% is found in the urine of cattle, indicating a greater initial retention by tissues in ruminants.

Birds

Radiocobalt levels of up to 5 to 8 pCi/kg ash and 28 to 39 pCi/kg ash of the eggshells and inner egg contents, respectively, have been found in Canada goose eggs on the Hanford Site (Rickard and Sweany 1977). No impacts on clutch size, hatching success, viability of the young, or population parameters have been associated with these levels of contamination when compared to uncontaminated goose populations.

7.2 Bioconcentration Factors and Trophic Transfer Coefficients

Cobalt is readily accumulated from the environment by aquatic and terrestrial biota. However, trophic transfer is low and no biomagnification of the radionuclide has been reported for either aquatic or terrestrial food chains.

7.2.1 Cobalt-60 Transfer in Aquatic Food Chains

The general aquatic concentration factors for cobalt are listed in Table 7.1. Cobalt is slightly more concentrated in invertebrates than in water but is markedly less concentrated in invertebrates than in algae and higher plants (Table 7.1).

Voshell et al. (1985) determined the uptake of cobalt at the various trophic levels of an aquatic insect community. Using this information in conjunction with the reported water and sediment concentrations of cobalt, the transfer coefficients at each trophic level were calculated. The algae and plankton concentrated cobalt from water and had transfer coefficients of 11,800 and 20,600, respectively. Herbivorous insects (coleopteran adults) feeding on the filamentous algae had an algae-to-insect transfer coefficient of 0.1. The transfer coefficient of saprovores (mayflies and chironomid midges) was 0.04. Saprovore-to-carnivore (damselflies and dragonflies) transfer was 0.01. The transfer coefficient for predators (*Notonectidae*) that consumed only the body fluids of their prey was 0.13. (Note: Voshell et al. (1985) suggest that emergent insects that leave the water as adults could be used as a biomarker of cobalt exposure.) The maximum transfer factors reported for fish that fed on cobalt-contaminated crustaceans (*Gammarus pulex*), midge larvae (*Chironomus* sp.), and the soft tissue of snails (*Lymnaea stagnalis*) ranged from 0.012 to 0.051 (Baudin et al. 1990). Midge

Table 7.1. Concentration Factors for Cobalt in the Aquatic Food Chain

<u>Trophic Level</u>	<u>Concentration Factor</u>	<u>Reference</u>
Algae and Higher Plants	2500-6200	Reichle et al. 1970a, 1970b
Invertebrates		
Saprovore(*)	325	Reichle et al. 1970a, 1970b
Fish		
Rainbow Trout (<i>Oncorhynchus mykiss</i>)		
eggs	7.0	Kimuru and Honda 1977b
fry	11.0	Kimuru and Honda 1977b
fingerlings	6.5	Kimuru and Honda 1977b
Smelt (<i>Osmerus mordax</i>)	48 -1000	Vanderploeg et al. 1975
Spottail Shiner (<i>Notropis hudsonius</i>)	220-630	Vanderploeg et al. 1975
Alewife (<i>Alosa pseudoharengus</i>)	190-420	Vanderploeg et al. 1975
Trout-Perch (<i>Percopsis omiscomaycus</i>)	130	Vanderploeg et al. 1975
Fathead Minnow (<i>Pimephales promelas</i>)	190	Harrison et al. 1990
Lake Trout (<i>Salvelinus namaycush</i>)	11	Harrison et al. 1990

(a) Detritus-Feeder.

larvae were reported to concentrate cobalt-60 from water by a factor of 30. Transfer of cobalt to larvae from sediment was low (0.62), but the sediment constituted a permanent exposure source to the larvae compared to the very transitory concentrations of cobalt in water. Virtually the entire amount of the cobalt-60 in an aquatic system was fixed rapidly to the sediment (Baudin and Nucho 1992; Lambrechts and Foulquire 1986). The sediment-to-plant transfer coefficient was 0.29 in a stream contaminated with cobalt (Stewart et al. 1992). The organic content of the sediment markedly influenced the uptake of cobalt-60 by midge larvae. An increase in the organic content of the sediment can lead to a two-fold increase in cobalt uptake (Baudin and Nucho 1992). The midge larvae daily ingested an amount of sediment equivalent to 9% of their dry weight (Gerking et al. 1976). The trophic transfer of cobalt-60 from planktonic algae to midge larvae was low, 0.0045, and did not lead to bioamplification of the radionuclide (Baudin and Nucho 1992).

In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for cobalt in the flesh of freshwater fish are 20 (NRCC 1983). A default bioconcentration factor of 1000 is recommended for fish in water of low mineral content and a default value of 100 is recommended for fish in water of high mineral content (CSA 1987). Myers et al. (1989) recommend a default value of 30 for eutrophic conditions.

7.2.2 Cobalt Transfer Through Terrestrial Food Chains

Cobalt progressively decreases in concentration through invertebrate food chains, generally averaging one-half the concentration of plants after two trophic exchanges (Reichle and Crossley 1969). The general terrestrial concentration factors for cobalt are listed in Table 7.2.

Table 7.2. Concentration Factors for Cobalt in the Terrestrial Food Chain (Reichle et al. 1970a, 1970b)

<u>Trophic Level</u>	<u>Consumer Type</u>	<u>Concentration Factor</u>
Plants		1.0
Invertebrates	Saprovore ^(a)	
	Herbivore	0.4
	Carnivore	0.5
Mammals	Herbivore	0.3

(a) Detritus-Feeder.

8.0 Chromium

8.1 Chromium Toxicity

Little is known about the relationship between concentrations of chromium in a given ecosystem and the biological effects on the component organisms. The same elemental concentration of chromium has a wide variety of mobilities and reactivities depending on the physical and chemical state of the ion. Therefore, the observed effects of chromium exposure vary widely. In addition, species' sensitivity to chromium differs greatly, even among closely related species (Steven et al. 1976). The toxicity of chromium ions is highly dependent on oxidation state. Only the trivalent and hexavalent chromiums are biologically significant. Trivalent chromium is the only form of chromium found in biological material. Trivalent chromium does not readily cross cell membranes, and it forms stable complexes with serum proteins. As a result, it has a low overall toxicity potential and is relatively inactive *in vivo*. In contrast, hexavalent chromium is readily taken up by living cells and is highly active in diverse biological systems. Although the known harmful effects of chromium in animals are attributed to exposure to the hexavalent form, it is the trivalent form that is ultimately damaging as it is formed from the reduction of hexavalent chromium and complexes with intracellular macromolecules.

8.1.1 Toxicity of Chromium in Aquatic Biota

The toxicity of chromium in aquatic biota is presented in the following sections.

Aquatic Plants

Hexavalent chromium is toxic to algae at concentrations of less than 10 mg/L (Shacklette et al. 1978). Growth of most species tested was reduced at concentrations of 10 to 45 ppb hexavalent chromium. Effects were most pronounced in water of a low alkalinity (Eisler 1985). Common duckweed (*Lemna minor*) is the most sensitive aquatic plant tested exhibiting reduced growth in water containing 10 ppb hexavalent chromium (Mangi et al. 1978). The LC50s for aquatic macrophytes range between 2.5 and 25 mg/L (Mangi et al. 1978).

Invertebrates

LC50 values for rotifers and crustaceans range between 0.4 and 67 mg/L. Hexavalent chromium was toxic to snails at 17 to 41 mg/L (Buikema et al. 1974; EPA 1980; Murti et al. 1973; Jouany et al. 1982). Reduced fecundity was observed in *Daphnia magna* at 10 ppb hexavalent chromium and 44 ppb trivalent chromium after 32 days of exposure (EPA 1980). Exposure to 1.8 mmol chromium solution resulted in a 100% death rate of the freshwater trematode, *Schistosoma haematobium*, in 1 h.

Sporocyst formation was also inhibited, and the number of miracidia penetrating snails (intermediate host) was reduced by 50% (Wolmarans et al. 1988). The acute toxicity values for aquatic invertebrates are listed in Table 8.1.

Fish

In general, adverse effects of chromium to sensitive fish species have been documented at 10 $\mu\text{g/L}$ (ppb) of hexavalent chromium and 30 $\mu\text{g/L}$ of trivalent chromium in freshwater (Eisler 1985). Growth rates of rainbow trout and chinook salmon fingerlings were reduced in fish exposed to 16 to 21 ppb hexavalent chromium for 14 to 16 weeks (EPA 1980). The half-life of chromium in rainbow trout was 1 day for the short-term component (34% of total chromium) and 25.6 days for the long-term component (Van der Putte et al. 1981). At a concentration of 0.23 ppm hexavalent chromium for 4 weeks, salinity tolerance and serum osmolality were impaired in migrating coho salmon (Sugatt 1980). The survival rate of alevins and juveniles of coho salmon was significantly reduced by exposure to 0.2 mg/L chromium (Oson 1958). Reproductive impairment in fathead minnows was observed after 10 months of exposure to 2.0 mg/L chromium (Pickering and Henderson 1966). The NOAEL and LOAEL for several species of freshwater fish are listed in Table 8.2.

Table 8.1. Acute Toxicity of Hexavalent and Trivalent Chromium in Aquatic Invertebrates

<u>Species</u>	<u>Water Hardness (mg CaCO₃/L)</u>	<u>LC50 (mg/L)</u>	<u>Test Duration</u>	<u>Reference</u>
Hexavalent				
Rotifers				
<i>Philodena acuticornis</i>	25	3.1	96 h	Buikema et al. 1974
	81	15	96 h	Buikema et al. 1974
Mollusks				
<i>Physa heterostropha</i> (Snail)	45	17.3	96 h	EPA 1980
	171	31.6-40.6	96 h	EPA 1980
Crustaceans				
<i>Gammarus pseudolimnaeus</i> (Amphipod)		67	96 h	EPA 1980
<i>Machrobrahium lamarrei</i> (Prawn)		1.8	96 h	Murti et al. 1983
<i>Daphnia magna</i> (Cladoceran)		0.4	24 h	Juoany et al. 1982
<i>Daphnia hyalina</i> (Cladoceran)	65	0.002	48 h	Baudouin and Scoppa 1974
<i>Cyclops abyssorum</i> (Copepod)	65	10.0	48 h	Baudouin and Scoppa 1974
<i>Eudaptomus padanus</i>	65	10.1	48 h	Baudouin and Scoppa 1974
Insects				
<i>Acroneuria lycurias</i> (Stonefly)		32	7 d	Warnick and Bell 1961
<i>Hydropsyche betteri</i> (Caddisfly)		32	7 d	Warnick and Bell 1961
<i>Ephemerella subvaria</i> (Mayfly)		16	7 d	Warnick and Bell 1961

Table 8.1. (contd)

<u>Species</u>	<u>Water Hardness (mg CaCO₃/L)</u>	<u>LC50 (mg/L)</u>	<u>Test Duration</u>	<u>Reference</u>
Trivalent				
Mollusks				
<i>Ampicula</i> sp. (Snail)		8.4	96 h	EPA 1980
Annelids				
<i>Nais</i> sp.		9.3	96 h	EPA 1980
Arthropods				
<i>Daphnia magna</i>	48	2.0	96 h	EPA 1980
	52	16.8	96 h	EPA 1980
	99	27.4	96 h	EPA 1980
	195	51.4	96 h	EPA 1980
<i>Gammarus</i> sp.		3.2	96 h	EPA 1980
Insects (4 spp.)		2.0-64.0	96 h	EPA 1980

Table 8.2. NOAELs and LOAELs Reported for Hexavalent and Trivalent Chromium in Freshwater Fish

<u>Species</u>	<u>NOAEL (µg/L)</u>	<u>LOAEL (µg/L)</u>	<u>Reference</u>
Hexavalent			
Rainbow Trout (<i>Oncorhynchus mykiss</i>)	51-200	105-350	Sauter et al. 1976
Brook Trout (<i>Salvelinus fontinalis</i>)	200	350	EPA 1980
Fathead Minnow (<i>Pimephales promelas</i>)	1000	3950	Pickering 1980
Channel Catfish (<i>Ictalurus punctatus</i>)	150	305	Sauter et al. 1976
Bluegill (<i>Lepomis macrochirus</i>)	522	1122	Sauter et al. 1976
White Sucker (<i>Catostomus commersoni</i>)	290	538	Sauter et al. 1976
Northern Pike (<i>Esox lucius</i>)	538	963	Sauter et al. 1976
Walleye (<i>Stizostedion vitreum</i>)	>2161		Sauter et al. 1976
Trivalent			
Rainbow Trout (<i>Oncorhynchus mykiss</i>)	30	157	EPA 1980
Fathead Minnow (<i>Pimephales promelas</i>)	750	1400	EPA 1980

Acute lethality (LC50) of chromium to fish varies between 17 and 171 mg/L for most fish species (Table 8.3). Stickelback (*Gasterosteus aculeatus*) appear to be more sensitive than other freshwater fish to chromium toxicity. Concentrations of 1.0 mg/L or more are lethal to this species (Anderson 1944; Murdock 1953; Jones 1939). Reported LC50 values for hexavalent and trivalent chromium are listed in Table 8.3.

8.1.2 Toxicity of Chromium to Terrestrial Biota

The toxicity of chromium to terrestrial biota is described below.

Plants

Chromium is beneficial, but not essential, to growth in higher plants. Residues in plants seldom exceed a few ppm. Growth inhibition noted in certain plant species on "serpentine soils" has been attributed to high chromium levels in the soils (Brooks 1972). Plants grown in these soils typically show symptoms of toxicity when concentrations of chromium in the leaves reach 4 to 8 ppm (dry weight) in corn leaves, 252 ppm in oat leaves, and 18 to 24 ppm in tobacco leaves or 375 to 400 ppm in tobacco roots (NAS 1974). Soil infertility has been associated with 1000 to 3900 ppm chromic acid in Maryland (Vokal et al. 1975) and with 2% to 3% chromic oxide in "poison spots" in Oregon (McMurtry and Robinson 1938). In native foliage, concentrations as high as 1390 ppm dry weight did not show adverse biological effects (Eisler 1985). Chromium concentrations in excess of 1 ppm in aqueous solution may inhibit germination of the seed and growth of roots and shoots (Towhill et al. 1978). Chromium salts, particularly hexavalent forms, are toxic to plants in very low concentrations. Bowen (1979) reports plant toxicity levels of 0.5 to 10 ppm. Severe plant damage occurs when chromium reaches levels of 9 ppm in plant ash (Brooks 1972).

Potted tobacco plants are sensitive indicators of chromium contamination, concentrating chromium rapidly and showing significant leaf growth reduction. They have been used as indicators of chromium contamination (Taylor and Parr 1978).

Invertebrates

Little information is available on invertebrate responses to chromium exposures. Concentrations of 10 to 15 ppm of hexavalent chromium in irrigation water applied to agricultural land were lethal to two species of earthworms by 58 to 60 days (Soni and Abbasi 1981; Abbasi and Soni 1983).

Amphibians/Reptiles

No information was found regarding the toxicity of chromium to amphibians and reptiles.

Table 8.3. Acute LC50 Values Reported for Chromium in Freshwater Fish

<u>Species</u>	<u>LC50 (mg/L)</u>	<u>Exposure Period</u>	<u>Reference</u>
Hexavalent			
Fathead Minnow <i>(Pimephales promelas)</i>	33	96 h	NAS 1974
	17.6	96 h	Pickering and Henderson 1966
	27.3	96 h	Pickering and Henderson 1966
Goldfish <i>(Carassius auratus)</i>	30	96 h	Pickering and Henderson 1966
Bluegill <i>(Lepomis macrochirus)</i>	170	96 h	Trama and Benoit 1960
	0.2	continuous	Surber 1965
	133	96 h	Pickering and Henderson 1966
	118	96 h	Pickering and Henderson 1966
Largemouth Bass <i>(Microterus salmoides)</i>	95	48 h	Fromm and Shiffman 1958
	94	80 h	Fromm and Shiffman 1958
Rainbow Trout <i>(Oncorhynchus mykiss)</i>	69	96 h	Benoit 1976
Brook Trout <i>(Salvelinus fontinalis)</i>	59	96 h	Benoit 1976
Trivalent			
Fathead Minnow <i>(Pimephales promelas)</i>	65	96 h	Pickering and Henderson 1966
	27	96 h	NAS 1974
Bluegill <i>(Lepomis macrochirus)</i>	72	96 h	Pickering and Henderson 1966

Mammals

The toxic effects of chromium on mammals are described in the following sections.

Toxic Response from Oral Exposure. Hexavalent chromium compounds are much more acutely toxic than trivalent compounds. A lethal single oral dose of hexavalent chromium in young rats was 130 mg/kg. However, 650 mg/kg of trivalent chromium was not toxic to rats (Samitz et al. 1962). Soluble hexavalent compounds (e.g., chromic and zinc chromates, calcium chromate, lead chromate, barium chromate, strontium chromate) are about 100- to 1000-fold less toxic than insoluble hexavalent compounds (e.g., chromic acid, the monchromates and dichromates of sodium, potassium, ammonium, lithium, cesium and rubidium) with oral toxicities of about 1500 mg/kg. These compounds, however, have an intermediate toxicity by dermal application of about 200 to 350 mg/kg (Samitz et al. 1962). Large doses (15 mg/kg) of both trivalent and hexavalent chromium compounds caused acute tubular necrosis in laboratory animals when administered parenterally (Kelly et al. 1982; Laborda et al. 1986; Mathur et al. 1977; Biber et al. 1968; Kramp et al. 1974). (Absorption from the gastrointestinal tract was low for both oxidation states.) Dietary exposure of mice for three generations to 20 ppm chromium oxide did not affect mortality, morbidity, growth, or fertility (Hutcheson et al. 1975). Levels up to 276 ppm chromium in the diet of growing rats for 20 weeks did not cause adverse effects in the animals (Mertz 1975; Mertz and Roginski 1975). Cats tolerated 1000 mg trivalent chromium without adverse effects (Venugopal and Luckey 1978). In rats, 1000 ppm dietary hexavalent chromium represented the toxic threshold (Steven et al. 1976). Trivalent chromium in drinking water at a concentration of 5 ppm trivalent chromium over the lifetime of the animals did not cause toxic responses in rats and mice (Schroeder et al. 1964, 1965). However, exposure to 5 ppm hexavalent chromium in drinking water decreased the growth rate of rats (Schroeder 1973). MacKensie et al. (1958) found that 50 ppm hexavalent chromium in drinking water caused liver and kidney damage along with growth depression. Exposure to trivalent chromium at the same level in food did not injure mice (Preston et al. 1976). Water concentrations of 25 ppm chromium as either trivalent or hexavalent chromium did not result in weight loss or pathology in rats (MacKensie et al. 1958). Adverse effects of chromium to sensitive wild mammals have been documented at 5.1 and 10 mg of hexavalent chromium and trivalent chromium, respectively, per kg of diet (ppm).

The major acute effect from ingested chromium is acute renal tubular necrosis. Chromium exposure at sublethal levels also damages the kidney. Low doses produce necrosis of the proximal convoluted tubule that leads to pronounced agluosuremia, ischemia, and tissue damage (Hook and Hewitt 1986). As the dose is increased, damage is observed throughout the proximal tubule. No studies have been conducted on the renal effects of low-level, long-term exposure to chromium (Wedeen and Qian 1991).

Toxic Response from Inhalation Exposure. Progressive pulmonary fibrosis and alterations in respiratory function can result from long-term exposure to both trivalent and hexavalent chromium compounds (Capodaglio et al. 1975; Sluis-Cremer and du Toit 1968). In rats, inhalation exposure to

calcium chromate dust at 13 mg/m³, 5 h per day, 5 days per week for life resulted in growth retardation, marked changes in the epithelium of the bronchia, changes in the tracheal submandibular lymph nodes, and atrophy of the spleen and liver after 2 years of exposure (Nettesheim et al. 1971). Cats were unaffected by aerosol exposures of 80 to 115 mg trivalent chromium for 1 h daily for 4 months. Humans experience strong irritation of nasal membranes at levels as low as 10 µg/m³ even after short exposures (Steven et al. 1976). Inhalation of hexavalent chromium compounds has been linked to kidney and liver damage in humans (Major 1922; Hunter and Roberts 1933).

Toxic Response from Dermal Exposure. Protein complexation accounts for the highly corrosive action in skin, ulcerations, and nasal and respiratory mucous membrane injury by hexavalent chromium (Browning 1969). Entry of chromium through wounds can result in deep ulcerations that penetrate to the underlying bone. The LOAEL for chromic acid, disodium salt, in guinea pigs is 206 mg/kg by dermal exposure (NIOSH 1987). Uptake of chromium from intact skin is very low. Application of 30,000 ppm hexavalent chromium caused skin lesions or ulcers in guinea pigs, but only if the skin at the site of application was abraded or stripped of its natural oils (Steven et al. 1976). Chromium is a potent skin sensitizer and can induce allergic skin reactions (Wahlberg 1973; Hicks et al. 1979).

Other Toxicity Information. Soluble hexavalent chromium compounds are highly toxic by subcutaneous, intraperitoneal, or intramuscular injection (10 to 50 mg/kg), but these routes of exposure have little environmental application.

At concentrations greater than 2 µM (0.3 mg/L) chromium is cytotoxic, causing modifications in the cell cycle (Bakke et al. 1984). The toxicity of chromium probably results from its ability to oxidize substances such as glutathione within the cell, producing trivalent chromium (DePamphilis and Cleland 1973; Cleland and Mildvan 1979; Marzilli 1981). The trivalent chromium ions have the ability to form complexes with macromolecules (e.g., proteins and nucleotides) modifying their structure and function (Denniston and Uyeki 1987; Sissoeff et al. 1976; Tamino et al. 1981; Balbi et al. 1981; DePamphilis and Cleland 1973). For example, formation of metallated nucleotides, such as CrATP, inhibits a number of enzyme systems (Danenberg and Cleland 1985; Cleland and Mildvan 1979; DePamphilis and Cleland 1973) resulting in abnormal cellular metabolism.

It should be noted that chromium is an essential element for humans and several species of laboratory animals. Data are incomplete for other organisms (Eisler 1985).

Immunological Effects of Chromium Exposure. Chromium is a potent sensitizer resulting in the induction of allergic skin reactions (Wahlberg 1973; Hicks et al. 1979). The ability of chromium to form very stable complexes with proteins is the probable mechanism of its toxic action in dermatitis and sensitization (Browning 1969).

Reproductive Effects of Chromium Exposure. Sterility in rats was induced at 1250 ppm zinc chromate in the feed. Potassium chromate caused sterility at 5000 ppm (Gross and Heller 1946).

Spermatogenic cell degeneration was observed in rabbits intraperitoneally injected with 2 mg/kg of either trivalent chromium or hexavalent chromium daily for 3 to 6 weeks (Behari et al. 1978; Tandon et al. 1979). Little placental transfer of chromium to the embryo occurs, indicating that reported malformations and fetal deaths (Iijima et al. 1979; Matsumoto et al. 1976) were likely caused by an action on the uterus or placenta (Leonard et al. 1984). Increased fetal death was observed at an intravenous dose of about 8 mg/kg, and malformations were observed at levels above 15 mg/kg (Matsumoto et al. 1976). Cleft palates and defects in skeletal ossification were observed in offspring of golden hamsters that received intravenous injections of 5 mg hexavalent chromium/kg body weight while pregnant (Gale 1978).

Carcinogenic Effects of Chromium Exposure. Water insoluble hexavalent chromium ions (e.g., chromic and zinc chromates, calcium chromate, lead chromate, barium chromate, strontium chromate) are carcinogenic, whereas the soluble forms (e.g., chromic acid, the monochromates and dichromates of sodium, potassium, ammonium, lithium, cesium, and rubidium) are not (Laskin et al. 1969). In contrast, the water-solubilized trivalent forms of chromium have caused cancers in mammals (Hatherhill 1981), but insoluble trivalent chromium is not biologically active (Gale 1978). Tumors of the lungs, nasal cavity, and paranasal sinus have been reported (Lewis 1992). Whether chromium compounds cause cancer at sites other than the respiratory tract is not clear (Casarett and Doulls 1986). Intratracheal administration of 40 mg/kg of calcium chromate for 34 weeks resulted in tumor formation in the lungs of rats. Lung tumors were also generated by oral uptake of 1600 mg/kg dipotassium chromate for 62 weeks (NIOSH 1987). Exposure to 500 to 1500 $\mu\text{g}/\text{m}^3$ of chromate for 6 to 9 years caused respiratory cancer in humans (ACGIH 1986). Dermal exposure to hexavalent chromium for 2 to 3 months resulted in local carcinomas of the muscle and skin in laboratory animals (Steven et al. 1976).

Genotoxicity. At high concentrations, chromium is a mutagen to a wide variety of organisms including plants, insects, microbes, and mammals (Leonard and Lauwerys 1980; Norseth 1981; Hatherhill 1981; Levis and Bianchi 1982; Bianchi et al. 1983; De Floro and Wetterhahn 1989; Hansen and Stern 1986). Chromium genotoxicity is a complex process involving active chromate ion transport across cell membranes, intracellular reduction via reactive Cr^{+5} and Cr^{+6} intermediates to stable Cr^{+3} species, and the binding of trivalent chromium with nucleic acids (Sissoeff et al. 1976; Tamino et al. 1981; Balbi et al. 1981). Although the ultimate mutagen is trivalent chromium, it is hexavalent chromium that produces positive responses in most test systems used to detect genetic effects. Positive responses are noted for trivalent chromium only in non-intact cell systems in which direct interaction with DNA is permitted (Levis and Bianchi 1982). This occurs because trivalent compounds are unable to cross cell membranes, but hexavalent compounds are actively transported across cell membranes and, once in the cell, are reduced to the genotoxic trivalent form. Trivalent chromium interferes with nucleotide biosynthesis, alters the structure of DNA, stimulates DNA repair, and reduces the fidelity of DNA synthesis (Bianchi and Levis 1986; Majone and Leis 1978; Nakamuro et al. 1978; Uyeki and Nishio 1983). A Chinese hamster ovary cell culture exposed to 52 ppb hexavalent chromium showed sister chromatid exchanges and inhibited cell proliferation. Trivalent chromium at 520 ppm did not affect cell proliferation of chromatid exchanges (Uyeki and

Nishio 1983). The NOAEL was 0.52 ppb Cr⁺⁶. Chromosomal rearrangements and aberrations were reported in rabbit cells after exposure to hexavalent chromium (Hatherhill 1981). Teratogenic effects induced by intravenous exposure to 5 mg hexavalent chromium/kg body weight to pregnant golden hamsters included cleft palates and defects in the ossification of the skeletal system (Gale 1978).

Biomarkers of Chromium Exposure/Effect. Chromium accumulator organs in mammals are the brain, for hexavalent chromium, and the kidney, for trivalent chromium. Tissue levels in excess of 4 mg total Cr/kg dry weight is indicative of chromium contamination (Eisler 1985). However, there is no correlation between the tissue concentration and dose or the extent of tissue damage for either valence state of chromium (Hatherhill 1986).

Exposure to chromium, even at low levels, induces a relatively specific necrosis in the proximal convoluted tubule of the kidney nephrons. This lesion could possibly be used as an indicator of sublethal, but injurious, exposure to the chromium. Mercury poisoning also induces necrosis of the proximal convoluted tubule, but differs in its localization (Goyer 1986).

It has been suggested that renal clearance of chromium can be used as an index of current exposure and body burden of chromium (Franchini et al. 1978; Borghetti et al. 1977).

Chromium Toxicokinetics: Metabolism and Distribution. In general, chromium compounds are poorly absorbed from the gastrointestinal tract. Less than 1% of an oral dose of trivalent chromium is absorbed from the digestive tract of rats; absorption of hexavalent chromium ranges from 1% to 6% (Langard and Norseth 1986; Visek et al. 1953; Underwood 1977; Ogawa 1976). Although hexavalent chromium can readily pass through cell membranes, the acid pH of the stomach reduces much of the ingested hexavalent chromium to the trivalent form. Trivalent chromium cannot pass through membranes, as absorption from the stomach is negligible (MacKensie et al. 1959; Donaldson and Barreras 1966). Trivalent chromium is not converted to hexavalent chromium in the body. In the alkaline pH of the duodenum, the chromium salts form insoluble polynucleate bridge hydroxo-aquo complexes that are excreted in feces. However, some of the complexes remain in solution and are absorbed into the blood (Hopkins and Schwarz 1964; Nelson et al. 1973).

About 0.1% to 1.2% of ingested trivalent chromium and 0.2% to 4% of hexavalent forms are excreted in the urine within 24 h. The bulk (84.7% to 96.7%) of the chromium is eliminated in the feces (Donaldson and Barreras 1966). Of the absorbed chromium, about 80% is excreted in the urine (Hopkins 1965; Mancuso and Hueper 1951). The estimated half-life of hexavalent chromium is 22 days; the half-life of whole-body elimination of trivalent chromium is 92 days (Yamaguchi et al. 1983).

At low doses (1 and 10 µg/kg body weight), hexavalent chromium accumulates in the bone marrow, spleen, testis, epididymis, and heart. At higher dose levels (60 and 250 µg/kg), the major accumulation sites are the liver, spleen, and bone marrow (Langard and Norseth 1986).

Trivalent chromium remains in the lungs after intratracheal application, whereas the soluble hexavalent compounds are absorbed into the bloodstream. About 80% of inhaled chromium is excreted in the urine (Shacklette et al. 1978).

Uptake of chromium through the intact skin is very low. Hexavalent chromium is converted to trivalent chromium on the skin.

No significant transfer of chromium from the mother to the fetus occurs unless the chromium is in a natural complex (Shacklette et al. 1978; Steven et al. 1976; Langard and Norseth 1979).

Birds

Adverse effects of chromium to sensitive species have been documented in wildlife at 5.1 and 10 mg of hexavalent chromium and trivalent chromium, respectively, per kg of diet (ppm). Aerosol concentrations in excess of 50 μg hexavalent chromium/ m^3 are potentially harmful to human health. In the absence of supporting data, this value is recommended for protection of sensitive species of wildlife, especially migratory waterfowl (Eisler 1985).

Dietary exposure to 10 or 50 ppm trivalent chromium for 5 months did not affect survival, reproduction, or blood chemistry of adult black ducks. However, duckling growth patterns were altered and survival rates were reduced by these diets (Heinz and Haseltine 1981). No change in fright-response behaviors was observed in either the adults or ducklings (Heinz and Haseltine 1981). However, exposure to 100 ppm hexavalent chromium in the diet for 3 months was fatal to ducks (Steven et al. 1976). Administration of 11.2 ppm in drinking water was not lethal over a 4-year period (Steven et al. 1976). Chickens appear to be more resistant to hexavalent chromium as exposure to 100 ppm in the diet did not cause any adverse effects (Rosomer et al. 1961).

Severe deformities were observed in chicken embryos following injection of 0.2 mg/kg (Giliani and Marano 1979). Embryo lethality (LD50) was observed when 1.7 mg/kg of hexavalent chromium or 22.9 mg/kg of trivalent chromium was injected into the eggs (Ridgeway and Karnofsky 1952). Deformities were produced by the hexavalent chromium, but no teratogenic effects were seen with injection of trivalent chromium (Ridgeway and Karnofsky 1952). No information is available on transfer and effects of chromium in eggs from adult exposure.

8.2 Bioconcentration Factors and Trophic Transfer Coefficients

The relationship between concentrations of chromium in a given environment and the biological effects on the organisms living there is poorly defined. Accumulation of chromium in organisms and tissues is highly dependent on the chemical form of the compound, route of entry, and exposure concentration (Yamaguchi et al. 1983). The highest concentrations of chromium are found at the lowest trophic levels. No biomagnification of chromium has been observed in food chains.

8.2.1 Chromium Transfer in Aquatic Food Chains

Concentration ratios of chromium in freshwater systems were reported by Vaughan et al. (1975) to be 250 for aquatic plants, 20 for invertebrates, and 40 for fish. Green algae accumulated hexavalent chromium at a rate of about 1000 times that of the water. In general, chromium is accumulated in large amounts by living or dead plant tissue. Mangi et al. (1979) suggest that accumulation by plant tissue occurs at a rate that linearly approximates concentration on a logarithmic basis. According to EPA guidelines, sediments are considered polluted when chromium concentrations are greater than or equal to 25 mg/kg. In annelid worms (*Tubifex* sp.), the concentration factor for trivalent chromium from sediments was 0.0057 (Neff et al. 1978).

8.2.2 Chromium Transfer through Terrestrial Food Chains

Generally, most of the chromium in soil and sediment is unavailable for uptake by biota. Bio-availability of chromium waste in soils is modified by soil pH and the presence of organic complexing substances (James and Bartlett 1983a, 1983b). Because organic material in soil will reduce any soluble hexavalent chromium to insoluble Cr_2O_3 , chromium in soil is found mainly in the trivalent form (Towill et al. 1978). A free trivalent ion is rapidly adsorbed or hydrolyzed and precipitated in soils lacking complexing substances. Organically complexed trivalent chromium remains soluble for at least 1 year.

The concentration ratio for plants grown on soils containing endogenous chromium was 0.002. A concentration ratio of 0.06 was observed in plants grown in amended soil. Distribution of the absorbed chromium was largely restricted to the lower stems (Cataldo and Wildung 1978).

Beetles and crickets collected near cooling towers of uranium enrichment facilities had 9 to 37 ppm chromium in gut contents. Assimilation rates were not measured. Cotton rats trapped in a fescue field adjacent to a cooling tower contained up to 10 times more chromium in fur, pelt, and bone than controls. No accumulation was seen in viscera and other internal organs. Licking of the coat by rats appeared to be a primary route of chromium uptake (Langard and Nordhagen 1980). Radiochromium uptake studies indicate low assimilation (0.8%) and rapid initial loss of hexavalent chromium (99% in 1 day) in rats. Mice fed 0.1 ppm hexavalent chromium in their food and water during a lifetime had 0.1 mg chromium/kg fresh weight in their liver and 0.7 mg/kg in the heart. A diet containing 5.1 ppm hexavalent chromium for a similar period resulted in liver and heart levels of 0.5 mg/kg and 1.8 mg/kg, respectively (Schroeder et al. 1964).

Few concentration factors are reported. Chromium levels considered to be high enough to consider the organism contaminated are listed in Table 8.4.

Table 8.4. Chromium Concentrations in Organisms from Contaminated Terrestrial Systems (Eisler 1985)

<u>Organism</u>	<u>Chromium Content (ppm)</u>
Plants	
Big Sagebrush	77-400 dw ^(a)
Fescue (grass)	15-342 dw
Rye	2.2-3.3 dw
Corn (kernels)	0.02 fw ^(b)
Insects	
Termites	1500 dw
Annelids	
Earthworms	1-13 dw
Mammals	
Pronghorn (hair)	0.3-640 dw
Coyote (hair)	0.7-12 dw
Elk (hair)	1.9-570 dw
Cotton Rat (whole body)	0.12 fw, (0.4) dw
Western Jumping Mouse (hair)	23-45 dw
Birds	
Ducks (liver)	0.02 fw
(eggs)	0.06 fw
Gull	<1 dw
Osprey (liver)	<1 fw

(a) dw dry weight.
 (b) fw fresh weight.

9.0 Technetium

9.1 Technetium Toxicity

Although technetium has a long half-life and is distributed more readily in the environment than most other radionuclides with long half-lives, technetium-99 as a beta-emitter is much less toxic than the alpha-emitting actinides. The toxicity of technetium in animals is low and appears to be related to the radioactive properties of the radionuclide rather than its chemical properties. However, a chemical toxicity has been associated with reduced fertility. Technetium is very toxic to plants. Its chemical properties affect the distribution, and biological half-life in plants, and may influence the retention of plutonium in target tissues (Roucoux and Colle 1986).

9.1.1 Toxicity in Aquatic Biota

The toxicity of technetium in aquatic biota is discussed below.

Aquatic Plants

Technetium exposure to 7.56×10^{-4} M (123 mg/L) (as pertechnetate) induced long lag periods in growth and bleaching of cells in blue-green algae (Gearing et al. 1971).

Invertebrates

No information is available regarding chemical toxicity. See Section 2.1 for radiation impacts.

Fish

No information is available regarding chemical toxicity. See Section 2.1 for radiation impacts.

9.1.2 Toxicity in Terrestrial Biota

The toxicity of technetium to terrestrial biota is presented in the following paragraphs.

Plants

Growth anomalies only occur in plants germinated in the presence of technetium, indicating that the toxicity of this radionuclide is probably associated with early stages of plant growth such as embryonic cell division. Adverse effects on germinating wheat seedlings were first observed at shoot-tissue concentrations of 0.68 to 2.8 $\mu\text{Ci/g}$ (a specific activity of 17 mCi/g corresponds to technetium levels in tissue of 40 to 165 ppm). The threshold dose rate that induced depression of shoot-tissue yield occurred at 2 rad/d. This low-dose rate suggests technetium toxicity is chemical rather than

radiological. Technetium-treated plants display similar symptomology to plants suffering from 2,4-D poisoning (Landa et al. 1977). In lettuce, the chemical toxicity threshold (growth reduction) was observed at concentrations of 0.2 ng/g dry weight of soil (Masson et al. 1989). The lethal concentration for Swiss chard was 0.05 µg technetium/g dry soil. Even at low concentrations of 0.1 µg technetium/g dry soil, technetium has been shown to inhibit plant growth and development in soybeans (Cataldo et al. 1978). Toxic effects were largely observed in buds and young leaves rather than in mature tissues (Finch 1983). It appears that incorporation of technetium results in technetium-cysteine, which is unable to form disulfide-like bridges. Formation of technetium-cysteine leads to nonfunctional proteins that accumulate and to increased production proteins (which end up defective) that, in turn, lead to metabolic dysfunction, especially in young tissue where protein synthesis is critical (Cataldo et al. 1989). Cellular effects of technetium have also been attributed to alteration of membrane permeability (Neel and Onasch 1989).

See Section 2.1 for additional information on radiation toxicity.

Invertebrates

No information is available regarding chemical toxicity. See Section 2.1 for radiation impacts.

Amphibians/Reptiles

No information is available regarding chemical toxicity. See Section 2.1 for radiation impacts.

Mammals

Because stable isotopes of this metal do not exist, there are few available data on the chemical toxicity of technetium in mammals. However, it is chemically similar to rhenium, and its toxicity is probably between manganese and rhenium. The toxicity of common manganese compounds varies from 90 to 934 mg/kg (Bowen 1979; NIOSH 1987). Rhenium toxicity is low. Intra-peritoneal injection of rhenium trichloride results in an LD50 of 280 mg/kg (Lewis 1992). The metastable isomer, technetium-99m, is used as a tracer and diagnostic tool in biology and medicine because of its low beta-particle energy, yet high specific activity, and poor absorption by mammals (Durbin 1960; Harper et al. 1964). Administration of very high concentrations of technetium (10 µg/g) in food is required to produce deleterious effects to thyroid function, fertility, and postnatal development (Van Bruwaene et al. 1986; Gerber et al. 1989). Because the radiation dose to the conceptus was only about 10 to 20 mGy, the fertility and fetal development impacts are likely caused by the chemical rather than radiation toxicity of technetium.

Biomarkers of Technetium Exposure/Effect. Technetium tends to concentrate in the thyroid and parathyroid (McGill et al. 1971). However, no relationship between tissue activity and exposure or

dose-response information is available. Other tissues with pronounced technetium concentration are the bone and skin. Hair also accumulates technetium and may be useful as a bioindicator of technetium exposure (Gerber et al. 1989).

Technetium Toxicokinetics: Metabolism and Distribution. In mammals, absorption of inorganic pertechnetate from the gastrointestinal tract is about 90%. However, biological half-life in humans is 2 days. Nearly all technetium is excreted within a week (Beasley et al. 1966). However, when technetium has been incorporated in plant tissue, the absorption rate is greatly reduced. About 75% of the ingested dose of technetium-95m incorporated in soybean tissue was excreted in the feces in 2 days in rats (Sullivan et al. 1979). Less than 10% was excreted in the urine. In guinea pigs, about 80% of the technetium-95m ingested as soybean-incorporated material was excreted in the feces and 10% in the urine within 2 days (Sullivan et al. 1979). Polygastric animals appeared to absorb less technetium than monogastric animals (Gerber et al. 1989). This reduced absorption may be due to the reduction of TcO_4^- in the rumen of polygastric animals that interferes with its reabsorption from the intestine (Jones 1989).

Birds

Although technetium is concentrated in avian oocytes (Roche et al. 1957; Thomas et al. 1984), no impacts to developing embryos have been noted.

No information is available regarding chemical toxicity. See Section 2.1 for radiation impacts.

9.2 Bioconcentration Factors and Trophic Transfer Coefficients

Technetium is very mobile, particularly in terrestrial ecosystems. It is poorly retained by aerated soil and accumulates in plants. Although assimilation of ingested technetium compounds can be high, retention of the radionuclide is low in animals. Transfer of technetium incorporated in plant tissue to animals and its retention in their tissues are even lower than for unincorporated technetium, indicating a low potential for food chain magnification.

9.2.1 Technetium Transfer in Aquatic Food Chains

Under anaerobic sediment conditions, technetium is reduced to Tc (IV), which is not absorbed by roots and, thus, macrophytes are unable to concentrate technetium in their tissues (Sheppard and Evendon 1991). The Commission of the European Communities (1979) suggests a technetium concentration factor for freshwater fish of 30 L/kg. This value is then multiplied by the concentration of technetium in the water (Zeevaert et al. 1989). In the absence of site-specific data, recommended default values for the water-based bioconcentration factor for technetium in the flesh of freshwater fish are 15 (NRCC 1983), 30 (CSA 1987), and 15 (Poston and Klopfer 1986; Myers et al. 1989).

No information was available on uptake of technetium in other components of the aquatic food chain.

9.2.2 Technetium Transfer through Terrestrial Food Chains

Plants readily concentrate technetium in their tissues and play an important role in technetium cycling in the environment. Plants are able to effectively accumulate technetium at soil levels as low as 0.01 $\mu\text{g/g}$. Hydroponically grown plants concentrate technetium in their tissues at culture levels as low as 0.02 pg/mL (Cataldo et al. 1989). In general, between 47% and 74% of the technetium applied to soil in water is assimilated by plants. Dicotyledon species appear to have a much higher root/shoot concentration ratio than is found in monocots. Cataldo and Wildung (1983) reported a 25% translocation of absorbed technetium from the root to the shoots, whereas about 5% to 7% of the technetium was found in root tissue and 42% to 67% appeared in the above-ground tissue of wheat seedlings (Landa et al. 1977). Concentration factors for technetium from the upper 15 cm of soil at field sites have been reported to range from 3 to 370 (Garland et al. 1983) and from 2 to 200 (Hoffman et al. 1980). Laboratory studies have produced concentration factors of 10 to 1200 (Landa et al. 1977; Wildung et al. 1977; Mousney and Myttenaere 1981). For native plants at the Hanford Site, Rouston and Cataldo (1977) have suggested a concentration factor of 76 to 390 for tumbleweed and 54 to 421 for cheat grass from five Hanford project soils. Swiss chard grown on different soil types had concentration factors of 11 to 2600. The variation in uptake appeared to be related to soil sorption of technetium in peat. Technetium uptake by the chard was four orders of magnitude higher in sand than for plants grown in peat (Sheppard et al. 1983). The final transfer coefficient was reached within 2 days of growing in contaminated soil and remained constant for the life of the plant (Masson et al. 1989). Van Loon et al. (1989) have described a general soil-to-plant transfer function for technetium.

Note that although technetium mobility is almost unretarded in aerated surface soils (and, therefore, readily accumulated by plants), technetium in unaerated soils is much less available (Sheppard et al. 1990; Garten 1987). This finding suggests that root depth is an important consideration in estimating plant uptake. Indeed, Sheppard and Evenden (1985) showed that technetium in the unaerated subsoil was not available to a terrestrial cereal.

Most soil parameters do not affect technetium uptake in plants. However, fertilized soil reduces technetium uptake. Concentration factors ($\mu\text{Ci/g tissue}/\mu\text{Ci/b soil}$) were 700 for plants grown in fertilized soil and 950 for plants grown in unfertilized soil (Landa et al. 1977). Nutrients effective in reducing uptake included manganese, sulfate, phosphate, and molybdenate (Cataldo et al. 1989). Plants grown on soils containing technetium concentrations less than 0.1 $\mu\text{g/g}$ were more effective at technetium uptake and removed up to 90% of the radionuclide from the soil. The presence of actinides has been reported to enhance technetium uptake in plants in some soil types (Masson et al. 1989). Technetium uptake in leaves of radish plants grown in calcareous soils was increased 4 times in the presence of uranium and 4.5 times in plutonium-amended soils. Plutonium also appeared to increase technetium uptake 1.5 times in the leaves and 3 times in roots of plants grown in acid soils.

The presence of americium in organic soils resulted in a sixfold increase in leaf uptake of technetium (Roucoux and Colle 1986; Masson et al. 1989). However, because no soil/plant concentrations or statistical information is presented, the validity of the reported technetium actinide relationship is unclear.

Incorporation of technetium in plant tissue alters the absorption and retention of the radionuclide in animal tissues. Whole body retention of incorporated technetium was less than 1% in rats and 0.5% in guinea pigs after 6 days (Sullivan et al. 1979). Although as much as 8.4% of ionic technetium was transferred to quail eggs, only 2% was transferred when the radionuclide was ingested in plant material (Thomas et al. 1984). Of the technetium deposited in the egg, 80% appeared in the yolk and 20% in the albumin.

10.0 Tritium

10.1 Tritium Toxicity

Tritium released from nuclear facilities enters the environment mainly as tritiated water and is, therefore, distributed rapidly throughout the biosphere. Tritium decays by beta particle emission with a maximum energy that is about one-hundredth of the energy of most beta emitters. Because beta particles of such low energy can only penetrate about 0.004 mm of tissue, an external radiation dose from tritium is not considered hazardous (Blaylock 1973). The short biological half-life of tritium also decreases its hazard relative to other radionuclides. The biological effects induced by tritium exposure do not differ in any qualitative way from those induced by external X- or gamma-radiation. Reference should be made to Section 2.1 for a description of the ionizing radiation toxicity.

10.1.1 Toxicity in Aquatic Biota

Exposure of fertilized eggs to beta radiation from tritiated water altered lifetime reproduction in mature medaka (*Oryzias latipes*), a small teleost fish (Taguchi and Etoh 1986). Reduced oviposition frequency and number of eggs laid per fish were observed in pairs composed of female fish exposed for 10 days during embryonic development and unirradiated males. However, those eggs produced were fertilized and hatched normally. When irradiated males were mated with unirradiated females, the number of fertilized eggs per fish and the hatchability of the fertilized eggs were reduced significantly. Both the reduced fecundity in females and fertility in males were observed at the lowest dose tested, 0.05 mCi/mL (total accumulated dose of 85 rad). Higher doses produced completely infertile fish. The response was dose-dependent. Fifty percent loss of female reproductive capacity occurred at an accumulated dose of tritium beta emissions of 400 rad.

See Section 2.1 for effects of ionizing radiation on aquatic biota.

10.1.2 Toxicity in Terrestrial Biota

The toxic effects of tritium on terrestrial biota are summarized below.

Plants

See Section 2.1 for effects of ionizing radiation on terrestrial biota.

Invertebrates

See Section 2.1 for effects of ionizing radiation on terrestrial biota.

Amphibians/Reptiles

See Section 2.1 for effects of ionizing radiation on terrestrial biota.

Birds

See Section 2.1 for effects of ionizing radiation on terrestrial biota.

Mammals

Because tritium, as tritiated water, is readily absorbed into the bloodstream from all routes of exposure and is distributed throughout the body, any radiation effects are comparable to whole-body irradiation (Osborne 1972; Stannard 1973). Entry of tritium into the body in organic form may lead to a concentration of the radionuclide within vital structures such as DNA. Once incorporated into DNA, tritium remains there until the cell's death. The genetic consequences of this incorporation, particularly in the most radiation-sensitive organs like ovary or testes, are of concern. When tritium is administered as tritiated water, less than 1% of the ingested tritium is incorporated into the organic compartment. However, administration of tritiated food results in a 4% to 11% incorporation of tritium in this compartment (Kirchmann et al. 1977). About 75% of the total hydrogen in the mammalian body is in the form of water, 14% is in proteins, and less than 0.5% is in nucleic acids (DNA and RNA) (Commerford 1984).

See Section 2.1 for effects of ionizing radiation on terrestrial biota.

Biomarkers of Tritium Exposure/Effect. Radioactivity of the exchangeable water component of the body can provide information on current exposure.

Tritium Toxicokinetic: Metabolism and Distribution. Only 0.004% of inhaled tritium dissolves in the blood and is transported throughout the body (Peterman 1982; Peterman et al. 1985). Uptake of tritiated water vapor is about equal along both the inhalation and dermal absorption route (Pinson and Langham 1957; Osburn 1978).

Distribution of organically bound tritium in rats, calves, and rabbits appears to be related to the metabolic activity of the individual tissues. The liver, kidney, and small intestine have much higher concentrations of tritium than the muscle and brain (Kirchmann et al. 1973; Pietrzak-Flis et al. 1978; Takeda and Iwakura 1992).

10.2 Bioconcentration Factors and Trophic Transfer Coefficients

As one would expect of water molecules, tritiated water is readily taken up by biota and incorporated into tissues. Plants appear to incorporate tritium more efficiently by photosynthetic

processes (Choi and Aronoff 1966; Kanazawa et al. 1982). Consumption of plant tissues by animals distributes the tritium into food webs. Retention of tritium in the food chain depends on the rate of catabolism at the trophic levels. Lipids are not completely catabolized, leading to retention of tritium, whereas carbohydrates are more completely catabolized, leading to loss of tritium from the food chain. The metabolic turnover rate of the carbon-associated tritium compounds then determines the concentration of tritium in the organism. Overall, neither plants nor animals concentrate tritium in their tissues, and tritium enrichment in food chains has not been observed.

10.2.1 Tritium Transfer in Aquatic Food Chains

Tritiated water can exchange with mobile chemical sites in fish flesh. In addition, tritium can be metabolically incorporated on less exchangeable sites in the fish from catabolic processes using contaminated organic matter that has moved up the food chain. In a study of tritium uptake by large-mouth bass in a stream near the center of the Savannah River Site, samples of fish flesh were freeze-dried, and the tritium content of the freeze-dried water was determined. Because tritiated water exchanges rapidly with fish, this measure reflects the concentration to which the fish had been exposed the previous day. The freeze-dried flesh was then combusted, and the water of combustion was analyzed for tritium content to provide information on the amount of organically bound tritium in the fish. Tritium activity of the stream water and vegetation was compared to levels of tritium in the fish. The results showed that the tritium content of the fish flesh was about equal to the tritium in the water of the previous year (Eaton and Murphy 1992).

10.2.2 Tritium Transfer through Terrestrial Food Chains

The biological half-life of tritium in plants can be defined by three components. The first component is rapidly excreted (0.3 to 2.0 h) and represents over 90% of the total incorporated tritium. Organically bound tritium turns over more slowly and has a half-life of about 17 to 30 h. The third component is from tritium in soil water and has a half-life of 80 to 270 h (Anspaugh et al. 1973; Koranda and Martin 1973; Belot et al. 1979; Guenot and Belot 1984). Plant concentration ratios are less than 1 for plants exposed to tritiated water (Diabate et al. 1990).

Significantly higher (10- to 60-fold) incorporation of tritium into mammalian tissues is seen after exposure to tritiated vegetation than to tritiated water (Kirchmann et al. 1977; Takeda and Iwakura 1992). Different incorporation rates of tritium into rat tissues were seen for different plant species. The difference appeared to be related to the chemical composition of the plants, with a higher protein content of the plant resulting in higher tritium incorporation into the rat tissues. High tritium incorporation into fat tissue of rats was also observed in rats fed plants with high fat content (e.g., soybean) (Takeda and Iwakura 1992). Concentration ratios of tritium in all tissues in rats were less than 1.0 (range 0.24 to 0.60) for rats fed tritiated rice. Higher concentration ratios were seen in rats fed tritiated soybean (0.32 to 1.10). Only the liver and lung of rats fed this higher fat food had concentration ratios greater than 1.0. Specific activity ratios between concentrations in milk of ruminants and drinking water were about 0.83 for milk water, 0.48 for lactose, 0.3 for milk fat, and 0.22 for

casein (Van Den Hoek et al. 1983). Ratios between milk and feed in ruminants were 0.10 for milk water, 0.84 for milk fat, 0.49 for casein, and 0.05 for lactose (Van Den Hoek et al. 1983). About 0.7% of the total ingested activity of tritiated water was incorporated into the main organs of pigs. When tritium was ingested as tritiated milk powder, 4.8% of the total activity ingested was incorporated into the tissues. Ingestion of tritiated potatoes resulted in an incorporation of 10.98% of the total activity into the major organs of the pig (Kirchmann et al. 1977). As shown in Table 10.1, no concentration of tritium is seen in animals exposed to tritiated water or feed. The one exception was the kangaroo rat, which has a unique water metabolism allowing it to maintain a positive water balance eating dry grain. The biological half-lives of tritium in mammals and birds are listed in Table 10.2.

Table 10.1. Specific Activity Ratios in Mammals after Continuous Tritium Intake (Diabate and Strack 1990)

<u>Species</u>	<u>Exposure</u>	<u>Specific Activity Ratio</u>	<u>Reference</u>
Rats	Tritiated water	0.2-0.47	Thompson and Ballou 1956 Laskey 1973
Rats	Tritiated meat	0.22-0.37	Pietrzak-Flis et al. 1982
Mice (liver and testes)	Tritiated water	0.25-0.4	Hatch and Mazrimas 1972
Kangaroo Rats	Tritiated environment	1.2-1.6	Martin and Koranda 1972
Rabbits	Tritiated water and feed	0.95-1.0	Moghissi et al. 1987

Table 10.2. Biological Half-Lives of Tritium in Body Water of Mammals and Birds (Van Den Hoek et al. 1983)

<u>Species</u>	<u>Biological Half-Life (days)</u>	
	<u>First Compartment</u>	<u>Second Compartment</u>
Mouse	1.1-1.6	23
Kangaroo Rat	13.3	114
Chicken	4.6	
Pig	3.8-4.3	
Cow (lactating)	3.1-4.0	33
Cow (non-lactating)	4.0	40

11.0 Europium

11.1 Europium Toxicity

Europium is a rare-earth element; i.e., an element in the lanthanide series. Because the lanthanide elements possess similar physical and chemical properties, their toxicities are also similar. Where data for europium are lacking, available information on similar rare-earth elements is reported to provide information on relative toxicity.

11.1.1 Europium Toxicity in Aquatic Biota

No information on the toxicity of europium to aquatic biota is available. For radioactive isotopes of europium, see Section 2.1 for a review of radiation toxicity.

11.1.2 Europium Toxicity in Terrestrial Biota

Europium toxicity in terrestrial biota is recorded below.

Plants

No information is available on the toxicity of europium to terrestrial plants.

Invertebrates

No information is available on the toxicity of europium to terrestrial invertebrates.

Mammals

Europium, along with the other lanthanide elements, is considered practically nontoxic when administered orally. In laboratory rats, the oral LD50 for nitrate compounds of the lanthanides is 5000 mg/kg. The oral LD50 for lanthanide chlorides in rats and mice is 5000 mg/kg (Rhone-Poulenc 1986, 1987). Oxides of rare-earth metals have LD50 values greater than 1000 mg/kg and may be as low as 10,000 mg/kg (Lewis 1992). No growth inhibition or histopathological damage was observed in mice and rats fed europium chloride at 1% of their diet for 3 months and europium at 0.05% of the diet caused no adverse effects through 3 generations of mice (Hutcheson et al. 1975a).

Europium is probably moderately toxic following parenteral administration of its soluble salts. Animal studies show that rare-earth oxides (cerium and neodymium) are less toxic than yttrium by inhalation. Intratracheal installation of 50 mg yttrium oxide produced granulomatous nodules and emphysematous changes in rats after 8 months (Mogilivskaya and Raikhlin 1963). Assuming

europium is less toxic than yttrium, no adverse effects from inhalation of the lanthanide element are anticipated in humans after a lifetime exposure to 1 mg/m³ (Stokinger 1981).

Rare-earth salts may irritate or damage eyes and abraded skin (Haley 1977).

Toxicokinetics: Metabolism and Distribution. Gastrointestinal absorption of the compounds of rare-earth metals is poor. In mammals, absorption of lanthanide salts is less than 0.05%. This low level of absorption has been recorded in all species studied so far. Species tested include rats and mice (Cochran et al. 1950; Haley 1965; Hutcheson et al. 1975b; Luckey et al. 1975), goats (Ekman and Aberg 1961), and cows (Gamer et al. 1960). Retention in mammals occurs mostly in the skeleton and less in soft tissues, except the liver. Low levels of absorbed lanthanides are distributed rapidly into the liver and kidneys followed by a gradual uptake and retention in the bones. Depositions of lanthanides are generally about 50% in liver and 50% in bone. The skeleton retains about 67% of the initial deposition after eight months (Hart et al. 1955). Intermediate concentrations of lanthanides separate out as colloidal hydroxides of phosphates and are removed by macrophages. They are then transported to the lymph nodes, bone marrow and liver. Subsequently, they are excreted into the digestive tract through the enteropathic circulation. Urinary excretion is also documented (Wald and Mode 1989).

Biomarkers of Radiation Exposure. No biomarkers of exposure were found in the literature. Liver is the target organ in lanthanide intoxication, and fatty liver degeneration and mitochondrial damage are observed at high concentrations (Snyder et al. 1959). However, the pathology is not unique to europium toxicity.

Birds

Teratogenic effects have been observed in chicken embryos after injection of europium chloride into the yolk sac of 8-day-old embryos. An injection of 20 mg europium as europium chloride caused leg deformities, joint damage, inhibition of feather formation, and edema (Zanni 1965). Female chicks were more susceptible to the teratogenic challenge than males.

11.2 Bioconcentration Factors and Trophic Transfer Coefficients

Little information is available on the accumulation and transfer of europium in aquatic and terrestrial food chains. Because lanthanide oxides are relatively insoluble and lack biological function, europium uptake and food chain transport are not expected.

11.2.1 Radionuclide Transfer in Aquatic Systems

No information is available on the bioaccumulation and trophic transfer of europium in freshwater systems.

11.2.2 Radionuclide Transfer in Terrestrial Systems

In general, plants do not absorb the lanthanides from the soil because of discrimination against their absorption by the roots. This effectively blocks the dietary transfer of lanthanides from soil to animals (Wald and Mode 1990). Shibuya and Nakai (1963) reported an accumulation of 0.21 ppm europium in terrestrial plants grown in areas where igneous rock is prevalent. Assuming a soil content of 1 to 2 ppm for soils of igneous rock origin (Bowen 1966), a bioaccumulation factor of about 0.014 may be estimated. Walnut species (*Carya*), which excrete complexing agents and have large concentrations of natural ligand in their rhizosphere, accumulate much higher levels of europium (Robinson et al. 1958). Calculated concentration factors for these species may range from 16 to 80.

No information is available for trophic transfer to higher organisms.

12.0 Nitrate

12.1 Nitrate Toxicity

Nitrate toxicity to both aquatic and terrestrial biota is low. Toxic levels have been shown to accumulate in some plant species under certain conditions. However, both acute and chronic poisonings are rare.

12.1.1 Nitrate Toxicity to Aquatic Biota

Nitrate toxicity to aquatic invertebrates is low (Hohreiter 1980). The LC50 for planaria (*Polycelia nigra*) is 1000 mg nitrate/L (Jones 1940). The acute 96-h LC50 for *Daphnia magna* is 665 mg/L (Dowden and Bennett 1965). Anderson (1944) reported immobilization of *Daphnia magna* after exposure to 5000 mg/L nitrate for 48 h. Reported 24-h LC50 for snails are 6000 mg/L for *Blomphalaria alexandrina*, and 3100 for *Pulinus truncatus* (Gohar and El-Gindy 1961). The 96-h LC50 for *Lymnes* species is 3251 (Dowden and Bennett 1965).

Nitrate levels in water that are harmful to freshwater fish are listed in Table 12.1.

Table 12.1 LC50 Values for Nitrate in Freshwater Fish

Species	Test Duration	LC50 (mg/L NO ₃ -N ^(a))	Reference
Chinook Salmon (<i>Oncorhynchus tshawytscha</i>)	96 h	1,310	Westin 1974
	7 d	1,080	Westin 1974
Rainbow Trout (<i>Oncorhynchus mykiss</i>) (fingerlings)	96 h	1,360	Westin 1974
	7 d	1,060	Westin 1974
Bluegills (<i>Lepomis macrochirus</i>)	96 h	2,000 (NaNO ₃)	Trama 1954
	96 h	420 (KNO ₃)	Trama 1954
	large	9,000	Cairns and Scheier 1959
	medium	10,000	Cairns and Scheier 1959
	small	9,400	Cairns and Scheier 1959
Mosquito Fish (<i>Gambusia affinis</i>)	96 h	6,650	Wallen et al. 1957
Goldfish (<i>Carassius auratus</i>)	14 h	1,282	Powers 1971
Warm water fish	life	90	Knepp and Arkin 1973

(a) 1.0% NO₃-N = 4.4% NO₃.

12.1.2 Nitrate Toxicity in Mammals

The nitrate toxicity in terrestrial biota is summarized below.

Mammals

Nitrate is of very low toxicity to animals and is rapidly excreted in the urine. It becomes a hazard only at high concentrations and under conditions that may convert it to nitrite, a much more toxic ion. Nitrite is readily absorbed into the bloodstream where it oxidizes ferrous iron in hemoglobin to the ferric state, forming methemoglobin. Methemoglobin cannot accept molecular oxygen, which reduces the oxygen-carrying capacity of the blood resulting in hypoxea or anoxia. Clinical signs of nitrate (nitrite) poisoning are dyspnea, cyanotic mucous membranes, and blood that is typically dark brown in color (Wolff and Wasserman 1972, Menzer 1991). Conditions that increase the hazard of nitrate (i.e., the conversion of nitrate to nitrite) are the presence of nitrogen-reducing microbes and low acidity in the gastrointestinal tract of warm-blooded animals. Human babies under 3 months have the lower acidity conditions in their digestive tracts characteristic of infant mammals and are at greater risk of nitrite formation than adults, assuming the presence of nitrogen-reducing microbes in the digestive tract. Illness has only been found in infants ingesting greater than 10 mg/L nitrate-nitrogen (NAS 1974). Therefore, a limit of 10 mg nitrate-nitrogen/L has been imposed on drinking water by the USEPA to prevent methemoglobinemia in bottle-fed infants (WHO 1978, 1985).

In animals other than humans, nitrate poisoning has occurred at concentrations between 1000 and 3000 ppm in water (Buck et al. 1976). Acute poisoning may also result from ingestion of plants that, under certain conditions, concentrate nitrate (Table 12.2). Conditions that lead to abnormal nitrate concentrations in these species of plants are high soil nitrate or ammonia levels, acid soil, low molybdenum, sulfur deficiency, low ambient temperature (55°F), soil aeration, drought conditions and decreased light (Buck et al. 1976).

Nitrates accumulate in the vegetative tissue, not in grain or fruits and, in general, the concentrations are greater in the stalks than in the leaves (Buck et al. 1976). Acute toxicosis occurs in herbivores consuming forage containing more than 1.0% nitrate (dry weight basis) (Dollahite and Rowe 1974).

Ruminants are 2 to 3 times more susceptible to nitrate toxicity than monogastric animals (Emerick 1974). The LD50 for nitrate fed to cattle in forage is about 1 g/kg body weight (Crawford et al. 1966). However, ruminants fed nitrate continuously become adapted to higher nitrate concentrations and nitrate levels in forage as high as 2 to 4% have been tolerated by ruminants (Buck et al. 1976).

Chronic nitrate poisoning is extremely rare and has not been readily verified in mammals (Turner and Kienhoz 1972, Emerick 1974, Ridder and Oehme 1974). A number of symptoms in domestic

Plants that Accumulate Nitrate (from Jones and Hunt 1983; Buck et al. 1976)

<u>Scientific Name</u>	<u>Common Name</u>
<i>Amaranthus retroflexus</i>	Pigweed
<i>Lactuca podium</i> spp.	Lamb's Quarters
<i>Cirsium arvense</i>	Canada Thistle
<i>Solanum</i> spp.	Jimsonweed
<i>Helianthus annuus</i>	Wild Sunflower
<i>Rumex crispus</i>	Fireweed
<i>Chenopodium parviflorum</i>	Cheeseweed
<i>Trifolium officinale</i>	Sweet Clover
<i>Amaranthus</i> spp.	Smartweed
<i>Rumex</i> spp.	Dock
<i>Cirsium</i> spp.	Russian Thistle
<i>Cirsium marianum</i>	Variegated or "Bull" Thistle
<i>Solanum</i> spp.	Nightshades
<i>Setaria</i> spp.	Johnson Grass

Crop Plants

<i>Avena sativa</i>	Oats
<i>Beta vulgaris</i>	Beets
<i>Brassica napus</i>	Rape
<i>Glycine max</i>	Soybean
<i>Linum usitatissimum</i>	Flax
<i>Medicago sativa</i>	Alfalfa
<i>Cereale</i>	Rye
<i>Sorghum vulgare</i>	Sudan grass
<i>Triticum aestivum</i>	Wheat
<i>Zea mays</i>	Corn

acute nitrate toxicosis, but no experimental evidence has been found to substantiate interference of thyroidal iodine uptake has been documented in some animals. However, it has not been demonstrated in cattle and dogs (Ridder and Oehme 1974). However, a hypothyroid effect has been noted, and thyroid function usually returns to normal after exposure. Nitrate may also dilate the arterioles causing lowered blood pressure.

12.2 Bioconcentration and Trophic Transfer Coefficients

With the exception of certain plants growing under specific conditions, nitrate is readily eliminated from organisms and, therefore, does not usually accumulate or magnify in concentration along food chains. The accumulation of nitrate in plants is discussed above (Section 12.1.2).

13.0 References

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Appendix A

**Radiological Units
and
International Multiples and Submultiples**

Appendix A

Radiological Units and International Multiples and Submultiples

In 1985, a new system of units based on fundamental physical quantities replaced the less precisely defined radiation units found in the older literature. Described below are the new International System (SI) units and the radiation units commonly found in the older literature. The international multiples and submultiples used in the text of this report are also provided.

<u>Symbol</u>	<u>Unit</u>	<u>Description</u>
Ci	Curie	A unit of activity of a radionuclide. A curie is equal to 3.7×10^{10} disintegrations (i.e. nuclear transformations) per second.
Bq	Becquerel	SI unit of activity of a radionuclide. One becquerel is equal to one disintegration (i.e. one nuclear transformation) per second.
R	Roentgen	A special unit of exposure for x- or gamma radiation that describes the quantity of ionization that these radiations produce in the air. An exposure of one roentgen results in 2.58×10^{-4} coulomb per kilogram of dry air.
rad	Rad	A unit of absorbed dose for any ionizing radiation. A rad is equal to 100 ergs absorbed per gram of any substance or 0.01 joule per kilogram. For water and soft tissues the absorbed dose per roentgen is between 0.93 and 0.98 rad. Therefore the roentgen and rad are nearly equivalent numerically.
Gy	Gray	SI unit of dose equal to 1 joule per kilogram. One gray = 100 rad.
Q (or QF)	Quality Factor	A unit used for radiation protection purposes in conjunction with the absorbed dose that accounts for the varying effectiveness of different radiations in producing a given biological effects.
rem	Rem	A unit of dose equivalent that is numerically equal to the dose in rads multiplied by the

<u>Symbol</u>	<u>Unit</u>	<u>Description</u>
		quality factor and any other modifying factors. (Under most conditions one rem is about equal to one rad.)
Sv	Seivert	SI a unit of dose equivalent that is numerically equal to the dose in grays multiplied by the quality factor and any other modifying factors.
K	kilo	10^3
m	milli	10^{-3}
μ	micro	10^{-6}
n	nano	10^{-9}
p	pico	10^{-12}

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