6. Injuries to Biological Resources

This chapter reflects the State Trustees' current understanding regarding injury to biological resources at the Arsenal, and identifies proposed approaches for completing an injury assessment for these resources.

Biological resources are defined in the DOI regulations as "those natural resources referred to in Section 101(16) of CERCLA as fish and wildlife and other biota. Fish and wildlife include marine and freshwater aquatic and terrestrial species; game, non-game, and commercial species; and threatened, endangered, and State sensitive species. Other biota encompass shellfish, terrestrial and aquatic plants, and other living organisms not listed in this definition" [43 CFR § 11.14 (f)].

Outside the central facilities area, the Arsenal consists largely of undeveloped, open grassland. Approximately 20% of the site is currently native grassland, and the rest of the area consists of exotic grasses and forbs, wetlands, riparian woodlands, intermittent streams, and permanent lakes (USFWS, 1997). A wide variety of wildlife use or inhabit the site and has been exposed to contamination. Wildlife at the Arsenal includes fish, reptiles, amphibians, small and large mammals, and more than 200 species of birds (Environmental Science and Engineering, 1989). Injured wildlife includes species that are resident at the site year-round and migrant species that use other habitats outside the Arsenal. Migrants include local migrants such as red fox and coyotes, seasonal migrants such as over-wintering bald eagles, and short-term migrants that use the site during annual migrations.

Section 6.1 of this chapter provides a summary of conclusions. Section 6.2 describes biological resources at the site. Section 6.3 presents relevant injury definitions from the DOI NRDA regulations and their application to the Arsenal. Section 6.4 discusses baseline conditions at the Arsenal. Section 6.5 describes approaches for determining injury for different biological resources. Section 6.6 presents preliminary information relevant for injury quantification. Section 6.7 describes the State Trustees' planned assessment activities, and references are included at the end of this chapter.

6.1 Summary of Conclusions

As described in preceding chapters, much of the habitat at the Arsenal was historically contaminated with elevated concentrations of hazardous substances in soil, surface water, and sediments. In addition, exposure to highly contaminated wastes in disposal basins such as Basin F proved acutely lethal to thousands of waterfowl. Contaminants include the toxic pesticides aldrin, DBCP, dieldrin, endrin, and isodrin. Prior to remediation, the pesticide dieldrin was

detected in surface soils across most of the Arsenal property (Rocky Mountain Arsenal Environmental Database, as described in Potomac Research International, 2006). The highest concentrations of dieldrin were found in the central area (Figure 6.1). Remediation of these areas, in accordance with the 1996 ROD, is anticipated to be complete in 2010.

The PASD and other preliminary observations indicate that injuries at the site include wildlife mortality, sub-lethal adverse effects, gamebird exceedences of U.S. Food and Drug Administration (FDA) safe levels for dieldrin, and exceedences of water quality criteria that are indicative of injuries to aquatic biota. These injuries have been reduced but not eliminated by past and ongoing remediation efforts. When completed, the remediation is expected to prevent any future biota exposures to harmful concentrations of hazardous substances.

In addition to injuries due to contamination exposure, some of the remedial activities themselves have caused injury, such as the construction of “biota barriers” to protect burrowing mammals from exposure to contamination in soils, and the excavation of borrow areas for the construction of landfill caps. Anticipated future assessment activities would expand the injury determination and quantify injuries and ecological service losses at the Arsenal.

Injury quantification will be used as inputs for a habitat equivalency analysis (HEA) or a resource equivalency analysis (REA), as described in Chapter 8. These approaches will allow the Trustees to determine the amount of restoration required to compensate for natural resource injuries at the Arsenal.

6.2 Biological Resources at the Arsenal

The Arsenal is located in the shortgrass prairie “ecoregion” in the Great Plains. The shortgrass prairie extends east from the Rocky Mountains and south from Montana into the high plains of Oklahoma, New Mexico, and Texas (Samson et al., 1998), and is characterized by ankle-high vegetation dominated by the characteristic grasses blue grama and buffalo grass. At a regional level, shortgrass prairie habitat is threatened by land conversion for urban development and agriculture (Neely et al., 2006). The Arsenal contains locally important habitat because of the extensive urban development that surrounds the site.

Within the Arsenal, there are three major habitat categories that each support different types of biological resources: upland prairie, perennial and intermittent surface water, and wetlands and riparian woodlands (Figure 6.2). The vast majority of the site (15,065 acres; 89% of total) is covered by prairie-type habitat, including weedy forbs and grasses, native perennial grasses, and shrubland/succulents. The area of surface water is limited (158 acres, 0.9%), but provides important habitat for waterfowl and other wildlife. Wetlands and riparian woodlands also cover a small area of the site (4%), but provide key habitat for wildlife, including large cottonwoods that are used as winter roosting sites for bald eagles (Table 6.1).

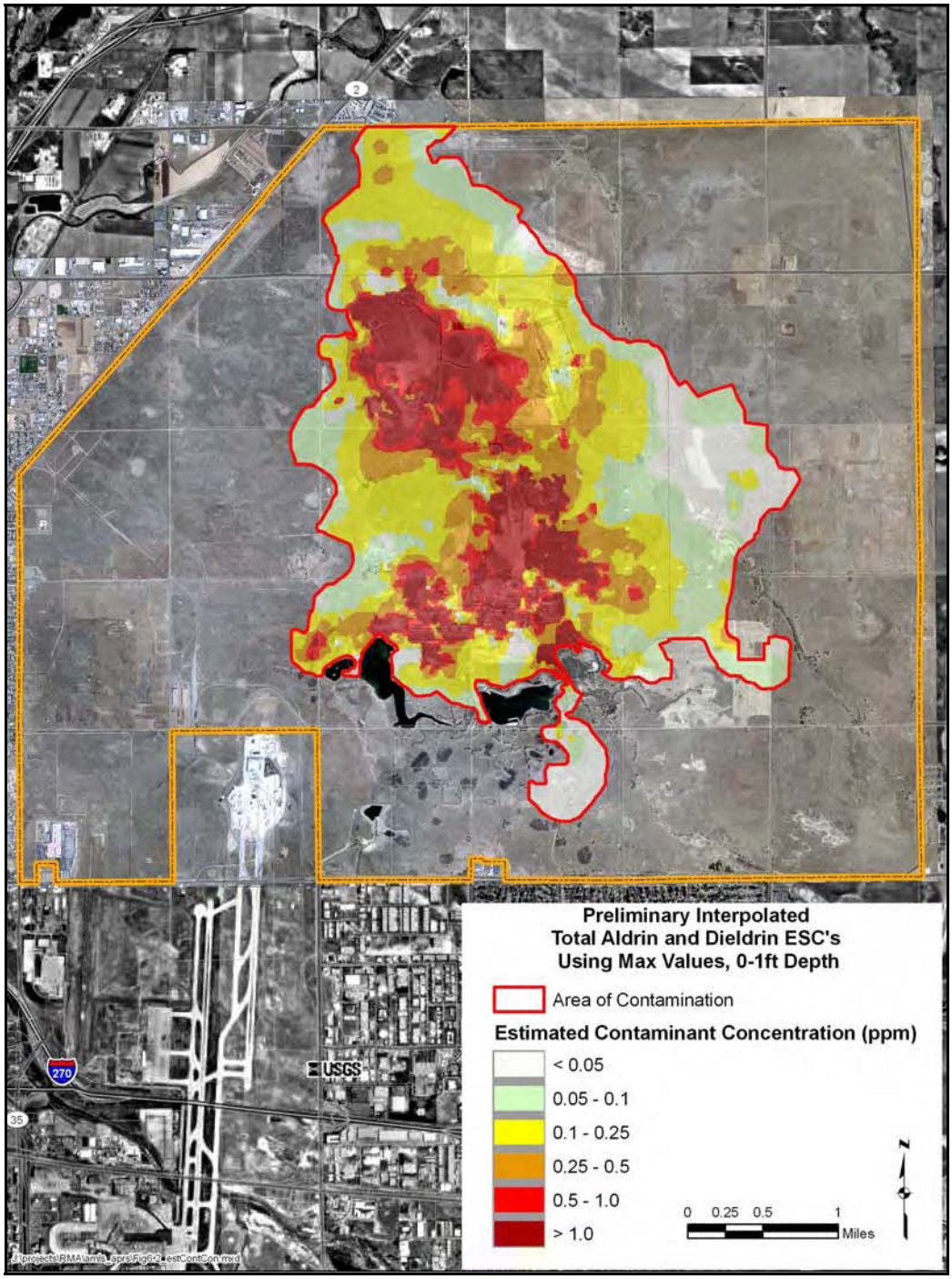

Figure 6.1. Estimated concentrations of aldrin and dieldrin in soils at the Arsenal before remediation.

Source data: BAS, 2002, Figure A1.6-2.

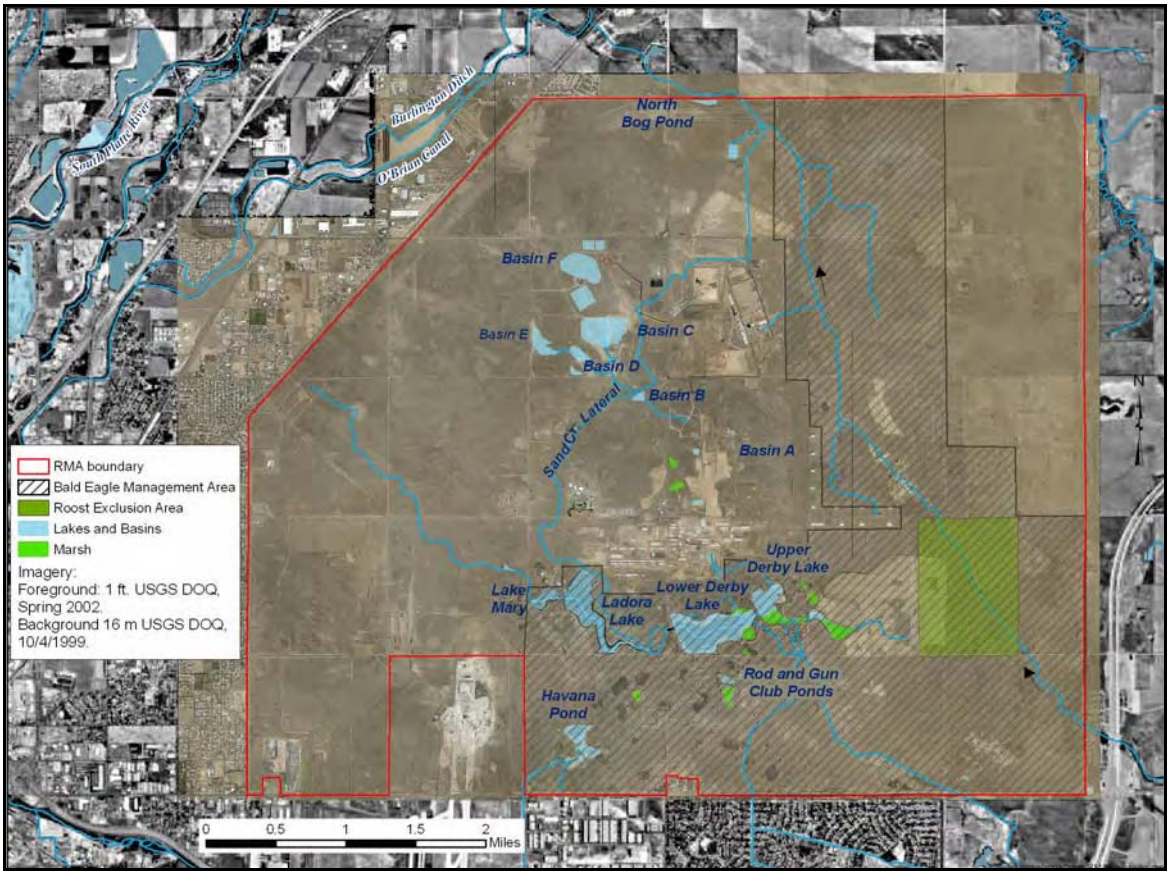

Figure 6.2. Map of the Arsenal showing major habitat features.

Table 6.1. List of habitat types at the Arsenal with associated acreages and percent of total area

Habitat type	Acres	Percent of site
Weedy forbs/grasses	10,002.1	58.9%
Native perennial grasses	4,032.3	23.8%
Shrubland/succulents	1,030.5	6.1%
Disturbed	458.2	2.7%
Wetlands	434.7	2.6%
Unclassified (e.g., Section 9 runway)	356.5	2.1%
Riverine/riparian	258.5	1.5%
Upland trees	240.6	1.4%
Lacustrine	157.9	0.9%
Total	**16,971.3**	**100%**

Source: Adapted from USFWS (1999).

6.2.1 Upland prairie

The upland prairie at the Arsenal includes areas classified as weedy forbs and grasses, native perennial grasses, and shrubland/succulents. Resident or migrant species found in upland prairie habitat at the Arsenal include a wide variety of birds, reptiles, and mammals (Table 6.2).

Table 6.2. Examples of species found in prairie habitat at the Arsenal

Wildlife category	Functional group	Species
Birds	Raptors	Bald eagle, sharp-shinned hawk, Cooper's hawk, Swainson's hawk, red-tailed hawk, ferruginous hawk, golden eagle, American kestrel, merlin, prairie falcon
	Breeding songbirds	Western meadowlark, horned lark, grasshopper sparrow, vesper sparrow, lark sparrow, lark bunting
	Game birds	Pheasant, mourning dove
	Migrants	Brewer's sparrow, clay-colored sparrow, Cassin's sparrow, chestnut-collared longspur
Reptiles	Snakes	Bull snake, rattlesnake, hognose snake
Mammals	Rabbits and hares	Eastern cottontail, desert cottontail, black-tailed jackrabbit
	Rodents	Black-tailed prairie dog, Northern pocket gopher, many species of mice and voles
	Carnivores	Coyote, red fox, least shrew, badger
	Ruminants	Mule deer, white-tailed deer

Sources: Morrison-Knudsen Environmental Services, 1989b; Denver Audubon Society, 1994; USFWS, 1994a, 1994b.

6.2.2 Perennial and intermittent surface water

Both perennial and intermittent bodies of water exist on or flow across the Arsenal (Figure 6.2). The Lower Lakes at the Arsenal (also called the "south lakes") are man-made: they include Lakes Mary, Ladora, Upper Derby, Lower Derby, and Rod and Gun Club Pond(s). Two natural ponds are also found on the Arsenal: North Bog Pond is on the northern edge of the Arsenal, and Havana Pond is on the southern edge. First Creek is the only natural stream at the site, but many man-made canals, including the Highline Lateral, Sand Creek Lateral, Uvalda Interceptor, and Havana Interceptor, also transported water at the site. First Creek is 5.9 miles long and is semi-perennial, with 39 acres of associated wetlands. It flows during the majority of the year in non-drought years (Ebasco Services et al., 1989), and discharges to O'Brian Canal approximately one-half mile north of the site. The creek and its associated wetland area pre-date the Arsenal. The size of the wetland and open water areas fluctuates based on hydrologic conditions and on manipulations by site personnel. Some of the wetland areas are ephemeral, particularly in dry years.

The water bodies on the Arsenal currently support a variety of organisms (Table 6.3). Phytoplankton, micro- and macro-zooplankton communities, and other aquatic plants known as macrophytes, as well as invertebrates, fish, and birds, are found in the Lower Lakes (BAS, 2003). Aquatic plants help provide habitat for aquatic invertebrates, fish, and water birds. Aquatic invertebrates are an important food source for fish, amphibians, reptiles, and birds. Fish populations at the Arsenal are primarily maintained by USFWS stocking from off-site hatcheries, although some species are able to reproduce. Reptiles, amphibians, waterfowl, and wading birds also make use of surface water resources at the Arsenal.

Table 6.3. Examples of species found in perennial and intermittent surface water at the Arsenal

Organism category	Species
Aquatic plants	American pondweed, leafy pondweed, sago pondweed, water-milfoil, watercress, water plantain, arrowhead, sedges, rushes, cattail, coontail
Aquatic invertebrates	Snails, dragonflies, damselflies, midges, crayfish
Fish	Rainbow trout, largemouth bass, smallmouth bass, northern pike, black crappie, bluegill, channel catfish, black bullhead, green sunfish, carp, yellow perch
Reptiles	Bullsnake, western hognose snake, common garter snake, western terrestrial garter snake, yellow-bellied racer, plains garter snake, rattlesnake, lesser earless lizard, short-horned lizards, many-lined skink
Birds – waterfowl	Canada goose, mallard, gadwall, blue-winged and green-winged teal, pintail, wigeon, shoveler, redhead, canvasback, ring-necked duck, lesser scaup, common goldeneye, bufflehead
Birds – wading birds	Great blue herons, black-crowned night herons, white pelican
Amphibians	Tiger salamander, plains spadefoot toad, Woodhouse's toad, striped chorus frog, bullfrog, northern leopard frog
Sources: Morrison-Knudsen Environmental Services, 1989a, 1989b; USFWS, 1994a; BAS, 2003.	

6.2.3 Wetlands, riparian woodland, and upland trees

For the purpose of this Assessment Plan, wetlands and riparian woodland habitat are distinguished from surface water areas. Substantial overlap of wildlife species is expected between the two habitat types because fluctuations in surface water levels can result in areas classified as surface water taking on the characteristics of wetlands. The wetlands and riparian woodland habitats currently support a variety of resident and migrant semi-aquatic and terrestrial organisms (Table 6.4). Migrant diversity through woodlands and upland groves is high: 33 migrant bird species have been noted, with yellow-rumped and yellow warblers, house wrens, and chipping sparrows the most common (Morrison-Knudsen Environmental Services, 1989b). Riparian woodland habitat includes large galleries of cottonwoods along intermittent stream channels and ditches. This habitat is somewhat rare in arid prairie environments and is very valuable for the over-wintering bald eagles that use the trees as roosts (USFWS, 1992).

Table 6.4. Examples of species found in wetlands, riparian woodland, and upland tree habitat at the Arsenal

Wildlife category	Habitat	Species
Birds	Wetlands	White pelican, double-crested cormorant, avocet, killdeer, sandpiper, white-faced ibis, migrant rail, migrant Wilson's phalarope, great blue heron, black-crowned night-heron, bobolink, marsh wren sandpiper, herring gull, ring-billed gull, Franklin's gull
	Marshes and wet meadows	Common yellow throat, red-winged blackbird, yellow-headed blackbird, song sparrow, grebe, American coot, common snipe, Virginia and sora rail, Canada goose, and a variety of ducks
	Riparian woodlands and upland groves	House wren, yellow-rumped and yellow warbler, chipping sparrow, American goldfinch, yellow-billed cuckoo, common nighthawk, downy woodpecker, western wood-peewee, violet-green swallow, blue jay, black-capped chickadee, gray catbird, red-eyed vireo, warbling vireo, black-headed grosbeak, blue grosbeak, indigo bunting, lazuli bunting, rufous-sided towhee
	Upland groves	Northern flicker, western kingbird, eastern kingbird, black-billed magpie, American robin, northern mockingbird, loggerhead shrike, lark sparrow, starling, Brewer's blackbird, common grackle, northern oriole, lesser goldfinch, house finch, house sparrow
Amphibians	Wetlands, floodplains	Northern chorus frog, great plains toad
Mammals	Riparian woodlands	Eastern cottontail, white-tailed deer, red fox, raccoon, fox squirrel, beaver, muskrat, badger, mink, and weasel
Reptiles	Wetlands and moist areas	Common garter snake, western terrestrial garter snake, western box turtle, racer snake, common and plains garter snake
Sources: Morrison-Knudsen Environmental Services, 1989b; USFWS, 1992		

In addition, a small area of upland trees (241 acres; 1.4%) was planted at the Arsenal by settlers. A number of bird species nest in this habitat (Table 6.4).

6.3 Injury Definitions

DOI regulations state that "an injury to a biological resource has resulted from the . . . release of a hazardous substance if the concentration of the substance is sufficient to:

- Cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 CFR § 11.62(f)(1)(i)]

- Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 USC 342, in edible portions of organisms [43 CFR § 11.62(f)(1)(ii)]

- Exceed levels for which an appropriate State health agency has issued directives to limit or ban consumption of such organism [43 CFR § 11.62(f)(1)(iii)].

The DOI regulations then go on to state that an injury can be demonstrated "if the biological response under consideration can satisfy all of the following acceptance criteria" [43 CFR § 11.62(f)(2)]. These criteria are:

- The biological response is often the result of exposure to . . . hazardous substances . . . [43 CFR § 11.62(f)(2)(i)]

- Exposure to . . . hazardous substances is known to cause this biological response in free-ranging organisms . . . [43 CFR § 11.62(f)(2)(ii)]

- Exposure to . . . hazardous substances is known to cause this biological response in controlled experiments . . . [43 CFR § 11.62(f)(2)(iii)]

- The biological response measurement is practical to perform and produces scientifically valid results . . . [43 CFR § 11.62(f)(2)(iv)].

In addition, DOI regulations list a number of biological responses that satisfy the criteria stated above. For the injury category of death, "biological responses" that meet the criteria for demonstrating that death resulted from exposure to hazardous substances include [43 CFR § 11.62(f)(4)(i)]:

- Brain cholinesterase (ChE) enzyme activity that has been inhibited by at least 50% compared to the mean for normal brain ChE activity for the wildlife species

- Wildlife kill investigations that indicate increased number of dead or dying birds or mammals

- Laboratory or field toxicity testing that reveals increased mortality.

For the injury category of behavioral abnormalities, the relevant biological response that has been found to meet the criteria is [43 CFR § 11.62(f)(4)(iii)]:

- Increased clinical behavioral signs of toxicity in the exposed population.

For the injury category of physiological malfunctions, the biological responses that have been found to meet the criteria include [43 CFR § 11.62(f)(4)(v)]:

- Eggshell thinning resulting from the adult bird having assimilated the hazardous substance
- Reduced avian reproduction
- ChE enzyme inhibition
- Delta-aminolevulinic acid dehydratase (ALAD) inhibition.

For the injury category of physical deformation, the biological responses that have been found to meet the criteria include [43 CFR § 11.62(f)(4)(vi)]:

- Overt external malformations
- Skeletal deformities
- Internal whole organ and soft tissue malformation
- Histopathological lesions.

In addition, according to DOI regulations, "injuries that are reasonably unavoidable as a result of response actions taken or anticipated" at a site are natural resource injuries for which damages can be recovered [43 CFR § 11.15(2)(1)]. Chapter 3 discussed response actions and remediation activities at the Arsenal. Section 6.5.4 provides a preliminary analysis of how specific response actions and remediation activities at the Arsenal have injured biological resources.

6.4 Baseline

For the purposes of defining injuries to biological resources, the baseline condition for the Arsenal is assumed to be a clean Army facility converted to a Wildlife Refuge. It is assumed the Refuge would have been created regardless of whether hazardous substances were released. Because baseline is assumed to be the Arsenal facility absent the releases of hazardous substances, impacts to wildlife from facilities and infrastructure on-site will not be included as part of the biological resource injury assessments. Only wildlife and habitat injuries resulting from the releases of hazardous substances and responses to those releases will be quantified. This assumption recognizes that the Arsenal was an industrial facility developed in a location with a history of agricultural use. Infrastructure such as buildings and roads reduced or eliminated wildlife habitat regardless of hazardous substance releases, while other infrastructure

such as surface water impoundments may have increased wildlife diversity. Under baseline conditions, it is assumed that this infrastructure would have existed with no releases of hazardous substances.

To evaluate injuries to wildlife from the hazardous substance releases, the Trustees may compare Arsenal data to data from control sites that represent baseline conditions. For example, evaluations of body burdens of contaminants in organisms might take into account background levels of contamination found in organisms at uncontaminated sites. Baseline conditions are discussed in more detail in subsequent sections.

6.5 Approaches for Determining Injury

The State has identified existing data that can be evaluated for each of the main categories of injury: exceedence of federal action levels for contaminants in edible portions of organisms, exceedence of levels that trigger consumption advisories, and exceedence of levels sufficient to cause adverse changes in viability, including wildlife kills. The Trustees included evidence of injury for biological resources in perennial and intermittent surface water. Each category is discussed further below.

6.5.1 Exceedence of FDA action levels in organisms

Injuries to wildlife occur when concentrations of hazardous substances in edible portions of an organism exceed FDA action or tolerance levels for safe consumption. Although the FDA exerts authority over domesticated poultry and not over wild gamebirds, exceedences of action levels for poultry are indicative of injuries to gamebirds.

Concentrations of dieldrin found in four different species of commonly consumed gamebirds at the Arsenal have exceeded the FDA action level of 0.3 milligrams per kilogram (mg/kg) for dieldrin residues in fatty tissue (Food Safety and Inspection Service, 1998) (Table 6.5). Moreover, the data shown in Table 6.5 were from carcasses (whole body) and muscle tissues. These concentrations would likely underestimate the concentrations of dieldrin in fatty tissue, which tends to have the greatest concentration of assimilated pesticide.

In addition, dieldrin concentrations in five largemouth bass samples from Lower Derby Lake in 1988 ranged from 0.067 to 0.644, with a mean concentration of 0.375 mg/kg (Environmental Science and Engineering, 1989, Table 4.3-5). This mean concentration exceeds the FDA action level of 0.3 mg/kg dieldrin for human consumption of fish (FDA, 2000).

Table 6.5. Examples of dieldrin concentrations in gamebirds at the Arsenal in excess of the FDA level of 0.3 mg/kg for poultry

Species	Tissue type	Maximum concentration (mg/kg)
Mallard	Adult carcass	4.53
Mallard	Juvenile carcass	0.52
Ring-necked pheasant	Juvenile carcass	1.33
Ring-necked pheasant	Adult carcass	2.92
Redhead	Muscle	0.32
American coot	Muscle	1.77

Source: Environmental Science and Engineering, 1989, Tables 4.3-1 and 4.3-2.

6.5.2 Exceedence of levels sufficient to trigger consumption advisories

Concentrations of hazardous substances in fish and wildlife at the Arsenal are sufficient to have triggered a ban on consumption. Elevated concentrations of pesticides in fish at the Arsenal led to the imposition of a catch-and-release policy in 1978, and a consumption ban for fish from the Arsenal lakes in 1984 (BAS, 2003). Consumption of all fish and wildlife was prohibited in the 1989 Federal Facilities Agreement, and these prohibitions were subsequently incorporated into the Arsenal National Wildlife Refuge Act of 1992. These bans indicate that fish resources and wildlife resources at the Arsenal have been injured by exposure to hazardous substances released at the site.

6.5.3 Adverse changes in viability

Concentrations of hazardous substances in biological resources have been sufficient to cause adverse changes in the viability of the organisms. In addition, extensive wildlife kills have been documented at the Arsenal. The State Trustees conducted a preliminary evaluation of these biological injuries by (1) collecting evidence related to wildlife kills, and (2) comparing the estimated exposure of wildlife at the Arsenal to toxicity benchmarks and injury thresholds for organochlorine pesticides.

This section is organized as follows: First, background is provided on the toxic effects of organochlorine pesticides. Next, available information on wildlife kills is described. Finally, toxicity benchmarks, injury thresholds, and exposure modeling are discussed and a comparison is made between estimated exposure levels and potential injury thresholds.

6.5.3.1 Toxicity of organochlorine pesticides

Many of the injuries to wildlife at the Arsenal have been caused by exposure to organochlorine pesticides, particularly the class of pesticides known as cyclodienes. Cyclodiene pesticides include aldrin, dieldrin, chlordane, endrin, and isodrin. Other injuries likely have been caused by pesticides from the class known as dichlorodiphenylethanes. This class includes DDT and associated metabolites DDE and dichlorodiphenyldichloroethane (DDD). Both classes of pesticides affect the neurological system of organisms by interfering with ion movements across nerve cell membranes (ATSDR, 1993, 1994, 1996, 2002). In birds and mammals, symptoms of toxicity include rigid paralysis, convulsions, respiratory failure and death. Emaciation is another characteristic symptom of organochlorine poisoning.

Over time, organochlorine pesticides have the potential to accumulate in fat deposits within a living organism and eventually reach toxic levels. These chemicals also tend to “biomagnify” up the food web, meaning that a top carnivore, for example, will accumulate the pesticide present in prey items. Therefore, lower concentrations of pesticides in prey items can become magnified into high concentrations in top predators. In addition, when organisms mobilize their fat stores during times of stress such as migration, they can be poisoned by the pesticides that are released into their circulatory system, even if the actual consumption of the pesticides happened days, weeks, or months previously (e.g., Henriksen et al., 1996).

The organochlorine pesticides discussed here all affect the neurological system in their toxic mode of action; thus the toxicity of multiple compounds may be additive. Additive toxicity means that the toxicity of a mixture of compounds will be approximately equal to the sum of the individual toxicities for each chemical present in the mixture. For example, the literature reports field collections of 425 birds killed by mixtures of dieldrin and chlordane at levels below the demonstrated lethality of either compound (Stansley and Roscoe, 1999). Additive toxicity in bobwhite quail also was reported based on feeding trials with chlordane and endrin (Ludke, 1976). An approach to evaluate the toxicity of compound mixtures is particularly important at the Arsenal, given the presence of multiple organochlorine pesticides that have been produced and identified at the site.

6.5.3.2 Evidence of wildlife kills

Wildlife mortality from chemical poisoning at the Arsenal was reported by the USFWS as early as 1951 (Finley, 1959), and continued at least through 1999 (USFWS, 2000). This mortality is consistent with numerous published accounts that describe poisoning of birds and other wildlife from agricultural use of aldrin and dieldrin. The introduction in 1956 of aldrin and dieldrin in the United Kingdom as seed treatments resulted in immediate poisonings of birds, particularly wood pigeons (*Columba livia*) and pheasants (*Phasianus colchicus*) (Peakall, 1996). Those poisonings

involved hundreds of incidents a year and thousands of individual birds. The deaths continued unabated until use of dieldrin as a wheat seed treatment was discontinued in 1975. At the Arsenal, other chemicals in addition to aldrin and dieldrin would have contributed to mortality.

The following sections give a brief overview of some of the reports of fish and wildlife mortality This is not a comprehensive review of all mortality data from the site.

1950s–1980

These injuries predate the enactment of CERCLA in 1981 and will not be included in the injury quantification for the site. However, they provide evidence of pesticide poisonings at the Arsenal.

- An estimated 1,200 ducks died in the spring of 1952 at the Lower Lakes. The USFWS reported that "experiments indicated that the cause of death was a toxic agent or agents carried on the surface of the water and probably entering the lakes through the process-water drain from the chemical plant area" (Finley, 1959, p. 1).

- In April 1959, 119 dead birds and animals were counted on a single day around the shore of Lake Ladora. An interview with a Shell employee revealed that he had gathered approximately 500 dead ducks for burial during the first three months of 1959 (Finley, 1959, p. 3).

- The USFWS stated that 2,000 ducks would be a conservative annual estimate of duck mortality in the Lower Lakes area, with 20,000 or more ducks dying over a 10-year period (Finley, 1959). The report noted that high wildlife mortality occurred at the lakes when extensive mud flats were exposed. In addition, the USFWS reported that Upper Derby, Lower Derby, and Ladora Lakes did not support fish, amphibians, or aquatic insects.

- At the Lower Lakes, more than 100 ducks were found dead on March 28, 1962, and 163 waterfowl deaths were reported between January and May 1966 (Environmental Science and Engineering, 1989, Table 1.3-1).

- In 1964, the Army removed contaminated sediments from Upper and Lower Derby Lakes and Lake Ladora, and waterfowl mortality declined from previous years. According to the Biota Remedial Investigation, "In subsequent years waterfowl and other wildlife continued to be found dead at the Lower Lakes but in smaller numbers" (Environmental Science and Engineering, 1989, p. 4-7).

- In a two-week period in early April 1973, approximately 750 dead ducks and grebes were collected from the area of Basin F in three visits, with several hundred additional carcasses observed on June 13 and 14, 1973 (Ward and Gauthier, 1973). The death of 136 ducks occurred in April 1973 at Basin C (Environmental Science and Engineering, 1989, Table 1.3-1).

- During two days in May 1975, 291 bird carcasses were removed from the shoreline of Basin F, including waterfowl, raptors, pheasants, and songbirds (Environmental Science and Engineering, 1989, Table 1.3-1).

- In June 1976, a die-off of juvenile starlings was noted at a roosting location on the Arsenal, outside of the contaminated basins area (Olds, 1976).

- Deaths of a great horned owl, five hawks, a coyote, and starlings were reported between 1976 and 1979 (Environmental Science and Engineering, 1989, Table 1.3-1).

- On May 7, 1980, the DOW, accompanied by the USFWS, inspected the perimeter of Basin F and found 389 wildlife carcasses, including 344 waterfowl, 40 birds other than waterfowl, and 5 small mammals (Seidel, 1980).

- An additional 49 waterfowl carcasses were collected at Basin F between October and December 1980 (Environmental Science and Engineering, 1989, Table 1.3-1).

In addition to waterfowl deaths, fish kills and amphibian deaths have been reported at the Lower Lakes:

- An unpublished USFWS report found an absence of frog choruses, egg masses, and tadpoles at the Lower Lakes in 1960 (USFWS, 1961).

- Stocking of channel catfish, bluegill, and northern pike in Lake Ladora in 1967 and 1968 was unsuccessful: only a single fish was caught in three 48-hour gill net attempts in 1968 (BAS, 2003).

- Catfish stocking in Lower Derby Lake in 1968 also was unsuccessful. A fish kill of largemouth bass, bluegill, and catfish was noted on May 16, 1973, following the release of aldrin into lake waters (Environmental Science and Engineering, 1989, Table 1.3-1).

- Death of rainbow trout in the Lower Lakes was noted on April 18, 1977 (Environmental Science and Engineering, 1989, Table 1.3-1).

Bird hazing devices, including flashing light pontoons, repeating-fire "Zon" guns, and "Avalarms," were installed around Basin F in 1975 in an attempt to reduce bird mortality (Environmental Science and Engineering, 1989, p. 4-4). In 1980, these devices were found to be inoperable during field inspections by the USFWS. In addition, USFWS personnel observed waterfowl landing and taking off from Basin F even when the devices were operating (Grieb, 1981).

1981–1988

Substantial wildlife mortality at the Arsenal continued in the 1981–1988 time period. Regular quarterly waterfowl mortality counts were conducted at Basin F from 1981 to 1987, and between 139 and 444 dead birds were found each year (Table 6.6). An IRA at Basin F in 1988, which moved contaminated liquids to storage tanks and lined holding basins, and covered the basin site with a clay cap, ended direct waterfowl mortality from exposure to contaminated liquid waste at Basin F.

Table 6.6. Reported waterfowl mortalities at Basin F (1981–1987)

Year	Number found dead
1981	202
1982	222
1983	444
1984	418
1985	140
1986	236
1987	139
Total	1,801

Source: Environmental Science and Engineering, 1989, Table 4.1-1.

1989–1999

Between 1989 and 1999, wildlife mortality data at the Arsenal were gathered through three programs: the fortuitous specimen program, the Building 111 program, and the avian mortality program. These sampling and collection programs indicate that substantial wildlife mortality continued at the Arsenal through 1999.

- From 1989 to 1993, 192 bird samples and 52 mammal samples were collected at the Arsenal by the USFWS as "fortuitous specimens" because the animals were either dead or dying (CDPHE, 1994). Bird species collected represented more than 30 different species, including raptors, waterfowl, and passerines.
- The 1999 USFWS Annual Progress Report for the Rocky Mountain Arsenal (USFWS, 2000) provides a cumulative list of the bird species for which mortalities were attributed to dieldrin or endrin poisoning between 1990 and 1998. A total of 102 bird mortalities in 19 species were attributed to dieldrin or endrin poisoning (USFWS, 2000, Table 1.8). The report also noted that "several birds had pronounced keels and displayed other symptoms of dieldrin poisoning" (USFWS, 2000, p. 28). A pronounced keel indicates emaciation.

These mortality reports are likely to underestimate substantially the total amount of wildlife mortality at the site caused by pesticide poisoning. For example, a bird that died from a collision with a building or power line would not be attributed to pesticide poisoning, even if exposure to pesticides decreased the bird's ability to avoid the obstacles. Interestingly, the USFWS reported that more birds were found dead at the Arsenal after periods of cold or foul weather, and dead birds were often emaciated (USFWS, 1997). These observations are consistent with the biological mechanisms and toxicokinetics of organochlorine pesticides.

The evidence presented above indicates that organochlorine pesticides have been the causative agent in wildlife mortalities from the mid-1950s through at least 1999.

6.5.3.3 Concentrations of pesticides in animal tissues above mortality thresholds at the Arsenal

In 1994, the USFWS developed guidelines for diagnosing contaminant-related deaths of birds at the Arsenal (USFWS, 1995, 1996, p. 1-35). Under these guidelines, mortality or morbidity of birds with brain levels of dieldrin greater than 9 mg/kg was attributed to dieldrin poisoning, while birds with clinical signs diagnostic of dieldrin poisoning, or with supporting necropsy data and brain dieldrin levels between 5 and 9 mg/kg, were considered evidence of suspected dieldrin poisoning. Levels of brain dieldrin of 1–5 mg/kg indicate a dangerous level of exposure, but it is likely that other factors contributed to or caused the death of the bird. Endrin was considered to be lethal at 0.8 mg/kg in the brain (USFWS, 1998, p. 6). The USFWS also appeared to apply these same diagnostic criteria to other animals collected through the fortuitous specimen program (USFWS, 1998).

As discussed previously, the number of mortalities attributed to dieldrin or endrin poisoning based on the USFWS guidelines is likely to be a substantial underestimate of the total number of mortalities resulting from pesticide exposure because of the additive toxicity of related organochlorine pesticides, among other reasons.

Tissue data available for dead or dying birds collected since 1955 indicate that bird tissue concentrations were high enough to cause or contribute to mortality.

- Chemical analysis in 1955 of a dead duck from the Lower Lakes revealed 261 mg/kg dieldrin in fat tissue and 32.7 mg/kg dieldrin in the liver (Jensen, 1955, p. 3).
- Chemical analysis of three ducks in 1959 found dieldrin concentrations between 30 and 64 mg/kg (Finley, 1959, Table 1).
- In 1982, 1 great blue heron, 1 black-billed magpie, 1 European starling, and 2 Brewer's blackbirds had dieldrin concentrations in brain tissue above 5 mg/kg, with additional specimens having brain concentrations between 1 and 5 mg/kg (McEwen, 1983).
- Between 1986 and 1988, contaminant analysis was completed for 5 ferruginous hawks, 3 red-tailed hawks, 4 great-horned owls, 2 golden eagles, and 2 mourning doves found dead (Environmental Science and Engineering, 1989, p. 4-37, p. 4-50). Contaminant levels in brain tissues of raptors ranged from < 0.175 to 15.6 mg/kg dieldrin and 0.475 to 10.3 mg/kg DDE. Both golden eagles and one ferruginous hawk had brain tissue dieldrin levels below detection. One additional ferruginous hawk had brain tissue dieldrin of less than 1 mg/kg. Two ferruginous hawks had brain tissue dieldrin levels between 5 and 9 mg/kg, and one ferruginous hawk, two red-tailed hawks, and two great horned owls had brain tissue dieldrin levels of more than 9 mg/kg. Brain tissue dieldrin was not reported individually for the remaining great horned owl and red-tailed hawk, but summary statistics indicate that one of these two samples had brain tissue dieldrin of 15.6 mg/kg.

Because many of the samples of dead wildlife were collected opportunistically, the actual wildlife mortality at the site is likely to have been substantially greater than the number reported. Additionally, the USFWS noted in 1997 that:

> …more birds are likely dying than what is found by the Service. Telemetry data in 1994 showed that some poisoned birds die in areas where they are unlikely to be found and not necessarily near contaminated areas. In 1996, a magpie that died from endrin poisoning was found in Section 10 on the abandoned Stapleton runway, a fair distance from contaminated soil areas. Birds that could not be evaluated due to decomposition may also have died from poisoning (USFWS, 1997, p. 53).

In addition to bird kills, evidence exists that pesticide poisoning resulted in the death of mammals at the Arsenal between 1989 and 1999. In 1997, the death of a cottontail rabbit found at Building 111 was attributed to endrin poisoning, based on a brain concentration of 1.05 mg/kg (USFWS, 1998, p. 11). Results of tissue analyses for fortuitous mammal specimens collected in

previous years indicated that mammals accumulate high concentrations of pesticides in their tissues. For example, two coyotes that were hit by a vehicle in 1994 had liver concentrations of dieldrin above 4 mg/kg (USFWS, 1996, Table 1-9). A badger collected in 1992 had a brain concentration of 2.46 mg/kg dieldrin and a liver concentration of 9.65 mg/kg, but the official cause of death reported on the necropsy was "undetermined" (USFWS, 1995, Tables 1-16, 1-17).

All of this information indicates that wildlife deaths have occurred at the Arsenal as a result of exposure to hazardous substances, including, but not limited to, aldrin and dieldrin. The anecdotal nature of the sampling likely substantially underestimates the actual wildlife deaths caused by pesticide poisoning on-site. Additional investigation of wildlife deaths will be undertaken as part of the State Trustees' assessment activities.

6.5.3.4 Historical benchmarks and potential injury thresholds for pesticide exposure

As discussed above, concentrations of pesticides in animal tissues above injury benchmarks can indicate that mortality of a specimen was caused by exposure to pesticides. It is also possible to develop injury thresholds based on exposure to pesticides in an animal's diet (or through dermal absorption or inhalation).

Mortality caused by short-term exposure to a hazardous substance is considered "acute toxicity." An acute oral dose, typically measured as a 50% Lethal Dose (LD_{50}), is the chemical dose sufficient to cause mortality in 50% of test animals in a laboratory study. Chemical doses are measured as mg of chemical per kg of body weight of the animal. When something is highly toxic, it means that ingestion of a small amount of the chemical will cause toxicity. Therefore, chemicals with higher toxicity have lower LD_{50} values – a lower LD_{50} value means that it takes a smaller amount of the chemical to cause mortality.

As shown in Table 6.7 for a variety of bird species, different pesticide compounds have different toxicities. Cyclodiene pesticides such as aldrin and dieldrin are among the most toxic organochlorine pesticides. However, aldrin is rapidly converted to dieldrin in environmental media, such as soil or water, and in the tissues of biological organisms. Therefore, aldrin is rarely detected at high concentrations. Endrin is about 10 times more toxic than dieldrin for common bird test species (Table 6.7). Other cyclodiene pesticides (chlordane, oxychlordane, heptachlor, heptachlor epoxide, and isodrin) are somewhat less toxic than dieldrin to birds (Friend and Trainer, 1974). For each compound, there also is marked variation in toxicity among bird species. For example, mallards are less sensitive to pesticides than California quail, meaning that mallards can ingest a higher dose of pesticide without being killed. The variation in toxicity among species is likely a result of differences in the birds' ability to metabolize these compounds (Ronis and Walker, 1989).

Table 6.7. Concentrations of different pesticides that cause mortality ("acute toxicity") in birds

Chemical	Test species	Number of samples	Sex	Age (months)	Acute oral dose (LD_{50} mg/kg)
Aldrin	Mallard	16	F	3–4	520
	Bobwhite	12	F	3–4	6.59
	Pheasant	12	F	3–4	16.8
Dieldrin	Mallard	12	F	6–7	381
	California quail	12	M	7	8.8
	Pheasant	9	M	10–23	79
	Rock dove	15	M, F	–	26.6
DDT	Mallard	8	F	3	> 2,240
	California quail	12	M	6	595
	Pheasant	15	F	3–4	1,334
Chlordane	Mallard	12	F	4–5	1,200
	California quail	12	M	12	14.1
	Pheasant	4	F	3	24.0–72.0
Endrin	Mallard	12	F	12	5.6
	California quail	12	F	9–10	1.2
	Pheasant	12	M	3–4	1.8
	Rock dove	16	M, F	–	2.0–5.0

Source: Hudson et al., 1984.

Low-level exposure to organochlorine pesticides (below lethal levels) has the potential to alter wildlife behavior and cause chronic health problems. Chronic low-level dosing results in a steady accumulation of dieldrin in an animal. For example, dogs fed a diet with just 0.01% dieldrin in food by weight (0.1 µg/g dieldrin) showed high concentrations of dieldrin accumulating in fat (Richardson et al., 1967). Similar results have been found in chronic dosing studies with birds. Altered behavior occurring as a result of this low-level dosing includes reduced alertness to predators, altered courtship behavior, and altered aggression (Sharma et al., 1976). Other chronic toxicity effects include increased genetic mutations, higher cancer rates, and endocrine disruption (WHO, 1989). Population effects of pesticide exposure for birds include delayed egg-laying, decreased egg production, reduced egg weights, and reduced eggshell thickness, all of which contribute to reduced hatchability and post-hatching mortality (Dahlgren et al., 1970; Sharma et al., 1976; Busbee, 1977; Newton, 1988; Walker and Newton, 1999).

The EPA (2005a) produced a comprehensive review of literature on dieldrin with the purpose of developing Ecological Soil Screening levels. EPA was particularly interested in papers reporting toxicity responses other than lethality, such as biochemical, behavioral, physiological, pathological, and reproductive impairment. These data were compiled to determine a Toxicity Reference Value (TRV) for birds. EPA defined the TRV as the "Dose above which ecologically relevant effects might occur to wildlife species following chronic dietary exposure and below which it is reasonably expected that such effects will not occur" (EPA, 2005b, p. 4-11). The TRV established by the EPA was 0.0709 mg dieldrin/kg body weight (bw)/day, based on adverse effects on reproduction.

The Rocky Mountain Arsenal Biological Advisory Subcommittee (BAS) established a TRV of 0.028 mg dieldrin/kg bw/day as an "estimated safe level" for small birds at the Arsenal. This number is lower than the TRV established by the EPA (BAS, 2002). The BAS TRV was based on a toxicity benchmark or "critical dose" of 0.28 mg dieldrin/kg bw/day from a study with homing pigeons (Robinson and Crabtree, 1969) and the application of a ten-fold uncertainty factor.

In summary, according to DOI regulations, injuries to wildlife can be evaluated based on the exposure of organisms to pesticides at levels that exceed toxicity thresholds or benchmarks. These thresholds vary by the type of pesticide, by species, and by toxicity endpoints, with sublethal effects occurring at lower exposure levels compared to lethal effects. Thresholds will be compared to Arsenal data to calculate the amount of lost ecological resources and services.

Exposure modeling

Exposure modeling is used to estimate contaminant exposures of different wildlife species, based on concentrations in environmental media such as soil and water. Measurements of pesticide concentrations in soil can be used to predict daily dietary doses of pesticides, based on assumptions about bioaccumulation of pesticides through the food web. Exposure modeling also can be used to predict tissue concentrations of pesticides in animals, based on assumptions about pesticide concentrations in the animal's diet.

More specifically, in an exposure model, a dose is calculated for each route of exposure: ingestion, inhalation, and dermal absorption. The total dose is compared to a TRV to determine whether it exceeds a safe level. All exposure routes are initially considered, but some may not be included in the exposure model if the route would not be expected to contribute very much to the overall dose. For a simple example of a dietary exposure model, the animal's average daily food intake is calculated in kilograms of food per day. Then, for each food type in the animal's diet, the animal's average daily food intake is multiplied by the fraction of the diet that each type represents. This gives the mass of each food type the animal consumes in an average day. This mass is multiplied by the concentration of pesticide in that type of food, to give a total amount of

the pesticide for that food. Finally, pesticide amounts for all foods are summed, giving a single total daily intake that is divided by the average body weight of the animal. Thus, dose is expressed as mg of pesticide per kilogram body weight per day.

This simple example of exposure modeling relies on single point estimates for average daily food intake, average food type mass, and contaminant concentration in the food source. In more complicated models, ranges and distributions of these variables are used rather than single point estimates. A computer runs multiple simulations, and the resulting dose estimate incorporates information on the uncertainty of variables included in the model.

An extensive risk assessment completed for the site (the IEA/RC; Ebasco Services et al., 1994) developed dietary exposure models for a range of organisms at the Arsenal. A refinement of this approach was subsequently presented in the Terrestrial Residual Ecological Risk (TRER) report (BAS, 2002). The exposure modeling in the TRER predicted that an average soil concentration of 0.065 mg/kg for aldrin/dieldrin across the home range of a small bird would result in a bird exposure dose of 0.028 mg aldrin/dieldrin per kg body weight per day. This exposure modeling was based on dietary intake and did not consider potential exposure through inhalation or dermal pathways, so it likely underestimates total exposure.

This exposure calculation is an example of an exposure calculation for a small bird. Similar exposure calculations can be conducted for other types of birds and wildlife to predict whether soil concentrations could result in wildlife exposure at levels exceeding injury thresholds and benchmarks for either dietary doses or tissue concentrations.

Comparison of estimated exposure to potential injury thresholds

The IEA/RC evaluated the dietary exposure of a variety of species or species groups to different chemicals, and compared exposure estimates to TRVs to quantify ecological risk (Ebasco Services et al., 1994). Comparisons were expressed as “hazard quotients,” where:

Hazard quotient = Exposure estimate / TRV.

The TRVs were developed by finding the lowest chemical dose associated with adverse effects to a particular class of organisms such as small birds, and then dividing this “critical dose” by a set of uncertainty factors to develop an estimated safe level, below which no adverse effects are expected to occur. For example, as mentioned above, the BAS established 0.028 mg dieldrin/kg bw/day as the TRV for small birds based upon a “critical dose” of 0.28 mg dieldrin/kg bw/day (Ebasco Services et al., 1994). The BAS divided the critical dose by an uncertainty factor of 5 to consider potential interspecies differences in sensitivity, and an additional uncertainty factor of 2 to account for differences between laboratory and field conditions and the potential for variability within a species (Ebasco Services et al., 1994). A calculated hazard quotient of 10 for

small birds would mean that an organism was exposed to a dose 10 times the TRV of 0.028 mg dieldrin/kg bw/day.

Figure 6.3 shows areas at the Arsenal where, prior to remedial activities, the hazard quotient for small birds exceeded 10 based on respective TRVs advocated by the different parties. Regardless of thresholds and exposure models used, the injury will be greatest in the central core of the Arsenal.

6.5.4 Injuries from response actions

Remediation work has occurred extensively across the site (Figure 6.4). Although this work has been necessary to reduce the pervasive contamination, collateral injuries to wildlife have been unavoidable. Soil remediation activities have included physically removing contaminated surficial soils using heavy equipment. Also, some areas with clean soils were excavated to use as soil covers for the landfills and in-place containment of soils. In addition, the Army, Shell, and the USFWS tilled soils for revegetation. These activities disturbed existing habitat on-site. Injuries to biological resources that would have inhabited or utilized this disturbed habitat will occur until the habitat is returned to baseline conditions.

Wildlife also has been injured by the intentional eradication of species at the Arsenal. In 1989, the Army exterminated prairie dogs in Section 36 and the southeast corner of Section 25 because high concentrations of dieldrin present in the prairie dogs were endangering wintering bald eagles that ate the prairie dogs (R.L. Stollar & Associates et al., 1992, p. IV-10).

Permanent remediation injuries to biological resources have also occurred. Specifically, biota intrusion layers are designed as part of the caps on hazardous landfills. For example, an 18-inch-thick layer consisting of crushed concrete is part of the landfill cap design. The crushed concrete layer is specially engineered to prevent burrowing animals such as prairie dogs and badgers from re-exposing buried hazardous substances (CDPHE, 1995; Foster Wheeler, 1996). While this cap is necessary to prevent additional exposure of wildlife to contaminants, the biota intrusion layer will limit habitat available to the Arsenal wildlife and constitutes a permanent injury to biological resources. Biota intrusion layers are also included in the acres of covers over contaminated South Plants soils. As part of the assessment, the State will consider these collateral injuries in addition to the habitat improvements that occur following remediation and revegetation.

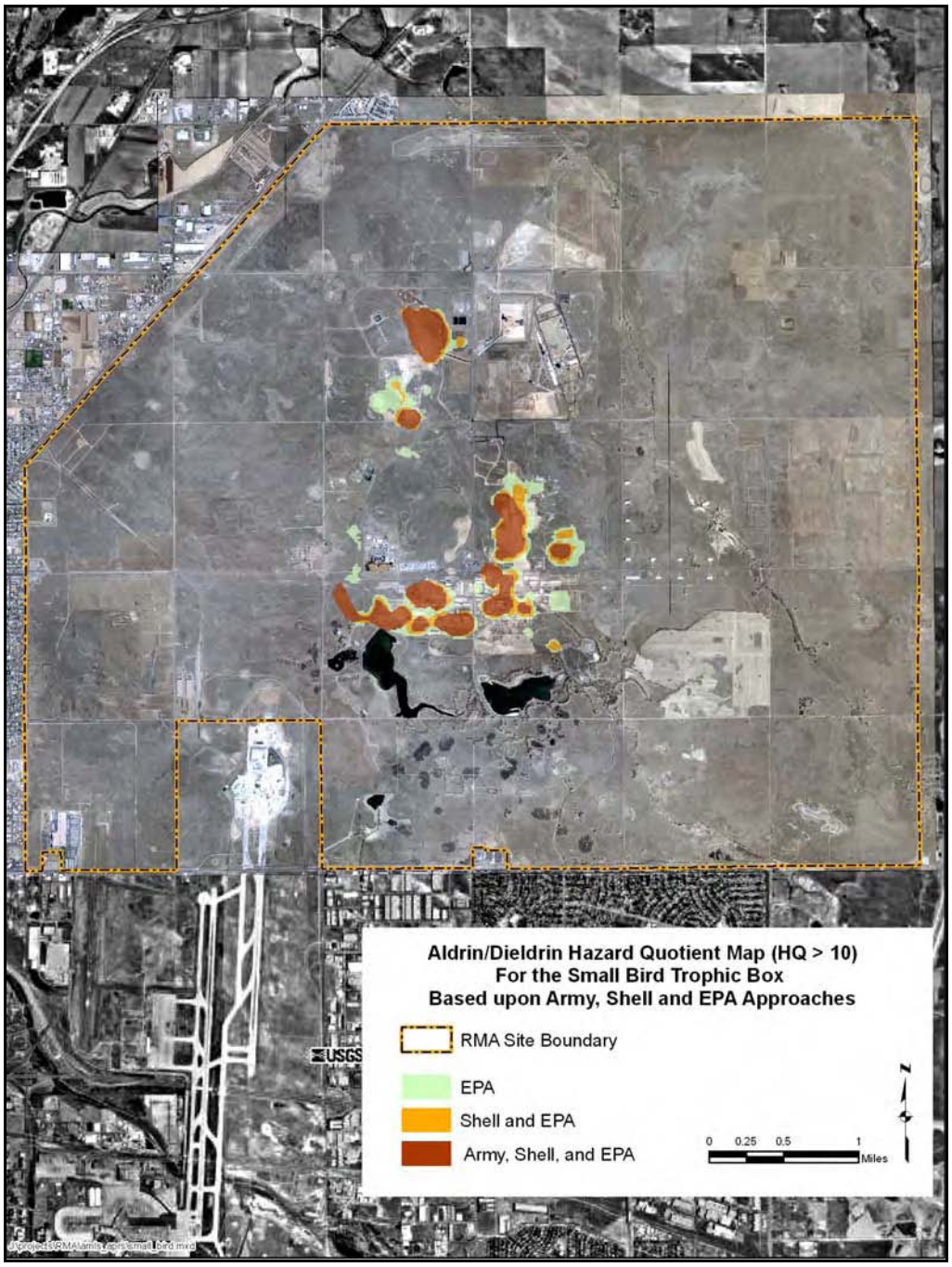

Figure 6.3. Example map of injured areas at the Arsenal for small birds exposed to dieldrin, based on data developed for the Arsenal risk characterization report.

Source: Ebasco Services et al., 1994, Figure C.3-33.

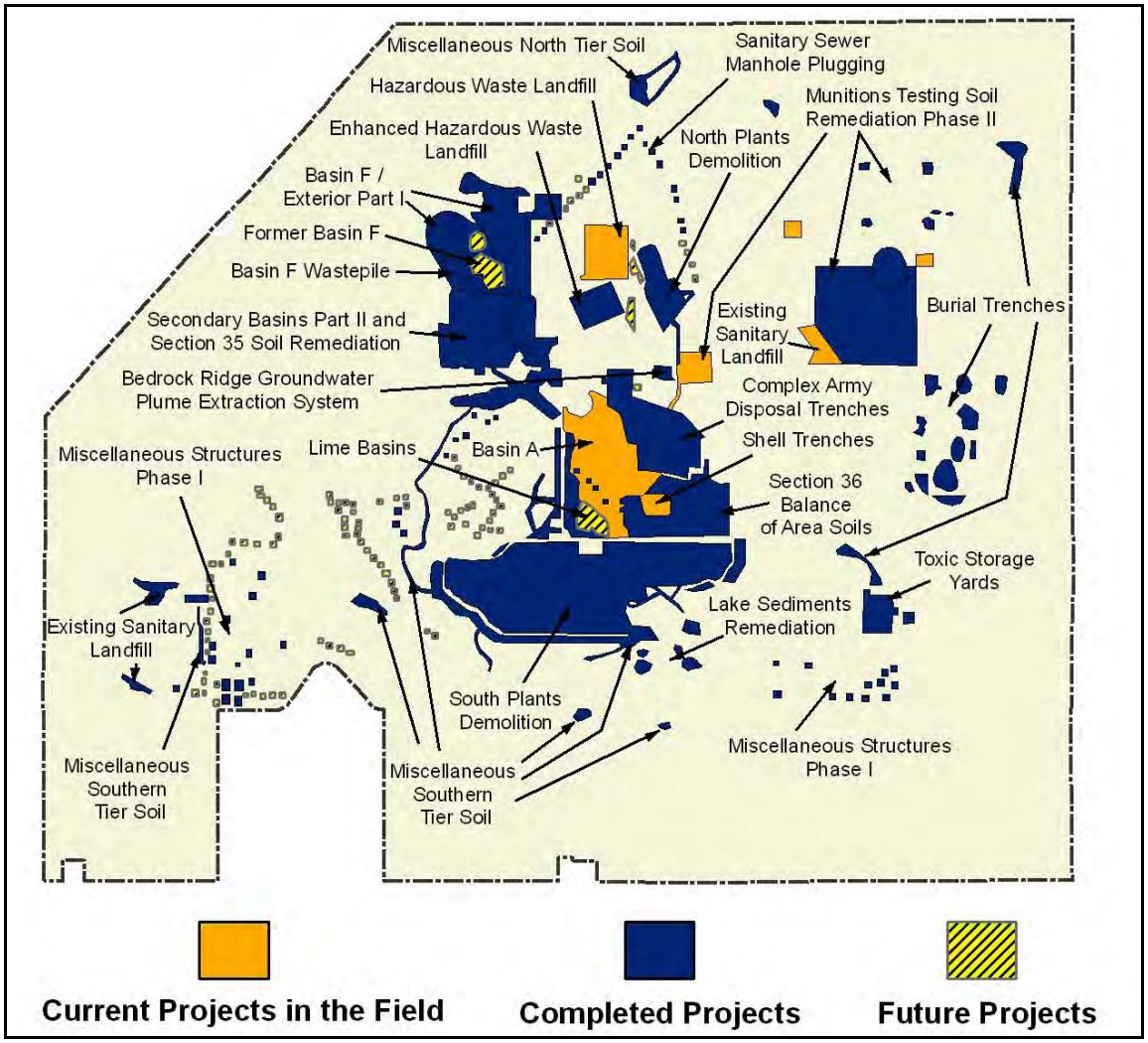

Figure 6.4. Spatial extent of soil remediation activities at the Arsenal as of September 2007.

Source: Rocky Mountain Arsenal, 2007.

6.5.5 Evidence of injury to biological resources associated with surface water

Biological resources at the Arsenal have been exposed to contaminants in surface water and associated sediments. Concentrations of contaminants that exceed established State and federal water quality criteria demonstrate potential injury to biological resources because water quality criteria are derived using exposure effects data from laboratory studies using a diverse assemblage of test organisms (Stephen et al., 1985).

Below is a brief list of water quality exceedences that have occurred in waters of the Arsenal. This is not a complete assessment, but an example using readily available data from the Arsenal reports.

- Surface water quality data for the Sand Creek Lateral drainage ditch indicated that aldrin and dieldrin were measured above respective water quality criteria between the fall of 1985 and the spring of 1987 (Ebasco Services et al., 1989, pp. 4-9–4-12)
- Chlordane was measured above the chronic criteria in Lower Derby Lake in April 1989 (Ebasco Services et al., 1994)
- Endrin was measured above the chronic criteria in Upper Derby Lake in September 1989 (Ebasco Services et al., 1994).

In addition, concentrations of contaminants in sediments have exceeded benchmark levels that indicate the probability of adverse effects on aquatic biota (Table 6.8). The sediment benchmarks that are used for comparison include consensus threshold effects concentrations (TECs), which represent concentrations below which adverse effects to sediment-associated biota would not be expected, and consensus probable effects concentrations (PECs), above which adverse effects to sediment-associated biota are likely (MacDonald et al., 2000). Additional aquatic sediment benchmarks criteria developed by the Ontario Ministry of Environment and Energy include the lowest observed effect levels, below which adverse effects would not be expected, and the severe effect levels (SEL), where “the sediment is considered heavily polluted and likely to affect the health of sediment-dwelling organisms” (Persaud et al., 1993).

6.6 Approaches to Injury Quantification

According to the DOI regulations, quantification of injuries is conducted to “quantify the effects of the discharge or release on the injured natural resources for use in determining the appropriate amount of compensation” [43 CFR § 11.70(c)]. Injuries to natural resources can cause reductions in the services those resources provide relative to baseline conditions, where baseline is defined as conditions that would have existed at the assessment area had the discharge of oil or release of the hazardous substance under investigation not occurred [43 CFR § 11.14(e)]. Services are the “physical and biological functions performed by the resource, including the human uses of those functions” [43 CFR § 11.14 (nn)].

Injury quantification involves determining the spatial extent where injuries have occurred as well as the timing and duration of injuries. Quantification also can involve estimates of the “degree of service loss” in an area – that is, what percentage of services has been lost at a site compared to baseline condition.

Table 6.8. Examples of exceedences of benchmark levels in contaminated sediments at the Arsenal

Water body	Date	Contaminant	Benchmark concentration (ppm)	Maximum sediment concentration (ppm)[a]	Benchmark type[b]
Lake Mary	1998	Arsenic	9.8	11.7	TEC
		Mercury	0.18	0.46	TEC
Lake Ladora	1995	Aldrin	0.08–0.8	0.15	SEL[c]
		Arsenic	9.8	11	TEC
		Dieldrin	0.0019	0.016	TEC
		Mercury	1.1	2.2	PEC
Lower Derby Lake	1985–1994	Aldrin	0.08–0.8	50	SEL[c]
	1995	Aldrin	0.08–0.8	0.56	SEL[c]
		Chlordane	0.018	0.103	PEC
		Dieldrin	0.062	0.063	PEC
		Mercury	1.1	3.2	PEC

ppm = parts per million.
a. All sediment data from the BAS (2003).
b. TEC = "Threshold Effects Concentration;" PEC = "Probable Effects Concentration" (both from MacDonald et al., 2000); SEL = "Severe Effects Level" from Persaud et al. (1993).
c. Severe effects levels based on a benchmark of 8 µg Aldrin/g of organic carbon. The benchmarks given here are calculated for a sediment sample with an assumed total organic carbon concentration between 1% and 10%.

To illustrate such potential quantifications, the Trustees estimated the spatial extent of injuries prior to remediation based on areas where soil concentrations of aldrin and dieldrin would result in small bird exposure that exceeds potential injury thresholds (Table 6.9). Three concentration ranges were defined based on the hazard quotient ranges for small birds in specified areas. All three ranges exceed the EPA (2005a) threshold for birds, while the > 0.65 ppm category also exceeds the critical dose threshold for small birds used in the Arsenal risk evaluation (Ebasco Services et al., 1994). Regardless of selected thresholds, the total spatial extent of injury will have decreased over time at the Arsenal, as remediation activities have cut off exposure pathways to contaminated soils and remediated areas have been revegetated.

Injury quantification also must be conducted for injuries caused by remedial activities. The USFWS estimated that 3,650 acres would be directly disturbed by remedial activities (USFWS, 1999). The duration of injury will depend on the time required to return the habitat to baseline conditions.

During the assessment process, the Trustees will conduct a full injury quantification that takes the timing and benefits of remediation into account more precisely and will also make sure that there is no "double-counting" of the same injured areas, using different injury tests.

Table 6.9. Comparison of injury thresholds for different concentrations of aldrin and dieldrin in soils at the Arsenal

Soil concentration (ppm aldrin/ dieldrin)	Acres at the Arsenal (pre-remediation)[a]	Estimated bird exposure dose (mg aldrin-dieldrin/kg bw/day)	Hazard quotient from Ebasco Services et al. (1994)	Exceeds EPA (2005a) threshold (0.0709 mg dieldrin/kg bw/day)	Exceeds the Arsenal threshold for small birds (0.28 mg dieldrin/kg bw/day)
0.130–0.325 (midpoint = 0.2275)	1,114	0.098	2–5	Yes	No
0.325–0.650 (midpoint = 0.4875)	672	0.21	5–10	Yes	No
> 0.65 (midpoint = 0.6500)	1,067	> 0.28	> 10	Yes	Yes
Total	**2,853**				

a. Acres estimated from a digitized version of Figure B-5 in BAS (2002).

6.7 Anticipated Assessment Activities

During the assessment phase of the NRDA, the Trustees will undertake activities to determine and quantify the full range of injuries to biological resources that have taken place at the Arsenal. Specific activities that the Trustees anticipate undertaking may include the following:

- **Injury determination**
  - **Revise and update bioaccumulation and dietary toxicity models.** The Trustees will review recent scientific literature and potentially expand the number of evaluated species, revise exposure scenarios, and identify additional toxicity references upon which to base injury thresholds. These may include those underlying EPA's Ecological Soil Screening Levels established for avian and mammalian herbivores, ground insectivores, and carnivores (EPA, 2005a), and any recent references regarding tissue concentrations related to adverse effects.
  - **Assess injuries to biological resources based on tissue and media concentrations.** The Trustees will assess injuries to biological resources by comparing tissue concentrations and media concentrations for different chemicals reported in the Rocky Mountain Arsenal Environmental Database to injury threshold values.

- **Address the additive toxicity of organochlorine pesticides.** The Trustees will use existing data to evaluate the potential additive toxicity of organochlorine pesticides. For example, wildlife specimens with brain concentrations of dieldrin below injury thresholds may also have had high concentrations of other pesticides that would result in an exceedence of injury thresholds when using additive toxicity models.

- **Assess injuries to reptiles, amphibians, fish, and bats.** The Trustees will assess injuries to reptiles, amphibians, fish, and bats based on a literature review to establish injury thresholds. Previous work at the Arsenal has focused on birds and ground-dwelling mammals, with little known about potential injuries to other types of wildlife.

- **Assess injuries to biological resources from exposure to metalloids.** The Trustees will assess injuries to biological resources from exposure to arsenic and other metalloids, using a literature review to establish injury thresholds. Previous work at the Arsenal has focused on organochlorine pesticides and also has evaluated risk from mercury exposure.

- **Assess injuries to biological resources associated with perennial and intermittent surface water and associated sediments.** The Trustees will assess injuries to biological resources associated with perennial and intermittent surface water and sediments, including the Lower Lakes. Trustees would use existing data on concentrations of contaminants in surface water, sediment, and biota to assess injury.

Injury quantification

- **Quantify spatial extent of injuries over time.** The Trustees will use GIS techniques to quantify the spatial extent of injury and how this injury has changed over time with remediation.

- **Quantify injuries from remediation.** The Trustees will examine information and maps of completed and anticipated remediation activities to determine the spatial extent and duration of injury resulting from loss of wildlife habitat during remediation as well as the degree of habitat improvement associated with the remediation.

- **Quantify resource losses over time.** The State Trustees would develop models to predict injury over time based on existing wildlife kill data, existing data on contaminant concentrations in environmental media, and toxicity thresholds.

References

ATSDR. 1993. Toxicological Profile for Heptachlor and Heptachlor Epoxide. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA. April.

ATSDR. 1994. Toxicological Profile for Chlordane. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA. May.

ATSDR. 1996. Toxicological Profile for Endrin. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA. August.

ATSDR. 2002. Toxicological Profile for Aldrin and Dieldrin. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA. September.

BAS. 2002. Rocky Mountain Arsenal Biological Advisory Subcommittee. Assessment of Residual Ecological Risk and Risk Management Recommendations at the Rocky Mountain Arsenal. Part I: Terrestrial Pathways and Receptors. Final Report. Prepared for the Arsenal Committee. April.

BAS. 2003. Rocky Mountain Arsenal Biological Advisory Subcommittee. Assessment of Residual Ecological Risk and Risk Management Recommendations at the Rocky Mountain Arsenal. Part II: Aquatic Pathways and Receptors. Prepared for the Arsenal Committee. April.

Busbee, E.L. 1977. The effects of dieldrin on the behaviour of young loggerhead shrikes. *The Auk* 94:28–35.

CDPHE. 1994. Response to Comments of Shell Oil Company on the Arsenal Fortuitous Specimen Collection and Necropsies Dated February 1994. Colorado Department of Public Health and Environment. August 11.

CDPHE. 1995. Equivalency Criteria for Alternative Cover – Rocky Mountain Arsenal. Colorado Department of Public Health and Environment. July 13.

Dahlgren, R.B., R.L. Linder, and K.K. Ortman. 1970. Dieldrin effects on susceptibility of penned pheasants to hand capture. *J Wildl Manage* 34(4):957–959.

Denver Audubon Society. 1994. Birds of Rocky Mountain Arsenal National Wildlife Refuge. U.S. Fish and Wildlife Service. Northern Prairie Wildlife Research Center Online, Jamestown, ND. Available: http://www.npwrc.usgs.govarsenal.htm/. Accessed 4/10/2007.

Ebasco Services, R.L. Stollar & Associates, Hunter/ESE, and Harding Lawson. 1989. Technical Support for Rocky Mountain Arsenal. Final Water Remedial Investigation Report, (Version 3.3). Prepared for U.S. Army Program Manager's Office for Rocky Mountain Arsenal Contamination Cleanup. July.

Ebasco Services, James M. Montgomery, International Dismantling & Machinery, Greystone Environmental, Hazen Research, Data Chem, BC Analytical, and Terra Technologies. 1994. Technical Support for Rocky Mountain Arsenal. Final Integrated Endangerment Assessment/ Risk Characterization. Version 4.2. Prepared for U.S. Army Program Manager's Office for the Rocky Mountain Arsenal. July.

Environmental Science and Engineering. 1989. Biota Remedial Investigation Final Report. Prepared for Office of the Program Manager, Rocky Mountain Arsenal Contamination Cleanup. May.

EPA. 2005a. Ecological Soil Screening Levels for Dieldrin. Interim Final.

EPA. 2005b. Guidance for Developing Ecological Soil Screening Levels. OSWER Directive 9285.7-55. November 2003. Revised February 2005.

FDA. 2000. Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed. U.S. Food and Drug Administration Industry Activities Staff Booklet. Available: http://www.cfsan.fda.gov/~lrd/fdaact.html#aldr. Accessed 8/3/2007.

Finley, R.B. 1959. Investigations of Waterfowl Mortality at the Rocky Mountain Arsenal. U.S. Fish and Wildlife Service, Wildlife Research Laboratory, Denver, CO.

Food Safety and Inspection Service. 1998. National Residue Program: Domestic Residue Data Book. Appendix 1: U.S. Residue Limits for Compounds in Meat and Poultry. Available: http://www.fsis.usda.gov/OPHS/red98/appendix1c.pdf. Accessed 8/3/2007.

Foster Wheeler. 1996. Record of Decision for the On-Post Operable Unit. Version 3.1. Foster Wheeler Environmental Corporation. July 2.

Friend, M. and D.O. Trainer. 1974. Experimental dieldrin – duck hepatitis virus interaction studies. *J Wildl Manage* 38(4):896–902.

Grieb, J.R. 1981. Letter re: Waterfowl deaths on Rocky Mountain Arsenal. To Don Minnich, Regional Director, U.S. Fish and Wildlife Service. January 28.

Henriksen, E.O., G.W. Gabrielsen, and J.U. Skaare. 1996. Levels and congeners patterns of polychlorinated biphenyls in kittiwakes (*Rissa tridactyla*) in relation to mobilization of body lipids associated with reproduction. *Environ Pollut* 92:27–37.

Hudson R.H., R.K. Tucker, and M.A. Haegele. 1984. *Handbook of Toxicity of Pesticides to Wildlife*. US Fish and Wildlife Service, Washington DC. *Resource Publ* 153.

Jensen, W.I. 1955. Waterfowl Mortality, Rocky Mountain Arsenal, Denver, Colorado. A Summary Report. U.S. Fish and Wildlife Service, Wildlife Research Laboratory, Denver, CO.

Ludke, J.L. 1976. Organochlorine pesticide residues associated with mortality: Additivity of chlordane and endrin. *B Environ Contam Tox* 16:253–260.

MacDonald, D.D., C. Ingersoll, and T. Berger. 2000. Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. *Archives of Environmental Contamination and Toxicology* 39:20–31.

McEwen, L.C. 1983. Letter re: Summary table showing organochlorine residues detected in brain tissues of some of the animals found dead at the Arsenal in 1982. To William McNeill, Rocky Mountain Arsenal. March 4.

Morrison-Knudsen Environmental Services. 1989a. Aquatic Resources of Rocky Mountain Arsenal, Adams County, Colorado. Prepared for Shell Oil Company, Holme Roberts & Owen. September 27.

Morrison-Knudsen Environmental Services. 1989b. Wildlife Resources of the Rocky Mountain Arsenal, Adams County, Colorado. Prepared for Shell Oil Company, Denver, CO.

Neely, B., S. Kettler, J. Horsman, C. Pague, R. Rondeau, R. Smith, L. Grunau, P. Comer, G. Belew, F. Pusateri, B. Rosenlund, D. Runner, K. Sochi, J. Sovell, D. Anderson, T. Jackson, and M. Klavetter. 2006. Central Shortgrass Prairie Ecoregional Assessment and Partnership Initiative. The Nature Conservancy of Colorado and the Shortgrass Prairie Partnership.

Newton, I. 1988. Determination of critical pollutant levels in wild populations, with examples from organochlorine insecticides in birds of prey. *Environ Pollut* 55:29–40.

Olds, K.L. 1976. Rocky Mountain Arsenal Bird Kill, Denver, Colorado, 4 June 1976. Entomological Special Study No. 44-123-76. U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD.

Peakall, D.B. 1996. Dieldrin and other cyclodiene pesticides in wildlife. In *Environmental Contaminants in Wildlife Interpreting Tissue Concentrations*, W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds.). Lewis Publishers, Boca Raton, FL, pp. 73–97.

Persaud, D., R. Jaagumuagi, and A. Hayton. 1993. Guidelines for the Protection and Management of Aquatic Sediment Quality in Ontario. Prepared by Water Resources Branch, Ontario Ministry of the Environment and Energy. August.

Potomac Research International. 2006. RTRAC: Rocky Mountain Arsenal Transfer File Check Program for the Arsenal Environmental Database. User's Manual (Data Dictionary). Prepared for U.S. Army Environmental Center, Aberdeen Proving Ground, MD. Revised by DPRA, Incorporated, Commerce City, CO, for use at Rocky Mountain Arsenal. June 14.

Richardson, L.A., J.R. Lane, W.S. Gardner, J.T. Peeier, and J.E. Campbell. 1967. Relationship of dietary intake to concentration of dieldrin and endrin in dogs. *Bull Environ Contam Toxicol* 2:207–219.

R.L. Stollar & Associates, Harding Lawson Associates, Ebasco Services, DataChem, Enseco-Cal Lab, and Midwest Research Institute. 1992. Comprehensive Monitoring Program: Biota Annual Report for 1990 and Summary Report for 1988 to 1990. Prepared for U.S. Army Program Manager for Rocky Mountain Arsenal. July.

Robinson, J. and A.J. Crabtree. 1969. The effect of dieldrin on homing pigeons. *Mededelingen van de Rijksfaculteit Landbouwwetenschappen te Gent* 34(3):413–427.

Rocky Mountain Arsenal. 2007. Map of arsenal cleanup. Available: http://www.pmtheArsenal.army.mil/cleanup/clnfrm.html/ Accessed 7/2007.

Ronis, M.J.J. and C.H. Walker. 1989. The microsomal monooxygenases of birds. *Rev Biochem Toxicol* 10:301–384.

Samson, F.B., F.L. Knopf, and W.R. Ostlie. 1998. Grasslands. In *Status and Trends of the Nation's Biological Resources*, M.J. Mac, P.A. Opler, C.E. Puckett Haecker, and P.D. Doran (eds.). Vol. 2. Northern Prairie Wildlife Research Center Online, Jamestown, ND, pp. 437–472. Available: http://www.npwrc.usgs.gov/resource/habitat/grlands/index.htm. Accessed 1/21/2000.

Seidel, J. 1980. Letter re: Tour of Rocky Mountain Arsenal with the intent of checking Reservoir "F" for duck mortality. To Darryl Todd, Colorado Division of Wildlife. May 8.

Sharma, R.P., D.S. Winn, and J.B. Low. 1976. Toxic, neurochemical and behavioral effects of dieldrin exposure in mallard ducks. *Arch Environ Contam Toxicol* 5(1):43–53.

Stansley, W. and D.E. Roscoe. 1999. Chlordane poisoning of birds in New Jersey, USA. *Environ Toxicol Chem* 18(9):2095–2099.

Stephen, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W. D. Brungs. 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses.* NTIS PB85-227049. U.S. EPA Research Laboratory, Duluth, MN.

USFWS. 1961. Special Pesticide Problems. Unpublished report, Denver Wildlife Research Center.

USFWS. 1992. Wildlife Habitat Assessment Report for Contaminant Remediation at Rocky Mountain Arsenal. Prepared by USFWS Rocky Mountain Arsenal Field Office, Commerce City, CO. November.

USFWS. 1994a. Amphibians and Reptiles of Rocky Mountain Arsenal National Wildlife Refuge. U.S. Fish and Wildlife Service. Northern Prairie Wildlife Research Center Online, Jamestown, ND. Available: http://www.npwrc.usgs.govarsenrep.htm/. Accessed 4/10/2007.

USFWS. 1994b. Mammals of Rocky Mountain Arsenal National Wildlife Refuge. U.S. Fish and Wildlife Service. Northern Prairie Wildlife Research Center Online, Jamestown, ND. Available: http://www.npwrc.usgs.govarsenmam.htm/. Accessed 4/10/2007.

USFWS. 1995. Rocky Mountain Arsenal National Wildlife Refuge Fiscal Year 1994 Annual Progress Report. February 15.

USFWS. 1996. Rocky Mountain Arsenal National Wildlife Refuge Fiscal Year 1995 Annual Progress Report. October.

USFWS. 1997. Rocky Mountain Arsenal National Wildlife Refuge Fiscal Year 1996 Annual Progress Report. January.

USFWS. 1998. Rocky Mountain Arsenal National Wildlife Refuge Fiscal Year 1997 Annual Progress Report. February.

USFWS. 1999. Habitat Restoration Plan for Rocky Mountain Arsenal National Wildlife Refuge. August. Available: http://www.pmthe Arsenal.army.mil/refuge/refgfrm.html. Accessed 5/2007.

USFWS. 2000. Rocky Mountain Arsenal National Wildlife Refuge Fiscal Year 1999 Annual Progress Report. February.

Walker, C.H. and I. Newton. 1999. Effects of cyclodiene insecticides on raptors in Britain – correction and updating of an earlier paper by Walker and Newton in *Ecotoxicology* 7:185–189 (1998). *Ecotoxicology* 8:425–429.

Ward, F.P. and D.A. Gauthier. 1973. Research Prospectus: Studies to Elucidate the Cause of Waterfowl Mortalities at Basin F and Vicinity, Rocky Mountain Arsenal, Colorado. Edgewood Arsenal/Dugway Proving Ground Ecological Research Team. August.

WHO. 1989. Aldrin and Dieldrin Health and Safety Guide. IPCS International Programme on Chemical Safety. Health and Safety Guide No. 21. World Health Organization. Available: http://www.inchem.org/documents/hsg/hsg/hsg021.htm. Accessed 5/10/2004.