APPENDIX J

Terrestrial White Paper

UPPER ARKANSAS RIVER BASIN SITE CHARACTERIZATION REPORT - SUPPORTING ANALYSIS: CHARACTERIZATION OF THE POTENTIAL FOR INJURY TO MAMMALIAN WILDLIFE

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1.0 INTRODUCTION

This paper supplements analysis of potential injury to mammalian wildlife presented in the Site Characterization Report (SCR) prepared for the 11-Mile Reach of the Upper Arkansas River Basin near Leadville, Colorado. The Consulting Team (CT) was tasked with reviewing the existing data for the 11-Mile Reach and describing injuries to natural resources based upon that information. This supplemental analysis is consistent with the Memorandum of Understanding (MOU) Work Plan for the SCR and considers the U.S. Department of the Interior (DOI) Natural Resource Damage Assessment (NRDA) regulations [43 CFR 11].

According to NRDA regulations [43 CFR 11.62(f)(3)], determining injury to biological resources, including mammalian wildlife, must be based on establishing a statistically significant difference in response levels between the population in the study area and that of a control area. The regulations also define specific categories of injury for biological resources and state that injury determination must be based on measurement methodologies that are capable of demonstrating the specific biological response under consideration. For the 11-Mile Reach, there are limited data that allow for direct determination of injury to mammalian wildlife. Because there are limitations in the amount and extent of injury-specific data, the existing information is combined with data from the nearby California Gulch Superfund Site and information from the ecotoxicological literature to characterize the *potential* for injury to mammals using a weight-of-evidence approach consistent with ecological risk assessment methodologies (e.g., USEPA 1997b). In general, this includes evaluation and comparison of known and estimated exposure of wildlife to ecotoxicological benchmarks corresponding to known levels of toxicity. This approach is consistent with the goals of the MOU, and the approach that the CT has taken in the SCR. This paper describes available information that is applicable to characterizing the potential for adverse effects in mammals, and summarizes the potential for injury in response to a series of questions from the MOU Parties (see Attachment A).

2.0 PATHWAYS

2.1 Conceptual Model

A conceptual model describes the sources of contamination and the pathways by which resources of concern could be exposed to contaminants. A complete pathway, which results in an exposure, does not necessarily constitute an injury to natural resources as defined in DOI's NRDA regulations. The exposure must elicit an effect or response which can be measured and which is statistically different between the study area and a control area.

Conceptual models for exposure of mammals in the 11-Mile Reach have been described in two ecological risk assessments (ERAs) (Woodward Clyde 1993; USEPA 1997a). The primary sources of contamination in the 11-Mile Reach are mining and mineral processing wastes from the Leadville mining district. ERAs have shown that the primary chemicals of concern in the 11-Mile Reach are the metals cadmium, lead, and zinc.

Historically, California Gulch has been a major pathway for transport of solid and soluble forms of the metals to downgradient areas; including the Arkansas River. Periodic flooding has resulted in deposition of mine wastes along the river. In addition, floodplain soils may have been affected by overland runoff and irrigation of pasturelands with contaminated water. Mammals may be exposed to metals through direct contact with mine wastes or secondarily through contaminated surface water, soil, or sediment; or through ingestion of forage or prey that may have accumulated metals from biotic and abiotic sources. Injuries to surface water, soil, sediment and vegetation that comprise habitat for mammalian wildlife are described and evaluated in the SCR.

The frequency and duration of contact with contaminated media are important in characterizing the potential for injury to mammalian wildlife. Habitat quality and availability in contaminated areas are major factors affecting the frequency and duration of contact. This is especially true for large, mobile species such as elk, deer, and coyotes that range over large areas and may spend only a portion of their time in the area of concern. Smaller, less mobile species such as rodents (e.g., mice and voles) and lagomorphs (e.g., rabbits) range over smaller areas and may contact contaminated media more frequently.

3.0 SUMMARY OF AVAILABLE DATA

Information available to characterize the potential for injury to mammals includes histopathological data for small mammals in the 11-Mile Reach, data on target organ metal concentrations, and metal concentrations in exposure media (e.g. soil, water, vegetation). In addition, similar data are available from nearby areas in the California Gulch Superfund Site (but outside the 11-Mile Reach), and information from the ecotoxicological literature can be used to characterize the potential for injury based on exposure estimates.

Site-specific information from the 11-Mile Reach and the upstream reference area (Reach 0) includes:

- Histopathological analyses of vole and short-tailed weasel tissue samples from Reaches 0 and 2 (WCC 1993);
- Metal concentrations in kidney and liver tissue from small mammals (WCC 1993); and
- Metal concentrations in vegetation, soil, and water in Reaches 1, 2, and 3 (Keammerer 1987; Levy et al. 1992; WCC 1993).

Data from outside the 11-Mile Reach, but within the nearby California Gulch Superfund Site include:

- Histopathological analyses of mouse, vole, and chipmunk tissue samples from several locations with varying levels of mine waste contamination (WCC 1993; Stoller 1996);
- Metal concentrations in kidney and liver tissue from small mammals (WCC 1993; Stoller 1996); and
- Metal concentrations in vegetation, soil, and water (WCC 1993; Stoller 1996).

Data available from the scientific literature include ecotoxicological benchmarks for:

- Metal concentrations in mammalian tissues from laboratory and field studies;
- Metal concentrations in mammalian tissues associated with specific effects;
- Metal intake rates [i.e., Toxicity Reference Values (TRVs)]; and
- Metal concentrations in forage and prey items.

The above information is used to help evaluate whether metal concentrations in mammal tissues or abiotic media in the 11-Mile Reach are consistent with nearby conditions for which more data on injury are available. In some cases, the data from the California Gulch Superfund Site reflect higher metal

concentrations and bioavailability than in the 11-Mile Reach, and represent a reasonable worst-case scenario. The characterization is further supported by literature-based ecotoxicological benchmarks that correspond to known levels of toxicity and/or injury.

Data on metal content and histopathology in larger mammals (e.g. elk, deer, fox) are lacking for the 11-Mile Reach. However, data on injury to small mammals are available to help characterize the potential for injury to larger species that are more mobile and spend less of their life cycles in the 11-Mile Reach.

4.0 CHARACTERIZATION OF POTENTIAL INJURY

The CT's characterization of the potential for injury to mammals follows a weight-of-evidence approach consistent with DOI's NRDA regulations and EPA's ecological risk assessment methodologies. Histopathological data from the 11-Mile Reach and from Reach 0 are used to directly assess injury to small mammals. The histopathologic data is important because the presence of lesions in biological tissues associated with contaminants exposure is specifically identified as an injury in the NRDA regulations [43 CFR 11.62(f)(4)(vi)(D)]. Because there is a limited amount of injury-specific data, other types of data are used as supporting weight-of-evidence to evaluate the uncertainty associated with the small histopathological data set.

Metal concentrations in kidney and liver samples are compared to ecotoxicological benchmarks that are associated with known levels of histopathological or physiological dysfunction. Internal organ and soft tissue malformation and histological lesions are the most common metals effects reported in the literature and associated with benchmark values. Several recently published secondary sources report benchmark values for wild mammals that are based on comprehensive reviews of field and laboratory studies (Hoffman et al. 1995; Beyer et al. 1996; Eisler 2000; Shore and Rattner 2001). A large number of laboratory and field studies were reviewed and it was determined that the majority of the literature is consistent with the benchmarks and associated effects presented in these secondary sources. Of the studies reviewed, most of the mammalian studies, which evaluated metals exposure and accumulation, were conducted on mining impacted sites and are therefore appropriate for consideration in the Upper Arkansas River Basin (UARB).

To further evaluate the level of uncertainty associated with limited histopathological data, metal concentrations in vegetation, small mammals, soil, and water are used to estimate the rate at which wildlife may ingest metals while feeding and drinking. These estimates are compared to TRVs, which are intake rates corresponding to known toxicological effects or lack thereof (EPA 1993, 1997b; Eisler 2000).

In addition to data collected from the 11-Mile Reach, information from California Gulch and the scientific literature are used to help evaluate the potential for injury to mammals in the 11-Mile Reach. Because of the proximity to mine-waste tailings and smelter residues, data from California Gulch reflect higher metal concentrations and greater bioavailability than conditions in the 11-Mile Reach and, therefore, represent a worse case scenario that can be used as a point of comparison. The characterization

was further developed by comparing non-injury specific data from both the 11-Mile Reach and California Gulch to literature-based ecotoxicological benchmarks that correspond to known levels of toxicity and/or injury. This includes benchmarks comparable to metal concentrations in tissues as described above, as well as dose-based (i.e., intake) benchmarks that are commonly used in ERAs.

4.1 Small Mammals

4.1.1 Histopathology

WCC (1993) sampled small mammals from locations in Reach 2, the California Gulch Superfund Site outside the 11-Mile Reach, and nearby reference areas along Tennessee Creek and the upper Arkansas River (Reach 0). Approximately 28 tissues from each of 36 animals were examined histologically using light microscopy. From Reach 2, WCC collected four southern red-backed voles (*Clethrionomys gapperi*), two long-tailed voles (*Microtus longicaudatus*), and two short-tailed weasels (*Mustela erminea*) for histological analysis. Samples from Reach 0 included four red-backed voles and five long-tailed voles.

The sampling locations in Reach 2 were irrigated pastures downstream of the confluence of California Gulch with the Arkansas River and reflect contamination from deposition of mine wastes, floodwater, and/or irrigation water. Data on soils and vegetation indicate that the animals were potentially exposed to elevated metal concentrations in soil and vegetation. The animals were collected from sites where mean metal concentrations in floodplain soils were 36 ppm cadmium, 968 ppm copper, 4,665 ppm lead, and 6,055 ppm zinc, representing some of the highest concentrations along the 11-Mile Reach. To a lesser extent, concentrations of the same metals were elevated in vegetation.

As noted, data from WCC (1993) are directly applicable to characterizing injury in the 11-Mile Reach. Besides the direct applicability for evaluating injury, these data were collected from an area of confirmed mine waste with contamination-level metal concentrations and from potentially mixed sources/transport mechanisms. In addition, vegetation from the area contains elevated metal concentrations creating a true metals exposure scenario. The data set also includes samples from reference areas of similar habitat.

Results of WCC's histologic analysis indicated no abnormal histopathology or injury that could be attributed to metals exposure. Although the kidney is the primary site of toxic action of cadmium,

WCC did not submit kidneys for histopathology from any of the animals collected. Based on the experience of the pathologist who conducted these analyses (Dr. Terry Spraker, Colorado State University), lesions associated with cadmium exposure would not be expected at the tissue concentrations found in these animals (T. Spraker, Pers. Comm.). This conclusion (that tissue lesions would not be expected to occur at the cadmium concentrations present in vole kidney tissues from Reaches 0 and 2) is also supported by the ecotoxicological literature (Cooke and Johnson 1996; Eisler 2000; Ma and Talmage 2001).

WCC (1993) also collected liver and kidney samples from areas on the Superfund Site (St. Josephs Cemetery and Hamm's Mill) in which soils metal concentrations were higher than those found in the 11-Mile Reach. Cadmium, lead, and zinc concentrations in kidney and liver samples from these Superfund Site locations were equal to or higher than concentrations from Reach 2. Despite the higher concentrations in soils and equivalent or higher concentrations in tissues, kidney and liver samples from the Superfund Site locations did not exhibit abnormal histopathology that could be attributed to metal toxicity. This supports the view that there is low risk of this type of injury for small mammals in the 11-Mile Reach. Based on this information, the absence of kidney histologic analysis is not critical to evaluating small mammal injury for the WCC data set.

Histopathological analyses of small mammal liver and kidney tissue are also available from Upper California Gulch (Stoller 1996). Upper California Gulch is part of the Superfund Site and contains soils, mill tailing and waste rock with varying metal concentrations. Sample locations included uplands as well as locations containing fluvial tailings deposits and waste rock in riparian areas. A total of twenty-five animals including primarily least chipmunks (*Eutamias minumus*) and deer mice (*Peromyscus maniculatus*), but also a southern red-backed vole and a long-tailed vole were collected from locations representing a wide range of metal contamination. Animals were collected directly from waste rock and tailing piles; from locations near, but not on waste piles; and from sites remote from waste piles in the study area and reference area (Iowa Gulch). In samples from all locations, results indicated "minimal" to "slight" occurrence of lesions. Neither the frequency nor intensity of lesions was correlated with proximity to mine waste deposits. These results indicate that these animals showed no signs of histopathological injury due to metals exposure.

Limitations affecting use of the WCCs (1993) histopathological data include low sample number (6 animals) and lack of data from other locations in the 11-Mile Reach. In addition, small mammal data from Reach 2 are for herbivorous vole species only. The Stoller (1996) data are not from the 11-Mile

Reach, but samples were collected from a gradient of contamination including animals inhabiting waste rock piles with high metal concentrations (cadmium over 100 ppm, lead over 40,000 ppm, and zinc over 15,000 ppm); as well as areas of nearby soils with lower metal concentrations. The histopathology results can be used in conjunction with data on metal concentrations in liver, kidney, soils, and vegetation in the weight of evidence approach to characterize the potential for injury. Limitations on use of the Stoller (1996) data include the lack of samples from the 11-Mile Reach and the lack of data from species other than granivorous/herbivorous species.

Insectivorous species such as shrews, may accumulate more cadmium than granivores (seed eaters) and herbivores (vegetation eaters) because terrestrial insects (their main dietary component) generally accumulate higher cadmium concentrations than vegetation (Cooke and Johnson 1996). Several shrew species potentially occur in the Upper Arkansas River Valley. WCC (1993) captured 3 montane shrews (*Sorex monticolus*) during their small mammal trapping effort to determine relative abundance, but they did not capture any shrews when they sampled for tissue collection.

4.1.2 Metal Concentrations in Tissues

It is generally accepted that diagnosis of metals poisoning as cause of death is established through a synthesis of necropsy observations, pathological findings, and tissue concentrations. Death (from metal poisoning) cannot be diagnosed on the basis of tissue concentrations only, but metal poisoning can be diagnosed from concentrations if sufficient data exist from the same or related species showing a relation between illness and concentrations (Franson 1996). Metal concentrations in biological tissues are not a direct measure of injury endpoints cited in DOI's NRDA regulations. However, toxicological studies of metal exposure have resulted in estimates of tissue metal concentrations that are correlated with histological effects and/or adverse physiological and biochemical effects. These types of effects are specific injury categories cited in the NRDA regulations. Thus, benchmark concentrations can be used in conjunction with tissue concentrations in samples from the site and reference areas to support the direct measures of injury and further characterize the potential for injury due to metals exposure. Metals are not degraded by metabolism; therefore, measure of metal concentrations in target organs is a relatively direct measure of exposure. As noted above, kidney and liver are important sites of metal toxicity in vertebrates. Therefore, data on metal residues in kidney and liver can be used to characterize the risk of adverse effects and the potential for injury.

Benchmark levels cited in the following discussion were taken from 1996 summaries of scientific literature on cadmium and lead toxicity in small mammals (Cooke and Johnson 1996, Ma 1996). For cadmium, Cooke and Johnson (1996) recommend 100 mg/Kg (wet wt) (350 mg/Kg dry wt) as critical concentration in kidneys of small mammals. No analogous recommendations for cadmium in liver tissue were provided since the kidney is the primary target organ in cadmium toxicity. For lead, Ma (1996) cites concentrations of 6 to 10 mg/Kg (dry wt) in kidney and 2.5 to 5 mg/Kg (dry wt) in liver as no-observed–adverse-effects levels (NOAELs) for effects ranging from changes in the somatic organ index to reproductive effects.

Data on metal residues in small mammal kidney and liver samples from Reach 0 and Reach 2 are available from WCC (1993). For Reach 2, there are individual and composite liver and kidney samples from voles and short-tailed weasels (Table 1). For Reach 0, there are kidney and liver composite samples from voles only (Table 2). Ideally, composite samples represent an average value of those individuals making up the composite sample. However, the variability around that average value is unknown as is the case with the composite samples submitted by WCC (1993). As with the histopathology studies, chemical residue data are also available from other sampling locations in California Gulch outside the 11-Mile Reach (WCC 1993; Stoller 1996). Data are available for red-backed voles, long-tailed voles, deer mice, and least chipmunks from locations representing a wide range of metal concentrations in soils.

Kidney cadmium concentrations in small mammals did not exceed NOAEL, lowest observed, adverse effect level (LOAEL), or critical concentrations associated with injury in small mammals (Figure 1). Maximum cadmium concentrations in kidneys from Reach 0 or Reach 2 did not approach the 350 mg/Kg (dry wt) critical concentration identified by Cooke and Johnson (1996). The maximum cadmium concentration identified in Reach 2 was 39 mg/Kg dry wt. (11.1 mg/Kg wet wt), while the maximum concentrations from the entire WCC study was 69 mg/Kg dry wt. (19.7 mg/Kg wet wt) (WCC 1993, Table 7-15). The maximum cadmium concentration in surface soils co-located with small mammal samples was approximately 55 mg/Kg. By contrast, the maximum cadmium concentration in chipmunk kidneys from Upper California Gulch was 119 mg/Kg dry wt. (34 mg/Kg wet wt.) and was associated with concentrations in surface soil of less than 15 mg/Kg (Stoller 1996, Figure 3-8).

These results indicate that maximum cadmium concentrations in small mammals from Reach 2, and from the overall Superfund site do not exceed the recommended threshold concentrations for physiological and histopathological effects (Cooke and Johnson 1996). In addition, the results suggest

that overall cadmium bioavailability from soils and plant materials may be less along the 11-Mile Reach than in some areas of the Superfund Site.

Lead concentrations in liver and kidney also did not exceed concentrations associated with ecologically important effects (Figure 2). The maximum concentration in kidney and liver was approximately equal to NOAEL-based concentrations for sublethal effects. Lead was detected in only one of three liver samples collected from Reach 0 and Reach 2 (Detection Limit <0.5 mg/Kg)(WCC 1993).

Limitations of chemical residue data from the WCC (1993) study are similar to those cited for histopathology. The sample size for 11-Mile Reach is small, and samples are not available from segments downstream of Reach 2. However, the samples represent maximal, or near maximal exposures for the 11-Mile Reach.

Only herbivorous voles were included in the WCC samples, and only granivorous/herbivorous chipmunks were collected from Upper California Gulch. Small mammals with more insectivorous diets, such as shrews, might be expected to accumulate greater amounts of cadmium (Cooke and Johnson 1996; Ma and Talmage 2001). Based on data from the literature, cadmium accumulation in shrew kidneys can be more than 25 times that of herbivorous rodents (Cooke and Johnson 1996). Tissues from the captured shrews in the 11-Mile Reach were not analyzed, but concentrations 25 times that of vole kidneys would exceed Cooke and Johnson's critical levels for adverse effects on renal function and structure, and may be consistent with a positive injury determination. However, the literature also indicates that in comparison to rodents, shrews may be more resistant to cadmium exposure through a greater metallothionein-related detoxication capacity of the target organs (i.e., liver and kidney) (Shore and Douben 1994; Cooke and Johnson 1996; Eisler 2000; Ma and Talmage 2001). Therefore, literature-based benchmarks used for rodents may not be appropriate for insectivores such as shrews and it is unclear whether the concentrations present at the site cause injury to shrews.

4.1.3 Conclusions - Small Mammals

Data are available for directly evaluating injury to small mammals in the 11-Mile Reach, although sample sizes are small and geographical coverage is limited. Six samples of voles are available from Reach 0 and Reach 2. Overall, histopathological analyses of these samples show no injury to small mammals in either Reach 0 or Reach 2. While the kidney is the primary organ associated with cadmium toxicity in mammals, kidneys were not analyzed for histopathology in samples collected from Reach 0 or Reach 2. The small sample size, limited geographical coverage, and lack of kidney histological analysis creates some uncertainty in the data; however, this uncertainty is reduced by the following:

- (1) Metal concentrations in kidney and liver samples from Reach 2 samples did not exceed benchmarks associated with physiological and histopathological effects;
- (2) Samples from the Superfund Site (areas of potentially higher exposure) also did not contain metal concentrations in excess of benchmarks and;
- (3) Samples from the Superfund site (areas of potentially higher exposure) did not exhibit liver or kidney histopathology, which could be related to effects from metals exposure.

The results from each of these three data sets are consistent with each other and support the results reported by WCC (1993). While none of these data sets alone are sufficient to evaluate injury, the weight-of-evidence provided by one injury-specific data set and three supporting data sets lends more confidence to the overall injury assessment.

Species with more insectivorous diets, such as shrews, may have higher exposures due to higher dietary cadmium concentrations, but data are not available for direct evaluation of these species. Existing literature indicates that benchmarks appropriate for rodents may not be appropriate for insectivores, as insectivores appear to be more tolerant of increased metals exposure.

The apparent lack of effects on individual small mammals receiving maximal or near-maximal metals exposure indicates a corresponding lack of injury to local populations in the 11-Mile Reach. Additional samples would reduce the uncertainty in evaluating potential injury, but may not significantly aid in restoration planning for the 11-Mile Reach (see SCR).

4.2 Large Mammals

Tissue metal data and histopathology analyses for larger mammals (e.g., elk, deer, fox) are not available for the 11-Mile Reach. However, the potential for injury to large mammals was characterized by evaluating exposure through the comparison of metal concentrations in forage and estimates of metal intake rates to benchmarks values. In addition, data on injury to small mammal populations was used to help evaluate the potential for injury to larger species that are more mobile and spend less of their lifecycles in the 11-Mile Reach. Because small mammals have a relatively small home range, they are likely to receive a more constant exposure to contaminated media. The 11-Mile Reach is a ribbon of habitat within the Upper Arkansas River Basin and it is expected that larger mammals will migrate through and may spend periods of time there. However, large mammal exposure will be considerably less as compared to that of small mammals.

Generally, data for direct measurement of injury to large mammals are not available, and characterizing the potential for injury is more difficult and uncertain. In the absence of data on histopathology or metal residues in tissues, the potential for injury can be estimated using risk assessment techniques in which the intake of metals is calculated and compared to benchmark intakes of known toxicity to the receptor or similar species. Data on metal content in food and other ingested materials is used along with estimates of the daily intake of each medium (Alldredge et al. 1974; EPA 1993, 1997; Beyer et al. 1994).

Large mammals of greatest concern in the 11-Mile Reach are elk (*Cervus elaphus*) and mule deer (*Odecoileus hemionus*). Elk and mule deer use the 11-Mile Reach seasonally during fall and winter, but migrate to higher elevations in spring and summer. However, a few individuals may remain through spring and summer. Elk feed both by grazing on grasses and forbs, and browsing on woody vegetation (Fitzgerald 1994). Deer are primarily browsers, but opportunistically feed on grasses and forbs (Fitzgerald 1994). Ungulates could be exposed to metals in forage plants, incidentally ingested soils, and, to a lesser extent, in surface waters. Data on metal content of grasses, forbs, and shrubs (e.g., willows) are available from Reach 0, 1, 2 and 3; grass and forb data are available from more downstream areas. Vegetation data are from Keammerer (1987) and were collected from locations in the floodplain, but distinct from mine waste deposits. Soils in these areas contain elevated metals concentrations, which tend to decrease with distance downstream from Reach 1.

Carnivorous mammals such as the coyote (*Canis latrans*), red fox (*Vulpes vulpes*), North American badger (*Taxidea taxus*), and short-tailed weasel (*Mustela erminea*) also inhabit the 11-Mile Reach. Individual fox and coyote occupy large areas ranging from several hundred to over 3,000 hectares (USEPA 1993; Fitzgerald 1994). Badgers and weasels have more restricted home ranges and individual may spend a large proportion of their time in the 11-Mile Reach. Coyotes, badgers, fox, and weasels are primary carnivorous, feeding on small mammals and birds. Small mammal whole-body data from California Gulch (Stoller 1996; USEPA 1997a) and other mine sites suggest that metals are not effectively translocated to the primary prey of these species, thus bioaccumulation is low limiting the potential for metals exposure to predators.

The potential for injury can be characterized by comparing the potential metals exposure of large mammals to ecotoxicologically-based benchmarks. This was conducted using two approaches: (1) comparing metal concentrations in forage plants to benchmarks from the scientific literature and (2) estimating daily intake of metals from forage foods and soils, and comparing the intakes to TRVs which represent rates corresponding to known levels of toxicity and injury (EPA 1993, 1997b, Eisler 2000).

4.2.1 Characterization of Potential Injury –Concentration-Based Benchmarks for Forage

There are few ecotoxicological benchmarks available for wild ungulates, therefore, benchmark metal concentrations recommended for ruminant wildlife and livestock forage or feed are used (Table 3). The most complete data set available for chemical concentrations in vegetation in the 11-mile reach is that of Keammerer (1987). Metal concentrations in vegetation for Reaches 0, 1, 2, and 3 are shown in Table 4. Except for lead, metal concentrations tended to be higher in forbs than in grasses. Mean metal concentrations were less than benchmarks for all metals except cadmium. Mean cadmium concentration exceeded the lowest benchmark of 0.5 mg/Kg for livestock (NAS 1980; Church 1988; Eisler 2000) in all reaches, including Reach 0. Cadmium exceeded the no-effects criterion (3-5 mg/Kg) in only Reach 3, and concentrations did not exceed those associated with mild renal dysfunction (10 mg/Kg) in any reach.

Cadmium concentrations in vegetation, especially forbs, were not significantly higher in Reach 1 or 2 than in Reach 0. Forb cadmium concentrations were highest in Reach 3, but differences may not be significant. These results suggest that concentrations in the downstream sampling locations are not different from Reach 0 (Figure 3), and that the corresponding potential for injury from cadmium in vegetation does not differ from baseline conditions represented by Reach 0.

4.2.2 Characterization of Potential Injury - Estimated Metal Ingestion Rates

The potential for injury to large mammals was characterized using standard risk assessment methodology to estimate the ingestion of metals in food and incidentally ingested soils (USEPA 1993, 1997b). The ingestion rates were then compared to TRVs representing known levels of potential toxicity and injury. The characterization was phased, starting with a screening-level analysis (USEPA 1997b),

which includes conservative assumptions about exposure (i.e., ingestion) and toxicity (i.e., most sensitive endpoints) in order to minimize the chance of underestimating the potential for injury.

The potential for injury was characterized for mule deer to represent grazing/browsing ungulates. Mule deer were selected because they feed on a variety of vegetation types in the area and may remain in the valley lowland for longer periods of time than elk, which migrate to higher elevations for a large proportion of the year. Mule deer food and incidental soil ingestion rates were taken from the scientific literature:

- Food ingestion rate: 0.02 Kg food/Kg bw/day (Alldredge et al. 1974)
- Soil ingestion rate: 0.0004 Kg soil/Kg bw/day (Beyer et al. 1994)

Conservative assumptions about bioavailability and contact rate were also used:

- Assuming that 100 percent of metal ingested in food is absorbed (i.e., 100 percent bioavailability)
- The receptor obtains 100 percent of food and incidentally ingested soil from the 11-Mile Reach

Both of these assumptions tend to overestimate exposure since only a small fraction of ingested metals is generally absorbed from the intestinal lumen (Klaassen 1995; Cooke and Johnson 1996; Eisler 2000), and individual animals are unlikely to feed exclusively in the 11-Mile Reach.

Estimated ingestion rates were compared to the following NOAEL-based TRVs:

- Cadmium: 0.27 mg/Kg bw/day (Sutou et al. 1980 as cited in Sample et al. 1996)
- Lead: 5 mg/Kg bw/day (Horwitt and Cowgill 1971)
- Zinc: 45 mg/Kg bw/day (Schlicker and Cox 1968 as cited in Sample et al. 1996)

Risk of adverse effect was characterized using the hazard quotient approach (HQ)(USEPA 1997b). The HQ is the ratio of estimated site exposure to the TRV (i.e. [site exposure] / [TRV]). An HQ greater than 1 indicates site exposures that exceed the TRV. NOAELs represent concentrations below those expected to elicit adverse effects; the threshold exposure for inducing effects is higher than the NOAEL and lies between it and the LOAEL. Since the TRVs used in this characterization are based on NOAELs for sublethal systemic or reproductive effects and chronic exposure durations, an HQ less than 1 is indicative of conditions under which no effects are expected. The conservatism of using NOAEL-

based TRVs is compounded by that associated with the exposure estimation methods (i.e., 100 percent bioavailability, 100 percent site use).

Exposures and risk were estimated for ingestion of cadmium, lead, and zinc in Reaches 0, 1, 2, and 3. The exposure point concentrations in forage plants and soil were assumed to be the mean +1 standard error for each reach (Tables 4 and 5). Since the exposure and risk estimates are based on conservative, screening-level assumptions, this approach is consistent with the methods used to characterize injury to plants in floodplain soils.

Calculations and HQs are shown in Table 6, and HQs are summarized in Figure 4. No HQs exceeded 0.6 for any metal or reach, indicating that exposures are not expected to exceed NOAELs. This result can be further interpreted as indicating a very low likelihood of injury to ungulates feeding in the 11-Mile Reach, and an even lower likelihood of significant injury or adverse effects on local populations.

4.2.3 Conclusions - Large Mammals

Data on metal concentrations in vegetation suggest that ruminant herbivores such as mule deer and elk are at some risk from cadmium concentrations in vegetation that exceed the lower range of recommended levels (0.5 mg/Kg) for livestock (NAS 1980; Church 1988; Eisler 2000). However, Church (1988) also indicates that cattle are not affected by cadmium concentrations of 3-5 mg/Kg, suggesting that this range may be more appropriate for characterizing injury. Although cadmium concentration tend to be higher in samples from downstream reaches, cadmium concentrations in grasses and forbs from Reach 1, 2, and 3 are not significantly higher than Reach 0 samples, suggesting that the risk of injury in downstream reaches may not be greater than baseline. Estimations of cadmium intake from ingestion of forage and soils from each reach do not exceed NOAEL-based TRVs for mammals, even when conservative screening-level assumptions are used to estimate intake.

Results from small mammals data also suggests that risk of injury to large mammals is minimal. Small mammals occupy much more restricted home ranges than larger, more mobile species. Therefore, individuals that occupy contaminated areas experience much longer duration exposures. The lack of effects observed in small mammals from Reach 2 and the Superfund site suggests that larger species are not at risk.

As noted above, this approach is not a direct measure of injury to large mammals. Rather, it helps characterize the *potential* for injury based on application of general toxicological information to site conditions. Conservative assumptions were used in the exposure and risk estimation process to minimize the likelihood that the risk of injury is underestimated. The effects of this conservatism are illustrated by comparing the estimates of exposure and risk generated by EPA in the site wide ERA (USEPA 1997), to the actual effects described in this document. EPA's assessments shows hazard indices between 10 and 100 in Reach 1 and 2. However, the exposure assessment presented above, combined with information on small mammals, is not consistent with significant risk to mammalian wildlife in the 11-Mile Reach.

Taken together, these results suggest that individual ruminants are not likely to be injured in the 11-Mile Reach unless they feed exclusively in the areas of highest contamination. Elk and deer populations that utilize the 11-Mile Reach are not likely to be injured due to the small proportion of the 11-Mile Reach that is covered by mine wastes and the fact that they do not continually utilize the contaminated areas.

5.0 **REFERENCES**

- Alldredge, A.W., J.F. Lipscomb, and F.W. Wicker. 1974. Forage intake rates of mule deer estimated with fallout cesium-137. J. Wildl. Manage. 38:508-816.
- Beyer, W.N., G Heinz, and A. W. Redmond-Norwood (Editors). 1996. Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. SETAC Special Publication Series. Lewis Publishers.
- Beyer, W.N., E.E. Connor, and S. Gerould. 1994. Estimates of soil ingestion by wildlife. J. Wildl. Manage. 58:375-382.
- Church, D.C. 1988. The Ruminant Animal-Digestive Physiology and Nutrition. Prentice Hall, New Jersey.
- Cooke, J.A. and M.S. Johnson. 1996. Cadmium in small mammals. *In:* Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. Beyer, W.N., G.H. Heinz, and A.W. Redmon-Norwood (eds.). SETAC Special Publication Series. Lewis Publishers.
- Eisler, R. 2000. Handbook of Chemical Risk Assessment: Health Hazards to Humans, Plants, and Animals. Volume 1 Metals, CRC Press, Boca Raton, FL, 738 pp.
- Eisler, R. 1985. Cadmium hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service, Biological Report 85(1.2) 46 pp.
- Eisler, R. 1988. Lead Hazards to Fish Wildlife And Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service, Biological Report 85 (1.14) 134 pp.
- Eisler, R. 1993. Zinc Hazards to Fish Wildlife And Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service, Biological Report 10, 106 pp.
- Fitzgerald, J.P., C.A. Meaney, and D.M. Armstrong. 1994. Mammals of Colorado. Denver Museum of Natural History, University Press of Colorado 467 pp.
- Franson, J.C. 1996. Cadmium in small mammals. *In:* Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. Beyer, W.N., G.H. Heinz, and A.W. Redmond-Norwood (eds.). SETAC Special Publication Series. Lewis Publishers.
- Hoffman, D.J., B.A. Rattner, G.A. Burton Jr., J. Cairns Jr. (Editors). 1995. Handbook of Ecotoxicology. CRC Press Inc., Boca Raton, Florida.
- Horwitt, M.K., and C.R. Cowgill. 1931. The effects of ingested lead on the organism: II. Studies on the dog. J. Pharmacol. Exper. Therapy. 66:289-301.
- Keammerer, W. 1987. Vegetation Studies in the Upper Arkansas River. Unpublished data collected for Asarco.
- Levy, D. E., K.A. Barbarick, E.G. Seimer, L.E. Somers. 1992. Distribution and Partitioning of Trace Metals in Contaminated Soils Near Leadville, CO. J. Environ. Quality 21:185-195.

- Ma, W., and S. Talmage. 2001. Insectivora. In: Ecotoxicology of Wild Mammals. Shore, R.F. and B.A. Rattner (eds.). pp123-158. Ecological and Environmental Toxicology Series. John Wiley and Sons LtD, West Sussex, England.
- Ma, W. 1996. Lead in mammals. *In:* Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. Beyer, W.N., G.H. Heinz, and A.W. Redmon-Norwood (eds.). pp. 281-296. SETAC Special Publication Series. Lewis Publishers.
- NAS. 1980. Mineral Tolerance of Domestic Animals. National Academy of Sciences, Washington D.C. 577 pp.
- Klaasen, C.D. 1995. Casarett and Doulls Toxicology, the Basic Science of Poisons, 5th edition. McGraw-Hill Health Professions Division.
- Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological benchmarks for wildlife. 1996 revision. Prepared for the US Department of Energy by Lockheed Martin Energy Systems, Inc. ES/ER/TM-86/R3.
- Schlicker, S. A. and D. H. Cox. 1968. Maternal dietary zinc, and development and zinc, iron, and copper content of the rat fetus. *J. Nutr.* 95: 287-294.
- Shore, R.F. and B. A. Rattner (Editors). 2001. Ecotoxicology of Wild Mammals. John Wiley and Sons, West Sussex, England.
- Shore, R.F. and P.E.T. Douben. 1994. The Ecotoxicological Significance of Cadmium Intake and Residues in Terrestrial Small Mammals. Ecotoxicology and Environmental Safety, 29:101-113.
- Spraker, T. 2001. Personal Communication with Dr. Terry Spraker, Pathologist, Veterinary Teaching Hospital, Colorado State University, Fort Collins, CO.
- Sutou, S., K. Yamamoto, H. Sendota, and M. Sugiyama. 1980. Toxicity, fertility, teratogenicity, and dominant lethal tests in rats administered cadmium subchronically. I. Fertility, teratogenicity, anddominant lethal tests. *Ecotoxicol. Environ. Safety*. 4:51–56.
- S.M. Stoller Corporation, 1996. Screening Level Ecological Risk Assessment, Operable Unit No. 4, California Gulch Superfund Site. Prepared for Resurrection Mining Company, December 19, 1996.
- USEPA (United States Environmental Protection Agency). 1993. *Wildlife Exposure Factors Handbook. Volume II.*" USEPA/600/R93/187b, Office of Research and Development, Washington, D.C.
- USEPA (United States Environmental Protection Agency). 1997a. *Ecological Risk Assessment for the Terrestrial Ecosystem, California Gulch NPL Site.* Prepared for USEPA by Roy F. Weston, Inc. and Terra Technologies. January.
- USEPA (United States Environmental Protection Agency). 1997b. *Ecological Risk Assessment Guidance* for Superfund: Process for Designing and Conducting Ecological Risk Assessments. Environmental Response Team, Edison, NJ. Interim Final. June 5, 1997.
- WCC (Woodward Clyde Consultants). 1993. Terrestrial Ecosystem Evaluation Report California Gulch Site, Leadville, Colorado. Prepared for ASARCO Inc., Denver, CO.

ATTACHMENT A

QUESTIONS FROM THE MOU PARTIES REGARDING TERRESTRIAL WILDLIFE

- 1. Clarify difference between literature benchmarks indicating *injury* and literature benchmarks indicating *risk of injury*. (i.e. : the difference between tissue residues that results in effects vs. soil/vegetation residues that pose a risk of injury)
- 2. Explain how the existing small mammal data can be used as an indicator of overall mammalian population health (i.e. based on small mammal life history, they could represent a worst-case scenario for all mammals) or explain if such an extrapolation is not appropriate.
- 3. Discuss the representativeness of using the existing small mammal data (primarily herbivores) to extrapolate to other small mammals (i.e. insectivores).
- 4. Explain the selection and application of specific benchmarks and why they are applicable to the Upper Arkansas River Basin.
- 5. Explain how the actual injury data is used in conjunction with benchmarks.
- 6. Present the range of benchmark values considered, their effects, and the basis for choosing specific ones.
- 7. Potential shortcomings of Woodward-Clyde not having sent the kidneys from the small mammal study to the pathologist
- 8. Discussion of other factors that affect/influence metals exposure and (i.e., species, home range, diet, etc)
- 9. How will soil ingestion by mammals be evaluated as a route of exposure?
- 10. Explain why co-location of Woodward-Clyde samples is a legitimate (conservative ?) approach

Tissue	Number of Individual Samples	Number of Composite Samples	Number of Individuals in Each Composite Sample	Total number of Samples	Total Number of Animals
vole					
Liver	2	1	4	3	6
Kidney	1	1	4	2	5
short-					
tailed					
weasel					
Liver	2	0	0	2	2
Kidney	2	0	0	2	2

Table 1. Number and Type of Vole and Short-Tailed Weasel Tissue Samples from Reach 2
(WCC 1993).

Tissue	Number of Individual Samples	Number of Composite Samples	Number of Individuals in Each Composite Sample	Total number of Samples	Total Number of Animals
Liver	0	3	10,10,6	3	26
Kidney	0	3	10,10,6	3	26

 Table 2. Number and Type of Vole Tissue Samples from Reach 0 (WCC 1993).

Metal and Criterion U	se	<u>Concentration</u>	Reference
'Maximum tolerated'	ruminants	0.5	Church (1988)
Maximum exposure w/o effect	ruminants	3-5	
Mild renal dysfunction	small mammals	10	Cooke & Johnson (1996)
Lead Recommended levels	horses cattle	<80 <200	Eisler (1988)
Zinc Recommended range for livestock Maximum tolerated	calves adult cattle	45-60 500 (DW) 1,000 (DW)	Eisler (1988)

Table 3. Recommended Metal Concentrations in Forage Protective of Wildlife and Livestock (units = mg/Kg)

Reach	Cadı (mg	nium ;/kg)	Cop (mg	per /kg)	Le (mg	ead (/kg)	Zi (mg	n ³					
	Grasses ² Forbs ²		Grasses Forbs		Grasses Forbs		Grasses Forbs						
0	0.8	3.8	5.1	11.2	0.1	2.9	82	255					
	(<u>+</u> 1.3)	(<u>+</u> 0.87)	(<u>+</u> 0.6)	(<u>+</u> 3.9)	(<u>+</u> 0)	(<u>+</u> 1.6)	(<u>+</u> 17.3)	(<u>+</u> 72)	9				
1	2.2	4.6	4.6	10.3	12.2	19.8	153	248					
	(<u>+</u> 0.2)	(<u>+</u> 1.4)	(<u>+</u> 0.4)	(<u>+</u> 2)	(<u>+</u> 5.2)	(<u>+</u> 8.3)	(<u>+</u> 71)	(<u>+</u> 74)	7				
2	1.6	3.4	4.9	7.7	9	13.1	147	186					
	(<u>+</u> 0.9)	(<u>+</u> 3.5)	(<u>+</u> 1.8)	(<u>+</u> 4.8)	(<u>+</u> 7)	(<u>+</u> 24)	(<u>+</u> 223)	(<u>+</u> 315)	8				
2	1.6	6.4	6.4	18.9	4.5	0.1	239	394					
5	(<u>+</u> 0.4)	(<u>+</u> 1.1)	(<u>+</u> 0.6)	(<u>+</u> 1.6)	(<u>+</u> 2.8)	(<u>+</u> 0)	(<u>+</u> 79)	(<u>+</u> 98)	8				

 Table 4

 Plant Tissue Metal Concentrations for Grasses and Forbs (reported on a dry-weight basis) from Sites Sampled along the Arkansas River ¹

¹Means and standard errors (\pm 1 s.e.) for sites sampled in 1987.

² The dietary concentration of cadmium that has been set as the maximum tolerable level for ruminants is 0.5 mg/kg (Church 1988). This concentration is exceeded for both grasses and forbs. This is most likely a result of the generally higher mineralization and metal content associated with soils in this region and does not translate to an injury to terrestrial trust resources. True toxicity to ruminants can only be determined with diet, physiological, and pathological studies of grazing animals.

 3 n = number of samples

Son Metal Concentrations (total) for Sites Sampled along the												
Arkansas River ¹												
Reach	Cadmium (mg/kg)	Copper (mg/kg)	Lead (mg/kg)	Zinc (mg/kg)	n ²							
	Total	Total	Total	Total								
0	3.3	29.9	238	428	•							
	(<u>+</u> 0.57)	(<u>+</u> 7.3)	(<u>+</u> 45)	(<u>+</u> 75)	9							
1	13.5	192	3,990	3,142								
'	(<u>+</u> 5.7)	(<u>+</u> 115)	(<u>+</u> 1,212)	(<u>+</u> 2,385)	7							
2	15.4	51.4	675	1180								
2	(<u>+</u> 3.9)	(<u>+</u> 15)	(<u>+</u> 241)	(<u>+</u> 451)	8							
3	7.4	58.5	626	959								
5	(<u>+</u> 2.9)	(<u>+</u> 31)	(<u>+</u> 435)	(<u>+</u> 407)	8							

 Table 5

 Soil Metal Concentrations (total) for Sites Sampled along the

¹ Means and standard errors (<u>+</u>1 s.e.) for sites sampled in 1987 by Keammerer.

 2 n = number of samples

Table 6. Screening Level Exposure and Risk Estimates for Cadmium, Lead, and Zinc by Mule Deer,Upper Arkansas River Drainage Study Area

	Vegetation				Soil				Water					Risk Es	timate			
Chemical	Conc. In Food (mg/kg) ¹	Food Ingestion Rate (kg/kg bw/d)	Fraction Food Ingested onsite	Fra ction Bioavailable	Intake from Food (mg/kg bw/day)	Conc. In Soil (mg/kg) ²	Soil Ingestion Rate (mg/kg bw/day)	Fraction Soil Ingested Onsite	Bioavailability	Intake from Soil (mg/kg bw/day)	Conc. In Water (mg/L) ³	Water Ingestion Rate (L/kg bw/day) ²	Fraction Water Ingested onsite	Bioavailability	Intake from Water (mg/kg bw/day)	Total Intake from Site (mg/kg bw/day)	TRV _{NOAEL} (mg/kg bw/day)	HQ
									REAC	40								
Cadmium	4.7	0.02	100%	100%	0.09	3.87	0.0004	100%	100%	0.002	0.0006	0.1	100%	100%	0.000	0.10	0.27	0.4
Lead	4.5	0.02	100%	100%	0.09	283	0.0004	100%	100%	0.1	0.001	0.1	100%	100%	0	0.20	5	0.04
Zinc	327.0	0.02	100%	100%	6.5	503	0.0004	100%	100%	0.2	0.03	0.1	100%	100%	0.003	6.74	45	0.1
									REAC	11								
Cadmium	6.0	0.02	100%	100%	0.12	19.2	0.0004	100%	100%	0.01	0.002	0.1	100%	100%	0.000	0.13	0.27	0.5
Lead	28.1	0.02	100%	100%	0.6	5202	0.0004	100%	100%	2.1	0.02	0.1	100%	100%	0.002	2.65	5	0.5
Zinc	322.0	0.02	100%	100%	6.4	5527	0.0004	100%	100%	2.2	0.8	0.1	100%	100%	0.08	8.73	45	0.2
									REAC	42								
Codmium	6.0	0.02	100%	100%	0.14	10.2	0.0004	100%	100%	0.01	0.0008	0.1	100%	100%	0.000	0.15	0.27	0.5
Caumum	0.9	0.02	100%	100%	0.14	19.5	0.0004	100%	100%	0.01	0.0008	0.1	100%	100%	0.000	0.15	5	0.5
Lead	37.1	0.02	100%	100%	0.7	916	0.0004	100%	100%	0.4	0.004	0.1	100%	100%	0	1.11	45	0.2
ZINC	501.0	0.02	100%	100%	10.0	1631	0.0004	100 %	100%	0.7	0.2	0.1	100%	100%	0.02	10.69	40	0.2
									REAC	43								
Cadmium	7.5	0.02	100%	100%	0.15	10.3	0.0004	100%	100%	0.004	0.0001	0.1	100%	100%	0.000	0.15	0.27	0.6
Lead	0.10	0.02	100%	100%	0.00	1061	0.0004	100%	100%	0.4	0.008	0.1	100%	100%	0.001	0.43	5	0.09
Zinc	492.0	0.02	100%	100%	9.8	1366	0.0004	100%	100%	0.5	0.2	0.1	100%	100%	0.02	10.41	45	0.2

¹ Mean + 1 standard error from Table 2

² From Table 3.

³ Values are mean total concentrations for low flow, 1994 to present (Period 3)



Figure 1. Cadmium Residues in Kidney Samples from the Upper Arkansas River Area



Figure 2. Lead Residues in Kidney and Liver and Associated Effects

NOAEL = No Observed Adverse Effects Level LOAEL = Lowest Observed Adverse Effects Level SOI = Somatic Organ Index



