Revised Final Baseline Risk Assessment for the Montana Pole NPL Site

Volume I: Baseline Risk Assessment

Submitted to:

Montana Department of Health and
Environmental Services
Helena, Montana

Submitted by:

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February 10, 1993

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February 12, 1993

Mr. Brian Antonioli
Montana Department of Health and Environmental Sciences
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Dear Mr. Antonioli:

Attached is the Final Baseline Risk Assessment for the Montana Pole site. The document has been revised to address comments submitted to MDHES by the Atlantic Richfield Company (ARCO). Below is a summary of changes to the document, and specific responses to ARCO comments which did not suggest changes in the document. The contents of this letter have been previously reviewed by yourself, and represent both the technical evaluations of CDM and the regulatory positions of the State.

ARCO Comments, General

A. A residential exposure scenario is inappropriate for assessing future exposures and risks at the Montana Pole site based on current zoning, flood plain designations, availability of more "desirable" locations, population trends in the Butte area and ordinances controlling the use of groundwater.

The State of Montana feels that evaluation of the residential scenario is appropriate for the Montana Pole site. Current zoning is a poor basis for assessing future land use. Zoning can be readily changed by local governments, and it might be anticipated that any development in the Butte area would be welcome and efforts made to accommodate. The flood plain designation for parts of the Site is also subject to change (e.g. with placement of flood control measures). This may be particularly applicable to the flood plain adjacent to the small drainage running from south to north through the middle of the site. The watershed for this area is expected to be small, and intermittent runoff might be easily controlled. The major area of the site which may be subject to restrictions based on the flood plain designation may be in the north, adjacent to Silver Bow Creek.

The desirability of the location for residences is only one of several criteria that might influence development. The fact that new homes are being built immediately adjacent to I-15 in Helena, MT suggests that factors such as price, proximity to work or family, etc. may override closeness to a major highway. Further, new homes continue to be built in Butte, despite the general lack of growth in the State. From the 1986-87 fiscal year to present, there were an average of 48 new residential home starts per year in Silver Bow County. In the latest year (1991-1992), 54 new starts were recorded. Thus, lack of population growth does not require that all new development cease.

Finally, city ordinances against the use of groundwater are, like zoning, subject to change, and do NOT prohibit residential development. Such ordinances might be considered in the evaluation of remedial alternatives as institutional controls, but it would be inappropriate to consider them as permanent limitations on the use of groundwater at the Montana Pole site for the purposes of a baseline risk assessment.

The State concludes that chances for residential development at the site are not demonstrably small enough to be ignored. Thus, future on-site exposures are assessed on the basis of a residential scenario which includes use of groundwater for all domestic purposes.

B. Documentation on plant uptake factors, the reference dose for dioxins and furans and the formation of TCDD in fires involving pentachlorophenol are not included in the document. Also, several references are missing from Section 9.

CDM has provided the requested documentation, along with extended discussions of plant uptake and the RfD for dioxins and furans, in the document text, and has placed the missing references into Section 9.

C. Documentation of the RME as defined in the text is not sufficient for complete evaluation. ARCO "statistics" suggest that the RME may be more conservative than suggested in the EPA definition.

CDM has added additional documentation to the text. Further, the "statistical analysis" provided in Table 1 (ARCO letter to Jane Stiles and Brian Antonioli, September 19, 1992) contains significant statistical errors which grossly overstate the conservativeness of the exposure calculations. Even ARCO's own consultants agree that the "ARCO method" is inappropriate and provide better (but still not completely appropriate) means to estimate how conservatism of an RME estimate (ARCO Risk Assessment Scoping Document for the Montana Pole and Treatment Plant, Appendix A, January 1991). This latter analysis, it should be noted, even with its flaws, predicted an RME estimate not more than 8 times the predicted upper 95 percent confidence limit. This finding is in accord with the position of the risk assessment, that uncertainty in the RME estimates themselves is unlikely to exceed an order of magnitude.

The State realizes that a degree of uncertainty approaching an order of magnitude in the risk assessment may be significant in terms of potential remediation costs. The State is prepared to considered uncertainties, both quantitative and qualitative, in decisions on risk management. The uncertainty analysis included in the risk assessment is intended to provide the State with the information necessary to appropriately interpret the quantitative risk estimates.

D. Documentation on how exposure point concentrations for dioxins and furans were calculated is not provided. The BRA should have considered that dioxin/furan samples were taken only from areas of know contamination, and that some detection limits for dioxins/furans were higher than the maximum actual detection. Inconsistencies between tables and text should be rectified.

The tables and text have been checked and modified as appropriate to provide a clearer explanation of the calculation of exposure point concentrations. In the original document, CDM did not use samples in calculation of exposure point concentrations if the samples were non-detect, and one-half the detection limit exceeded the maximum detect for the site. Moreover, the original document expressly acknowledges that the dioxin/furan data is likely to overestimate site-wide concentrations (page 7-30, original BRA). This text is retained.

E. Calculation of exposure point concentrations may have used incorrect statistical formulas, incorporated some inappropriately reported data for pentachlorophenol, and may have inappropriately used results from duplicates and samples from the same location in the calculations.

The text, tables and appendices have been modified as appropriate to make the calculations of exposure point concentrations more clear. In particular, the typographical error which left out the square root sign (page 5-28, original BRA) has been corrected, the exposure point concentration for PCP in groundwater has been recalculated without the misreported values, and an explanation of the incorporation of duplicates and samples for the same location has been provided. Where sample numbers exceed 30, the t value set at infinity is still used. Using actual t values when samples sizes are large makes essentially no difference in the calculated values, and, in fact, many tables of t values are truncated at n=30. For the sake of statistical "purity", where samples sizes are less than 30, exposure point concentrations were recalculated using the appropriate t value, although the changes in the resulting exposure point concentrations were minimal.

F. ARCO suggests that the BRA should provide more specific information on toxicity of organic chemicals to ecologic receptors.

Substantial additional information (e.g. see Table 8-3) has been added to Section 8 in all subsections where appropriate. The information is relevant to both organic and inorganic chemicals.

ARCO Comments, Specific

page 1-1 An accurate description of the operating period of the plant has been substituted into this section.

page 2-1 The text has been modified to provide an unambiguous description of the relationship between the Priority Soils OU and the Montana Pole site.

page 2-2 The document has been modified to clarify the discussions on air exposures. The changes are intended to present first the logic for considering such exposures (e.g. lack of vegetation and surficial contamination), then the screening level assessment for demonstrating that, in fact, the air pathway is not significant.

page 2-4 Citations are provided to support the statement that there is continued input of mining related contamination to the Silver Bow Creek drainage basin.

page 2-6 Reference to the groundwater use ordinance has been added to this section.

page 2-7 Reference to logging, and residential and industrial development, as well as mining/smelting, has been added to the discussion of impacts to local vegetation.

page 2-8 The reference to the Hydrometrics (1983) inventory has been deleted.

page 2-11 The vegetation unit A5 has been added to Figure 2-2.

page 2-12 ARCO again suggests that the residential scenario is inappropriate.

A response is provided above in A. Reference to the trespasser and recreational scenarios has been added to this section.

page 3-1 ARCO description of the butt treating process has been incorporated.

page 3-2 ARCO suggested addition regarding the conveyance by grant deed has been incorporated.

page 3-2 ARCO suggests that 2,3,7,8-TCDD should not be considered a COC for the site.

The State disagrees. TCDD was detected in screening quality data, is known to be formed in fires involving PCP, and has been reliably detected at many wood treating sites both in Montana and in other states. It should be noted that the inclusion of 2,3,7,8-TCDD in the calculation of exposure point concentrations makes essentially no difference in the final result. From a risk and remediation standpoint, 2,3,7,8-TCDD is not an issue.

page 4-1 ARCO requests copies of all data reports used in the preparation of this document.

Only data supplied by Keystone (1992) have been used in the quantitative risk evaluation. No additional data were used or are supplied here. The sample IDs presented in the BRA have been checked to ensure they reflect the appropriate samples in the RI report.

page 4-2 More detail on the impacts of screening quality data on the BRA has been included in Section 7.6.13. CDM feels that the current discussions in Section 4 are adequate.

page 4-3 ARCO suggests that metals should not be considered chemicals of concern for the site, and that text in this Section is inconsistent with their inclusion.

The rationale for including inorganic compounds is well described in the document and is carried consistently through the various sections. However, text has been modified slightly to ensure no misunderstanding.

page 4-22 The data for 2,3,7,8-TCDD has been rechecked and appropriate detections used in the estimation of total toxicity equivalents for dioxins/furans. It should be noted that TCDD contributes insignificantly to the total TEF for dioxins/furans.

page 4-22 ARCO again suggests that metals should not be considered chemicals of concern for the site, arguing that they are not site related, and that including them will provide no additional useful information to the management of the hazardous wastes sites in the Butte area.

Although the State agrees that the site is not a source of inorganics, it disagrees that this BRA will provide no additional useful information. The area of concern is

unlikely to be specifically assessed in other risk evaluations, and the activities necessitated by contamination related to the operations at the Montana Pole site could, in theory, interfere with activities necessitated by contamination from past mining/smelting operations. Thus, it is important to consider inorganic compounds in this assessment so that risk management decisions can be coordinated as necessary.

page 4-25 A section on data representativeness of dioxins/furans has been added to the text.

page 4-26 ARCO suggests that results from other risk assessments are not appropriately applied to the Montana Pole site.

This statement is in direct conflict with ARCO's position on evaluation of arsenic, "Since risks associated with inorganic chemicals are being assessed in separate investigations, no information will be lost if they are omitted for the MPTP BRA". In fact, the several operable units of the Silver Bow Creek NPL site and the Montana Pole NPL Site cannot be addressed in isolation, and various risk investigations must be applied over large areas of the site. Thus, references to the Lower Area One BRA are appropriate.

The State also recognizes that ARCO has submitted comments on the Lower Area One BRA, but neither agrees with all of ARCO's criticisms, nor feels that submission of these comments reflects on the appropriateness of citing the document.

page 4-28 The statement that the "site has a history of mining activity" was intended to refer to the area in general, not specific operations at the Montana Pole plant. The text has been changed to make this clear.

page 4-29 Smelter wastes could have migrated to the site in several ways, probably the most significant being via air emissions while smelters were active. The text has been expanded for additional clarity.

page 4-30 The statement that "it is not unreasonable to assume that data for other organic chemicals might be similar", was intended to reflect the expectation that similar seasonal patterns of contamination might be expected (i.e. that concentrations would be high in early summer and low in mid-fall). The text has been modified to clarify this interpretation.

page 5-2 The text has been changed to reflect the appropriate orientation of pole barns and fence.

page 5-2 The term "frequently" has been removed from the cited phrase, mainly because it is ill-defined, and is reflected quantitatively in the exposure assumptions.

page 5-3 ARCO implies that "swimming" requires constant total body immersion and that use of exposure assumptions based on "swimming" are inappropriate for Silver Bow Creek.

The State uses a broader definition to include activities such as inner-tubing, water-fights, "dam"-building, etc, where total body exposure and intimate contact with the water is likely though intermittent. It is likely that the assumptions used for exposed body surface area and incidental surface water ingestion overestimate actual likely exposures, but probably not as dramatically as suggested by ARCO. Additional text has been added to the document to clarify the interpretation of "swimming". A change in the rating for potential for overestimating actual exposures has not been made. The ARCO suggestion would indicate that ingestion rates are likely to be less than 0.5 ml/hr, less than one-tenth of a teaspoon for each hour of active play.

page 5-4 Table 5-1 has been changed to reflect no current potential for exposure to groundwater.

page 5-9 ARCO suggests an inconsistency in exposure frequencies for air and other exposure scenarios.

ARCO is incorrect in asserting that the document contains an air exposure scenario. The calculations for potential air exposure are clearly identified as a worst case screen. This fact is recognized by ARCO in other comments. As a screening calculation, 365 days/year is appropriate. No changes have been made to the text or calculations.

page 5-11 See response to comment on page 5-3. Text has been changed to clarify interpretation of "swimming".

page 5-11 ARCO suggests that the dermal exposure factor for PCP is too high, and that 0.01 should be substituted.

CDM considered ARCO's arguments in developing the original document, but concluded that the lower factor would not be representative of the site. The factor of 0.01 applies to PCP in ionized form, as might be expected for PCP *dissolved* in water. The unionized form is expected in fuel oil solution. Since PCP is found in association with fuel oil in soil and at the point of discharge to Silver Bow Creek, it is felt that a significant fraction of the exposure might be to the unionized form. Current evidence suggests that unionized PCP may have a dermal uptake factor as high as 0.5 (EPA 1991). Use of the generic value of 0.1 was thought appropriate, then, for a situation where exposures were likely for both ionized and unionized forms. Expanded discussion and additional references are provided in the new text.

page 5-12 Exposure duration for workers has been changed to 25 years.

page 5-13 ARCO suggests that the soil ingestion assumption for workers is too high.

Information referred to by ARCO (1991a) is inappropriate for evaluation of worker ingestion rates, and the rate of 10 mg/day cannot be accepted. Further, the rate of 50 mg/day is for a typical worker. For an industrial worker in a dusty outdoor environment, this may underestimate potential ingestion rates. EPA (1991) guidance

allows for modification of the worker ingestion rate based on site specific conditions, so long as exposure frequencies are appropriately adjusted. In fact, where contact is anticipated to be high (e.g. construction, landscaping, etc.) rates as high as 480 mg/day are suggested. The rate of 100 mg/day is thought to be a reasonable compromise between the "typical" worker, and the worker actively working in and with soil. Exposure frequencies have been adjusted downwards, since extensive soil contact is not expected on every workday.

page 5-5 Footnote "g" has been removed from Table 5-5.

page 5-15 ARCO suggests that a shorter exposure duration should be used for assessing non-cancer risks.

An appropriate exposure duration for assessing chronic toxicity is often a matter of judgement. For some chemicals, e.g. cadmium, it may be inappropriate to consider exposure durations less than the majority of a lifetime as chronic because of the way in which the RfD was derived. For others, such as methylmercury, a shorter time may be justified due to rapid bioaccumulation. For PCP, it is difficult to judge the shortest time in which chronic low level exposure might produce adverse effects. CDM assumed that 10 years was a reasonable exposure duration. ARCO is correct in suggesting that soil ingestion rates should be prorated for the time period chosen. This was, in fact, done for the original calculations. Exposure assumption tables and text have been changed to make this clear.

Here, and in other places in the text, ARCO also suggests that soil ingestion rates for adults and older children should be in the range of 10 mg/day.

EPA has addressed the issue of soil ingestion for sites in the Clark Fork drainage (EPA 1992), and this assessment uses the values for soil ingestion recommended. ARCO is referred to this document for the discussion of current evidence and the appropriateness of the values used.

page 5-17 ARCO again suggests that assumptions for body surface area for exposure to surface water are too conservative.

This issue has been addressed in response for comment on page 5-3.

In addition, the assumptions of 2 hour/day visitations and one-half of each visitors time spent in the vicinity of Montana Pole site are criticized as overestimates.

The State feels that the exposure time is realistic; depth of water may make some difference, but any waterway is viewed as attractive, especially to children. Moreover, spending time in the vicinity of the Montana Pole site may not be unusual, and represents best judgement. It is difficult, if not impossible, to accurately predict which sections of a creek will be the most attractive.

page 5-17 ARCO suggests that the BRA incorrectly assumes a total soil and sediment ingestion rate of 150 mg/day.

ARCO fails to consider the fraction of soil ingested from a contaminated source. If a child spent time in contact with both soil and sediment in one site visit, the scenario assumes that he/she would ingest 100 mg/day x 0.5, or 50 mg/day from each source. This would equal the total expected daily ingestion for the child for that day. This is thought reasonable, since visitors to the site are expected to engage in active play in or near the water, and can be envisioned to consume the bulk of their daily average ingestion for that day at the site.

Further, and more importantly, exposure frequency and time in the risk assessment are meant to be general averages. Some children may visit the site more often, but for shorter periods, others less frequently, but for longer times. Some children may play exclusively in the creek, others mainly on the banks and away from the creek, and still others would split time between the two. Thus, in an actual exposed population, sediment and soil exposures may not occur on the same days, may be more or less intense than the average assumptions indicate, and may vary significantly from exposure to exposure even for the same child. However, there are no data available to support a more detailed treatment of the trespasser scenario, and the assumptions used in the risk assessment present a pathway reduced to a minimum of assumptions. This is felt adequate for the purposes of the assessment and no quantitative changes have been incorporated in the revision.

page 5-21 ARCO requests additional explanation of the factor 0.7 used to represent fraction of "total soil contacts" attributed to contaminated sources.

This information has been added to the appropriate section in the document.

page 5-22 ARCO suggests that lack of correction for chemical loss from plants during washing and cooking is unrealistic.

The State disagrees in this case for two reasons. First, no contribution from chemicals deposited on or absorbed to plants is assumed. Only chemicals taken up into plant tissue are evaluated. Thus, one might consider that the BRA assumed that all chemicals adhered to the surface of the plants would be removed by washing. Second, many garden vegetables, including the leafy and root crops likely to be grown in the cool climate of Butte are often consumed raw. For many, this is the preferred form for consumption. Thus, loss of chemicals during cooking may not be of great significance. Moreover, there is little or no information on the efficiency of removal of chemicals of any kind during cooking. No correction for loss during cooking is thus considered appropriate both because of the tendency to eat many garden vegetables raw, and because of lack of data on which to base loss of PCP and other COCs.

page 5-33 ARCO again comments on a few PCP data points erroneously reported in the RI (Keystone 1991).

This issue was addressed in response to comment D above.

page 5-39 All apparent discrepancies have been corrected.

page 5-43 All apparent discrepancies have been corrected.

page 5-45 All calculations have been checked and corrected as necessary.

page 5-50 Table 5-20 has been footnoted to indicate which PAH compounds are included in the heading "PAH (Total non-carcinogen)".

page 5-51 The requested identifications for the North and South exposure areas have been added to Tables 5-23 and 24, the value for FI appropriately corrected and a description of skin surface areas added to the discussion of Table 5-4.

page 5-56 Plant uptake factors have been added to Tables 5-25 and 5-27. In addition, body weights for all uptake calculations have been checked and corrected as necessary.

page 5-65 The State's position on the likelihood of the future residential scenario has been added to the discussion of uncertainties in Section 7.6.20. This was deemed a more appropriate place for the discussion.

page 5-65 The soil ingestion rate for the farm worker has been deleted from Table 5-29.

page 5-66 A rating of low for uncertainty for the adult produce ingestion rate has been added to Table 5-29.

page 5-69 ARCO suggests that the 35% bioavailability for arsenic in soil in rabbits is incorrect.

The 35% represents a comparison with the absorption factor used in the BRA (80%). This is deemed appropriate. The text has been edited to clarify this calculation.

page 5-69 ARCO suggests that compounded overestimates of exposure might lead to estimates which overstate exposure by a factor of 40.

The State disagrees with ARCO's simplistic calculations and again refers to quantitative analyses of uncertainty (see response to general comment C) which indicate that the approaches used in the risk assessment are likely to produce estimates which meet the definition of RME. The discussion here has been modified slightly to provide clarity on the interpretation of uncertainty.

page 6-30 ARCO suggests addition of recent bioavailability studies to the lead toxicity profile.

These have been added along with appropriate discussion.

page 7-2 Text has been corrected.

page 7-6 Table 7-3 has been checked and corrected as appropriate.

page 7-10 The explanation of worker exposures in the north and south portions of the site has been clarified.

pages 7-10, 11, 13, 17, 22, 23 & 25 Text has been corrected.

page 7-29 ARCO requests additional mention of the fact that many exposure parameters are more likely to overestimate than underestimate risks.

An appropriate interpretation of the "conservativeness" of the BRA is included in the document. Additional text has been added to make clear the connection between the choice of exposure parameters and the RME.

page 7-32 ARCO again suggests that the residential drinking water scenario is unlikely.

This comment has been addressed above and in changes in the text, and appropriate discussion has been added to this section to provide consistency with earlier modifications.

page 8-2 ARCO complains that the Metro Storm Drain is misrepresented as surface water.

The State is does not believe that non-perennial drainages are excluded from the definition of surface water. The Storm Drain is appropriately described as an intermittent drainage in the text. No change has been incorporated into the revised document.

page 8-4 ARCO complains that Silver Bow Creek is not confined within the slag wall as described in the text.

The State believes that the description is accurate for much of the reach of the Creek in question. However, the description is of no consequence for the risk assessment, and ARCO's suggested description has been substituted in the revision.

page 8-5 ARCO complains that descriptions of organic pollutants and sources for mine wastes are incorrect.

The State disagrees, but has listed other potential sources of contaminants as appropriate in the revised text.

page 8-6 ARCO suggests that impacts due to activities other than mining are in part responsible for the lack of a fishery in Silver Bow Creek, and that German Gulch is not representative of stream conditions in unimpacted areas.

The State continues to believe that the major impacts to Silver Bow Creek are a result of mining activities. However, mention is made in the revised text to other possible contributors. The State also believes that German Gulch is an appropriate reference

stream for the purposes of this assessment (identification of appropriate target species). No change has been incorporated to the text.

page 8-6 ARCO requests reference to the lack of fishery between MPTP and Warm Springs Ponds.

Reference has been provided.

ARCO also suggests that references to Phillips (1985) are incorrect.

The State disagrees, but has modified the language slightly to better represent its beliefs and the findings reported by Phillips.

ARCO again suggests that factors other than metals have had significant impact on life in Silver Bow Creek.

The State believes that the impacts of metals on the Creek are appropriately presented in the revised document. No changes were made to specifically address this comment.

page 8-10 ARCO suggests that the BRA is inconsistent in describing fisheries in Silver Bow Creek.

The State disagrees. The few trout which may be found at Warm Springs Ponds do not suggest a viable fishery either here or elsewhere in the Creek. No change has been incorporated into the revised text.

page 8-11 ARCO again suggests contaminant sources other than mine wastes as contributors to impacts on stream life.

See response to comment on page 8-6.

page 8-13 ARCO suggests that Figure 8-3 should be modified to include a direct soil ingestion pathway.

This change has been made.

page 8-16 ARCO suggests that the description of the contribution of tailings to surface soil is overstated.

The comment was intended to refer only to the northern area of the site, where tailings have been deposited as sediment from the Creek. The text has be changed to make this clear.

page 8-18 ARCO again suggests including and using toxicologic information appropriate for ecological receptors for the site.

See response to general comment F.

ARCO also requests literature references for the terrestrial toxicity data.

The requested references have been emphasized, and uncertainties clarified in the revised text.

page 8-23 ARCO requests specific discussion of various toxicity values referenced in Table 8-2.

The requested information has been added to the Table, and discussed in the text.

page 8-26 ARCO requests additional text on critical toxicity values used for calculating toxicity criteria.

The State believes these are adequately described in the toxicity profile, exposure assessment and uncertainty analysis subsection of the ecological assessment.

page 8-27 ARCO requests that the aquatic toxicity data for PCP should be updated.

The original document used AWQC from EPA (1986) as suggested. References have been checked to ensure their accuracy.

Appendix C ARCO suggests that the plant uptake factor for PCP is too high, and references a value of 0.001 discussed in their scoping document.

CDM has investigated plant uptake of PCP in additional depth and a detailed discussion of the plant uptake factor is now included in Appendix C, and is summarized in the exposure assessment. Changes made in the assessment of potential exposures via the garden vegetable pathway include an increase in the plant uptake factor for PCP to 3, a decrease in the exposure duration to 6 years, and elimination of leafy and vine crops from consideration. The result is a decrease in exposures and risks for this pathway of over 10 fold for PCP. Rationale for all changes is provided in Appendix C.

Sincerely,

CAMP DRESSER & McKEE INC.

James M. LaVelle
Senior Toxicologist

Executive Summary

EXECUTIVE SUMMARY

INTRODUCTION

The Montana Pole National Priority List (NPL) site was identified by EPA as a Superfund site in 1986. The site obtained NPL status as a result of chemical contamination associated with a timber treatment plant that operated nearly continuously from 1947 through 1983. The site is approximately 45 acres located in the SE 1/4, Section 24, T3N, R8W; specifically, at 202 W. Greenwood Avenue in Butte, Montana. With the exception of coal tar creosote used to a limited extent in 1969, the primary solution used to treat timber products consisted of 5 percent pentachlorophenol (PCP) combined with 95 percent petroleum (various fuel oils). Past use of PCP, creosote and petroleum-related products, including dioxin and polycyclic aromatic hydrocarbons (PAHs), have contaminated surface soils, subsurface soils, sediments, groundwater, and surface water at the Montana Pole site. Inorganic wood treating chemicals have apparently never been used at the site.

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended by the Superfund Amendment and Reauthorization Act (SARA), stipulates that remedies at Superfund sites must be protective of both human health and environmental receptors. To evaluate the degree to which a remedial alternative is protective, it is necessary to assess both existing environmental and human health risks (no action alternative or baseline) and potential risks for proposed remediation alternatives. To determine final clean-up criteria, these risks are evaluated by regulatory authorities in conjunction with applicable or relevant and appropriate requirements (ARARs), technological limitations, and other site-specific factors.

Scope of Human Health Risk Assessment

The overall approach to the human health risk assessment follows guidance provided in "Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A) (EPA 1989a). This document provides guidance on all aspects of human health risk assessment, including: evaluating available data and identifying chemicals selected for quantitative analysis, developing exposure scenarios that depict expected exposure conditions, assessing toxicity of chemicals under

expected exposure conditions, and combining this information to estimate carcinogenic and noncarcinogenic health risks.

Site-specific exposure parameters and assumptions provided by MDHES, and Region VIII EPA, have also been incorporated into this assessment.

Scope of Ecological Risk Assessment

The ecological risk assessment generally follows guidance provided in "Risk Assessment Guidance for Superfund: Volume II — Environmental Evaluation Manual (Part B)" (EPA 1989b). This document provides guidance regarding the nature of an ecological risk assessment and the types of information that should be included. It does not, however, provide detailed guidance regarding the implementation of specific ecological risk assessments. Ecological risk assessments are, by their nature, very site-specific. Issues such as exposure point concentrations, potential direct and indirect ecological receptors, bioaccumulation and biomagnification within ecosystems, and chemical toxicity to specific ecological receptors must be considered on a site-specific basis. The degree to which an ecological risk assessment should be qualitative or quantitative also depends upon many site-specific ecological factors as well as chemical-specific factors. This ecological risk assessment contains both qualitative and quantitative aspects.

Uncertainty Analysis

There is uncertainty associated with each estimated exposure parameter or toxicity value. In order to perform a quantitative risk assessment, it is necessary to make numerous quantitative assumptions regarding the type and extent of exposure that an individual or organism may receive, and the amount of exposure required to elicit an adverse effect.

In this assessment, uncertainties are addressed qualitatively in each major section of the report. For many of the selected parameter values, review of the associated uncertainties indicates that the selected value will tend to overestimate exposure or risk. Thus, the selected values are "conservative," or likely to be overprotective rather than underprotective of potential receptors.

ENVIRONMENTAL SETTING

The Montana Pole site is located in Butte, Montana within the area designated as the Butte Priority Soils operable unit of the Silver Bow Creek NPL site. In general, the environmental setting for the site is similar to that for the Butte area as a whole. Major exceptions include wetland areas along Silver Bow Creek and current vegetation, both of which are more specific to the site. In this section, Climate, geology, hydrology, vegetation, wetlands, and current land use and demography are discussed in relation to possible exposure pathways for current and future visitors, workers or residents on the site.

Climate

The climate within Butte and the surrounding vicinity is characterized by short, cool, dry summers and cold winters. Total annual precipitation measured at the Butte airport averages 11.7 inches.

Average annual temperatures measured at the Butte airport range between 34.0 and 42.6°F, with a mean of 38.9°F. The normal frost-free period is approximately 60 days.

The cool climate, short frost-free period and long winters all suggest that (1) exposure to soils outdoors may be limited by frozen ground conditions and snow cover, and (2) gardening may be limited both in types of crops and in extent by a short growing seasons.

Geology

The Butte area adjacent to the Montana Pole site is underlain by granitic rocks of the Boulder Batholith. A weathered zone is generally present in the upper 100 to 200 feet of the bedrock. Unconsolidated/alluvial sediments of fluvial or alluvial fan origin and Tertiary to Quaternary age are present in the valleys and drainages throughout the area. Alluvial thickness at the Montana Pole site ranges from 11 to over 47 feet. The water table is found at approximate depths of 5 to 10 feet below ground surface.

Hydrology

The Montana Pole site lies within the upper Silver Bow Creek drainage basin. Silver Bow Creek originates in the mountains northeast of Butte. Both above and below the Montana Pole site, the drainage basin has been subjected to contamination (primarily arsenic, copper and lead) by historic mining and mineral processing activities in and around Butte.

Surface Water

Surface water runoff is characterized by high snowmelt flows in April through early June and low flows during the late summer months of July and August. Silver Bow Creek is a losing stream adjacent to the site. However, groundwater discharge occurs along the northern boundary of the site. Direct seepage of non-aqueous phase liquids (NAPLs) and dissolved phase contaminants into Silver Bow Creek occurs from the site. The Montana Pole site drains from the south to the north into Silver Bow Creek. Surface discharge occurs during storm events primarily through a drainage ditch which runs through the site, including through contaminated areas.

Groundwater

Groundwater in the Butte area occurs in two water-bearing units: 1) the unconsolidated sediments associated with the Tertiary and Quaternary age valley fill deposits (alluvial aquifer); and 2) the weathered and fractured bedrock deposits associated with the Boulder Batholith. The depth to water in the unconsolidated valley fill ranges from two to greater than 30 feet (CH2M-Hill and Chen-Northern 1990). Well yields for the valley typically range from 3 gallons per minute (gpm) to over 30 gpm.

The depth and porosity of the alluvial aquifer indicate that wells completed in this zone could yield sufficient water for domestic purposes. Thus, it is reasonable to assume future use of the groundwater for drinking water.

Vegetation

Plant communities associated with Silver Bow Creek have been extensively affected by past urban and industrial activity. The major impact to the plant communities near the Montana Pole site has been from industrial facility construction. Inspection of the floodplain boundary of the site indicates that another major impact to plant communities has been caused by deposition of metal-enriched waste materials (mill tailings) covering the original alluvial soils.

The disturbed, sparse vegetation which occurs over much of the Montana Pole site suggests that wind-blown dust could be a significant transport mechanism for site-related contaminants. As previously mentioned, low annual precipitation may also contribute to dusty conditions.

Wetlands

An intermediate-level wetland delineation performed at the Montana Pole site indicated nine vegetation units with four delineated as wetlands. These four include:

- An isolated depression approximately 1 acre in size bordered by a transformer storage yard and a railroad track embankment.
- An approximately 1,800-foot segment of Silver Bow Creek with associated streambank vegetation occupying approximately 2.5 acres and composed entirely of herbaceous emergent vegetation.
- An isolated stand of shrubs and herbaceous vegetation, approximately 0.1 acres in size, bordering Silver Bow Creek.
- An isolated depression approximately 0.05 acres in size which collects runoff from surrounding higher ground including the interstate embankment.

Land Use/Demography

Much land use in the vicinity of the Montana Pole site is industrial, usually associated with past and present mining activities. Colorado Smelter wastes and mill tailings are located to the west and north of the Montana Pole site. A federal manganese stockpile site and the former Butte Reduction Works are located directly north, while the Montana Power Company's transformer maintenance and storage

facility is located to the north and east of the site. A partially reclaimed gravel pit and a blasting and explosive powder company (LaVelle Powder) are located to the south of the site. An overpass for U.S. Interstates 15 and 90 crosses the middle of the site, in an east-west direction. The site is surrounded on both the east and west sides by active railroad lines, some of which served the facility.

Residential areas are located within one quarter mile east and west of the site. Though population of Silver Bow County has been steadily decreasing since about 1960, new residential development continues with about 48 new housing starts per year since 1986. There is one on site resident whose house is located within the property line noted adjacent to Greenwood Avenue. There is also an auto body shop and an architect's office located on site.

The proximity of commercial and residential properties is indicative of mixed land use in the area. Future use of the area, including the Montana Pole site is also likely to be mixed. This suggests for future land use that both worker and residential exposure scenarios need be provided to the risk manager to assist in risk management decisions. For assessing current land use, a recreation/trespasser scenario is also indicated.

DATA EVALUATION

Sampling Efforts

The most recent sampling program, conducted by ARCO Coal Company under the direction of MDHES, includes priority pollutant analyses according to Contract Laboratory Program (CLP) procedures. These sampling results provide data that are used in this BRA because this sampling program provides a more current and accurate indication of the extent of contamination present at the site. Chemicals found in soil and groundwater include phenolics, polycyclic aromatic hydrocarbons (PAHs), dioxins/furans, and various metals. In surface water, phenolic compounds, PAHs, and metals are found; in sediments two dioxin/furan isomers, two phenolic compounds, and several metals were detected.

Chemicals of Concern For Human Health

Chemicals that are present on site as a result of Montana Pole operations and that are likely to contribute to risk are identified as COCs. In addition, some metals not believed to be associated with Montana Pole operations are evaluated to provide a basis for comparison with mining-related sites immediately adjacent. Chemicals that are selected after this screening are quantitatively evaluated.

Cocs for Human Health

Chemicals detected on the Montana Pole site are screened as COCs based upon their toxicity to humans or laboratory animals (when human data were unavailable), their maximum concentrations measured in each media, and their frequency of detection. The same screening criteria applied to soil and groundwater are also applied to surface water and sediment. This makes the screen very conservative for these media, since it is unlikely that exposures to either surface water or sediment would occur over an extended time period on a daily basis.

Based on the above described process and some special considerations, the chemicals listed in Table 1 are considered COCs for human health for the Montana Pole site.

EXPOSURE ASSESSMENT

Potential pathways by which human and environmental receptors could be exposed to contaminants at, or originating from, the Montana Pole site are provided in Table 2, and include incidental exposure to soil, surface water and sediment, use of groundwater for domestic purposes and consumption of vegetables grown in contaminated soils. In identifying potential pathways of exposure, both current and likely future land use of the site and surrounding study area are considered. Proximity to Silver Bow Creek and lack of access control for much of the site suggests that trespassers may frequent the site and be exposed to contamination. Past industrial use of the site suggests that future on site workers might be exposed to site-related contaminants while at work. Finally, the existence of residential land use immediately adjacent to and on site suggest the potential for future residential development.

TABLE 1

COCs FOR HUMAN HEALTH AT THE MONTANA POLE SITE

GROUNDWATER

Arsenic
Chromium (VI)
Copper
Lead
Manganese
2-chlorophenol
4-chloro-3-methylphenol
2,4-dichlorophenol
2,4-dinitrophenol
2,4-dinitrotoluene
Dioxins/Furans
2-methyl-4,6-dinitrophenol
Acenaphthene
Anthracene
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(g,h,i)perylene
Benzo(k)fluoranthene
Chrysene
Dibenzo(a,h)anthracene
Fluoranthene
Fluorene
Indeno(1,2,3-cd)pyrene
2-methyl naphthalene
Naphthalene
Phenanthrene
Pyrene
Pentachlorophenol
2,3,5,6-tetrachlorophenol
2,4,6-trichlorophenol

SOIL

Arsenic
4-chloro-3-methylphenol
Dioxins/Furans
2-methyl-4,6-dinitophenol
Anthracene
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Indeno(1,2,3-cd)pyrene
Pentachlorophenol
2,4,6-trichlorophenol

SURFACE WATER

Arsenic
Copper
Lead
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Chrysene
Dibenzo(a,h)anthracene
Pyrene
Pentachlorophenol
Zinc

SEDIMENTS

Arsenic
Dioxins/Furans
Lead

TABLE 2

POTENTIAL PATHWAYS OF EXPOSURE TO CHEMICALS FROM THE MONTANA POLE NPL SITE UNDER FUTURE LAND USE CONDITIONS

Exposure Medium	Potential Routes of Exposure	Potential Receptors	Potential for Chemical Exposure
Soil	Dermal absorption, incidental ingestion	Future on-site residents, workers	High. Children are especially likely to play on soils.
Surface Water and Sediments in Silver Bow Creek	Dermal absorption, incidental ingestion	Future on-site residents, workers	High. Children are especially likely to swim and wade in creek.
Air	Inhalation of volatile organics and fugitive dust	Future on-site residents, workers	High. Potential for fugitive dust generation and volatilization of organics from soil is high.
Groundwater	Ingestion	Future on-site residents, workers	High. Contaminants are present in groundwater.[a]
Produce	Ingestion	Future on-site residents, workers	Moderate. Uptake of contaminants in groundwater and soils by plants is likely to occur.[b]

[a] Assumes that drinking water wells may be installed in the future. Actual potential for on site residential development appears to be low.

[b] Assumes that gardening in the Butte area will be limited by climate.

The highest exposures are estimated for future on site residents, and this is expected since such individuals are expected to contact contamination much more frequently than either workers or site trespassers. For residents, exposure via the groundwater pathway is much greater than for any other pathway (Table 3). Potential future use of the alluvial aquifer for domestic purposes represents the highest exposure potential for the site. Chemicals for which exposure is highest include pentachlorophenol (PCP), the major wood-treating chemical used on site, and PAHs which are constituents of creosote. Creosote was also used to treat wood at the Montana Pole site for a brief period.

TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to examine the potential for each chemical to cause adverse effects in exposed individuals and to provide an estimate of the dose-response relationship between the extent of exposure to a particular contaminant and adverse effects. Adverse effects include both noncarcinogenic and carcinogenic health effects in humans.

Carcinogenic Effects

Of the COCs for the site, several, including PCP, dioxins/furans, 2,4,6-trichlorophenol, some PAHs and arsenic, are known or suspected human carcinogens. The most potent of these chemicals are the dioxins/furans. Significantly less exposure to these compounds is predicted to be necessary to produce the same level of cancer risk. Some of the PAHs are also relatively potent carcinogens, though less so than the dioxins/furans. PCP, for which site-related exposures may be greatest, is a less potent carcinogen than either dioxins/furans or the carcinogenic PAHs. Arsenic, while a potent carcinogen, is not considered to have been released from the Montana Pole and Treatment plant in the past.

Noncarcinogenic Effects

The potential for COCs to produce noncancer effects varies widely. Dioxins/furans are extremely potent compounds, and only small exposures may be associated with increased risk of adverse effects. Other compounds, such as copper, are relatively non-toxic, and only produce adverse effects at much

TABLE 3

ESTIMATED CHRONIC DAILY INTAKES FROM INGESTION OF GROUNDWATER FOR FUTURE ON-SITE RESIDENTS

Chemical	Chemical Concentration Cs(ug/L)	Ingestion Rate IR(L/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(mg/ug)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	6.5E+03	2	350	30	1.0E-03	NA	1.0E+00	59	25550	9.06E-02
Dioxins/Furans(TEFs)	5.3E-02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	7.35E-07
2,4,6-Trichlorophenol	2.3E+02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	3.23E-03
Benzo(a)pyrene(TEFs)	3.0E+02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	4.23E-03
Arsenic	2.3E+01	2	350	30	1.0E-03	NA	1.0E+00	59	25550	3.22E-04
Noncarcinogenic Exposure										
Pentachlorophenol	6.5E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	6.57E-01
Dioxins/Furans (TEFs)	5.3E-02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	5.33E-06
2,4,6-Trichlorophenol	2.3E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.34E-02
PAH (Total non-carcinogen)(a)	3.0E+05	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.02E+01
2-chlorophenol	4.0E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	4.08E-03
Arsenic	2.3E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.36E-03
Copper	1.4E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	1.41E-02
Manganese	2.5E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.52E-01
Lead	3.0E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.00E-03
Chromium	2.8E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.87E-03
2,4-Dichlorophenol	9.9E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	9.94E-02
2,4-Dinitrotoluene	2.2E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.23E-02
4-Chloro-3-methylphenol	3.3E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.34E-02
2-Methyl-4,6-dinitrophenol	3.8E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.86E-02
2,3,5,6-Tetrachlorophenol	3.1E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.12E-01

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x FI x ABS/ BW x AT

NA = Not Applicable

(a) All PAHs detected except benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(g,h,i)perylene, chrysene, dibenz(a,h) anthracene and indeno(1,2,3-cd)pyrene

higher exposure levels. In general, exposures estimated in this assessment for noncarcinogenic effects are sufficiently low such that only the more potent toxicants could present a significant risk.

RISK CHARACTERIZATION

Cancer Risk Estimates

To evaluate potential cancer health risks related to the Montana Pole site, chemical exposures calculated are multiplied by cancer slope factors to develop upper range incremental lifetime cancer risks. Incremental cancer risks in the range of 10^{-6} to 10^{-4} may be characterized as acceptable by the EPA depending on the nature of the site and the COCs.

Cancer risks for exposure to COCs in groundwater are the greatest for any pathway. Only future residents are evaluated for this exposure. Risks exceed 1×10^{-2}, the upper limit for risk predictions using current models (Table 4). Significant risk is attributable to PCP, even though this chemical is one of the least potent carcinogens among the COCs. This finding attests to the very high concentrations of PCP found in the groundwater beneath the Montana Pole site. Dioxins/furans also contribute significantly to risks. These compounds are expected contaminants of technical grade PCP which is used for wood treating.

The consumption of homegrown produce also contributes significant potential risk for future residents (Table 5). Risks for this pathway, however, may be only about 1 percent of the risks from drinking contaminated groundwater. This is due to a reduction in exposure concentration for most COCs (produce concentrations are estimated to be less than soil concentrations), and fewer days of exposure (the growing season in Butte is limited by climate). Risks from exposure to PCP and dioxins/furans are the greatest for this pathway (risks of 9×10^{-4} and 1×10^{-4}, respectively) for the southern area of the site. Exposures in the northern area, between the Interstate and Silver Bow Creek, gave rise to similar overall cancer risk estimates, although the risks for individuals compounds varied somewhat.

Risks associated with direct contact with soil (incidental ingestion and dermal contact) are associated with significantly less risk for all exposure scenarios than those estimated for groundwater and produce consumption. However, for workers and trespassers, these pathways are major contributors

TABLE 4

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF GROUNDWATER FOR FUTURE ON-SITE RESIDENTS

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	9.06E-02	1.20E-01	1.09E-02
Dioxins/Furans(TEFs)	7.35E-07	1.50E+05	1.10E-01
2,4,6-Trichlorophenol	3.23E-03	1.10E-02	3.55E-05
Benzo(a)pyrene(TEFs)	4.23E-03	7.30E+00	3.09E-02
Arsenic	3.22E-04	1.75E+00	5.64E-04
		Total Cancer Risk	1.53E-01
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	6.57E-01	3.00E-02	2.19E+01
Dioxins/Furans (TEFs)	5.33E-06	1.00E-09	5.33E+03
2,4,6-Trichlorophenol	2.34E-02	NA	NA
PAH (Total non-carcinogen)	3.02E+01	4.00E-02	7.54E+02
2-chlorophenol	4.08E-03	5.00E-03	8.17E-01
Arsenic	2.36E-03	3.00E-04	7.86E+00
Copper	1.41E-02	4.00E-02	3.52E-01
Manganese	2.52E-01	1.00E-01	2.52E+00
Lead	3.00E-03	NA	NA
Chromium	2.87E-03	5.00E-03	5.73E-01
2,4-Dichlorophenol	9.94E-02	3.00E-03	3.31E+01
2,4-Dinitrotoluene	2.23E-02	6.80E-01	3.27E-02
4-Chloro-3-methylphenol	3.34E-02	NA	NA
2-Methyl-4,6-dinitrophenol	3.86E-02	NA	NA
2,3,5,6-Tetrachlorophenol	3.12E-01	3.00E-02	1.04E+01
		Total Hazard Index	6.16E+03

NA = Not Applicable

TABLE 5

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF HOME-GROWN VEGETABLES FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Total Vegetable Pathway CDI (mg/kg-day)	Slope Factor (mg/kg-day)	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	7.43E-03	1.20E-01	8.92E-04
Dioxins/Furans (TEFs)	7.20E-10	1.50E+05	1.08E-04
2,4,6-Trichlorophenol	1.90E-03	1.10E-02	2.10E-05
Benzo(a)pyrene(TEFs)	6.35E-07	7.30E+00	4.63E-06
Arsenic	2.65E-04	1.75E+00	4.64E-04
	Total Cancer Risk		1.49E-03
		RfD (mg/kd-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	1.62E+00	3.00E-02	5.39E+01
Dioxins/Furans(TEFs)	5.21E-09	1.00E-09	5.21E+00
2,4,6-Trichlorophenol	1.38E-02	NA	NA
Anthracene	7.97E-06	3.00E-01	2.66E-05
Arsenic	1.92E-03	3.00E-04	6.40E+00
Cadimum	7.03E-04	5.00E-04	1.41E+00
	Total Hazard Index		6.69E+01

NA=Not Applicable

to overall risks, since groundwater and produce ingestion are not considered (Tables 6 and 7). Overall, cancer risk estimates for workers and trespassers are up to one thousand times less than those for future residents, and fall near the upper limit of the EPA risk range of 10^{-4} to 10^{-6}.

Noncarcinogenic Health Risks

To evaluate non-cancer health risks, chemical exposure is compared to one of several types of toxicity criteria to determine if the exposure is within a range of exposure which is unlikely to cause adverse health effects. The potential for noncarcinogenic health effects is evaluated by dividing a chemical-specific exposure level by a chemical-specific reference dose. The resulting hazard index (HI) assumes that there is a level of exposure (RfD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the CDI exceeds the RfD (i.e., HI > 1), a potential for non-cancer health effects may exist.

The pattern for non-cancer risks is similar to that for carcinogenic risks. Risks are greatest for future residents and for groundwater and produce ingestion pathways. For groundwater, dioxins/furans, non-carcinogenic PAHs and PCP all have hazard indices (HIs) exceeding unity (533, 75, and 22 respectively, Table 4). Risks for adverse effects, which could include effects on the liver, kidneys, adrenal glands and other organs may be significant for these compounds.

For produce ingestion, HIs for dioxins/furans are smaller, but still exceed one. For example the HI for dioxins/furans is 6 (Table 5). However, because of an high estimate for PCP absorption through plant roots, the HI for PCP is higher (64) for this pathway. Only anthracene among the PAHs is a COC for soil, and it is present in quantities too small to present significant risk. The only other possible contributor to risk via this pathway is arsenic (HI=7). This chemical is not thought to emanate from the Montana Pole site.

For the direct soil contact pathways, risks (HIs) are substantially lower. For future residents, HIs for most chemicals are less than one, and no increased risk for adverse effects in anticipated. However, in several instances (Tables 6 and 7), exposures to dioxins/furans could result in HIs at or slightly above one. This suggests that dioxin/furan concentrations in soils at the Montana Pole site may be associated with potential exposure at the threshold for possible adverse effects.

Table 6

Summary of Estimated Total Risks
for Current On-site Trespassers

Chemical	Soil Ingestion	Dermal Contact with Soil	Sediment Ingestion	Surface water Ingestion	Dermal Contact with Surface Water
	Incremental Lifetime Cancer Risk				
Carcinogenic Exposure					
Pentachlorophenol	1.25E-06	9.40E-06	NA	3.33E-06	3.65E-07
Dioxins/Furans(TEFs)	6.44E-07	4.82E-07	2.47E-09	NA	NA
2,4,6-Trichlorophenol	5.38E-10	4.03E-09	NA	NA	NA
Benzo(a)pyrene(TEFs)	7.27E-09	NA	NA	4.35E-07	NA
Arsenic	1.88E-06	1.76E-06	NA	NA	NA
Total Cancer Risk	3.78E-06	1.16E-05	2.47E-09	3.77E-06	3.65E-07
			Total Cancer Risk for all Media		1.96E-05
Noncarcinogenic Exposure	Hazard Index	Hazard Index	Hazard Index	Hazard Index	Hazard Index
Pentachlorophenol	2.03E-03	1.52E-02	NA	5.40E-03	5.90E-04
Dioxins/Furans (TEFs)	2.50E-02	1.88E-02	9.59E-05	NA	NA
2,4,6-Trichlorophenol	NA	NA	NA	NA	NA
2-methyl-4,6-dinitrophenol	NA	NA	NA	NA	NA
Anthracene	3.25E-08	NA	NA	NA	NA
Arsenic	2.09E-02	1.96E-02	NA	NA	NA
Cadmium	3.02E-04	2.26E-04	NA	NA	NA
4-Chloro-3-methylphenol	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	1.24E-05	NA
Total Hazard Index	4.82E-02	5.38E-02	9.59E-05	5.41E-03	5.90E-04
			Total Hazard Index for all Media		1.08E-01

NA = Not Applicable

TABLE 7

Summary of Estimated Total Risks for Future On-Site Workers

Chemical	Soil Ingestion	Dermal Contact with Soil	Sediment Ingestion	Surface water Ingestion	Dermal Contact with Surface Water
	Incremental Lifetime Cancer Risk				
Carcinogenic Exposure					
Pentachlorophenol	8.03E-06	3.63E-05	NA	NA	NA
Dioxins/Furans(TEFs)	4.12E-06	1.86E-06	NA	NA	NA
2,4,6-Trichlorophenol	3.44E-09	1.56E-08	NA	NA	NA
Benzo(a)pyrene(TEFs)	4.65E-08	NA	NA	NA	NA
Arsenic	1.20E-05	6.80E-06	NA	NA	NA
Total Cancer Risk	2.42E-05	4.50E-05	0.00E+00	0.00E+00	0.00E+00
			Total Cancer Risk for all Media		6.92E-05
Noncarcinogenic Exposure	Hazard Index	Hazard Index	Hazard Index	Hazard Index	Hazard Index
Pentachlorophenol	6.24E-03	2.82E-02	NA	NA	NA
Dioxins/Furans (TEFs)	7.69E-02	3.48E-02	NA	NA	NA
2,4,6-Trichlorophenol	NA	NA	NA	NA	NA
2-methyl-4,6-dinitrophenol	NA	NA	NA	NA	NA
Anthracene	9.99E-08	NA	NA	NA	NA
Arsenic	6.42E-02	3.63E-02	NA	NA	NA
Cadmium	9.72E-04	4.19E-04	NA	NA	NA
4-Chloro-3-methylphenol	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	NA	NA
Total Hazard Index	1.48E-01	9.97E-02	0.00E+00	0.00E+00	0.00E+00
			Total Hazard Index for all Media		2.48E-01

NA = Not Applicable

ECOLOGICAL ASSESSMENT

The objective of the ecological risk assessment (ERA) is to evaluate the potential effects of contaminated surface water, soils, sediments and groundwater from the Montana Pole NPL site on terrestrial and aquatic plants and animals. Protection of the non-human population, community, or ecosystem is the usual focus of ecological risk assessments. The lack of appropriate toxicity data for wildlife and other environmental receptors at the population level makes quantitative inferences at this level or above difficult. This assessment therefore, addresses effects on populations and communities in a more qualitative fashion.

The ecological risk assessment is complementary to the human health risk assessment for this site. Many initial steps used to evaluate human risks are similar for assessment of ecological impacts. These include:

- Identification of potential receptors (e.g., wildlife, fisheries, and threatened and endangered species)
- Identification of valued habitats such as wetlands in the project area or off-site areas that could be affected by contaminant movement off-site
- Assessment of the potential for exposure; discussion of the toxicity of the site contaminants to potential receptors
- Characterization of the potential current and future risk or threat to the environment from contaminants at the site.

Potential Receptors

Aquatic Communities

Silver Bow Creek adjacent to the Montana Pole site and downstream to the Warm Springs Ponds does not support a fisheries population. Westslope cutthroat trout (*Oncorhynchus clarki lewisi*) and bull trout (*Salvelinus confluentus*) are reported to have once been caught in the vicinity of Butte prior to intensive mining activities. Prior to 1975, severe mining-related pollution in much of the upper Clark Fork Rivers drainage had rendered the system incapable of supporting a viable fishery. Excessive metals deposits still prevent the establishment of a fishery in Silver Bow Creek.

Benthic invertebrate communities and algae have re-established themselves within the study area since the cessation of direct mine waste water discharges to Silver Bow Creek. Mayflies, caddis flies, and stoneflies have been collected, although they demonstrate low density and limited diversity. No known surveys on benthic communities have been conducted within the study area since about 1984. The current density and diversity of this aquatic community is unknown.

No terrestrial communities within the Montana Pole site have been identified as critical habitat or communities of special concern. No rare or endangered plants were identified within the study area boundaries of the Lower Area One (LAO) Operable Unit of the Silver Bow Creek NPL site, nor downstream of this study area. Vegetation growing adjacent to Silver Bow Creek within the Montana Pole site is limited to willows (*Salix exigua*) and grasses. Shrubs indicative of dry conditions are found throughout the area.

Chemicals Selected for the Ecological Risk Assessment

From the list of chemicals expected to occur at the Montana Pole site, seven chemicals or chemical groups are selected for evaluation in this ERA, based upon mobility and persistence, bioaccumulation potential, adequacy of toxicological data to evaluate risks, comparisons of maximum detected concentrations with toxicity criteria values, and the use of these chemicals in the wood-treating process at the Montana Pole and Treatment Plant site. These chemicals are:

- Polycyclic Aromatic Hydrocarbons (PAHs)
- Pentachlorophenol (PCP)
- Dioxin/Furans
- Arsenic
- Cadmium
- Copper
- Zinc

Ecological Toxicity Assessment

Toxicity assessment is typically comprised of two elements. The first, hazard identification, is intended to characterize the nature and extent of biota health hazards associated with chemical exposures. The second, a dose-response assessment, determines the relationship between the magnitude of exposure to a chemical and the occurrence of adverse health effects. For the Montana Pole site, each chemical of concern is evaluated for toxicity values for use in risk characterization.

Ecological Risk Characterization

The ecological risk evaluation is similar to human risk evaluation, in that exposure assumptions and toxicological data are combined with site data to estimate risk. However, nonhuman receptors vary greatly in physiology and behavior, and thus it is difficult to quantify risk. Thus, this ecological risk assessment is a qualitative discussion of potential risks and how these risks might affect biological receptors at the Montana Pole site.

Risks to Aquatic Life

Metals and arsenic found in sediments and surface water in Silver Bow Creek may be a primary reason for the lack of diversity and productivity of the reaches of Silver Bow Creek adjacent to the site. Elevated concentrations of these contaminants come from historical mining activity in the upper reaches of the Silver Bow Creek drainage. The Montana Pole wood treating plant is not considered to be a source of metals contamination in the area.

Dioxins/furans, PAHs and PCP have all been detected in surface water and/or sediments in stream reaches adjacent to the Montana Pole site. A seep where groundwater discharges into the creek can be detected visually near the location of surface water sampling station SW-05. Thus chemicals are currently being released to surface water, and may pose a threat to aquatic life.

The stress on the Silver Bow Creek system from inorganic contamination limits the potential receptors for exposure to organic chemicals. In particular, the lack of fish greatly shortens the aquatic food chain by eliminating higher trophic levels. Further, lack of food sources (aquatic plants, insects and

other invertebrates, small fish) make upper Silver Bow Creek unattractive for larger animals such as migratory water fowl or raptors. It is unlikely that such animals would spend any significant time in stretches of the creek near the Montana Pole site. Any impact of organic contamination from the Montana Pole site should be considered only potential, especially when such impacts are due to hypothetical biomagnification of chemicals near the top of the food web.

Maximum concentrations of PCP detected in surface water exceed both the acute and chronic ambient water quality criteria (AWQC). Water concentrations of PCP as high as 591 μg/kg could limit the recovery of aquatic life in the impacted stretch of the creek.

Maximum PCP concentrations were found in the area of a major seep and are considered to represent "worst case" conditions at the site since rapid dilution of PCP is expected in the Creek below this seep; the reach of the Creek subjected to PCP concentrations above the chronic AWQC (5.6 μg/L) may be quite limited.

PAHs, including lower molecular weight compounds such as anthracene, pyrene and naphthalene, are present only in low concentrations even at the area of the seep. The highest concentration reported was 12.7 μg/L for acenaphthene. Acute and chronic toxicity values for acenaphthene and many other PAHs are not available, however, the concentration of PAHs in surface water at the Montana Pole site and downstream of the site are below observed chronic toxicity values for aquatic organisms. Although individual PAHs are not specifically addressed in this assessment, the generally low concentrations found in surface water and sediments suggest that a more refined assessment would reach similar conclusions. For this reason, PAHs are discussed only as a group, even though individual members of the group vary considerably in their toxicity to aquatic life.

Risks to Terrestrial Life

Because organic COC concentrations appear to diminish rapidly with distance downstream from the Montana Pole site, potential future impacts from Montana Pole site-related chemicals are likely to be limited to a short reach of stream starting at the region of discharge of contaminated groundwater. Wildlife and/or domestic animals using the downstream portions of the creek as a drinking water

source are not expected to be exposed to significant concentrations of organic COCs, unless discharge of contaminated groundwater significantly increases.

Significant exposure of major wildlife species to surface water, sediments, and soils in the impacted reach of the creek are also unlikely. The Montana Pole site is heavily disturbed by past human activity, and is surrounded by residential housing, industrial development and an Interstate freeway. The site is unlikely to be attractive to wildlife, and larger animals (predators, deer, elk) are not expected to use the site, or the adjacent reach of the creek.

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1 Section One

1.0 INTRODUCTION

The Montana Pole National Priority List (NPL) site was identified by EPA as a Superfund site in 1986. It is one of several NPL sites within the Clark Fork drainage (Figure 1-1). The site obtained NPL status as a result of chemical contamination associated with a timber treatment plant that operated nearly continuously from 1946 through 1984. The site is approximately 45 acres located in the SE 1/4, Section 24, T3N, R8W; specifically, at 202 W. Greenwood Avenue in Butte, Montana (see Figures 1-2 and 1-3). With the exception of coal tar creosote used to a limited extent in 1969, the primary solution used to treat timber products consisted of 5 percent pentachlorophenol (PCP) combined with 95 percent petroleum (various fuel oils). Past use of PCP, creosote and petroleum-related products, including dioxins and polycyclic aromatic hydrocarbons (PAHs), have contaminated surface soils, subsurface soils, sediments, groundwater, and surface water at the Montana Pole site. Inorganic wood treating chemicals have apparently never been used at the site.

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended by the Superfund Amendment and Reauthorization Act (SARA), stipulates that remedies at Superfund sites must be protective of both human health and environmental receptors. To evaluate the degree to which a remedial alternative is protective, it is necessary to assess both existing environmental and human health risks (no action alternative or baseline) and potential risks for proposed remediation alternatives. To determine final clean-up criteria, these risks are evaluated by regulatory authorities in conjunction with applicable or relevant and appropriate requirements (ARARs), technological limitations, and other site-specific factors.

At sites where litigation is anticipated, such as the Montana Pole site, both environmental and public health risk assessments are typically included in a single, stand-alone litigation document entitled the Baseline Risk Assessment (BRA). The baseline risk assessment defines the baseline risks posed by the site in the absence of any remediation. These baseline risks are subsequently used as one criteria to evaluate proposed remedial alternatives. Detailed guidance for performing risk assessments, provided in the Risk Assessment Guidance for Superfund (RAGS) (EPA 1989), was followed in preparing this Baseline Risk Assessment (BRA).

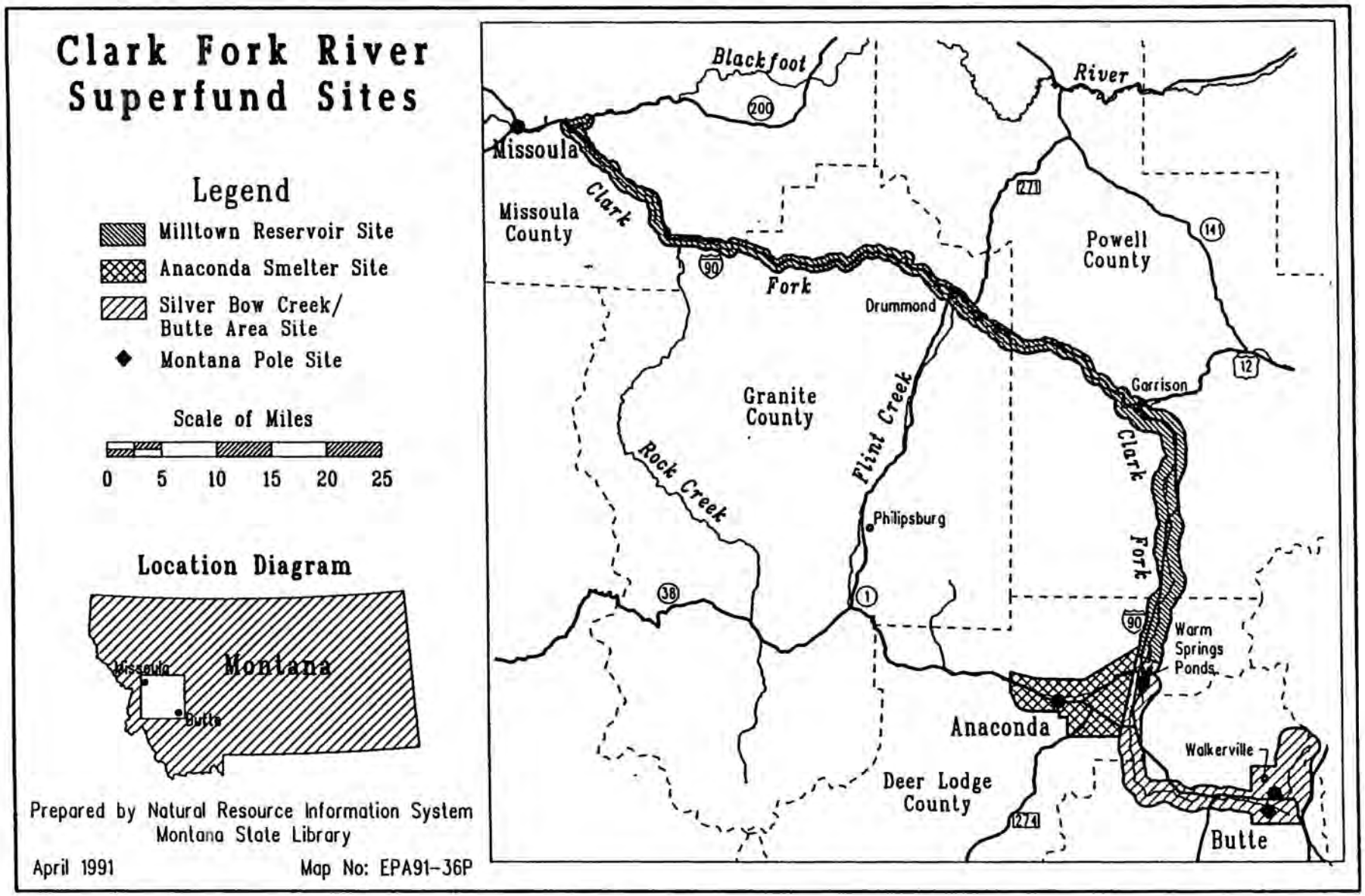
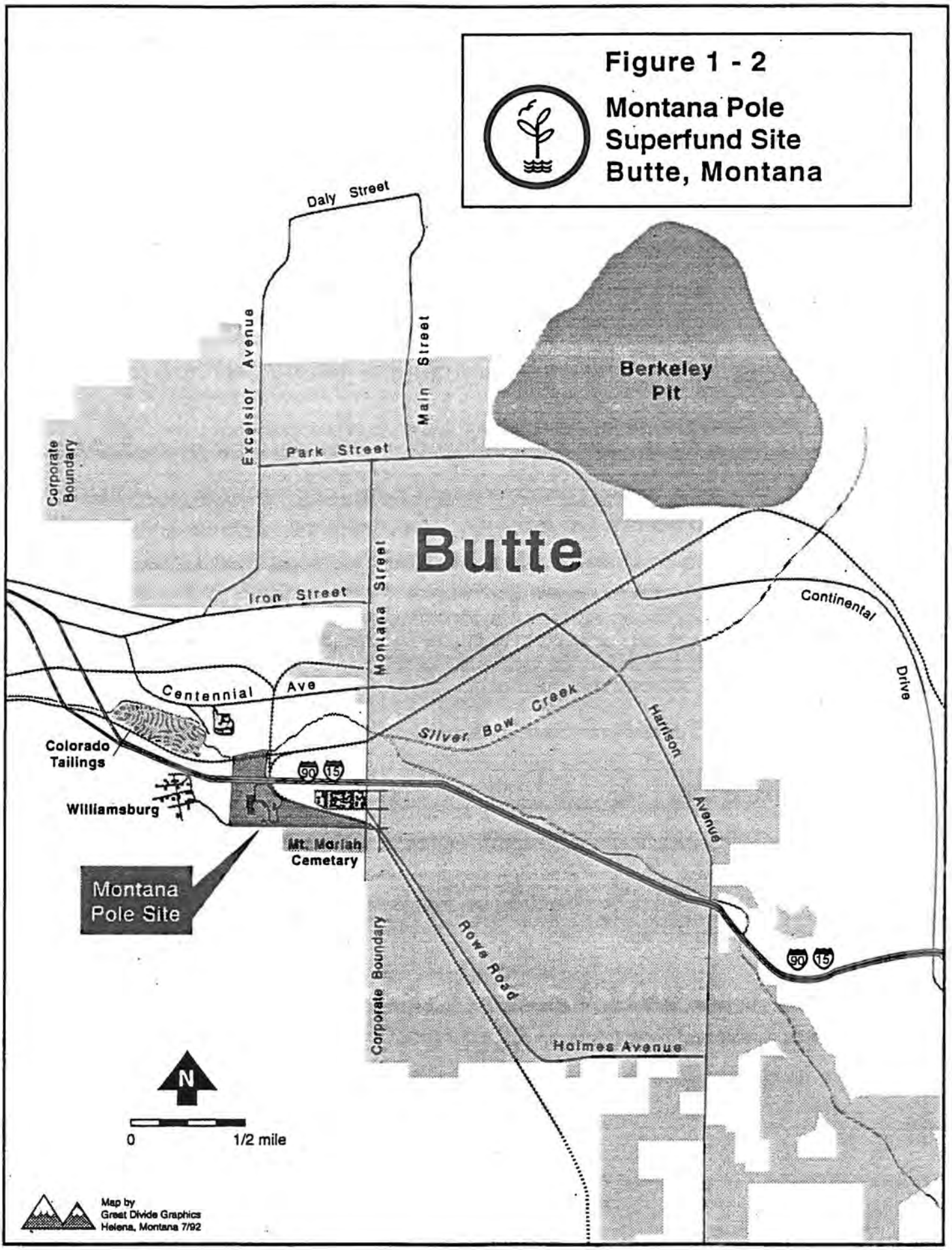
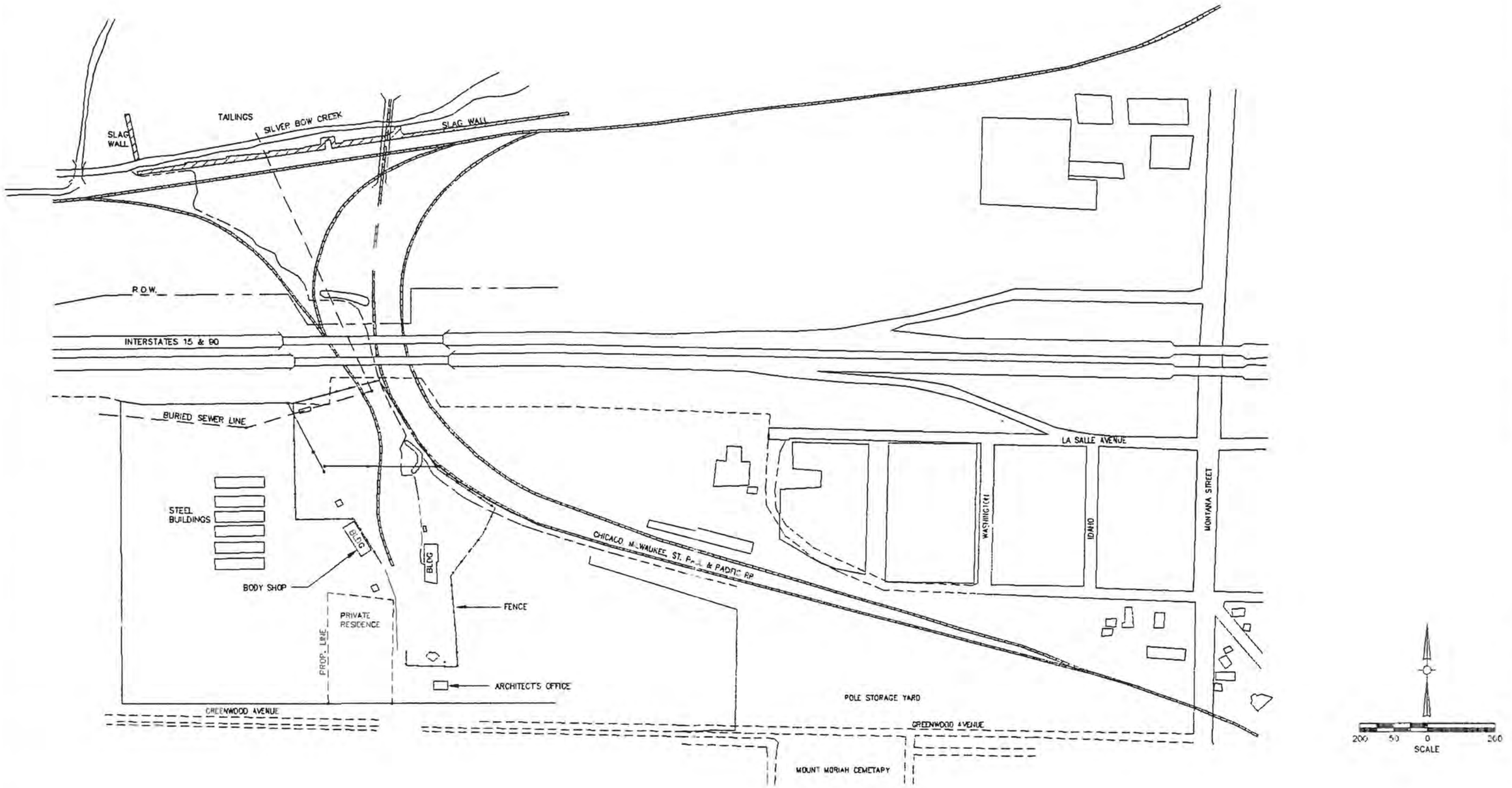

Clark Fork River Superfund Sites
Legend
Milltown Reservoir Site
Anaconda Smelter Site
Silver Bow Creek/ Butte Area Site
Montana Pole Site
Scale of Miles
0 5 10 15 20 25
Location Diagram
Montana
Missoula
Butte
Prepared by Natural Resource Information System
Montana State Library
April 1991
Map No: EPA91-36P
Blackfoot
River
Missoula
Clark Fork
Missoula County
Powell County
Drummond
Granite County
Rock Creek
Flint Creek
Philipsburg
Garrison
Clark Fork
Warm Springs Ponds
Anaconda
Walkerville
Deer Lodge County
Butte
200
271
141
90
12
38
1
274

FIGURE 1-1

Figure 1 - 2

Montana Pole Superfund Site Butte, Montana





MONTANA POLE CERCLA SITE

SITE LOCATION MAP

CAMP DRESSER & McKEE INC.

CDM

FIGURE NO. 1-3

The goals of this BRA for the Montana Pole site are the following:

- Characterize contamination at the site by media using site data, primarily those collected for the ongoing Remedial Investigation/Feasibility Study (Keystone 1991).
- Identify the chemicals of concern.
- Identify potential exposure pathways for both current and future scenarios, by which human or environmental receptors may be exposed to site contaminants, should no remedial action occur.
- Assess exposure for the pathways deemed most significant.
- Assess the toxicity of the chemicals of concern.
- Characterize risk to human health and impacts to the environment.

Methods for accomplishing these goals are discussed in the document "Preliminary Endangerment Assessment Montana Pole NPL Site" (CDM 1990). Comments on this document from the Montana Department of Health and Environmental Services (MDHES) and Atlantic Richfield Company (ARCO) were considered in the preparation of this BRA.

1.1 SCOPE OF HUMAN HEALTH RISK ASSESSMENT

The overall approach to the human health risk assessment follows guidance provided in "Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A) (EPA 1989a). This document provides guidance on all aspects of human health risk assessment, including: evaluating available data and identifying chemicals selected for quantitative analysis, developing exposure scenarios that depict expected exposure conditions, assessing toxicity of chemicals under expected exposure conditions, and combining this information to estimate carcinogenic and noncarcinogenic health risks.

Site-specific exposure parameters and assumptions provided by MDHES, and Region VIII EPA, have also been incorporated into this assessment.

1.2 SCOPE OF ECOLOGICAL RISK ASSESSMENT

The ecological risk assessment generally follows guidance provided in "Risk Assessment Guidance for Superfund: Volume II — Environmental Evaluation Manual (Part B)" (EPA 1989b). This document provides guidance regarding the nature of an ecological risk assessment and the types of information that should be included. It does not, however, provide detailed guidance regarding the implementation of specific ecological risk assessments. Ecological risk assessments are, by their nature, very site-specific. Issues such as exposure point concentrations, potential direct and indirect ecological receptors, bioaccumulation and biomagnification within ecosystems, and chemical toxicity to specific ecological receptors must be considered on a site-specific basis. The degree to which an ecological risk assessment should be qualitative or quantitative also depends upon site-specific ecological factors as well as chemical-specific factors. The ecological risk assessment contains both qualitative and quantitative aspects.

Exposure point concentrations for the ecological risk assessment are based on data for soils, surface water and sediments collected as part of the remedial investigation. Field observations, consultation with local biologists and fisheries personnel, and toxicological literature surveys are also important sources of information for this ecological risk assessment.

Exposure point concentrations are compared to EPA Ambient Water Quality Criteria (AWQC) for aquatic life (EPA 1986b); Montana Water Quality Standards; and toxicological literature values where no-observed-adverse-effect-levels (NOAEL) and lowest-observed-adverse-effect-levels (LOAEL) are identified for selected species. These comparisons are used to determine chemical concentrations in various media that would be protective of the potential ecological receptors. Potential for biomagnification is evaluated qualitatively.

1.3 UNCERTAINTY ANALYSIS

There is uncertainty associated with each estimated exposure parameter or toxicity value. In order to perform a quantitative risk assessment, it is necessary to make numerous quantitative assumptions regarding the type and extent of exposure that an individual or organism may receive, and the amount of exposure required to elicit an adverse effect.

In this assessment, uncertainties are addressed qualitatively in each major section of the report. For many of the selected parameter values, review of associated uncertainties indicates that the selected value will tend to overestimate exposure or risk. Thus, the selected values are "conservative," or likely to be overprotective rather than underprotective of potential receptors.

1.4 ORGANIZATION OF THE RISK ASSESSMENT

The remainder of the risk assessment is organized as follows:

Section 2.0: Environmental Setting
Section 3.0: Site History
Section 4.0: Data Evaluation
Section 5.0: Human Health Exposure Assessment
Section 6.0: Human Health Toxicity Assessment
Section 7.0: Human Health Risk Characterization
Section 8.0: Ecological Assessment
Section 9.0: References

2 Section Two

2.0 ENVIRONMENTAL SETTING

The Montana Pole site is located in Butte, Montana and is wholly surrounded by the Priority Soils operable unit of the Silver Bow Creek NPL Site, a separate Superfund site incorporating large areas of Butte. In general, the environmental setting for the site is similar to that for the Butte area as a whole. Major exceptions include wetland areas along Silver Bow Creek and current vegetation, both of which are more specific to the site. In this section, climate (Section 2.1), geology (Section 2.2), hydrology (Section 2.3), vegetation (Section 2.4), wetlands (Section 2.5), and current land use and demography (Section 2.6) are discussed in relation to possible exposure pathways for current and future visitors, workers or residents on the site. Additional discussion of environmental setting as it pertains to ecological receptors is provided in Section 8.2.1.

2.1 CLIMATE

The climate within Butte and vicinity is characterized by short, cool, dry summers and cold winters. Total annual precipitation measured at the Butte airport averages 11.7 inches. Records dating back to 1905 indicate that annual precipitation varies between 6.4 and 20.6 inches. May and June are generally the wettest months, during which approximately 35 percent of the total annual precipitation occurs. During an average year, more than two-thirds of the precipitation falls between April and September. The net annual evaporation is estimated at 26 inches per year (NOAA 1939-1987).

The low annual precipitation and high annual evaporation suggest dry soil conditions for much of the year. In turn, this suggests that dust blowing from the site, especially from areas with little vegetation, may carry contaminants to human receptors in the area. This potential source of exposure is evaluated in Section 5.2.1.1.

Based on records from 1951 to 1984, average annual temperatures measured at the Butte airport range between 34.0 and 42.6°F, with a mean of 38.9°F. The lowest recorded temperature was -55°F during February 1933, and the highest was 100°F during July 1931. July and August are the warmest months with average temperatures above 60°F. January, with an average temperature of 15.5°F, is the coldest month. The normal frost-free period is approximately 60 days (NOAA 1939-1987).

Climate in the higher elevations surrounding the study area is alpine to subalpine, characterized by colder temperatures and heavier precipitation, often in the form of snow. Melting of the mountain snowpack in spring and early summer provides the majority of the surface water supply within the study area (MultiTech 1987). Snow cover in the lower valleys usually melts during March to early April, with the mountain snowpack remaining through May into June.

The cool climate, short frost-free period and long winters all suggest that (1) exposure to soils outdoors may be limited by frozen ground conditions and snow cover, and (2) gardening may be limited both in types of crops and in extent by a short growing seasons. These suggestions are reflected in Section 5.2 in choices for fraction of contaminated material ingested and amount of garden vegetables consumed.

Wind speed and direction data for Butte are available from five locations, including information dating back to 1956. The terrain around Butte exerts a controlling effect on the wind patterns and causes marked differences over very short distances. Butte has winds characteristic of mountain valleys, with a primary flow along the valley axis and with a secondary air flow up the valley walls in the daytime and down at night. A windrose pattern for the Butte-Walkerville area is shown in Figure 2-1. The potential importance of winds for resuspending contaminants in air near the Site is addressed in Section 5.2.1.1.

2.2 GEOLOGY

The Butte area adjacent to the Montana Pole site is underlain by granitic rocks of the Boulder Batholith. These rocks are primarily quartz monzonite intersected by porphyritic dikes and plugs. The granitic rocks are fractured and faulted with resulting mineralization and alteration. A weathered zone is generally present in the upper 100 to 200 feet of the bedrock which, in mineralized zones, is underlain by a deep sulfide zone containing disseminated and vein deposits of copper and other metals.

Unconsolidated/alluvial sediments of fluvial or alluvial fan origin and Tertiary to Quaternary age are present in the valleys and drainages throughout the area, specifically along Silver Bow Creek and the drainages along the East Ridge (MultiTech 1987). Published reports (CH2M Hill and Chen-Northern

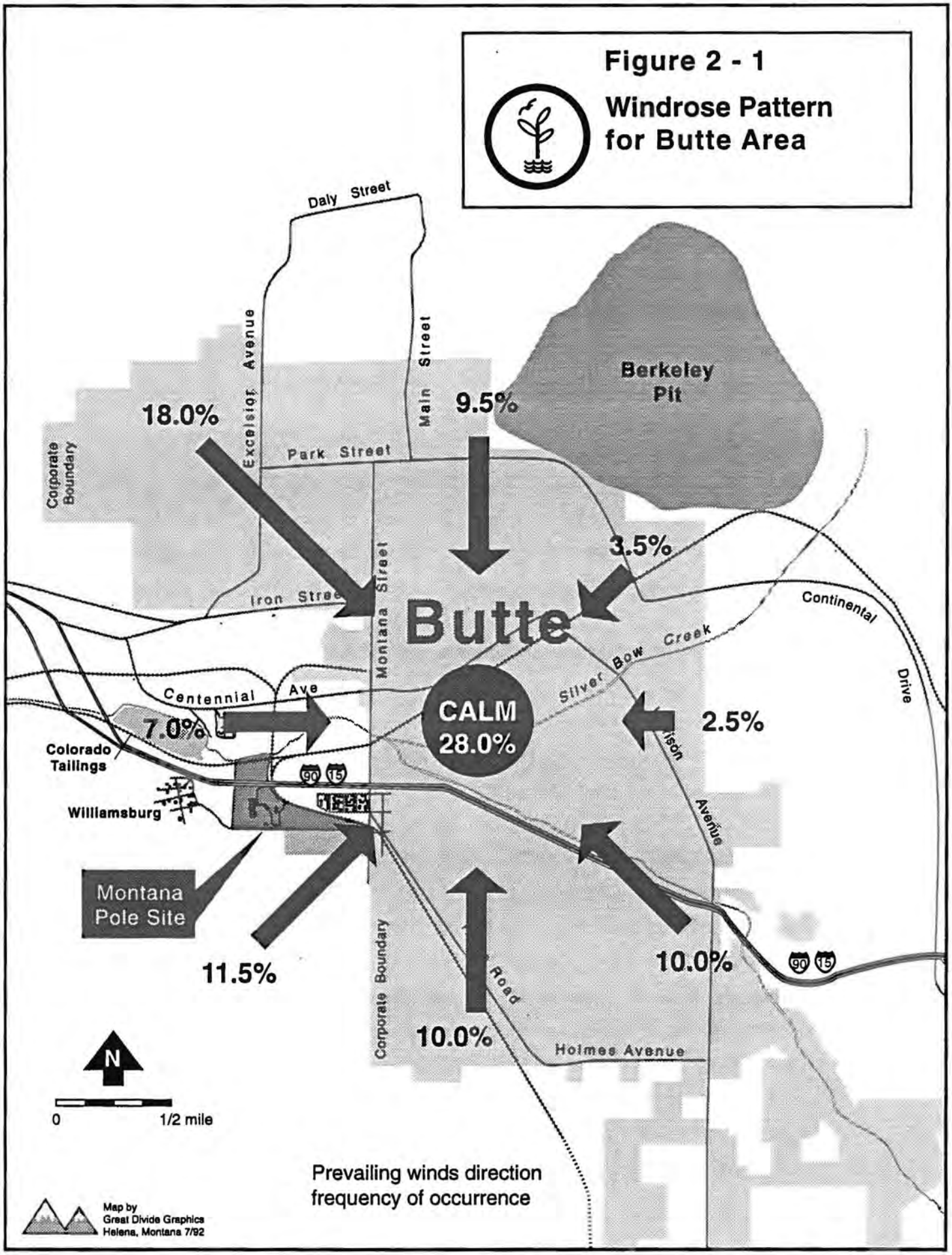


Figure 2 - 1
Windrose Pattern for Butte Area
Daly Street
Excelsior Avenue
Main Street
Park Street
Corporate Boundary
Berkeley Pit
18.0%
9.5%
3.5%
Iron Street
Montana Street
Butte
Continental Drive
Silver Bow Creek
Centennial Ave
CALM
28.0%
7.0%
2.5%
Colorado Tailings
Williamsburg
Montana Pole Site
Harrison Avenue
11.5%
10.0%
10.0%
Corporate Boundary
Holmes Avenue
N
0
1/2 mile
Prevailing winds direction
frequency of occurrence
Map by
Great Divide Graphics
Helena, Montana 7/92

1990) suggest that valley fill and alluvial deposits range in thickness from over 300 feet near the Butte Civic Center area on the east, to less than 40 feet within the area of the Colorado Tailings. The Colorado Tailings, located immediately northwest and hydrologically downgradient of the Montana Pole site, is within the area of the valley constriction.

The unconsolidated deposits consist of discontinuous layers, and lenses of sandy clay, clayey silty sand, and scattered sand and gravel (Botz 1969). The thickness of individual sand and gravel layers is reported to range from 2 to 20 feet. Based on 1985 drill logs, and data generated by Keystone, Inc. (April, 1991), alluvial thickness at the Montana Pole site ranges from 11 to over 47 feet. The water table is found at approximate depths of 5 to 10 feet below ground surface.

2.3 HYDROLOGY

The Montana Pole site lies within the upper Silver Bow Creek drainage basin. Silver Bow Creek originates in the mountains northeast of Butte. The creek is a major tributary of the upper Clark Fork River. Major tributaries to Silver Bow Creek above the Montana Pole site include Yankee Doodle Creek, originating northwest of Yankee Doodle tailings pond, and Blacktail Creek, originating south of Butte.

Silver Bow Creek flows west about 22 miles, terminating in the Warm Springs Ponds. Below Warm Springs Ponds the stream course is called the Upper Clark Fork River.

The Silver Bow Creek drainage basin, both above and below the Montana Pole site, has been subjected to contamination (primarily arsenic, copper and lead) by historic mining and mineral processing activities in and around Butte. Continued input of mining related contamination to the Silver Bow Creek drainage basin occurs from historic tailings impoundments, smelting and refining facilities and waste disposal areas. The continued input of contaminants results from direct contact of materials with ground and surface waters and from stormwater runoff events. For example, the preliminary baseline risk assessment for Lower Area One of the Silver Bow Creek/Butte Area NPL Site states that due to past mining and smelting activities, the water quality in Silver Bow Creek has deteriorated, and has existed in this condition for many decades. It further states that "...it is unlikely that any improvement in water quality can occur without direct action to mitigate the release of

contaminants into Silver Bow Creek" (FPC 1991). Other facilities near the Montana Pole site which could impact surface and groundwater quality in the basin include a Montana Power transformer maintenance facility just above the Montana Pole site, and the Butte Sewage Treatment Plant just below the Montana Pole site.

2.3.1 SURFACE WATER

Surface water runoff is characterized by high snowmelt flows in April through early June and low flows during the late summer months of July and August. Average annual flow between 1984 and 1986 at USGS Station 12323170 (Silver Bow Creek above Blacktail Creek), was 0.09 cubic feet per second (cfs) with a maximum flow of 1.7 cfs in April 1985 and a minimum flow of 0 cfs which occurred at least one day in all months of record. The drainage area for this gauge is 20 square miles. At USGS station 12323250 (Silver Bow Creek below Blacktail Creek) average annual flow over the 1984 to 1986 period of record was 24 cfs. A maximum flow of 100 cfs occurred in April 1985 and a minimum flow of 14 cfs occurred in August and September 1985. The drainage area for this gauge, including the Blacktail and Missoula Gulch areas, is 125 square miles (Hydrodata 1984-1986).

According to most recent data, Silver Bow Creek is a losing stream adjacent to the site. On June 27, 1990, the flow was 6.15 cfs and 4.8 cfs just upstream and just downstream of the site, respectively. On November 12, 1990, the flows were 12.6 cfs upstream and 9.07 downstream. Even so, data clearly indicate that contaminants in groundwater are discharging into the creek at SW-005, and perhaps other areas (Keystone 1991).

The Montana Pole site drains from the south to the north into Silver Bow Creek. Surface discharge occurs during storm events primarily through a drainage ditch which runs through the site, including through contaminated areas. Groundwater discharge occurs along the northern boundary of the site. Direct seepage of non aqueous phase liquids (NAPLs) and dissolved phase contaminants into Silver Bow Creek occurs from the site.

Changes is surface water flow may influence the concentrations of contaminants from the Montana Pole site in Silver Bow Creek. When combined with changes in groundwater discharge to the creek,

seasonal variations may add considerable uncertainty to exposure estimates for visitors to the Creek. This uncertainty is discussed further in Section 5.6.

2.3.2 GROUNDWATER

Groundwater in the Butte area occurs in two water-bearing units which are: 1) the unconsolidated sediments associated with the Tertiary and Quaternary age valley fill deposits (alluvial aquifer); and 2) the weathered and fractured bedrock deposits associated with the Boulder Batholith. The depth to water in the unconsolidated valley fill ranges from two greater than 30 feet (CH2M-Hill and Chen-Northern 1990). Well yields for the valley typically range from 3 gallons per minute (gpm) to over 30 gpm.

There has been little development of the water-yielding zones in the Butte area since treated surface water is available through the Butte Water Company. However, several households have recently installed irrigation wells which derive water from the alluvial aquifer (CH2M-Hill and Chen-Northern 1990).

The bedrock system in the Butte area is not as well understood as the alluvial system. Groundwater in the bedrock system occurs in fractures and in weathered zones near the top of the competent rock. Hydraulic characteristics of the bedrock system are variable because of major faulting which has occurred in the area and because of the large network of underground mines in the area. Impacts from the Montana Pole site appear to be limited to the alluvial aquifer system.

Groundwater velocity calculations for the contaminated alluvial aquifer system at the Montana Pole site were made by Keystone, Inc. (April 1991). The calculated average linear flow velocity beneath the site was 0.3 ft/day (108 ft/year). As mentioned in Section 2.3.1, direct seepage of non aqueous phase liquids (NAPLS) and dissolved phase contaminants into Silver Bow Creek occurs from the site.

The depth and porosity of the alluvial aquifer indicate that wells completed in this zone could yield sufficient water for domestic purposes. Thus, it is reasonable to assume future use of the groundwater for drinking water. [It should be noted, however, that Butte-Silver Bow County has enacted an ordinance (No. 431) prohibiting occupants of property connected to the public water

supply system from using water from wells for domestic purposes (ARCO 1992).] Further, the indication that Silver Bow Creek receives groundwater seepage in the vicinity of the Montana Pole site indicates that contaminants in the groundwater will continue to migrate into surface waters. Continued exposure to contaminants in groundwater is expected for visitors to the creek.

2.4 VEGETATION

Vegetation in the Butte area has been characterized by the Montana Department of State Lands (1981), and Hydrometrics (1983). The bluebunch wheatgrass (*Agropyron spicatum*)/bluegrass (*Poa spp.*)/rubber rabbitbrush (*Chrysothamnus nauseosus*) plan community is most predominant and best describes the pre-disturbed vegetation for the Montana Pole site. Other major plant species included in the community type are Idaho fescue (*Festuca idahoensis*), needle-and-thread (*Stipa comata*), prairie Junegrass (*Koeleria cristata*), western wheatgrass (*Agropyron smithii*), threadleaf sedge (*Cares filifolia*), and big sagebrush (*Artemisia tridentata*).

Plant communities associated with Silver Bow Creek have been extensively affected by past urban and industrial activity. The major impact to the plant communities near the Montana Pole site has been from industrial facility construction. Inspection of the floodplain boundary of the site indicates that another major impact to plant communities has been caused by deposition of metal-enriched waste materials (mill tailings) covering the original alluvial soils. In areas with extensive tailings deposition, vegetative cover is sparse with only intermittent areas supporting communities of inland salt grass (*Distichlis stricta*), scorpion plant (*Phacelia hastata*), and willows. Where the mill tailings have eroded away (exposing original alluvial soil), willows, tufted hairgrass (*Deschampsia caespitosa*), and bentgrass (*Agrostis spp.*) have recolonized the substrate (Hydrometrics 1983).

Additional disturbances to vegetation resulted from activities associated with the construction of the railroad and treatment plant facility buildings located on the site. A storage yard, previously used for stockpiling treated and untreated timbers, is an additional important associated disturbance. Traffic and mechanical activities in the facility and storage yard areas eliminated the original vegetation and hindered natural regrowth. Surface soils within the plant area were unvegetated during most of the site's operations, exposing the soils to wind and water erosion.

Much of the vegetative cover in and around the Butte study area had disappeared by 1890 as a result of air pollution from smelting and heap roasting. Other factors that have contributed to this loss include extensive logging and urban/industrial development. Existing vegetation represents an early successional stage that has developed since the demise of heap roasting and relocation of smelting to Anaconda (from 1890 to 1910) (Hydrometrics 1983).

2.5 WETLANDS

An intermediate-level wetland delineation was performed at the Montana Pole site in May 1990 (Keystone 1990). The guidance followed for the delineation was developed by a federal interagency committee composed of the U.S. Army Corps of Engineers, EPA, U.S. Fish and Wildlife Service, and USDA Soil Conservation Service (FICWD 1989).

Keystone used a vegetation unit method for the delineation that involves a visual separation of vegetation types into units followed by further definition of species and physical characteristics present at each unit. Nine vegetation units were identified and four were delineated as wetlands as shown in Figure 2-2. Table 2-1 lists the dominant vegetation from all the vegetation units and their indicator status according to the U.S. Fish and Wildlife Service "Wetland Plant List, Northwest Region." A brief description of each of the units delineated as a wetland is presented below.

A-4

This area is an isolated depression approximately 1 acre in size bordered by a transformer storage yard and a railroad track embankment. A-4 lies within the floodplain of Silver Bow Creek and is susceptible to periodic or seasonal inundation. Dominant vegetation includes great bulrush and spotted knapweed.

A-5

This unit is an approximately 1,800-foot segment of Silver Bow Creek with associated streambank vegetation occupying approximately 2.5 acres and composed entirely of herbaceous emergent vegetation. It provides wildlife habitat and streambank erosion protection. Dominant vegetation

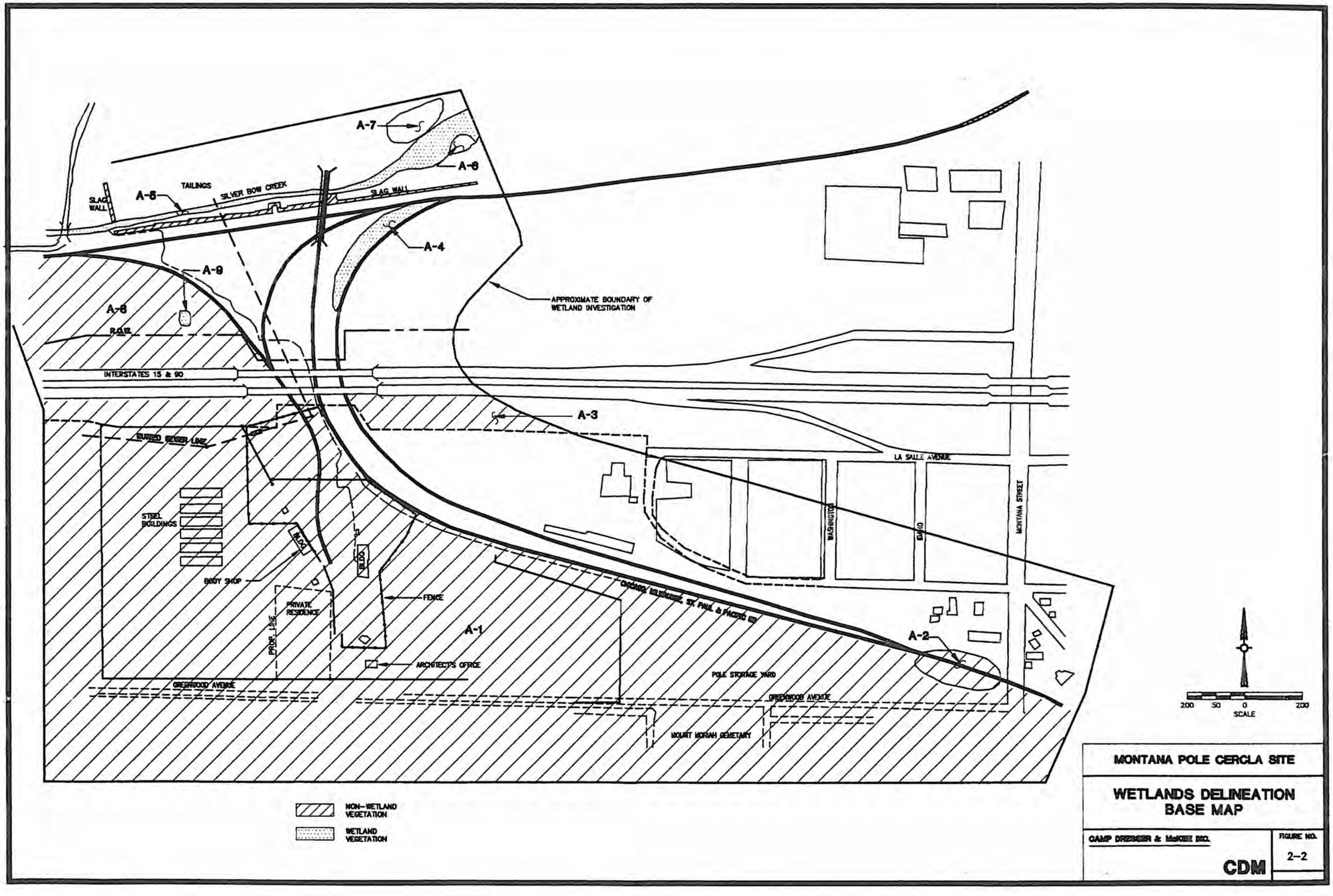

A-7
A-8
A-5
TAILINGS
SILVER BOW CREEK
SLAG WALL
SLAG WALL
A-4
A-9
A-6
ROW
APPROXIMATE BOUNDARY OF WETLAND INVESTIGATION
INTERSTATES 15 & 90
A-3
BURIED SEWER LINE
LA SALLE AVENUE
WASHINGTON
IDAHO
MONTANA STREET
STEEL BUILDINGS
BLDG
BLDG
BODY SHOP
FENCE
PRIVATE RESIDENCE
PROP. LINE
A-1
CHICAGO, MILWAUKEE, ST. PAUL & PACIFIC RR
A-2
ARCHITECT'S OFFICE
POLE STORAGE YARD
GREENWOOD AVENUE
GREENWOOD AVENUE
MOUNT MORIAH CEMETARY
200 50 0 200
SCALE
NON-WETLAND VEGETATION
WETLAND VEGETATION
MONTANA POLE CERCLA SITE
WETLANDS DELINEATION BASE MAP
CAMP DRESSER & McKEE INC.
CDM
FIGURE NO.
2-2

TABLE 2-1

LIST OF ALL DOMINANT VEGETATION[a]

Scientific Name	Common Name	Stations Found	Indicator Status[b]
Elymus cinereus	Great Basin Wild Rye	A-5, A-8	FAC
Equisetum arvense	Field Horsetail	A-6	FAC
Scirpus validus	Great Bulrush	A-6, A-4, A-9	OBL
Populus tremula	Quaking Aspen	A-3	FAC
Populus deltoides	Cottonwood	A-3, A-8	FAC
Salix sessifolia	River Willow	A-6, A-9	FACW
Erigeron compositus	Cutleaf Daisy	A-1	UPL
Melilotus officinalis	Sweet Clover	A-1	FACU
Ambrosia trifida	Great Ragweed	A-1	FAC
Acorus calamus	Sweet Flag	A-5	OBL
Elodea spp.	Common Waterweed	A-5	OBL
Avena fatua	Wild Oat	A-2, A-6	UPL
Conyza canadensis	Horseweed	A-3	FACU
Bromus secalinus	Cheat Grass	A-3	UPL
Bromus pompellianus	Brome Grass	A-2, A-3	UPL
Campanula spp.	Bellflower	A-1, A-3	FACU
Achiellea millefolium	Yarrow	A-2	FACU
Centerrea maculosa	Spotted Knapweed	A-1, A-2, A-4, A-8	UPL
Chyrsothamnus nauseosus	Rubber Rabbitbrush	A-1, A-2, A-3	UPL
Solidago graminifolia	Lance-leaved Goldenrod	A-1, A-2	UPL
Agrostis alba	Redtop	A-2	FACW

[a] Source: Keystone Environmental Resources (1990).

[b] Obligate (OBL); plants that occur almost always (probability > 99%) in wetlands under natural conditions.

Facultative Wetlands (FACW); plants that usually (probability 67-99%) occur in wetlands, but occasionally are found in nonwetlands.

Facultative (FAC); plants that are equally likely (probability 34-66%) to occur in wetlands or nonwetlands.

Facultative Upland (FACU); plants that usually (probability 67-99%) occur in nonwetlands, but occasionally are found in wetlands (probability 1-33%).

Upland (UPL); plants that occur almost always (probability > 99%) in nonwetlands under natural conditions.

includes great basin wild rye, sweet flag, and common water weed. Wildlife observed here includes muskrats, semi-palmated plovers, and mallard ducks.

A-6

This unit is an isolated stand of shrubs and herbaceous vegetation, approximately 0.1 acres in size, bordering Silver Bow Creek. It provides wildlife habitat and streambank erosion protection. Dominant vegetation includes field horsetail, great bulrush, river willow, and wild oat.

A-9

A-9 is an isolated depression approximately 0.05 acres in size which collects runoff from surrounding higher ground including the interstate embankment. Only the vegetation is indicative of wetlands. Dominant vegetation includes great bulrush and river willow.

2.6 LAND USE/DEMOGRAPHY

Much of the land in the vicinity of the Montana Pole site has been used industrially, usually associated with past and present mining activities, though commercial and residential areas are immediately adjacent to the Site. Colorado Smelter wastes and mill tailings are located to the west and north of the Montana Pole site. A federal manganese stockpile site and the former Butte Reduction Works are located directly north, while the Montana Power Company's transformer maintenance and storage facility is located to the north and east of the site. A partially reclaimed gravel pit and a blasting and explosive powder company (LaVelle Powder) are located to the south of the site. An overpass for U.S. Interstates 15 and 90 crosses the middle of the site, in an east-west direction. The site is surrounded on both the east and west sides by active railroad lines, some of which served the facility.

The population of Silver Bow County has been steadily decreasing since about 1960. From 1960 to 1980, the population decreased approximately 9.5 percent per decade, while from 1980 to 1990 the decrease was almost 11 percent. Preliminary 1990 census figures indicate that the population of Silver Bow County at that time was 34,000 (ARCO 1992). Even with the decrease in population,

however, there were 54 housing starts in the county in 1991-1992, and an average of 48 per year since 1986 (Walker 1992). Thus, population growth does not seem prerequisite for modest development of new residential structures. Residential areas are located within one quarter mile east and west of the site as shown on Figure 1-2. There is one on site resident whose house is located within the property line noted adjacent to Greenwood Avenue on Figure 1-3. There is also an auto body shop and an architect's office located on site. These facilities are also located near the on site residence along Greenwood Avenue noted on Figure 1-3.

The proximity of commercial and residential properties is indicative of mixed land use in the area. Future use of the area, including the Montana Pole site is also likely to be mixed. This suggests that both worker and residential exposure scenarios need be provided to the risk manager to assist in risk management decisions.

Because the site is currently mostly open space and assessable to visitors/trespassers, a recreational or trespasser scenario is also considered in this analysis. This is included to assess potential current needs for time-critical remediation, and risks associated with future open space land use.

3 Section Three

3.0 SITE HISTORY

This section is intended as an overview of historical activities, both operational and remedial, which have occurred since wood-treating operations began on the site in 1946-47. A brief discussion of operations, past emergency removal actions and current status of remedial investigations are provided here to summarize the nature of past releases of contaminants and to describe briefly past attempts to characterize contamination and to limit its spread.

Construction of the Montana Pole and Treatment Plant (MPTP) commenced in July 1946. Initial plant facilities included a pole peeling machine, two butt treating vats, and related ancillary facilities. Butt treating of poles involved immersing untreated poles in the butt treating vats in a heated mixture of treating oil and pentachlorophenol. In April 1947, the first load of treated timbers was shipped off-site.

Major modifications to the MPTP occurred in August 1949 and again in 1956. In 1949, a 73-foot long, 6-foot diameter retort was installed to increase timber treatment production efficiency. A second retort was installed in 1956, which was 66-feet long with a 7-foot diameter. The retorts were used both to dry green timber using the Boulton process, and to pressure treat timber with the petroleum/pentachlorophenol (PCP) mixture. Drying timber using the Boulton process generates steam, which is condensed and discharged. At the MPTP site, Boultonized water was reportedly discharged into an on-site drainage ditch that flows northward to Silver Bow Creek.

The butt treatment vats and retorts were in operation until May 1969. On the evening of May 5, 1969, an explosion occurred while a charge of poles was being treated in the east butt treating vat. The explosion immediately generated a very hot fire that destroyed the east vat, boiler room, and retort building. Although the boiler, retorts, and auxiliary equipment were badly damaged, reconstruction of the MPTP was performed and the plant was functional by December 1969. The west butt treatment vat was not destroyed by the fire. It was used for timber treatment and for mixing the petroleum/PCP product used in the retorts. While butt treating of poles continued throughout the life of the plant, sometime after the second retort was added in the mid-1950s, a majority of logs was treated in the retort. As a result of the explosion and fire, considerable spillage

of petroleum/PCP fluid occurred from the east treating vat. Additional seepage occurred from both retorts as a result of broken pipes and damaged valves.

It should be noted that fires at wood-treating sites using PCP may create lower molecular weight dioxin/furan isomers (e.g. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)), probably through dechlorination of higher molecular weight congeners such as octachlorodibenzo-p-dioxin (OCDD) present in technical grade PCP (Rappe et al. 1986). Because of the major fire at the Montana Pole site, the presence of TCDD was assumed likely, and was taken into consideration in the selection of chemicals of concern for the site (Section 4.5.1).

Construction of a small on-site sawmill commenced in the fall of 1978 and was fully operational in the fall of 1979. Additionally, in response to implementation of the Resource Conservation and Recovery Act (RCRA), a closed-loop process water system was constructed in 1980. The primary function of this system was to eliminate overland discharges of Boultonized water (generated from the drying of green timber). The closed-loop water recovery system operated by collection of Boultonized water in storage tanks, recirculation of this water through the condensing system, and evaporation of excess water using aeration sprays.

In 1983, the Montana Pole site was proposed to be added to the list of Superfund sites as a result of PCP/petroleum contamination present at the site. In May 1984 the MPTP officially went out of business, and most of its real property was conveyed by grant deed to the Miners Bank of Butte, the primary creditor. To recover capital, Miners Bank conducted an auction of on-site plant equipment and other salvageable resources on June 14, 1984.

In 1985, EPA began an emergency removal action which consisted of soil and groundwater actions. The two most significant emergency removal action activities that occurred during the 1985 and 1986 field seasons were:

1) Excavation, removal, and on-site storage of approximately 10,000 cubic yards of contaminated soils; and

2) Interception of the groundwater contamination plume, recovery of non aqueous phase liquids (NAPLS) from the groundwater and reinjection of the groundwater (minus the NAPLs) into

infiltration galleries located near and upgradient to the main process area of the site. This system is still in operation.

In July, 1987, the Montana Pole site was added to the NPL, primarily as a result of contamination (including NAPLs) seeping into Silver Bow Creek.

Between 1983 and 1989, investigations were conducted at the site by the EPA Technical Assistance Team, Environmental Response Team, United States Coast Guard Pacific Strike Team, MDHES, Montana Bureau of Mines and Geology, Emergency Response Cleanup Service Contractor (Riedel Environmental Services), and Montana College of Mineral Science and Technology. The results of these investigations are summarized by CDM (1989).

Through a cooperative agreement between MDHES and EPA, MDHES is administering RI/FS activities at the site which are being performed by the Atlantic Richfield Company (ARCO). ARCO completed RI field sampling tasks in 1991, and has produced a preliminary draft RI Report (Keystone 1991). The components of the RI include:

- identification of local geologic and hydrogeologic conditions around the site
- delineation of soil quality in and around the treating plant, historical runoff areas, and treated wood storage areas
- determination of groundwater hydraulic properties
- determination of groundwater quality
- determination of surface water and sediment quality within Silver Bow Creek
- characterization of the removed soils, dismantled equipment and various oils and sludges stored on the site
- determination of air quality upwind, downwind, and on the site.

The data set generated as part of the RI is the most complete and most reliable set of data generated to date at the site. This data set provides the basis for this Risk Assessment, although information from other sources is used and referenced throughout this document.

4

Section Four

4.0 DATA EVALUATION

This section briefly discusses and summarizes previous and current sampling efforts that have taken place at the Montana Pole NPL Site. The results of sampling performed during these investigations are summarized in this section to determine chemicals to be considered for evaluation in the risk assessment. The purpose of this screening step is to ensure that only those chemicals attributable to contamination in the study area (i.e., chemicals that are not associated with blank contamination, and are present above background concentrations), and that are likely to contribute to risk are carried through the risk assessment. Chemicals that remain after this screening are called chemicals of concern (COCs). Section 4.1 discusses previous sampling efforts at the Montana Pole site and Section 4.2 discusses more recent sampling. Section 4.3 discusses the use of enforcement and screening quality data. Section 4.4 presents information on background concentrations of some on site chemicals. Section 4.5 lists the methods used for selection of COCs and the resulting lists of COCs for human and ecological receptors. Uncertainties in the database are addressed in Section 4.6.

4.1 PREVIOUS SAMPLING EFFORTS

Previous sampling and analytical data have been compiled from data submitted by ARCO to MDHES and to CDM. CDM personnel have reviewed information to determine what records are available regarding sampling events, sample chain of custody, quality assurance/quality control (QA/QC), analytical results, and related criteria. A detailed summary of these data and their evaluation is provided in Volume I of the Work Plan for the Montana Pole site (CDM 1989).

Sampling activities at the Montana Pole site began in March 1983, and have continued to the present. Sampling was performed by the EPA Technical Assistance Team (TAT) and Environmental Response Team (ERT) personnel, as well as the U.S. Coast Guard Pacific Strike Team (PST), MDHES, Montana Bureau of Mines and Geology (MBMG) personnel, Emergency Response Cleanup Services Contractor (ERCS) Riedel Environmental Services, and Montana College of Mineral Science and Technology (MT Tech) personnel. Sampling has included the collection of solid, sludge, liquid and gaseous samples for analysis. Much of the previous sampling data is unvalidated and often samples were only analyzed for pentachlorophenol, dioxins and furans making these data of somewhat limited reliability, accuracy, and scope.

4.2 RECENT SAMPLING EFFORTS

The most recent sampling program, conducted by ARCO Coal Company under the direction of MDHES, includes priority pollutant analyses according to Contract Laboratory Program (CLP) procedures. These sampling results provide data that are used in this BRA because this sampling program provides a more current and accurate indication of the extent of contamination present at the site. Three sets of data were collected by ARCO during their ongoing RI. Round 1 data were collected in June 1990, round 2 in November 1990, and round 3 in May/June 1991. Sampled media include soil (surface and subsurface), groundwater, surface water, sediment, and air. Results of sampling rounds 1, 2, and 3 have undergone a Quality Assurance/Quality Control (QA/QC) review and have been qualified as either enforcement or screening quality.

Chemicals found in soil and groundwater include phenolics, polycyclic aromatic hydrocarbons (PAHs), dioxins/furans, and various metals. It should be noted that volatile organic compounds (e.g. benzene, ethylbenzene, toluene, xylenes) reported in investigations previous to the RI were generally found only in very low concentrations during the RI sampling. This may be due to loss of these compounds over time via volatilization.

In surface water, phenolic compounds, PAHs, and metals are found; in sediments two dioxin/furan isomers, two phenolic compounds, and several metals are detected. Data are reported at four sampling stations for two sampling rounds for surface water. The same stations were sampled only once for sediments.

4.3 USE OF ENFORCEMENT AND SCREENING QUALITY DATA

Because of the potential for litigation involving the Montana Pole site, an effort was made to base the RA on data of the highest quality. Where possible, exposure point concentrations were estimated from enforcement quality data. Moreover, data qualifiers are retained throughout the document so that the reader can easily associate conclusions and data quality.

The text includes specific mention where conclusions are based on screening quality data. The potential impact of screening quality data on the overall conclusions of the risk assessment is discussed in Section 7.6.12.

4.4 POTENTIAL BACKGROUND CONCENTRATIONS

Several factors, including background concentrations, must be considered when selecting COCs. Certain chemicals, while not naturally occurring, may be present due to widespread use and anthropogenic sources. For example, PAHs are ubiquitous at low levels in urban and industrial areas such as the Montana Pole site. They are emitted by combustion sources such as the burning of coal, oil, refuse, and diesel fuel. Other sources of PAHs include vehicle tires, leaching from coal storage piles, creosote-treated lumber, or asphalt surfaces. For this BRA, background can be adequately estimated from detection limits in samples where chemicals are not detected.

Inorganic chemicals are present naturally in soils, and limited information on concentrations is available for the Montana Pole site. As a result, the primary criteria for inclusion of inorganic chemicals are frequency of detection and a toxicity screen. Frequently detected chemicals with maximum concentrations exceeding typical health based standards or criteria are retained for evaluation in the risk assessment. Elevated metals concentrations are present at the site, particularly near Silver Bow Creek. Elevated metals concentrations are considered to be due to historical mining and ore processing activities in Butte, not from operations at the Montana Pole facilities. However, associated risks are evaluated.

4.5 CHEMICALS OF CONCERN

This section discusses the methods used to identify chemicals that are present on site as a result of Montana Pole operations and that are likely to contribute to risk based on toxicity. In addition, some chemicals not believed to be related to Montana Pole were also evaluated to provide consistency with risk assessment efforts for the Silver Bow Creek NPL site. Chemicals that are selected after this screening are quantitatively evaluated. Section 4.5.1 presents COCs for human health risk assessment and the method by which they were selected, and Section 4.5.2 evaluates available data for calculation

of exposure point concentrations. Contaminants of concern for ecological risks are addressed in Section 8.2.7, and summarized in Section 4.5.3.

4.5.1 COCs FOR HUMAN HEALTH

Chemicals detected on the Montana Pole site are screened as COCs based upon their toxicity to humans or laboratory animals (when human data were unavailable), their maximum concentrations measured in each media, and their frequency of detection. Tables 4-1 through 4-4 present the results of this screening procedure. Details for the screening are provided below. Numerical results are provided in Appendix A.

For each chemical considered, the Toxicity Value column presents the available cancer slope factor and corresponding carcinogenic group classification and/or the oral reference dose (RfD). These values are defined as follows by EPA (1989):

Cancer slope factor

A cancer slope factor is an estimate of cancer risks per unit daily dose derived from the application of low-dose extrapolation procedures to data from either human or animal exposures. Slope factors are derived for carcinogenic substances in three groups, based on a weight-of-evidence judgement of carcinogenic potential. These groups are:

Group A - Sufficient evidence for carcinogenesis in humans (human data available).
Group B - Insufficient evidence for carcinogenesis in humans, sufficient evidence in experimental animals. This group is further divided into Group B1 (suggestive or limited evidence in humans and Group B2 (contradictory or no evidence in humans).
Group C - Insufficient evidence for carcinogenesis in humans, limited evidence in experimental animals.
Group D - Insufficient data to classify.

Slope factors are presented in units of risk per mg/kg-day.

Reference Dose (RfD)

A reference dose (RfD) is an estimate, with an uncertainty spanning perhaps an order of magnitude or more, of a daily exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. RfDs are presented as a daily dose rate in mg/kg-day.

TABLE 4-1

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SOIL AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative[c] Toxicity	Decision
Inorganics			
Aluminum	No toxicity values available	Low	Omit - low toxicity
Arsenic	Group A carcinogen Slope factor 1.75 $(mg/kg\text{-}day)^{-1}$ Oral RfD 3×10^{-4} (mg/kg-day)	High	Retain - known human carcinogen. Low RfD
Barium	Oral RfD 7×10^{-2} (mg/kg-day)	Low	Omit - low toxicity, high RfD
Beryllium	Group B2 carcinogen Slope factor 8.4 $(mg/kg\text{-}day)^{-1}$ inhalation 4.3 $(mg/kg\text{-}day)^{-1}$ oral Oral RfD 5×10^{-3} (mg/kg-day)	High	Omit - maximum detected concentration is below risk based criteria
Cadmium	Oral RfD 5×10^{-4} (mg/kg-day)	High	Omit - maximum detected concentration is below risk based criteria
Calcium	No toxicity values available	Low	Omit - generally considered safe
Chromium (as Cr (VI))	Oral RfD = 5×10^{-3} (mg/kg-day)	High	Omit - maximum detected concentration is below risk based criteria
Cobalt	No toxicity values available	NA	Omit - maximum detected is below expected natural background
Copper	Oral RfD 4×10^{-2} (mg/kg-day)[d]	Low	Omit - low toxicity, high RfD
Iron	No toxicity values available	Low	Omit - generally considered safe
Lead	Toxicity evaluated using an integrated uptake biokinetic model (IUBK)	High	Omit - maximum detected concentration is near regional background

TABLE 4-1 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SOIL AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative[c] Toxicity	Decision
Magnesium	No toxicity values available	Low	Omit - low toxicity
Manganese	Oral RfD 1 x 10^{-1} (mg/kg-day)	Low	Omit - low toxicity, high RfD
Nickel	Oral RfD 2 x 10^{-2} (mg/kg-day) Group A carcinogen (based on inhaled refinery dust) Slope factor 8.4 x 10^{-1} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration is below risk based concentration
Potassium	No toxicity values available	NA	Omit - generally considered safe
Sodium	No toxicity values available	NA	Omit - generally considered safe
Vanadium	Oral RfD 7 x 10^{-3} (mg/kg-day)[d]	Low	Omit - low toxicity, high RfD
Zinc	Oral RfD 3 x 10^{-1} (mg/kg-day)	Low	Omit - low toxicity, high RfD
Organics			
Carbon disulfide	Oral RfD 1 x 10^{-1} (mg/kg-day)	Low	Omit - low toxicity, high RfD
2-chlorophenol	Oral RfD 5 x 10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based concentration
4-chloro-3-methylphenol	No toxicity values available	NA	Retain (will address qualitatively only)
2,4-dichlorophenol	Oral RfD 3 x 10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based concentration
2,4-dimethylphenol	Oral RfD 2 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based concentration

TABLE 4-1 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SOIL AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative[c] Toxicity	Decision
2,4-dinitrophenol	Oral RfD 2 x 10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based concentration
Dioxins/furans	Based on 2,3,7,8 TCDD. Group B2 carcinogens. Slope factor = 1.5 x 10^{5} $(mg/kg\text{-}day)^{-1}$[d] oral	High	Retain - high potential toxicity environmentally persistent
2-methyl-4,6-dinitrophenol	No toxicity values available	NA	Retain (will address qualitatively only)
2-nitrophenol	No toxicity values available	NA	Omit
4-nitrophenol	No toxicity values available	Low	Omit
Pentachlorophenol	Group B2 carcinogen Slope factor = 1.2 x 10^{-1} $(mg/kg\text{-}day)^{-1}$ (oral) Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Phenol	Oral RfD 6 x 10^{-1} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based concentration
2,3,5,6-tetrachlorophenol	Oral RfD 3 x 10^{-2} [e]	Low	Omit - maximum detected concentration is below risk based concentration Based on 2,3,4,6-tetrachloropehnol
2,4,6-trichlorophenol	B2 carcinogen Slope factor = 1.1 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria

TABLE 4-1 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN SOIL AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative[c] Toxicity	Decision
PAHs			
Acenaphthene	Oral RfD 6 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based concentration
Acenaphthylene	No toxicity values available	NA	Omit - maximum detected concentration is below risk based concentration (based on acenaphthene)
Anthracene	Oral RfD 3 x 10^{-1} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Benzo(a)anthracene[f]	Group B2 carcinogen Slope factor = 7.7 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Retain - maximum detected concentration exceeds risk based criteria
Benzo(a)pyrene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$ (oral)	High	Retain - high toxicity: maximum detected concentration exceeds risk based criteria
Benzo(b)fluoranthene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$ (oral)	High	Retain - high toxicity maximum detected concentration exceeds risk based criteria
Benzo(g,h,i)perylene	Group D carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Omit - maximum detected concentration is below risk based concentration
Benzo(k)fluoranthene	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Retain - maximum detected concentration exceeds risk based criteria
Chrysene	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Omit - maximum detected concentration is below risk based concentration

TABLE 4-1 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SOIL AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative[c] Toxicity	Decision
Dibenzo(a,h)anthracene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$ (oral)	High	Omit - maximum detected concentration is below risk based concentration
Fluoranthene	Oral RfD 4 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based concentration
Fluorene	Oral RfD 4 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based concentration
Indeno(1,2,3-cd)pyrene[e]	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria
Naphthalene	Oral RfD 4 x 10^{-2} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based concentration
2-methylnaphthalene	No toxicity values available	NA	Omit - maximum detected concentration is below risk based concentration (based on naphthalene)
Phenanthrene	No toxicity values available	NA	Omit - maximum detected concentration is below risk based concentration (based on naphthalene)

[a] Toxicity Reference Value — Slope factors are given in units of $(mg/kg\text{-}day)^{-1}$, RfDs in mg/kg/day.
[b] All toxicity values are from the EPA's Integrated Risk Information System (IRIS) unless otherwise noted (EPA 1992c).
[c] Relative Toxicity — High: RfD <0.001, slope factor >1; Moderate: RfD >0.001, <0.01; Low: RfD >0.01, slope factor <1
[d] Toxicity criteria from EPA Health Effects Assessment Summary Tables (HEAST FY 92) (EPA 1992d).
[e] Based on RfD for 2,4,5,6-tetrachlorophenol.
[f] For carcinogenic PAHs, slope factor for benzo(a)pyrene is multiplied by the appropriate toxicity equivalence factor (TEF) to estimate chemical specific slope factors. See Section 6.3.11 for discussion of TEFs.
NA = Not applicable

TABLE 4-2

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
Inorganics			
Antimony	Oral RfD 4 x 10^{-4} (mg/kg-day)	High	Omit - only one detect near screening criteria
Arsenic	Group A carcinogen Slope factor 1.75 $(mg/kg\text{-}day)^{-1}$ Oral RfD 3 x 10^{-4} (mg/kg-day)	High	Retain - known human carcinogen, high toxicity
Barium	Oral RfD 7 x 10^{-2} (mg/kg-day)	Low	Omit - low toxicity, high Rfd
Cadmium	Oral RfD 5 x 10^{-4} (mg/kg-day)	High	Omit - one high detect related to sediment in Silver Bow Creek
Calcium	No toxicity values available	NA	Omit - generally considered safe
Chromium (as Cr(VI))	Oral RfD = 5 x 10^{-3} (mg/kg-day)	High	Retain - high toxicity. Maximum detected concentration exceeds risk based criteria
Copper	Oral RfD 4 x 10^{-2} (mg/kg-day)[d]	Low	Retain - maximum detected concentration exceeds risk based criteria.
Iron	No toxicity values available	NA	Omit - not toxic
Lead	Toxicity evaluated using an integrated uptake biokinetic model (IUBK)	High	Retain - maximum detected concentration exceeds risk based criteria
Magnesium	No toxicity values available	NA	Omit - low toxicity
Manganese	Oral RfD 1 x 10^{-1} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria

TABLE 4-2 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
Nickel	Oral RfD 2 x 10^{-2} (mg/kg-day) Group A carcinogen (based on inhalation of refinery dust) Slope factor 8.4 x 10^{-1} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration is below risk based criteria
Potassium	No toxicity values available	NA	Omit - generally considered safe
Silver	Oral RfD 5 x 10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based criteria
Sodium	No toxicity values available	NA	Omit - generally considered safe
Vanadium	Oral RfD 7 x 10^{-3} (mg/kg-day)[d]	Low	Omit - maximum detected concentration below risk based criteria
Zinc	Oral RfD 2 x 10^{-1} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria
Organics			
2-chlorophenol	Oral RfD 5 x 10^{-3} (mg/kg-day)	Moderate	Retain - maximum detected concentration exceeds risk based criteria
4-chloro-3-methylphenol	No toxicity values available	NA	Retain - (will address qualitatively)
2,4-dichlorophenol	Oral RfD 3 x 10^{-3} (mg/kg-day)	Moderate	Retain - maximum detected concentration exceeds risk-based criteria
2,4-dimethylphenol	Oral RfD 2 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria

TABLE 4-2 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
2,4-dinitrophenol	Oral RfD 2 x 10^{-3} (mg/kg-day)	Moderate	Retain - maximum detected concentration exceeds risk based criteria
2,4-dinitrotoluene	(2,4/2,6)B2 carcinogen Oral slope factor 6.8 x 10^{-1} $(mg/kg\text{-}day)^{-1}$	Moderate	Retain - maximum detected concentration exceeds risk based criteria
Dioxins/furans	Based on 2,3,7,8 TCDD Group B2 carcinogens Slope factor = 1.5 x 10^{5} $(mg/kg\text{-}day)^{-1}$[d] inhalation or oral	High	Retain - high potential toxicity, environmentally persistent
2-methyl-4,6-dinitrophenol	No toxicity values available	NA	Retain (will address qualitatively only)
2-nitrophenol	No toxicity values available	NA	Omit - maximum detected concentration was less than risk based criteria (based on 4-nitrophenol)
4-nitrophenol	Oral RfD 6.2 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration was less than risk based criteria
Pentachlorophenol	Group B2 carcinogen Slope factor = 1.2 x 10^{-1} $(mg/kg\text{-}day)^{-1}$ (oral) Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Phenol	Oral RfD 6 x 10^{-1} (mg/kg-day)	Low	Omit - maximum detected concentration less than risk-based criteria
2,3,5,6-tetrachlorophenol	Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeding risk-based criteria

TABLE 4-2 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
Toluene	Oral RfD 2 x 10^{-1} (mg/kg-day)	Low	Omit - low toxicity maximum detected concentration less than risk-based criteria
2,4,6-trichlorophenol	B2 carcinogen Slope factor = 1.1 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria
Xylene	Oral RfD 2 x 10^{0} (mg/kg-day)	Low	Omit - low toxicity, maximum detected concentration less than risk-based criteria
PAHs			
Acenaphthene	Oral RfD 6 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Acenaphthylene	No toxicity values available	NA	Omit - maximum detected concentration less than risk-based criteria
Anthracene	Oral RfD 3 x 10^{-1} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk-based criteria
Benzo(a)anthracene[f]	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Retain - maximum detected concentration exceeds risk-based criteria
Benzo(a)pyrene	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk-based criteria
Benzo(b)fluoranthene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk-based criteria
Benzo(g,h,i)perylene	Group D carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk-based criteria

TABLE 4-2 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
Benzo(k)fluoranthene	Group B2 carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk-based criteria
2-chloronaphthalene	Oral RfD 8×10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration less than risk based criteria
Chrysene	Group C carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria
Dibenzo(a,h)anthracene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk based criteria
Fluoranthene	Oral RfD 4×10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Fluorene	Oral RfD 4×10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Indeno (1,2,3-cd)pyrene[c]	Group B2 carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria
2-methylnaphthalene	No toxicity values available	Moderate (based on naphthalene)	Retain - (will address qualitatively only)
Naphthalene	Oral RfD 4×10^{-2} (mg/kg-day)	Moderate	Retain - maximum detected concentration exceeds risk based criteria
Phenanthrene	No toxicity values available	NA	Retain - maximum detected concentration exceeds risk based criteria (based on naphthalene)

TABLE 4-2 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN GROUNDWATER AT THE MONTANA POLE SITE

Chemical	Toxicity[a,b] Values	Relative[c] Toxicity	Decision
Pyrene	Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria

[a] Toxicity Reference Value Slope factors are given in units of $(mg/kg\text{-}day)^{-1}$, RfDs in (mg/kg-day).

[b] All toxicity values are from the EPA's Integrated Risk Information System (IRIS) unless otherwise noted (EPA 1992c).

[c] Relative Toxicity
High: RfD < 0.001, slope factor > 1
Moderate: RfD > 0.001, < 0.01
Low: RfD > 0.01, slope factor < 1

[d] Toxicity criteria from EPA Health Effects Assessment Summary Tables (HEAST FY 92) (EPA 1992d).

[e] Based on RfD for 2,4,5,6-tetrachlorophenol.

[f] For carcinogenic PAHs, slope factor for benzo(a)pyrene is multiplied by the appropriate toxicity equivalence factor (TEF) to estimate chemical specific slope factors. See Section 6.3.11 for discussion of TEFs.

NA = Not applicable1

TABLE 4-3

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SURFACE WATER AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative Toxicity[c]	Decision
Inorganics			
Arsenic	Group A carcinogen Slope factor 1.75 $(mg/kg\text{-}day)^{-1}$ Oral RfD 3×10^{-4} (mg/kg-day)	High	Retain - known human carcinogen. High toxicity
Cadmium	Oral RfD 5×10^{-4} (mg/kg-day)	High	Omit - a single detected concentration was not greater than risk-based criteria
Copper	Oral RfD 4×10^{-2} (mg/kg-day)[d]	Low	Retain - maximum detected concentration exceeds risk based criteria
Lead	Toxicity evaluated using an integrated uptake biokinetic model (IUBK)	High	Retain - maximum detected concentration exceeds risk based criteria
Zinc	Oral RfD 2×10^{-1} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Organics			
Carbazole	Slope factor 2×10^{-2} $(mg/kg\text{-}day)^{-1}$[d]	Low	Omit - maximum detected concentration is below risk based criteria
2-chlorophenol	Oral RfD 5×10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based criteria
2,4-dichlorophenol	Oral RfD 3×10^{-3} (mg/kg-day)	Moderate	Omit - maximum detected concentration is below risk based criteria
2,4-dimethylphenol	Oral RfD x 1 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based criteria

TABLE 4-3 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN SURFACE WATER AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative Toxicity[c]	Decision
2-nitrophenol	No toxicity values available	NA	Omit - maximum detected concentration is below risk based criteria
4-nitrophenol	Oral RfD 6.2 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based criteria
Pentachlorophenol	Group B2 carcinogen slope factor = 1.2 x 10^{-1} $(mg/kg\text{-}day)^{-1}$ Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria
Phenol	Oral RfD 6 x 10^{-1} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria
PAHs			
Acenaphthene	Oral RfD 6 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based criteria
Acenaphthylene	No toxicity values available	Moderate (based on naphthalene)	Omit - maximum detected concentration is below risk based criteria
Anthracene	Oral RfD 3 x 10^{-1} (mg/kg-day)	Low	Omit - maximum detected concentration is below risk based criteria
Benzo(a)anthracene[f]	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$ (oral)	Low	Retain - maximum detected concentration exceeds risk based criteria
Benzo(a)pyrene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk based criteria

TABLE 4-3 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN
FOR HUMAN HEALTH IN SURFACE WATER AT THE MONTANA POLE SITE

Chemical	Toxicity Values[a,b]	Relative Toxicity[c]	Decision
Benzo(b)fluoranthene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk based criteria
Benzo(g,h,i)perylene	Group D carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration below risk based criteria
Benzo(k)fluoranthene	Group B2 carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration below risk based criteria
Chrysene	Group C carcinogen Slope factor = 7.3×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Retain - maximum detected concentration exceeds risk based criteria
Dibenzo(a,h)anthracene	Group B2 carcinogen Slope factor = 7.3 $(mg/kg\text{-}day)^{-1}$	High	Retain - maximum detected concentration exceeds risk based criteria
Fluoranthene	Oral RfD 4×10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria
Fluorene	Oral RfD 4×10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria
Indeno(1,2,3-cd)pyrene	Group B2 carcinogen Slope factor = 5.8×10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration below risk based criteria
Naphthalene	Oral RfD 4×10^{-3} (mg/kg-day) Under review	Moderate	Omit - maximum detected concentration below risk based criteria
Phenanthrene	No toxicity values available	NA	Omit - maximum detected concentration below risk based criteria (based on naphthalene)
Pyrene	Oral RfD 3×10^{-2} (mg/kg-day)	Low	Retain - maximum detected concentration exceeds risk based criteria

TABLE 4-3 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SURFACE WATER AT THE MONTANA POLE SITE

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[a] Toxicity Reference Value
Slope factors are given in units of $(mg/kg\text{-}day)^{-1}$, RfDs in (mg/kg-day).

[b] Relative Toxicity — High: RfD < 0.001, slope factor > 1
Moderate: RfD > 0.001, < 0.01
Low: RfD > 0.01, slope factor < 1

Note: Metals are not quantitatively evaluated in this analysis since exposure to metals in Silver Bow Creek was evaluated in the "Lower Area One" risk assessment. Results are summarized in Section 7.

[c] For carcinogenic PAHs, slope factor for benzo(a)pyrene is multiplied by the appropriate toxicity equivalence factor (TEF) to estimate chemical specific slope factors. See Section 6.3.11 for discussion of TEFs.

NA = not applicable

TABLE 4-4

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SEDIMENT AT THE MONTANA POLE SITE

Chemical	Toxicity Values	Relative Toxicity	Decision
Inorganics			
Arsenic	Oral RfD 3 x 10^{-4} (mg/kg-day)	High	Retain - maximum detected concentration exceeds risk based criteria
Cadmium	Oral RfD 5 x 10^{-4} (mg/kg-day)	High	Omit - maximum detected concentration below risk based criteria
Chromium (as Cr(VI))	Oral RfD = 5 x 10^{-3} (mg/kg-day)	High	Omit - maximum detected concentration below risk based criteria
Lead	Toxicity evaluated using an integrated uptake biokinetic model (IUBK)	High	Retain - maximum detected concentration may be of concern (based on IUBK model)
Organics			
Dioxins/furans	Based on 2,3,7,8 TCDD. Group B2 carcinogens Slope factor = 1.5 x 10^{5} $(mg/kg\text{-}day)^{-1}$[d] inhalation or oral	High	Retain - high toxicity, environmentally persistent
2-methyl-4,6,-dinitrophenol	No toxicity values available	NA	Omit - maximum detected concentration below risk based criteria based on 2,4-dinitrophenol
Indeno(1,2,3-cd)pyrene[f]	Group B2 carcinogen Slope factor = 7.3 x 10^{-2} $(mg/kg\text{-}day)^{-1}$	Low	Omit - maximum detected concentration below risk based criteria
Pentachlorophenol	Group B2 carcinogen Slope factor = 1.2 x 10^{-1} $(mg/kg\text{-}day)^{-1}$ (oral) Oral RfD 3 x 10^{-2} (mg/kg-day)	Low	Omit - maximum detected concentration below risk based criteria

TABLE 4-4 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR HUMAN HEALTH IN SEDIMENT AT THE MONTANA POLE SITE

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[a] Toxicity Reference Value Slope factors are given in units of $(mg/kg\text{-}day)^{-1}$, RfDs in (mg/kg-day).

[b] All toxicity values are from the EPA's Integrated Risk Information System (IRIS) unless otherwise noted (EPA 1992).

[c] Relative Toxicity
- High: RfD < 0.001, slope factor > 1
- Moderate: RfD > 0.001, < 0.01
- Low: RfD > 0.01, slope factor < 1

[d] Toxicity criteria from EPA Health Effects Assessment Summary Tables (HEAST FY 92) (EPA 1992).

[e] Based on RfD for 2,4,5,6-tetrachlorophenol.

[f] For carcinogenic PAHs, slope factor for benzo(a)pyrene is multiplied by the appropriate toxicity equivalence factor (TEF) to estimate chemical specific slope factors. See Section 6.3.11 for discussion of TEFs.

NA = Not applicable

Reference Concentration (RfC)

A reference concentration (RfC) is analogous to an RfD and is an estimate of an ambient air concentration, with an uncertainty spanning perhaps an order of magnitude or more, that is likely to be without an appreciable risk of deleterious effects for a lifetime of exposure. RfCs are presented as a concentration in air in units of $\mu g/m^3$.

The Relative Toxicity column notes whether the toxicity of a chemical is considered "high" or "low," relative to the other contaminants evaluated. Reference doses equal to or less than 0.001 mg/kg-day are rated as "high," indicating they are relative toxic (i.e., only a small dose is required to elicit adverse health effects); those that are greater than 0.001 but less than 0.01 mg/kg-day are rated as "moderate"; and those greater than 0.01 mg/kg-day are rated as "low". Similarly, cancer slope factors greater than 1.0 mg/kg-day^{-1} are rated as "high," and those less than 1.0 mg/kg-day^{-1} are rated as "low" for purposes of this assessment. These ranges are arbitrary and judged by CDM as adequate for this screening.

4.5.1.1 Toxicity Screening

For the toxicity screen, a spreadsheet developed by Smith (1992) was used to generate risk-based toxicity screening criteria. This database contains RfDs and cancer slope factors for a large number of chemicals, including all chemicals currently on the EPA Integrated Risk Information System (IRIS) and in the EPA Health Effects Assessment Summary Tables (HEAST). CDM maintains and updates this database to ensure that the most current toxicity reference values are used. As originally supplied, this spreadsheet contained several RfDs and slope factors not listed on IRIS or in the HEAS tables. Such values were not used in the toxicity screen, since the spreadsheet supplies no documentation for the values, and the CDM is not aware of such information from other sources.

Toxicity values in the spreadsheets are used to estimate media concentrations associated with an increased cancer risk of 1×10^{-6} or a hazard index of 1, based on standard residential exposure parameters (EPA 1989a). These media concentrations (the screening criteria) are then compared with maximum detected media concentrations found in the RI data. Where maximum detected values exceed the screening criteria, the chemical is retained as a chemical of concern, except as indicated below.

The same screening criteria applied to soil and groundwater are also applied to surface water and sediment. This makes the screen very conservative for these media, since it is unlikely that exposures to either surface water or sediment would occur over an extended time period on a daily basis. The screen for these media thus includes some chemicals which are unlikely to contribute significantly to risks.

4.5.1.2 Special Considerations

For groundwater, the maximum (and only) detected concentrations of both antimony and cadmium exceed the toxicity screening criteria. Antimony exceeded its screening level only slightly (22 vs. 14 μg/L), was detected only once and is not a known contaminant of concern for either mining activities in Butte, or operations at the Montana Pole site. For these reasons, antimony was not considered a chemical of concern.

Cadmium exceeded its screening criteria by a factor of about three (66 μg/L vs. 18 μg/L), which may be significant. However, it was detected in only 1 of 24 samples. Moreover, the single detect was from a shallow well immediately adjacent to Silver Bow Creek. Since the creek and the shallow groundwater are in close communication in this area (Keystone 1990), it is likely that this detect is influenced by the high concentrations of cadmium found in sediments (up to 21900 μg/kg). The low frequency of detection and the location of the single detect thus suggest that cadmium is not a chemical of concern for groundwater.

2,3,7,8-TCDD is considered a COC for this assessment. It was detected only once in surface soil up to a depth of 2 feet (0.0106 (S) μg/kg) and this detection was given an S qualifier. Presence of lower molecular weight chlorinated dioxins/furans at many wood-processing sites, and a fire at the Montana Pole site, suggest that some TCDD might be found. For these reasons, the single S value is assumed to reflect the presence of small amounts of TCDD on the Montana Pole site.

Finally, no consideration of source is incorporated into the selection of COCs. (Antimony is the lone exception, and other criteria also entered in the decision to eliminate this chemical from the list.) Inorganic chemicals, including many associated with historical mining activity, are not known to have been used at the Montana Pole site during wood-treating operations, and EPA (1989a) suggests that

lack of association with source can be a criteria for elimination of chemicals. However, exposure to inorganic contaminants in the scenarios addressed in this assessment could add significantly to baseline risks imposed by exposures to site-related chemicals. Significant risks might have an impact on any remedial measures that might be considered necessary for the site. For this reason, inorganic chemicals are retained, where appropriate, for quantitative assessment. The assessment is careful to note throughout that risks associated with exposure to inorganic chemicals are not believed to be related to the Montana Pole site.

Based on current sampling data (Keystone 1992), the above described process and these special considerations, the chemicals listed in Table 4-5 are considered COCs for human health for the Montana Pole site.

4.5.2 ADEQUACY OF DATABASE FOR CALCULATION OF EXPOSURE POINT CONCENTRATIONS

4.5.2.1 Detection Limits

Samples in which compounds were not detected at the detection limit are evaluated for this analysis according to EPA guidance (EPA 1989a). This guidance states that, when only some samples in a medium test positive for a chemical, samples that are not positively detected at the detection limit should be considered present at one-half of the detection limit. This should be done only if it is reasonable to believe that the chemical should be present. When the chemical is found in some samples in a given medium, it is conservatively assumed that the chemical is present also in the non-detect samples. An exception to this approach occurs when detection limits for a particular chemical are unusually high and exceed the maximum positive results for the same chemical from the same data set. In this case, EPA guidance states that samples with very high detection limits should be excluded from the analysis if they cause the calculated exposure concentration to exceed the maximum detected concentration for a particular sample set (EPA 1989a).

For most compounds, adequately low detection limits were realized. Failure to detect compounds, in general, suggests that significant concentrations from a risk standpoint are not present. For two of the major COCs, (PCP and dioxins/furans) detection limits present no significant problems in assessing nature and extent of contamination.

TABLE 4-5

COCs FOR HUMAN HEALTH AT THE MONTANA POLE SITE

GROUNDWATER

Arsenic
Chromium (VI)
Copper
Lead
Manganese
2-chlorophenol
4-chloro-3-methylphenol
2,4-dichlorophenol
2,4-dinitrophenol
2,4-dinitrotoluene
Dioxins/Furans
2-methyl-4,6-dinitrophenol
Acenaphthene
Anthracene
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(g,h,i)perylene
Benzo(k)fluoranthene
Chrysene
Dibenzo(a,h)anthracene
Fluoranthene
Fluorene
Indeno(1,2,3-cd)pyrene
2-methyl naphthalene
Naphthalene
Phenanthrene
Pyrene
Pentachlorophenol
2,3,5,6-tetrachlorophenol
2,4,6-trichlorophenol

SOIL

Arsenic
4-chloro-3-methylphenol
Dioxins/Furans
2-methyl-4,6-dinitophenol
Anthracene
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Indeno(1,2,3-cd)pyrene
Pentachlorophenol
2,4,6-trichlorophenol

SURFACE WATER

Arsenic
Copper
Lead
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Chrysene
Dibenzo(a,h)anthracene
Pyrene
Pentachlorophenol
Zinc

SEDIMENTS

Arsenic
Dioxins/Furans
Lead

Some significant problems in detection limits were found in the data for PAHs. In some cases, non-detects were reported with detection limits over an order of magnitude greater than reported detections in other samples. The dataset for PAHs also contains a great deal of screening-qualified data. These data problems reduce the usefulness of the data in estimation of exposure point concentrations. However, data appear sufficient for evaluation in the BRA, and for development of risk-based clean-up goals as necessary (see Section 4.5.2.2 below).

4.5.2.2 Data Representativeness, Soils

PCP

Data for PCP are considered representative of surface soil concentrations. Samples taken from the 0- to 0.5-foot interval are available for much of the site. At other locations, only samples from a depth interval of 0- to 2-feet are available. However, for these samples it appears that PCP has moved into the soil with its fuel oil solvent, and concentrations do not decrease rapidly with depth. Thus, the 0- to 2-foot interval is likely to adequately represent surface soil concentrations in these areas.

PAHs

Data for PAHs are more limited than those for PCP. Mostly, PAH samples are clustered in certain areas of the site (for example, near the steel buildings where samples MOPO-SS-A23 through A27 were taken). Large areas of the site were not sampled for PAHs since PCP data points were relied upon in large part for delineation of extent of soil contamination. Exposure point calculations for PAHs are therefore more uncertain than for PCP. Since the sampling locations for PAHs were taken from areas where PCP concentrations were generally high, overestimation may be more likely than underestimation.

Dioxin/Furan

Data is limited for dioxins and furans. Samples taken were mainly clustered and cannot be said to be representative of the site. An accurate exposure point concentration is difficult to calculate due to the small sample size and the limited sampling area.

Other Organic Chemicals

Data representativeness for the phenolic chemicals other than PCP is similar to that for PAHs. As described in later sections, however, phenolics other than PCP appear to contribute little to site risks.

Thus, data limitations for phenolics are not expected to significantly impact the results of this assessment.

Inorganic Chemicals

Data may be least representative for the inorganic COCs since they were not addressed in the same detail as organic COCs and since other data on inorganics in and around Silver Bow Creek are available from other sources.

Apparently, no inorganic wood treating chemicals were used in processing of timbers at the site, and no large on site sources of inorganics are anticipated. Moreover, any elevated levels of arsenic and cadmium are likely to have come from off-site. These off-site sources (e.g., mine and smelter wastes in the Lower Area One Operable Unit of the Silver Bow Creek NPL site) are the subjects of other risk assessments for the area. Results from the Lower Area One assessment are incorporated into this document where appropriate.

Sample Depth for Organic COCs

Many samples for organic COCs were taken from a depth interval of 0 to 2 feet. In many cases, this depth interval is too large for purposes of estimating exposures via ingestion of or dermal contact with surface soils. Many chemicals, such as dioxins/furans, PCP, PAHs, and many metals bind strongly to soil particles and do not migrate effectively beyond the first few inches of soil. Compositing over too large a depth interval can dilute surficial soil concentrations leading to an underestimation of exposure point concentrations.

At the Montana Pole site, however, chemical concentrations for organic COCs do not appear to decrease rapidly with depth in many areas. Table 4-6 provides sample data for PCP at various depth

TABLE 4-6

PCP CONCENTRATIONS VS. SAMPLING DEPTH

Sample Location	Depth Interval (ft.)	PCP (μg/kg)
SL-206-C	0-0.5 1-6	46100 E 46000 E
SL-209C	0-1 1-3 3-6	8480 E 86500 E 49900 E
SL-211-C	0-3 3-6 6-9 9-12 12-15	344000 E 356000 E 435000 E 317000 E 539000 E
SL-214-C	0-1.5 1.5-3	8910 E 38.1 E
SL-208C	0-3 3-7	20900 E 17900 E
SL-209c	0-1 1-3 3-6	8480 E 86500 E 49900 E
SS-A8	0-2 6-8 12-14 41-43	295000 E 355000 E 312000 E 1850 E
SS-A2	0-2 4-6 28-30	131000 S 13500 S 36.8 US
SS-A3	0-2 6-8 33-36	38900 S 91600 S 73 S
SL-207C	0-3 3-6 6-9 9-12 12-15	3100 E 189000 E 135000 E 478000 E 207000 E

intervals from ten sampling locations. Significant decrease with depth is seen only for location SL-214-C. At other locations high PCP levels are found below 2 feet and often down to the maximum depth interval sampled. It appears, for these locations, that binding to soil particles does not greatly impede movement of PCP into the subsurface. For these samples, use of data taken from depth intervals up to 2 feet can be considered adequate for generating surficial soil exposure point concentrations.

The high mobility of PCP in soils at this site may be related to bulk flow of fuel oils used in the wood treating process. The amount of PCP and fuel oil spilled in some locations appears to have overwhelmed the retention capacity of the soil. Fuel oil and associated PCP and other COCs are present in some areas of the site down to groundwater.

In other areas of the site, for example the south end of the site which was used for treated timber storage, did not receive large amounts of spillage, PCP has not penetrated as deeply into the soil. Fortunately, for much of the site, data from the depth interval of 0 to 0.5 feet are available from the round 1 sampling grid. Even though PCP migration into soil may be limited over much of the site, these shallower samples provide a representative picture of surficial soil contamination.

The above considerations may hold true, in part, for other organic COCs. Data from opportunistic samples available for trichlorophenol (TCP), methyldinitrophenol (MDNP), and PAHs are similar to those available for PCP. Data taken from areas where large amounts of fuel oil were released suggest that little decrease of chemical concentrations occurs with depth.

Sample Depth for Inorganic COCs

Data for arsenic and cadmium concentrations are from composites over a depth interval of 0 to 2 feet. Neither of these compounds is expected to migrate rapidly through soil, and neither should dissolve significantly in fuel oil. For these COCs, available data could lead to underestimation of concentrations at the surface and exposure point concentrations.

This underestimation, however, may be mitigated to some extent by two factors. First, the Montana Pole site lies on alluvium in the historical flood plain for Silver Bow Creek. Because the creek drains

a region of high natural mineralization and since the site has a history of mining activity, the alluvium is likely to contain relatively high background levels of arsenic.

Second, it is unlikely that the Montana Pole site is a significant source of arsenic or cadmium contamination. Neither chemical is known to have been used in the wood treating process on site. Instead, metal contamination is likely to come from off-site sources, particularly from dust blowing from tailings piles and/or mine wastes located in the vicinity and upstream along Silver Bow Creek or from historical smelter emissions. This suggests that no significant sources of inorganic contaminants are likely on site, and that concentrations in general should be less on the Montana Pole site than in areas more directly affected by mining activities. An exception may be the area adjacent to the creek which may have in the past received significant contamination from upstream sources.

For both arsenic and cadmium, exposure point concentrations are likely to underestimate actual surficial soil concentrations, but the absolute magnitude of this underestimate is likely to be small.

4.5.2.3 Data Representativeness, Groundwater

Groundwater has been a focus of site characterization efforts, and a large database has been generated. The extent and level of groundwater contamination, in general, have been adequately defined for purposes of calculating exposure point concentrations. Data for COCs are discussed below.

Organic Chemicals Excluding Dioxins/Furans

Relatively large numbers of data points are available for most organic COCs in groundwater beneath the site. Sampling rounds have included both summer and late fall sampling times so that seasonal variability is reflected, to some extent, in the data.

Most on-site wells are located in or near the groundwater plume, such that the area of impact for groundwater has been adequately sampled. In addition, wells have been screened over several depth intervals in the shallow aquifer, so that both areal and vertical extent of contamination is reflected in the data.

Dioxins/Furans

Data for dioxins/furans is limited to a subset of wells which were sampled during the RI.

Inorganic Chemicals

In general, fewer data points from on site wells are available for inorganic chemicals than for most organics. However, sample sizes are still relatively high (about 30) and a variety of well locations and screening depths are represented. The available data is considered to provide a reasonable approximation of potential exposure point concentrations.

4.5.2.4 Data Representativeness, Surface Water

Inorganic Chemicals

Surface water data generated during the RI for the Montana Pole site are limited and are probably of little use in determination of exposure point concentrations for inorganic COCs. However, a risk assessment based on other, more extensive data, for these COCs in the creek as it passes the Montana Pole site has recently been completed (CDM-FPC 1991). Risks estimated in that report can be assumed to adequately define risks from exposure to metals in the reach of Silver Bow Creek adjacent to the Montana Pole site.

Organic Chemicals

Two widely spaced sampling events (June 1990 and November 1990) suggest that, for PCP, the available data are sufficient to provide order-of-magnitude estimates for surface water exposure point concentrations. The two reported concentrations from the sampling station at the site of contaminant seepage into the creek are approximately 600 μg/L(S) and 200 μg/L(S) for June and November respectively, a threefold difference. It is not unreasonable to assume that data for other organic chemicals might show similar seasonal fluctuation.

Data also indicate that concentrations of PCP and some PAHs decrease rapidly with downstream distance from the site seepage. It seems likely that significant impacts to Silver Bow Creek are limited to a stream reach immediately downstream. This suggests that potential impacts to receptors visiting the creek will also be limited, unless particularly high use is made of the reach in question.

4.5.2.5 Data Representativeness, Sediments

Chemicals Other than Dioxins/Furans

Single data points from five sampling locations are available for sediments in Silver Bow Creek. These data provide some indication of the quantities of contaminants found in the creek. They are also useful in determining the possible extent of downstream contaminant migration. Both types of information are useful for the risk assessment. Calculation of exposure point concentrations from these data is, however, deemed inappropriate due to the small sample size. Instead, concentrations at SD-005 are used to estimate exposures. These may be interpreted as providing a "worst case" estimate for chronic daily intakes. Further discussion is provided in Section 5.4.

Dioxins/Furans

For dioxins/furans, data are limited to a single sample take from SD-005. These are not representative of the impacts to the creek. However, the data do provide an estimate of maximum impact. SD-005 is located at the site of a significant seep of non-aqueous phase liquid (NAPL) from the site into the creek. Based on data for PAHs and PCP, concentrations of dioxins/furans are expected to decrease rapidly downstream (see Figures 8-5 and 8-6 and accompanying text). Exposure point concentrations based on (OCDD) for sediment can be assumed to provide a "worst case" exposure estimate.

4.5.3 CHEMICALS OF CONCERN FOR ECOLOGICAL RISKS

A detailed description of the selection of COCs for ecological risk are provided in Section 8.2.7. Chemicals of concern for ecological risks selected for the Montana Pole site include:

Arsenic
Cadmium
Copper
PAHs
Pentachlorophenol
Lead
Dioxins/Furans
Zinc

A large number of chemicals for which little data on ecological risks exist, were eliminated on the basis of lack of information. Elimination of these chemicals is expected to have little effect on overall impacts to the Creek (Section 8.2.7).

4.6 UNCERTAINTIES ASSOCIATED WITH THE DATABASE

Major uncertainties in data representativeness are discussed in detail in Sections 4.5.2.2 through 4.5.2.5. For PCP, environmental concentrations and distributions appear to be well characterized, and exposure point concentrations can be assumed to be relatively accurate. For other COCs with fewer useable data points, confidence in exposure point concentrations calculated is reduced. The potential effects of uncertainties are further discussed in Section 7.6.2.

5 Section Five

5.0 EXPOSURE ASSESSMENT

This section addresses potential pathways by which human and environmental receptors could be exposed to contaminants at, or originating from, the Montana Pole site. In identifying potential pathways of exposure, both current and likely future land use of the site and surrounding study area are considered. This chapter identifies potential exposure pathways under both current and future scenarios (Section 5.1), provides quantitative exposure assumptions (Section 5.2), defines exposure units (Section 5.3), determines exposure point concentrations (Section 5.4), calculates chemical intake rates for selected exposure scenarios (5.5), and disucsses major uncertainties (Section 5.6).

This exposure assessment has been prepared in accordance with US EPA guidance (EPA 1989a). This guidance states that, for Superfund exposure assessments, intake variables should be selected so that the combination of all intake variables results in an estimate of the reasonable maximum exposure (RME) for that pathway. EPA defines the RME as the maximum exposure that is reasonably expected to occur at a site. With this approach, some intake variables may not be at their individual maximum values, but when in combination with other variables will result in estimates of the RME. For this analysis, upper confidence limits on chemical concentrations in each media are calculated. These concentrations are used as exposure point concentrations and are combined with upper range and average values for other exposure parameters. Based on the experience of CDM and others (e.g. HLA 1992), the approach taken appears to provide exposure estimates that meet EPA's RME definition. Providing that site contamination is well characterized, exposures are expected to fall in the 95th to 99th percentile range of those possible.

5.1 IDENTIFICATION OF EXPOSURE PATHWAYS

An exposure pathway (the sequence of events leading to contact with a chemical) is defined by the following four elements:

- A source and mechanism of chemical release to the environment
- An environmental transport medium for the released chemical

Northern Area

Intakes from ingestion of homegrown produce are slightly higher in this area relative to the southern area, except for PCP. There were fewer high detections of PCP in the northern area than in the southern area. PCP does, however, result in the second highest intake for the northern area, 1.5×10^{-3} mg/kg-day for cancer and the highest noncancer CDIs of 3.8×10^{-1}. Intakes from other chemicals for cancer CDIs ranged from 8.9×10^{-8} for dioxins/furans to 9.3×10^{-3} for 2,4,6-trichlorophenol.

Chronic daily intakes for produce grown in the northern area are provided in Tables 5-31 and 5-32. Calculations for estimation of chemical concentrations in plants are provided in Appendix C.

5.6 MAJOR UNCERTAINTIES ASSOCIATED WITH EXPOSURE ASSESSMENT

Quantitative evaluation of chemical exposures for a risk assessment may be the largest single source of uncertainty in the risk assessment. The procedures and assumptions used in this exposure assessment were derived from a combination of EPA guidance, site-specific information, and professional judgment, and are subject to various amounts of uncertainty depending upon the type of assumption or estimate considered.

Uncertainties from different sources may be compounded in an exposure assessment. For example, if a chronic daily intake (CDI) for a chemical measured in the environment is evaluated to determine whether there is a potential health hazard, the uncertainties in the concentration measurements and exposure assumptions will be expressed in the result. To ensure that human health is adequately protected, the exposure assessment incorporates conservative (likely to overestimate risk) estimates and approaches. The aim of the assessment is to estimate exposure well above the average, but still within the range of possible exposures. Therefore, actual exposures posed by a site are unlikely to be higher, but may be lower than those predicted in the assessment. Several of the key exposure assumptions which illustrate this approach are discussed below and are also presented in Table 5-33.

5.0 EXPOSURE ASSESSMENT

This section addresses potential pathways by which human and environmental receptors could be exposed to contaminants at, or originating from, the Montana Pole site. In identifying potential pathways of exposure, both current and likely future land use of the site and surrounding study area are considered. This chapter identifies potential exposure pathways under both current and future scenarios (Section 5.1), provides quantitative exposure assumptions (Section 5.2), defines exposure units (Section 5.3), determines exposure point concentrations (Section 5.4), calculates chemical intake rates for selected exposure scenarios (5.5), and disucsses major uncertainties (Section 5.6).

This exposure assessment has been prepared in accordance with US EPA guidance (EPA 1989a). This guidance states that, for Superfund exposure assessments, intake variables should be selected so that the combination of all intake variables results in an estimate of the reasonable maximum exposure (RME) for that pathway. EPA defines the RME as the maximum exposure that is reasonably expected to occur at a site. With this approach, some intake variables may not be at their individual maximum values, but when in combination with other variables will result in estimates of the RME. For this analysis, upper confidence limits on chemical concentrations in each media are calculated. These concentrations are used as exposure point concentrations and are combined with upper range and average values for other exposure parameters. Based on the experience of CDM and others (e.g. HLA 1992), the approach taken appears to provide exposure estimates that meet EPA's RME definition. Providing that site contamination is well characterized, exposures are expected to fall in the 95th to 99th percentile range of those possible.

5.1 IDENTIFICATION OF EXPOSURE PATHWAYS

An exposure pathway (the sequence of events leading to contact with a chemical) is defined by the following four elements:

- A source and mechanism of chemical release to the environment
- An environmental transport medium for the released chemical

- A point of potential exposure by the receptor with the medium (i.e., the "exposure point")
- A route of exposure (e.g., inhalation, ingestion)

An exposure pathway is considered "complete" only if all of these elements are present. The first two elements of an exposure pathway, a source and transport of chemical have been addressed previously. In this section, the last two elements are discussed. Human populations potentially exposed to site contaminants under current and possible future land use conditions are discussed, and the routes through which they may be exposed are identified.

5.1.1 CURRENT LAND USE CONDITIONS

The Montana Pole site includes several abandoned buildings and six pole barns in which contaminated soils are stored. Only small portions of the site are currently restricted from public access. A fenced area is located south of the interstate and east of the pole barns. This area cannot easily be accessed by trespassers or other unauthorized individuals. Silver Bow Creek is the northern boundary of the Montana Pole site and is used for recreational purposes. The site is mostly open space with loose sand, gravel and small brush occupying much of the land making it suitable for dirt bike-riding or other outdoor activities. It has been reported that people frequent the site to pick wildflowers (Appleman 1990).

The majority of the Montana Pole site is zoned M-2 (heavy industrial) with the remainder zoned M-1 (light industrial). These zoning designations do permit single family residences only in special circumstances, i.e. for a property/business owner, and future land use plans do not indicate that a change in zoning is currently being considered (ARCO 1992a). One residence is located at the south-eastern corner of the site, as described in Section 2.6, and is occupied by Mr. Torger Oass, previous president and owner/operator of the Montana Pole and Treatment Plant. Specific exposure pathways for Mr. Oass are not evaluated because (1) there are no sampling data for his property, (2) he does not use an on-site well for drinking water, and (3) potential risks are assumed to be accounted for by the pathways evaluated. There is also an autobody shop on-site with one to two workers and an architect's office with one employee, as described in Section 2.6. Their activities, however, are restricted to the buildings which they occupy and it is assumed that their exposures would not exceed those estimated in the future on-site worker scenario.

The Montana Pole site is located within the City of Butte, which currently has a population of approximately 34,000 residents. There are several residences located approximately one-quarter mile east and west of the site. Residences located east of the site are downwind. A transformer storage facility owned by the Montana Power Company is located directly north of the Chicago, Milwaukee, St. Paul & Pacific Railroad, but is not on-site. A cemetery is located southeast of the site across Greenwood Avenue.

Based on the above information, three human populations may be currently exposed to COCs in various media at the Montana Pole site:

- Trespassers that use the site for recreational purposes
- Residents that live downwind of the site and who may be exposed to contaminants present in dust and air
- On-site workers (non-remedial)

Table 5-1 summarizes current potential exposure pathways evaluated at the Montana Pole site. Each receptor population is discussed individually below.

Current On-Site Trespassers. Local children and residents are reported to use Silver Bow Creek for swimming and wading. While it is recognized that Silver Bow Creek is too shallow for typical swimming, the state uses a broader definition to include activities, such as inner-tubing, water fights, "dam" building, etc. where total body exposure and intimate contact with the water is likely though intermittent. It has also been reported that people trespass on the site to pick wildflowers. Therefore, exposure to COCs is evaluated for an individual using the creek and trespassing on the site. COCs were detected in surface water and sediments and persons contacting sediments and surface water (e.g., while wading in the creek) may dermally absorb or ingest these chemicals. Dermal absorption and incidental ingestion of COCs present in surface water and sediments are, therefore, also evaluated. Trespassers may also be exposed to COCs in the soil via dermal absorption and incidental ingestion of surface soil and inhalation of dust generated via wind erosion of surface soils. Inhalation of COCs present in air is also an exposure pathway for persons trespassing on-site. Exposure to COCs via ingestion of groundwater is not a likely exposure pathway as there are no wells currently used for drinking water purposes on-site. Individuals ages 6 through 18 are evaluated as they are a

TABLE 5-1

POTENTIAL PATHWAYS OF EXPOSURE TO CHEMICALS FROM THE MONTANA POLE NPL SITE UNDER CURRENT LAND USE CONDITIONS

Exposure Medium	Potential Routes of Exposure	Potential Receptors	Pathway Complete	Potential for Chemical Exposure
Soil	Dermal absorption, incidential ingestion	Trespassers	Yes	High. Potential for trespassers to contact surface soil high.
Surface Water and Sediments in Silver Bow Creek	Dermal absorption, incidential ingestion	Trespassers	Yes	High for trespassers. Children are reported to swim in Silver Bow Creek, contaminants are present in surface water and sediment.
Air	Inhalation of volatile organics and fugitive dust	Residents located down wind of the site	Yes	Moderate. Potential for fugitive dust generation and volatilization of organics from soil is moderate.
Groundwater	Ingestion, dermal absorption, and inhalation while showering	Trespassers	No	Low. Groundwater is not used for drinking purposes.

likely age group to trespass on the site, and their behavior and activities are likely to result in more exposure than would be the case for other age groups.

Current Downwind Residents. Residents located directly west of the site are downwind and are, therefore, susceptible to exposure to COCs generated from the site via inhalation. COCs may be present in air due to wind-blown surface soil and organics volatilized from the soils. However, site sampling data of ambient air indicates that there is little potential for exposure via this route. In order to estimate an upper bound of potential exposure (screening analysis), current residents located downwind are evaluated for exposure to COCs via this pathway conservatively assuming that air concentrations downwind are the same as those on-site.

Current On-Site Workers. Workers in the autobody shop and the architect's office may be exposed to contaminated soil via incidental ingestion and dermal contact. Their exposure, however, is expected to be minor compared to a future on-site worker. The on-site worker scenario is evaluated under future land use conditions.

Summary of Current Land Use Exposure Pathways

- Inhalation of contaminants in air by trespassers, residents located downwind (east) of the site, and on-site workers (as discussed in Section 5.2.1.1, risk for this pathway is not quantified but likelihood of significant exposure is evaluated by a screening analysis)
- Dermal absorption and incidental ingestion of surface water and sediments by trespassers and on-site workers
- Dermal absorption and incidental ingestion of surface soils by trespassers and on-site workers

5.1.2 FUTURE LAND USE CONDITIONS

In evaluating the no-action alternative, possible future land uses of the study area must also be considered. Potential future pathways that are additional to those evaluated under current land use conditions are discussed below and are summarized in Table 5-2. As stated previously, the study area currently has mixed residential and industrial land use. Future land use plans for the site area have been prepared by the Butte/Silver Bow County Planning Board in conjunction with the Butte/Silver

TABLE 5-2

POTENTIAL PATHWAYS OF EXPOSURE TO CHEMICALS FROM THE MONTANA POLE NPL SITE UNDER FUTURE LAND USE CONDITIONS

Exposure Medium	Potential Routes of Exposure	Potential Receptors	Potential for Chemical Exposure
Soil	Dermal absorption, incidental ingestion	Future on-site residents, workers	High. Children are especially likely to play on soils.
Surface Water and Sediments in Silver Bow Creek	Dermal absorption, incidental ingestion	Future on-site residents, workers	High. Children are especially likely to swim and wade in creek.
Air	Inhalation of volatile organics and fugitive dust	Future on-site residents, workers	High. Potential for fugitive dust generation and volatilization of organics from soil is high.
Groundwater	Ingestion	Future on-site residents, workers	High. Contaminants are present in groundwater.[a]
Produce	Ingestion	Future on-site residents, workers	Moderate. Uptake of contaminants in groundwater and soils by plants is likely to occur.[b]

[a] Assumes that drinking water wells may be installed in the future. Actual potential for on site residential development appears to be low.

[b] Assumes that gardening in the Butte area will be limited by climate.

Bow County Council of Commissioners (ARCO 1992a). The plan for preferred future land uses in the vicinity of the Lower Area One study area of the Colorado tailings operable unit recommends that the northern portion of the site remain primarily industrial and commercial. Existing land use of the southern portion of the site is highly mixed and the land use plan recommends that such use continue (ARCO 1992a). Even though residential land use may be less likely than industrial, there are currently no absolute restrictions that would prevent the future development of the site for residential purposes. Therefore, receptor populations considered for future land use are:

- Future on-site industrial workers
- Future on-site residents.

Future On-Site Industrial Workers. Persons working outdoors on or near areas of contaminated soils may be exposed to COCs in the soil via incidental ingestion, dermal absorption, inhalation of dust generated via wind erosion, and inhalation of volatile organics emitted from the surface and subsurface soil. Exposure via ingestion of groundwater may occur in the future and is evaluated for future workers at the site. Incidental ingestion of surface water is not considered for a worker at the site, since it seems unlikely that workers would spend significant amounts of time near Silver Bow Creek as part of their job. Exposure via sediments is considered even less likely for a worker and is not evaluated.

Future On-Site Resident. The Montana Pole site may be developed in the future for residential use. Exposure assumptions for an adult and a child are used to evaluate the future land use scenarios in this risk assessment.

Future residents at the Montana Pole site may be exposed to contaminants in the soils via dermal absorption, incidental ingestion, inhalation of volatile organic chemicals from the soils, and inhalation of fugitive dust generated from wind erosion of the soils. It is also likely that future on-site residents will use Silver Bow Creek for recreational purposes; however, incidental ingestion and dermal absorption from surface water and sediments are assumed to be adequately assessed by the recreational/trespasser scenario. Ingestion of groundwater is evaluated for future residents as installation of a well for private use is possible. Home-grown produce grown in contaminated soils also presents an exposure pathway for future residents and is evaluated.

Summary of Future Land Use Exposure Pathways

- Dermal absorption and incidental ingestion of surface soil by future workers and future on-site residents.
- Dermal absorption and incidental ingestion of surface water and sediments by future on-site residents.
- Inhalation of ambient air by future industrial workers and future on-site residents.
- Ingestion of home-grown produce by future on-site residents.
- Ingestion of groundwater by future on-site residents and industrial workers.

5.2 EXPOSURE ASSUMPTIONS

This section describes the exposure assumptions used for each exposure scenario. Exposure estimates are expressed in terms of the mass of substance in contact with the body per unit body weight per unit time (e.g., mg chemical per kg body weight per day, mg/kg-day). These exposure estimates are termed Chronic Daily Intakes (CDIs). To determine CDIs, the assumptions concerning measured or estimated chemical concentrations, exposed populations, and exposure conditions such as frequency and duration of exposure are used together with intake parameters.

5.2.1 CURRENT LAND USE SCENARIOS

5.2.1.1 Current Off-Site Resident - Inhalation of Ambient Air

The following discussion presents a screening (upper bound) analysis for the likelihood of significant exposure via this route and is not used in quantifying risks. Table 5-3 presents exposure assumptions for inhalation of ambient air by a hypothetical individual located downwind of the site. For this exposure pathway it is conservatively assumed that inhalation exposure could occur every day (365 days/yr). It is also assumed that residents will live in the area for 30 years (EPA 1989a). An average inhalation rate of 20 m^3/day for adult males and females is used (EPA 1989a). The equation used to estimate exposure via inhalation is as follows:

TABLE 5-3

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INHALATION OF AMBIENT AIR FOR RESIDENTS (FUTURE)

Parameter	Exposure Assumption
Frequency of Exposure — Residents (future)	365 d/yr[a]
Exposure Duration — Residents (future)	30 yr[b]
Inhalation Rate — Residents (future)	20 m^3/day[b]
Average Body Weight — Residents (future)	70 kg
Fraction Inhaled — Residents (future)	1.0[c]

[a] Based on exposure occurring every day of the year.
[b] EPA (1989a).
[c] Assumes all air inhaled is contaminated.

$$\text{Intake (mg/kg/day)} = \frac{\text{CA x IR x EF x ED}}{\text{BW x AT}}$$

where: CA = Chemical Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Using these exposure assumptions and the above equation, it is possible to calculate an air concentration associated with a given "acceptable" chronic intake. Such an intake can be estimated simply for pentachlorophenol by assuming a target cancer risk of 1×10^{-6}, and a slope factor of 0.12 $(mg/kg\text{-}day)^{-1}$. An acceptable chronic daily intake can be estimated as:

$$\textbf{Intake (mg/kg-day) = Target Risk/Slope Factor = } 8.3 \times 10^{-6} \textbf{ mg/kg-day}$$

If this value is substituted into the first equation, and the equation solved for CA, the result is an estimate of the air concentration (6×10^{-5} mg/m^3) for PCP associated with an increased cancer risk of 1×10^{-6}.

This can be converted to a concentration of particulates in air by assuming that 100 percent of all particulates come from the site. The exposure point concentration for PCP in surface soils is 320 mg/kg (Section 5.4.1.4). Approximately 2×10^{-7} kg, or 200 μg, of this soil would have to be present in each cubic meter of air to reach a concentration of 6×10^{-5} mg PCP/m^3. This value can be interpreted as the long-term annual average dust concentration necessary to produce an exposure associated with an increased cancer risk of 1×10^{-6} in a population exposed 24 hours/day, 365 days/year for 30 years. The highest ambient dust level measured on or near the Montana Pole site was 40.9 $\mu g/m^3$ (Keystone 1991), about 5 times less than the 200 $\mu g/m^3$ associated with a one-in-a-million increased cancer risk. Even "worst case" exposure assumptions as presented here thus require long-term annual ambient dust concentrations significantly in excess of those which might be associated with risks above the lower limit of the EPA risk range.

It seems highly unlikely that dust blowing from the Montana Pole site could have a substantial impact on any receptor group either now or in the future. For this reason, the air pathway is not further addressed quantitatively in this assessment.

5.2.1.2 Current On-Site Trespasser - Dermal Absorption and Incidental Ingestion of Soil

Individuals trespassing at the Montana Pole site may be directly exposed to chemicals by dermal absorption of contaminated surface soil. Further exposure could occur by inadvertent ingestion of soil through activities such as eating. Tables 5-4 and 5-5 present the assumptions used in assessing exposure by dermal absorption and incidental ingestion of contaminated surface soils by trespassers. It is expected that during winter months, frozen ground, snow cover, and/or heavy clothing will limit the period during which exposure through direct contact may occur. Based on climatological data for Butte for a period of 96 years (EarthInfo Inc. 1989), there are approximately seven months (April - October) during which exposure to surface soils is considered possible. November through March have average maximum temperatures of 5°C or less indicating that the ground is likely to be frozen and/or covered with snow. Based on these data there are approximately 210 days of possible exposure to soil. Individuals are assumed to trespass on-site approximately two times a week resulting in an exposure frequency of 60 days/year (210 x 2/7 = 60). Exposure duration is 12 years for a child ages 6 through 18.

Dermal absorption of soils assumes exposure to the hands, forearms and legs and a 50th percentile body part-specific surface area for males (EPA 1989a), resulting in a skin surface area of 5,165 cm^2. Fiftieth percentile values are recommended by RAGS (EPA 1989a). Due to lack of site-specific information on soils, an adherence factor for commercial potting soil of 1.45 mg/cm^2 is used (EPA 1989a). A dermal absorption factor of 0.01 is used for inorganics and 0.1 for organics except as noted. These values reflect the generally recognized poor dermal absorption of most compounds through intact, nonabraded, nonoccluded skin.

A default absorption factor of 0.1 for uptake of dioxins/furans may over estimate actual absorption across the skin. A lower value of 0.03 for dioxins/furans has been suggested for dermal absorption (Poiger and Schlatter 1986). Recently, EPA has used a value of 0.01 for absorption from soil (EPA 1990c). This value is adopted here.

TABLE 5-4

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA DERMAL CONTACT WITH SURFACE SOIL FOR WORKERS (FUTURE) AND TRESPASSERS (CURRENT)

Parameter	Exposure Assumption
Frequency of Exposure	
Workers (future)	150 d/yr[a]
Trespassers (current)	60 d/yr[b]
Period of Exposure	
Workers (future)	25 yr[c]
Trespassers (current)	12 yr[d]
Skin Surface Area	
Workers (future)	3,120 cm^2 [f]
Trespassers (current)	5,165 cm^2 [f]
Average Body Weight	
Workers (future)	70 kg[d]
Trespassers (current)	43 kg[g]
Averaging Time	
Noncarcinogens	365 d/yr x 25 yr[d] (worker) 365 d/yr x 12 yr (trespasser)
Carcinogens	365 d/yr x 70 yr[d]
Skin Adherence Factor	1.45 mg/cm^2 [d]
Fraction Contaminated	
Workers (future)	1.0
Trespassers (current)	0.5
Absorption Factor	
Organics	0.1
Inorganics & Dioxins/Furans	0.01

[a] Based on exposure occurring 5 days a week for 7 months of the year (5/7 x 210 = 150).

[b] Based on exposure occurring 2 times a week (2/7 x 210 = 60).

[c] EPA (1991b). Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Supplemental Guidance. "Standard Default Exposure Factors." Interim Final. OSWER Directive: 9285.6-03.

[d] EPA (1989a).

[e] Current exposure is for trespassers ages 6 through 18.

[f] EPA (1989a). 50th percentile body surface area for adult forearms and hands were used for workers; children; and forearms, hands, and legs were used for trespassers ages 6 through 18.

[g] EPA (1989i). Exposure Factors Handbook. Office of Health and Environmental Assessment. EPA/600-8-89/043.

TABLE 5-5

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INCIDENTAL INGESTION OF SURFACE SOIL FOR WORKERS (FUTURE) AND TRESPASSERS (CURRENT)

Parameter	Exposure Assumption
Frequency of Exposure	
Workers (future)	150 d/yr[a]
Trespassers (current)	60 d/yr[b]
Exposure Duration	
Workers (future)	25 yr[c]
Trespassers (current)	12 yr[d]
Ingestion Rate	
Workers (future)	100 mg/day[c,e]
Trespassers (current)	100 mg/day[c,e]
Average Body Weight	
Workers (future)	70 kg[c]
Trespassers (current)	43 kg[f]
Fraction Ingested	
Workers (future)	1.0
Trespassers (current)	0.5
Fraction Absorbed	1.9

[a] Based on exposure occurring 5 days a week for 7 months of the year (5/7 x 210 = 150)

[b] Based on exposure occurring 2 times a week (2/7 x 210 = 60)

[c] EPA (1989a, 1991b).

[d] Current exposure is for trespassers ages 6 through 18.

[e] EPA (1989j). Interim Final Guidance for Soil Ingestion Rates. Office of Solid Waste and Emergency Response. (OSWER Directive 9850.4)

[f] EPA (1989j). Exposure Factors Handbook. Office of Health and Environmental Assessment. EPA\600\8-89\043.

[g] 0.8 Absorption is assumed for arsenic in soil (ICF/Clement 1988).

Absorption of phenolic compounds from soil theoretically could be greater than the 0.1 default value. Gastrointestinal absorption of PCP may be greater than 90 percent and dermal absorption may vary from 10 to 50 percent (ATSDR 1989c). Relative absorption may range from 11 to 55 percent. The higher value, however, would be appropriate for PCP in oil formulations. Though this was the original form of release into the environment, it is not clear that PCP in surficial soils is still in an oil solution, and some binding of PCP to soil particles is expected. In addition, PCP may be present at neutral pH predominantly in anionic form which would tend to limit absorption. It was assumed that a value of 0.1 would adequately assess PCP absorption.

An incidental soil ingestion rate of 100 mg/day is used as recommended by RAGS (EPA 1989a) and the fraction of contaminated soil ingested is assumed to be 50 percent since individuals trespassing do not live on-site. A substantial fraction of soil ingested by these trespassers may thus come from areas other than the Montana Pole site. A body weight of 43 kg is used which is the average of male and female body weights for children ages 6 through 18 (EPA 1989i). Averaging times of 12 and 70 years (4,380 and 25,550 days) are used for noncarcinogens and carcinogens respectively. The equations for estimating exposure via dermal absorption and incidental ingestion of surface soil are as follows:

$$\text{Relative Dose (mg/kg/day)} = \frac{\text{CS x CF x SA x AF x ABS x EF x ED x FC}}{\text{BW x AT}}$$

where:
- CS = Chemical Concentration in Soil (mg/kg)
- CF = Conversion Factor (10^{-6} kg/mg)
- SA = Skin Surface Area Available for Contact (cm^2/day)
- AF = Soil to Skin Adherence Factor (mg/cm^2)
- ABS = Absorption Factor (unitless)
- EF = Exposure Frequency (days/year)
- FC = Fraction Contaminated (unitless)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT = Averaging Time (period over which exposure is averaged - days)

$$\text{Intake (mg/kg/day)} = \frac{\text{CS x IR x CF x FI x FA x EF x ED}}{\text{BW x AT}}$$

where: CS = Chemical Concentration in Soil (mg/kg)
IR = Ingestion Rate (mg/day)
CF = Conversion Factor (10^{-6} kg/mg)
FI = Fraction Ingested (unitless)
FA = Fraction Absorbed (unitless)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

5.2.1.3 Current On-Site Trespassers — Dermal Absorption and Incidental Ingestion of Surface Water and Sediments

Tables 5-6 and 5-7 present exposure assumptions used for quantifying exposure via direct contact and incidental ingestion of surface water and sediments in Silver Bow Creek. It has been reported that children often use Silver Bow Creek for recreational activities such as swimming and wading. (See Section 5.1.5 for the State's definition of "swimming"). Based on climatological data discussed previously, it is expected that exposure to surface water in Silver Bow Creek may occur for approximately five months of the year (May-September) when temperatures are sufficiently high. This results in 150 days of possible exposure to surface water and sediments. It is assumed for this scenario that children may swim in the creek approximately two times a week resulting in an exposure frequency of 43 days/year (150 x 2/7 = 43). An exposure time of 2 hrs/day is used. The national average time for swimming is 2.6 hrs/day (USDOI 1973 in EPA 1988c, EPA 1989a). However, given the shallow depth of the creek and the cool temperatures of Montana surface water, 2 hrs/day is a reasonable assumption. Additionally, a trespasser is assumed to spend only one half of the time in the reach of the creek contaminated by the Montana Pole site.

An exposure duration of 12 years is used for children ages 6 through 18. For dermal contact with surface water, dermal absorption estimates assume exposure to total body surface area using average 50th percentile values for males and females (ages 6-18). This value (13,050 cm^2) is provided by RAGS (EPA 1989a). A default value of 0.1 is used for absorption of organic chemicals from water sediments (EPA 1989a). Additional discussion is included in sections 5.2.2.1 and 5.2.2.2. An

TABLE 5-6

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA DIRECT CONTACT AND INCIDENTAL INGESTION OF SURFACE WATER

Parameter	Exposure Assumption
Frequency of Exposure Trespassers (current)	43 d/yr[a]
Exposure Duration Trespassers (current)	12 yr[b]
Skin Surface Area Trespassers (current)	13,050 cm^2 [c]
Exposure Time Trespassers (current)	2 hr/day[c]
Average Body Weight	43 kg[d]
Ingestion Rate	50 ml/hr[c]
Fraction Contaminated	0.5
Permeability Constant	8.4 E-04 cm/hr[c]

[a] Based on exposure occurring twice a week for 150 days (2/7 x 150 = 43)
[b] Exposure is for a trespasser ages 6 through 18
[c] EPA (1989a)
[d] EPA (1989i). Exposure Factors Handbook. Office of Health and Environmental Assessment. EPA\600\8-89\043.

TABLE 5-7

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA DIRECT CONTACT WITH SEDIMENTS

Parameter	Exposure Assumption
Frequency of Exposure Trespassers (current)	43 d/yr[a]
Period of Exposure Trespassers (current)	12 yr[b]
Skin Surface Area Trespassers (current)	13,050 cm^2 [c]
Average Body Weight Trespassers (current)	43 kg[d]
Skin Adherence Factor	2.0 mg/cm^2 [c]
Absorption Factor	0.1[e] 0.01[f]
Ingestion Rate Trespassers (current)	50 mg/day[b]
Fraction Contaminated	0.5

[a] Based on exposure occurring twice a week for 150 days (2/7 x 150 = 43)
[b] EPA (1989a)
[c] Current exposure is for a trespasser ages 6 through 18
[d] EPA (1989i). Exposure Factors Handbook. Office of Health and Environmental Assessment. EPA\600\8-89\043.
[e] For organic compounds other than dioxins/furans.
[f] For inorganic compounds and dioxins/furans.

average of body weight for male and female children ages 6 through 18 is used. This value is 43 kg and is obtained from the Exposure Factors Handbook (EPA 1989i). For incidental ingestion of surface water, a recommended ingestion rate of 50 ml/hour is used (EPA 1989a).

With the exception of exposure duration, frequency, and ingestion rate, the same exposure assumptions and equations used to evaluate exposure to surface soil are used for evaluating exposure to sediment. These are described in Section 5.2.1.2. For incidental ingestion of sediment, an ingestion rate of 50 mg/day (1/2 the daily soil ingestion rate for 6 to 18 year-olds) is used. This rate is based on the assumption that play in the shallow creek would involve constant intimate contact with the sediments and that play activities would stir sediment into the water column where it might be ingested incidentally with surface water (see above). The equations for estimation of exposure via dermal absorption and incidental ingestion of surface water are as follows:

$$\text{RelativeDose (mg/kg/day)} = \frac{CW \times SA \times PC \times ET \times EF \times ED \times FC \times CF}{BW \times AT}$$

where: CW = Chemical Concentration in Water (mg/liter)
SA = Skin Surface Area Available for Contact (cm^2)
PC = Dermal Permeability Constant (cm/hr)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
FC = Fraction Contaminated (unitless)
CF = Volumetric Conversion Factor for Water (1 liter/1000 cm^3)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

$$\text{Intake (mg/kg/day)} = \frac{CW \times IR \times ET \times EF \times ED \times FC}{BW \times AT}$$

where: CW = Chemical Concentration in Water (mg/liter)
IR = Ingestion Rate (liters/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
FC = Fraction Contaminated (unitless)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

5.2.2 FUTURE LAND USE SCENARIOS

5.2.2.1 Future On-Site Worker - Direct Contact and Incidental Ingestion of Soil

Future industrial workers at the Montana Pole site may be directly exposed to chemicals by dermal contact with contaminated surface soil. Further exposure could occur by inadvertent ingestion of soil through activities such as eating or smoking. Tables 5-4 and 5-5 present the assumptions used in assessing exposure by direct contact and incidental ingestion of contaminated surface soils by workers. As discussed previously, exposure to surface soils is considered likely for only seven months of the year (April - October) as the ground is likely to be frozen and/or covered with snow for the months of November through March (see Section 5.2.1.2). This results in approximately 210 days of possible exposure to soils. It is assumed under the exposure conditions evaluated that outside workers may be exposed to soil five days a week for seven months. Therefore, exposure to soil could occur on a total of 150 days per year (210 x 5/7 = 150). It is further assumed that a worker performs this type of job for 25 years (EPA 1991b). For dermal absorption, exposure to the hands and forearms and using 50th percentile body part-specific surface areas for males (EPA 1989a), a skin surface area of 3,120 cm^2,is used. Due to lack of site-specific information on soils, an adherence factor for commercial potting soil of 1.45 mg/cm^2 is used (EPA 1989a, 1988c).

For incidental ingestion of soil, a soil ingestion rate of 100 mg/day is used (EPA 1989a). Standard assumptions for average adult body weight (70 kg) (EPA 1989a) and averaging times (25 and 70 years for noncarcinogens and carcinogens, respectively), are used for both direct dermal contact with and incidental ingestion of soils. The equations used to calculate exposure via dermal absorption and incidental ingestion of surface soils are presented in Section 5.2.1.2.

5.2.2.2 Future Resident — Dermal Absorption and Incidental Ingestion of Soil

Future residents are likely to be exposed to contaminants present in surface soils when gardening and/or when engaging in recreational activities. As with other soil exposure pathways, there are approximately 350 days during which exposure to surface soils may occur. It is assumed that exposure might occur on each of these days, either directly to outdoor soils or indirectly to contaminated dust that has migrated into the house. A fraction of contaminated soil contacted of 0.7

is used since it is reasonable to assume that some contact/ingestion will occur away from home. In essence, this reduces exposure frequency to 245 days, about the number of days per year when the ground is not snow covered and/or frozen. Fraction of contaminated soil contacted is not provided in EPA guidance and is based on professional judgement and site-specific considerations.

Body weights of 19 kg and 59 kg are used for a young child and an adult, respectively (EPA 1989a). For incidental ingestion, ingestion rates of 160 and 120 mg/day are used for young children and adults, respectively. These values are based on the most recent EPA guidance (1989a). The exposure duration for a young child is 10 years starting from birth and incidental ingestion is prorated for ingestion of 200 mg/day for 6 years and 100 mg/day for 4 years. An exposure duration of 30 years is used for an adult resident. Incidental ingestion is prorated for 200 mg/day for 6 years and 100 mg/day for 24 years. All other exposure assumptions and the equations used to estimate exposure are the same as those presented in Section 5.2.1.2. Tables 5-8 and 5-9 present exposure assumptions for this pathway.

5.2.2.3 Future Resident — Ingestion of Home-grown Produce

Ingestion of vegetables grown within the study area could result in human exposure to the chemicals of potential concern. Vegetables could contain site-related chemicals through two routes:

- Uptake of chemicals from contaminated soils
- Deposition of contaminated windborne soil and dust particles onto plant surfaces.

Ingestion of produce grown on-site is evaluated under future use conditions for adults and children.

Evaluation of deposition of soil contaminants onto vegetables has consistently been shown to contribute only a small fraction to total vegetable contaminant loads (ICF/Clement 1988). For this reason, deposition of soil contaminants onto plant surfaces was not considered significant, and is not quantitatively evaluated. The only major source of contamination of vegetables is considered to be via root uptake.

TABLE 5-8

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA DERMAL CONTACT WITH SURFACE SOIL FOR RESIDENTS (FUTURE)[a]

Parameter	Exposure Assumption
Frequency of Exposure	
Children (future)	350 d/yr[b]
Residents (future)	350 d/yr
Exposure Duration	
Residents (future)	30 yr[b]
Children (future)	10 yr[b]
Skin Surface Area	
Residents (future)	3,476 cm^2 [c]
Children (current)	4,187 cm^2 [c]
Average Body Weight	
Residents (future)	59 kg[b]
Children (future)	19 kg[b]
Averaging Time	
Noncarcinogens	365 d/yr x 30 yr[b] (resident) 365 d/yr x 10 yr (child)
Carcinogens	365 d/yr x 70 yr[b]
Skin Adherence Factor	1.45 mg/cm^2 [c]
Fraction Contaminated	0.7
Absorption Factor	
Organics	0.1
Inorganics & Dioxins/Furans	0.01

[a] Chronic daily intakes (CDIs) for estimating cancer risks to future residents are conservatively based on exposure during the first 30 years of life. CDIs for estimating non-cancer risks are conservatively based on exposure for children ages 0 to 10 years old.

[b] EPA (1989a).

[c] EPA (1989a,i). 50th percentile body surface area for adult forearms and hands were used for adult residents; forearms, hands, and legs were used for children ages 0 through 10.

TABLE 5-9

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INCIDENTAL INGESTION OF SURFACE SOIL FOR RESIDENT (FUTURE)[a]

Parameter	Exposure Assumption
Frequency of Exposure	
Children (future)	350 d/yr[b]
Residents (future)	350 d/yr[b]
Exposure Duration	
Residents (future)	30 yr[b]
Children (future)	10 yr[b]
Ingestion Rate	
Residents (future)	120 mg/day[c,d]
Children (future)	160 mg/day[c,e]
Average Body Weight	
Residents (future)	59 kg[b]
Children (future)	19 kg[b]
Fraction Ingested	
Residents (future)	0.7
Children (future)	0.7

[a] CDIs for estimating cancer risks to future residents are conservatively based on exposure during the first 30 years of life. CDIs for estimating non-cancer risks are conservatively based on exposure for children ages 0 to 10 years old.

[b] EPA (1989a).

[c] EPA (1989k). Interim Final Guidance for Soil Ingestion Rates. Office of Solid Waste and Emergency Response. (OSWER Directive 9850.4)

[d] Soil ingestion prorated for incidental ingestion of 200 mg/day for ages 0 to 6 and 100 mg/day for ages 6 to 30.

[e] Soil ingestion prorated for incidental ingestion of 200 mg/day for ages 0 to 6 and 100 mg/day for ages 6 to 10.

Table 5-10 presents exposure assumptions for ingestion of home-grown produce by future on-site residents for COCs other than PCP. Exposure assumptions used for PCP are presented in Table 5-11, and explained in detail in Appendix C. It is assumed that chemicals in home-grown vegetables would not be removed by peeling or cooking prior to ingestion. This is considered reasonable since contamination of plant surfaces is not considered significant, and since many home gardeners may prefer to consume many common crops raw.

Based on climatological data discussed previously (see Section 2.1), it is assumed that ingestion of home-grown produce only occurs over a six-month period based on a growing season of four months. It is further assumed that an individual ingests vegetables twice weekly. This results in an exposure frequency of approximately 52 days (180 x 2/7 = 52). The small number of exposure days reflects the expectation that gardening will be limited by climate. These assumptions are similar to those used in previous assessments for Silver Bow Creek NPL site operable units (Clement Associates 1989).

An exposure duration of 30 years is assumed as recommended by RAGS (EPA 1989a). Ingestion of vine, leafy, and root crops is evaluated. Ingestion rates for these crops are obtained from USDA (1982). Average ingestion rates for vine, leafy, and root crops are 151, 144, and 114 grams/day, respectively. Vine crops consist of such vegetables as tomatoes, cucumbers, and string beans; leafy crops include lettuce, broccoli and spinach; and root crops include carrots, potatoes, and onions. For adults, a body weight of 59 kg is used and for children ages 1 through 10, a body weight of 19 kg is used.

Plant uptake factors for soil COCs were selected as described in Appendix C. The major focus of this analysis was on PCP. PCP was selected for emphasis because it is a major COC for soil at the site, and is the best characterized of all COCs. Estimates for soil concentrations of dioxins and furans are biased towards areas where higher concentrations are expected, and few data points are available. Data for other phenolics is similar. Inorganic compounds are included only for reference to other risk assessments for Butte area NPL sites and operable units and are not believed to be associated with the Montana Pole site.

TABLE 5-10

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INGESTION OF HOME GROWN VEGETABLES[b]

Parameter	Exposure Assumption
Frequency of Exposure	
Residents (future)	52 d/yr
Children (future)	52 d/yr
Exposure Duration	
Residents (future)	30 yr[b]
Children (future)	10 yr
Ingestion Rate	
Vine Crops	151 g/day
Leafy Crops	144 g/day
Root Crops	114 g/day
Body Weight	
Residents (future)	59 kg
Children (future)	19 kg
Averaging Time	
Noncarcinogen	365 d/yr x 10 yrs
Carcinogen	365 d/yr x 70 yrs
Fraction Absorbed	1[d]

[a] For compounds other than pentachlorophenol.

[b] CDIs for estimating cancer risk to future residents are conservatively based on exposure during the first 30 years of life. CDIs for estimating non-cancer risks are conservatively based on exposure for children ages 0 to 10 years old.

[c] EPA (1989a).

[d] For arsenic, absorption is assumed to be 80 percent.

TABLE 5-11

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INGESTION OF HOME GROWN VEGETABLES GROWN IN SOIL CONTAINING PENTACHLOROPHENOL[a]

Parameter	Exposure Assumption
Frequency of Exposure	
Residents (future)	52 d/yr
Children (future)	52 d/yr
Exposure Duration	
Residents (future)	6 yr[b]
Children (future)	6 yr
Ingestion Rate	
Root Crops	114 g/day
Body Weight	
Residents (future)	59 kg
Children (future)	19 kg
Averaging Time	
Noncarcinogen	365 d/yr x 6 yrs[a]
Carcinogen	365 d/yr x 70 yrs[a]
Fraction Absorbed	1[b]

[a] EPA (1989a).

[b] For arsenic, absorption is assumed to be 80 percent.

In addition, significant information on PCP degradation in soil and on uptake into plants is available to support a more rigorous analysis. A complete description of the interpretation of PCP literature is provided in Appendix C.

The equation used to evaluate this pathway is as follows:

$$\text{Intake (mg/kg/day)} = \frac{\text{CF x IR x FA x EF x ED}}{\text{BW x AT}}$$

where: CF = Contaminant Concentration in Food (mg/kg)
IR = Ingestion Rate (kg/day)
FA = Fraction Absorbed from Contaminated Source (unitless)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

It should be noted that no reliable vegetable ingestion rates for young children were located. Using values based on adult consumption may overestimate childhood exposure. However, for families with gardens, home grown vegetables, when available, may become the focus of meals. Consumption could increase for the few days when home grown produce is consumed. Adult ingestion rates, therefore, may not greatly overestimate children's consumption.

5.2.2.4 Future Resident - Ingestion of Groundwater

Although on-site groundwater wells are not currently used for drinking water, it is assumed that future residents could use on-site wells. To assess this exposure for future residents, it is assumed that individuals could be exposed for 350 days per year. An ingestion rate of 2 L/day is also assumed. Body weights of 19 kg and 59 kg were used for a child and adult, respectively (EPA 1989a). It is conservatively assumed that water ingested in the future would have the same degree of contamination as present groundwater.

The equation used to evaluate this pathway is as follows:

$$\text{Intake (mg/kg/day)} = \frac{\text{CW x IR x EF x ED x CF}}{\text{BW x AT}}$$

where: CW = Contaminant Concentration in Water (μg/L)
IR = Ingestion Rate (L/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
CF = Conversion Factor (mg/μg)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Exposure parameters for groundwater ingestion are provided in Table 5-12.

Exposure to COCs in groundwater could also occur during showering, bathing and dishwashing. Though potentially significant, this pathway is not quantified. Data are available for volatile organic compounds (see Section 7.6.8) which indicate that this source of exposure might be approximately equal to that from ingestion of contaminated water in residential settings. The bulk of this exposure (perhaps 70%) is thought to be due to inhalation of contaminants volatilized from the water. Thus, only about 30% of the exposure is expected to come from dermal uptake of chemicals. Since the COCs for the Montana Pole site are metals and semi-volatiles, little inhalation exposure is expected. Instead, intake would be through dermal uptake.

The volatile compounds studied for exposure via showering (e.g. chloroform) are absorbed through the skin relatively easily compared to dioxins/furans which are assigned a permeability factor of 0.01 in this assessment. PCP in water was also assigned a relatively low permeability in this assessment (0.1). These major contaminants would be expected to be taken up to a lesser extent through the skin than chemicals such as chloroform, resulting in relatively smaller exposures.

Since it is expected that exposure via showering, bathing and dishwashing for COCs for Montana Pole are likely to be small (less than 30% of those for exposure via ingestion of groundwater), this pathway was not quantitatively evaluated. Uncertainties for this pathway are further discussed in Section 7.6.8.

TABLE 5-12

ASSUMPTIONS USED TO ESTIMATE EXPOSURE VIA INGESTION OF GROUNDWATER[a]

Parameter	Exposure Assumption
Frequency of Exposure	
Residents (future)	350 d/yr[b]
Children (future)	350 d/yr[b]
Exposure Duration	
Residents (future)	30 yr[b]
Children (future)	10 yr
Ingestion Rate	
Residents (future)	2L/d[b]
Children (future)	2L/d[b]
Body Weight	
Residents (future)	59 kg[b]
Children (future)	19 kg[b]
Averaging Time	
Noncarcinogen	365 d/yr x 10 yrs[b]
Carcinogen	365 d/yr x 70 yrs[b]

[a] CDIs for estimating cancer risk to future residents are conservatively based on exposure during the first 30 years of life. CDIs for estimating non-cancer risks are conservatively based on exposure for children ages 0 to 10 years old.

[b] EPA (1989a)

5.3 EXPOSURE AREAS

5.3.1 SOILS

The Montana Pole site is relatively small. Few easily defined subareas, based on potential site access, were identified for which separate exposure calculations seemed justified. Instead, it is reasonable to assume that trespassers would visit fairly broad areas of the site. It is, however, appropriate that the northern part of the site, between the freeway and Silver Bow Creek, be considered separately since it is isolated from the rest of the site by Interstate 90. Further, this area of the site, because it is closer to the creek, is likely to be more attractive to children who might visit the site. Separate exposure point concentrations, thus, seem justified for this area. Future on-site workers are assumed to be exposed only in the southern area, based on current industrial conditions.

Because a small section of the site is currently fenced, trespassers may be less likely to visit this area. Over the long term, however, the fence cannot be considered a permanent barrier. Since this risk assessment is based on a no-action scenario, it is reasonable to assume greater access to the fenced area in the future. Still, risks may be lower for current site trespassers, and this is further discussed under uncertainties.

5.3.2 GROUNDWATER

Because of the relatively small size of the groundwater plume as currently defined (Keystone 1991), it was deemed reasonable to include all on-site wells in the shallow aquifer in calculations of exposure point concentrations. Since most wells were installed in the area affected by Montana Pole site-related contamination, this averaging is not expected to underestimate possible exposure point concentrations for groundwater. Separate exposure point concentrations were generated for some COCs from potentially downgradient wells located outside the plume as currently defined. These exposure points are the focus of a qualitative discussion of potential off-site risks (Section 7.4).

5.4 EXPOSURE POINT CONCENTRATIONS

Where large numbers of samples (> 30) were available, exposure point concentrations were calculated using the methods presented below. In all instances where sample sizes were large, data was log-normally distributed (see Appendix B for graphs of data by chemical). For these data sets, the 95 percent upper confidence limit (UCL) was calculated from the mean of the ln transformed data using the following equation.

$$UCL = e^{(x + 0.5s^2 + sH\sqrt{n-1})}$$

where: x = mean of ln transformed data
s = standard deviation
H = H statistic from statistical tables
n = number of data points

There is one exception. The estimated UCL for PCP was more than 1,400,000 μg/L for groundwater. This value exceeds the maximum detect value by a factor of 20. Since 57 samples were available for this calculation, the high UCL is not believed to reflect poor site characterization, and use of the maximum observed value was considered overly conservative. For this compound, an exposure point concentration was calculated using an upper 95 percent confidence limit on the arithmetic mean without *ln* transformation. The exposure point concentration for PCP in groundwater was calculated excluding Round 3 data containing transcriptional errors in the data summary tables supplied by Keystone. Methods and rationale for this calculation are provided in Section 5.4.1.1.

Exposure point concentrations for log-normally distributed data where sample numbers were < 30 were calculated using the methods presented in Gilbert's *Statistical Methods for Environmental Pollution Monitoring*. For these data sets, the 95 percent upper confidence limit (UCL) was calculated from the mean of the ln transformed data using the following equation.

$$UCL = e^{(x + (t \times (s.d. /\sqrt{n})}$$

Where: x = Mean of ln transformed data
t = Student-t statistic
s.d. = Standard deviation
n = Sample size

Where sample sizes are small (generally 6 to 16 data points), the first method (*ln* transformation) is unstable and often yields exposure point concentrations much higher than the maximum detected concentration. An effort was made to estimate a conservative exposure point concentration, without defaulting to the highest observed concentration. A discussion supporting final exposure point concentrations is provided for each chemical.

Where only one to a few data points were available, the maximum (or only) observed value is used as the exposure point concentration. This was necessary for all COCs in surface water and sediments.

No exposure point concentrations are estimated for inorganic COCs in surface water or sediment. A previous risk assessment addressed human health risks for these chemicals (CDM-FPC 1991) and this was deemed sufficient for evaluation of risks due to metal exposure in the reach of the creek adjacent to the Montana Pole site. Results of this assessment are summarized in Section 7.

Two analytical methods were used to analyze the samples for PAHs and phenols. The first, gas chromatography (GC) provides low detection limits, but the identification of the compounds in a complex matrix can be uncertain. The second, gas chromatography mass spectrometry (CGMS) provides precise compound identification in complex sample matrices but at higher detection limits. The GCMS data was included in the dataset used to calculate the 95 percent upper confidence limit when relatively high concentrations of the compounds were reported. The GC data was used when low concentrations of the compounds were reported.

Samples in which compounds were not detected at the detection limit are evaluated for this analysis according to EPA guidance (EPA 1989a). This guidance states that, when only some samples in a medium test positive for a chemical, samples that were not positively detected at the detection limit should be considered present at one-half of the detection limit. This value was included in the exposure point concentration calculation if it was less than the maximum concentration observed.

5.4.1 SOILS

Sampling points used for estimation of exposure point concentrations for soil COCs are provided in Appendix B. Only data from depth intervals from 0 to 2 feet were included in an attempt to assess possible surficial contamination. Sample depth is further discussed in Section 4.5.2.2.

5.4.1.1 Dioxins/Furans

Limited analyses for dioxins and furans were available for calculation of an exposure point concentration. Samples taken were mainly clustered and cannot be said to be representative of the site. The small sample size, and the lack of samples from many areas of the site make generation of an accurate exposure point concentration difficult. For the purposes of this assessment the rationale presented below was adopted.

Concentrations of OCDD were compared with concentrations of PCP from samples taken at the same locations, or from sampling points nearby (Table 5-9). OCDD is expected to be, by weight, the major dioxin/furan on-site, because it is the major dioxin contaminant of technical grade PCP, and because PCP is believed to be the major source of dioxins/furans. OCDD is, hence, used as a surrogate for all dioxin/furan congeners. It might be expected that the highest concentrations of OCDD would occur in locations where PP concentrations were also high. No clear relationship is apparent in Table 5-13, but the highest OCDD concentration does coincide with a co-located PCP sample that is also high (sample location MOPO-SS-A14).

The data are at least consistent with the expectation that high OCDD levels may correlate spatially with areas of heavy PCP contamination. This suggests that areas with high concentrations of PCP, but which have not been sampled for dioxins/furans, may also contain high concentrations of OCDD and other dioxin/furan congeners. Such areas are found at certain sample locations within the fenced area of the site (opportunistic samples designated SL-0xx-x where x's indicate existence of several sample sites).

Uncertainty in available data suggests a conservative approach to estimating exposure point concentrations for dioxins/furans. Due to the limited number of samples in the northern area (SS-A13

TABLE 5-13

COMPARISON OF CO-LOCATED PCP & OCDD SAMPLE RESULTS

Sample Location	PCP (µg/kg)	OCDD (µg/kg)
MOPO-SS-A13	88,200	17.16E
MOPO-SS-A14	300,000	5020E
MOPO-SS-A23	216/201[a]	0.0176UE
MOPO-SS-A260	216/201[a]	40.55
MOPO-SS-A6	148	65.45
SS-A60	216/201[a]	37E
SS-B3	1,700	33.1
SS-B6	1,800	20.1

[a] Not co-located. Values are from two closest sampling points (SL-W17-D & SL-W18-D) from which data are available.

and SSA-14) the maximum value observed was used as the exposure point concentration. Use of the maximum concentration observed was considered for the southern area; however, the existence of many sampling points with relatively low PCP concentrations suggests that high levels of OCDD and other congeners will not be found over large areas of the site. A less restrictive assumption is that dioxin/furan concentrations are normally distributed on the site. Assuming such a distribution, an exposure concentration can be generated as the upper 95 percent confidence limit on the arithmetic mean as:

$$UCL = mean + (t \times (s.d. / \sqrt{n}))$$

where: UCL = Upper 95 percent confidence limit
t = Student-t statistic
s.d. = Standard deviation
n = Sample size

The resulting exposure point concentration is significantly influenced by the higher dioxin/furan concentrations, but is also significantly lower than the maximum values. This allows the exposure point concentrations to reflect the potential for additional "hot spots" while still recognizing that many unsampled areas of the site are likely to have relatively low dioxin/furan soil concentrations.
It should be noted that it is likely that the actual distribution of dioxin/furan concentrations in soil is log-normal, and a more extensive data set would likely bear this out. A geometric mean is the appropriate measure of central tendency for log-normally distributed data. However, little confidence could be placed in a geometric mean from the eight non-random samples currently available. The small sample size suggests that the actual distribution of dioxin/furans is not known with accuracy.

5.4.1.2 PAHs

Data for PAHs, though more extensive, are qualitatively similar to those for dioxins/furans. It was deemed appropriate to estimate exposure point concentrations using the methods described for dioxins/furans.

5.4.1.3 Inorganic COCs

Limited data for inorganic COCs (see Section 4.5.2) make accurate estimation of exposure point concentrations difficult. No source for inorganic COCs has been identified on-site and no substantial "hot spots" are predicted to occur, except near Silver Bow Creek which has been affected by historical mining activity. Exposures to metals in surface water and sediment are addressed in CDM-FPC (1991) and these results are discussed in Sections 7.2 and 7.3.

5.4.1.4 PCP

Four types of surficial soil data are available for PCP. These include opportunistic grab samples taken in areas where contamination was visible or likely, grid samples, backhoe pit samples and soil borings. Averaging of opportunistic soil samples with those from more systematic sampling may bias the exposure point concentration upwards by emphasizing areas with high soil levels of COCs. Eliminating opportunistic samples, however, would, in this case, mean that no data would be included from areas known to have high contamination, especially in the northern portion of the site. For this reason, opportunistic samples were included in the calculation of exposure point concentrations for PCP.

The effect of including opportunistic samples could be large. When only grid point samples were included in an estimate, the resulting value (0.69 mg/kg) was significantly lower than the exposure point concentration used in the assessment (319 mg/kg). Apparently, levels of contamination in areas where opportunistic samples were taken are substantially greater than in areas covered by the grid. A more representative exposure point concentration probably lies somewhere between these two values. Adjustment of the exposure point concentration would, however, be arbitrary and difficult to support. Thus, the higher value is adopted to ensure protectiveness.

Exposure point concentrations for soil COCs are provided in Table 5-14.

TABLE 5-14

EXPOSURE POINT CONCENTRATIONS
SURFICIAL SOILS (μg/kg)

	Southern Area	Northern Area
Anthracene	51.07	224.95
Benzo(a)anthracene	20.25	6825.61
Benzo(b)fluoranthene	18.30	476.06
Benzo(k)fluoranthene	8.74	457.42
Benzo(a)pyrene	12.04	270.23
Indeno(1,2,3-cd)pyrene	15.99	338.89
4-chloro-3-methylphenol	765.09	6605.55
2-methyl-4,6,-dinitrophenol	11445.54	14759.28
Pentachlorophenol	319070.4	61943.0
2,4,6-trichlorophenol	1492.55	7212.23
OCDD	46.79	5020
1234678-HpCDD	4.23	469
1234789-HpCDF	.013	12.9
123789HxCDD	.019	1.7
123678HxCDD	.126	14.9
123478HxCDD	.015	1.4
12378PeCDD	.004	0.0067
2378-TCDF	.002	0.421
2378-TCDD	.008	0.0106
1234678-HpCDF	.298	81.8
123678HxCDF	.0371	2.6
234678HxCDF	.0142	2
123789HxCDF	0	0.00056
123478HxCDF	.037	17.1
OCDF	.787	433
23478PeCDF	.0049	2.2

TABLE 5-14 (Cont.)

EXPOSURE POINT CONCENTRATIONS
SURFICIAL SOILS (μg/kg)

	Southern Area	Northern Area
12378PeCDF	.0064	2
Arsenic	40985.21	147177.10
Cadmium	789.25	1862.56
Chromium	11047.69	9829.16

5.4.2 GROUNDWATER

Wells used for estimation of exposure point concentrations for groundwater COCs are provided in Appendix B. Only on-site wells completed above the bedrock were included in the calculation of exposure point concentrations. A few wells (e.g. MW-15) screened across the alluvial/bedrock interface were thus excluded from the calculations. Since two nearby residential wells are completed in the shallow alluvial aquifer above bedrock, it was assumed that any well installed on-site would be of similar depth. Wells included in the calculations still cover a wide range of depths in the alluvial aquifer and are thought to represent water quality in this aquifer adequately.

The exclusion of the deepest wells screened at or below bedrock is further supported by depths reported from nearby residential wells. The closest well to the site is completed in the alluvial aquifer (CDM 1989). Two residential wells sampled in the RI (Keystone 1991), are completed at depths of 25 feet and 55 feet. These depths are generally less than the depth to bedrock found beneath the Montana Pole site. Both are likely mostly or wholly dependent on the alluvial aquifer.

Exposure point concentrations for groundwater COCs are provided in Table 5-15.

5.4.3 SURFACE WATER AND SEDIMENT

Because of small sample sizes, maximum observed concentrations at sample station SW/SD-005 are used as exposure point concentrations. These data are presented in Table 5-16, and are illustrated in Figures 8-2, 8-5, and 8-6 for PCP and dibenzo(a,h)anthracene. As previously discussed, use of data from SW/SD-005 is very conservative, since the location is a seep of NAPLs, and calculated CDIs are considered "worst case" rather than reasonable maximum exposures.

5.5 CHRONIC DAILY INTAKES

Estimates of chronic daily intakes are discussed in the following subsections by scenario and by medium for each of the COCs.

TABLE 5-15

EXPOSURE POINT CONCENTRATIONS FOR GROUNDWATER

	µg/L
Acenaphthene	474.08
Acenaphthylene	238,069.08
Anthracene	259.85
Benzo(a)pyrene	69.63
Benzo(a)anthracene	7,199.97
Benzo(b)fluoranthene	0.18
Benzo(g,h,i)perylene	9.62
Benzo(k)fluoranthene	35.89
Chrysene	19,805.83
Dibenzo(a,h)anthracene	18.75
Fluoranthene	421.12
Fluorene	42,850.20
Indeno(1,2,3-cd)pyrene	1.29
2-methylnaphthalene	4,039.26
Naphthalene	4,259.54
Phenanthrene	3,817.27
Pyrene	848.02
4-chloro-3-methylphenol	331.13
2,4-dichlorophenol	985.15
2-methyl-4,6-dinitrophenol	381.94
2,3,5,6-tetrachlorophenol	3,090.53
2,4,6-trichlorophenol	231.89
Pentachlorophenol	6,506.98
2-chlorophenol	40.47
2,4-dinitrotoluene	220.51
1234678-HpCDD	1.66

TABLE 5-15 (Cont.)

EXPOSURE POINT CONCENTRATIONS
FOR GROUNDWATER

	µg/L
1234678-HpCDF	0.182
1234789-HpCDF	0.0156
123678HxCDD	0.0653
123789HxCDD	0.0097
123478HxCDF	0.0468
123678HxCDF	0.0085
234678HxCDF	0.0179
OCDD	14.96
OCDF	0.543
12378PeCDF	0.0072
23478PeCDF	0.007
Arsenic	23.14
Chromium	28.39
Copper	139.51
Lead	29.68
Manganese	2,493.35

[a] 95 percent upper confidence limit on geometric mean unless otherwise noted.
[b] 95 percent upper ocnfidence limit on arithmetic mean.
[c] Maximum detected concentration due to limited sample numbers.

TABLE 5-16

EXPOSURE POINT CONCENTRATIONS
SURFACE WATER AND SEDIMENTS

	Surface Water µg/L	Sediments µg/kg
Pentachlorophenol	591	—
Benzo(a)anthracene	1.5	—
Benzo(a)pyrene	0.2	—
Benzo(b)fluoranthene	0.4	—
Chrysene	9.0	—
Dibenzo(a,h)anthracene	0.6	—
Pyrene	1.36	—
Dioxins/Furans	—	1.4
Arsenic	24.9	—
Copper	220	—
Lead	30	—

— = not considered a COC for this medium

5.5.1 CURRENT ON-SITE TRESPASSERS

5.5.1.1 Dermal Contact with and Incidental Ingestion of Soil

Intake rates for PCP for estimation of carcinogenic risks are estimated to be 1.1×10^{-5} for ingestion of contaminated soil, and 7.8×10^{-5} for dermal absorption from contaminated soil. Total exposure to PCP via these routes is 8.9×10^{-5} mg/kg-day. Other exposures are significantly less. In particular, intakes of dioxins/furans are expected to be 4.3×10^{-12} and 3.2×10^{-12} mg/kg-day for oral and dermal routes, respectively. Intakes of 2,4,6-trichlorophenol and arsenic are 4.9×10^{-8} and 1.1×10^{-6} respectively for the oral route, and 3.7×10^{-7} and 1.0×10^{-6} for the dermal route.

Intakes for estimation of non-cancer risks are somewhat (about six times) higher since these intakes are averaged over ages 6 to 18, when body weights are lower. Chronic daily intakes for these pathways are summarized in Tables 5-17 and 5-18 .

Future on-site trespassers are expected to receive similar exposures. No separate intakes were estimated for this scenario.

It should be noted here that for all dermal exposure pathways, PAHs are not quantified. As discussed in detail in Section 6.3.10, route of entry effects and metabolism make it difficult to justify extrapolating cancer slope factors from the oral to the dermal routes. Potential contributions of PAHs to risks via dermal exposures are further discussed in Section 7.6.14.

5.5.1.2 Dermal Contact with and Incidental Ingestion of Surface Water & Sediment

Only PCP is assessed for exposure via dermal contact with surface water, since it was the only organic COC for this media for which extrapolation to dermal exposure is appropriate. PAHs were not evaluated because of route of entry effects and metabolism. Intake of PCP is estimated to be 3.0×10^{-6} mg/kg-day based on exposures for carcinogenic risks. Exposures for assessing non-cancer risks are about 6-fold higher due to the much shorter averaging time. This holds true for intake estimates for other chemicals and pathways for this scenario.

TABLE 5-17

ESTIMATED CHRONIC DAILY INTAKES FROM SOIL INGESTION FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chemical Concentration Cs(mg/kg)	Ingestion Rate IR(mg/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	3.19E+02	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	25550	1.05E-05
Dioxins/Furans (TEFs)	1.31E-04	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	25550	4.29E-12
2,4,6-Trichlorophenol	1.49E+00	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	25550	4.89E-08
Benzo(a)pyrene (TEFs)	3.04E-02	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	25550	9.96E-10
Arsenic	4.10E+01	100	60	12	1.0E-06	8.00E-01	5.0E-01	43	25550	1.07E-06
Noncarcinogenic Exposure										
Pentachlorophenol	3.19E+02	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	6.10E-05
Dioxins/Furans (TEFs)	1.31E-04	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	2.50E-11
2,4,6-Trichlorophenol	1.49E+00	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	2.85E-07
4-chloro-3-methylphenol	7.65E-01	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	1.46E-07
2-methyl-4,6-dinitrophenol	1.14E+01	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	2.18E-06
Anthracene	5.11E-02	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	9.76E-09
Arsenic	4.10E+01	100	60	12	1.0E-06	8.00E-01	5.0E-01	43	4380	6.27E-06
Cadmium	7.89E-01	100	60	12	1.0E-06	1.00E+00	5.0E-01	43	4380	1.51E-07

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x FI x ABS/ BW x AT

NA = Not Applicable

TABLE 5-18

ESTIMATED CHRONIC DAILY INTAKES FOR DERMAL CONTACT WITH SOIL FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chemical Concentration Cs(mg/kg)	Surface Area SA(cm^2/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contam. FI	Body Weight BW(kg	Averaging Time AT(d/yrxyr)	Adherance Factor AF (mg/cm^2)	Chronic Daily Intake (mg/kg-day)
Pentachlorophenol	3.19E+02	5165	60	12	1.0E-06	0.10	5.0E-01	43	25550	1.45	7.83E-05
Dioxins/Furans (TEFs)	1.31E-04	5165	60	12	1.0E-06	0.01	5.0E-01	43	25550	1.45	3.21E-12
2,4,6-Trichlorophenol	1.49E+00	5165	60	12	1.0E-06	0.10	5.0E-01	43	25550	1.45	3.66E-07
Arsenic	4.10E+01	5165	60	12	1.0E-06	0.01	5.0E-01	43	25550	1.45	1.01E-06
Noncarcinogens											
Pentachlorophenol	3.19E+02	5165	60	12	1.0E-06	0.10	5.0E-01	43	4380	1.45	4.57E-04
Dioxins/Furans (TEFs)	1.31E-04	5165	60	12	1.0E-06	0.01	5.0E-01	43	4380	1.45	1.88E-11
2,4,6-Trichlorophenol	1.49E+00	5165	60	12	1.0E-06	0.10	5.0E-01	43	4380	1.45	2.14E-06
4-chloro-3-methylphenol	7.65E-01	5165	60	12	1.0E-06	0.10	5.0E-01	43	4380	1.45	1.10E-06
Arsenic	4.10E+01	5165	60	12	1.0E-06	0.01	5.0E-01	43	4380	1.45	5.87E-06
Cadmium	7.89E-01	5165	60	12	1.0E-06	0.01	5.0E-01	43	4380	1.45	1.13E-07

CDI(mg/kg-day)=Cs x SA x AF x AbF x EF x ED x CF x FI/BW/AT

Dioxins/Furans (specifically OCDD) are the only organic COCs for sediments. Intakes from dermal absorption are estimated to be 8.6×10^{-14} mg/kg-day (cancer risk).

Inorganic COCs are not addressed since a more comprehensive risk assessment for metals in this reach of Silver Bow Creek has recently been completed (CDM-FPC 1991). Results of this assessment are summarized in Section 7.

Dermal intakes from surface water and sediments are summarized in Table 5-19.

Intakes of PCP from ingestion of surface water are estimated to be 2.8×10^{-5} mg/kg-day (cancer risk). This is about 10 times larger than intakes from the dermal route. Intakes of PAHs were much less, about 6×10^{-8} mg/kg-day, based on benzo(a)pyrene equivalents.

Ingestion of sediments is expected to result in a chronic daily intake of 1.6×10^{-14} mg/kg-day of dioxins/furans (based on TCDD equivalents) for cancer risks. This is about 4 times less than exposures from dermal contact.

Tables 5-20 and 5-22 summarize chronic daily intakes from incidental ingestion of surface water and sediments.

5.5.2 FUTURE ON-SITE WORKER

Intake rates for future on-site workers are estimated to be about six times that for on-site trespassers. For example, intake of PCP for workers via soil ingestion is estimated to be 6.7×10^{-5} mg/kg-day, compared with 1.1×10^{-5} for trespassers. The difference lies in the greater exposure frequency and higher fraction of soil ingested from a contaminated source.

Intakes for estimation of non-cancer risks exceed intakes for estimation of cancer risks by a factor of about three. The difference is accounted for by the shorter averaging time for non-cancer (25 years) than for carcinogenic (70 years) effects.

Intakes for this pathway are provided in Tables 5-22 and 5-23.

TABLE 5-19

ESTIMATED CHRONIC DAILY INTAKES FROM DERMAL CONTACT WITH SURFACE WATER AND SEDIMENTS FOR ON-SITE TRESPASSERS

SURFACE WATER

Chemical	Chemical Concentration Cs(mg/L)	Surface Area SA(cm2)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(L/cm3)	Permeability Constant PC(cm/hr)	Exposure Time ET(hr/d)	Fraction Contaminated FC	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure											
Pentachlorophenol	5.9E-01	13050	43	12	1.0E-03	8.4E-04	2.0E+00	0.5	43	25550	3.04E-06
Noncarcinogenic											
Pentachlorophenol	5.9E-01	13050	43	12	1.0E-03	8.4E-04	2.0E+00	0.5	43	4380	1.77E-05

CDI(mg/kg-day) = Cs*SA*EF*ED*CF*PC*ET*FC/BW/AT

SEDIMENTS

Chemical	Chemical Concentration Cs(mg/kg)	Surface Area SA(cm2)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Adherence Factor AF(mg/cm2)	Fraction Contaminated FC	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure											
Dioxins/Furans	1.4E-06	13050	43	12	1.0E-06	1.0E-02	2.0E+00	0.5	43	25550	8.58E-14
Noncarcinogenic											
Dioxins/Furans	1.4E-06	13050	43	12	1.0E-06	1.0E-02	2.0E+00	0.5	43	4380	5.01E-13

CDI (mg/kg-day) = Cs*SA*EF*ED*CF*ABS*AF*FC/BW/AT

TABLE 5-20

ESTIMATED CHRONIC DAILY INTAKES FROM INGESTION OF SURFACE WATER FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chemical Concentration Cs(ug/l)	Ingestion Rate IR(L/hr)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(mg/ug)	Absorption Factor ABS	Exposure Time (hr/day)	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	5.91E+02	0.05	43	12	1.0E-03	NA	2.0E+00	43	25550	2.78E-05
Benzo(a)pyrene (TEFs)	1.27E+00	0.05	43	12	1.0E-03	NA	2.0E+00	43	25550	5.96E-08
Noncarcinogenic Exposure										
Pentachlorophenol	5.91E+02	0.05	43	12	1.0E-03	NA	2.0E+00	43	4380	1.62E-04
Pyrene	1.36E+00	0.05	43	12	1.0E-03	NA	2.0E+00	43	4380	3.73E-07

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x ABS x ET /BW/AT

TABLE 5-21

ESTIMATED CHRONIC DAILY INTAKES FROM SEDIMENT INGESTION FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chemical Concentration Cs(mg/kg)	Ingestion Rate IR(mg/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Dioxin/Furans	1.4E-06	50	43	12	1.0E-06	NA	5.0E-01	43	25550	1.64E-14
Noncarcinogenic Exposure										
Dioxin/Furans	1.4E-06	50	43	12	1.0E-06	NA	5.0E-01	43	4380	9.59E-14

CDI (mg/kg-day) = Cs x IR x EF x ED x CF x ABS x FI /BW/AT

5.5.3 FUTURE ON-SITE RESIDENTS

Chronic daily intakes for these receptors that involve surface soils (Sections 5.5.3.2 and 5.5.3.3) are discussed first for the southern area and secondly for the northern section. This is primarily due to predicted land use patterns (see Section 5.1.2).

5.5.3.1 Ingestion of Groundwater

Chronic daily intakes from ingestion of groundwater are greater than those from other direct exposure pathways. For example, PCP intakes are estimated to be 9.1×10^{-2} mg/kg-day (cancer risk) compared with 1.9×10^{-4} mg/kg-day from incidental soil ingestion.

Indirect exposure via ingestion of home produce grown in contaminated soils (see Section 5.5.3.3) is lower than that from ingestion of groundwater, but does contribute significantly to risks. The chronic daily intake of PCP is expected to be 0.24 mg/kg-day (cancer risk) or about 5 percent of the intake from groundwater.

Chronic daily intakes from ingestion of groundwater are provided in Table 5-24.

5.5.3.2 Dermal Contact with and Incidental Ingestion of Soil

Southern Area

As expected from the larger exposure frequencies, intakes for this scenario (residential) are higher than for others. For example, PCP intakes for soil ingestion are 1.9×10^{-4} and 1.8×10^{-3} for estimation of carcinogenic and non-cancer risks, respectively. These are about three times higher than those expected for on-site workers, and nearly ten times higher than those for on-site trespassers. This scenario is thus the most restrictive for dermal contact and soil ingestion pathways.

Chronic daily intakes for these pathways in the southern area are provided in Tables 5-25 and 5-26.

TABLE 5-22

ESTIMATED CHRONIC DAILY INTAKES FROM SOIL INGESTION FOR FUTURE ON-SITE WORKERS OF THE SOUTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Ingestion Rate IR(mg/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	3.19E+02	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	25550	6.69E-05
Dioxins/Furans (TEFs)	1.31E-04	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	25550	2.75E-11
2,4,6-Trichlorophenol	1.49E+00	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	25550	3.13E-07
Benzo(a)pyrene (TEFs)	3.04E-02	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	25550	6.37E-09
Arsenic	4.10E+01	100	150	25	1.0E-06	8.00E-01	1.0E+00	70	25550	6.87E-06
Noncarcinogenic Exposure										
Pentachlorophenol	3.19E+02	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	1.87E-04
Dioxins/Furans (TEFs)	1.31E-04	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	7.69E-11
2,4,6-Trichlorophenol	1.49E+00	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	8.76E-07
4-chloro-3-methylphenol	7.65E-01	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	4.49E-07
2-methyl-4,6-dinitrophenol	1.14E+01	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	6.72E-06
Anthracene	5.11E-02	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	3.00E-08
Arsenic	4.10E+01	100	150	25	1.0E-06	8.00E-01	1.0E+00	70	9125	1.92E-05
Cadmium	7.89E-01	100	150	25	1.0E-06	1.00E+00	1.0E+00	70	9125	4.63E-07

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x FI x ABS/ BW x AT

NA = Not Applicable

TABLE 5-23

ESTIMATED CHRONIC DAILY INTAKES FOR DERMAL CONTACT WITH SOIL
FOR FUTURE ON-SITE WORKERS OF THE SOUTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Surface Area SA (cm^2/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contam. FI	Body Weight BW(kg	Averaging Time AT(d/yrxyr)	Adherance Factor AF (mg/cm^2)	Chronic Daily Intake (mg/kg-day)
Pentachlorophenol	3.19E+02	3120	150	25	1.0E-06	0.10	1.0E+00	70	25550	1.45	3.03E-04
Dioxins/Furans (TEFs)	1.31E-04	3120	150	25	1.0E-06	0.01	1.0E+00	70	25550	1.45	1.24E-11
2,4,6-Trichlorophenol	1.49E+00	3120	150	25	1.0E-06	0.10	1.0E+00	70	25550	1.45	1.42E-06
Arsenic	4.10E+01	3120	150	25	1.0E-06	0.01	1.0E+00	70	25550	1.45	3.89E-06
Noncarcinogens											
Pentachlorophenol	3.19E+02	3120	150	25	1.0E-06	0.10	1.0E+00	70	9125	1.45	8.47E-04
Dioxins/Furans (TEFs)	1.31E-04	3120	150	25	1.0E-06	0.01	1.0E+00	70	9125	1.45	3.48E-11
2,4,6-Trichlorophenol	1.49E+00	3120	150	25	1.0E-06	0.10	1.0E+00	70	9125	1.45	3.96E-06
4-chloro-3-methylphenol	7.65E-01	3120	150	25	1.0E-06	0.10	1.0E+00	70	9125	1.45	2.03E-06
Arsenic	4.10E+01	3120	150	25	1.0E-06	0.01	1.0E+00	70	9125	1.45	1.09E-05
Cadmium	7.89E-01	3120	150	25	1.0E-06	0.01	1.0E+00	70	9125	1.45	2.10E-07

CDI(mg/kg-day)=Cs x SA x AF x AbF x EF x ED x CF x FI/BW/AT

TABLE 5-24

ESTIMATED CHRONIC DAILY INTAKES FROM INGESTION OF GROUNDWATER FOR FUTURE ON-SITE RESIDENTS

Chemical	Chemical Concentration Cs(ug/L)	Ingestion Rate IR(L/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(mg/ug)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	6.5E+03	2	350	30	1.0E-03	NA	1.0E+00	59	25550	9.06E-02
Dioxins/Furans(TEFs)	5.3E-02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	7.35E-07
2,4,6-Trichlorophenol	2.3E+02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	3.23E-03
Benzo(a)pyrene(TEFs)	3.0E+02	2	350	30	1.0E-03	NA	1.0E+00	59	25550	4.23E-03
Arsenic	2.3E+01	2	350	30	1.0E-03	NA	1.0E+00	59	25550	3.22E-04
Noncarcinogenic Exposure										
Pentachlorophenol	6.5E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	6.57E-01
Dioxins/Furans (TEFs)	5.3E-02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	5.33E-06
2,4,6-Trichlorophenol	2.3E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.34E-02
PAH (Total non-carcinogen)(a)	3.0E+05	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.02E+01
2-chlorophenol	4.0E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	4.08E-03
Arsenic	2.3E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.36E-03
Copper	1.4E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	1.41E-02
Manganese	2.5E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.52E-01
Lead	3.0E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.00E-03
Chromium	2.8E+01	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.87E-03
2,4-Dichlorophenol	9.9E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	9.94E-02
2,4-Dinitrotoluene	2.2E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	2.23E-02
4-Chloro-3-methylphenol	3.3E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.34E-02
2-Methyl-4,6-dinitrophenol	3.8E+02	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.86E-02
2,3,5,6-Tetrachlorophenol	3.1E+03	2	350	10	1.0E-03	NA	1.0E+00	19	3650	3.12E-01

$$CDI(mg/kg\text{-}day) = Cs \times IR \times EF \times ED \times CF \times FI \times ABS / BW \times AT$$

NA = Not Applicable

(a) All PAHs detected except benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(g,h,i)perylene, chrysene, dibenz(a,h) anthracene and indeno(1,2,3-cd)pyrene

Northern Area

Chronic daily intakes from incidental soil for future residents are highest for arsenic (cancer) and (noncancer CDI). These estimated intakes are 6.9×10^{-5} and 6.7×10^{-5} mg/kg-day, respectively. For dermal contact with soil PCP exposures were 1.5×10^{-4} and 1.3×10^{-3} mg/kg-day for cancer and noncancer CDIs, respectively Chronic daily intakes for these pathways in the northern area are provided in Tables 5-27 and 5-28.

5.5.3.3 Ingestion of Homegrown Produce

Southern Area

Intakes from ingestion of homegrown produce are from one to four orders of magnitude greater than those from direct soil ingestion or dermal absorption. Total chronic daily intake of PCP, for example, is 7.4×10^{-3} mg/kg-day compared with 1.9×10^{-4} for soil ingestion and 7.8×10^{-4} for dermal absorption, based on exposures for estimating carcinogenic risks. Efficient transfer of phenolics to plant tissue accounts for these high intakes. Intakes from homegrown produce may contribute significantly to total exposures for the phenolics, and for cadmium.

For intakes for dioxins/furans and arsenic, exposures via the ingestion of homegrown produce are closer to those from soil ingestion or dermal absorption. For dioxins/furans, exposure is estimated at 7.2×10^{-10} mg/kg-day for produce ingestion and 7.6×10^{-11} for incidental ingestion of soil, based on exposures for estimating carcinogenic risks. Arsenic intakes are 2.7×10^{-4} and 2.0×10^{-5} mg/kg-day for produce and soil ingestion, respectively.

For benzo(a)pyrene toxicity equivalency factors (TEFs), a similar comparison yields chronic intake estimates of 6.4×10^{-7} and 1.8×10^{-8} mg/kg-day. For these COCs, intake via the ingestion of homegrown produce is expected to contribute equally to overall exposures. (For discussion of TEFs see Section 6.3.10).

Intakes for homegrown produce from the southern area are provided in Table 5-29. Table 5-30 provides a summary of intake estimates for all crop types from the southern area.

TABLE 5-25

ESTIMATED CHRONIC DAILY INTAKES FROM SOIL INGESTION FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Ingestion Rate IR(mg/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	3.19E+02	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	1.86E-04
Dioxins/Furans (TEFs)	1.31E-04	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	7.64E-11
2,4,6-Trichlorophenol	1.49E+00	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	8.70E-07
Benzo(a)pyrene (TEFs)	3.04E-02	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	1.77E-08
Arsenic	4.10E+01	120	350	30	1.0E-06	8.00E-01	7.0E-01	59	25550	1.91E-05
Noncarcinogenic Exposure										
Pentachlorophenol	3.19E+02	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	1.80E-03
Dioxins/Furans (TEFs)	1.31E-04	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	7.40E-10
2,4,6-Trichlorophenol	1.49E+00	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	8.44E-06
4-chloro-3-methylphenol	7.65E-01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	4.32E-06
2-methyl-4,6-dinitrophenol	1.14E+01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	6.44E-05
Anthracene	5.11E-02	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	2.89E-07
Arsenic	4.10E+01	160	350	10	1.0E-06	8.00E-01	7.0E-01	19	3650	1.85E-04
Cadmium	7.89E-01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	4.46E-06

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x FI x ABS/ BW x AT

NA = Not Applicable

TABLE 5-26

ESTIMATED CHRONIC DAILY INTAKES FOR DERMAL CONTACT WITH SOIL FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Surface Area SA(cm^2/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contam. FI	Body Weight BW(kg	Averaging Time AT(d/yrxyr)	Adherance Factor AF (mg/cm^2)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure											
Pentachlorophenol	3.19E+02	3476	350	30	1.0E-06	0.10	7.0E-01	59	25550	1.45	7.84E-04
Dioxins/Furans (TEFs)	1.31E-04	3476	350	30	1.0E-06	0.01	7.0E-01	59	25550	1.45	3.22E-11
2,4,6-Trichlorophenol	1.49E+00	3476	350	30	1.0E-06	0.10	7.0E-01	59	25550	1.45	3.67E-06
Arsenic	4.10E+01	3476	350	30	1.0E-06	0.01	7.0E-01	59	25550	1.45	1.01E-05
Noncarcinogenic Exposure											
Pentachlorophenol	3.19E+02	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	6.84E-03
Dioxins/Furans (TEFs)	1.31E-04	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	2.81E-10
2,4,6-Trichlorophenol	1.49E+00	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	3.20E-05
4-chloro-3-methylphenol	7.65E-01	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	1.64E-05
Arsenic	4.10E+01	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	8.79E-05
Cadmium	7.89E-01	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	1.69E-06

CDI(mg/kg-day)=Cs x SA x AF x AbF x EF x ED x CF x FI/BW/AT

TABLE 5-27

ESTIMATED CHRONIC DAILY INTAKES FROM SOIL INGESTION FOR FUTURE ON-SITE RESIDENTS OF THE NORTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Ingestion Rate IR(mg/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contaminated FI	Body Weight BW(kg)	Averaging Time AT(d/yrxyr)	Chronic Daily Intake CDI(mg/kg-day)
Carcinogenic Exposure										
Pentachlorophenol	6.19E+01	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	3.61E-05
Dioxins/Furans (TEFs)	1.63E-02	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	9.50E-09
2,4,6-Trichlorophenol	7.21E+00	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	4.21E-06
Benzo(a)pyrene (TEFs)	8.13E-01	120	350	30	1.0E-06	1.00E+00	7.0E-01	59	25550	4.74E-07
Arsenic	1.47E+02	120	350	30	1.0E-06	8.00E-01	7.0E-01	59	25550	6.87E-05
Noncarcinogenic Exposure										
Pentachlorophenol	6.19E+01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	3.50E-04
Dioxins/Furans (TEFs)	1.63E-02	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	9.21E-08
2,4,6-Trichlorophenol	7.21E+00	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	4.08E-05
4-chloro-3-methylphenol	6.61E+00	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	3.73E-05
2-methyl-4,6-dinitrophenol	1.48E+01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	8.34E-05
Anthracene	2.25E-01	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	1.27E-06
Arsenic	1.47E+02	160	350	10	1.0E-06	8.00E-01	7.0E-01	19	3650	6.66E-04
Cadmium	1.86E+00	160	350	10	1.0E-06	1.00E+00	7.0E-01	19	3650	1.05E-05

CDI(mg/kg-day)=Cs x IR x EF x ED x CF x FI x ABS/ BW x AT

NA = Not Applicable

TABLE 5-28

ESTIMATED CHRONIC DAILY INTAKES FOR DERMAL CONTACT WITH SOIL FOR FUTURE ON-SITE RESIDENTS OF THE NORTHERN AREA

Chemical	Chemical Concentration Cs(mg/kg)	Surface Area SA(cm^2/d)	Exposure Frequency EF(d/yr)	Exposure Duration ED(yrs)	Conversion Factor CF(kg/mg)	Absorption Factor ABS	Fraction Contam. FI	Body Weight BW(kg	Averaging Time AT(d/yrxyr)	Adherance Factor AF (mg/cm^2)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure											
Pentachlorophenol	6.19E+01	3476	350	30	1.0E-06	0.10	7.0E-01	59	25550	1.45	1.52E-04
Dioxins/Furans (TEFs)	1.63E-02	3476	350	30	1.0E-06	0.01	7.0E-01	59	25550	1.45	4.01E-09
2,4,6-Trichlorophenol	7.21E+00	3476	350	30	1.0E-06	0.10	7.0E-01	59	25550	1.45	1.77E-05
Arsenic	1.47E+02	3476	350	30	1.0E-06	0.01	7.0E-01	59	25550	1.45	3.62E-05
Noncarcinogenic Exposure											
Pentachlorophenol	6.19E+01	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	1.33E-03
Dioxins/Furans (TEFs)	1.63E-02	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	3.50E-08
2,4,6-Trichlorophenol	7.21E+00	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	1.55E-04
4-chloro-3-methylphenol	6.61E+00	4187	350	10	1.0E-06	0.10	7.0E-01	19	3650	1.45	1.42E-04
Arsenic	1.47E+02	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	3.16E-04
Cadmium	1.86E+00	4187	350	10	1.0E-06	0.01	7.0E-01	19	3650	1.45	3.99E-06

CDI(mg/kg-day) = Cs x SA x AF x AbF x EF x ED x CF x FI/BW/AT

Northern Area

Intakes from ingestion of homegrown produce are slightly higher in this area relative to the southern area, except for PCP. There were fewer high detections of PCP in the northern area than in the southern area. PCP does, however, result in the second highest intake for the northern area, 1.5×10^{-3} mg/kg-day for cancer and the highest noncancer CDIs of 3.8×10^{-1}. Intakes from other chemicals for cancer CDIs ranged from 8.9×10^{-8} for dioxins/furans to 9.3×10^{-3} for 2,4,6-trichlorophenol.

Chronic daily intakes for produce grown in the northern area are provided in Tables 5-31 and 5-32. Calculations for estimation of chemical concentrations in plants are provided in Appendix C.

5.6 MAJOR UNCERTAINTIES ASSOCIATED WITH EXPOSURE ASSESSMENT

Quantitative evaluation of chemical exposures for a risk assessment may be the largest single source of uncertainty in the risk assessment. The procedures and assumptions used in this exposure assessment were derived from a combination of EPA guidance, site-specific information, and professional judgment, and are subject to various amounts of uncertainty depending upon the type of assumption or estimate considered.

Uncertainties from different sources may be compounded in an exposure assessment. For example, if a chronic daily intake (CDI) for a chemical measured in the environment is evaluated to determine whether there is a potential health hazard, the uncertainties in the concentration measurements and exposure assumptions will be expressed in the result. To ensure that human health is adequately protected, the exposure assessment incorporates conservative (likely to overestimate risk) estimates and approaches. The aim of the assessment is to estimate exposure well above the average, but still within the range of possible exposures. Therefore, actual exposures posed by a site are unlikely to be higher, but may be lower than those predicted in the assessment. Several of the key exposure assumptions which illustrate this approach are discussed below and are also presented in Table 5-33.

TABLE 5-29

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS FROM INGESTION OF HOME-GROWN VEGETABLES AT THE SOUTHERN RESIDENTIAL AREA

I. Leafy Vegetables

Chemical	Uptake Factor	Contam. Conc. in Lettuce Cs (mg/kg)	Ingest. Rate IR (g/day)	Freq. of Exp. EF (days/yr)	Exp. Duration ED (years)	Body Weight BW (kg)	Avg. Time AT (days)	Conversion Factor CF (mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Dioxins/Furans (TEFs)	0.013	1.7E-06	144	52	30	59	25550	1.00E-03	2.53E-10
2,4,6-Trichlorophenol	3	4.5E+00	144	52	30	59	25550	1.00E-03	6.71E-04
Benzo(a)pyrene(TEFs)	0.05	1.5E-03	144	52	30	59	25550	1.00E-03	2.24E-07
Arsenic	0.006	2.5E-01	144	52	30	59	25550	1.00E-03	3.73E-05
Noncarcinogenic Exposure									
Dioxins/Furans(TEFs)	0.013	1.7E-06	144	52	10	19	3650	1.00E-03	1.84E-09
2,4,6-Trichlorophenol	3	4.5E+00	144	52	10	19	3650	1.00E-03	4.86E-03
Anthracene	0.05	2.6E-03	144	52	10	19	3650	1.00E-03	2.81E-06
Arsenic	0.006	2.5E-01	144	52	10	19	3650	1.00E-03	2.70E-04
Cadimum	0.55	4.3E-01	144	52	10	19	3650	1.00E-03	4.64E-04

CDI (mg/kg-day) = Cs x IR x EF x ED x CF/BW/AT

TABLE 5-29 (CONT.)

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS
FROM INGESTION OF HOME-GROWN VEGETABLES
AT THE SOUTHERN RESIDENTIAL AREA

III. Vine Crop

Chemical	Uptake Factor	Contam. Conc. in Tomato Cs(mg/kg)	Ingest. Rate IR(g/day)	Freq. of Exp. EF(days/yr)	Exp. Duration ED(years)	Body Weight BW(kg)	Avg. Time AT(days)	Conversion Factor CF(mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Dioxins/Furans (TEFs)	0.013	1.7E-06	151	52	30	59	25550	1.00E-03	2.66E-10
2,4,6-Trichlorophenol	3	4.5E+00	151	52	30	59	25550	1.00E-03	7.03E-04
Benzo(a)pyrene(TEFs)	0.05	1.5E-03	151	52	30	59	25550	1.00E-03	2.34E-07
Arsenic	0.006	2.5E-01	151	52	30	59	25550	1.00E-03	3.91E-05
Noncarcinogenic Exposure									
Dioxins/Furans(TEFs)	0.013	1.7E-06	151	52	10	19	3650	1.00E-03	1.92E-09
2,4,6-Trichlorophenol	3	4.5E+00	151	52	10	19	3650	1.00E-03	5.10E-03
Anthracene	0.05	2.6E-03	151	52	10	19	3650	1.00E-03	2.94E-06
Arsenic	0.006	2.5E-01	151	52	10	19	3650	1.00E-03	2.83E-04
Cadimum	0.15	1.2E-01	151	52	10	19	3650	1.00E-03	1.36E-04

CDI (mg/kg-day) = Cs x IR x EF x ED x CF/BW/AT

TABLE 5-29(CONT.)

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS
FROM INGESTION OF HOME-GROWN VEGETABLES
AT THE SOUTHERN RESIDENTIAL AREA

II. Root Crops

Chemical	Uptake Factor	Contam. Conc. in Carrot Cs (mg/kg)	Ingest. Rate IR (g/day)	Freq. of Exp. EF (days/yr)	Exp. Duration ED (years)	Body Weight BW(kg)	Avg. Time AT(days)	Conversion Factor CF (mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Pentachlorophenol(a)	3	9.6E+02	114	52	1	59	25550	1.00E-03	3.78E-03
Pentachlorophenol		4.8E+02	114	52	1	59	25550	1.00E-03	1.89E-03
Pentachlorophenol		2.4E+02	114	52	1	59	25550	1.00E-03	9.44E-04
Pentachlorophenol		1.2E+02	114	52	1	59	25550	1.00E-03	4.72E-04
Pentachlorophenol		6.0E+01	114	52	1	59	25550	1.00E-03	2.36E-04
Pentachlorophenol		3.0E+01	114	52	1	59	25550	1.00E-03	1.18E-04
Pentachlorophenol(SUM)					6				7.43E-03
Dioxins/Furans (TEFs)	0.013	1.7E-06	114	52	30	59	25550	1.00E-03	2.01E-10
2,4,6-Trichlorophenol	3	4.5E+00	114	52	30	59	25550	1.00E-03	5.31E-04
Benzo(a)pyrene(TEFs)	0.05	1.5E-03	114	52	30	59	25550	1.00E-03	1.77E-07
Arsenic	0.04	1.6E+00	114	52	30	59	25550	1.00E-03	1.89E-04
Noncarcinogenic Exposure									
Pentachlorophenol	3	9.6E+02	114	52	1	19	365	1.00E-03	8.21E-01
Pentachlorophenol		4.8E+02	114	52	1	19	365	1.00E-03	4.10E-01
Pentachlorophenol		2.4E+02	114	52	1	19	365	1.00E-03	2.05E-01
Pentachlorophenol		1.2E+02	114	52	1	19	365	1.00E-03	1.03E-01
Pentachlorophenol		6.0E+01	114	52	1	19	365	1.00E-03	5.13E-02
Pentachlorophenol		3.0E+01	114	52	1	19	365	1.00E-03	2.56E-02
Pentachlorophenol(SUM)					6				1.62E+00
Dioxins/Furans(TEFs)	0.013	1.7E-06	114	52	10	19	3650	1.00E-03	1.45E-09
2,4,6-Trichlorophenol	3	4.5E+00	114	52	10	19	3650	1.00E-03	3.85E-03
Anthracene	0.05	2.6E-03	114	52	10	19	3650	1.00E-03	2.22E-06
Arsenic	0.04	1.6E+00	114	52	10	19	3650	1.00E-03	1.37E-03
Cadimum	0.15	1.2E-01	114	52	10	19	3650	1.00E-03	1.03E-04

CDI (mg/kg-day) = Cs x IR x EF x ED x CF/BW/AT

(a) See Appendix C for explanation of exposure concentration

TABLE 5-30

SUMMARY OF ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS FROM INGESTION OF HOME-GROWN VEGETABLES AT THE SOUTHERN RESIDENTIAL AREA

Chemical	Leafy Vegetable CDI mg/kg-day	Vine Crops CDI mg/kg-day	Root Crops CDI mg/kg/day	Total Vegetable Pathway CDI (mg/kg-day)
Carcinogenic Exposure				
Pentachlorophenol	NA	NA	7.43E-03	7.43E-03
Dioxins/Furans (TEFs)	2.53E-10	2.66E-10	2.01E-10	7.20E-10
2,4,6-Trichlorophenol	6.71E-04	7.03E-04	5.31E-04	1.90E-03
Benzo(a)pyrene(TEFs)	2.24E-07	2.34E-07	1.77E-07	6.35E-07
Arsenic	3.73E-05	3.91E-05	1.89E-04	2.65E-04
Noncarcinogenic Exposure				
Pentachlorophenol	NA	NA	1.62E+00	1.62E+00
Dioxins/Furans(TEFs)	1.84E-09	1.92E-09	1.45E-09	5.21E-09
2,4,6-Trichlorophenol	4.86E-03	5.10E-03	3.85E-03	1.38E-02
Anthracene	2.81E-06	2.94E-06	2.22E-06	7.97E-06
Arsenic	2.70E-04	2.83E-04	1.37E-03	1.92E-03
Cadimum	4.64E-04	1.36E-04	1.03E-04	7.03E-04

NA=Not Applicable

TABLE 5-31

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS FROM INGESTION OF HOME-GROWN VEGETABLES AT THE NORTHERN RESIDENTIAL AREA

I. Leafy Vegetables

Chemical	Uptake Factor	Contam. Conc. in Lettuce Cs (mg/kg)	Ingest. Rate IR (g/day)	Freq. of Exp. EF (days/yr)	Exp. Duration ED (years)	Body Weight BW (kg)	Avg. Time AT (days)	Conversion Factor CF (mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Dioxins/Furans (TEFs)	0.013	2.1E-04	144	52	30	59	25550	1.00E-03	3.13E-08
2,4,6-Trichlorophenol	3	2.2E+01	144	52	30	59	25550	1.00E-03	3.28E-03
Benzo(a)pyrene(TEFs)	0.05	4.1E-02	144	52	30	59	25550	1.00E-03	6.11E-06
Arsenic	0.008	8.8E-01	144	52	30	59	25550	1.00E-03	1.31E-04
Noncarcinogenic Exposure									
Dioxins/Furans(TEFs)	0.013	2.1E-04	144	52	10	19	3650	1.00E-03	2.27E-07
2,4,6-Trichlorophenol	3	2.2E+01	144	52	10	19	3650	1.00E-03	2.38E-02
Anthracene	0.05	1.1E-02	144	52	10	19	3650	1.00E-03	1.19E-05
Arsenic	0.008	8.8E-01	144	52	10	19	3650	1.00E-03	9.50E-04
Cadimum	0.55	1.0E+00	144	52	10	19	3650	1.00E-03	1.08E-03

CDI (mg/kg-day) = Cs x IR x EF x ED x CF/BW/AT

TABLE 5-31 (CONT.)

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS
FROM INGESTION OF HOME-GROWN VEGETABLES
AT THE NORTHERN RESIDENTIAL AREA

III. Vine Crop

Chemical	Uptake Factor	Contam. Conc. in Tomato Cs(mg/kg)	Ingest. Rate IR(g/day)	Freq. of Exp. EF(days/yr)	Exp. Duration ED(years)	Body Weight BW(kg)	Avg. Time AT(days)	Conversion Factor CF(mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Dioxins/Furans (TEFs)	0.013	2.1E-04	151	52	30	59	25550	1.00E-03	3.28E-08
2,4,6-Trichlorophenol	3	2.2E+01	151	52	30	59	25550	1.00E-03	3.44E-03
Benzo(a)pyrene(TEFs)	0.05	4.1E-02	151	52	30	59	25550	1.00E-03	6.41E-06
Arsenic	0.006	8.8E-01	151	52	30	59	25550	1.00E-03	1.38E-04
Noncarcinogenic Exposure									
Dioxins/Furans(TEFs)	0.013	2.1E-04	151	52	10	19	3650	1.00E-03	2.38E-07
2,4,6-Trichlorophenol	3	2.2E+01	151	52	10	19	3650	1.00E-03	2.49E-02
Anthracene	0.05	1.1E-02	151	52	10	19	3650	1.00E-03	1.25E-05
Arsenic	0.006	8.8E-01	151	52	10	19	3650	1.00E-03	9.96E-04
Cadimum	0.15	2.8E-01	151	52	10	19	3650	1.00E-03	3.17E-04

CDI (mg/kg-day)= Cs x IR x EF x ED x CF/BW/AT

TABLE 5-31(CONT.)

ESTIMATED CHRONIC DAILY INTAKES FOR FUTURE RESIDENTS
FROM INGESTION OF HOME-GROWN VEGETABLES
AT THE NORTHERN RESIDENTIAL AREA

II. Root Crops

Chemical	Uptake Factor	Contam. Conc. in Carrot Cs (mg/kg)	Ingest. Rate IR (g/day)	Freq. of Exp. EF (days/yr)	Exp. Duration ED (years)	Body Weight BW(kg)	Avg. Time AT(days)	Conversion Factor CF (mg/ug)	Chronic Daily Intake (mg/kg-day)
Carcinogenic Exposure									
Pentachlorophenol(a)	3	1.9E+02	114	52	1	59	25550	1.00E-03	7.47E-04
Pentachlorophenol		9.30E+01	114	52	1	59	25550	1.00E-03	3.66E-04
Pentachlorophenol		4.60E+01	114	52	1	59	25550	1.00E-03	1.81E-04
Pentachlorophenol		2.30E+01	114	52	1	59	25550	1.00E-03	9.04E-05
Pentachlorophenol		1.20E+01	114	52	1	59	25550	1.00E-03	4.72E-05
Pentachlorophenol		5.80E+00	114	52	1	59	25550	1.00E-03	2.28E-05
Pentachlorophenol(SUM)					6				1.45E-03
Dioxins/Furans (TEFs)	0.013	2.1E-04	114	52	30	59	25550	1.00E-03	2.48E-08
2,4,6-Trichlorophenol	3	2.2E+01	114	52	30	59	25550	1.00E-03	2.60E-03
Benzo(a)pyrene(TEFs)	0.05	4.1E-02	114	52	30	59	25550	1.00E-03	4.84E-06
Arsenic	0.04	5.9E+00	114	52	30	59	25550	1.00E-03	6.96E-04
Noncarcinogenic Exposure									
Pentachlorophenol	3	1.9E+02	114	52	1	19	365	1.00E-03	1.62E-01
Pentachlorophenol		9.30E+01	114	52	1	19	365	1.00E-03	7.95E-02
Pentachlorophenol		4.60E+01	114	52	1	19	365	1.00E-03	3.93E-02
Pentachlorophenol		2.30E+01	114	52	1	19	365	1.00E-03	1.97E-02
Pentachlorophenol		1.20E+01	114	52	1	19	365	1.00E-03	1.03E-02
Pentachlorophenol		5.80E+00	114	52	1	19	365	1.00E-03	4.96E-03
Pentachlorophenol(SUM)					6				3.16E-01
Dioxins/Furans(TEFs)	0.013	2.1E-04	114	52	10	19	3650	1.00E-03	1.80E-07
2,4,6-Trichlorophenol	3	2.2E+01	114	52	10	19	3650	1.00E-03	1.88E-02
Anthracene	0.05	1.1E-02	114	52	10	19	3650	1.00E-03	9.40E-06
Arsenic	0.04	5.9E+00	114	52	10	19	3650	1.00E-03	5.04E-03
Cadimum	0.15	2.8E-01	114	52	10	19	3650	1.00E-03	2.39E-04

CDI (mg/kg-day)= Cs x IR x EF x ED x CF/BW/AT

(a) See Appendix C for explanation of exposure concentration

TABLE 5-32

SUMMARY OF ESTIMATED CHRONIC DAILY INTAKES
FOR FUTURE RESIDENTS FROM INGESTION OF HOME-GROWN VEGETABLES
AT THE NORTHERN RESIDENTIAL AREA

Chemical	Leafy Vegetable CDI mg/kg-day	Vine Crops CDI mg/kg-day	Root Crops CDI mg/kg/day	Total Vegetable Pathway CDI (mg/kg-day)
Carcinogenic Exposure				
Pentachlorophenol	NA	NA	1.45E-03	1.45E-03
Dioxins/Furans (TEFs)	3.13E-08	3.28E-08	2.48E-08	8.89E-08
2,4,6-Trichlorophenol	3.28E-03	3.44E-03	2.60E-03	9.31E-03
Benzo(a)pyrene(TEFs)	6.11E-06	6.41E-06	4.84E-06	1.74E-05
Arsenic	1.31E-04	1.38E-04	6.96E-04	9.65E-04
Noncarcinogenic Exposure				
Pentachlorophenol	NA	NA	3.16E-01	3.16E-01
Dioxins/Furans(TEFs)	2.27E-07	2.38E-07	1.80E-07	6.44E-07
2,4,6-Trichlorophenol	2.38E-02	2.49E-02	1.88E-02	6.75E-02
Anthracene	1.19E-05	1.25E-05	9.40E-06	3.37E-05
Arsenic	9.50E-04	9.96E-04	5.04E-03	6.99E-03
Cadimum	1.08E-03	3.17E-04	2.39E-04	1.64E-03

NA=Not Applicable

5.6.1 ADEQUACY OF CHEMICAL DATA BASE

As discussed in Section 4.5.2, data for PCP in soils are adequate for estimating exposure point concentrations, and uncertainties for such values are considered low. Actual exposure points are probably within a factor of 10.

For other soil COCs, data are more limited and confidence in exposure point concentrations is decreased. Given the high variability seen in the PCP data, it is considered that exposure point concentrations may for some COCs be off by a factor exceeding 10. Uncertainty is thus rated moderate in Table 5-33.

Data for groundwater is extensive for all COCs and some confidence can be placed in the exposure point concentrations for this medium. Uncertainty is rated low. It should be noted, however, that variability in these data is high and actual exposures would be highly dependent on placement of any future well.

Data for sediments and surface water are very limited and exposures of trespassers to these media must be considered approximate. Accurate estimation of exposure point concentrations would require a more detailed knowledge of the extent of contamination in the stream, and the likely use of this area by trespassers. However, it is recognized that site characterization data are often those available for risk assessment. Maximum possible use of these data is made in this BRA.

5.6.2 EXPOSURE PATHWAYS AND RECEPTORS

Several exposure pathways are omitted from the quantitative analysis. These include ingestion of surface water used as a residential drinking supply, watering of livestock with surface water and subsequent ingestion of meat and dairy products and inhalation of soil and dust containing resuspended chemicals. Lack of a quantitative treatment for any of these pathways could lead to an underestimation of site-related risks.

Such underestimation is, however, expected to be small — generally much less than an order of magnitude. Silver Bow Creek is too small and too contaminated with metals from upstream sources

TABLE 5-33

EXPOSURE ASSUMPTIONS AND POTENTIAL EFFECT ON EXPOSURE ASSESSMENT

Exposure Assumption	Potential Magnitude for Over-Estimation of Exposure	Potential Magnitude for Under-Estimation of Exposure	Potential Magnitude for Over- or Under-Estimation of Exposure
Adequacy of Chemical Data Base			
Estimation of exposure point concentrations for PCP.			Low
Estimates of exposure point concentrations for other CDLs.			Low to Moderate
Exposure Pathway Analysis			
No quantitive evaluation of beef and dairy, surface water ingestion and inhalation pathways.		Low	
Exposure Parameters			
The use of reasonable maximum exposure (RME) scenarios for receptor populations	Low		
The assumptions regarding body weight, period exposed, population characteristics, and lifestyle may not be representative for any actual exposure situation.			Low
Exposure to contaminants remains constant over exposure period.	Moderate		
Concentration of contaminants remains constant over exposure period.	Moderate		
Fraction contaminated for intake soil is assumed to be 70 percent.	Low		
Absorption factors for chemicals not based on measured absorption from soil matrices	Low to Moderate		
Exposure frequency (24 hours/day for 365 days/yr)	Low to Moderate		
Exposure duration	Low		
Soil Ingestion Parameters			
Ingestion Rates: Child (160 mg/day) Adult (100 mg/day) On-Site Worker (100 mg/day)			 Low Low Low

TABLE 5-33 (Cont.)

EXPOSURE ASSUMPTIONS AND POTENTIAL EFFECT ON EXPOSURE ASSESSMENT

Exposure Assumption	Potential Magnitude for Over-Estimation of Exposure	Potential Magnitude for Under-Estimation of Exposure	Potential Magnitude for Over- or Under-Estimation of Exposure
Soil Ingestion Parameters (Cont.)			
Assumption that soil ingestion rate includes ingestion of household dust.			Low
Produce Ingestion Parameters			
Ingestion Rates: Adult or child Vine Crops (151 g/day) Leafy Crops (144 g/day) Root Crops (114 g/day)			Low
Use of plant uptake factors			Low to High
Dermal Exposure to Soil Parameters			
Use of absorption factors			Low to High
Soil adherence factor (1.45 mg/cm^2)			Low

Key:

Low = uncertainty is estimated to be less than one order of magnitude.
Moderate = uncertainty is estimated to be between one and two orders of magnitude.
High = uncertainty is estimated to be greater than two orders of magnitude.

to be used as a source of drinking water. Further, Silver Bow Creek near the Montana Pole site is not likely, due to current and likely future land use, to be used for watering cattle or other livestock. Neither pathway is likely to be of any significance for exposure now or in the future.

Resuspension of contaminated soil and dust is also not expected to contribute significantly to risks, based on the screening analysis in Section 5.2.1.1. Ambient dust levels would have to be unrealistically high just to reach chronic exposure levels based on a target risk of 1×10^{-6} for increased cancer incidence. Since risks due to inhalation of contaminants are unlikely to be higher than this target, this pathway is unlikely to contribute significantly to site-related risks.

5.6.3 GENERAL EXPOSURE ASSUMPTIONS

Exposure parameters were chosen in a fashion intended to err on the side of protectiveness for human health. This was done intentionally to comply with current guidance (EPA 1989a). Parameters such as exposure frequencies, exposure durations and intake or contact rates were chosen as upper range values, perhaps in the range of 90th or 95th percentiles. As a result, exposure estimates are expected to also be upper range values. Use of such parameters are necessary to produce estimates which meet the definition of RME. Overall uncertainty in exposure assumptions is expected to be less than an order of magnitude. This is based on experience within CDM and elsewhere which indicates that estimates of 95th percentile exposures using quantitative uncertainty techniques seldom are more than an order of magnitude different than deterministic estimates such as those used in this report. In many instances, such values differ by less than a factor of two or three.

A greater source of uncertainty may be the assumption that exposure to chemicals remains constant over the exposure period. Because the site is no longer active, chemical concentrations may gradually decrease over time. Depending on factors such as wind and water erosion, degradation (for organic chemicals), uptake into plants and animals and subsequent transport away from the site, leaching into the subsurface and groundwater, and discharge of groundwater to Silver Bow Creek, there could be substantial redistribution of exposures to different media. It is difficult to determine how exposures might change with time, though in the long term an overall decrease in exposures is expected.

There is some uncertainty associated with the assumption of hypothetical residential land use. The assumption, however, is based on the State's position on the likelihood of this land use. The State's position is that current zoning is a poor basis for assessing future land use since it can be readily changed by local governments. The flood plain designation is also subject to change due to factors such as flood control measures. It should also be noted that the lack of population growth in the Butte area does not imply that all new development will cease. The State concludes that chances for residential development at the Site are not demonstrably small enough to be ignored. Thus, future on-site exposures are assessed in this analysis.

Relative absorption factors of one (default) were used throughout the assessment for ingestion exposures (with the exception of 0.8 used for arsenic ingested in soil), due to lack of information on relative absorption of chemicals from soil and produce compared to absorption of chemicals in studies used to generate reference doses, reference concentrations, and slope factors. Where absorption of a material is less in the exposure setting than in the experimental study, exposures may be overestimated. Although the converse may also be true, in general experimental studies are done with forms of chemicals and exposure protocols that maximize chemical absorption. Thus, lack of specific absorption factors is thought to lead to overestimation of exposures. However, overestimation is likely to be less than an order of magnitude based on CDM's experience at other sites. For example, inorganic arsenic is expected to be almost completely absorbed in the gastrointestinal tract when in soluble form. The slope factor for arsenic is based on exposure to such a form in a large Taiwanese population (see Section 5.3.2 for a description of this study). Recent studies in rabbits suggest that arsenic in insoluble form (from mining wastes) could be significantly less available for absorption, but still nearly 28 percent of an administered dose is absorbed (i.e., 38 percent of the absorption used in this baseline RA) (Johnson et al. 1991). Thus, for arsenic, the magnitude of any error due to differences in absorption would be about a factor of three.

[It should be noted that rabbits may not be the most appropriate animal model for bioavailability studies, and that errors, if any, may be less than that predicted by more appropriate models (Weis and LaVelle 1991)].

5.6.4 SOIL INGESTION PARAMETERS

Soil ingestion rates were chosen to represent an upper range value for the different age groups. As such, they provide for an estimate of exposure in the upper range of possible exposures on the site. Keeping in mind that this upper range estimate is the goal of the risk assessment, these values are appropriate. Still, there is uncertainty in each value, especially for the older age groups where data are almost totally lacking. Thus, use of these ingestion rates may either over- or underestimate reasonable maximum exposures. It seems unlikely that these estimates would be off by more than an order of magnitude (for instance, this would require an on-site worker to consume one gram of soil/day on the high side, or 10 mg/day on the low side). The potential uncertainty is thus estimated as low.

5.6.5 PRODUCE INGESTION PARAMETERS

Produce ingestion rates are selected as upper range values for adults. These may overestimate upper range consumption rates for children. However, it seems reasonable that homegrown produce might be emphasized for those few meals when such produce is eaten. Overall, uncertainty is expected to be low.

Information on plant uptake factors for Montana Pole site COCs is limited. Potentially, a large degree of uncertainty may be associated with these factors. Plant uptake is detailed in Appendix C and uncertainty is further discussed in Section 7.6.12.

The choice of 6 years is different from the 10 years used to assess chronic exposure (noncarcinogenic effects) in other pathways. This conflict arises because, as discussed above, loss of PCP from garden soil might be much greater than assumed for other soils, and exposure is expected to become insignificant after longer time periods. Whether 6 years is sufficient time for chronic toxicity to develop from exposure to PCP cannot be assessed from current data. It is possible that the shorter exposure duration results in an overestimate of potential exposure.

5.6.6 GROUNDWATER AND SURFACE WATER INGESTION PARAMETERS

Drinking water ingestion rates for residential scenarios are well established. It is unlikely that the magnitude of any uncertainty is greater than a factor of 10 and is probably much lower. Surface water ingestion rates are, however, much less reliable, especially for the current situation where the creek is not deep enough for swimming. It is possible that the ingestion rate of 50 ml/hr overestimates actual upper range values for the Montana Pole site situation, but the degree of uncertainty is difficult to determine. A low to moderate rating is considered reasonable. An error of more than 100 would mean, as a lower bound, an intake rate of less than 0.5 ml/hr (about 1/8 of a teaspoon).

5.6.7 DERMAL EXPOSURE PARAMETERS

Relative absorption factors were used for estimating exposures from dermal absorption. Uncertainty is great for such factors due to lack of data on absorption from different soils, and for many chemicals. Absorption factors used may either under or overestimate actual exposures. The magnitude of this uncertainty is rated low to moderate. It should be noted that dermal absorption is strongly affected by the organic content of soil and could range from the value given to essentially zero depending on soil conditions.

The soil adherence factor is thought to be a reasonable estimate for soil loading, but considerable controversy surrounds its choice. Factors as low as 0.6 mg/cm^2 and as high as 1.4 mg/cm^2 have been considered. For kaolin clay, a factor of 2.77 mg/cm$_2$ may be appropriate (EPA 1989i). This is a fairly tight range of estimates and suggests that the value chosen is within a factor of two or three of actual possible soil loading.

Surface areas chosen for this BRA are best estimates (50th percentiles) based on national norms. Little uncertainty is associated with these estimates per se. Choice of exposed body parts is, however, based on professional judgment and is potentially subject to greater uncertainty. The magnitude of this uncertainty, which could result in either under or over estimation of risk, is judged to be low.

5.7 SUMMARY

Intakes of COCs are highest for the ingestion of groundwater by future residents. For a few compounds, notably PCP, exposures from homegrown produce could also be significant for these receptors. Other pathways do not contribute significantly relative to groundwater to overall exposures.

Intakes are much less for on-site trespassers or future workers. For both groups, dermal exposures contribute a significant portion of total chronic daily intake.

6 Section Six

6.0 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to examine the potential for each chemical to cause adverse effects in exposed individuals and to provide an estimate of the dose-response relationship between the extent of exposure to a particular contaminant and adverse effects. Adverse effects include both noncarcinogenic and carcinogenic health effects in humans.

Sources of toxicity information include EPA's Integrated Risk Information System (IRIS), Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles, Health Effects Assessment Summary Tables (HEAST), and EPA criteria documents. The hierarchy of toxicological information sources used in this risk assessment is based on EPA guidance (EPA 1989a).

For each chemical, a brief toxicity profile is included, based on information in the documents cited above and any more recent information from the literature. These profiles outline major adverse effects, describe important toxicokinetic findings (absorption into, distribution in, metabolism by, and excretion from the body), discuss uncertainties and important data gaps, and summarize important studies used in the derivation of critical toxicity criteria.

Criteria for carcinogens are provided as slope factors in units of risk per milligram of chemical exposure per kilogram body weight per day. These factors are based on the assumption that no threshold for carcinogenic effects exists and any dose, no matter how small, is associated with some finite cancer risk. On the other hand, criteria for noncarcinogens, or for significant noncarcinogenic effects caused by carcinogens, are provided as reference doses in units of milligrams of chemical exposure per kilogram body weight per day. Such doses may be interpreted as thresholds, below which adverse effects are not expected, even in the most sensitive populations.

Quantitative dose-response information, in the form of critical toxicity criteria, for each chemical is presented in Section 5.1. Uncertainties associated with toxicity criteria estimates are discussed in Section 5.2. Individual chemical profiles in support of toxicity criteria and uncertainty analysis are presented in Section 5.3.

6.1 TOXICITY REFERENCE VALUES

6.1.1 CARCINOGENS

Evidence of Carcinogenicity

The EPA has developed a system for stratifying weight of evidence. This classification system characterizes the overall weight of evidence of carcinogenicity based on the availability of human, animal, and other supportive data (EPA 1992b). Three major factors are considered in characterizing the overall weight of evidence of carcinogenicity:

- The quality of evidence from human studies
- The quality of evidence from animal studies, which are combined into a characterization of the overall weight of evidence for human carcinogenicity
- Other supportive data that are assessed to determine whether the overall weight of evidence should be modified.

The EPA classification system for the characterization of the overall weight of carcinogenicity has the following five categories.

- Group A - Human Carcinogen. This category indicates that there is sufficient evidence from epidemiological studies to support a causal association between an agent and cancer.
- Group B - Probable Human Carcinogen. This category generally indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).
- Group C - Possible Human Carcinogen. This category indicates that there is limited evidence of carcinogenicity in animals in the absence of adequate data on humans.
- Group D - Not Classified. This category indicates that the evidence for carcinogenicity in animals is inadequate.
- Group E - Evidence of Noncarcinogenicity to Humans. This category indicates that there is evidence for noncarcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

Cancer Slope Factors

The EPA Cancer Assessment Group (CAG) (now the Cancer Review and Validation Effort, or CRAVE Committee) has used a variety of specialized models to estimate the upper bound risk of carcinogenesis for more than 50 compounds. Data from animal or epidemiological studies are used to determine slope factors, which are expressed as (milligram of chemical per kilogram of body weight per day)$^{-1}$. The slope factor describes the increase in an individual's risk of developing cancer over a 70-year lifetime per unit of exposure where the unit of exposure is expressed as milligrams of chemical per kilogram of body weight per day.

The term "upper bound" indicates that slope factors are generally calculated using methodology intended to be protective of human health. For example, slope factors are based on the assumption that cancer risks decrease linearly with decreasing dose, and that use of a 95 percent upper confidence limit estimate for the slope is appropriate in most cases to compensate for animal to human extrapolation and other uncertainties. The resulting slope factors are considered to be upper range estimates that are unlikely to underestimate carcinogenic potential in humans.

When the upper-bound cancer slope factor is multiplied by the lifetime average dose of a potential carcinogen, the product is the upper-bound lifetime individual cancer risk associated with exposure at that dose. The calculated risk is thus an estimate of the increased likelihood of cancer over existing levels resulting from exposure to the COC. For example, if the product of the slope factor and the average daily dose is 1×10^{-6}, the predicted upper bound cancer risk for the exposed population is one in one million, or 0.0001 percent. This risk would be in addition to any "background" risk of cancer not related to the chemical exposure. Slope factors for carcinogenic chemicals (except lead) selected as Chemicals of Concern (COCs) for the Montana Pole NPL Site are listed in Table 6-1. The data used to develop each slope factor are found in the corresponding EPA health assessment documents for each chemical and are summarized in the toxicity profiles (Section 6.3).

It should be noted that calculation of risk relies on data derived from the results of human epidemiological studies or chronic animal bioassays. The likelihood that a pollutant is a human carcinogen is a function of the weight of evidence of animal and/or human studies relating to:

TABLE 6-1

TOXICITY VALUES FOR COCs AT THE MONTANA POLE NPL SITE

Chemical of Concern	EPA Carcinogenic Classification/Slope Factor $(mg/kg\text{-}day)^{-1}$		RfD[a] (Chronic) (mg/kg-day)	
	Oral	Inhalation	Oral	Inhalation
2,3,7,8-Tetrachlorodibenzo-p-dioxin	B2/1.5×10^{5}	B2/1.5×10^{5}	NA	NA
Pentachlorophenol	B2/1.2×10^{-1}	ND	3×10^{-2}	ND
4-chloro-3-methyl phenol	ND	ND	ND	ND
2-methyl-4,6 dinitrophenol	ND	ND	ND	ND
2-chlorophenol	ND	ND	5×10^{-3}	ND
2,4-dichlorophenol	ND	ND	3×10^{-3}	ND
2,4-dinitrophenol	ND	ND	2×10^{-3}	ND
2,4-dinitrotoluene	B2/6.8×10^{-1}	ND	ND	ND
2,3,5,6-tetrachlorophenol	ND	ND	3×10^{-2} [b]	ND
2,4,6-trichlorophenol	B2/1.1×10^{-2}	1.1×10^{-2}	ND	ND
PAHs				
Carcinogenic				
Chrysene	B2/7.3×10^{-2}	B2	DI	DI
Benzo(a)anthracene	B2/7.3×10^{-2}	B2	NA	NA
Benzo(a)pyrene	B2/7.3	B2/6.1	NA	NA
Benzo(b)fluoranthene	B2/7.3	B2	NA	NA
Benzo(ghi)perylene	D/7.3×10^{-2}		NA	NA
Benzo(k)fluoranthene	B2/7.3×10^{-2}	B2	NA	NA
Dibenzo(a,h)anthracene	B2/7.3	B2	NA	NA
Indeno(1,2,3-cd)pyrene	B2/7.3×10^{-2}	B2	NA	NA
Noncarcinogenic				
Acenaphthene	NC	NC	6×10^{-2}	ND
Anthracene	NC	NC	3×10^{-1}	ND
Fluoranthene	NC	NC	4×10^{-2}	ND
Fluorene	NC	NC	4×10^{-2}	ND
Naphthalene	NC	NC	4×10^{-2}	ND
Phenanthrene	NC	NC	DI	DI
Pyrene	NC	NC	3×10^{-2}	ND
2-Methyl naphthalene	ND	ND	ND	ND
INORGANICS				
Arsenic	A/1.75	A/1.5×10^{1}	3×10^{-4}	ND
Cadmium	ND	6.3	Water - 5×10^{-4} Food - 1×10^{-3}	ND
Chromium (VI)	ND/ND	A/4.1×10^{1}	5×10^{-3}	ND
Copper	D/NC	NC	1.3 mg/L[d]	ND
Zinc	D/NC	NC	2×10^{-1}	ND
Lead	B2/ND[e]	ND	ND	ND
Manganese	D/NC	NC	1×10^{-1}	1.1×10^{-4}

TABLE 6-1 (Cont.)

TOXICITY VALUES FOR COCs AT THE MONTANA POLE NPL SITE

Notes:

[a] RfD = reference dose
[b] Based on 2,3,4,6-tetrachlorophenol
[c] See Table 6-3 for potency factors used in this assessment
[d] Current Drinking Water Standard
[e] Exposure determined by integrated uptake biokinetic model

key =

ND - Not determined
NC - Noncarcinogenic via this route of exposure
DI - Data inadequate for risk assessment
NA - Not applicable, RfDs are not typically calculated for carcinogens

- Increase in the number of tissues affected by the chemical.
- Increase in the number of animal species, strains, sexes, and number of experiments and doses showing a carcinogenic response.
- Occurrence of clear-cut dose-response relationships as well as a high level of statistical significance of the increased tumor incidence in treated compared to control groups.
- A dose-related shortening of time-to-tumor occurrence or time-to-death with tumor.
- A dose-related increase in the proportions of tumors that are malignant.

Animal studies are usually conducted using relatively high doses in order to observe possible adverse effects. Because humans are expected to be exposed at lower doses, the data are adjusted by using a mathematical model. The data from animal studies are fitted to the linearized multi-stage model and a dose-response curve is obtained. The low-dose slope of the dose-response curve is subjected to various adjustments (e.g., calculation of 95 percent upper confidence limit), and inter-species scaling factors are often applied to derive slope factors for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an individual basis. These models provide rough, but plausible, estimates of the upper limits on lifetime risk. Although the actual risk is unlikely to be higher than the estimated risk, it could be considerably lower.

Cancer slope factors for COCs for the Montana Pole site are presented in Table 6-1.

6.1.2 NONCARCINOGENS

Reference doses (RfDs) are critical toxicity values developed by the EPA for chemicals exhibiting noncarcinogenic effects. RfDs are usually derived from no-observable-adverse-effect levels (NOAELs) taken either from human studies, often involving workplace exposures, or from animal studies, and are adjusted downward using uncertainty or safety factors.

Generally, uncertainty factors are applied to correct for the possibilities that humans are more sensitive than experimental animals, and that there may be sensitive subpopulations of humans (e.g., children, pregnant women, individuals with hay fever or asthma). Depending upon the information available, other factors may also be applied.

The RfD is an estimate of the daily exposure to a chemical that would be without adverse effects even if the exposure occurs continuously over a lifetime. An RfD is probably associated with an uncertainty spanning an order of magnitude or more. RfDs are presented in units of milligram of chemical per kilogram of body weight per day for comparison with rates of intake into the body. Intakes that are less than the RfD are not likely to be of concern. Chronic intakes that are greater than the RfD indicate a possibility for adverse effects, at least in sensitive populations. However, whether such exposures actually produce adverse effects will (depending on the chemical) be a function of a number of factors such as the accuracy of uncertainty factors applied to the NOAEL, the appropriateness of animal models used in studies extrapolated to humans, and the potential for the chemical to cause effects in organs or systems (e.g., reproductive and immune systems) that have not been adequately studied. None of the above are quantifiable, such that it is not possible to discuss the risk of adverse effects in numerical terms. However, it is generally accepted that the protective assumptions made by EPA in deriving RfDs will, in most cases, mean that exposures slightly in excess of the RfD will be associated with a low risk for adverse effects, with the probability of adverse effects increasing with increasing exposure.

The RfDs for COCs are presented in Table 6-1.

6.2 UNCERTAINTIES ASSOCIATED WITH TOXICITY ASSESSMENT

There are many uncertainties associated with the use of toxicology information in health risk assessments that are related to uncertainties intrinsic to toxicology. Chief among these uncertainties are:

- The use of dose-response information high-dose studies to predict adverse health effects at low doses
- Applicability of experimental animal studies to predict effects in humans.

However, these and other uncertainties are intrinsic limitations to the risk assessment process which cannot be resolved quantitatively given the current understanding of toxicology and human health. These uncertainties are addressed in part by consistent application of conservative assumptions regarding the toxic effects of chemicals, such as uncertainty factors for reference doses and upper

bound estimates for cancer slope factors. Such procedures are intended to protect public health and are expected, in many cases, to overstate potential impacts on human health.

An assumption incorporated into this risk assessment is the selection of 2,3,7,8-TCDD equivalents as a surrogate for all dioxins and furans that might be present at the site. These toxicity equivalents are based on limited and somewhat indirect data. Two sets of equivalency factors are still available (EPA 1989h). Especially for the higher molecular weight congeners, such as OCDD, these two sets of TEFs provide significantly different estimates of relative potency. The most recent TEFs project higher risks at sites where the higher molecular weight congeners predominate.

Also, reference concentrations (RfCs) and/or reference doses (RfDs) for some chemicals are not available from established sources and cannot be easily derived from existing data. Thus, several compounds are not included in the quantitative analysis of potential risks of systemic (non-cancer) effects. This could lead to an underestimation of risks, if a chemical is sufficiently toxic and expected to be present in sufficiently high concentrations. Missing values for RfCs and RfDs are discussed in the toxicologic profiles in Section 6.3, and their impact on risk characterization is addressed in Section 7.6.1.

Finally, not all chemicals detected at the Montana Pole site were included in the quantitative assessment. Elimination of these chemicals was based on a toxicity screen as described in Section 4.5.1.1. Based on available data, these chemicals appeared unlikely to contribute significantly to site risks. The database, especially for soils, is limited and it is possible that hot spots for some chemicals were missed in the RI investigation. The likelihood of such hot spots is not considered great, though, since the chemicals eliminated are generally process related, and often occur as contaminants or breakdown products of process chemicals. These chemicals are expected to be present in relatively small concentrations in areas where process wastes were released.

A brief profile of each chemical of concern identified for the Montana Pole site is presented in this section. Critical adverse health effects and the basis for the derivation of the health criteria are discussed for each COC.

6.3 TOXICITY PROFILES

6.3.1 ARSENIC

Arsenic (As) is a naturally occurring metalloid which can be present in a number of different valence states and as a constituent in both inorganic and organic compounds. Elemental arsenic is used in industry as an alloying agent; both inorganic and organic arsenical compounds have been used as pesticides and pharmaceuticals. At the Montana Pole site, arsenic contamination most likely comes from historical mining and ore processing.

Toxicokinetics

Absorption of arsenic from the gastrointestinal tract is dependent on the solubility of the arsenic compound. Soluble forms of both As(III) and As(V) are essentially completely absorbed in laboratory animals (Vahter 1981) and humans (EPA 1984c). Insoluble forms may be essentially nonavailable for absorption in humans as indicated by the lack of increase in urinary excretion of arsenic in human volunteers administered arsenic selenide orally (Mappes 1977).

Following inhalation, adsorption is dependent on particle size; larger particles are quickly cleared from the lungs with little absorption. Smaller particles penetrate into alveolar spaces and may remain there for extended periods, increasing the chances for inhaled arsenic to be absorbed (EPA 1984c). Absorption from the lung may be rapid for soluble arsenic forms, but much slower for more insoluble forms (ATSDR 1989a). Forms of arsenic associated with mining activities are generally less soluble.

Arsenic is efficiently metabolized to methylated forms in the liver in both animals (ATSDR 1989a) and humans (Buchet et al. 1981). Because acute toxicity of these methylated forms is much less than for inorganic arsenic, methylation is considered detoxification. At high arsenic doses, methylation may become saturated (Lovell and Farmer 1985; Buchet et al. 1981). This may result in a "threshold" determined by the ability to metabolize arsenic, where low doses are relatively non-toxic due to conversion to methylated forms, and higher doses are more toxic since greater amounts of inorganic arsenic will be available for distribution to target tissues. This is especially important for carcinogenesis following oral exposure, where small daily intakes could be much less effective in

inducing cancer than higher doses that saturate metabolism. Unfortunately, information insufficient to determine the saturation point in humans (EPA 1988a) and it is not possible at this time to make adjustments to the oral slope factor for low chronic daily intakes.

Arsenic is primarily excreted in the urine in both animals and humans (ATSDR 1989a). This is true for both inorganic and methylated forms. Biliary excretion has been noted to be highly variable in animals, but due to reabsorption in the intestines, does not contribute significantly to overall excretion (Klassen 1974).

Qualitative Description of Health Effects

Toxicological information on arsenic has been reviewed by EPA in its ambient water quality criteria document (EPA 1980b) and health assessment document (EPA 1984c) and, more recently, by EPA's Risk Assessment Forum (EPA 1985c) and ATSDR (1989a). Acute poisoning of humans with arsenic may result in gastrointestinal effects, hemolysis, and neuropathy. Chronic exposure is associated with characteristic toxic effects on the peripheral nervous system and, in children, on the central nervous system. In humans, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular injury frequently follow chronic exposure to arsenic. Arsenic has been found to be embryotoxic, fetotoxic, and teratogenic in several animal species at high doses. One report suggests that children of women working in a Swedish copper smelter had lower birth weights than expected (Nordström et al. 1978). Though arsenic exposure was involved, women were also exposed to a variety of heavy metals and sulfur dioxide. Thus, it is not possible to link fetal effects with arsenic exposure.

Arsenic induces chromosome aberrations and impairs DNA repair but has not been shown to be a point mutagen. Epidemiological studies have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma, while ingestion has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer (Tseng et al. 1968; Chen et al. 1986). Although arsenic's potential as a human carcinogen has long been recognized, reliable induction of cancer in animal models has not yet been achieved. Arsenic exposure has been reported to increase the neurotoxic effects of lead in children as measured by aggressive behavior (Marlowe et al. 1985). Arsenic and aluminum may interact in similar fashion, promoting aggressive behavior (ATSDR 1989a). Arsenic and cigarette smoke are reported to have multiplicable effects on lung

cancer mortality in smelter workers (Pershagen et al. 1983). Arsenic and cadmium together had a greater effect on reduced weight gain in rats than expected from the simple sum of their individual effects (Mahaffey and Fowler 1977).

Quantitative Description of Health Effects

The EPA (1984c) has classified arsenic as a Group A — Human Carcinogen. This category applies to chemical agents for which there is sufficient evidence of carcinogenicity in humans.

Oral Toxicity

To estimate the risks posed by ingestion of arsenic, the EPA (1980b) used data obtained in Taiwan by Tseng et al. (1968). Tseng et al. used the Weibull model to relate skin cancer incidence, age, and level of arsenic exposure via drinking water. Based on a study population of 40,421 individuals that had obtained drinking water from wells contaminated with varying levels of arsenic for 45 years, age-specific cancer prevalence rates were found to be correlated with both local arsenic concentrations and age (duration of exposure). Extrapolation to low dose levels yielded a risk of 4.29×10^{-3} associated with lifetime exposure to 10 μg/liter of arsenic in drinking water (EPA 1984c). In the same area, Chen et al. (1986) reported an association between bladder, lung, and liver tumors and ingestion of arsenic-contaminated drinking water.

A recent study (Astolfi et al. 1981) has shown an association between the ingestion of arsenic in drinking water (at concentrations around 1 ppm) and skin cancer. Epidemiological studies conducted in the United States have not yet shown such an association, but the reported studies were generally too insensitive to have shown such an association if it had existed at the predicted magnitude (EPA 1984c).

One possible complicating factor for risk assessment of ingested arsenic is possible variation in carcinogenic potency according to the chemical form of arsenic. Trivalent inorganic arsenic compounds are generally more toxic than pentavalent inorganic arsenic compounds or organic arsenic compounds (EPA 1980b). However, recent studies have shown that water samples from the area of Taiwan, where Tseng et al.'s (1968) studies were carried out, contain primarily pentavalent inorganic

arsenic and no organic arsenicals (EPA 1984c). The CAG unit risk is therefore applicable to other circumstances in which pentavalent arsenic compounds are ingested.

A second complicating factor is the possibility that inorganic arsenic at low doses is detoxified by methylation in the body. If such detoxification was saturated at doses received by the Taiwanese study group, a slope factor based on the Taiwan study could overstate arsenic risks at lower doses. However, the study on which human detoxification kinetics might be based (Buchet et al. 1981) is weak and subject to a variety of interpretations (EPA 1988b). Thus, until better data become available, the pending EPA oral slope factor seems the best estimate for its cancer potency following arsenic ingestions.

The EPA has developed an oral reference dose based on studies by Tseng et al. (1968) and Tseng (1977). Data in these studies show an increased incidence of blackfoot disease in arsenic exposed individuals in Taiwan. Hyperpigmentation and keratosis of the skin were also reported. Based on average arsenic concentrations in wells used by these individuals, a NOAEL of 0.8 μg/kg-day has been estimated. An uncertainty factor of three was applied to the NOAEL to yield an RfD of 3×10^{-4} (mg/kg-day) (EPA 1992b).

The EPA interim primary drinking water standard for arsenic is 50 μg/liter (EPA 1992b). This value was established as a maximum allowable level for arsenic in drinking water by the U.S. Public Health Service in 1942, and it continues to be used in the current EPA regulations (EPA 1985c). The EPA's Office of Drinking Water is considering maintaining the present maximum contaminant level (MCL) of 50 μg/liter for arsenic in municipal drinking water supplies (EPA 1992b).

Inhalation Toxicity

The health risks posed by airborne arsenic compounds have been reviewed in considerable detail by the EPA (1984c), and studied on the carcinogenicity of arsenic compounds were reviewed by the International Agency for Research on Cancer (IARC) in 1980. Risk assessments for exposure to airborne arsenic were presented by OSHA (1983) and EPA (1984c). The following summary is based on these reviews and risk assessments and on review of the primary literature.

It is well established that inhalation of certain arsenic compounds can cause cancer in humans. Several studies of workers in smelters and plants that manufacture arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma (EPA 1984c).

The EPA (1984c) based its quantitative risk assessment for inhaled arsenic on five studies of three exposed worker populations (Lee-Feldstein 1983; Brown and Chu 1982, 1983a,b; Enterline and Marsh 1980; Ott et al. 1974). All five studies showed excess risks of lung cancer that were related to the intensity and duration of exposure and the duration of follow-up (latency). The estimates of unit risk (unit risk is the risk associated with lifetime exposure to 1 unit (generally 1 (mg/kg-day), 1 mg/L, or 1 $\mu g/m^3$ of a substance) obtained from the five studies were in reasonable good agreement, ranging from 1.2×10^{-3} to 1.36×10^{-2} $(\mu g/m^3)^{-1}$. The EPA omitted the highest value, derived from the study of Ott et al. (1974), which was considered least reliable, and calculated the geometric mean for each of the two remaining populations and then an overall geometric mean to obtain a best estimate of 4.3×10^{-3} $(\mu g/m^3)^{-1}$ for the unit risk.

No reference concentration is available for inorganic arsenic. Extrapolation from the oral value is deemed inappropriate based on the following considerations. First, the relative sensitivity of various tissues to arsenic exposure via oral and inhalation routes is not clear. Certainly, the skin is in the critical target for carcinogenic response following oral exposure, while the lung is the target after inhalation. Since it cannot be determined if the target organ is the same for the two exposures, route-to-route extrapolation is not appropriate. Further, metabolism may influence relative doses by the two routes. Inorganic arsenic is methylated *in vivo* by a saturable process in the liver. Because of first pass effects, the differences in the rate and extent of absorption following exposure by the two routes, the concentrations of inorganic arsenic which reach critical targets may differ. Again, this suggests that route-to-route extrapolation is inappropriate. Lack of RfC requires that inhalation exposures to arsenic be assessed qualitatively for systemic effects.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 0.2 mg/m^3 for arsenic and soluble compounds of arsenic.

Summary of Arsenic Criteria		Source
EPA carcinogenic classification	Group A	EPA 1992c
Oral slope factor (pending)	$1.75 \times 10^{+0}$ (mg/kg-day)$^{-1}$	EPA 1992c
Inhalation slope factor	$1.5 \times 10^{+1}$ (mg/kg-day)$^{-1}$	EPA 1992c
RfD (Oral)	3×10^{-4} (mg/kg-day)	EPA 1992b
Maximum Contaminated Level (MCL)	0.05 mg/L	EPA 1992d
EPA Drinking Water Health Advisories	Not available	EPA 1992d
Ambient Water Quality Criteria (AWQC) (concentration associated with a 10^{-6} excess lifetime cancer risk)		EPA 1992b
Ingestion of water and aquatic organisms	2.2 ng/liter	EPA 1992b
Ingestion of aquatic organisms	17.5 ng/liter	EPA 1992b
Freshwater aquatic life chronic toxicity	0.19 mg/L	EPA 1992b

6.3.2 CHROMIUM

Chromium is a naturally occurring metal present in low concentrations in the earth's crust, primarily in chromite ore. Chromium is used extensively in industry, mainly for plating metals such as stainless and alloy steels and aluminum. It is also used as an additive in cleansing agents, paints, catalysts, leather tanning agents, fungicides, and wood preservatives. The sources of chromium at the Montana Pole site are not known since chromium was not mined or smelted in Butte, and is not known to have been used for wood treating.

No data are available on species of chromium present at the Montana Pole site. For this reason, the conservative assumption is made that all is present in the more toxic Cr(VI) form. Toxicity of Cr(VI) is discussed in this profile.

Toxicokinetics

Absorption of chromium (VI) takes place following exposure by any route. However, except for very large single doses, this absorption does not seem to lead to systemic affects. After inhalation, toxic effects appear to be limited to the upper respiratory tract and lung. Following dermal exposure, only hypersensitivity reactions of the skin have been noted. After oral exposure, not even local effects are seen from doses that produce no acute toxic effects.

This lack of systemic effects may be due, in part, to low absorption rates. For oral exposure, absorption of chromium (VI) might range from about 2 percent (Ogawa 1976) to about 10 percent (Donaldson et al. 1966). Absorption is probably limited by reduction of chromium (VI) to chromium (III) in the low pH of the gastric juice (ATSDR 1989b), although *in vivo* measurements have not been made. Chromium (III) absorption may be as little as one-tenth that for chromium (VI) (Donaldson et al. 1966).

Dermal absorption is implied by experiments using human volunteers in which some chromium (VI) was found in urine following dermal exposure (Samitz and Shrager 1966). In a single animal study, a dermal absorption rate of 0.69 to 0.725 μmol/h/cm^2 was estimated for Na_2CrO_4 for guinea pig skin (Wahlberg and Skog 1965). This flux may be compared to that of water vapor for humans (28 μmol/h/cm^2) (Scheuplein and Blank 1973, anisole 9 μmol/h/cm^2) (all from Barry et al. 1984). All the latter are relatively water soluble organic compounds with molecular weight similar to that of chromate anion (93-122 versus 114), and have flux rates 10 times greater or more.

Metabolism and sequestration of chromium (VI) in the body may also play an important role in limiting systemic toxicity. Chromium (VI) can be rapidly reduced to chromium (III) inside red blood cell and much absorbed chromium (VI) may be "detoxified" by this route (Wiegand et al. 1984; Korallus 1986). It is unlikely that significant oxidation of chromium (III) takes place *in vivo* (Petrilli et al. 1986; Hertel 1986), and chromium (III) appears to be permanently trapped once inside red blood cells (RBCs) (ATSDR 1989b). Moreover, chromium (III) is also effectively reduced in the plasma and can subsequently undergo olation and polymerization to high molecular weight complexes which have little biologic activity (Anderson 1981). If the capacity to reduce chromium (VI) is not exceeded, and chromium (III) polymerization proceeds at a sufficient rate, little active chromium (VI) of chromium (III) will be delivered to potential target organs followed absorption into the bloodstream.

Qualitative Description of Health Effects

Hexavalent chromium (chromium [VI]) compounds are strong oxidizing agents and are severely irritating and corrosive (EPA 1980e; ATSDR 1989b). Chronic inhalation of dust containing chromium (VI) in the form of chromic acid or as soluble salts may cause respiratory irritation,

perforation or ulceration of the nasal septum and decreased spirometric values (Lindberg and Hedenstierna 1983). In addition, several investigators have associated chronic exposure to chromium (VI) dust with emphysema, chronic bronchitis, polyps, chronic inflammation and other respiratory conditions in occupational settings (ATSDR 1989b).

No systematic adverse effects have been reported in humans following chronic oral exposure to chromium (VI) compounds. However, ingestion of large single doses ($\leq$2 g) can cause rental tubular necrosis (Langard and Norseth 1986). Similarly, chronic systemic effects have not been reported in animals even after lifetime oral exposure to chromium (VI) (Mackenzie et al. 1958). Interperitoneal injection of chromium (VI) can, however, cause a variety of effects, including rental tubular necrosis, in animals (EPA 1984e). Thus, the lack of systemic toxicity in humans and animals following chronic oral exposure is not due to the lack of intrinsic toxicity. Rather, as discussed below, it is likely due to the kinetics of absorption and distribution of chromium (VI) following ingestion.

Dermal exposure to chromium (VI) can cause irritation and ulceration when exposures are large. Further, smaller exposures may lead to hypersensitivity reactions. Recent reports in abstract form suggest that 10 percent of sensitized individuals will respond to 10 ppm K_2CrO_4 in a patch test (Mylavarapu and Sun 1991) and that to protect the most sensitive individuals, a clean-up level to 5 $\mu g/cm^2$ on surfaces would have to be achieved (Symms 1991).

Finally, chronic exposure to chromium-bearing dusts via inhalation has been associated with lung cancer in occupationally exposes workers in a number of studies (reviewed in ATSDR 1989b). Unfortunately, exposure data have not been sufficient to clearly establish the form(s) of chromium responsible for the increases in lung cancer (ATSDR 1989b). However, it has been generally accepted that chromium (VI) compounds are likely to be the key etiologic agents. This is consistent with the findings that *in vitro* chromium (VI) compounds can enter cells readily, while chromium (III) is effective at low concentrations in induction of chromosome aberrations and sister chromatic exchanges (SCEs), gene mutation and cell transformation (reviewed in Bianchi and Levis 1985). A few studies have measured increases in chromosome aberrations and SCEs in the peripheral lymphocytes of workers exposed to soluble chromium (VI) compounds (reviewed in ATSDR 1989b).

Chronic oral exposure to chromium (VI) compounds did not cause increased tumor incidence in rats (Mackenzie et al. 1958). This result, combined with the lack of evidence for cancer following oral exposure in humans has led the EPA to the conclusion that chromium (VI) is not carcinogenic via this route.

Quantitative Description of Health Effect

The EPA (1984e) based its quantitative risk assessment for inhaled hexavalent chromium on a study by Mancuso (1975). Mancuso's study showed excess risks of lung cancer in workers exposed to chromates between 1931 and 1937 and followed until 1974. Lung cancer risks increased with duration of exposure and with age. Estimates of cumulative exposure to soluble, insoluble, and total chromium were derived from a single set of industrial hygiene measurements taken in 1949. Smoking habits of the workers were not determined or discussed. For lifetime exposure the "unit risk" was calculated to be 1.2×10^{-2} $(\mu g/m^3)^{-1}$. Expressed in terms of total intake via inhalation, the cancer potency factor was calculated as 41 (mg/kg-day) (EPA 1984e).

Confidence in the EPA's unit risk is attenuated by several factors. Although results of studies of chromium exposure are consistent across locations and investigators and a dose-response relationship has been established, the Mancuso study based its exposure calculations on the assumption that the ratio between chromium (III) and chromium (VI) was 6:1. This was the assumed minimum chromium (VI) content and could underestimate risks. On the other hand, the 1949 hygiene data may have underestimated actual exposures which could lead to overestimation of risk. Finally, the implicit assumption in the study that smoking rates were similar in the worker and general populations may cause an overestimation or risk, since smoking rates are often higher among industrial workers (EPA 1992b).

Based on exposure via inhalation, IARC (1980) classified chromium (VI) as Group A — Human Carcinogen. EPA classified chromium (VI) as Group A — Human Carcinogen (EPA 1984e,f) via the inhalation route. Chromium compounds which are ingested are classified as Group D (EPA 1984g).

Hexavalent chromium's potent carcinogenic effects when inhaled make calculation of subchronic or chronic allowable intakes by inhalation for noncarcinogenic endpoints of toxicity inappropriate (EPA

1984e). For trivalent chromium, a chronic allowable intake (AIC) by inhalation of 0.005 (mg/kg-day) was calculated in the Health Effects Assessment for Trivalent Chromium (EPA 1984f). This number was derived from a TLV. The studies used for derivation of the TLV involved workers concomitantly exposed to other dusts and fumes (ACGIH 1986).

For oral exposure to chromium (VI), a subchronic allowable intake (AIS) of 0.025 (mg/kg-day) was derived in the Health Effects Assessment for Hexavalent Chromium (EPA 1984d). The AIS was based on a one-year study in which rats were exposed to 0-25 mg/L chromium VI as potassium chromate in drinking water. Increased tissue concentrations of chromium, but no adverse health effects were reported at the highest dose (Mackenzie et al. 1958 as cited in EPA 1984d). An oral chronic allowable intake (AIC) of 0.005 (mg/kg-day) was derived from the same study, with application of an additional safety factor of five to adjust for less than lifetime exposure (EPA 1984d). This AIC has been adopted by the EPA as the RfD for soluble chromium (VI) compounds (EPA 1992b).

An RfC for inhalation of chromium (VI) has been established by EPA (1992b), but is currently under review. The value is based on a human study (occupational) that established a $LOAEL_{HEC}$ of 0.7 $\mu g/m^3$ for production of atrophy in the nasal mucosa. An uncertainty factor of 300 was applied to convert the LOAEL to a NOAEL, to protect sensitive subpopulations and to account for less-than-chronic exposures in the worker population studied. The resulting RfC is 2×10^{-6} mg/m^3. An RfD of 6×10^{-7} (mg/kg-day) is generated by multiplying the RfC by the inhalation rate (20 m^3/day) and dividing by body weight (70 kg).

A final Maximum Contaminant Level Goal (MCLG) of 0.10 mg/L was adopted for total chromium, based on an Adjusted Allowable Daily Intake (AADI) of 0.17 mg/L, and with exposure by other routes (0.10 mg/day via the diet and 0 mg/day via air) factored in (EPA 1992b).

Summary of Chromium Criteria		Source
EPA carcinogen classification (inhalation of Chromium VI only)	Group A	EPA 1992b
Inhalation carcinogenic potency factor	42 $(mg/kg\text{-}day)^{-1}$	EPA 1992b
Oral RfD (Chromium VI)	0.005 (mg/kg-day)	EPA 1992b
RfC (Chromium VI) (under review)	3.0×10^{-4} $\mu g/m^3$	EPA 1992b
Maximum Contaminant Level (MCL) (Total Chromium)	0.1 mg/L	EPA 1992d
Maximum Contaminant Level Goal (MCLG) (Total Chromium)	0.1 mg/L	EPA 1992d
EPA Drinking Water Health Advisories		
Lifetime Health Advisory (HA)	0.1 mg/L	EPA 1992d
Longer-term HA		
Child	0.2 mg/L	EPA 1992d
Adult	0.8 mg/L	EPA 1992d
Shorter-term HA		
10-day HA (child)	1 mg/L	EPA 1992d
One-day HA (child)	1 mg/L	EPA 1992d
Ambient Water Quality Criteria (AWQC)		
Water and fish consumption	50 μg/liter	EPA 1992b

6.3.3 COPPER

Copper (Cu) is a reddish colored metal with the atomic number 29 and an atomic weight 63.5 g/mole. It is widely used as a structural metal, particularly when high electrical and thermal conductivity are required. Copper salts are used as fungicides, in ceramics, and for electroplating, and have a wide variety of other industrial uses (ACGIH 1986).

Toxicokinetics

Copper can be absorbed following dermal, oral, or inhalation exposure. The levels of copper in the body are held constant by alterations in the rate and amount of copper absorbed, its distribution, and rate of excretion. Little is known about distribution of absorbed copper, except that it binds to some plasma proteins and is transported to the liver. It is released back to plasma from the liver. Copper is primarily excreted in the feces following oral exposure (ATSDR 1990b).

Qualitative Description of Health Effects

Copper is an essential element in human nutrition. A daily copper intake of 2 mg is considered to be adequate for health and normal copper metabolism. The normal daily adult intake of copper from food in the United States is reported to range from 2 to 4 mg per day. The reported average intake of copper in young children is 1.5 mg/day; the minimum dietary requirement is 0.10 μg/kg of body weight per day (EPA 1985e).

Toxic effects resulting from acute over-exposure to copper in laboratory animals and humans include gastrointestinal disturbances, hemolytic anemia, renal damage, liver damage, and glucose-6-phosphate dehydrogenase inhibition. Limited data are available on the chronic toxicity of copper; however, chronic over-exposure may cause anemia. Efficient homeostatic mechanisms generally protect mammals from the adverse effects of dietary copper excess. In humans, individuals with Wilson's disease are at additional risk from the toxic effects of copper. Wilson's disease is an inborn error of copper metabolism in which copper accumulates in the liver, brain, and kidney, resulting in hemolytic anemia, neurological abnormalities, and corneal opacities. In addition, individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency are likely to be at increased risk to the toxic effects of copper (EPA 1985e).

Ingestion of copper salts by humans can cause salivation, gastrointestinal irritation, nausea, vomiting, gastric pain, hemorrhagic gastritis, and diarrhea. Dermal exposure to copper salts can produce skin irritation and eczema. Ocular contact with copper salts may produce conjunctivitis and corneal lesions. Inhalation of dusts and mists of copper salts may result in irrigation of the mucous membranes and pharynx, as well as ulceration and perforation of the nasal septum. A condition similar to metal fume fever has been observed in workers exposed by inhalation to metallic copper dust at concentrations of approximately 0.2 mg/m^3. Metal fume fever is characterized by short-term chills, fever, aching muscles, dryness of throat and mouth, and headache. Extensive industrial experience with copper-welding operations and copper-metal refining suggests that no adverse effects result from exposure to copper fumes at concentrations of up to 0.4 mg Cu/m^3 (ACGIH 1986).

Copper compounds have generally provided negative results in microbial mutation assays. Copper sulfate was observed to increase the frequency of recessive lethal mutations in *D. melanogaster* at

high concentrations. Equivocal results have been obtained from carcinogenicity studies. Administration of copper compounds to mice by subcutaneous injection has been reported to induce tumor formation. Orally-administered copper compounds were not found to increase tumor incidence in several studies (EPA 1985e). There is little evidence in the literature to suggest that copper has a teratogenic effect in either animals or humans (EPA 1980c)

The health effects of copper in man and animals are reviewed in more detail by EPA (1985a, 1984i, 1980c).

Quantitative Description of Health Effects

The International Agency for Research on Cancer (IARC) has not evaluated the carcinogenic potential of copper. Applying the criteria for carcinogenicity as proposed by EPA's Carcinogen Assessment Group, copper is classified in Group D — Not Classified. This category applies to chemical agents for which there is inadequate evidence of carcinogenicity in animals and humans (EPA 1984i, 1992b).

The EPA (1992a) has established a maximum contaminant level goal (MCLG) of 1.3 mg/L for drinking water (EPA 1992b). This value is based on the observation that copper exposure at high levels may cause gastrointestinal disturbances and other acute toxic effects at oral doses greater than 5.3 mg. An oral RfD of 4×10^{-2} (mg/kg-day) can be derived from this analysis. However, the EPA Office of Drinking Water concluded that toxicity data were inadequate for calculation of an RfD (EPA 1992b).

Copper is included in the National Drinking Water Regulations with a secondary standard of 1.0 mg/L based upon taste and odor. The World Health Organization (WHO) has not proposed a guideline for copper based upon health effects; however they have proposed a guideline value of 1 mg/L based upon the ability of copper to stain laundry and plumbing fixtures at concentrations above 1 mg/L (EPA 1985e).

In the Health Effects Assessment for copper (EPA 1984i), an allowable intake chronic (AIC) of 3.7×10^{-2} (mg/kg-day) was derived based on the human lowest-observed-adverse-effect level (LOAEL) of

5.3 mg/day and an uncertainty factor of two. This analysis is consistent with the analysis presented by the Office of Drinking Water.

The American Conference of Governmental Industrial Hygienists (ACGIH) recommended time-weighted average threshold limit value (TLV) for copper is 1 mg/m^3 (copper dust or mist). ACGIH also recommends a time-weighted average TLV of 0.2 mg/m^3 for copper fume.

Summary of Copper Criteria		Source
EPA carcinogen classification	Group D	EPA 1984i
MCLG	1.3 mg/L	EPA 1992b

6.3.4 2,4-DINITROTOLUENE

Dinitrotoluene (DNT) does not occur naturally, but is produced when toluene and nitric acid are mixed. DNT is primarily used to create flexible polyurethane foams for the bedding and furniture industry. The chemical is also used in the production of dyes, ammunition and explosives. Industrially-used dinitrotoluene generally consists of a mixture of the 2,4- and 2,6-isomers. Technical grade DNT is composed of approximately 76 percent 2,4-DNT, 19 percent 2,6-DNT and small amounts of other isomers. Pure grade 2,4-DNT consists of 98 percent 2,4-DNT and 25 percent of 2,6-DNT and/or other isomers. The following information regarding the health effects of DNT is condensed from EPA (1992b) and ATSDR (1989d).

Toxicokinetics

Dinitrotoluene is absorbed through the lungs, the gastrointestinal tract, and the skin. No quantitative information regarding absorption via inhalation and dermal exposure is available. Upon oral exposure at least 55-90 percent of DNT is absorbed (Rickert and Long 1980). Once absorbed, DNT is temporarily distributed into blood, liver, kidney, lungs, and intestines. Low levels of DNT also have been detected in the brain, heart, and spleen of mice (Schut et al. 1983).

DNT is readily metabolized and excreted. The major metabolite excreted in the urine of individuals exposed to DNT is 2,4-dinitrobenzoic acid (Woollen et al. 1985).

Qualitative Description of Health Effects

The available data concerning human health effects due to inhalation of DNT stem from studies of occupational exposure. No animal data regarding DNT inhalation are available. workers in case studies were exposed to technical grade DNT, and the effects of the individual isomers in DNT have not been separated. Chronic inhalation exposures of workers to dinitrotoluene resulted in increased mortality from heart disease (Levine et al. 1986), cyanosis and anemia (Perkins 1919; McGee et al. 1942), liver tenderness (McGee et al. 1947), dizziness and headaches (McGee et al. 1942), and reduction in sperm counts (Ahrenholz 1980). No carcinogenic effects of inhalation exposure to DNT were reported. The results of occupational exposure studies are limited by small numbers of cohorts, and by lack of consideration of individuals' lifestyles.

Available data regarding health effects due to oral exposures to DNT stem from laboratory experiments with animals. No information regarding ingestion of DNT is available for humans. Ingestion of 2,4-DNT causes mortality in rats, mice and dogs (Lee et al. 1978) with sensitivities differing greatly between different types of animals. High incidences of mortality were noted in dogs, rats, and mice following ingestion of 25, 206, and 441 (mg/kg-day) of 2,4-DNT administered over a 13 week period, (Lee et al. 1978). In another study in which animals were exposed for 24 months, doses of 10, 40, and 898 (mg/kg-day) caused mortality in dogs, rats, and mice, respectively (Ellis et al. 1979).

Hematological effects due to oral exposure to 2,4-DNT included: methemoglobinemia, anemia, and reticulocytosis (Ellis et al. 1979), and cyanosis (Lane et al. 1985). In rats, anemia was observed at a dose of 100 (mg/kg-day) in a study with subchronic exposure duration, and a dose of 14 (mg/kg-day) in a study with a chronic exposure duration (Hazleton Laboratories 1982; Hazleton Laboratories 1977). Hepatoxic effects due to ingestion of 2,4-DNT included hepatic lesions, hepatic dysplasia, and hepatocellular carcinoma (Ellis et al. 1979). Exposure also resulted in liver discoloration and inflammation (Hazleton Laboratories 1982). Renal effects due to oral exposure to 2,4-DNT included renal dysplasia in male mice, cystic degeneration, anaplastic epithelium, and renal tumors (Ellis et al. 1979). Neurological effects included tremors, convulsions, ataxia, and paralysis (Lee et al. 1978). Reproductive effects due to ingestion of 2,4-DNT included decreased sperm production, testicular and ovarian atrophy, and degeneration of the seminiferous tubules.

Oral exposure to DNT was shown to induce cancer in laboratory animals. In one study 76 percent of male mice fed daily doses of 97 mg of 2,4-DNT (98 percent 2,4-DNT, 2 percent 2,6-DNT) per kg developed renal tumors within 2 years. Increased incidences of hepatocellular carcinomas in rats were also noted (Ellis et al. 1979).

Data regarding dermal exposure to DNT are very limited. Mortality was observed in two cats that received dermal applications of 3.3 g/kg of 2,4-DNT (Zieger 1913). In rabbits 2,4-DNT was reported to be a mile dermal irritant (Lee et al. 1975).

Quantitative Description of Health Effects

EPA has developed an oral reference dose for 2,4-dinitrotoluene. Ellis et al. (1985) gave beagle dogs doses of up to 10 (mg/kg-day) 2,4-DNT in gelatin capsules. Neurotoxic effects and lesions of the central nervous system were commonly observed. The LOAEL of 1.5 (mg/kg-day) is based on neurotoxicity and biliary tract hyperphasia. The NOAEL is 0.2 (mg/kg-day) to which an uncertainty factor of 100 was applied (EPA 1992b). The human health ambient water quality criteria for 2,4-dinitrotoluene are 1.1×10^{-1} μg/L for water and fish consumption, and 9.1 μg/L for fish consumption. These criteria are based on a 1×10^{-6} estimated incremental increase in lifetime cancer risk (EPA 1992b).

While some data are available for the health effects of 2,4-dinitrotoluene, more extensive data exist for the 2,4-/2,6-dinitrotoluene mixture. Ellis et al. (1979) tested the mixture for carcinogenicity in Sprague-Dawley rats. Doses of 0, 15, 100, or 700 ppm of the mixture (98 percent, 2,4-dinitrotoluene and 2 percent 2,6-dinitrotoluene) were administered daily in food. After treatment significant increases in hepatocellular carcinomas were observed in male and female rats. In additions, increases in mammary gland adenomas, fibroadenomas, fibromas, and adenocarcinomas were noted in female rats. EPA combined the data for tumor incidence with dose information to calculate an oral slope factor of 6.8×10^{-1} (mg/kg-day), and a drinking water unit risk of 1.9×10^{-5} $\mu g/L^{-1}$.

No inhalation slope factor is currently available.

Summary of 2,4-dinitrotoluene criteria		Source
Oral Reference Dose	0.2 (mg/kg-day)	EPA 1992b
Ambient Water Quality Criteria:	9.1 (μg/L)	EPA 1992b
Fish ingestion	1×10^{-1} (μg/L)	EPA 1992b1c
Fish and water ingestion	1.1×10^{-1}	

Summary of 2,4-/2,6-dinitrotoluene criteria		Source
EPA Carcinogen Classification	Group B2	EPA 1992b
Oral RfD (2,4-DNT)	2.0×10^{-3} (mg/kg-day)	EPA 1992b
Inhalation RfC	Not Available	EPA 1992b
Oral carcinogenic potency (2,6-DNP)	6.8×10^{-1} (mg/kg-day)$^{-1}$	EPA 1992b
Inhalation carcinogenic potency	Not Available	EPA 1992b
Ambient Water Quality Criteria:		
Fish ingestion	9.1 (μg/L)	EPA 1992b
Fish and water ingestion	1.1×10^{-1} (μg/L) 10^{-1}	EPA 1992b

6.3.5 DIOXINS AND FURANS

Polychlorinated dibenzo-p-dioxins (PCDDs) are a family of 75 congeners, each of which is an isomer of one of eight homologous PCDDs with varying degrees of chlorination. Polychlorinated dibenzofurans (PCDFs) are a closely related family of components comprised of 135 compounds containing up to eight chlorine atoms. These families are referred to generically as "dioxins" and "furans." PCDDs and PCDFs are considered together because of their similar toxic effects. PCDD/PCDF isomers are not naturally occurring substances, but are formed as contaminants or impurities during chemical production or pyrolysis. Although there is general agreement that PCDD/PCDF isomers are produced by burning wood with added HCl and by incinerators burning chlorinated wastes (Tiernan et al. 1985), PCDD/PCDF isomer production from combusting coal and hydrocarbons (such as occurs in gas burners and auto and truck engines) has not been confirmed (NRCC 1981). Experiments indicate that dioxin is produced during the burning of specific chemicals such as chlorinated phenols, polychlorinated benzenes, and polychlorinated diphenyl esters (Rappe et al. 1986).

Qualitative Description of Health Effects

Toxicokinetics

Absorption of 2,3,7,8-TCDD appears to be efficient (> 87 percent) based on a study involving a single human volunteer (Poiger and Schlatter 1986). Studies in animals support this finding (e.g., Piper et al. 1973) and also indicate that the presence of food in the gastrointestinal tract may limit absorption. Fries and Marrow (1975a,b) found that absorption was about 50 percent when TCDD was administered in the diet of rats. When TCDD is administered bound to soil, absorption is also attenuated suggesting that binding to soil constituents can lower bioavailability (Lucier et al. 1986). When administered bound to activated charcoal, no TCDD absorption could be measured suggesting that the organic content of soil may be a limiting factor in determining absorption (Poiger and Schlatter 1986).

Absorption of TCDD following inhalation has not been well studies and the literature contains no data on which to base estimates of absorption efficiency (ATSDR 1987). However, the EPA assumed that absorption through the lungs would be efficient in extrapolating the oral slope factor for TCDD to the inhalation route.

Mechanisms of Action

Much current knowledge on the toxic effects of dioxins and furans comes from studies using the single potent dioxin congener, 2,3,7,8-tetrachlorodibenzo(p)dioxin. Generalization to other agents is assumed based on similar biochemical and molecular properties and on a few studies using other congeners.

The current consensus on mechanism of action for dioxins and furans is that selective binding to a high affinity receptor protein in the cytosol of mammalian cells is responsible for the exceptionally high toxicity and unusual spectrum of effects seen following exposure.

2,3,7,8-TCDD binds selectively to a high affinity "receptor" protein in the cytosol of mammalian cells (Roberts et al. 1985; Poland et al. 1979; Carlstedt-Duke 1978). The TCDD-receptor complex is

translocated to the nucleus of the cell where it binds to DNA and alters gene expression as indicated by increased mRNA synthesis. Receptor binding is associated with the induction of aryl hydrocarbon hydroxylase (AHH) and a variety of other enzymes. This induction has been demonstrated in a number of different tissues, but is particularly marked in the liver, kidney, thymus, and skin, which are important target organs for 2,3,7,8-TCDD toxicity. The affinity of the cytosolic receptor for 2,3,7,8-TCDD varies widely among and within species. At least in mice, this variability is genetically controlled and is associated with the Ah gene locus (Poland et al. 1976), a locus that is also associated with induction of AHH. A number of researchers have recently determined that the sensitivity of experimental animals to many of the biological effects of 2,3,7,8-TCDD is associated with AHH inducibility and segregates with the Ah locus during cross-breeding experiments in mice (Poland et al. 1976). These findings are important for risk assessment because they show genetic variability in susceptibility to biological effects of 2,3,7,8-TCDD. Especially wide variations in susceptibility are expected in a genetically heterogenous species such as humans.

Acute Toxicity

The most frequently observed adverse health effect resulting from acute exposure to dioxins in humans is chloracen. TCDD is known to be one of the most potent compounds in producing chloracne; however, sufficient data on exposure are not available to define the dose necessary to produce this effect. Chloracne develops several days to months after exposure to dioxins and may persist for as long as 29 years after exposure (Suskind 1985). Although chloracne has been reported in most or all cases of occupational exposure, in many cases only a portion of the workers subject to exposure developed chloracne, suggesting variability susceptibility. It is believed that humans can develop chloracne following exposure to 2,3,7,8-TCDD by any route.

Effect on the immune system also appear to be associated with exposure to 2,3,7,8-TCDD. In a clinical study of 154 former residents of a mobile home park (Quail Run, Missouri) where the soil was contaminated with 2,3,7,8-TCDD, Hoffman et al. (1986) reported a significant reduction in delayed hypersensitivity responses to standard antigens among a subgroup of 51 residents, compared with 93 controls. Measures of T-cell functioning were also depressed, alghouth not significantly, among the residents. These results suggest an association between impairment of the immune system

and exposure to 2,3,7,8-TCDD. However, actual exposures were not documented or measured in this study.

A number of studies investigated reproductive outcomes in human populations exposed to 2,3,7,8-TCDD, but these studies are severely compromised by difficulties in documenting exposure, and in establishing rates of adverse reproductive outcomes in comparison populations. for example, Hanify et al. (1981) found a statistical association between incidence of birth defects (heart defects and talipes) and wide-area spraying of 2,4,5-T. Overall, however, the evidence for an association between exposure to 2,3,7,8-TCDD and adverse reproductive outcomes is inconclusive.

Carcinogenicity

Several Swedish epidemiological studies have reported an association between occupational exposure to phenoxy acid herbicides or chlorophenol and increased incidence of certain cancers, including soft tissue sarcomas, non-Hodgkin's lymphomas, and nasopharyngeal cancers (Eriksson et al. 1981; Hardell et al. 1981; Hardell and Standstrom 1979). The presumptive link between these exposures and cancer is the presence of 2,3,7,8-TCDD or other dioxin isomers as impurities in phenoxy acids and chlorophenol.

A case-control study of similar design in New Zealand failed to demonstrate a significantly increased relative risk for soft tissue sarcoma among individuals exposed to phenoxy herbicides or chlorophenol (Smith et al. 1982, 1983). Lynge (1985) reported excess incidences of soft-tissue sarcomas among Danish workers employed in the manufacture of phenoxy herbicides, but most of the herbicides involved were not contaminated with 2,3,7,8-TCDD. There have been several case reports of soft-tissue sarcomas among U.S. workers exposed to phenoxy acids and/or dioxins (Zack and Gaffey 1983; Cook 1981; Johnson et al. 1981; Zack and Suskind 1980. However, Fingerhut et al. (1984) showed that some of these reports were based on erroneous pathological diagnoses. In a small but well-controlled study, Thiess et al. (1982) reported a significant excess of stomach cancers among worker presumptively exposed to 2,3,7,8-TCDD in a chemical reactor accident 23 years earlier. Other cancer studies have been inadequate to show either positive or negative results. Although some of these results suggest a possible association between exposure to 2,3,7,8-TCDD and increased risk of cancer, the evidence taken as a whole is inconclusive.

Several factors complicate the interpretation of the toxic effects of 2,3,7,8-TCDD, especially the extrapolation of animal data to predict likely effects in humans.

The studies regarding the toxicity of 2,3,7,8-TCDD in humans did not include adequate characterization of exposure. Many studies were of human populations exposed to phenoxy acids or chlorophenol, in which contamination with 2,3,7,8-TCDD is likely but was not verified or measured. In addition, quantitative characterization of exposure was not provided in any of the studies. Therefore, the human data are useful only for qualitative comparison with the animal data.

Another factor that complicates the extrapolation of the toxicity data of 2,3,7,8-TCDD in animals to humans is that the persistence of 2,3,7,8-TCDD in humans is not known. 2,3,7,8-TCDD is relatively persistent in the environment and in many living systems. It is concentrated in the fat and liver of most species following absorption. In rodents, the biological half-life of 2,3,7,8-TCDD ranges from 10 to 43 days. However, McNulty et al. (1982) reported much longer persistence in the tissue of rhesus monkeys, probably greater than one year. Poiger and Schlatter (1986) estimated that about 90 percent of the body burden of 2,3,7,8-TCDD in a single volunteer was sequestered in fat, and calculated a half-life of 2,120 days assuming first order kinetics. This data is consistent with the high bioconcentration potential of 2,3,7,8-TCDD as calculated by Geyer et al. (1986). Though there is little hard data on which to estimate persistence of dioxins and furans in humans, it seems reasonable from the above, to assume a relatively long half-life.

A final factor which complicates extrapolation of animal toxicity data to humans is that toxic responses to 2,3,7,8-TCDD vary widely among and within species. For example guinea pigs, rhesus monkeys and chickens are extremely sensitive to the acute toxic effects of 1,3,7,8-TCDD, rats and mice are intermediate in sensitivity, and hamsters are relatively insensitive. The significance of intraspecies variability is that some individuals may be much more susceptible than others, requiring the use of large safety factors to protect the most sensitive individuals.

Quantitative Description of Health Effects

The oral slope factor for 2,3,7,8-TCDD is based on a feeding study in rats in which dose-dependent increase in tumors were seen at various sites depending on the sex of the animal (Kociba et al.

1978a,b). Extrapolation of these data using a linear multistage model results in a slope factor 1.5×10^{5}. Toxicologically, there seems little reason to believe that route of entry effects toxicologic outcome. Thus, the slope factor for inhalation exposure was assumed to be the same as for oral. However, the slope factors are under review by the EPA (EPA 1992b). The EPA has approved a method to assess the carcinogenicity of other dioxin isomers by applying toxicity equivalency factors (TEFs) to these isomers (EPA 1989h). These TEFs are multiplied by the cancer slope factor for 2,3,7,8-TCDD to estimate the cancer slope factor for other dioxin isomers. These TEFs are listed in Table 6-2.

No RfD or RfC have been established to TCDD. However, the proposed MCL (5×10^{-8} mg/L in EPA 1991a.) suggests that an acceptable daily intake (ADI) might be 1×10^{-9} mg/kg-day assuming that a 70 kg human consumes 2L of contaminated water per day [$(5 \times 10^{-8}$ mg/L x 2L/day)/70 kg]. This is consistent with the estimate for a chronic daily dose (1×10^{-9} mg/kg-day) associated with "minimal risk for effects other than cancer" (ATSDR 1987). This RfD is based on a three-generation study in rats exposed to 2,3,7,8-TCDD diets at doses of 0.001, 0.01, and 0.1 μg/kg-day (Murray et al. 1979). The lowest dose resulted in renal effects, decreased fetal weight, and changes in the gestational index. The low dose, when adjusted by an uncertainty factor of 1,000, resulted in an RfD of 1×10^{-9} mg/kg-day^{-1}. For subchronic effects, ATSDR (1987) suggests that a daily dose of 1×10^{-6} mg/kg-day is also associated with minimal risk for effects other than cancer provided the exposure period is 14 days or less.

Summary of 2,3,7,8-TCDD Criteria		Source
EPA carcinogen classification	B2	EPA 1992b
Oral slope factor	$1.5 \times 10^{+5}$ (mg/kg-day)$^{-1}$	EPA 1992c
Inhalation slope factor	$1.5 \times 10^{+5}$ (mg/kg-day)$^{-1}$	EPA 1992c
Maximum Contaminant Level (MCL)	5×10^{-8} (mg/L)	EPA 1992d
Maximum Contaminant Level Goal (MCLG)	Zero	EPA 1992d
Ambient Water Quality Criteria (AWQC) (10^{-4} to 10^{-7})	1.3×10^{-9} to 1.3×10^{-12} (mg/L)	EPA 1984k

TABLE 6-2

TOXICITY EQUIVALENCY FACTORS FOR CHLORINATED DIBENZO-P-DIOXINS AND -DIBENZOFURANS[a]

Compound	TEF
Mono, Di, and TriCDDs	0
2,3,7,8-TCDD Other TCDDs	1 0
2,3,7,8 - PeCDD Other PeCDDs	0.5 0
2,3,7,8 - HxCDD Other HxCDDs	0.1 0
2,3,7,8 - HpCDD Other HpCDDs	0.01 0
OCDD	0.001
Mono, Di-, and TriCDFS	0
2,3,7,8 - TCDF Other TCDFs	0.1 0
1,2,3,7,8 - PeCDF 2,3,4,7,8 - PeCDF Other PeCDFs	0.05 0.5 0
2,3,7,8 - HxCDF Other HxCDFs	0.1 0
2,3,7,8 - HpCDF Other HpCDFs	0.01 0
OCDF	0.001

a EPA 1989. Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDDs and CDFs) and 1989 Update. EPA/625/3-89/016.

6.3.6 LEAD

Lead is a ubiquitous metal in nature. Concentrations in rocks and soils from natural sources range from 10 to 30 mg/kg. High concentrations of lead are usually found in conjunction with cadmium, zinc, and silver ores. Lead is widely used in industry because of its softness, resistance to corrosion and radiation, and high density. Major uses of lead have been in batteries, as an additive in gasoline, as a pigment in paint, in solders, and in other alloys. The combustion of leaded gasoline has been the major source of environmental pollution. Because of its extensive use, the potential for exposure to lead is great.

Toxicokinetics

Absorption of lead from the gastrointestinal tract is estimated at 10-15 percent for adults. For young children, absorption of dietary lead may be much greater (~50 percent) (Hammond 1982). Absorption of lead from soil is probably less, and may be in the range of 25-30 percent based on animal bioassay data (ATSDR 1990c; EPA 1990a; Drill et al. 1979). Two recent studies on bioavailability of lead from mining/milling wastes have been published. Freeman et al. (1991) examined uptake of lead by rats in a dietary feeding study. Lead was supplied in the form of a composite of lead contaminated soils taken from Butte, Montana. Estimates of absorption from this study range from 2 to 10 percent. In contrast, LaVelle et al. (1991) measured uptake of lead by young pigs following intubation of an aqueous slurry of a mixture of soil and mill tailings taken from Midvale, Utah. Absorption estimates from this study ranged up to 40 percent, falling in a similar range as those cited above for other soils. A paper by Weis and LaVelle (1991) provides evidence that rats may be a poor model for lead absorption by young children, and that data from young pigs may be more appropriate for extrapolation to children less than 6 years old.

Essentially 100 percent of the lead deposited in the deep lung following inhalation is eventually absorbed. Respiratory uptake in children appears to be greater than adults on the basis of body weight. Once absorbed, lead is stored in the body in kidney, liver, and bone where it may remain for long periods of time. Lead is primarily excreted by the kidneys into the urine, with lesser amounts eliminated via biliary excretion (EPA 1984h,m).

Qualitative Description of Health Effects

The following information has been summarized from EPA (1984m, 1990a) and ATSDR (1990c). Lead has diverse biological effects in humans and animals. Considerable data exists on the effects of lead exposure in humans, and these effects are generally related to a direct measure of human exposure (e.g., blood lead levels). Currently, the EPA is refining an uptake biokinetic model for use in predicting blood lead concentration from intake of lead by various routes and from various media.

A major problem associated with lead exposure is the ubiquitous nature of the compound. Unlike many other contaminants for which exposure may be related to a specific route or situation, substantial background lead exposure primarily occurs through diet. This background exposure is an important determinant of blood lead level and must be considered when additional significant exposure routes are identified.

Major toxic effects caused by chronic, low-level exposure to lead are alterations in the hematopoietic and nervous systems. In addition, low levels of lead in blood appear to be associated with small increases in diastolic blood pressure, and higher doses of lead can produce damage to kidney, G.I. tract (colic), liver, and endocrine glands (ATSDR 1990c; Tsuchiya et al. 1978). Heme synthesis is inhibited by the effects of lead on several steps in the biosynthetic pathway. Specifically, lead stimulates delta-aminolevulinic acid synthetase (ALA-S), thereby increasing the production of delta-aminolevulinic acid dehydrase (ALA-D). Lead also inhibits ALA-D leading to accumulation of ALA. Finally, lead inhibits ferrochetalase (heme synthetase), thereby inhibiting insertion of iron into the protoporphyrin ring. This in turn leads to the generation of zinc protoporphyrin (ZPP) due to substitution of zinc for iron in the porphyrin moiety. ZPP remains in erythrocytes throughout their lifetimes in blood and can be used as a clinical sign of recent lead exposure (ATSDR 1990c). No threshold has been identified for this effect on heme production. Decreased heme production may result in significantly decreased hemoglobin production and anemia when exposures are large enough. Decreased heme production can also have deleterious effects on other heme-containing proteins, such as cytochrome P450, which detoxify certain chemicals in the body. Impaired heme synthesis has been reported in adults at levels of less than 30 μg/dl lead in the blood (EPA 1984m).

Exposure to lead which results in levels of lead in the blood (PbB) of over 80 μg/dL in children and over 100 μg/dL in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possibly death. Persons with these high levels may be asymptomatic or show only slight signs of intoxication, but rapid deterioration can occur. In children, permanent learning disabilities are seen at these levels, even if there are no overt signs of lead poisoning (EPA 1984m).

At lower blood lead levels, effects on the nervous system can be much more subtle. At blood lead levels as low as 30 to 70 μg/dL in adults, nerve conduction velocities can be reduced, and these effects can lead to neuromotor dysfunction in the extremities (foot-drop and wrist-drop syndromes). In children, significant deficits in IQ and behavioral indices were noted in children with pre and/or postnatal blood levels of 70 μg/dL extending down to at least 10-15 μg/dL. Accompanying these low level effects on cognitive function were retardation of bone growth and hearing deficits. As with inhibition of heme synthesis, there has been no indication of a threshold for lead effects on the nervous system in data from epidemiologic studies. Thus, it is possible that adverse effects could occur at blood lead levels less than 10-15 μg/dL. Although there is still some controversy over the interpretation of effects in children with the lowest blood lead levels, there seems to be a general consensus that very low blood lead levels in young children may produce undesirable effects and attempts should be made to reduce lead exposure prenatally and in the youngest age groups as much as possible.

Other adverse effects are associated with exposure to low levels of lead. Slow nerve conduction in peripheral nerves has been seen in adults at 30-40 μg/dl blood lead levels (PbB); altered testicular function was observed in men with PbB levels as low as 40-50 μg/dl; and renal dysfunction has been associated with PbB levels as low as 40 μg/dL (EPA 1984m).

The voluminous literature on lead is difficult to summarize briefly. The Toxicological Profile for Lead (ATSDR 1990c) includes over 520 references and is not comprehensive. The above synopsis is taken from the following reviews: EPA 1984m; ATSDR 1990c; EPA 1989c.

Quantitative Description of Health Effects

Oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) have been associated with increased renal tumor frequency in rats (EPA 1985f), but no quantitative estimate of excess cancer risk has been performed by the Carcinogen Assessment Group of the EPA. The EPA (1985f) has noted that the available data provide an insufficient basis on which to regulate lead acetate, lead phosphate, and lead subacetate as human carcinogens. However, applying the criteria described in EPA's Guidelines for Carcinogenic Risk Assessment (EPA 1986c), these lead salts have been classified by the EPA (1992b) in Group B2 — Probable Human Carcinogen. This category applies to agents for which there is inadequate evidence from human studies and sufficient evidence from animal studies.

There is no current maximum contaminant level (MCL) for lead (EPA 1992b). The Treatment Technique Action Level of 0.015 mg/L was recently finalized (June 1991) by the Office of Drinking Water (EPA 1992d). The maximum contaminant level goals (MCLGs) for lead at the source and at the tap are both zero.

The EPA Office of Drinking Water issued a draft health advisory of 20 μg/day for all extended periods of exposure (EPA 1985f). Blood levels above 15 μg/dl were identified as the level of concern, and fetuses and infants under 2 years of age are the sensitive subpopulation. In order to protect the fetus, it was considered advisable to limit the blood lead level in women of child-bearing age to below 15 μg/dl since studies indicate that the ratio of fetal/maternal blood lead values is close to 1:1 (Hubermont et al. 1978 as cited in EPA 1985f).

The Clean Air Act National Ambient Air Quality Standard for lead is 1.5 $\mu g/m^3$ (EPA 1992b).

Acceptable intakes for chronic or subchronic periods of exposure were not calculated for either inhalation or oral ingestion in the Health Effects Assessment Document (EPA 1984m) because the general population is already accruing unavoidable background exposures through food, water, and dust. Any significant increase above background exposure would represent a cause for concern. In lieu of AICs or RfDs, EPA is currently refining a computer model for prediction of blood lead levels in children exposed to lead from a variety of sources (EPA 1991a).

At present, human health criteria for lead in soil have not been established in the United States. The United Kingdom Directorate of the Environment has developed a tentative guideline of 550 ppm for lead in soil in residential areas (Smith 1981). Vernon Houk of the Centers for Disease Control has been quoted as indicating that levels of lead in soil of 300-400 ppm are acceptable based on studies of childhood lead poisoning (Mielke et al. 1984).

No RfC is available for lead, and, as discussed above, it is not clear that there is a threshold below which there are no risks from exposure to lead. Since RfCs are based on the assumption that such a threshold exists, estimation of an RfC for lead is not appropriate at this time.

However, the impact of inhalation of lead in incinerator emissions can be assessed by the use of the IU/BK model discussed above. This model allows for the impact of lead in air on blood lead levels in children to be estimated. Thus, estimated blood lead levels can then be compared to targetblood lead concentrations to assess possible risks. This approach was taken in assessing risks from inhalation of lead.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 0.15 mg/m^3 lead in air.

Summary of Lead Criteria		Source
EPA Carcinogen Classification	Group B2	EPA 1992b
Oral RfD	Not available	EPA 1992b
Inhalation carcinogenic potency	Not available	EPA 1992b
Oral carcinogenic potency	Not available	EPA 1992b
Maximum contaminant level (MCL)	None	EPA 1992d
Treatment Technique Action Level (TT)	0.015 mg/L	EPA 1992d
Maximum Contaminant Level Goal (MCLG)	0 mg/L	EPA 1992d
EPA Drinking Water Health Advisories	Not available	EPA 1992d
Ambient Water Quality Criteria (AWQC)	Varies with hardness	EPA 1986b
Water and fish consumption	50 μg/L	EPA 1986b
National Ambient Air Quality Standard (NAAQS)	1.5 μg/m	EPA 1984h

6.3.7 MANGANESE

Elemental manganese is a grey-white metal resembling iron, with atomic number 25 and an atomic weight of 55 g/mole. It is highly reactive and can be present in 7 oxidation states. Manganese if often used as an alloy to impart hardness.

Toxicokinetics

Manganese compounds are practically insoluble in water or body fluids; therefore, only manganese in particles small enough to reach the alveolar lining can be absorbed into the blood. The degree of absorption by inhalation is unknown. Absorption of manganese by the oral route is controlled by homeostatic mechanisms. The absorption rate will depend on the amount ingested and on tissue levels of manganese. Limited information on humans indicates that absorption is only about 3 percent of the administered dose (Saric 1986).

Absorbed manganese is rapidly eliminated from the blood and distributed to the liver. Manganese is distributed in the body in constant concentrations that are characteristic of the individual tissues. In blood, manganese is bound to proteins. Absorbed manganese is almost totally excreted in the feces (Saric 1986).

Qualitative Description of Health Effects

The toxic effects of manganese have been studies primarily in workers who have inhaled manganese containing dust (EPA 1984j). Exposure to high levels of manganese causes pneumonitis in exposed workers. Chronic exposure has also been associated with manganism — a progressive neurological disease similar to Parkinson's disease, manifested by speech disturbances, a masklike face, tremors, difficulties in walking, and sexual disturbances (EPA 1984j). Although exposure in the cases of manganism reported by the EPA (1984j) was by inhalation, some of the manganese that is inhaled can be removed by mucociliary clearance and consequently swallowed (EPA 1984j), becoming available for absorption from the gastrointestinal tract. One case study reported apparent manganism associated with extremely high levels of manganese in a drinking water well, further suggesting that ingestion, as well as inhalation is an important route of exposure (Kawamura et al. 1941). Chronic exposure to

manganese also causes increased production of erythrocytes, with consequent increases in hemoglobin values and erythrocyte counts.

Quantitative Description of Health Effects

The EPA has derived an oral RfD for manganese based on extensive studies by the RC (1989), the HO (1973), and Schroeder et al. (1966). The World Health Organization reported no adverse effects in humans consuming supplements of 0.11 to 0.13 mg manganese/kg/day. The NRC determined "safe and adequate" levels to be 0.03 to 0.07 (mg/kg-day), and Schroeder et al. reported a chronic human NOAEL of 0.16 (mg/kg-day). From these studies, the EPA derived a NOAEL of 0.14 (mg/kg-day) and a RfD of 0.1 (mg/kg-day) since both uncertainty and modifying factors were 1 (EPA 1992b).

An inhalation RfC has also been developed based on the epidemiological study by Roels et al. (1987). In this cross-sectional study 141 male workers were exposed to manganese dioxide, tetroxide and various salts. The median time-weighted average (TWA) was identified as a LOAEL, converted to a human equivalent concentration, and corrected by uncertainty/modifying factors of 300 and 3, respectively. The resulting RfC is 1.1×10^{-4} (mg/kg-day) (EPA 1992c).

The EPA has assigned manganese a weight-of-evidence classification of D — not classifiable as to human carcinogenicity. This indicates that the existing data are inadequate to assess the carcinogenicity of manganese.

The NAS (1972) recommended that 0.05 mg/L soluble manganese not be exceeded in public water sources to prevent staining of plumbing fixtures and spotting of laundered clothes.

Summary of Manganese Criteria

EPA Carcinogen Classification	Group D	EPA 1992c
Inhalation RfC	1.1×10^{-4}(mg/kg-day)	EPA 1992c
Oral RfD	1×10^{-1} (mg/kg-day)	EPA 1992c
Inhalation carcinogenic potency	Not available	EPA 1992c
Oral carcinogenic potency	Not available	EPA 1992c
Secondary Maximum contaminant level	0.05	EPA 1992d
EPA Ambient Water Quality Criteria Water or fish consumption	100 µg/L	EPA 1992b

6.3.8 PENTACHLOROPHENOL

Pentachlorophenol (PCP) does not occur naturally in the environment. It is principally used as a wood preservative and the majority of use is in treatment of utility poles. In the past, PCP was one of the most widely used pesticides in the United States. However, its use is now restricted. Currently, the major releases to the environment occur during production and use as a wood preservative, with the greater portion released to air. The following information has been summarized from EPA (1992b) and ATSDR (1989c).

Toxicokinetics

PCP is readily absorbed through the lungs, gastrointestinal tract, and skin. Once absorbed, it is cleared from the body very rapidly, but low levels may remain in the liver, kidney, lung, brain, and plasma. Data on distribution to tissues following chronic low level exposure is extremely limited. Limited distribution of PCP to tissues has been observed and is through to be the result of binding of PCP to plasma proteins.

PCP is not readily metabolized and is excreted in the urine primarily in the unchanged state. Limited metabolism does occur in the liver. Little information is available on excretion of PCP following chronic low doses, but results of one study in exposed workers indicated that it was significantly slower than excretion following an acute dose. Urinary levels of PCP decreased only by 60 to 80 percent when the workers were absent from work for up to 18 days (Casarette et al. 1969).

Qualitative Description of Health Effects

Adverse health effects in humans following chronic exposure to PCP by inhalation include bronchitis, hematological effects (including aplastic anemia, increased numbers of immature leukocytes and basophils), decreased renal function and irritation of the skin and eyes (Begley et al. 1977, in ATSDR 1989c; Klemmer et al. 1980; and Roberts 1963).

Information on adverse effects following chronic oral exposure is based on animal bioassays. Effects include changes in erythrocyte and hemoglobin levels, multiple hepatic effects (including enlargement and degeneration), increased kidney weights, impaired renal function, and immunosuppression (Kerkvliet et al. 1982 in ATSDR 1989c; Kinzell et al. 1981; and Johnson et al. 1973). Animal bioassays indicate that pentachlorophenol is not teratogenic, but is embryo- and fetotoxic. Rats receiving up to 50 (mg/kg-day) on gestation days 8-11, 12-15, or 6-15 had significantly decreased body weights and an increased incidence of resorptions. Bioassays conducted by the National Toxicology Program (NTP 1989) indicate that two different technical grades of PCP given in the diet, were carcinogenic in mice. Male mice showed dose-related increases in the incidence of tumors of the adrenal medulla and hepatocellular adenomas and carcinomas. Female mice developed the same types of tumors while receiving one PCP commercial product (Dowicide EC-7), but developed only hemangiosarcomas of the spleen and liver when consuming TG-Penta.

Most information on adverse health effects following dermal exposure is contained in case reports of individuals exposed either occupationally or in the home. This information is limited in that dose and duration were not quantified, and concurrent exposure by inhalation probably occurred. Effects reported include: hematological disorders (aplastic anemia, red cell aplasia, and decreased hematocrit and leukocyte counts), hepatic disfunction (elevated SGOT and SGPT levels), impaired renal function, dermatitis, and corneal damage.

All of the data discussed in the preceding paragraphs have a common confounding factor, i.e., that technical grades of pentachlorophenol commonly contain impurities that could have contributed significantly to the toxic effects observed. The impurities include polychlorinated dibenzo-p-dioxins and dibenzofurans. However, humans are generally exposed to the technical grades of this compound so that available data may be indicative of environmental exposures.

It is generally considered that the mechanism of systemic toxicity of pentachlorophenol is the uncoupling of mitochondrial oxidative phosphorylation, resulting in accelerated aerobic metabolism and increased heat production (hyperthermia) (Weinbach and Garbus 1965 in ATSDR 1989c). Toxic effects following acute exposures (multiple central nervous system effects and hepatic effects at the cellular level) support his hypothesis (Gray et al. 1985 in ATSDR 1989c). However, the role of this mechanism in adverse effects observed following chronic low level exposure is less clear. The site of action of PCP is reportedly the cell membrane. The compound destablizes cell membranes by disrupting the membrane bilayer, resulting in changes in hydrogen ion permeability of the lipid matrix and loss of membrane function.

Quantitative Description Of Health Effects

As previously stated, the NTP has tested two different pentachlorophenol commercial products for carcinogenicity in mice. The compounds (technical grade (TG) and EC-7) were administered daily in food at doses of 0, 100, or 200 ppm (TG) or 0, 100, 200, or 600 ppm (EC-7). Male mice receiving TC or EC-7 developed hepatocellular adenomas and carcinomas, and benign and malignant tumors of the adrenal medulla. Females receiving TG showed a possible increase in lever tumors (statistical significance was not reached), and a statistically significant increase in hemangiosarcomas. Those receiving EC-7 developed benign and malignant liver tumors, tumors of the adrenal medulla, and vascular tumors (EPA 1992b). EPA has developed risk estimates using data on the female mice, combining the incidences of tumors of the liver, adrenal medulla, and vascular system. An oral slope factor was calculated as the geometric mean of the slope factors for the two PCP commercial products. The oral slope factor is 1.2×10^{-1} (mg/kg-day) and the drinking water unit risk is 3×10^{-6} $(\mu g/L)^{-1}$. No inhalation slope factor is currently available.

EPA has also developed an oral reference dose (RfD) based on a 2-year study in rats (Schwetz et al. 1978 in EPA 1991). Male and female rats received 3, 10, or 30 mg pentachlorophenol/kg/day. Adverse effects observed in females receiving the two higher doses included pigmentation of the liver and kidneys, while those receiving the highest dose had decreased body weight gains. Males exhibited pigmentation of the liver and kidneys only at the highest dose. The RfD was derived using a chronic NOAEL of 3 (mg/kg-day) and applying an uncertainty factor of 100 for intra- and interspecies variability, resulting in a RfD of 3×10^{-2} (mg/kg-day). EPA rated confidence in the RfD

as medium because more chronic studies are needed. The Agency has not developed an inhalation RfC.

Summary of Pentachlorophenol Criteria		Source
EPA Carcinogen Classification	Group B2	EPA 1992b
Inhalation RfC	Not available	EPA 1992b
Oral RfD	3×10^{-2} (mg/kg-day)	EPA 1992b
Inhalation carcinogenic potency	Not available	EPA 1992b
Oral carcinogenic potency	1.2×10^{-1} (mg/kg-day)$^{-1}$	EPA 1992d
Maximum contaminant level	0.001 mg/L	EPA 1992d
Maximum contaminant level goal	0 mg/L	EPA 1992d
EPA Drinking Water Health Advisories		EPA 1992d
1-day	1.0 mg/L	EPA 1992d
10-day	0.3 mg/L	EPA 1992d
Longer term		EPA 1992d
Adult	1 mg/L	EPA 1992d
Child	0.30 mg/L	EPA 1992d
Lifetime	0.22 mg/L	EPA 1992d
EPA Ambient Water Quality Criteria		
(Ingestion of water and aquatic		EPA 1992b
organisms)	1.01 mg/L	EPA 1992b
Water or fish consumption	100 μg/L	

6.3.9 PHENOLIC COMPOUNDS OTHER THAN PCP

A phenolic compound is one that contains a hydroxyl (-OH) group attached to an aromatic ring. Chlorinated phenols represent a group of commercially produced substituted phenols and cresols referred to as chlorophenols and chlorocresols. Most chlorophenols are synthesized by the direct chlorination of phenol. As a group, chlorophenols are characterized by a odor described as unpleasant, medicinal, pungent, phenolic, strong or persistent (Sittig 1980). Trichlorophenols and tetrachlorophenols have been used as fungicides, wood preservatives, and bactericides (Deichman and Keplinger 1981). Phenols with nitro (-N) groups on the aromatic ring are referred to as nitrophenols and those with methyl substitutions (-CH3) are methylphenols.

Toxicokinetics

Phenols, in general, are readily absorbed following oral, inhalation, and dermal exposure. In human subject approximately 90 percent of orally administered phenol, and 13 percent of dermally applied phenol, was absorbed based on urinary excretion. Distribution is rapid, with the largest fraction

found in the liver (ASTDR 1989c). Chlorophenols are metabolized and excreted as conjugates of sulfate and glucuronic acids (Deichman and Keplinger 1981). They are excreted rapidly via the urine regardless of the route of exposure. For example, rats exposed to 2,4,6-trichlorophenol in the diet eliminate approximately 80 percent of the dose in urine and 20 percent in feces (EPA 1980d).

It is important to note that it is unlikely that an individual would be exposed to a single phenolic compound. For example, there are six isomers of dimetheylphenol and it is usually found as a mixture of all isomers (EPA 1980d).

Qualitative Description of Health Effects

In rats high oral doses of the chlorophenols produce similar signs of poisoning. Restlessness and an increased rate of respiration appear a few minutes after administration of 2- and 3-chlorophenol, followed rapidly by motor weakness. Tremors, clonic convulsions which can be induced by noise or touch, difficult breathing, and coma set in promptly and continue until death. Similar signs are produced by 4-chlorophenol, but the convulsions are more severe. In the rat, monochlorophenols produce injury to the kidneys with red blood cell casts in the tubules, fatty infiltration of the liver, and hemorrhages in the intestines (Deichman and Keplinger 1981).

Dichlorophenols and trichlorophenols also produce these signs but, decreased activity and motor weakness do not appear quite so promptly. The tremors are much less severe, but may continue until a few minutes before death. Tetrachlorophenols produce signs similar to those caused by the mono-, di, and trichlorophenols, except that tremors and convulsions probably are due to asphyxia or hypoglycemia which result from a different mechanism (inhibition of oxidation phosphorylation) than those noted with the lower chlorinated phenols.

From the work of several investigators, it can be concluded that the increasing the chlorination of phenol results in a reduction of the convulsant action but increases inhibition of oxidative phosphorylation. Pentachlorophenol, for example, does not produce convulsion.

Dermatoses, including photoallergic contact dermatitis, have been reported in man after exposure to 2,4,5-trichlorophenol, chloro-2-phenylphenol, and tetrachlorophenols. Effects included elevated

lesions of the skin, blackheads, sebaceous cysts, and marked hyperkeratosis (Deichman and Keplinger 1981). A condition known as chloracne can occur following dermal exposure to several chlorinated phenols, especially those contaminated with dioxin.

2-Chlorophenol There is little information available on the acute or chronic effects of 2-chlorophenol in humans. This compound upon contact may produce skin and eye irritation. 2-Chlorophenol is considered to be an uncoupler of oxidative phosphorylation and a convulsant via acute exposure. Among the species tested by various routes of acute exposure the toxic effects observed are very similar. Effects observed are similar to those reported following chlorophenol exposure. Autopsies of animals have revealed kidney and liver damage as well as hemorrhages in the intestines.

No information is available on the mutagenic potential of 2-chlorophenol, nor are there data on reproductive or teratogenic effects of 2-chlorophenol in humans or laboratory animals (EPA 1980d).

4-Chlorophenol There are no reports on the effects associated with acute or chronic exposure to 4-chlorophenol in humans. Effects in animal studies are similar to those exhibited following chlorophenol exposure. There is no information of the mutagenic, reproductive or teratogenic effects of 4-chlorophenol.

2,4-Dichlorophenol 2,4-Dichlorophenol is a skin and eye irritant (ATSDR 1990d). Effects observed in animal studies are the same as those following exposure to chlorophenols. Few studies are available that address the mutagenic potential of 2,4-dichlorophenol in mammalian systems. In one assay this compound did not exhibit mutagenic potential with or without activation (EPA 1980h).

2,4,6-Trichlorophenol Exposure to trichlorophenol causes a response similar to that of other chlorophenols. 2,4,6-Trichlorophenol appears to be a weak mutagen based on the results of several mutagenicity studies using different cell cultures (EPA 1992b).

2-Methylphenol No specific information is available, but toxicity is believed to be same as observed with other isomers (Deichmann and Keplinger 1981). (See 4-methylphenol).

4-Methylphenol Physiologic response to this compound is similar to those produced by phenol. High dose acute exposures by all routes may cause muscular weakness, gastrointestinal disturbances, severe depression collapse, and death. Although the effects are primarily on the central nervous system, edema of the lungs and injury of the kidneys, liver, pancreas, and spleen may also occur. Methylphenols have a marked corrosive action on tissues, producing burns and dermatitis. 3-Methylphenol is generally considered the least toxic isomer; however, it is unclear whether 2- or 4-methylphenol is the more toxic of these two isomers. Although all three isomers are considered to have the same general degree of toxicity (Deichmann and Keplinger 1981). Three cresol isomers produced positive results in genetic toxicity studies both alone and in combination (EPA 1992b).

2,4-Dimethylphenol 2,4-Dimethylphenol is considered to be a strong dermal irritant with exposure resulting in dermatitis based dermal studies in laboratory animals. Clinical signs of poisoning from 2,4-dimethylphenol exposure include dyspnea, motor coordination disturbances, onset of rapid clonic spasms, and asymmetrical body position. Most of the animals exhibiting these symptoms died within 24 hours (EPA 1980i). 2,4-Dimethylphenol was negative in a reverse mutation assay with *E. coli* (EPA 1987b). There is a lack of chronic toxicity data, as well as reproductive and developmental studies with this compound.

Quantitative Description of Health Effects

2-Chlorophenol There are no data available on the carcinogenic potential of this compound in humans. Two initiation-promotion trials in animals found an association between 2-chlorophenol exposure and a high incidence of papillomas. It is unclear what the primary carcinogenic effects of the compound are since the assays were designed to study promoting activity (EPA 1980g).

The EPA has developed an oral reference dose (RfD) based on a 10 week study of 2-chlorophenol treatment (Exon & Koeller 1982 in EPA 1992b). Weanling female rats were exposed to 0, 5, 50, or 500 ppm of 2-chlorophenol in drinking water. The rats were bred after 10 weeks of treatment, which continued during breeding, gestation, and weaning. An increase in the conception rate and in the number of stillborns as well as a decrease in litter size was observed in the rats exposed to 500 ppm, which was chosen as the LOAEL. No effects were observed at 50 ppm which yielded a dose of 5 (mg/kg-day). An uncertainty factor of 1,000 was applied to account for interspecies extrapolation,

intraspecies variability and for the use of subchronic data to estimate a RfD of 0.005 (mg/kg-day). Confidence in the oral RfD was noted as being low because the study evaluated only reproductive and hematological effects from the oral subchronic study. No inhalation RfC is available at this time (EPA 1992b).

4-Chlorophenol There are no data available on the carcinogenic potential of 4-chlorophenol in humans and it has not been tested in laboratory animals (EPA 1992b). The EPA states that available data are inadequate for quantitative derivation of an RfD or RfC.

2,4-Dichlorophenol There are no data available on the carcinogenic potential in humans. One study involving two trials of 2,4-dichlorophenol in mice found it to be similar to phenol in promoting activity. However, no statistical analysis or dose-response data were included to support this comparison. No data are available for laboratory animals on the primary carcinogenic potential of this compound (EPA 1980h).

The EPA has developed an oral reference dose (RfD) based on a 1985 study by Exon & Koeller (EPA 1992b). Female rats were exposed to 3, 30, or 300 ppm 2,4-dichlorophenol in drinking water from weaning age through breeding at 90 days, birth, and weaning of pups. Pups were randomly selected from the exposed groups and administered 2,4-dichlorophenol for an additional 15 weeks. Increases in serum antibody levels were found to be treatment related. The increase was statistically significant in the high-dose group as were increases in spleen and liver weights. Exon & Koeller reported that exposure of dams to 300 ppm dichlorophenol resulted in a significant decrease in litter sizes. The NOEL for the study was determined to be 3 ppm or 0.3 (mg/kg-day). An uncertainty factor of 100 was applied to account for extrapolation from animal data to humans and for protection of sensitive human subpopulations. the immunological functions measured in the study are not commonly used endpoints in deriving human health risk. The confidence in the oral RfD was therefore noted as being low. No inhalation RfC is available at this time (EPA 1992b). This substance has not been evaluated by the EPA for evidence of human carcinogenic potential (EPA 1992b).

2,4,6-Trichlorophenol The EPA has classified 2,4,6-trichlorophenol as a probable human carcinogen and placed it in weight-of-evidence Group B2. This value was based on sufficient evidence in animals

which demonstrated increased incidences of lymphomas or leukemias in male rates and hepatocellular adenomas or carcinomas in male and female mice. The study was performed by the National Toxicity Program (NTP) which administered 2,4,6-trichlorophenol in the diet of male and female mice. Make mice received 5,000 or 10,000 ppm for 105 weeks, female mice received 10,000 or 20,000 ppm of 2,4,6-trichlorophenol in feed. Rats were also exposed to 2,4,6-trichlorophenol at 5,000 or 10,000 ppm in feed for 106 or 107 weeks. Animals were observed to have decreased body weights and doses were lowered at week 38. Dose-related decreases in mean body weight were reported but no increase in mortality nor other toxic signs were observed. In males but not females, there were significant dose-related increases in lymphomas or leukemias over controls. In both males and females there were statistically significant trends in the incidence of combined hepatocellular adenomas and carcinomas. Additional studies were considered to derive the carcinogenic slope factor (CSF) for 2,4,6-trichlorophenol. Adequate numbers of animals were observed for their lifetime. The inhalation CSF was based on data from the oral study (EPA 1992b).

2-Methylphenol A RfD was established for this compound based on a 90-day oral subchronic neurotoxicity study in rats (EPA 1986a). Sprague-Dawley rats were gavaged daily with 0, 50, 175, or 600 (mg/kg-day). Critical effects reported were decreased body weights, neurotoxicity, and mortality. A NOAEL and LOAEL were derived from this study, 50 and 150 (mg/kg-day), respectively. An uncertainty factor of 1000 was applied to achieve the oral RfD of 5×10^{-2} mg/kg-day) (EPA 1992b).

The EPA has assigned this compound a cancer weight-of-evidence classification of Group C - possible human carcinogen. This is based on an increased incidence of skin papillomas in mice in an initiation-promotion study. However, these data are not adequate for quantifying cancer slope factors (EPA 1992b).

4-Methylphenol A cancer slope factor was not derived for this compound but the EPA has classified this compound as a Group C carcinogen, a possible human carcinogen. This was based on inadequate human carcinogenicity data and limited animal data. An increased incidence of skin papillomas was observed in mice in an initiation-promotion study. Exposure was to a mixture of methylphenol isomers with benzene or polycyclic aromatic hydrocarbons (EPA 1992b).

The oral RfD has been withdrawn for this compound pending further review by the RfD/RfC Work Group (EPA 1992b).

2,4-Dimethylphenol There are no data available on the human carcinogenic potential of 2,4-dimethylphenol and the EPA has not evaluated this compound.

The RfD for 2,4-dimethylphenol was derived from a 90-day gavage study in mice (EPA 1992b). 2,4-Dimethylphenol was administered to male and female mice by gavage at doses of 5, 50, or 250 (mg/kg-day). Toxicology relevant clinical signs observed after week six in the high-dose group of both genders included squinting, lethargy, prostration, and ataxia with onset shortly after dosing. Statistically significant hematological changes were observed. Blood urea nitrogen (BUN) levels were significantly below vehicle controls in female mid- and high-dose groups. The LOAEL and NOAEL for this study were 250 and 50 (mg/kg-day) respectively. An uncertainty factor of 3000 was used in deriving a RfD. A value of 10 was applied for inter- and intraspecies variability and 30 for lack of chronic toxicity data, data in a second species and reproductive/developmental studies. An additional 14-day gavage study was also performed. Reported results included lethargy, prostration, and ataxia in male and female mice in the 250 (mg/kg-day) dose group. This corresponded with the results from the previous study. Confidence in the study is medium since it examined appropriate endpoints and identified both a LOAEL and a NOAEL. An inhalation reference concentration is not available at this time. This compound has not been evaluated by the EPA for evidence of human carcinogenic potential. Some data are available to indicate that 2,4-dimethylphenol may exhibit tumor-promoting activity and may act as a whole carcinogen on mouse skin (EPA 1987b).

2,4-Dinitrophenol The oral RfD is based on the incidence of cataracts in exposed human. Over 100 anecdotal cases of cataracts resulting in therapeutic use of 2,4-dinitrophenol (Horner 1942). Data were insufficient to allow for calculation of a NOEL. However, cataracts were observed in patients receiving as little as 2 mg/kg-day. An uncertainty factor of 1000 was applied to the LOAEL of 2 mg/kg-day and confidence in the oral RfD is listed as low (EPA 1992b).

p-Nitrophenol A risk assessment for this compound is under review by the EPA work group. The derivation of an inhalation reference concentration was determined to be inadequate. This compound has also not been evaluated by the EPA for evidence of human carcinogenic potential (EPA 1992b).

2,3,5,6-Tetrachlorophenol In this analysis 2,3,4,6-tetrachlorophenol (TCP) is used as a surrogate for 2,3,5,6-tetrachlorophenol (for which there are no toxicity criteria). The RfD is based on a subchronic gavage study in which rats received 0 to 200 mg\kg-day 2,3,4,6-TCP for up to 90 days (EPA 1986d). Numerous health effects were reported at 200 mg/kg-day. These included decreased body weights, increased liver and kidney weights, and centrilobular hypertrophy in livers (also observed in the 100 mg/kg-day dose group). Based on the NOAEL of 25 mg/kg-day and an uncertainty factor of 1000, the oral RfD of 3×10^{-2} was developed (EPA 1992b).

Summary of Criteria

Source: EPA 1992b

2-Chlorophenol

Oral Reference Dose	5×10^{-3} (mg/kg-day)	EPA 1992b
EPA Ambient Water Quality Criteria - Aquatic Health		
Freshwater Acute	$9.7 \times 10^{+2}$ µg/L	
Freshwater Chronic	none	

2,4-Dichlorophenol

Oral Reference Dose	3×10^{-3} (mg/kg-day)	EPA 1992b
EPA Ambient Water Quality Criteria - Human Health		
Ingestion of water and aquatic organisms	3,090 µg/L	
Ingestion of water only	3,090 µg/L	
EPA Ambient Water Quality Criteria - Aquatic Health		
Freshwater Acute	2,020 µg/L	
Freshwater Chronic	365 µg/L	

2,3,4,6-Tetrachlorophenol

Oral Reference Dose	3×10^{-2}	

2,4,6-Trichlorophenol

EPA Classification	B2	EPA 1992b
Oral Cancer Slope Factor	1.1×10^{-2} (mg/kg-day)$^{-1}$	EPA 1992b
EPA Ambient Water Quality Criteria - Human Health		
Ingestion of water and aquatic organisms	1.2 µg/L	
Ingestion of aquatic organisma only	3.6 µg/L	
EPA Ambient Water Quality Criteria - Aquatic Health		
Freshwater Acute	none	
Freshwater Chronic	$9.7 \times 10^{+2}$ µg/L	

2-Methylphenol

Oral Reference Dose	5×10^{-2} (mg/kg-day)	

4-Methylphenol

Oral Reference Dose	5×10^{-2} (mg/kg-day)	EPA 1992b
EPA Classification	C	
Oral Cancer Slope Factor	not available	

2,4-Dimethylphenol

Oral Reference Dose	2×10^{-2} (mg/kg-day)	EPA 1992b
EPA Ambient Water Quality Criteria - Aquatic Health		
Freshwater Acute	$2.12 \times 10^{+3}$ µg/L	
Freshwater Chronic	none	

2,4-Dinitrophenol

Oral Reference Dose	2×10^{-3}	EPA 1992b

6.3.10 POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic aromatic hydrocarbons (PAHs), are a class of compounds consisting of two or more fused aromatic (benzene) rings. They form as a result of incomplete combustion of organic compounds or by the partial breakdown of hydrocarbon compounds due to ultraviolet radiation. PAHs are commonly found as components of coal tar, soot, vehicle exhaust, creosote, refuse and wood burning emissions, and petroleum oils (EPA 1984n).

There are over one hundred different PAH compounds, but only a few have been adequately characterized toxicologically. Information in this profile has been summarized from the ATSDR profile on PAHs (ATSDR 1990a) and other sources, as indicated.

Toxicokinetics

PAHs are absorbed through the lungs, gastrointestinal tract, and skin. The rates of absorption vary among the different compounds and are also affected by the type of material in which the PAH is carried (e.g. water, food, oil compounds). Limited information indicates that PAHs absorbed from

the lungs or gastrointestinal tract distribute primarily to soft tissues including the lungs, liver, kidney, and fatty tissue. There is little distribution of dermally absorbed PAHs.

Metabolism of PAHs occurs in all tissues. Enzymatic activity, however, varies among tissues and affects the degree of metabolism and bioavailability of PAHs. The primary method of metabolism is via oxidation by microsomal enzymes. PAHs are known enzyme inducers, that is, they cause enhanced enzymatic activity by increasing the rate of enzyme synthesis.

Excretion of PAHs following inhalation exposure is reportedly rapid. The larger portion is excreted in the feces for both inhalation and oral routes of exposure.

Qualitative Description of Health Effects

Several PAHs, especially those with four or more benzene rings, have been established as carcinogens in animals. Among the most potent and best studied carcinogenic PAHs is benzo(a)pyrene (B(a)P). A significant amount of knowledge of toxicologic actions of PAHs is based on extrapolation of studies with B(a)P to other carcinogenic members of the class. PAHs are carcinogenic in various species and by all routes of exposure. In most cases (e.g., after dermal exposures), tumors develop both at the site of contact and systemically.

Metabolism plays a critical role in carcinogenesis induced by PAHs. These compounds are activated to "ultimate" carcinogens, which can react directly with DNA, via mixed function oxidase enzymes in many tissues. Differences in metabolic capabilities probably is the basis for many differences in sensitivity to carcinogenic effects of PAHs both among species and among organ systems.

Although PAHs are among the more potent animals carcinogens found in tobacco smoke, the presence of many other potential etiologic compounds in smoke makes it impossible to determined the quantitative association between PAH exposure and lung cancer in humans. A similar argument can be made for other complex mixtures containing PAHs that have been associated with increased cancer incidence (e.g., soot, coal tar). Thus, data on human cancer are indirect and weak. On this basis, the EPA has classified several PAHs as Group B2 carcinogens (B(a)P, Indeno[1,2,3-c,d]pyrene, dibenzo[a,h]anthracene, chrysene, benzo[k]flouranthene, benzo[b]flouranthene, and

benzo[a]anthracene) indicating sufficient evidence for carcinogenesis in animals, but inadequate evidence in humans.

Because potency factors for the other carcinogenic PAHs have not been developed, the potency factor for B(a)P is used as a basis for potency factors for other carcinogenic PAHs. This is accomplished by using Toxicity Equivalence Factors for PAHs that were developed by one agency of the EPA (Bennett 1989i).

Carcinogenesis assays using lower molecular weight PAHs have been generally negative, and many of the compounds have been classified into Group D — Not Classified (acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene). However, chronic high doses of Group D PAHs can produce toxicity in renal, hepatic, and hematologic systems, and RfDs have been developed for most of the above. In addition, several PAHs, notably, pyrene, act as cancer promoters or co-carcinogens in animal studies. Thus, especially since Group D compounds are usually present in much greater quantities than the Group B2 agents, exposure to mixtures of PAHs may pose more risk exposure to single compounds.

Quantitative Description of Health Effects

Toxicological investigation of complex mixtures such as PAHs is difficult and does not always identify which compound(s) is responsible for an adverse effect. Of the PAHs tested individually, B(a)P has been the most extensively tested. These test results, combined with other empirical data and structure activity relationships with other PAHs, indicate that B(a)P probably has the greatest carcinogenic potential of any chemical in this class. Therefore, one EPA agency has used cancer potency estimates for B(a)P as a "benchmark" to determine relative carcinogenic potential for other PAHs (Bennett 1989). Studies on the carcinogenicity of B(a)P are discussed in the following paragraph.

Neal and Rigdon (1967) administered B(a)P in the diet at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, or 100 ppm to Swiss mice. Treatment time was variable up to a maximum of 197 days. Stomach tumors were observed in mice receiving 20 ppm or more B(a)P. Brune et al. (1981) administered B(a)P to Sprague-Dawley rats by caffeine gavage resulting in annual doses of 6, 18, or 9 mg/kg.

Untreated and gavage controls were included. There was a statistically significant association between dose and the proportions of rats with tumors of the forestomach, esophagus, or larynx. These data were used to derive an oral slope factor of 7.3 (mg/kg-day) based on the geometric mean from all four data sets (male and female rats and mice) (EPA 1992b).

An interim inhalation slope factor of 6.1 (mg/kg-day) was derived based on a study by Thyssen et al. (1981) (EPA 1992c). Hamsters were exposed to B(a)P at concentrations of 2.2, 9.5 or 45 mg/m^3 for up to 675 days. Hamsters receiving the mid-dose developed tumors of the nasal cavity, larynx, trachea, and pharynx, while those in the highest dose group developed tumors of the upper digestive tract. It should be noted that these are provisional slope factors that have not yet undergone final review by the EPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) work group.

No slope factors are available for dermal exposure to PAHs. Further it is not appropriate to extrapolate slope factors from oral exposure to the dermal route for two reasons. First, as discussed above, the skin is a major target organ for carcinogenic effects of PAHs following dermal exposure. Route of entry effects compromise route-to-route extrapolation, and EPA (1989a) uses benzo(a)pyrene as an example of a chemical for which route of entry effects preclude the use of the oral slope factor.

Second, the skin is also a site of metabolism of PAHs. Thus, even for chemical absorbed into the blood stream, the form of the chemical, and hence its biological activity, may be altered. Thus, it is not appropriate to consider quantitatively risks for internal cancers based on absorption estimates from dermal exposure. Dermal absorption is generally measured using radioactive tracers which do not provide an indication of the form of the chemical which reaches the blood stream.

For the above reasons, quantitative evaluation of toxicity of PAHs following dermal exposure is not appropriate, and such exposures need to be addressed qualitatively. Further discussion of risks due to dermal exposures to PAHs is provided in Section 7.6.13.

As previously stated, one EPA agency, the Office of Emergency and Remedial Response (OERR), has developed "toxicity equivalency factors" to rank the relative carcinogenic potential of other PAHs relative to B(a)P. In this evaluation, the approximate distribution of PAHs by carcinogenic potency is as follows:

- Category 1 — PAHs known or suspected to have high or moderately high carcinogenic potential (9 percent);
- Category 2 — PAHs known or suspected to have weak or marginal to moderate carcinogenic potential (21 percent);
- Category 3 — PAHs known or judged to be non-carcinogenic (70 percent).

OERR has proposed assigning slope factors equivalent to those for B(a)P for category 1 PAHs, equivalent to 0.01 B(a)P for all category 2 PAHs, and zero for all category 3 PAHs (Bennett 1991). These toxicity equivalence factors are listed in Table 6-3.

EPA has developed oral reference doses (RfDs) for several of the noncarcinogenic PAHs. These RfDs and associated references are listed on Table 6-4.

Summary of PAH Criteria		Source
EPA Carcinogen Classification	B2	EPA 1992b
Oral Carcinogenic Potency Factor (B(a)P)	$7.3 \times 10^{+0}$ (mg/kg-day)$^{-1}$	EPA 1992c
Inhalation Carcinogenic Potency Factor (B(a)P)	$6.1 \times 10^{+0}$ (mg/kg-day)$^{-1}$	EPA 1992d
Proposed Maximum Contaminated Level (MCL)	0.0002 mg/L	EPA 1992d
Proposed Maximum Contaminant Level Goal (MCLG)	Zero	EPA 1992b
Ambient Water Quality Criteria (AWQC)	Not available	

6.3.11 ZINC

Introduction

Zinc is a naturally occurring element in water, soil and air, which is used predominantly in the manufacture of metal alloys. Zinc is also used in fluorescent screens, manufacture of pigments and as a photo conductor in copying machines.

Zinc is released into the environment by metallurgic smelter and refining operations. Upon release, zinc tends to absorb to soil and sediment. Transport in water is therefore limited and contamination is generally restricted to areas close to release sources.

TABLE 6-3

ESTIMATED TOXICITY EQUIVALENCE FACTORS AND POTENCY ESTIMATES FOR PAHs

Chemical	Relative Potency[a] (Chu/Chen 1984)	EPA Classification	TEF (OSWER)	Resulting OSWER Potency (oral slope factor) $(mg/kg/day)^{-1}$
Benzo(a)anthracene	0.0134	B2[b]	0.01	7.3×10^{-2}
Benzo(b)fluoranthene	0.0800	B2	1.0	7.3
Benzo(k)fluoranthene	0.0044	B2	0.01	7.3×10^{-2}
Benzo(a)pyrene	1	B2	1.0	7.3
Benzo(g,h,i)perylene	ND	D	0.01	7.3×10^{-2}
Chrysene	0.0012	B2	0.01	7.3×10^{-2}
Dibenz(a,h)anthracene	0.6900	B2	1	7.3
Indeno(1,2,3-cd)pyrene	0.0171	B2	0.01	7.3×10^{-2}

[a] Relative to BaP
[b] Probable human carcinogen

TABLE 6-4

ORAL RfDs FOR PAHs

Compound Status	Exposure	Species	Critical Effect	Uncertainty Factor	Modifying Factor	Reference Dose	Reference
Acenapthene/Verified (11/15/89)							
	175 $(mg/kg\text{-}day)^{-1}$ daily by gavage for 90 days (NOAEL); 350 $(mg/kg\text{-}day)^{-1}$ (LOAEL)	Mouse	Hepatotoxicity	3,000	1	6E-2 $(mg/kg\text{-}day)^{-1}$	EPA 1989a
Anthracene/Verified (11/15/89)							
	1000 $(mg/kg\text{-}day)^{-1}$ daily by gavage for 90 days (NOEL)(HDT)	Mouse	No effects	3,000	1	3E-1 $(mg/kg\text{-}day)^{-1}$	EPA 1989b
Fluoranthene/Verified (11/15/89)							
	125 $(mg/kg\text{-}day)^{-1}$ daily by gavage via corn oil for 13 weeks (NOAEL); 250 $(mg/kg\text{-}day)^{-1}$ (LOAEL)	Mouse	Nephropathy, increased relative liver weights, hematological and clinical effects	3,000	1	4E-2 $(mg/kg\text{-}day)^{-1}$	EPA 1988
Fluorene/Verified (11/15/89)							
	Gavage via corn oil 125 $(mg/kg\text{-}day)^{-1}$ for 13 weeks (NOAEL); 250 $(mg/kg/day)^{-1}$ (LOAEL)	Mouse	Decreased RBC, packed cell volume and hemoglobin	3,000	1	4E-2 $(mg/kg\text{-}day)^{-1}$	EPA 1989c
Naphthalene							
	50 $(mg/kg\text{-}day)^{-1}$ in diet for 5 days/week for 13 weeks (35.7 $(mg/kg/day)^{-1}$)	Rat	Decreased body weight	10,000	1	4E-2 $(mg/kg\text{-}day)^{-1}$	NTP study (1980)

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TABLE 6-4 (Cont.)

ORAL RfDs FOR PAHs

Compound Status	Exposure	Species	Critical Effect	Uncertainty Factor	Modifying Factor	Reference Dose	Reference
Pyrene/Verified (11/15/89)							
	75 $(mg/kg\text{-}day)^{-1}$ by gavage via corn oil for 13 weeks (NOAEL)	Mouse	Nephropathy and decreased kidney weight	3,000	1	3E-2 $(mg/kg\text{-}day)^{-1}$	EPA 1989d

HDT = Highest Dose Tested

EPA. 1989d.
_____. 1989e.
_____. 1989f.
_____. 1989g.
NTP. 1980.

Toxicokinetics

Zinc is absorbed gastrointestinally, dermally and via the lungs. Gastrointestinal zinc absorption is more efficient in people with zinc-deficiencies than in people with adequate nutritional levels of zinc.

Zinc is the most abundant trace metal in humans and is distributed throughout the body. Zinc is toxic, however, when ingested in large amounts. Distribution of zinc is limited following ingestion of large doses, probably due to the fact that absorption of zinc decreases at high levels in the gastrointestinal system.

Elimination via the intestine is the predominant excretion mechanism for zinc in humans.

Qualitative Description of Health Effects

Inhalation of high doses of zinc chloride smoke released through the explosion of smoke generators and smoke bombs has reportedly caused death in humans (Evans 1945; Milliken et al. 1963; Hjortso et al. 1988). The common limiting factor in the above cited reports is, that while the fumes consisted predominately of zinc chloride, other substances may also have been present. It is therefore difficult to distinguish between the effects of the different chemicals. Inhalation of zinc oxide particles, which is commonly due to occupational exposures, has also been shown to elicit toxic effects in humans. Inhalation of zinc oxide is associated with respiratory system effects ranging from cough and nasal passage irritation (Sturgis et al. 1927) to metal fume fever (Brown 1988).

Ingestion of large doses of zinc oxide (850 mg/kg/day) was shown to cause mortality in ferrets (Straube et al. 1980). At doses, which are not fatal, gastrointestinal distress, alteration in gastrointestinal tissues and pancreatic abnormalities are commonly observed following ingestion of zinc (Samman and Roberts 1987). Ingestion of zinc has also been linked to anemia in humans (Moore 1978) and animals (Straube et al. 1980).

Additionally, hepatitic effects (necrotic hepatocytes) and renal effects (diffuse nephrosis) have been observed in animals upon oral exposure to zinc (Straube et al. 1980).

Quantitative Description of Health Effects

Zinc is a group D carcinogen (EPA 1992b). Chemicals in this group are not classifiable as to human carcinogenicity.

An oral Reference Dose (RfD) for zinc has been developed by EPA, but is currently being reviewed (EPA 1993). The RfD was based on a human exposure study (EPA 1992c). The RfD was determined from the lowest observed adverse effect level (with anemia being the adverse effect) after humans had been therapeutically exposed to zinc at doses of 2.14 mg/kg/day for an unspecified period of time. An uncertainty factor of 10 was assumed in the development of the RfD (EPA 1992c). The oral RfD is 0.2 mg/kg/day (EPA 1992c).

The lifetime Health Advisory (HA) for zinc is 2.1 mg/kg/day (EPA 1992c).

Summary of Zinc Criteria

EPA Carcinogen Classification	Group D	EPA 1992b
RfD (under review)	0.2 mg/kg/day	EPA 1992c
Health Advisories		
Lifetime	2.1 mg/kg/day	EPA 1992c

7

Section Seven

7.0 RISK CHARACTERIZATION

7.1 INTRODUCTION

In this section, chemical exposure values calculated in Section 5.0 are combined with toxicity values summarized in Section 6.0 to develop quantitative health risk estimates for exposure to Montana Pole site chemicals of concern. Both cancer and non-cancer health risks are evaluated for each of the exposure pathways that are likely to exist in the vicinity of the plant site. Risks from these exposure routes and pathways are then combined to provide a total estimate of carcinogenic and noncarcinogenic health risks for the site. Carcinogenic health risks are presented in Section 7.2, and noncarcinogenic risks are present in Section 7.3.

This section provides quantitative risk estimates for three groups of receptors exposed or potentially exposed to COCs from the Montana Pole site. The three receptor groups are current and future on-site trespassers, future on-site workers and future on-site residents. Trespassers are evaluated for dermal contact with and incidental ingestion of soils, surface water and sediments while visiting the site. Future on-site workers are evaluated for dermal contact with and incidental ingestion of soils. Future residents are evaluated for dermal contact with and incidental ingestion of soils, ingestion of groundwater used for domestic purposes and ingestion of home grown produce. Although all receptors are potentially exposed to contaminants in dust blown from the site, initial screening calculations suggest that this pathway is unlikely to contribute significantly to exposures and risks. Thus, no quantitative estimates of risks due to inhalation of site contaminants is provided.

7.2 CANCER RISK ESTIMATES

To evaluate potential cancer health risks related to the Montana Pole site, chemical exposures calculated in Section 5.0 are multiplied by cancer slope factors identified in Section 6.0 to develop upper range incremental lifetime cancer risks. Incremental cancer risks in the range of 10^{-6} to 10^{-4} may be characterized as acceptable by the EPA depending on the nature of the site and the COCs.

7.2.1 CURRENT ON-SITE TRESPASSERS

7.2.1.1 Incidental Ingestion of Soil

Estimated incremental lifetime risks for incidental ingestion of surficial soils range from 7×10^{-9} to 2×10^{-6}, with an aggregate risk of 4×10^{-6}. The largest components of these risks (83 percent) are from arsenic and PCP. Potential cancer risks from incidental ingestion of soil are presented in Table 7-1.

7.2.1.2 Dermal Contact with Soil

Incremental lifetime cancer risks for dermal contact with soil are presented in Table 7-2. These risks range from 4×10^{-9} to 2×10^{-6} for 2,4,6-TCP and arsenic, respectively. Two of the four chemicals evaluated had associated risks exceeding 1×10^{-6}, the lower limit of the EPA risk range. Total cancer risk for this pathway is estimated to be 1×10^{-5}.

As previously noted (Section 5.2.1.2), exposures and, hence, risks due to dermal exposure to PAHs were not quantified in this assessment. Data showing both dermal effects and metabolism of PAHs indicate that extrapolation of the oral cancer slope factor to dermal exposure is inappropriate (Section 6.3.10). Risks due to dermal exposure to PAHs are discussed qualitatively in Section 7.6.14.

7.2.1.3 Dermal Contact with and Incidental Ingestion of Surface Water

Incremental lifetime cancer risks for incidental ingestion of PCP in surface water (Silver Bow Creek) are estimated to be 3×10^{-6}. Estimated risks from ingestion of PAHs are about an order of magnitude less (4×10^{-7}). These risks reflect the conservative assumption that exposure occurs at the most highly contaminated surface water sampling location where NAPL seeps into the creek. Incremental lifetime cancer risks are summarized in Tables 7-3 and 7-4. Cancer risks for dermal contact with surface water are quantified for pentachlorophenol only. Estimated cancer risk for the trespasser scenario is 4×10^{-7}. Because of the conservative assumption that exposure point concentrations are equal to concentrations at the seep at SW-005, these estimates should be taken as worst-case, rather than reasonable maximums.

TABLE 7-1

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF SOIL FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg/day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	1.05E-05	1.20E-01	1.25E-06
Dioxins/Furans(TEFs)	4.29E-12	1.50E+05	6.44E-07
2,4,6-Trichlorophenol	4.89E-08	1.10E-02	5.38E-10
Benzo(a)pyrene(TEFs)	9.96E-10	7.30E+00	7.27E-09
Arsenic	1.07E-06	1.75E+00	1.88E-06
		Total Cancer Risk	3.79E-06
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	6.10E-05	3.00E-02	2.03E-03
Dioxins/Furans (TEFs)	2.50E-11	1.00E-09	2.50E-02
2,4,6-Trichlorophenol	2.85E-07	NA	NA
4-Chloro-3-methylphenol	1.46E-07	NA	NA
2-methyl-4,6-dinitrophenol	2.18E-06	NA	NA
Anthracene	9.76E-09	3.00E-01	3.25E-08
Arsenic	6.27E-06	3.00E-04	2.09E-02
Cadmium	1.51E-07	5.00E-04	3.02E-04
		Total Hazard Index	4.83E-02

NA = Not Applicable

TABLE 7-2

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH DERMAL CONTACT WITH SOIL FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	7.83E-05	1.20E-01	9.40E-06
Dioxins/Furans (TEFs)	3.21E-12	1.50E+05	4.82E-07
2,4,6-Trichlorophenol	3.66E-07	1.10E-02	4.03E-09
Arsenic	1.01E-06	1.75E+00	1.76E-06
		Total Cancer Risk	1.16E-05
		RfD (mg/kg-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	4.57E-04	3.00E-02	1.52E-02
Dioxins/Furans (TEFs)	1.88E-11	1.00E-09	1.88E-02
2,4,6-Trichlorophenol	2.14E-06	NA	NA
4-chloro-3-methylphenol	1.10E-06	NA	NA
Arsenic	5.87E-06	3.00E-04	1.96E-02
Cadmium	1.13E-07	5.00E-04	2.26E-04
		Total Hazard Index	5.38E-02

TABLE 7-3

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARDS INDICES ASSOCIATED WITH INGESTION OF SURFACE WATER FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	2.78E-05	1.20E-01	3.33E-06
Benzo(a)pyrene (TEFs)	5.96E-08	7.30E+00	4.35E-07
		Total Cancer Risk	3.77E-06
		RfD (mg/kg-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	1.62E-04	3.00E-02	5.40E-03
Pyrene	3.73E-07	3.00E-02	1.24E-05
		Total Hazard Index	5.41E-03

TABLE 7-4

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH DERMAL CONTACT WITH SURFACE WATER FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chronic Daily Intake CDI(mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	3.04E-06	1.20E-01	3.65E-07
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	1.77E-05	3.00E-02	5.90E-04

7.2.1.4 Incidental Ingestion of Creek Sediment

Estimated incremental lifetime cancer risks from ingestion of creek sediments are presented in Table 7-5. Incidental ingestion of dioxins/furans, the only chemicals of concern found in sediments, resulted in estimated cancer risks of 2×10^{-9}. This risk estimate is low even though this exposure pathway was assessed assuming exposure at the most highly contaminated sediment sampling location. This pathway is unlikely to be of concern for on-site trespassers.

7.2.1.5 Exposures to Inorganic Chemicals in Surface Water

The risk assessment for Lower Area One (CDM-FPC 1991) addressed risks to trespassers who frequent Silver Bow Creek in the vicinity of the Montana Pole site. The LAO assessment is based on a more extensive data set than that available for this BRA, and its findings are briefly summarized here.

Risks from ingestion of surface water ranged from 2×10^{-7} to 8×10^{-7} varying with location in the stream. These risks are lower than the lower end of the EPA risk range and at least two orders of magnitude less than those associated with organic COCs at SW-005. Risks from ingestion of surface water appear insignificant for this scenario.

7.2.2 FUTURE ON-SITE WORKERS

7.2.2.1 Incidental Ingestion of Soil

Table 7-6 presents estimated incremental lifetime cancer risks from incidental ingestion of soil. These risks range from 3×10^{-9} to 1×10^{-5}. The highest risk is from ingestion of soil containing arsenic which contributes 50 percent of risks. Ingestion of soil contaminated with PCP resulted in a risk estimate 1.5 times less (8×10^{-6}).

TABLE 7-5

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH SEDIMENT INGESTION FOR CURRENT ON-SITE TRESPASSERS

Chemical	Chronic Daily Intake CDI(mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Dioxin/Furans	1.64E-14	1.50E+05	2.47E-09
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Dioxin/Furans	9.59E-14	1.00E-09	9.59E-05

TABLE 7-6

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF SOIL FOR FUTURE ON-SITE WORKERS OF THE SOUTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg/day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	6.69E-05	1.20E-01	8.03E-06
Dioxins/Furans(TEFs)	2.75E-11	1.50E+05	4.12E-06
2,4,6-Trichlorophenol	3.13E-07	1.10E-02	3.44E-09
Benzo(a)pyrene(TEFs)	6.37E-09	7.30E+00	4.65E-08
Arsenic	6.87E-06	1.75E+00	1.20E-05
		Total Cancer Risk	2.42E-05
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	1.87E-04	3.00E-02	6.24E-03
Dioxins/Furans (TEFs)	7.69E-11	1.00E-09	7.69E-02
2,4,6-Trichlorophenol	8.76E-07	NA	NA
4-Chloro-3-methylphenol	4.49E-07	NA	NA
2-methyl-4,6-dinitrophenol	6.72E-06	NA	NA
Anthracene	3.00E-08	3.00E-01	9.99E-08
Arsenic	1.92E-05	3.00E-04	6.42E-02
Cadmium	4.63E-07	5.00E-04	9.27E-04
		Total Hazard Index	1.48E-01

NA = Not Applicable

7.2.2.2 Dermal Contact with Soil

Incremental lifetime cancer risks for workers exposed via this pathway ranged from 2×10^{-8} to 4×10^{-5}. For three of the four contaminants considered in analysis of this pathway, however, cancer risks exceed 1×10^{-6}. These compounds include PCP, dioxins/furans, and arsenic. PCP contributes 80 percent of the total risk. These results are presented in Table 7-7.

7.2.3 FUTURE ON-SITE RESIDENT

7.2.3.1 Ingestion of Groundwater

The more potent carcinogenic PAHs predominate in groundwater and, using the TEF approach, result in a risk of 3×10^{-2}. Dioxin/furan related risks are slightly higher than those for PAHs (1.1×10^{-1}), and both greatly exceed the EPA risk range (Table 7-8). Risks for pentachlorophenol are approximately 1×10^{-2}. Risk estimates for trichlorophenol and arsenic are at least an order of magnitude less and contribute little to overall estimates.

The risk estimates for PAHs and dioxins/furans are higher than the range which current risk models are capable of predicting. Thus, it may be more accurate to consider risk estimates of 1×10^{-2} and higher as "greater than 1 in 100." Additional discussion is provided in Section 7.6.4.

Groundwater beneath the site is clearly heavily contaminated. Risk estimates for groundwater ingestion far exceed the EPA risk range of 1×10^{-4} to 1×10^{-6}.

7.2.3.2 Incidental Ingestion of Soil

Incremental lifetime cancer risks for incidental ingestion of soil are presented in Table 7-9 for the southern area of the site. These risks range from 1×10^{-8} for 2,4,6-TCP to 3×10^{-3} for arsenic. For this pathway, risks exceed 1×10^{-6} for exposure to PCP, dioxins/furans, and arsenic. These results indicate that soils at Montana Pole site contain significant levels of all three contaminants with combined risks approaching the upper limit of the EPA risk range of 1×10^{-4} to 1×10^{-6}. Arsenic is a significant contributor to total risk (50 percent), but is not considered to be site-related.

TABLE 7-7

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH DERMAL CONTACT WITH SOIL FOR FUTURE ON-SITE WORKERS OF THE SOUTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	3.03E-04	1.20E-01	3.63E-05
Dioxins/Furans (TEFs)	1.24E-11	1.50E+05	1.86E-06
2,4,6-Trichlorophenol	1.42E-06	1.10E-02	1.56E-08
Arsenic	3.89E-06	1.75E+00	6.80E-06
		Total Cancer Risk	4.50E-05
		RfD (mg/kg-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	8.47E-04	3.00E-02	2.82E-02
Dioxins/Furans (TEFs)	3.48E-11	1.00E-09	3.48E-02
2,4,6-Trichlorophenol	3.96E-06	NA	NA
4-chloro-3-methylphenol	2.03E-06	NA	NA
Arsenic	1.09E-05	3.00E-04	3.63E-02
Cadmium	2.10E-07	5.00E-04	4.19E-04
		Total Hazard Index	9.97E-02

TABLE 7-8

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF GROUNDWATER FOR FUTURE ON-SITE RESIDENTS

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	9.06E-02	1.20E-01	1.09E-02
Dioxins/Furans(TEFs)	7.35E-07	1.50E+05	1.10E-01
2,4,6-Trichlorophenol	3.23E-03	1.10E-02	3.55E-05
Benzo(a)pyrene(TEFs)	4.23E-03	7.30E+00	3.09E-02
Arsenic	3.22E-04	1.75E+00	5.64E-04
		Total Cancer Risk	1.53E-01
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	6.57E-01	3.00E-02	2.19E+01
Dioxins/Furans (TEFs)	5.33E-06	1.00E-09	5.33E+03
2,4,6-Trichlorophenol	2.34E-02	NA	NA
PAH (Total non-carcinogen)	3.02E+01	4.00E-02	7.54E+02
2-chlorophenol	4.08E-03	5.00E-03	8.17E-01
Arsenic	2.36E-03	3.00E-04	7.86E+00
Copper	1.41E-02	4.00E-02	3.52E-01
Manganese	2.52E-01	1.00E-01	2.52E+00
Lead	3.00E-03	NA	NA
Chromium	2.87E-03	5.00E-03	5.73E-01
2,4-Dichlorophenol	9.94E-02	3.00E-03	3.31E+01
2,4-Dinitrotoluene	2.23E-02	6.80E-01	3.27E-02
4-Chloro-3-methylphenol	3.34E-02	NA	NA
2-Methyl-4,6-dinitrophenol	3.86E-02	NA	NA
2,3,5,6-Tetrachlorophenol	3.12E-01	3.00E-02	1.04E+01
		Total Hazard Index	6.16E+03

NA = Not Applicable

TABLE 7-9

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF SOIL FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	1.86E-04	1.20E-01	2.23E-05
Dioxins/Furans(TEFs)	7.64E-11	1.50E+05	1.15E-05
2,4,6-Trichlorophenol	8.70E-07	1.10E-02	9.57E-09
Benzo(a)pyrene(TEFs)	1.77E-08	7.30E+00	1.29E-07
Arsenic	1.91E-05	1.75E+00	3.35E-05
		Total Cancer Risk	6.74E-05
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	1.80E-03	3.00E-02	6.01E-02
Dioxins/Furans (TEFs)	7.40E-10	1.00E-09	7.40E-01
2,4,6-Trichlorophenol	8.44E-06	NA	NA
4-Chloro-3-methylphenol	4.32E-06	NA	NA
2-methyl-4,6-dinitrophenol	6.44E-05	NA	NA
Anthracene	2.89E-07	3.00E-01	9.62E-07
Arsenic	1.85E-04	3.00E-04	6.18E-01
Cadmium	4.46E-06	5.00E-04	8.92E-03
		Total Hazard Index	1.43E+00

NA = Not Applicable

For the northern area of the site, risks are higher, reflecting the larger exposure point concentrations. Risk estimates range from 5×10^{-8} for TCP to 1×10^{-3} for dioxins/furans. The latter contribute over 90 percent of the total carcinogenic risk for this pathway. The remaining risk is attributable mainly to arsenic. PCP is a relatively minor contributor (less than 1 percent).

Risks for this pathway and are provided in Table 7-10.

7.2.3.3 Dermal Contact with Soil

Incremental lifetime cancer risks for dermal contact with soil in the southern area range from 4×10^{-8} to 9×10^{-5}. The largest risks are attributable to PCP (80 percent). However, three of the four compounds considered had associated risks greater than 1×10^{-6}. These results are presented in Table 7-11.

In the northern area, risk estimates range from 2×10^{-7} to 6 to 10^{-4} for TCP and dioxins/furans, respectively. Nearly 90 percent of the total risk of 7×10^{-4} is attributable to dioxins/furans, and only 3 percent to PCP. Results are presented in Table 7-12.

7.2.3.4 Ingestion of Home-grown Vegetables

Incremental lifetime cancer risks from ingestion of home-grown vegetables in the southern area are presented in Table 7-13. The highest risk, 9×10^{-4}, is from ingestion of vegetables containing PCP. Other risks range from 5×10^{-6} for PAHs to 5×10^{-4} for arsenic.

High risks are estimated for this pathway for the northern area. Risks are highest for dioxins/furans (1×10^{-2}) and arsenic (2×10^{-3}). Total cancer risks are estimate to be approximately 1×10^{-2}. Results are presented in Table 7-14.

7.3 NONCARCINOGENIC HEALTH RISKS

To evaluate non-cancer health risks, chemical exposure is compared to one of several types of toxicity criteria to determine if the exposure is within a range of exposure which is unlikely to cause adverse

TABLE 7-10

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF SOIL FOR FUTURE ON-SITE RESIDENTS OF THE NORTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	3.61E-05	1.20E-01	4.33E-06
Dioxins/Furans(TEFs)	9.50E-09	1.50E+05	1.43E-03
2,4,6-Trichlorophenol	4.21E-06	1.10E-02	4.63E-08
Benzo(a)pyrene(TEFs)	4.74E-07	7.30E+00	3.46E-06
Arsenic	6.87E-05	1.75E+00	1.20E-04
		Total Cancer Risk	1.55E-03
Noncarcinogenic Exposure		RfD (mg/kg-day)	Hazard Index
Pentachlorophenol	3.50E-04	3.00E-02	1.17E-02
Dioxins/Furans (TEFs)	9.21E-08	1.00E-09	9.21E+01
2;4,6-Trichlorophenol	4.08E-05	NA	NA
4-Chloro-3-methylphenol	3.73E-05	NA	NA
2-methyl-4,6-dinitrophenol	8.34E-05	NA	NA
Anthracene	1.27E-06	3.00E-01	4.24E-06
Arsenic	6.66E-04	3.00E-04	2.22E+00
Cadmium	1.05E-05	5.00E-04	2.11E-02
		Total Hazard Index	9.44E+01

NA = Not Applicable

TABLE 7-11

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH DERMAL CONTACT WITH SOIL FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	7.84E-04	1.20E-01	9.41E-05
Dioxins/Furans (TEFs)	3.22E-11	1.50E+05	4.83E-06
2,4,6-Trichlorophenol	3.67E-06	1.10E-02	4.03E-08
Arsenic	1.01E-05	1.75E+00	1.76E-05
		Total Cancer Risk	1.17E-04
		RfD (mg/kg-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	6.84E-03	3.00E-02	2.28E-01
Dioxins/Furans (TEFs)	2.81E-10	1.00E-09	2.81E-01
2,4,6-Trichlorophenol	3.20E-05	NA	NA
4-chloro-3-methylphenol	1.64E-05	NA	NA
Arsenic	8.79E-05	3.00E-04	2.93E-01
Cadmium	1.69E-06	5.00E-04	3.39E-03
		Total Hazard Index	8.05E-01

NA=Not Applicable

TABLE 7-12

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH DERMAL CONTACT WITH SOIL FOR FUTURE ON-SITE RESIDENTS OF THE NORTHERN AREA

Chemical	Chronic Daily Intake (mg/kg-day)	Slope Factor (mg/kg-day)-1	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	1.52E-04	1.20E-01	1.83E-05
Dioxins/Furans (TEFs)	4.01E-09	1.50E+05	6.01E-04
2,4,6-Trichlorophenol	1.77E-05	1.10E-02	1.95E-07
Arsenic	3.62E-05	1.75E+00	6.33E-05
		Total Cancer Risk	6.83E-04
		RfD (mg/kg-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	1.33E-03	3.00E-02	4.43E-02
Dioxins/Furans (TEFs)	3.50E-08	1.00E-09	3.50E+01
2,4,6-Trichlorophenol	1.55E-04	NA	NA
4-chloro-3-methylphenol	1.42E-04	NA	NA
Arsenic	3.16E-04	3.00E-04	1.05E+00
Cadmium	3.99E-06	5.00E-04	7.99E-03
		Total Hazard Index	3.61E+01

NA=Not Applicable

TABLE 7-13

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF HOME-GROWN VEGETABLES FOR FUTURE ON-SITE RESIDENTS OF THE SOUTHERN AREA

Chemical	Total Vegetable Pathway CDI (mg/kg-day)	Slope Factor (mg/kg-day)	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	7.43E-03	1.20E-01	8.92E-04
Dioxins/Furans (TEFs)	7.20E-10	1.50E+05	1.08E-04
2,4,6-Trichlorophenol	1.90E-03	1.10E-02	2.10E-05
Benzo(a)pyrene(TEFs)	6.35E-07	7.30E+00	4.63E-06
Arsenic	2.65E-04	1.75E+00	4.64E-04
	Total Cancer Risk		1.49E-03
		RfD (mg/kd-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	1.62E+00	3.00E-02	5.39E+01
Dioxins/Furans(TEFs)	5.21E-09	1.00E-09	5.21E+00
2,4,6-Trichlorophenol	1.38E-02	NA	NA
Anthracene	7.97E-06	3.00E-01	2.66E-05
Arsenic	1.92E-03	3.00E-04	6.40E+00
Cadimum	7.03E-04	5.00E-04	1.41E+00
	Total Hazard Index		6.69E+01

NA=Not Applicable

TABLE 7-14

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARD INDICES ASSOCIATED WITH INGESTION OF HOME-GROWN VEGETABLES FOR FUTURE ON-SITE RESIDENTS OF THE NORTHERN AREA

Chemical	Total Vegetable Pathway CDI (mg/kg-day)	Slope Factor (mg/kg-day)	Incremental Lifetime Cancer Risk
Carcinogenic Exposure			
Pentachlorophenol	1.45E-03	1.20E-01	1.75E-04
Dioxins/Furans (TEFs)	8.89E-08	1.50E+05	1.33E-02
2,4,6-Trichlorophenol	9.31E-03	1.10E-02	1.02E-04
Benzo(a)pyrene(TEFs)	1.74E-05	7.30E+00	1.27E-04
Arsenic	9.65E-04	1.75E+00	1.69E-03
	Total Cancer Risk		1.54E-02
		RfD (mg/kd-day)	Hazard Index
Noncarcinogenic Exposure			
Pentachlorophenol	3.16E-01	3.00E-02	1.05E+01
Dioxins/Furans(TEFs)	6.44E-07	1.00E-09	6.44E+02
2,4,6-Trichlorophenol	6.75E-02	NA	NA
Anthracene	3.37E-05	3.00E-01	1.12E-04
Arsenic	6.99E-03	3.00E-04	2.33E+01
Cadimum	1.64E-03	5.00E-04	3.27E+00
	Total Hazard Index		6.81E+02

NA=Not Applicable

health effects. As discussed in the previous chapter, the toxicity criteria which are relevant to this risk assessment are the EPA Reference Doses (RfDs). The potential for noncarcinogenic health effects is evaluated by comparing a chemical-specific exposure level with a chemical-specific reference dose. This ratio of exposure to toxicity for a single chemical is called a hazard index (HI) and is calculated as follows:

$$\text{Hazard Index} = \frac{\text{CDI}}{\text{RfD}}$$

where: CDI = Chronic Daily Intake (mg/kg-day)
RfD = Reference Dose (mg/kg-day)

The hazard index assumes that there is a level of exposure (RfD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the CDI exceeds the RfD (i.e., CDI/RfD > 1), a potential for non-cancer health effects may exist.

The potential for toxic health effects for each of the exposure pathways quantitatively evaluated for this risk assessment is discussed below.

7.3.1 CURRENT ON-SITE TRESPASSER

7.3.1.1 Incidental Ingestion of Soil

Hazard indices from incidental ingestion of contaminated soil were all low. The highest HI for dioxins/furans was approximately 3×10^{-2}. HIs for incidental soil ingestion are listed in Table 7-1. Section 7.6.3 discusses uncertainties in the approach used in assessing dioxin/furan noncancer effects.

7.3.1.2 Dermal Contact with Soil

Hazard indices resulting from dermal contact for the on-site trespasser are all less than one. The highest are those for dioxins/furans and arsenic of 2×10^{-2}. That for PCP is only slightly lower. These results, presented in Table 7-2, indicate that it is unlikely that trespassers would experience increased risk of noncarcinogenic effects from exposure via this route.

7.3.1.3 Dermal Contact with and Incidental Ingestion of Surface Water

The hazard index resulting from incidental ingestion of PCP in surface water was 5×10^{-3}, indicating that no adverse effects would be expected by exposure via this route. The hazard index for ingestion of pyrene is significantly less. These hazard indices are summarized in Table 7-3.

Dermal contact with water considered only pentachlorophenol, since, as previously indicated, extrapolation of the oral reference dose to dermal PAH exposure is considered inappropriate. The HI is 6×10^{-4}, indicating that it is unlikely that any adverse non-cancer effects would result from such exposure. These HIs are presented in Table 7-4.

7.3.1.4 Incidental Ingestion of Creek Sediments

The hazard index resulting from ingestion of creek sediments is presented in Table 7-5. Ingestion of dioxin/furans by this pathway resulted in an estimated hazard index of 1×10^{-4}, indicating that noncarcinogenic adverse health effects are unlikely.

7.3.1.5 Exposures to Inorganic Chemicals in Surface Water

The risk assessment for Lower Area One (CDM-FPC 1991) addressed risks to trespassers who frequent Silver Bow Creek in the vicinity of the Montana Pole site. The LAO assessment is based on a more extensive data set than that available for this BRA, and its findings are briefly summarized here.

No hazard indices for individuals chemicals exceeded unity, and even total hazard indices summed across chemicals were significantly less than one for all stream locations. For ingestion of surface water, no significant risk of non-cancer effects is expected due to exposure to inorganic chemicals.

7.3.2 FUTURE ON-SITE WORKER

7.3.2.1 Incidental Ingestion of Soil

Hazard indices for incidental ingestion of contaminated soil are presented in Table 7-6. These were all significantly below their associated RfDs. The HI for dioxins/furans for this pathway is 8×10^{-2}, indicating little potential for noncancer adverse health effects.

7.3.2.2 Dermal Contact with Soil

Hazard indices for this pathway are low, the largest being approximately 4×10^{-2} for dioxins/furans, a reflection of the low estimated RfD used for dioxins/furans. These results indicate that noncarcinogenic adverse health effects are unlikely to occur in workers exposed via this pathway. These HIs are presented in Table 7-7.

7.3.3 FUTURE ON-SITE RESIDENTS

7.3.3.1 Ingestion of Groundwater

Virtually all risk for this pathway for threshold toxic effects (non-cancer health risks) can be attributed to dioxins/furans and PAHs. The hazard index for dioxins/furans alone is over 5,000 indicating that exposures are expected to exceed the "RfD" (1 pg/kg-day) by over three orders of magnitude. A HI of 754 is estimated for non-carcinogenic PAHs (see below for further explanation). PCP intakes are also expected to exceed the RfD significantly (HI = 22). Exposures to any of these compounds could result in adverse health impacts for future residential receptors. HIs at or slightly exceeding one are estimated for arsenic and manganese. All HIs are presented in Table 7-8.

It should be noted that all non-carcinogenic PAHs are evaluated together using the single RfD for naphthalene. This approach is taken since exposure to a number of PAH compounds is expected, many or even most of which are not evaluated due to lack of analytical data, lack of toxicity data, or both. Using the lowest RfD for non-carcinogenic PAHs is an attempt to compensate for non-evaluated PAHs.

In addition, one should note that CDIs for non-carcinogenic PAHs are over 1,000 times those of the carcinogenic PAHs. This indicates that not including the higher molecular weight PAHs in the estimation of HIs does not significantly effect results. The carcinogenic PAHs would have to have extraordinarily low RfDs (approaching 1×10^{-5} mg/kg-day) to contribute significantly to non-cancer risks.

7.3.3.2 Incidental Soil Ingestion

Hazard indices resulting from incidental ingestion of soil for the southern area are generally low. The highest hazard index is for dioxins/furans and is estimated to be 7×10^{-1}. This result is in part due to the low RfD estimate for dioxins/furans, as discussed in Section 7.6.3. All HIs for this pathway for on-site residents are presented in Table 7-9. Based on an additive HI of 1.4 and the conservative nature of the dioxin RfD, the likelihood of adverse effects should be low.

A similar pattern is found for the northern area, though hazard indices are generally higher. An HI of 90 is estimated for dioxins/furans, and of 2 for arsenic, as shown in Table 7-10. This would indicate that this pathway, in the northern area, could impose an increased risk for adverse health effects. See Section 7.6.2 for a discussion of uncertainties in the data base, particularly for dioxins in the northern area.

7.3.3.3 Dermal Contact with Soil

Hazard indices for this pathway in the southern area are all less than one. The HI of 0.3 for arsenic is the largest, and the total (0.8) suggests that adverse health effects are unlikely via this exposure route. HIs for dermal contact with soil for on-site residents are presented in Table 7-11.

A slightly higher HI of 35 is found for dioxins/furans for the northern area, as presented in Table 7-12. Risks for non-cancer health effects are higher for this area, predominantly from exposure to dioxins/furans.

7.3.3.4 Ingestion of Home-grown Vegetables

Hazard indices for ingestion of home-grown vegetables in the southern area are presented in Table 7-13. Some of these HIs are quite high with the largest resulting from uptake of PCP into vegetables (HI = 64). The next highest HI is for ingestion of arsenic contaminated vegetables (HI = 7). It is, thus, possible that consumption of home-grown produce could lead to non-cancer health effects. Similarly high HIs for dioxins/furans (HI = 722) and arsenic (HI = 27) are estimated for the northern area of the site (Table 7-14). Risks for non-cancer health effects are expected to be increased in both areas due to exposure to these COCs.

7.4 COMBINING RISKS ACROSS CHEMICALS AND PATHWAYS

7.4.1 ON-SITE TRESPASSERS

Ingestion of soil and surface water and dermal contact with these media and with sediments all contribute significantly to overall cancer risks from these pathways. Total cancer risks, summed across all pathways is estimated to be 2×10^{-5} (Table 7-15). Risks are almost totally due to exposure to PCP, dioxins/furans, and arsenic.

For non-cancer risk, adding HIs for dioxins/furans for ingestion of and dermal contact with soils accounts for approximately one-half. The total HI is 1×10^{-1}, suggesting that total exposure should not be expected to result in adverse noncancer effects.

7.4.2 FUTURE WORKER SCENARIO

Combining soil ingestion and dermal contact pathways for future workers suggests a total incremental cancer risk of 7×10^{-5} due mostly to exposure to PCP (64 percent of total risk) and arsenic (27 percent). The total HI for these pathways is approximately 0.3. The significant contributors are dioxins/furans and arsenic. Combined risks across pathways for this scenario are presented in Table 7-16.

Table 7-15

Summary of Estimated Total Risks
for Current On-site Trespassers

Chemical	Soil Ingestion	Dermal Contact with Soil	Sediment Ingestion	Surface water Ingestion	Dermal Contact with Surface Water
	Incremental Lifetime Cancer Risk				
Carcinogenic Exposure					
Pentachlorophenol	1.25E-06	9.40E-06	NA	3.33E-06	3.65E-07
Dioxins/Furans(TEFs)	6.44E-07	4.82E-07	2.47E-09	NA	NA
2,4,6-Trichlorophenol	5.38E-10	4.03E-09	NA	NA	NA
Benzo(a)pyrene(TEFs)	7.27E-09	NA	NA	4.35E-07	NA
Arsenic	1.88E-06	1.76E-06	NA	NA	NA
Total Cancer Risk	3.78E-06	1.16E-05	2.47E-09	3.77E-06	3.65E-07
			Total Cancer Risk for all Media		1.96E-05
Noncarcinogenic Exposure	Hazard Index	Hazard Index	Hazard Index	Hazard Index	Hazard Index
Pentachlorophenol	2.03E-03	1.52E-02	NA	5.40E-03	5.90E-04
Dioxins/Furans (TEFs)	2.50E-02	1.88E-02	9.59E-05	NA	NA
2,4,6-Trichlorophenol	NA	NA	NA	NA	NA
2-methyl-4,6-dinitrophenol	NA	NA	NA	NA	NA
Anthracene	3.25E-08	NA	NA	NA	NA
Arsenic	2.09E-02	1.96E-02	NA	NA	NA
Cadmium	3.02E-04	2.26E-04	NA	NA	NA
4-Chloro-3-methylphenol	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	1.24E-05	NA
Total Hazard Index	4.82E-02	5.38E-02	9.59E-05	5.41E-03	5.90E-04
			Total Hazard Index for all Media		1.08E-01

NA = Not Applicable

7.4.3 FUTURE RESIDENTIAL SCENARIO

Because of the overwhelming dominance of the groundwater pathway, combining of risks across pathways for the residential exposure pathway would not be informative. Contributions from other pathways are too small to significantly affect total risk estimates. Cancer risks are combined across carcinogenic chemicals for the groundwater pathway. Total risk for this pathway, and for this scenario, is estimated to be approximately 2×10^{-1}.

For non-cancer health effects, the dominance of two chemicals, dioxins/furans and phenols in groundwater suggest that combining of HIs across pathways would also not be informative. HIs from other pathways would not contribute significantly to risk. In addition, dioxins/furans and phenols do not affect the same organ systems at low doses and combining of HIs for these chemicals is not considered appropriate.

7.5 RISKS ASSOCIATED WITH EXPOSURE TO LEAD

Elevated levels of lead found in groundwater beneath the Montana Pole site are thought to be due to historical mining activities in Butte. However, because it was found in high concentrations in on-site wells, lead is evaluated in this BRA.

Risk due to exposure to lead cannot be assessed using standard methods because of the lack of a reference dose, reference concentration, and slope factor. The EPA feels that current data are insufficient for determination of a NOAEL and, thus, no reference dose or concentration should be derived. Further, the EPA feels that the primary threat to human health from lead exposure is subtle neurological effects in young children. For this reason, the EPA has refused to derive a cancer slope factor, despite lead's Group B2 status as a probable human carcinogen.

The only quantitative tool currently available for use in the assessment of lead risks is the Integrated Uptake Biokinetic (IUBK) model (EPA 1991a), or one of a number of permutations based upon the same principles. This model uses current information on the uptake to lead following exposure from different routes, the distribution of lead among various internal body compartments, and the excretion of lead to predict impacts of lead exposure on blood lead concentrations. The predicted blood lead

Table 7-16

Summary of Estimated Total Risks for Future On-Site Workers

Chemical	Soil Ingestion	Dermal Contact with Soil	Sediment Ingestion	Surface water Ingestion	Dermal Contact with Surface Water
	Incremental Lifetime Cancer Risk				
Carcinogenic Exposure					
Pentachlorophenol	8.03E-06	3.63E-05	NA	NA	NA
Dioxins/Furans(TEFs)	4.12E-06	1.86E-06	NA	NA	NA
2,4,6-Trichlorophenol	3.44E-09	1.56E-08	NA	NA	NA
Benzo(a)pyrene(TEFs)	4.65E-08	NA	NA	NA	NA
Arsenic	1.20E-05	6.80E-06	NA	NA	NA
Total Cancer Risk	2.42E-05	4.50E-05	0.00E+00	0.00E+00	0.00E+00
			Total Cancer Risk for all Media		6.92E-05
Noncarcinogenic Exposure	Hazard Index	Hazard Index	Hazard Index	Hazard Index	Hazard Index
Pentachlorophenol	6.24E-03	2.82E-02	NA	NA	NA
Dioxins/Furans (TEFs)	7.69E-02	3.48E-02	NA	NA	NA
2,4,6-Trichlorophenol	NA	NA	NA	NA	NA
2-methyl-4,6-dinitrophenol	NA	NA	NA	NA	NA
Anthracene	9.99E-08	NA	NA	NA	NA
Arsenic	6.42E-02	3.63E-02	NA	NA	NA
Cadmium	9.72E-04	4.19E-04	NA	NA	NA
4-Chloro-3-methylphenol	NA	NA	NA	NA	NA
Pyrene	NA	NA	NA	NA	NA
Total Hazard Index	1.48E-01	9.97E-02	0.00E+00	0.00E+00	0.00E+00
			Total Hazard Index for all Media		2.48E-01

NA = Not Applicable

concentrations can then be compared with target blood lead concentrations associated with subtle neurological effects.

Risks from ingestion of lead in groundwater are assessed using the IUBK model, version 0.6 (EPA 1991a). This model predicts blood lead concentrations in children 0-7 years of age, using a combination of exposure and biokinetic parameters. Since children are thought to be most susceptible to the adverse effects of lead, protection for this age group is assumed to also protect older individuals. Protection of young children is considered achieved when the model predicts that less than five percent of children will have blood levels greater than 10 μg/dL.

Use of the model is directed toward assessing the impact of lead in drinking water on blood lead levels. To accomplish this goal, the model was run in default mode to provide a baseline for comparison. The model predicts that average blood lead levels in a population with no unusual source of lead exposure will be 3.24 μg/dL with only 0.06 percent of the population with blood lead concentrations above 10 μg/dL.

In contrast, average blood lead levels increase to 5.18 μg/dL when the exposure point concentration for lead (34.2 μg/L) is input to the model. Almost three percent of the population is predicted to have blood lead concentrations above 10 μg/dL.

The potential impact of lead in groundwater on blood levels is significant, increasing average levels by almost 2 μg/dL and greatly increasing the predicted proportion of children with concentrations above 10 μg/dL. The impact on the high end of the distribution could be even greater since the default geometric standard deviation (GSD) in the model is less than that found in a recent study of lead exposure in children living in Butte. The GSD calculated from this study was 1.8. If this value is input to the model, 2.6 percent and 12 percent of children are predicted to have blood lead concentrations above 10 μg/dL for default and site-specific groundwater runs respectively.

Results of IUBK modeling are presented in Appendix D.

7.6 UNCERTAINTIES ASSOCIATED WITH RISK CHARACTERIZATION

7.6.1 UNCERTAINTIES IN TOXICITY CRITERIA

A potentially large source of uncertainty is inherent in the derivation of the EPA toxicity criteria (i.e. RfDs, and cancer slope factors). In many cases, data must be extrapolated from animals to sensitive humans by the application of uncertainty factors to an estimated NOAEL or LOAEL for noncancer effects. While designed to be protective, it is likely in many cases that uncertainty factors overestimate the magnitude of differences that may exist between human and animals, and among humans.

In some cases, however, toxicity criteria may be based on studies that did not detect the most sensitive adverse effects. For example, many past studies have not measured possible toxic effects on the immune system. Moreover, some chemicals may cause subtle effects not easily recognized in animal studies. The effects of lead on cognitive function and behavior at very low levels of exposure serve as examples.

In addition, derivation of cancer slope factors often involves linear extrapolation of effects at high doses to potential effects at lower doses commonly seen in environmental exposure settings. Currently, it is not known whether linear extrapolation is appropriate. Probably, the shape of the dose response curve for carcinogenesis varies with different chemicals and mechanisms of action. It is not possible at this time, however, to describe such differences in quantitative terms.

It is likely that the assumption of linearity is conservative and yields slope factors that are unlikely to lead to underestimation of risks. Yet, for specific chemicals, current methodology could cause slope factors, and, hence, risks, to be underestimated.

Use of the EPA toxicity criteria could either over or underestimate potential risks, but it is difficult to determine either the direction or magnitude of any errors. In general, however, it is likely that the criteria err on the side of protectiveness for most chemicals.

7.6.2 UNCERTAINTIES IN THE DATA BASE

The database has limitations as described in Section 4.5.2. For many chemicals, especially dioxins/furans, few sample data points are available for estimation of potential exposure concentrations. This could lead to either under- or over-estimation of risk. Dioxins/furans are emphasized since they contribute substantially to risks from incidental soil ingestion and ingestion of groundwater. The small numbers of samples, taken opportunistically, may bias exposure point concentrations for these compounds. Although there may be unidentified "hot spots" (areas with anomalously high contaminant concentrations) for dioxins/furans, there are probably also large areas of the site with little contamination. Data limitations are such that it may be advisable to place more emphasis on risk estimates for PCP for which data was much more extensive. PCP contributed substantially to overall site risks.

This may be especially true when considering risks associated with the northern portion of the site. Soil concentrations for this area are considerably influenced by high concentrations of OCDD and other congeners found in samples taken near the location of the old oil/water separator. These are probably not representative of the site as a whole, and might better be interpreted as indicative of a "hot spot".

7.6.3 UNCERTAINTIES IN QUANTITATIVE TOXICOLOGY FOR DIOXINS/FURANS

ARCO (1992) has provided a synopsis of current literature and professional judgement on the toxicologic properties of dioxins/furans. CDM does not agree with all of the conclusions of this document, but certainly recognizes the potential for the current dioxin/furan cancer slope factor to overestimate impacts of these compounds on human health. It is likely that, some time in the future, the dioxin/furan slope factor will be reduced. The magnitude of this reduction may be a factor of 3 to 10 or more, based on current information available to CDM. This agrees in a general way with the information provided by ARCO (1992). Risk managers may wish to keep these uncertainties in mind in evaluating the cancer risk estimates for dioxins/furans.

The "RfD" for dioxins/furans used in this assessment is basically a consensus value. ARCO (1992) presents the wide range of alternative values which have been adopted by various agencies and

countries. The range of values indicates the variety of interpretations of current information on the toxicology of dioxins/furans. The adopted value falls at the midpoint of the range which can be considered to be based on a threshold model for dioxin/furan toxicity. This range is appropriate for an "RfD", since the RfD methodology is based on the concept of a threshold for systemic toxicity. The midpoint value has been used by EPA (1990b) for assessing dioxin risks at pulp and paper mills. A value near 1 pg/kg-day seems reasonable for addressing threshold effects for dioxin/furan. Risk managers may wish to give more weight to HIs than to cancer risks for dioxins/furans in evaluating risks at the Montana Pole site.

7.6.4 RISK ESTIMATES EXCEEDING 1×10^{-2}

Risks for ingestion of contaminated groundwater exceed 1×10^{-2} for dioxins/furans and PAHs. These values exceed the ability of current risk models to predict cancer incidence and cannot be accepted as realistic estimates. For values exceeding 1 in 100 increased cancer risk, it should be assumed that, though risks are likely to exceed 1 in 100, quantification is inaccurate.

7.6.5 RISKS DUE TO INGESTION OF GROUNDWATER

Risks from groundwater ingestion should be considered hypothetical. The concentrations of PCP and other contaminants are high enough to greatly exceed sensory thresholds for taste and odor. This water would be unpalatable and actual exposure potential low, at least for ingestion. Risk estimates are, however, useful for providing an illustration of the severity of contamination, and for eventual generation of remediation goals.

In addition, groundwater concentrations beneath the site are highly variable. Actual risks due to ingestion of groundwater would likely depend heavily on well placement and pumping rates. It is conceivable that a well placed near the edge of the plume would pull in enough water to dilute contamination to below sensory thresholds. Such water might sill contain sufficient quantities of contamination to pose an unacceptable health risk.

7.6.6 LACK OF A GROUNDWATER INGESTION PATHWAY FOR THE WORKER SCENARIO

Groundwater exposure for the on-site worker was not addressed quantitatively. If an on-site well were installed, the high concentrations of contaminants beneath the site would likely pose an unacceptable risk. This exposure pathway was omitted from the worker scenario, since it was assumed that residential use of the aquifer does not require residential development of the land. It is conceivable that a well to supply all or part of drinking water to a future nearby home or development (i.e., trailer park) could be installed on or near the property even if the property itself was not developed. Stated in another way, it was assumed that water rights and development might be independent of either current or future land use. The residential scenario was, thus, deemed the most appropriate for assessing the groundwater pathway.

Risks for on-site workers, however, can be estimated from current assumptions. Differences in exposure scenarios between on-site residents and workers occur in exposure frequency, intake rate and body weight. Since the exposure and risk calculations are linear, worker exposure and risk (for carcinogenic effects) would be 250/350 x 1/2 x 59/70 x 1/0.7 or 0.4 times the exposures and risks for the residential scenario. This calculation assumes exposure frequencies of 250 and 350 days/yr, ingestion rates of 1 and 2 liters/day, body weights of 70 and 59 kg, and fractions of ingested water contaminated of 1 and 0.7 for worker and residential exposure scenarios respectively. Worker risk from ingestion of PCP would be estimated at 8×10^{-2} (0.4 times the total residential risk estimate of 2×10^{-1}). (See Section 7.6.4 for discussion of risks exceeding 1×10^{-2}).

7.6.7 LACK OF QUANTITATIVE ASSESSMENT OF SUBCHRONIC AND ACUTE EXPOSURES

Generally, site remediation is based on the more restrictive clean-up levels established for chronic exposures. Such exposures are the focus of this assessment. The high concentrations of contaminants found in surface soils and especially groundwater at the Montana Pole site suggest, however, that short-term exposures could be significant. Risks from short-term exposures could be important in determining the need for interim remedial actions to remove threats to public health before final remediation is implemented. In addition, risks from short-term exposures may be significant for

workers engaged in remedial activities at the site. Such exposures would be especially important if works might contact contaminated groundwater.

Since much of the heavily contaminated soil has been removed and stored on-site, and the area of this removal fenced, it seems likely that immediate threats to on-site trespassers have been addressed until a final remedy is implemented. Failure to address subchronic and acute exposures for this receptor population is, thus, unlikely to significantly underestimate risks for current visitors to the site, or those that may visit in the near future.

Further, there is no ready source of exposure to groundwater, except in the area of the SW/SD-005 sampling station where groundwater seeps into Silver Bow Creek. Here, concentrations of contaminants are greatly reduced and effects from short-term exposures seem unlikely. Exposure to more contaminated groundwater is also unlikely unless the shallow aquifer is tapped, for example, during implementation of additional remediation. Again, risks from short-term exposure to groundwater seem unlikely in the near future.

7.6.8 LACK OF QUANTITATIVE ASSESSMENT OF DERMAL ABSORPTION OF CONTAMINANTS WHILE SHOWERING WITH CONTAMINATED GROUNDWATER

Residential use of groundwater would entail not only ingestion of water, but also dermal contact while showering, washing dishes and hands, etc. For organic compounds such as PCP and dioxins/furans, dermal contact can lead to significant intake. Groundwater exposures and risks may be underestimated by failure to quantify this pathway for ingestion alone.

As discussed in Section 5.2.2.4, exposures via dermal absorption while showering are unlikely to exceed 0.3 times the exposures expected from drinking the same water (Maxwell et al. 1991), and may be significantly lower. Groundwater risk estimates may, therefore, be a maximum of about one third higher than those presented.

7.6.9 UNCERTAINTIES IN EXPOSURE ESTIMATES FOR RESIDENTS AND WORKERS

As discussed in Section 5.6.3, overall uncertainty in exposure assumptions is expected to be low. CDMs experience in combining uncertainties using quantitative methods (e.g., Monte Carlo simulation) suggests that deterministic estimates based on the RME concept often fall in the upper range of possible exposure estimates suggested by the simulation. Generally, deterministic estimates are within a factor of 10 and frequently within a factor of 2 or 3 of the 95th percentile of the quantitative analysis. Such findings indicate that the RME is likely to achieve its stated goals of estimating an exposure in the upper range of those possible.

7.6.10 UNCERTAINTIES IN EXPOSURE ESTIMATES FOR ON-SITE TRESPASSERS

Visitors to Silver Bow Creek may be exposed to site-related contamination near and downstream from the seep at SW/SD-005. Exposure to these visitors will be a complex function of time spent in the contaminated portion of the stream, the concentrations of contaminants in this reach of the stream, types of activities engaged in, the frequency of visits to the stream, and the number of years visiting the site remains attractive. Little or no data is available to quantitatively estimate any of these parameters. Therefore, this pathway is evaluated based mostly on professional judgement, and is subject to considerable uncertainty.

Since the water and sediment concentrations used to assess risk in this scenario were taken from the area of the seep, it may be reasonable to consider these estimates as upper bounds, rather than RMEs. The reach of the stream which is contaminated with organic compounds from the Montana Pole site appears to be limited (see discussion in Section 8.2.6), and concentrations in both surface water and sediment appear to diminish with distance down stream. Unless children were to play, swim and/or wade excessively in the area of the seep, their exposure is likely to be considerably less than that estimated.

For exposure to on-site soils, data are less limiting, but professional judgement must be used to estimate frequency of visits to the site, and the nature of activities likely to be engaged in. If children were to be attracted to the more contaminated areas of the site or visit the site more often than assumed, the estimates presented could underestimate actual risks.

One of the most contaminated areas on-site is currently fenced, and access to this area is apparently limited now and into the near future. For current site trespassers, opportunities for visiting contaminated areas is limited, and it seems less likely that they would receive exposures higher than those estimated. The risk estimates for this scenario probably do not overestimate risks, but the degree of conservativeness in these scenarios is difficult to determine.

7.6.11 ESTIMATES FOR DERMAL ABSORPTION AND PLANT UPTAKE FOR ARSENIC

The exposure point concentration of arsenic in this assessment is elevated, but is considered to reflect contamination moving onto the Montana Pole site from arsenic released during historical mining activities. Since this arsenic may be found in mineral forms and matrices that might limit availability for plant uptake, or for dermal absorption, it is likely that the intake estimates for arsenic for these pathways overestimate potential exposures. For both pathways, the exposure and risk estimates provided might be considered upper bound, rather than RME, estimates.

7.6.12 UNCERTAINTIES IN THE PRODUCE INGESTION PATHWAY

Potential risks from ingestion of produce grown in contaminated soil emphasized PCP. Risk estimates based on this COC are, thus, likely to be the most reliable for use in risk management for the site. As described in Appendix C, interpretation of pertinent literature on PCP was done conservatively, and it is believed that the resulting exposure and risk estimates for the produce ingestion pathway are unlikely to underestimate risks. However, due to the large uncertainty associated with PCP soil half-life, it is difficult to ascertain the degree of conservativeness in the estimates. If the actual soil half-life proves to be much less than that used in the assessment, risk could be greatly overstated.

7.6.13 USE OF SCREENING LEVEL DATA

Most data for the major site-related COCs contain a large proportion of enforcement quality data. Estimates for exposures to PCP and dioxins/furans especially are not likely to be greatly influenced by uncertainties in screening quality data.

For other COCs, such as the PAHs, a great deal of screening quality data had to be used to generate exposure point concentrations. Problems of high detection limits also compromise the usefulness of this data (Section 4.5.2.1). Exposure point concentrations for these compounds are assumed to be associated with high uncertainty. However, PAHs are relatively minor contributors to overall site-related risks. Unless exposure point concentrations are greatly underestimated, the effect of data problems for PAHs on the results of the risk assessment is probably small.

7.6.14 DERMAL ABSORPTION OF PAHS

No quantitative assessment of dermal exposure to PAHs was carried out. As discussed in Section 6.3.10, use of oral slope factor is considered inappropriate for dermal exposure to PAHs because of route of entry effects and metabolism. However, some PAHs are known animal carcinogens following dermal exposure, and mixtures containing large amounts of PAHs (e.g. soot) are known human carcinogens. Risks due to exposure to PAHs may, thus, be underestimated.

7.6.15 INTERPRETATION OF ARSENIC RISKS

Because of uncertainties in the metabolism of arsenic and the derivation of its oral slope factor, EPA (1992b) suggests that risk managers may choose to assume that arsenic risks are overestimated by up to an order of magnitude. Additional uncertainties related to bioavailability and exposure parameters for on-site trespassers have been identified above and elsewhere in the document. These uncertainties and the conclusion that arsenic contamination is not site-related could be used to support such consideration in risk management decisions for the Montana Pole site.

7.6.16 COCS IN OFF-SITE GROUNDWATER

Risks are not assessed for exposure to groundwater from wells downgradient, but off-site. No significant contamination from COCs related to operations at the Montana Pole site were found in off-site wells, except for those immediately adjacent to and on the fringes of the current plume. Wells currently outside the plume could be contaminated in the future, but no plume modeling has been done which would allow prediction of future contaminant concentrations in downgradient, off-site wells.

The very high concentrations of PCP and other COCs in the groundwater beneath the site suggest that future risks from ingestion of groundwater downgradient from the site could be substantial.

7.6.17 LACK OF RfDs AND SLOPE FACTORS FOR SOME COCs

Several phenolic compounds do not currently have RfDs and/or slope factors. Risks from exposure to these chemicals could not be addressed quantitatively. Phenolics other than PCP, however, are present in relatively small quantities. None are expected to be so toxic that the quantities found would contribute significantly to site-related risks. It seems unlikely that lack of toxicity values significantly effects the results of this assessment.

7.6.18 NO QUANTITATIVE ANALYSIS OF USE OF GROUNDWATER FOR IRRIGATION

Future on-site residents could use contaminated groundwater to irrigate home gardens. Contaminants could be taken into plant tissues and consumed. Failure to quantify this pathway may underestimate risks imposed by ingestion of home-grown produce. Since, next to ingestion of groundwater, this pathway contributes most to potential risks from ingestion of PCP, some caution is necessary in interpreting risks and establishing remediation goals. This is particularly true if the final decision for on-site remediation allows residential development, but not use of groundwater as drinking water. In this case, consumption of contaminated home grown produce could be the dominant source of exposure and risk. If a decision to restrict drinking water uses, but allow irrigation is contemplated, additional analysis of this pathway would be necessary.

7.6.19 USE OF UPPER CONFIDENCE LIMITS FOR EXPOSURE POINT CONCENTRATIONS

Exposure point concentrations were calculated as 95% upper confidence limits on geometric or arithmetic means (Section 5.4). The intention of this calculation is, in part, to ensure that uncertainty in the database is addressed in exposure estimates to ensure protectiveness. Calculation of upper confidence limits, however, is based on statistical procedures which can be highly unstable (see Section 5.4) and yield unrealistic exposure point concentrations. Moreover, current statistical procedures due not take into consideration the size of a site in determining exposure point concentrations. It is intuitively obvious that 60 samples taken over a one acre site are associated with

much less uncertainty in terms of generating an exposure point concentration than the same number taken on a 1000 acre site, even if the variability in the two data sets is the same. Because the Montana Pole site is relatively small (45 acres), only part of the site appears to be heavily contaminated, and relatively large numbers of samples are available for the major risk driver, PCP, for all media, upper confidence limits could overestimate the potential for exposure to PCP.

To determine the possible size of any over prediction, appropriate measures of central tendency in the different data sets can be compared to exposure point concentrations used in the BRA. Two such comparisons, for soil and groundwater, are provided below for PCP.

7.6.19.1 Soil

The exposure point concentration for PCP in soil in the southern area is 319 mg/kg, and is associated with a cancer risk of about 2×10^{-5} from incidental ingestion of soil by future on-site residents. The geometric and arithmetic mean concentrations in soils on-site are calculated as 1.3 and 83 mg/kg. These concentrations range from 246 to 3.8 times less than the exposure point concentration used. This in turn suggests that risks could be as low as 9×10^{-8} to 5×10^{-6}.

The high variability in the soil data for PCP results in an exposure point concentration significantly higher than either above measure of central tendency, even though the number of data points (74) is high. This could lead to an overestimate of potential exposures. One should recall, however, that calculation of exposure point concentrations in this BRA assumes that future human activity on the site will result in more or less random exposure to the sampled areas of the site. In fact, human behavior may be much different and result in more frequent activity in either more heavily or less heavily contaminated areas. Thus, risks need not be overestimated at all, or could be overestimated to an even greater extent.

7.6.19.2 Groundwater

The exposure point concentration for groundwater on-site is estimated to be 6,500 μg/L. Compared to the geometric mean of 200 μg/L, risks from future groundwater ingestion could be overestimated by a factor of 30. This suggests that risks could be as low as 5×10^{-3}.

Risks due to ingestion of groundwater are difficult to assess, however, because actual exposure point concentrations will reflect well placement and aquifer conditions, as well as concentrations of contaminants. For the placement of any given well, potential exposure point concentrations cannot easily be calculated. Thus, it is not possible to say with confidence whether potential future risks are under- or overestimated in the current risk assessment.

It should again be noted that risks from ingestion of groundwater should be considered hypothetical, since heavy contamination is expected to render this water unpalatable. Instead, the high exposures and potential risks should be used as an indication of the degree of aquifer contamination and as a basis for setting of remediation goals.

7.6.20 UNCERTAINTIES IN ASSUMPTIONS FOR FUTURE LAND USE

Current land use in the vicinity of the Montana Pole site is mixed. Thus, residential development of the site may not take place. In such case, risk estimates for the residential scenario would not be relevant. It is not possible to assign a probability to this event, however, and several factors discussed the letter preface to this document suggest that the possibility of residential development may not be negligible. Application of the risk estimates for the residential scenario to any cleanup decisions must be based on a qualitative evaluation of possible future land use.

7.7 SUMMARY

Risks for exposure to COCs are greatest for the groundwater pathway where only residential exposure is evaluated quantitatively. Estimated risks for future residents are higher than can be predicted by current risk assessment models (Sections 7.2.3.1). Consumption of produce grown in contaminated soil also contributes significantly to overall site risks (Section 7.2.3.4), although relative to ingestion of contaminated groundwater risks, contributions from this pathway are only about one percent as great for the southern area soils. For both pathways, virtually all risk is due to exposures to PCP and dioxins/furans.

Pathways related to direct exposure to soil are associated with significantly less risk for all exposure scenarios. Estimated risks for exposure to PCP, dioxins/furans and other COCs were either within or less than the EPA risk range of 1×10^{-4} to 1×10^{-6} (Sections 7.2.1.1, 7.2.2.1, and 7.2.3.2).

Risks due to exposure to surface water and sediment were lowest for all pathways evaluated (Sections 7.2.1.3 and 7.2.1.4). Exposures are assumed to occur only for on-site trespassers. The lower exposure frequencies and ingestion rates for these receptors serve to reduce potential risks.

For the major contributor to site-related risks, PCP, uncertainties are expected to be low, and exposures are thought to provide an acceptable estimate of the RME. For other COCs, uncertainties in available data, toxicity and exposures are expected to be higher. Particularly for dioxins/furans, exposure and risk estimates could be associated with considerable error. Based on current information, risk management decisions based on compounds other than PCP are more difficult to support scientifically.

8 Section Eight

8.0 ECOLOGICAL ASSESSMENT

8.1 INTRODUCTION

The objective of the ecological risk assessment (ERA) is to evaluate the potential effects of contaminated surface water, soils, sediments and groundwater from the Montana Pole NPL site on terrestrial and aquatic plants and animals. Protection of the non-human population, community, or ecosystem is the usual focus of ecological risk assessments. The lack of appropriate toxicity data for wildlife and other environmental receptors at the population level makes quantitative inferences at this level or above difficult. This assessment will, therefore, address effects on populations and communities in a qualitative fashion.

The ecological risk assessment is complementary to the human health risk assessment for this site. Many initial steps used to evaluate human risks are similar for assessment of ecological impacts. These include:

- Identification of potential receptors (e.g., wildlife, fisheries, and threatened and endangered species)
- Identification of valued habitats such as wetlands in the project area or off-site areas that could be affected by contaminant movement off-site
- Assessment of the potential for exposure; discussion of the toxicity of the site contaminants to potential receptors
- Characterization of the potential current and future risk or threat to the environment from contaminants at the site.

This assessment follows the most recent EPA guidance for performing ecological risk assessments at Superfund sites (EPA 1989b).

The format of this report is based upon the above guidance, and includes the following elements:

- Section 8.2 Ecological Exposure Assessment, which includes an identification of potential aquatic and terrestrial receptors, identification of potential exposure pathways, and a discussion of uncertainties associated with the exposure assessment

- Section 8.3 Ecological Toxicity Assessment, including toxicity profiles for the potential COCs for ecological receptors, and a discussion of the uncertainties associated with the toxicity assessment

- Section 8.4 Ecological Risk characterization, including a comparison of media-specific COC concentrations to benchmark toxicity values, a qualitative assessment of possible population and community level effects, discussions of risks to aquatic life, terrestrial wildlife, and vegetation, and a discussion of associated uncertainties; and

- Section 8.5 Summary of Ecological Risks.

8.2 ECOLOGICAL EXPOSURE ASSESSMENT

8.2.1 ENVIRONMENTAL SETTING

The environmental setting for the ecological and human health risk assessment is discussed in Section 2.0 of this document. Environmental factors that apply specifically to the ERA are discussed in more detail in the following subsections.

8.2.1.1 Surface Water

The primary surface water features within and adjacent to Montana Pole site include Silver Bow Creek, Blacktail Creek, and the Metro Storm Drain (MSD) (Figure 8-1).

The MSD generally follows the historic Silver Bow Creek channel from just below the Weed Concentrator (about 1.3 miles northeast of Montana Pole site) to its confluence with Blacktail Creek. Upper MSD is typically dry, except during snowmelt or precipitation events (CH2M Hill and Chen Northern 1990).

Several storm drain outfalls enter the MSD as it flows from the Weed Concentrator to its confluence with Blacktail Creek. These outfalls collect storm water from various areas on Butte Hill, and are also typically dry except during snowmelt or precipitation events. The MSD becomes a perennial surface water course near the middle reaches of the system, in the vicinity of Harrison Avenue, due to its interception of shallow alluvial groundwater in the area. Flows in the MSD near its mouth during non-runoff conditions are typically 0.4 to 0.5 cubic feet per second (MultiTech 1987).

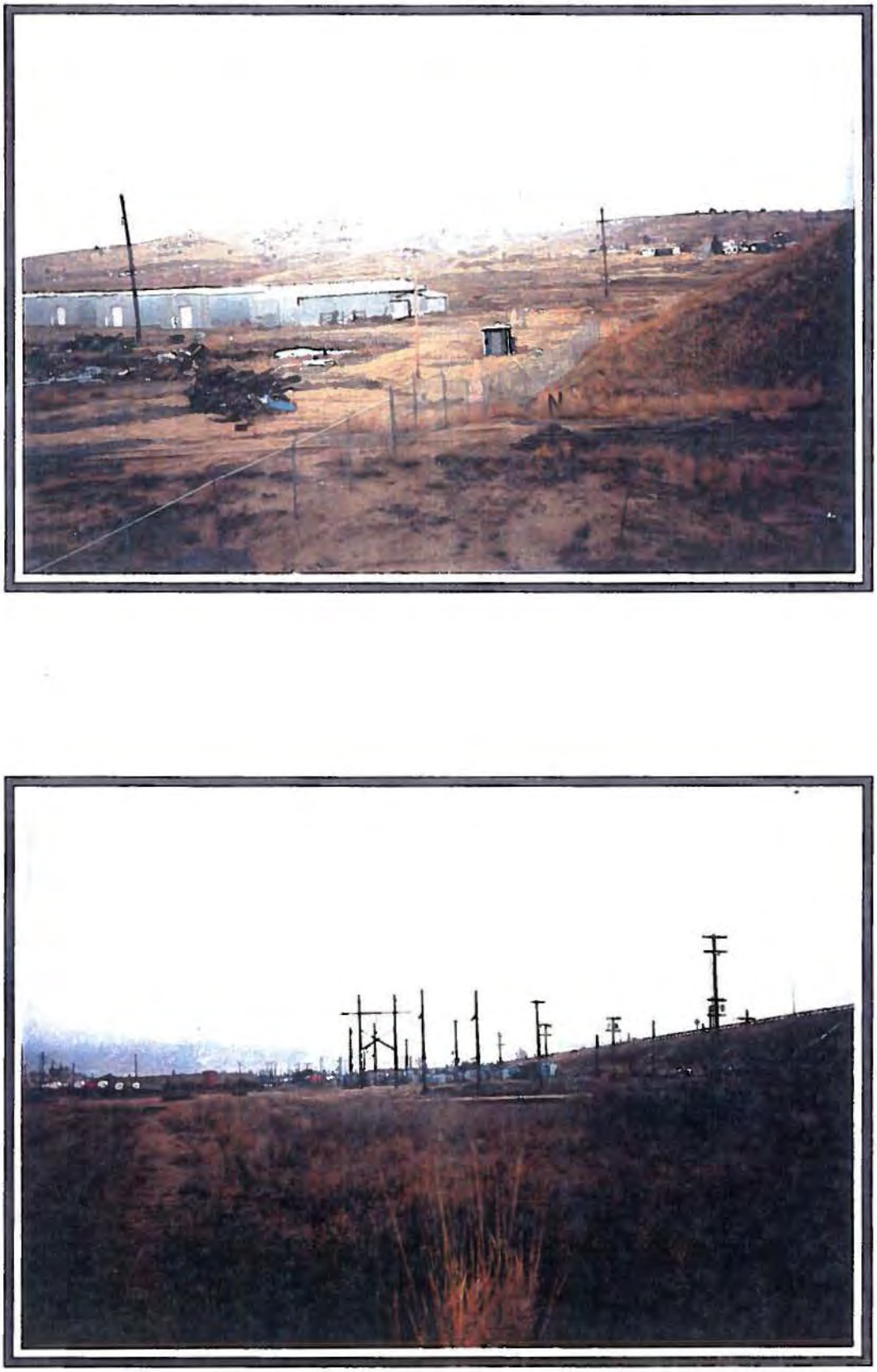


Figure 8-2 Typical Terrestrial Habitat at the Montana Pole Site

Blacktail Creek originates about 15 miles south of its confluence with the MSD in the Highland Mountains and drains approximately 75 square miles. Blacktail Creek is a perennial stream that supplies the majority of flow in modern-day Silver Bow Creek. Average flow in Blacktail Creek at its mouth is approximately 11.2 cfs.

Silver Bow Creek begins immediately east of the study area at the confluence of the MSD and Blacktail Creek, and is adjacent to historic mineral processing slag walls as it flows past the Montana Pole site.

Additional storm drain outfalls enter Silver Bow Creek below the confluence of Blacktail Creek and the MSD. Overland runoff from areas along the floodplain, including the Butte Reduction Works and the Colorado Tailings, enters the creek during local snowmelt runoff and precipitation events.

Flows in Silver Bow Creek, just below the Montana Pole site, have been recorded by the U.S. Geological Survey (USGS) since 1983. The average flow measured from 1983 to 1988 was 23.8 cfs; the highest recorded flow was 424 cfs on May 25, 1987, and the lowest recorded flow was 9.7 cfs on September 1 and 5, 1988.

There are no known domestic, agricultural, or industrial uses of surface waters within the Montana Pole site. However, surface waters serve as habitat for algae and benthic invertebrates in Silver Bow Creek. Aquatic biology investigations have shown that these organisms are present in the creek along the reach within this area (Chadwick and Associates 1985).

The flow regime of Silver Bow Creek at Montana Pole site can be divided into three general categories: spring snowmelt runoff, baseflow or low flow, and storm runoff due to localized precipitation events.

Spring snowmelt runoff occurs annually from approximately March 15 to June 15, and is due to seasonal melting of the snowpack in the mountainous upper watershed of Blacktail Creek. Average daily streamflows in Silver Bow Creek at the gaging station just west of the Colorado Tailings typically range from 26 to 33 cfs during this period. Major influences on water quality in Silver Bow Creek within the Montana Pole site during spring snowmelt runoff include:

- contributions from off-site sources, including Blacktail Creek, and the MSD;
- entrainment and solubilization of channel bank materials due to higher stream levels, both within and upstream from the Montana Pole site; and
- groundwater discharge to the creek.

Baseflow or low flow in Silver Bow Creek occurs annually from approximately June 15 to March 15. During this time, flow in Silver Bow Creek originates mainly from groundwater discharge, and average daily streamflows typically range from 18 to 22 cfs. Groundwater discharge to the creek, both within and upstream from the study area, is a major influence on water quality. Groundwater from beneath the Montana Pole site contributes to the organic pollutant load in Silver Bow Creek during both spring snowmelt runoff and baseflow conditions.

Storm runoff is the result of thunderstorms, frontal rain storms, and localized snowmelt runoff that occurs during the winter months. Thunderstorms and frontal storms occur most frequently from May through August. May and June are the months of greatest precipitation in Butte, with average monthly precipitation of 1.7 and 2 inches, respectively. Storm runoff events often occur simultaneously with spring snowmelt runoff or baseflow, and can last from one half hour to a few days. During storm runoff, water quality in Silver Bow Creek within the Montana Pole site is likely to be impacted by areas on Butte Hill outside of the Montana Pole site, including areas of mine tailings, slag, and waste rock.

8.2.1.2 Wetlands

Emergent and shrub-scrub wetlands are the dominant wetland types within the Montana Pole site. Wetland areas are characterized by willows (*Salix sp.*), cattails (*Typha sp.*), rushes (*Juncus sp.*), emergent grasses and other hydrophytic vegetation (Figure 8-1). Wetlands are primarily located along Silver Bow Creek, which flows by the Montana Pole site. Isolated pockets of small wetlands less than 1 acre occur sporadically along the northern boundary of the site. Wetlands at the Montana Pole site are further described in Section 2.5.

8.2.1.3 Terrestrial Areas

Terrestrial habitat to support biological receptors at the Montana Pole site is very limited. Wood-treating operations have disturbed much of the original grassland that was present prior to Montana Pole site development. Presently terrestrial habitat consist of "slag" piles, open grassy areas along fences and buildings, and grassy areas adjacent to wetlands (Figure 8-2). These habitats probably support rabbits, field mice, and other small rodents.

8.2.2 POTENTIAL RECEPTORS

Aquatic Communities

Silver Bow Creek adjacent to the Montana Pole site and downstream to the Warm Springs Ponds does not support a fisheries population (FPC 1992). Westslope cutthroat trout (*Oncorhynchus clarki lewisi*) and bull trout (*Salvelinus confluentus*) are reported to have once been caught in the vicinity of Butte prior to intensive mining activities (Knudsen 1984). Phillips (1985) notes that prior to 1975, mining-related pollution (including raw sewage from mining camps, sedimentation, and elevated metals concentrations) in much of the upper Clark Fork Rivers drainage contributed to the system being incapable of supporting a viable fishery. In addition, he stated that excessive metals deposits still prevent the establishment of a fishery in Silver Bow Creek.

Five species of trout have been recorded within Silver bow Creek watershed and, therefore, are considered potential ecological receptors. These include the westslope cutthroat trout, rainbow trout (*Oncorhynchus mykiss*), brook trout, bull trout, and brown trout (*Salmo trutta*). Although no trout are found in Silver Bow Creek near the Montana Pole site, due primarily to historical contamination associated with mining activities, there is potential for trout to occur because they are present in tributaries to Silver Bow Creek. A viable aquatic community does occur in Blacktail Creek, a tributary to Silver Bow Creek, just above the site. Blacktail Creek contributes the larger flow of the confluence, and fish and other aquatic animals may move downstream into the study area. German Gulch, which is located downstream of Butte, is considered representative of relatively unimpacted streams in this area. The fishery in this creek may be useful as a biological measure for streams, that have not been substantially impacted by human activities.

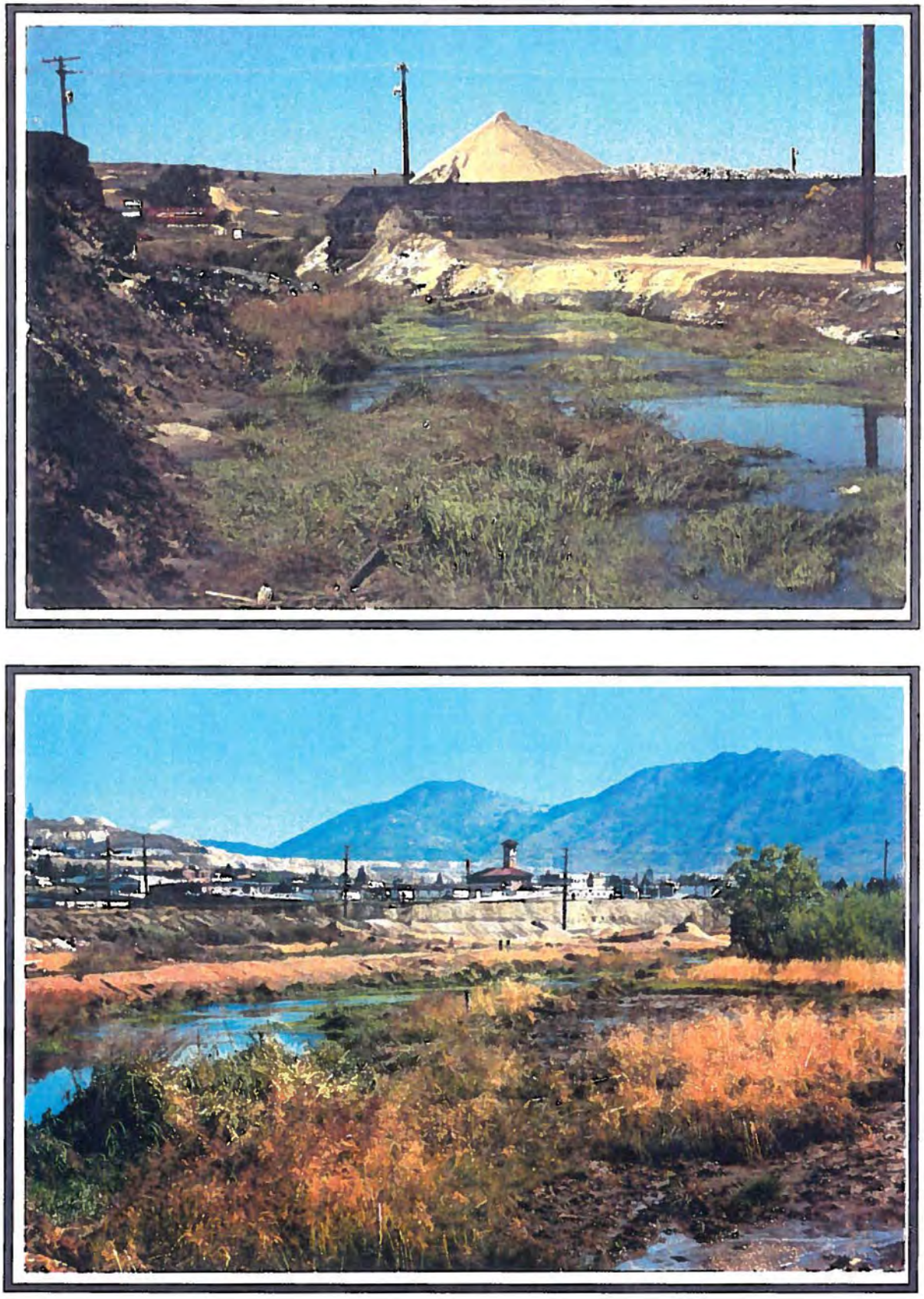


Figure 8-1 Wetland Areas Along Silver Bow Creek, Montana Pole Site

To evaluate the potential impacts to trout from the chemicals of concern in their environment, information on their life history is useful. Information on spawning, diet, and habitat are briefly summarized below.

Westslope Cutthroat Trout

Westslope cutthroat trout are classified as a Species of Special Concern by the State of Montana. This species has been strongly affected by introduced species and its range has been severely reduced. Pure strains of westslope cutthroat trout have been documented for only 25 Montana Streams, representing 1.1 percent of its historic range (MDFWP 1984).

Westslope cutthroat trout are adapted to cold, clear, oxygenated streams of moderate gradient. Many cutthroats now found are hybrids, probably a result of past stocking efforts, however, the Montana Department of Fish, Wildlife, and Parks (MDFWP) has discontinued stocking of streams in the state (MDFWP 1991). The nest (redds) of this species is similar to other trout species, in that a well-defined structure is constructed on the bottom of gravelly streams, and is abandoned after spawning (Lagler et al. 1977). This species spawns in the spring with benthic invertebrates comprising the majority of the fish's diet.

Brook Trout

Brook trout were introduced to Montana in the late 1800s from the eastern coast of North America. They thrive in cold, high elevation streams and are unable to tolerate the higher temperatures tolerated by brown, rainbow, and cutthroat trout. Brook trout, however, can tolerate a wider variety of chemical conditions including less fertile waters, and acid/alkaline and alkaline waters. Brook trout have a tendency to overpopulate an area, resulting in high numbers of fish that are generally of reduced size. In some areas, they may occur at densities up to 3,500 fish per acre (Varley and Schullery 1983).

Brook trout spawn in the fall and prefer gravel bottoms, but use a wider variety of substrates than other trout. They are short-lived, living only up to five years. They also apparently consume a wider variety of food that other trout, and their diet includes insects, other aquatic invertebrates, and

snails. Newly hatched fish usually maintain small feeding territories within a stream. In general, the males mature in the first year, and females mature in their second year. An 8-inch female produces about 800 eggs; 12-inch females can produce up to 1,200 eggs (Woodling 1984). The nest is similar to the westslope cutthroat trout, and is constructed in swift water that is usually less than one foot deep.

Brook trout do not occur in the vicinity of the Montana Pole site, but are found in tributaries to Silver Bow Creek. Brook trout were observed in Blacktail Creek upstream of the Montana Pole site, and in both German Gulch and Willow Creek, which empty into Silver Bow Creek downstream of the City of Butte.

Rainbow Trout

Rainbow trout were introduced into Montana during the same period that brook trout were introduced. They are native to the Pacific coast. Under ideal conditions rainbow trout can live up to eight years and may weigh up to five kilograms. They are the most adaptable of the trout species with respect to habitat conditions. Rainbows prefer clear, cold, rocky-bottom streams. Cover is extremely important and includes overhanging vegetation, submerged vegetation, undercut banks, instream objects, rocky substrates, and surface water turbulence. Trout production is highest in streams with pool-riffle ratios of 1:1. Flow regime is also very important, with average base flows being ideal if it is 50 percent of the average annual daily flow — less than 25 percent is considered poor. Siltation of spawning substrate is the single most destructive factor in affecting the survival of fish embryos.

Male rainbows reach maturity at two years of age; females at three years. Spawning occurs in the spring, although some hatchery varieties spawn in the fall. The next site is at the head of a riffle or downstream of a pool. The nest is typically longer and deeper than the female's body. The eggs are deposited and then covered with gravel and sand to an average depth of 15 cm. The number of eggs produced varies from about 400 to 3,000 depending on the female's size (Eddy and Underhill 1974). Eggs hatch in about one month at 51°F and in 18 days at 60°F. The yolk sac fry stay in the gravel about two weeks, until the yolk is used up (Varley and Schullery 1983).

Rainbow trout feed early in the morning and late in the evening. They are opportunistic feeders and feed on a wide variety of foods depending on what is locally available. In general, they consume more algae than other trout species, and they also feed at midwater and at the surface more than other trout species.

Rainbow trout have been observed in the lower portion of Silver Bow Creek, near the confluence with the Clark Fork River. They potentially could move up into the Butte area should water quality conditions improve in Silver Bow Creek.

Brown Trout

Brown trout are native to Europe and were introduced into Montana in the early 1900s. This species has been very successful and is abundant from high mountain streams to broad rivers flowing to the plains. Brown trout are able to tolerate higher temperatures than other trout and thus are found at lower elevations than other trout species. Most brown trout spawn in the fall, although some spawn in the spring. They move into small feeder tributaries in October and November in search of spawning beds with adequate gravel and rubble. The nest is a well-defined structure in the gravelly bottom of the stream and the eggs are covered with gravel after they are deposited (Raleigh et al. 1986).

The young feed primarily on aquatic insects, whereas the adults will also feed on other fish, as well as terrestrial insects, crustaceans, mollusks, and earthworms (Varley and Schullery 1983). Pisciveorous behavior generally occurs at about 3 years of age or about 12 inches. Adult trout are known to also consume crayfish, mice, frogs, birds, and snakes as part of their diet (Varley and Schullery 1983). Pools and deeper water are generally preferred by brown trout more than other trout species. Like rainbow trout, brown trout have been observed in the lower reaches of Silver Bow Creek, and have the potential to move upstream in response to improved water quality.

Bull Trout

Bull trout, which are similar to Dolly Varden (*Salvelinus malma*), are extremely predacious and rely mostly on smaller fish for a food source. Bull trout inhabit deep pools in large cold rivers and lakes,

and are more common in high mountain areas where snowfields and glaciers are present (Page and Burr 1991). The bull trout is also a Species of Special Concern in the State of Montana, and there is the potential for bull trout to move into Silver Bow Creek from the Clark Fork River (MDFWP 1991). Considerable effort is being expended by the MDFWP to increase the distribution of this species and the wetslope cutthroat trout in Montana.

Benthic Invertebrates

Benthic invertebrate communities have re-established themselves within the study area since the cessation of mine waste water discharges and other pollution sources associated with human activities to Silver Bow Creek. Mayflies, caddis flies, and stoneflies have been collected, although they demonstrate low density and limited diversity (Chadwick and Associates 1985). No known surveys on benthic communities have been conducted within the study area since about 1984. The current density and diversity of this aquatic community is unknown.

Aquatic Vegetation

Aquatic vegetation and algae have also been collected from the creek within the study area. Downstream of the study area boundary, the emergent aquatic grass *Alopecurus sp.* was abundant in 1984 (Chadwick and Associates 1985). Aquatic vegetation, both upstream and downstream of the Montana Pole site, has recently been observed in abundance by CDM representatives.

Terrestrial Communities

No communities within the Montana Pole site have been identified as critical habitat or communities of special concern. No rare or endangered plants were identified within the study area boundaries of the Lower Area One (LAO) Operable Unit of the Silver Bow Creek NPL site, nor downstream of this study area (IntraSearch Engineering, Inc. 1984). Vegetation growing adjacent to Silver Bow Creek within the Montana Pole site is limited to willows (*Salix exigua*) and grasses. Shrubs indicative of dry conditions are found throughout the area.

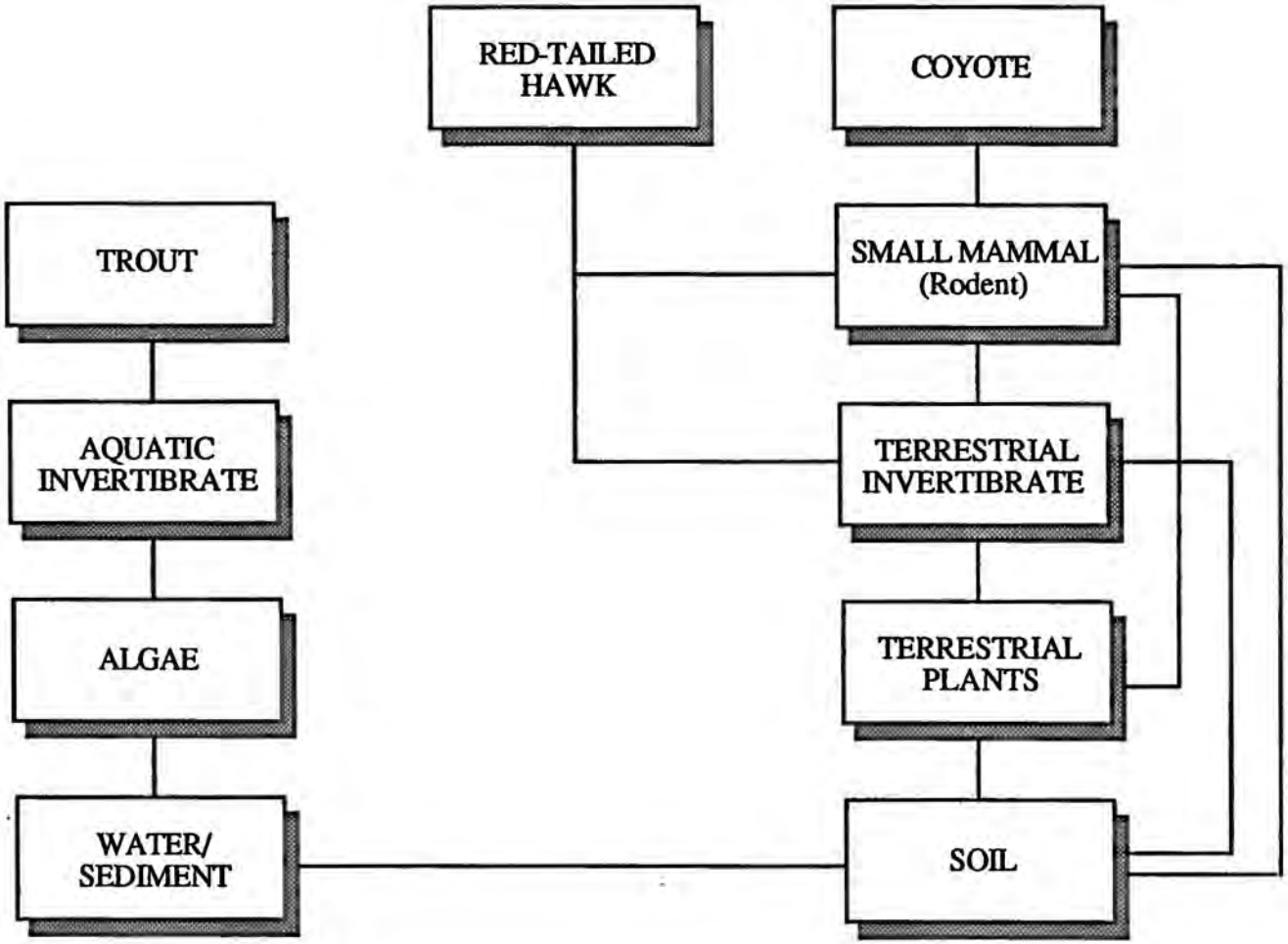
The U.S. Fish and Wildlife Service has stated that there are no threatened or endangered wildlife species present in LAO or in the near vicinity, which would include the Montana Pole site (Harms 1986). Although no wildlife surveys have been conducted in the area, it is anticipated that wildlife typical of disturbed and rural residential areas would be found. This could include medium-sized mammals such as rabbits and foxes, and small mammals that are commonly found in disturbed areas such as field mice and rats. It is possible that burrowing animals live in the area, although most of the area is continually subjected to disturbances from human activity. Domestic animals such as cats and dogs could also come onto the site due to its close proximity to residential areas.

Birds may be exposed to chemicals indirectly by ingesting contaminated food. A large portion of the diet of smaller birds consists of insects such as grasshoppers and crickets. Exposure via this pathway is likely to be of most concern to those species that consume food items that tend to bioaccumulate or bioconcentrate contaminants. Examples are hawks, which rely on rodents (e.g., field mice), and ducks which rely on aquatic species for a portion of the dietary intake. Predators using the habitat around the Montana Pole site exclusively may be at greatest risk as they may be exposed both to contaminated biota and to contaminated drinking water.

Downstream of the study area, as impacts from human activities decrease, larger mammals such as deer and coyote may be found.

8.2.3 EXPOSURE PATHWAYS

An environmental exposure pathway is the means by which chemicals are transported from a source to an ecological receptor. In this risk assessment, the source of potential contamination is from the Montana Pole site. This risk assessment is limited to evaluating contamination from this site. Contaminants can be present in soil, surface water, sediments, groundwater, and air at the Montana Pole site. Figure 8-3 shows potential environmental pathways and routes of exposure for ecological receptors at the site. It is assumed that exposure pathways for ecological receptors will be from ingestion of contaminated surface water, soils, vegetation and prey. Inhalation exposure was not considered to be an important exposure pathway for biota within the Montana Pole site. As discussed in more detail in Section 5.2.1.1, the air pathway contributes little to overall exposures for human receptors. This is expected to be true for non-human receptors as well.



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Exposure Pathways and Potential Biological Receptors for the Montana Pole Site**	Figure No. 8-3

In accordance with EPA guidance (EPA 1988c), sediment and surface water are considered as an integrated exposure medium because of complex chemical equilibrium between these two media. Groundwater is considered a source for surface water contamination and is not considered directly in assessment of exposures.

Groundwater

The only direct ecological exposure pathway from contaminated groundwater is through uptake of contaminants by vegetation rooted in the shallow aquifer. However, in the Montana Pole site area, in places where vegetation exists, it does not appear to be in contact with the shallow aquifer. Therefore, this pathway is not considered complete and will not be evaluated.

Terrestrial animals that burrow or inhabit dens live near the surface, and it is also unlikely they would contact contaminated groundwater. Therefore, the groundwater exposure pathway for wildlife receptors is considered to be incomplete, and will not be discussed further.

Groundwater does discharge to surface water in the vicinity of sampling station SW-005 on Silver Bow Creek. Aquatic life may be exposed to contaminants in this area and downstream. This potential exposure is discussed in the next section.

Surface Water

Aquatic organisms in Silver Bow Creek could be exposed to contaminants in the water column or sediments. Contaminants have reached the creek via discharge of contaminated groundwater and, perhaps, by erosion of contaminated soils from the Montana Pole site. Exposure can occur when contaminated water is ingested or passes over gill surfaces of animals, or is taken up by plants. Animals may also be exposed indirectly by ingesting contaminated plants. Aquatic organisms from Blacktail Creek may enter Silver Bow Creek and be exposed in the same way as other organisms in Silver Bow Creek. Birds, mammals, amphibians, or reptiles could become exposed through the food chain through ingestion of aquatic organisms or insects that have incorporated contaminants, through ingestion of surface water as drinking water, or through dermal contact with contaminated surface

water while foraging for food. In addition, both wild and domestic animals downstream from the Montana Pole site could be exposed through the ingestion of surface water.

Riparian vegetation in contact with contaminated surface water could take up contaminants in solution. Terrestrial wildlife could then ingest plants that have taken up contaminants, thereby bioaccumulating these contaminants. Such bioaccumulation, however, is not expected to be great for the major organic chemicals of concern — PAHs and PCP. Wildlife also use the creek for a water resource, thereby ingesting contaminants present in solution or in suspension within the water column.

Contaminant migration pathways are discussed in Section 5.1. For the purposes of this risk assessment, the conclusion of this discussion is that contaminants from the Montana Pole site leave the site in surface water, either through erosion and runoff, or through precipitation and infiltration into the groundwater, followed by discharge of groundwater to surface water.

Soils

The site, disturbed by human activity, is sparsely vegetated with mainly grasses and weeds. Some animals will be deterred by the fencing which surrounds a portion of the Montana Pole site. Should any terrestrial animals venture into the area, they may be directly exposed to contaminants in the soil. Direct contact with contaminated soil and incidental ingestion could occur among dustbathing animals, such as many bird species. Indirect exposure of animals to contaminants in soil may occur via ingestion of grasses and other land plants that may have bioaccumulated contaminants. Incidental ingestion of soil is a possible exposure route for fastidious animals such as raccoons who may ingest soil while grooming; herbivorous animals such as rabbits who may ingest soil while feeding on plants; or seed-eating bird species who may ingest soil while foraging for seeds on the ground. As with other exposure routes, the importance of this exposure route varies from species to species because of behavioral differences. Populations of animals such as rabbits, which are both herbivorous and frequent groomers, may be more affected by contaminated soil than other populations which contact soil less often. Also of concern are domestic animals such as dogs and cats that may venture onto the site from nearby residential areas. Cats and dogs are likely to ingest some contaminated soil upon grooming of their coats. It is also possible for individuals coming in contact with these pets to come in contact with the contaminated soil as well.

Plants may be directly exposed to contaminants in soil via uptake through the roots. Chemicals may accumulate in various portions of different species of plants. Because plant uptake values vary greatly from species to species, exposures via this pathway are difficult to quantify. At the site, tailings material, which makes up a substantial portion of the surface material near the creek, is not favorable for plant growth, as evidenced by the sparse plant growth on the north end of the site along Silver Bow Creek.

8.2.4 SUMMARY OF POTENTIAL RECEPTORS AND EXPOSURE ROUTES

As stated previously, the direct groundwater pathway is considered to be incomplete and will not be evaluated. For the surface water pathway, potential receptors and exposure routes to constituents present in surface water at the Montana Pole site or leaving the site include:

- Riparian vegetation within and downstream of the study area
- Wildlife and livestock that use the creek as a source of water, either at the site or downstream of the site, including both resident and migratory species
- Wildlife that feed on riparian vegetation that may have bioconcentrated contaminants from surface water or groundwater
- Aquatic vegetation and benthic invertebrates present in the creek adjacent to the site and downstream of the site
- Fish and other aquatic organisms that may move from Blacktail Creek into Silver Bow Creek
- Wildlife that feed on aquatic vegetation or animals that may have bioaccumulated contaminants from surface water
- Wildlife that experience dermal contact with contaminated surface water while foraging for food

8.2.5 BIOCONCENTRATION, BIOACCUMULATION, AND BIOMAGNIFICATION

One approach to predicting risks to ecological receptors, is to determine the relationship between tissue concentrations of contaminants within organisms and tissue concentrations associated with adverse impacts. Particularly for animals, it may be useful to distinguish between a contaminant that

is taken directly from the abiotic environment and that which is "transported" through the food chain. Three terms are commonly used to make these distinctions: bioconcentration, bioaccumulation, and biomagnification. Food webs of potential importance for the Montana Pole site are provided in Figure 8-3.

Bioconcentration is defined as the results of exposure to the contaminant in the species' physical environment, such as the direct uptake of a contaminant by a fish in contaminated surface water. A unitless bioconcentration factor (BCF) describes the numerical relationship between the chemical concentration in the media and the chemical concentration in the organism.

Bioaccumulation is defined as the uptake of a contaminant as a result of exposure to the contaminant through the ingestion of contaminated vegetation or prey species. A unitless bioaccumulation factor (BAF) is the ratio of the concentration of contaminant in the organism to that in the vegetation or prey species. When this ratio exceeds one, bioaccumulation of contaminants can increase exposure to contaminant for those species feeding higher in the food chain.

Biomagnification is defined as the total accumulation occurring in a food chain or food web. A unitless biomagnification factor (BMF) is the ratio of the concentration of contaminant in the top predator to that in the source medium.

Determining bioconcentration, bioaccumulation, and biomagnification factors for contaminants may be useful in determining potential exposures. In this risk assessment, BAFs, BCFs, and BMFs are derived from the literature.

Bioconcentration, bioaccumulation, and biomagnification are both chemical- and species-specific and depend on the physical/chemical characteristics of contaminants. The impact each chemical has will depend entirely on the physiological processes of organisms that comprise the ecosystem's food web. The specific dynamics of the biological communities near the Montana Pole site have not been investigated. Thus, terrestrial and aquatic risks presented in this document are qualitative rather than quantitative.

8.2.6 ASSESSMENT METHODS

Assessments of risks to biota are based upon the availability of appropriate toxicological data (e.g., BAFs, BMFs, LOAELs) found in the literature. When sufficient toxicological data are available, a quantitative assessment (ecological modeling) may be appropriate, depending on assessment objectives. A less rigorous approach is used to evaluate exposures when toxicological information is limited, which is the case for most ecological risk assessments. Such assessments generally consist of simple analyses, such as calculation of a Toxicity Quotient (TQ) (Barnhouse et al. 1986), and/or the use of Federal and State ambient water quality criteria or professional judgment to describe the potential for environmental impacts.

8.2.6.1 Aquatic Life

Ambient Water Quality Criteria (EPA 1986b) for the protection of freshwater aquatic life have been established for a number of chemicals under the U.S. Clean Water Act. These criteria are developed to be protective of 95 percent of all aquatic species, and are based on a diverse group of aquatic species. Therefore, not only are fish protected, but aquatic invertebrates and plants are protected as well. These criteria are compared with average and maximum surface water concentrations in the risk characterization section (Section 8.5.1) to determine the likelihood of adverse effects to aquatic life.

EPA Ambient Water Quality Criteria (EPA 1986b) and other related aquatic toxicity data were used to qualitatively evaluate risks to aquatic organisms. For example, species-specific aquatic toxicity data for the three site-related organic COCs (PAHs, PCP, dioxin), based on species known or expected to occur in or near study area surface waters were also evaluated.

8.2.6.2 Terrestrial Wildlife

No criteria have been developed specifically for the protection of terrestrial wildlife. Peer-reviewed scientific literature or documents summarizing literature (such as USFWS Hazard Review documents) are the main sources of most of the toxicity data for terrestrial wildlife. For purposes of assessment, toxicity values are obtained from studies reporting NOELs, LOELs, or median lethal doses (LD_{50}s)

for terrestrial or avian species and are presented in this section. Most terrestrial toxicity values are based on domestic birds or mammals. The NOEL represents the highest dietary concentration of a chemical not associated with an adverse effect in an animal, while the LOEL represents the lowest dietary concentration reported to cause an effect. The LD_{50} represents the dose which was lethal to 50 percent of an experimental population over a specified time period.

8.2.7 CHEMICALS SELECTED FOR THE ECOLOGICAL RISK ASSESSMENT

From the list of chemicals expected to occur at the Montana Pole site and adjacent vicinity seven chemicals or chemical groups are selected for evaluation in this ERA. Selection of these chemicals is based upon mobility and persistence, bioaccumulation potential, adequacy of toxicological data to evaluate risks, comparisons of maximum detected concentrations with toxicity criteria values, and the use of these chemicals in the wood-treating process at the Montana Pole and Treatment Plant site. The chemicals selected for evaluating qualitative risks to ecological receptors are:

- Polycyclic Aromatic Hydrocarbons (PAHs)
- Pentachlorophenol (PCP)
- Dioxin/Furans
- Arsenic
- Cadmium
- Copper
- Zinc

Not all chemicals detected were considered in the screen for COCs for ecological risk. The toxicological database for many chemicals detected on the Montana Pole site is limited, and no quantitative assessment of potential ecological effects is possible. The chemicals detected on site were initially screened, using professional judgement, for those which might be addressed using quantitative methods, and for those which, from experience and the literature, are likely be of concern for aquatic systems. Chemicals not addressed in the formal screening are discussed qualitatively in Section 8.5.3 which evaluates uncertainties.

Table 8-1 is a summary of the screening process, providing rationale for the inclusion or omission of chemicals from the list of COCs. Table 8-2 provides the comparisons among maximum detected media concentrations and toxicity criteria values, where available. The soil toxicity criteria values are calculated from NOAEL's found in the literature, using the following equation.

$$\frac{\text{NOAEL } (\mu\text{g/kg/day}) \times \text{BW (kg)}}{\text{IR (kg/day)}}$$

Where: NOAEL = observed no observable adverse effect level
BW = body weight
IR = soil ingestion rate

The following assumptions were used to provide conservative (screening level) toxicity values for the several species on which the NOAELs are based.

RATS	BW	=	0.25 kg
	IR	=	0.0005 kg/day
CHICKENS	BW	=	2.5 kg
	IR	=	0.01 kg
DOGS	BW	=	20 kg
	IR	=	0.01 kg/day
GOATS	BW	=	30 kg
	IR	=	0.01 kg
COWS	BW	=	360 kg
	IR	=	0.4 kg

For manganese, where the NOAEL is based on a dietary concentrations, it was assumed that rats might consume 5 percent of their dietary intake in soil each day, and the toxicity value was estimated as NOAEL (μg/day) / 0.05.

TABLE 8-1

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR ECOLOGICAL RISK ASSESSMENT

Chemical	Sufficient Toxicological Data Available[a]	Ambient Water Quality Criteria Available[b]	Maximum Detected Concentration Exceeds Available Toxicity Values[c]	Persistence/ Mobility Bioaccumulation Potential	Associated with Montana Pole Wood-treating Process	Decision
ORGANICS						
Carbon disulfide	Limited	No	No	Low/High/Low	No	Omit — no evidence of unacceptable media concentrations
Dioxin/Furans	Yes	No	Yes-soil	High/Low/High	Yes	Retain — maximum soil concentration exceeds sediment/soil criteria values
PAHs	Yes	Yes - for some PAHs	No	High/Low/High	Yes	Retain — maximum on site and groundwater concentrations are sufficiently high to warrant assessment
PCPs	Yes	Yes	Yes-water	High/Low/High	Yes	Retain — maximum water concentrations exceed criteria value
INORGANICS						
Aluminum	Limited	Yes - 1988	NA	High/Low/NA	No	Omit — no evidence of unacceptable media concentrations
Arsenic	Yes	Yes	Yes-sediment	High/Medium/Low	No	Retain — maximum soil and sediment concentrations exceed criteria values
Barium	Limited	No	NA	High/Low/NA	No	Omit — no evidence of unacceptable media concentrations
Beryllium	Yes	Yes	No	High/Low/NA	No	Omit — no evidence of unacceptable media concentrations
Cadmium	Yes	Yes	Yes-water Yes-sediment	High/Low/Low	No	Retain — maximum surface water and sediment concentrations exceed criteria values
Chromium	Yes	Yes	No	High/Low/Low	No	Omit — no evidence of unacceptable media concentrations
Cobalt	Limited	No	No	Medium/Medium/NA	No	Omit — no evidence of unacceptable media concentrations
Copper	Yes	Yes	Yes-water Yes-sediment	High/Low/Low	No	Retain — maximum surface water, soil and sediment concentrations exceed criteria values
Iron	Yes	Yes	No	Medium/Medium/Low	No	Omit - no evidence of unacceptable media concentrations

TABLE 8-1 (Cont.)

SUMMARY OF CRITERIA USED TO SELECT CHEMICALS OF CONCERN FOR ECOLOGICAL RISK ASSESSMENT

Chemical	Sufficient Toxicological Data Available[a]	Ambient Water Quality Criteria Available[b]	Maximum Detected Concentration Exceeds Available Toxicity Values[a]	Persistence/ Mobility Bioaccumulation Potential	Associated with Montana Pole Wood-treating Process	Decision
INORGANICS (Cont.)						
Lead	Yes	Yes	Yes-water Yes-sediment	High/Low/Low	No	Retain — maximum surface water, soil and sediment concentrations exceed criteria values
Manganese	Limited	4/6	No	High/Low/NA	No	Omit - no evidence of unacceptable media concentrations
Nickel	Yes	Yes	No	High/Low/Low	No	Omit — no evidence of unacceptable media concentrations
Vanadium	Yes	No	No	High/Low/NA	No	Omit — no evidence of unacceptable media concentrations
Zinc	Yes	Yes	Yes-water Yes-sediment	High/Low/Low	No	Retain - maximum surface water, soil and sediment concentrations exceed criteria values

[a] Based upon available toxicity values for wildlife, laboratory, and/or domestic animals (i.e., NOAELs, LOAELs, LC_{50}s). See Table 8-2.

[b] Ambient Water Quality Criteria (AWQC) are provided by the US EPA and represent the most current recommendations on acceptable limits for aquatic life (EPA 1986b).

[c] Persistence/Mobility: Persistence is described by a qualitative estimate of how long the chemical will remain in the environment. Mobility is described by a qualitative estimate of how readily the chemical will move away from its first site of deposition. For volatile compounds, no appreciable deposition may take place.

NA - No data available

TABLE 8-2

SUMMARY OF MAXIMUM CHEMICAL DETECTIONS AND ECOLOGICAL TOXICITY VALUES

Chemical	Maximum Concentration in Sediment (µg/kg)	Sediment Criteria Value[a] (µg/kg)	Maximum Concentration in Surface Water[d] (µg/L)	AWQC[b] Acute/Chronic (µg/L)	Maximum Concentration in Soil (µg/kg)	Toxicity Value (µg/kg)	Reference for Toxicity Value
ORGANICS							
Dioxins/Furans	1.4	NA	—	NA	5,020	5E-06 µg/kg based on NOAEL: 1E-08 µg/kg-bw; rat	Murray et al. 1979
Polycyclic Aromatic Hydrocarbons	139 (Indeno-(1,2,3-CD) pyrene)	NA	12.7 (Acenaphthene)	NA	236,000 (Nathphalene)	15,000,000 µg/kg based on NOAEL: 30,000 µg/kg-bw; rat LC_{87} for Bluegill (Benzo(a)anthracene) = 1000 µg/L LC_{50} for Rainbow Trout (Fluorene) = 820 µg/L	IARC 1973 Eisler 1987
Pentachlorophenol	1,820	NA	16.5	8.9/5.6 at pH = 7.0	1,510,000	1,500,000 µg/kg based on NOAEL: 3,000 µg/kg-bw; rat and rabbit	EPA 1980d
INORGANICS							
Aluminum	—	NA	—	NA	10,400,000	NA	
Arsenic	842,000	57,000	25.2	360/190	356,000	2,400,000 µg/kg based on NOAEL: 1,200 µg/kg-bw/day; dog	Byron et al. 1967
Barium	—	NA	—	NA[c]	190,000	NA	
Beryllium	—	NA	—	130/5.3	650	500,000 µg/kg based on NOAEL: 1,000 µg/kg-bw/day;rat	NAS 1977
Cadmium	21,900	5,100	25.2	3.9/1.1	3310	22,500,000 µg/kg based on NOAEL: 7,500 µg/kg-bw;goat	Anke et al. 1970
Carbon disulfide	—	NA	—	NA	8	NA	
Chromium	18,700	260,000	—	16/11 (VI)	15,000	20,000,000 µg/kg based on NOAEL: 40,000 µg/kg-bw; rat	Mackenzie et al. 1958

TABLE 8-2 (Cont.)

SUMMARY OF MAXIMUM CHEMICAL DETECTIONS AND ECOLOGICAL TOXICITY VALUES

Chemical	Maximum Concentration in Sediment (µg/kg)	Sediment Criteria Value[a] (µg/kg)	Maximum Concentration in Surface Water[d] (µg/L)	AWQC[b] Acute/Chronic (µg/L)	Maximum Concentration in Soil (µg/kg)	Toxicity Value (µg/kg)	Reference for Toxicity Value
INORGANICS (Cont.)							
Cobalt	5,210,000	NA	—	NA	8,900	1,150,000 µg/kg based on NOAEL: 5,000 µg/kg-bw; chicken	NRC 1977
Copper	—	3,900,000	1,340	18/12	1,140,000	9,000,000 µg/kg based on NOAEL: 10,000 µg/kg-bw; cow	Engle et al. 1982
Iron	—	NA	—	1,000/NA	17,300,000	NOAEL: 2,000,000 µg/kg-bw; calves	Koong et al. 1970
Lead	714,000	450,000	30.3	82/3.2	134,000	5,000,000 µg/kg based on NOAEL: 10,000 µg/kg-bw; rat	Azar et al. 1973
Manganese	—	—	—	—	541,000	2,000,000 µg/kg based on NOAEL: 100,000 µg/kg in diet of rats	Clayton 1981-1982
Nickel	—	NA		1,800/96	8,800	1,250,000 µg/kg based on NOAEL: 2,500 µg/kg-bw; rat	Ambrose et al. 1976
Vanadium	—	NA	—	NA	35,700	10,000,000 µg/kg based on NOAEL: 20,000 µg/kg-bw/day; rat	Paternain 1987
Zinc	6,220,000	410,000	7,510	320/47	1,500,000	50,000,000 µg/kg based on 100,000 µg/kg-bw; rat	Slicker & Cox 1968

[a] Source: Washington State Administrative Code (1991).
[b] Source: EPA 1986b.
[c] 50,000 µg/L as reported.
[d] Includes enforcement-quality data only.

NA = Not available
- = Not a COC for this medium

8.2.8 UNCERTAINTIES ASSOCIATED WITH THE EXPOSURE ASSESSMENT

Exposures of Riparian Plants and Wildlife

While exposure occurs along the stream banks, the bioavailability of organics to riparian plants from contaminated soils and water at Montana Pole site is not known. Similarly, the frequency and duration of wildlife exposure and amounts of contaminated water and food consumed are not known. Therefore, in the absence of site-specific biota tissue concentration data, a discussion of potential risks to these organisms may be under-estimated or over-estimated.

8.3 ECOLOGICAL TOXICITY ASSESSMENT

In accordance with the RAGS (EPA 1989a), toxicity assessments were prepared for each of the ecological COCs identified in Section 8.2.7. The toxicity assessment is typically comprised of two elements. The first, hazard identification, is intended to characterize the nature and extent of biota health hazards associated with chemical exposures. The second, a dose-response assessment, determines the relationship between the magnitude of exposure to a chemical and the occurrence of adverse health effects.

Dose-response estimates, such as RfDs and cancer potency slope factors, however, have not been developed for biota. Therefore, a survey of the toxicological literature for biota toxicity values and comparisons of surface water concentrations to AWQC for the protection of freshwater aquatic life are used to assess toxicity to biota on or near Montana Pole site.

The AWQC for the protection of aquatic life for each COC are presented in Table 8-2. These criteria provide water quality "adequate for the protection of 95 percent of all aquatic life." This includes fish, invertebrates, and aquatic plants. Criteria are based solely on data and scientific judgments on the relationship between contaminant concentration and potential effect, and do not consider economics or technology (EPA 1986b). These criteria, along with other toxicological reference values, were used to assess the potential for adverse impacts to aquatic life posed by the Montana Pole site.

In addition, aquatic toxicity data specific for species known or expected to occur in or near study area surface waters are evaluated for the three chemicals associated with Montana Pole (PAHs, PCP, and dioxin). These data, presented in Table 8-3, are compared to ambient surface water concentrations for estimating risks to species specific to study area or adjacent surface waters.

Water quality criteria values do not exist specifically for the protection of aquatic plants or terrestrial plants and animals. For these organisms, the toxicological literature commonly reports the NOAEL, the LOAEL, the medium lethal concentration to 50 percent of the population (LC_{50}), the LD_{50}, and the effective concentration (EC) where a measured effect is seen in some percentage of the population (i.e., EC_{50} or EC_{10} for measured effects in 50 percent of 10 percent of the population, respectively). These values are determined in controlled laboratory experiments and may not reflect the effects that may occur between multiple chemicals — particularly the interactions among metals and other constituents. However, additive effects are generally accepted as those best describing the behavior of complex chemical mixtures, and synergistic or antagonistic effects are not well-documented for most chemical mixtures studies to date. Toxicity values for the selected COCs and metals for terrestrial organisms are presented in Table 8-2.

The following guidelines were used to select critical toxicity values for use in this ecological risk assessment. Chronic values are chosen over acute toxicity values when available. Acute toxicity values are used only if no chronic data are available for any species. If a chronic NOEL and a chronic LOEL are available, the NOEL is chosen as the critical toxicity value. The NOEL selected is the highest NOEL reported, and the LOEL selected is the lowest LOEL reported. If only an LD_{50} is available, the value selected is the lowest LD_{50} reported in the literature. These values are used to estimate risk in Section 8.5 by comparing them with estimated dietary concentrations in birds and mammals. Appropriate safety factors are applied in some cases, based on the type of toxicity value available.

Brief toxicity profiles for site-specific COCs and metals found in surface water at Silver Bow Creek were prepared for aquatic plants, terrestrial plants, aquatic animals, and terrestrial animals. These profiles are presented below.

TABLE 8-3

SPECIES-SPECIFIC ECOLOGICAL EFFECTS FOR ORGANIC COCs

Species	Measurement Endpoint	Endpoint Conc. (µg/L)	Ref	Assessment Endpoint
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)				
Cladoceran (*Daphnia*)	48-hr LC50 (Acenaphthene)	870	1	Decreased population size
Mayfly (*Hexagenia*)	120-hr LC50 (Fluorene)	5,800	2	Decreased population size
Rainbow Trout	96-hr LC50 (Fluorene)	820	2	Decreased population size
PENTACHLOROPHENOL				
Freshwater Alga (various species)	50% growth inhibition/reduction	80-760	3, 4	Decreased primary productivity
Cladoceran (*Ceriodaphnia*)	Reduction in number of young	4.1	5	Decreased population size
Cladoceran (*Ceriodaphnia*)	48-hr LC50	164	6	Decreased population size
Midge (*Chironomus*)	24-hr LC50 (pH 6)	465	7	Decreased population size
Midge (*Chironomus*)	50% locomotion inhibition (temp 35° C)	631	8	Increased predation Decreased population size
Midge (*Chironomus*)	50% locomotion inhibition (temp 15° C)	1176	8	Increased predation Decreased population size
Cladoceran (*Daphnia*)	96-hr LC50	475	2	Decreased population size
Cladoceran (*Daphnia*)	50% immobilization	370-440	9	Increased predation Decreased population size
Cladoceran (*Simocephalus*)	96-hr LC50 (temp 24° C)	204	5	Decreased population size
Cladoceran (*Simocephalus*)	96-hr LC50 (temp 18° C)	670	5	Decreased population size
Tubificid Worm (*Tubifex*)	24-hr LC50 (pH 7.5)	286	2	Decreased population size
Amphipod (*Gamarus*)	30-day LC50	860	10	Decreased population size
Amphipod (*Gamarus*)	96-hr LC50	1,150	11	Decreased population size
Caddisfly (*Philarctus*)	96-hr LC50	1,200	12	Decreased population size
Mayfly (*Callibaetes*)	96-hr LC50	1,700	12	Decreased population size
Amphipod (*Crangonyx*)	96-hr LC50	1,900	12	Decreased population size

TABLE 8-3 (Cont.)

SPECIES-SPECIFIC ECOLOGICAL EFFECTS FOR ORGANIC COCs

Species	Measurement Endpoint	Endpoint Conc. (µg/L)	Ref	Assessment Endpoint
PENTACHLOROPHENOL (Cont.)				
Isopod (*Ascellus*)	96-hr LC50 (temp 8.6° C)	2,300	12	Decreased population size
Rainbow Trout	27% growth inhibition	7.4	2	Decreased population size
Rainbow Trout	100% mortality (DO 3 mg/L)	10	13	Decreased population size
Rainbow Trout	100% mortality (DO 5 mg/L)	20	13	Decreased population size
Rainbow Trout	Significant mortality, decreased growth	19	14	Decreased population size
Rainbow Trout	48% reduction in egg viability	22	15	Decreased population size
Rainbow Trout	11-19% growth inhibition	28	2	Decreased population size
Rainbow Trout	96-hr LC50	34-121	2, 14, 16, 17, 18	Decreased population size
Rainbow Trout	100% mortality (72 days post fertilization)	40	13	Decreased population size
Rainbow Trout	41-day LC100	46	2	Decreased population size
Rainbow Trout	81% reduction in egg viability	49	15	Decreased population size
Rainbow Trout	Eye abnormalities in embryos	60	15	Decreased population size
Rainbow Trout	3.5-hr LC50	10,000	19	Decreased population size
Brook Trout	96-hr LC50	126	2	Decreased population size
DIOXIN				
Rainbow Trout	Growth retardation in fry at 72 days following 96 hr exposure of eggs	0.0001	20	Decreased population size
Rainbow Trout	26% mortality in fry at 72 days following 96 hr exposure	0.01	20	Decreased population size
Rainbow Trout	Significant fry mortality at 78 days following 6 hr exposure	0.107	21	Decreased population size

1 Rogers et al. 1984
2 Finger et al. 1985
3 Crossland and Wolff 1985
4 Smith et al. 1987
5 Hedtke et al. 1986
6 Huber et al. 1982
7 Fisher and Wadleigh 1986
8 Fisher 1986
9 Berglind and Dave 1984
10 Graney and Giesey 1986
11 Graney and Giesey 1987
12 Hedtke and Arthur 1985
13 Chapman and Shumway 1978
14 Dominguez and Chapman 1984
15 Nagler et al. 1986
16 Johnson and Finley 1980
17 Mayer and Ellersieck 1986
18 Mckim et al. 1987
19 Cote 1972
20 Helder 1981
21 Branson et al. 1985

8.3.1 PENTACHLOROPHENOL (PCP)

PCP is a commercially produced organochlorine compound used as a preservative of wood and wood products, herbicides, insecticides, fungicides, molluscicides, and bactericides (Eisler 1989).

Phytotoxicity

PCP is toxic to plant mitochondria. At 267 μg/L, 50 percent uncoupling was noted in insulated mitochondria of potato (*Solanum tuberosum*) and mung bean (*Phaseolus aureus*) (Ravanel and Tissut 1986). Ehrlich et al. (1987), in addition, reported that cell growth and synthesis of RNA and ribosome in yeast, *Saccharomyces* sp., were adversely affected by PCP in a dose-related manner. A concentration of 0.3 mg/L caused a 50 percent reduction of root growth in rice seedlings (Nagasawa et al. 1981).

Uptake of PCP was observed in rice (*Aryza sativa*) grown over a 2-year period under flooded conditions with a single application (23 kg/ha) of radiolabled PCP to soil (Weiss et al. 1982). Uptake was 12.9 percent of the application during the first year, and roots contained the largest concentration — 5 mg PCP/kg. Uptake was reduced to 2.5 percent and soil residues corresponded to 8.4 kg/ha in the second year. Increased amounts of unextractable residues in plants and lower chlorinated conjugated phenols were also identified during the second year (Weiss et al. 1982).

Aquatic Toxicity

PCP is most toxic and most rapidly metabolized in aquatic environments at elevated temperatures and reduced pH (Eisler 1989). Adverse effects occur at 3-100 μg/L for invertebrates, and < 1-68 μg/L for fishes (Eisler 1989). In one particular study, 21 day chronic mortality of *Daphnia magna* was produced at 320 μg/L, but not at 180 μg/L (EPA 1980d). Largemouth bass (*Micropterus salmoides*) were exposed to concentrations of PCP ranging from 1.6 to 88 μg/L for 8 weeks. Over the final 3 weeks, fish reared in concentrations of 67 and 88 μg/L performed significantly less feeding acts and had a lower rate of prey capture than did control fish (Brown et al. 1987). Table 8-3 presents aquatic toxicity data specific to study area species.

PCP is expected to bioconcentrate because of its low water solubility. However, the BCF is dependent on (1) the pH of the water, since PCP becomes more dissociated at higher pH, and (2) the rate of depuration. Reported log BCF values include: 2-89 in fathead minnows; 2.4-3.73 in rainbow trout; and 0.7-1.7 in sheepshead minnows (Veith et al 1979; Niimi 1982; and Parish et al. 1978). PCP is also known to bioaccumulate. Bioaccumulation of PCP in fish occurs rapidly with uptake from water rather than through the food chain or diet (Eisler 1989). PCP absorbed by goldfish from water was rapidly excreted as a sulfate conjugate, the biological half-life was approximately 10 hours (USFWS 1980).

AWQC for PCP are pH dependent. At pH 7.0, which is the average pH recently measured in Silver Bow Creek, AWQC to protect freshwater life are 8.9 μg/L (acute) and 5.6 μg/L (chronic) (EPA 1986c). The chronic value was used in this ERA to assess potential risks to aquatic organisms. In addition, toxicity values for potentially resident species were used to evaluate potential PCP toxicity.

Terrestrial Wildlife Toxicity

Signs of PCP intoxication in birds include excessive drinking and regurgitation, rapid breathing, wing shivers or twitching, jerkiness, shakiness, ataxia, tremors, and spasms (Hudson et al. 1984). Mortality from PCP has been observed in various species of birds at single oral doses of 380 to 504 mg/kg BW and dietary concentrations of 3,850 mg/kg (Eisler 1989). An acute oral LD_{50} of 380 mg/kg BW was reported for mallard (*Anas platyrhynchos*) (Hudson et al. 1984). No deaths, however, were observed in Japanese quail (*Coturnix japonica*) fed 3,100 mg/kg (Hill and Camardese 1986). A criterion for protection of birds against adverse effects of PCP was proposed as 1.0 mg/kg in the diet (Eisler 1989).

Little data is available on the effects of PCP to mammalian wildlife. However, studies have been conducted on livestock and small laboratory animals. Some signs of acute PCP intoxication in domestic and laboratory animals include elevated blood sugar, vomiting, elevated blood pressure, increased respiration rate, high fever, collapse, and death (Eisler 1989). Acute toxicity was observed in rats at doses >5 mg/kg BW, however, no effects were observed at levels ≤ 5 mg/kg BW (IARC 1979). Ingestion of 3 mg/kg BW of a commercially available purified grade of PCP did not produce any adverse effects in rats (IARC 1979). EPA (1980) reported a NOAEL for rats and rabbits of 3

mg/kg BW. Rats were administered this dose for 24 months while rabbits were only exposed for 90 days. Cattle fed up to 20 mg/kg doses of technical grade PCP experienced decreased weight gain, progressive anemia, and immune effects. Only minimal adverse effects were observed after exposure to analytical grade PCP (NRC 1986).

PCP tends to accumulate in mammalian tissues unless it is efficiently conjugated into readily excretable form (Kinzell et al. 1985). The ability to conjugate PCP varies widely among species (Braun and Sauerhoff 1976; EPA 1980d). Rodents are the most efficient, however, excreting as much as 40 percent unchanged PCP in the urine (NRC 1986). For evaluating risks to terrestrial organisms, the NOAEL of 3 mg/kg/bw for the rat was used.

8.3.2 POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

There are thousands of PAH compounds, each differing structurally in the number and position of aromatic rings. They also differ toxicologically in the nature and severity of systemic effects and carcinogenicity produced. Environmental concern focuses on PAHs with lower molecular weights because these compounds tend to be more acutely toxic and cause more adverse effects to some organisms than higher molecular weight PAHs. However they are considered noncarcinogenic. The higher molecular weight PAHs are generally less toxic, but are carcinogenic, mutagenic, or teratogenic to organisms such as fish and other aquatic life, birds, and mammals (Eisler 1987).

Phytotoxicity

Some PAHs have growth-promoting effects on plants. Higher plants, including tobacco, rye, and radish and algae (*Chlorella vulgaris, Scenedesmus deligurus,* and *Ankistrodesmus*) all demonstrated increased growth when exposed to PAHs. Growth-promotion was near 100 percent in some cases, and benzo(a)pyrene was the most effective of the PAHs studied (Graf and Nowak 1968). Edwards (1983) states that some plants contain chemicals to protect against PAH effects and once plants have synthesized PAHs, these compounds may act as plant growth hormones.

Aquatic Toxicity

PAHs vary substantially in their toxicity to aquatic organisms. Toxicity of PAHs increases with increasing molecular weight and with increasing alkyl substitution of the aromatic ring. For example, no toxicity was observed in minnows (*Poeiciliopsis spp.*) at a water concentration of 250 μg of 7,12-dimethylbenz(a)anthracene/L while 150,000 μg naphthalene/L killed 50 percent of exposed mosquitofish (*Gambusia affinis*). AWQC are generally unavailable for PAH compounds. LC_{50} values for some PAHs and for some aquatic organisms are available in the literature such data are shown in Table 8-2. The data show that PAH toxicity varies extensively among aquatic organisms and among PAHs. In general, crustaceans are most sensitive to PAH toxicity and teleost fish are least sensitive (Eisler 1987). Table 8-3 lists species-specific toxicity data for local species.

Most species of aquatic organisms rapidly accumulate (bioconcentrate) PAHs from low concentrations in the ambient medium (Eisler 1987). Bioaccumulation of PAHs is species specific, with organisms that are incapable of metabolizing PAHs having the greatest bioaccumulation rates. Species in this category include algae and mollusks. The bioaccumulation potential of PAHs increases with increasing molecular weight and with increasing octanol/water partition coefficients (K_{ow}). Table 8-4 shows BCFs for different PAHs in *Daphnia pulex*, and gives molecular weights for PAHs shown. The data illustrate the relationship between bioconcentration potential and molecular weight of PAHs. In general, PAHs such as naphthalene, which have relatively low molecular weights, are associated with lower BCFs than PAHs with higher molecular weight (Table 8-4).

Terrestrial Wildlife Toxicity

A limited amount of data are available that relate to the effects of PAHs in birds. One study involved feeding 4,000 mg PAH/kg in the diet to mallards, *Anas platyrhynochos*, for seven months. There were no signs of toxicity during exposure, however, liver weight increased 25 percent and blood flow to liver increased 30 percent when compared to controls (Eisler 1987). A study conducted by Hoffman and Gay (1981) measured embryotoxicity of various PAHs to mallard eggs. 7,12-Dimethylbenz(a)anthracene was the most toxic PAH of those studied, causing 26 percent mortality in 18 days at a dose of 0.002 μg/egg. Incomplete skeletal ossification, and defects in eye, brain, liver, feathers and bill were observed in survivors. The same dose (0.002 μg/egg) of

TABLE 8-4

BIOCONCENTRATION FACTORS OF PAH COMPOUNDS IN *DAPHNIA PULEX*

Daphnia pulex				
	BCF	**Exposure Period**	**Reference[a]**	**Molecular Weight**
Anthracene	720-1200	24 hours	Southworth et al., 1978, 1979, Neff 1985	178
9-Methylanthracene	4583	24 hours	Neff 1985	102
Benz(a)anthracene	10,109	24 hours	Southworth et al. 1978	228
Benzo(a)pyrene	134,248	3 days	Lu et al. 1977	252
Naphthalene	131	24 hours	Neff 1985	128
Perylene	7191	24 hours	Neff 1985	252
Phenanthrene	325		Neff 1985	178
Pyrene	2702	24 hours	Gerhart and Carlson 1978	202

[a] As cited in Eisler 1987.

benzo(a)pyrene did not affect mallard survival, but did cause embryonic growth reduction and an increased incidence of abnormal survivors.

Toxic and carcinogenic effects of PAHs have been observed in various mammalian species. Acute and chronic exposure to various carcinogenic PAHs have caused destruction of hematopoietic and hymphoid tissues, ovotoxicity, antispermatogenic effects, adrenal necrosis, and changes in the intestinal and respiratory epithelia (EPA 1980a; Eisler 1987). Carcinogenicity has been observed in rodents at chronic oral doses between 0.00004 to 3,300 mg/kg BW, depending on the PAH compound. Rodents were most sensitive to 7,12-dimethylbenz(a)anthracene (0.00004 mg/kg BW) (Sims and Overcash 1983; Lo and Sandi 1978). Rats exposed to 30 mg benzo(a)phrene/kg in the diet for 110 days did not develop stomach tumors, however, tumors were observed in rats exposed to 40-45 mg/kg (IARC 1973) (Table 8-3). For this ERA, the NOAEL (30 mg/kg bw) was used to evaluate risk.

8.3.3 DIOXINS/FURANS

Phytotoxicity

No information is available on the phytotoxicity of dioxin/furan isomers.

Aquatic Toxicity

There are a limited number of studies dealing with dioxin toxicity to aquatic organisms and no toxicity information is available for furans. The summary document of Eisler (1986b) reported that no data were available on lethal or sub-lethal effects to aquatic organisms for any PCDD isomer except 2,3,7,8-TCDD and 1,3,6,8-TCDD. Sensitive species of teleosts exhibited reduced growth and fin necrosis at concentrations as low as 0.1 μg/L of 2,3,7,8-TCDD after exposure for 24 to 96 hours. Concentrations of 1.0 μg/L and higher were eventually fatal, and exposure to lower concentrations (0.01 μg/L) for 24 hours had no measurable effect. Guppies that survived exposure to 0.1 or 10 μg/L 2,3,7,8-TCDD for 10 days, also demonstrated reduced growth and fin necrosis (Murty 1986). Aquatic toxicity data for local species are presented in Table 8-3.

Eisler (1986b) also noted that aquatic invertebrates, plants, and amphibians were comparatively resistant to 2,3,7,8-TCDD. For example, there were no adverse effects on growth, reproduction, or food consumption of algae, daphnids, and snails during immersion for 32 days in solutions containing 2.4 to 4.2 mg/L of 2,3,7,8-TCDD (Yockim et al. 1978). An NOAEL for guppy (*Poecilia reticulatus* was reported at 0.01 μg 2,3,7,8-TCDD/L (Miller et al. 1979). The LOAEL for northern pike (*Esox lucius*) was 0.1 μg/L (Helder 1980). Bullfrog (*Rana catesbeiana*) demonstrated no effect when administered a dose of 0.5 mg/kg-bw (Neal et al. 1979).

Accumulation of 2,3,7,8-TCDD from the aquatic environment was evident for all species that have been examined (Eisler 1986b). Accumulation of 1,3,6,8-TCDD was also observed, but was lower and was eliminated 10 to 15 times more rapidly than 2,3,7,8-TCDD (Corbet et al. 1983). Studies conducted with teleosts demonstrated that body burdens of 2,3,7,8-TCDD increased with increasing concentration in the water column and with increasing duration of exposure. Upon removal to uncontaminated water, less than 50 percent was lost in 109 days (Miller et al. 1979). Bioconcentration factors for various aquatic species are: 2,083 (alga, *Odegonium cardiacum*) 3,731 (snail), 7,125 (*Daphnia magna*), 1,482 (Mosquito fish, *Gambusia affinis*), and 2,181 (channel catfish, *Ictalurus punclatus*) (Eisler 1986b). The LOAEL (0.1 μg/L) for northern pike was used to evaluate risks to aquatic receptors.

Terrestrial Wildlife Toxicity

Information is lacking or scarce on the biological properties of dioxin/furan isomers, except 2,3,7,8-TCDD (Eisler 1986b). The latter has been associated with lethal, carcinogenic, teratogenic, reproductive, mutagenic, histopathologic, and immunotoxic effects. There are substantial inter- and intraspecific differences in sensitivity and toxic responses to 2,3,7,8-TCDD. Typically, animals poisoned by 2,3,7,8-TCDD exhibit weight loss, atrophy of the thymus gland, and eventually death. The toxicological mechanisms are imperfectly understood (Eisler 1986b).

Domestic chickens were relatively sensitive to dioxins/furans (Eisler 1986b) especially 2,3,7,8-TCDD. Chickens fed 1 or 10 μg of 2,3,7,8-TCDD, 1,2,3,7,8-CDD, or hepta-CDDs per kg of body weight daily for 21 days showed signs of chick edema disease, i.e., pericardial, subcutaneous, and peritoneal edema; liver enlargement and necrosis with fatty degeneration; and frequently resulted in

death (NRCC 1981). Although there presently is no evidence of biomagnification of dioxins/furans in birds, it is speculated that piscivorous birds have a greater potential to accumulate dioxins/furans than the fish that they eat (NRCC 1981).

The greatest toxicity of dioxin/furan isomers to mammals is caused by those isomers with halogen atoms occupying at least 3 of the 4 lateral ring positions (2,3,7,8 positions) and at least one of the adjacent ring positions being nonhalogenated (Kociba and Schwetz 1982a,b). This was observed in studies involving mouse and guinea pig. Both of these species were most sensitive to 1,2,3,4,6,7,8-hepta CDD and least sensitive to 2,8-di CDD. LC_{50} values ranged from $<300,000$ to >600 μg/kg-bw (Kociba and Schwetz 1982b).

Almost all of the information on the toxicity of dioxins/furans to mammals is for the isomer 2,3,7,8-TCDD. It has been found that very small amounts of 2,3,7,8-TCDD can be toxic to rats and other animals. Doses as low as 0.100 mg/kg administered to rats showed biphasic decline in body weight with cessation of food and water consumption and urine production (Courtney et al. 1978). LD_{50} values of 0.0006 - 0.002, 0.1 - 0.2, and 1.15 - 5.05 mg/kg were reported for guinea pig, dog, and hamster, respectively (Eisler 1986b).

Accumulation of 2,3,7,8-TCDD was observed in rats fed 0.45 and 1.3 μg/kg TCDD/day. On day 42 of the study, total accumulation, mainly in the liver, was 10 times the daily intake (The Chemical Society 1974-1975). Elimination rates of 2,3,7,8-TCDD are similar for most species. The estimated retention times in small laboratory mammals (rats, mice, guinea pigs, and hamster) extended from 10.8 to 30.2 days for 50 percent elimination (Eisler 1986b). Most 2,3,7,8-TCDD is eliminated in the feces (IARC 1977). The LD_{50} (0.0006 μg/kg bw) for the guinea pig was used to evaluate risks to wildlife.

8.3.4 ARSENIC

Phytotoxicity

To be absorbed by plants, arsenic must be in a mobile form. Arsenic can be absorbed through the roots or the leaves, although translocation of arsenic is species dependent (Eisler 1988a). When

translocation does occur, arsenic acts to degrade chlorophyll, thus reducing photosynthesis and eventually productivity (Salisbury and Ross 1969). Sensitive plant species can experience decreased productivity in the presence of 1 mg/L water soluble arsenic (Eisler 1988a). The EPA has recommended that effluent to be used for irrigation purposes contain no more than 0.1 mg/L arsenic (EPA 1976). It is recommended that water used for irrigation contain no more than 10 mg/L arsenic for short term use or 1 mg/L for continuous use (Geonomics, Inc. 1978).

Exposure to arsenic resulted in 100 percent mortality in three freshwater species of alga (*Cladophoro sp.*, *Spirogyra sp.*, and *Zygnema sp.*) and 95 percent mortality in a freshwater submerged plant species (*Potamogeton sp.*) at concentrations of 2,320 μg/L (Arsenic[III]) (EPA 1985c). Richter (EPA 1985c) reported that *Selanastrum capricornutum* was more sensitive to pentavalent arsenic than trivalent arsenic; 50 percent growth inhibition occurred in 4-days at 690 μg/L pentavalent arsenic and at 31,200 μg/L trivalent arsenic.

Water milfoil, waterweed, and blue-green algae tolerate between 6,000 and 10,000 μg/L sodium arsenate before toxicity becomes apparent. Pondweeds will tolerate up to 5,000 μg/L of the tioxide form before growth is hindered (Becker and Thatcher 1973).

Anderson et al. (1980) reported BCFs of 2 to 5 for four species of aquatic plants, after 42 days of exposure. The plants evaluated were *Hydrophila lacustris*, water hyacinth (*Eichhlornia crassipes*), alligator weed (*Alternanthera philoxeroides*), and duckweed (*Lemna minor*).

Aquatic Toxicity

The toxicity of arsenic to aquatic and terrestrial wildlife has been recently reviewed by Eisler (1988a). Arsenic is toxic to aquatic animal species, and induces its toxic effects via enzyme inhibition. In aquatic species, arsenic has induced death following acute exposures and has caused death and deformity following chronic exposures. Some fish species have shown some indication of acclimation to arsenic exposure, that is, toxicity has decreased in organisms previously exposed to arsenic (Rand and Petrocelli 1985).

Amphipods and cladocerans are the most sensitive tested aquatic animals to arsenic. Mean acute values for these species are *Gammarus pseudolimnaeus* 874 μg/L, *Simocephalus sp.* 1,175 μg/L, *Ceriodaphia reticulata* 1,800 μg/L, and *Daphnia sp.* 2,444 μg/L (EPA 1985c). Stoneflies are relatively tolerant of arsenic; the acute value for *Pteronarcys californica* is 22,040 μg/L (EPA 1985c). No acute toxicity values were reported by EPA (1985c) for mayflies or caddisflies, and toxicity information for these groups is limited.

Trout are among the most sensitive fish tested with arsenic. In rainbow trout (*Oncorhynchus mykiss*) acute effects occurred at 10,800 μg/L (as arsenic [V]) in fish 2 months old under static conditions (Hale 1977). An LC_{50} (144 hours) for adult rainbow trout of 13,340 μg/L was reported for static conditions by Johnson and Finley (1980). An acute LC_{50} for brook trout (*Salvelinus fontinalis*) of 14,960 μg/L for flow-through conditions was reported by Cardwell et al. (EPA 1985c). An acute LC_{50} of greater than 16,010 μg/L was reported for cutthroat trout (*Salmo clarki*), based on a static test (Johnson and Finley 1980; EPA 1985c). The 28-day EC_{50} (based on death and deformity) for embryo-larval rainbow trout exposed to arsenic (III) is 550 μg/L (EPA 1985c; EPA 1985c). The EC_{10} for this study was 134 μg/L (EPA 1985c).

Information on the toxicity of arsenic-contaminated sediment is limited. Pavlou and Weston (1983 in Pavlou 1987) reviewed concentrations for arsenic in sediments and found values ranging from 3-8 mg/kg.

There is no evidence for bioaccumulation through the food chain with respect to arsenic content in plants. In species that exhibit tolerance, arsenic is stored as an insoluble residue in roots. In nontolerant species, toxicity first appears in root systems and limits growth before arsenic can be translocated to other plant parts.

The EPA (1985c) reported that arsenic may bioaccumulate in lower forms of aquatic life for readily than in fish, and that bioaccumulation potential is similar for Arsenic (III) and Arsenic (V). BCFs of 3 to 17 have been reported for the snails *Stagnicola emarginata* and *Helisoma campanulatum*, respectively (EPA 1985c). Stonefly (*Pteronarcys dorsata*), after 28 days of exposure, had BCFs of 9 and 7 for arsenic (III) and arsenic (V), respectively (EPA 1985c). The EPA (1985c) found no accumulation of arsenic (both inorganic and organic forms) in rainbow trout (whole body) after 28-

Amphipods and cladocerans are the most sensitive tested aquatic animals to arsenic. Mean acute values for these species are *Gammarus pseudolimnaeus* 874 μg/L, *Simocephalus sp.* 1,175 μg/L, *Ceriodaphia reticulata* 1,800 μg/L, and *Daphnia sp.* 2,444 μg/L (EPA 1985c). Stoneflies are relatively tolerant of arsenic; the acute value for *Pteronarcys californica* is 22,040 μg/L (EPA 1985c). No acute toxicity values were reported by EPA (1985c) for mayflies or caddisflies, and toxicity information for these groups is limited.

Trout are among the most sensitive fish tested with arsenic. In rainbow trout (*Oncorhynchus mykiss*) acute effects occurred at 10,800 μg/L (as arsenic [V]) in fish 2 months old under static conditions (Hale 1977). An LC_{50} (144 hours) for adult rainbow trout of 13,340 μg/L was reported for static conditions by Johnson and Finley (1980). An acute LC_{50} for brook trout (*Salvelinus fontinalis*) of 14,960 μg/L for flow-through conditions was reported by Cardwell et al. (EPA 1985c). An acute LC_{50} of greater than 16,010 μg/L was reported for cutthroat trout (*Salmo clarki*), based on a static test (Johnson and Finley 1980; EPA 1985c). The 28-day EC_{50} (based on death and deformity) for embryo-larval rainbow trout exposed to arsenic (III) is 550 μg/L (EPA 1985c). The EC_{10} for this study was 134 μg/L (EPA 1985c).

Information on the toxicity of arsenic-contaminated sediment is limited. Pavlou and Weston (1983 in Pavlou 1987) reviewed concentrations for arsenic in sediments and found values ranging from 3-8 mg/kg.

There is no evidence for bioaccumulation through the food chain with respect to arsenic content in plants. In species that exhibit tolerance, arsenic is stored as an insoluble residue in roots. In nontolerant species, toxicity first appears in root systems and limits growth before arsenic can be translocated to other plant parts.

The EPA (1985c) reported that arsenic may bioaccumulate in lower forms of aquatic life for readily than in fish, and that bioaccumulation potential is similar for Arsenic (III) and Arsenic (V). BCFs of 3 to 17 have been reported for the snails *Stagnicola emarginata* and *Helisoma campanulatum*, respectively (EPA 1985c). Stonefly (*Pteronarcys dorsata*), after 28 days of exposure, had BCFs of 9 and 7 for arsenic (III) and arsenic (V), respectively (EPA 1985c). The EPA (1985c) found no accumulation of arsenic (both inorganic and organic forms) in rainbow trout (whole body) after 28-

day exposures. A BCF of 4 and a biologic half-life of 1 day was derived for bluegills (*Lepomis macrochirus*) for arsenic (III) (EPA 1978). A BCF of 3 was reported for fathead minnow when exposed to arsenic (V) for 30 days (EPA 1985c). Eisler (1988a) also indicated that bioconcentration of arsenic by aquatic organisms is generally low (BCF less than 17). Oladimeji et al. (EPA 1985c) found that pre-exposure of rainbow trout to arsenic (III) enhanced the elimination of subsequent dosing. These studies suggest that some elimination pathway is induced in freshwater fish through exposure to low (nonlethal) doses of arsenic (III). Reduced growth and survival has been reported in immature bluegills when arsenic concentrations in their muscle tissue exceed 1.3 mg/kg fresh weight (Eisler 1988a).

In an early life-stage test, the narrow-mouthed toad (*Gastrophryne carolinenesis*) had an EC_{50} of 40 μg/L (As [III]) with a 7-day exposure (Birge 1978). An EC_{50} of 4,450 μg/L based on fatalities and deformities was reported for the marbled salamander (*Ambystoma opacum* after 8 days of exposure (EPA 1984c).

Terrestrial Wildlife Toxicity

LD_{50}s for birds of 2,000 mg arsenic/kg body weight showed signs of poisoning within 10 minutes. Death was determined to be caused by destruction of blood vessels lining the gut which resulted in decreased blood pressure and shock. Sensitive species of birds include brown-headed cowbirds (*Molothrus ater*) with an LD_{50} of 99.8 mg/kg body weight of copper acetoarsenite. Mallards have an intermediate value of 323 mg sodium arsenite per kg body weight.

No chronic toxicity studies on mallards were located in the literature reviewed for this report. After 56 days of exposure to arsenic in the diet, the NOAEL for 22 week old chickens was 10 ppm (approximately 1.25 mg/kg-body weight) and at 100 ppm there was decreased body weight, feed intake, and egg production (Clement I.C. 1990).

Chronic arsenic poisoning is seldom seen in mammals (Eisler 1988a). Detoxication and excretion (inorganic arsenics are oxidized, biomethylated and excreted in the urine) are rapid, and therefore, the probability of chronic poisoning from continuous ingestion of small doses is rare.

Inorganic arsenic is more acutely toxic than organic arsenic and can cross the placenta in most species of mammals. Early developmental stages are the most sensitive to the effects of arsenic. Malformation has been documented at single oral doses of 2.5 to 33 mg arsenic per kg body weight and at chronic doses of 1 to 10 mg arsenic per kg body weight (animals species not given) (Eisler 1988a). Toxicokinetics varies from species to species and toxicity may vary as well; therefore, extrapolation of animal data from one species to another must be done with caution.

It has been suggested that arsenic may have negligible effects on wildlife; however, a reduction in species diversity may result due to selective destruction of vegetation (Eisler 1988a).

Summary

Very little data are available on the chronic effects of arsenic to biota that could occur in Silver Bow Creek. The toxicity assessment shows that aquatic invertebrates are the most organisms tested, with chronic values (EC_{xx}) ranging from 874 to 22,0404 μg/L. Acute toxicity to fish from arsenic exposure ranges from 10,800 μg/L to 16,010 μg/L. Acute and chronic AWQC for arsenic are 360 μg/L and 190 μg/L, respectively. The established AWQC would be expected to protect all biota in Silver Bow Creek.

8.3.5 CADMIUM

Phytotoxicity

Plant tissue normally contains up to 1 mg cadmium per kg tissue, but it is not essential for growth. Translocation can occur, with the effects of toxicity appearing in leaves (wilting and chlorosis) before growth is retarded (Salisbury and Ross 1969). Wetland plant species such as sedge and alkali bulrush have the capacity to uptake cadmium from the soils. Sedge translocates cadmium rapidly, while alkali bulrush concentrates it in the roots and lower stems. Tule, saltgrass (which is found in the floodplains of Silver Bow Creek), and arrowgrass have the ability to take up cadmium, slowly translocate it, and concentrate it to an average of 74 ppm in their tissues (Lee et al. 1976).

Aquatic Toxicity

The toxicity of cadmium to aquatic life has been reviewed by Eisler (1985) and Korte (1983). There is no evidence that cadmium is an essential mineral for animals (Eisler 1985). For aquatic organisms, the toxicity of cadmium generally decreases as hardness increases. Chronic toxicity values for fathead minnows (*Pimephales promelas*) and *Daphnia magna* tested over a range of hardness values found a significant correlation between hardness and toxicity (EPA 1985d). Thus, water quality criteria have been developed to reflect the relationship of cadmium toxicity and hardness.

The genus *Daphnia* (including *D. magna* and *D. pulex*) ranked third in sensitivity to cadmium, of 44 genera of freshwater animals (EPA 1985d). The genus mean value for *Daphnia* (at a hardness of 50 mg/L as $CaCO_3$) is 26.06 μg/L (EPA 1985d). Aquatic insects are relatively tolerant of cadmium. Mean acute values (hardness of 50 mg/L as $CaCO_3$) for mayflies are 322.8 μg/L (*Paraleptophlebia praepedita*) and 2,310 μg/L (*Ephemerella grandis*)(EPA 1985d). The mean acute value (hardness of 50 mg/l as $CaCO_3$) for caddisflies is 3,400 μg/L and for damselflies is 8,100 μg/L (EPA 1985d).

For fish species, genera *Salmo* and *Onchorhynchus* (based on brown and rainbow trout results) ranked number one in acute sensitivity to cadmium (EPA 1985d). The acute LC_{50} for rainbow trout, based on flow-through tests was 1.75 μg/L at a hardness of 31 mg/L as $CaCO_3$ (Davies 1976). The species mean acute value (for hardness, 50 mg/L as $CaCO_3$) is about 3.6 μg/L (EPA 1985d). A 28-day EC_{50} (based on death and deformity) of 140 μg/L at a hardness of 104 mg/L as $CaCO_3$ has been reported for rainbow trout (Birge 1978; EPA 1985d). The LC_{50} for brown trout, based on static tests is 1.4 μg/L at a hardness of 39 to 48 mg/L as $CaCO_3$ (EPA 1985d). Holcombe et al. (EPA 1985d), reported an LC_{50} of 5,080 μg/L for brook trout at a hardness of 47.4 mg/L as $CaCO_3$ (EPA 1985d). However, Carroll et al. (as cited in Eisler 1985), reported an LC_{50} of less than 1.5 μg/L at a hardness of 42 mg/L as $CaCO_3$. At a hardness of 330 to 350 mg/L, the 96-hour LC_{50} was only slightly higher at 3.8 to 4.4 μg/L (Carroll et al. 1979 as cited in Eisler 1985). Seven-day studies with brook trout resulted in 4.4 percent mortalities at 3.6 μg/L and 30.6 percent mortalities at 60 μg/L (Lehnertz 1989).

A chronic toxicity value (based on early life-stage effects) of approximately 6.7 μg/L was reported for brown trout (*Salmo trutta*) at a hardness of 44 mg/L (as $CaCO_3$) (Eaton et al. 1978 as cited in EPA

1985d). Chronic values for brook trout (*Salvelinus fontinalis*) of 2.045 μg/L and 1.732 μg/L were reported for hardness values (as $CaCO_3$) of 44 and 3 mg/L, respectively (EPA 1985d). Testicular damage was reported for brook trout exposed to 10 μg/L cadmium for 21 days at a hardness of 120 mg/L as $CaCO_3$ (EPA 1985d). A number of long-term exposure studies have been conducted with rainbow trout, but chronic toxicity values have not been determined. Birge et al. (EPA 1985d), observed reduced survival in rainbow trout exposed for 18 months to 0.2 μg/L at a hardness of 112 mg/L as $CaCO_3$. Hughes et al. (EPA 1985d) reported increased gill diffusion in rainbow trout exposed for 234 days to 2 μg/L cadmium at a hardness of 240 mg/L as $CaCO_3$. Physiological effects were reported by Arillo et al. (EPA 1985d), in rainbow trout exposed to 10 μg/L cadmium for 4 months at a hardness of 320 mg/L as $CaCO_3$. Reduced growth and survival were observed by Woodworth and Pascoe (EPA 1985d) in rainbow trout exposed to 100 μg/L cadmium for 47 days at a hardness of 98.6 mg/L as $CaCO_3$. Physiological effects were also reported by Majewski and Giles (EPA 1985d) for rainbow trout exposed for 178 days to 3.6 to 6.4 μg/L cadmium, at a hardness of 82 mg/L as $CaCO_3$. Reduced survival of embryo-larval rainbow trout was reported at a concentration of less than 5 μg/L and a hardness of 100 mg/L as $CaCO_3$ after 62 days of exposure (In EPA 1985d).

Limited information is available on the toxicity of sediments contaminated with cadmium (see Long and Morgan, 1989). Pavlou and Weston (In Pavlou 1987) reviewed proposed limits for cadmium in (marine) sediments and found values ranging from 1 to 6 mg/kg. Francis et al. (1984), conducted static toxicity tests with embryo-larval goldfish (*Carassius auratus*), largemouth bass (*Micropterus salmoides*) and leopard frogs (*Rana pipiens*) exposed to cadmium-enriched freshwater sediments. Cadmium concentrations in sediments ranged from 1 to 1,000 mg/kg, and the concentrations in the overlying water ranged from 1.1 to 76.5 μg/L, respectively. Only largemouth bass exposed to the highest concentration (average measured concentrations were 1,079 mg/kg in sediments and 43.9 μg/L in water) resulted in statistically significant mortality (25 percent). Birge et al. (In Birge 1978), reported statistically significant mortality in rainbow trout (early eyed-egg to 4-days post-hatch) exposed to cadmium-enriched sediments containing 2.15 mg cadmium/kg; the overlying water contained 6.8 μg/L cadmium. Midge larvae exhibited lower survival rates, reduced size, and decreased rates of emergence after exposure to sediments contaminated with cadmium, chromium, and zinc (In Francis et al. 1984). Midge larvae have also shown avoidance behavior to sediments contaminated with more than 422 mg/kg cadmium and more than 8,330 mg/kg zinc (In Francis et al. 1984).

1985d). Chronic values for brook trout (*Salvelinus fontinalis*) of 2.045 μg/L and 1.732 μg/L were reported for hardness values (as $CaCO_3$) of 44 and 3 mg/L, respectively (EPA 1985d). Testicular damage was reported for brook trout exposed to 10 μg/L cadmium for 21 days at a hardness of 120 mg/L as $CaCO_3$ (EPA 1985d). A number of long-term exposure studies have been conducted with rainbow trout, but chronic toxicity values have not been determined. Birge et al. (EPA 1985d), observed reduced survival in rainbow trout exposed for 18 months to 0.2 μg/L at a hardness of 112 mg/L as $CaCO_3$. Hughes et al. (EPA 1985d) reported increased gill diffusion in rainbow trout exposed for 234 days to 2 μg/L cadmium at a hardness of 240 mg/L as $CaCO_3$. Physiological effects were reported by Arillo et al. (EPA 1985d), in rainbow trout exposed to 10 μg/L cadmium for 4 months at a hardness of 320 mg/L as $CaCO_3$. Reduced growth and survival were observed by Woodworth and Pascoe (EPA 1985d) in rainbow trout exposed to 100 μg/L cadmium for 47 days at a hardness of 98.6 mg/L as $CaCO_3$. Physiological effects were also reported by Majewski and Giles (EPA 1985d) for rainbow trout exposed for 178 days to 3.6 to 6.4 μg/L cadmium, at a hardness of 82 mg/L as $CaCO_3$. Reduced survival of embryo-larval rainbow trout was reported at a concentration of less than 5 μg/L and a hardness of 100 mg/L as $CaCO_3$ after 62 days of exposure (EPA 1985d).

Limited information is available on the toxicity of sediments contaminated with cadmium (see Long and Morgan 1989). Pavlou and Weston (Pavlou 1987) reviewed proposed limits for cadmium in (marine) sediments and found values ranging from 1 to 6 mg/kg. Francis et al. (1984), conducted static toxicity tests with embryo-larval goldfish (*Carassius auratus*), largemouth bass (*Micropterus salmoides*) and leopard frogs (*Rana pipiens*) exposed to cadmium-enriched freshwater sediments. Cadmium concentrations in sediments ranged from 1 to 1,000 mg/kg, and the concentrations in the overlying water ranged from 1.1 to 76.5 μg/L, respectively. Only largemouth bass exposed to the highest concentration (average measured concentrations were 1,079 mg/kg in sediments and 43.9 μg/L in water) resulted in statistically significant mortality (25 percent). Birge et al. (Birge 1978), reported statistically significant mortality in rainbow trout (early eyed-egg to 4-days post-hatch) exposed to cadmium-enriched sediments containing 2.15 mg cadmium/kg; the overlying water contained 6.8 μg/L cadmium. Midge larvae exhibited lower survival rates, reduced size, and decreased rates of emergence after exposure to sediments contaminated with cadmium, chromium, and zinc (Francis et al. 1984). Midge larvae have also shown avoidance behavior to sediments contaminated with more than 422 mg/kg cadmium and more than 8,330 mg/kg zinc (Francis et al. 1984).

In general, cadmium does not readily accumulate in edible fish tissues (Heiskary and Helwig 1983). Accumulated cadmium is slowly excreted by freshwater organisms (EPA 1985d; EPA 1985d). Kumada et al. (EPA 1985d), found faster elimination of cadmium consumed in the diet than that taken up from the water column.

BCFs are reported in EPA (1985d) for a variety of aquatic invertebrates. Whole-body BCFs for snails (*Physa integra*) and clams (*Corbicula fluminea*) range from 1,750 to 3,770. BCFs for insects (including mayfly, dragonfly, damselfly, stonefly, beetle, caddisfly, and midge) range from 164 to 4,190. Whole-body values for crustaceans are 320 to 484 for cladocerans and 184 for crayfish. BCFs for plants include 603 for duckweed (*Lemna valdivians*) and 960 for fern (*Salvinia natans*) (EPA 1985d). A BCF of 2,500 has been reported for the algae, *Chlorella vulgaris* (Eisler 1985).

BCFs for cadmium in freshwater fish range from 3 for muscle tissue of brook trout (based on 490 days of exposure) (EPA 1985d) to 2,213 for whole body mosquitofish (*Gambusia affinis*) (based on 180 days of exposure) (EPA 1985d). BCFs for brook trout muscle were somewhat higher for shorter exposure durations. A BCF of 151 was determined for 84 days of exposure (EPA 1985d), and a BCF of 22 was reported for 93 days of exposure for brook trout (EPA 1985d). Whole body BCFs of 33 and 540 were reported for rainbow trout based on 70 and 140 days of exposure, respectively (EPA 1985d). Cadmium is preferentially accumulated in the liver, thus reducing concentrations in the muscle. Rainbow trout exposed to 10 μg/L cadmium for 3 months had BCFs of 4,900 for the liver, 1,740 for gill tissue, 740 for kidney, 160 for spleen, and 100 for heart tissues (Eisler 1985).

Water quality criteria derived to protect freshwater animals should also be protective of freshwater plants according to EPA (1985d), since the lowest toxicity values for plants are higher than the lowest toxicity values for fish and invertebrates. Rachlin et al. (EPA 1985d), determined 96-hour EC_{50} values for a number of species, including: 105 μg/L for the green alga *Chlorella saccarophila*, 120 μg/L for the alga *Anabaena flos-aquae*, 310 μg/L for the diatom *Navicula incerta*, and 480 μg/L for the diatom *Nitzschia costerium*.

Terrestrial Wildlife Toxicity

Cadmium may be accumulated through the food chain in sufficient quantities to be harmful to higher trophic levels (Thoman et al. 1974). It has a toxic effect on a variety of birds and mammals. The know effects on ducks are all sublethal, primarily resulting in growth retardation, anemia, and testicular damage. Studies by White and Finley (1978a,b) and White, Finley, and Farrell (1978) show that no weight loss or mortality occurred in adult mallards fed concentrations of cadmium up to 200 ppm in the diet. Egg production was suppressed in ducks fed 200 ppm dietary cadmium, but not in those fed lower concentrations; however, mild to severe kidney lesions developed in ducklings fed 20 ppm cadmium in the diet. Altered avoidance behavior was observed in young ducks who parents were fed 4 ppm dietary cadmium for about 4 months before egg laying. This behavior is considered detrimental to wild birds (Eisler 1985).

Cadmium may compete for binding sites on proteins and thus may inhibit enzymatic reactions. Zinc, iron, and selenium have an antagonistic effect on the toxicity of cadmium whereas lead and mercury exacerbate it (Eisler 1985).

The lowest oral dose of cadmium producing death in tested mammals was 150 mg/kg body weight cadmium fluoride in guinea pigs (Eisler 1985).

Summary

Chronic toxicity of cadmium to aquatic receptors in Silver Bow Creek is dependent upon the hardness of the water. Literature values for cadmium toxicity (chronic) were reflective of low hardness conditions (<50 mg/L $CaCO_3$). Chronic values for brook trout, which is the most sensitive trout, range from 1.7 to 2.0 μg/L at water hardness less than 50 mg/L $CaCO_3$. Chronic AWQC that would be protective of trout is 1.1 μg/L at a water hardness of 100 mg/L $CaCO_3$. Therefore, the AWQC should be protective of fish species residing in Silver Bow Creek.

8.3.6 CHROMIUM

Phytotoxicity

Chromium is beneficial but not essential to growth of terrestrial plants. Chromium residue in plants seldom exceed 1 to 2 ppm except for those grown on chromium amended soils or in serpentine areas. Even plants with elevated levels of chromium do not show any signs of toxicity. However, concentrations of 1 mg/L soluble chromium have been shown to inhibit growth of roots and shoots (Eisler 1986).

In general, the toxicity of trivalent chromium to mammals is low because its membrane permeability is poor and it is noncorrosive. There is little tendency for trivalent chromium to biomagnify in the foodchain in the inorganic form. Hexavelent chromium is more toxic because of its oxidizing potential and its ease in penetrating the biological membranes. Little hexavelent chromium is anticipated to be present in surface waters as it is readily reduced to the trivalent form in the presence of organic compounds (Eisler 1986).

Generally, invertebrates are more sensitive to chromium (VI) than fish. Acute toxicity information for chromium (VI) is available for at least seven species of fish. The LC_{50}s range from 17,600 μg/L for fathead minnow to 249,000 μg/L for goldfish. Wallen et al. (as cited in EPA 1984d), conducted a study with mosquitofish on the effects of chromate and dichromate potassium and sodium salts. Base don chromium, the dichromate salts at 95,000 μg/L were more toxic than the chromate salts at 120,000 μg/L. In another study of potassium dichromate and potassium chromate, the 96 hour LC_{50} was 110,000 μg/L and 170,000 μg/L, respectively, for bluegill (EPA 1984f).

Studies on the chronic toxicity of brook and rainbow trout have been completed. Benoit (EPA 1984b) found that, based on survival, the limits of chromium (VI) are 200-350 μg/L with a chronic value of 265 μg/L. Growth retardation was noted, but this was a temporary effect. Sauter et al. (EPA 1984f) also noted a temporary size reduction in their study on rainbow trout. They also determined that in an early life study, 51 μg/L and 105 μg/L of chromium (VI) with a chronic value of 73 μg/L were the limits for rainbow trout. Benoit calculated the acute-chronic ratios for chromium (VI) with brook and rainbow trout as 220 and 260 respectively.

Chromium (III) has been found to more acutely toxic than chromium (VI) in four fish species (EPA 1984f). Fifteen 96 hour LC_{50} values for chromium (III) have been calculated for eleven freshwater fish species. These LC_{50}s range from 3,330 μg/L for guppies in soft water to 71,900 μg/L for bluegill in hard water. A chronic value for chromium (III) in hard water for fathead minnow is 1,020 μg/L. The mean acute-chronic ratio for fish is 27.

Acute toxicity data indicates that hexavalent chromium is more toxic than chromium (III) in comparatively soft and acidic freshwaters, that younger organisms are more sensitive, and that 96 hours is insufficient to attain stable mortality patterns (Eisler 1986).

The toxicity of chromium to resident biota in Silver Bow Creek is based upon chromium (VI). Chronic values for fish from the literature show that chromium VI concentrations range from 73 μg/L to 265 μg/L. The chronic AWQC value for chromium VI is 11 μg/L, and therefore would be protective of resident trout species.

8.3.7 COPPER

Phytoxicity

Copper is an essential micronutrient for plant life (EPA 1976). It is absorbed as a divalent cupric or monovalent cuprous ion, existing in plants primarily in the cupric form (Salisbury and Ross 1969). Copper is vital in the functioning of certain enzymes and essential in the synthesis of chlorophyll molecules (EPA 1976). Normal content in plants varies between 5 and 15 ppm, but higher concentrations can be found (Antonovics et al. 1971). Roots are the primary storage site for copper, with high concentrations also found in chloroplasts (Reuther 1957). Copper uptake is regulated by an internal mechanism for tolerance. At low copper concentrations, uptake in above ground parts is low. As concentrations increase, uptake remains low until a threshold concentration is reached and uptake increases abruptly. The threshold concentration of copper is different for each species. The recommended safe levels for irrigation water are 0.2 to 5.0 mg/L (Geonomics 1978).

Many emergent marsh species are able to absorb and accumulate copper from solution. Bulrush, sedge, iris, cattail, and reed absorb concentrations of 4.8, 5.6, 5.7, 4.7, and 4.2 mg copper per kg

dry weight, respectively (Lee et al. 1976). Although copper can concentrate to high levels in plant tissue, no indication of bioaccumulation in the food chain has been noted.

Some submergent plants (waterweed, water milfoil, pickerelweed, pondweed, and blue-green algae) are unaffected by copper concentrations of 10 mg/L at a pH 8.1. Many blue-green algae species can tolerate high copper concentrations as long as conditions remain aerobic (Becker and Thatcher 1973). However, copper is toxic to aquatic plants and has been widely used as an algicide and aquatic herbicide (EPA 1985e). No "final plant value" has been derived by the EPA (1985e) because of insufficient data. In general, concentrations that cause growth inhibition in plants are much higher than chronic toxicity values that are available for fish and invertebrates (EPA 1985e). The 7-day EC_{50} reported for duckweed (*Lemna minor*) is 119 μg/L (EPA 1985e). The 33-day EC_{50} (based on growth effects) is 180 μg/L for the alga *Chlorella vulgaris* (Rosko and Rachlin 1977 as cited EPA 1985e). Rachlin et al. (EPA 1985e) reported a 96-hour EC_{50} of 550 μg/L for the alga *Chlorella saccharophila*. A 14-day EC_{50} (based on cell volume) of 85 μg/L was reported for the green alga *Selenastrum capricornutum* (EPA 1985e).

Aquatic Toxicity

The toxicity of copper to aquatic animals was reviewed by Harrison (1986) and Demayo et al. (1982a). Copper is an essential mineral for animals, since it comprises an essential part of many enzymes and it is important in hemoglobin formation (NAS 1980; Rand and Petrocelli 1985). The primary mechanism of acute copper toxicity in fish is probably osmoregulatory disruption and failure, rather than gill destruction and hypoxia (Rand and Petrocelli 1985). Data suggest that acclimation increases tolerance to copper. Continued ingestion of copper in excess of nutritional requirements leads to accumulation, especially in the liver (Rand and Petrocelli 1985). Copper toxicity decreases with increasing water hardness (Rand the Petrocelli 1985) and water quality criteria have been developed to reflect this relationship.

In acute aquatic tests, the most sensitive invertebrate species are cladocerans; the average acute value for the genus *Daphnia* (including *D. magna, D. pulex*, and *D. pulicaria*) is 17.08 μg/L (hardness 50 mg/L as $CaCO_3$), and the mean genus value of *Ceriodaphnia* is 18.77 μg/L (hardness 50 mg/L as $CaCO_3$) (EPA 1985e). Some aquatic insects are among the least sensitive to copper of the species

tested. Mean acute values (adjusted to hardness of 50 mg/L as $CaCO_3$) are 10,240 μg/L for stoneflies, 6,200 μg/L for caddisflies, and 4,600 μg/L for damselflies (EPA 1985e).

Trout are among the most sensitive to copper of tested fish species. The mean acute toxicity value for rainbow trout is 42.5 μg/L at a hardness of 50 mg/L as $CaCO_3$ (EPA 1985e). This value is based on the results of 40 acute tests and the pooled slope of toxicity to hardness. The mean acute value for cutthroat trout (*Oncorhynus clarki*) at a hardness of 50 mg/L as $CaCO_3$ is 66.26 μg/L, based on nine acute tests (EPA 1985e). The LC_{50} is 100 μg/L for brook trout at a hardness of 45 mg/L as $CaCO_3$ (EPA 1985e). Rainbow trout are apparently more sensitive to the acute effects of copper than cutthroat or brook trout.

Based on available information, it is not definitive which trout species is most sensitive to chronic exposure to copper. The lowest maximum acceptable toxicant concentration reports for aquatic animals is 3.873 μg/L for the brook trout (*Salvelinus fontinalis*), at a hardness of 37.5 mg/L as $CaCO_3$ (EPA 1985e). However, rainbow trout are more sensitive to copper than brown and brook trout, based on results of early life stage studies by McKim et al. (1978 as cited in EPA 1985e). The chronic value for rainbow trout is 19.01 μg/L at a hardness of 45.4 mg/L as $CaCO_3$; at the same hardness, the chronic values are 30.83 μg/L for brown trout and 31.15 μg/L for brook trout (McKim et al. 1978 as cited in EPA 1985e). Thus, in this study brown trout and brook trout had similar sensitivities to copper. McKim and Benoit (1971 in EPA 1985e) reported a chronic value for brook trout (life-cycle test) of 12.86 μg/L at a hardness of 45 mg/L as $CaCO_3$ that is lower than the values reported above by McKim et al. (1978 as cited in EPA 1985e) at the same hardness. Literature values for copper show that chronic levels for copper in trout species range from 12.86 μg/L to 31.15 μg/L. The most sensitive trout was the brook trout (12.86 μg/L), which is found in the Silver Bow Creek drainage. The chronic AWQC established for aquatic species (12 μg/L), based upon a water hardness of 100, should be protective of brook trout and other trout species in Silver Bow Creek.

Limited information is available on the toxicity of freshwater sediments contaminated with copper. Redox potential, pH, and organic carbon content can affect the bioavailability and toxicity of metals in sediments (Besser and Rabeni 1987). Pavlou and Eston (1983 as cited Pavlou 1987) reviewed proposed limits for copper in sediments and found values ranging from 25 to 50 mg/kg. In a laboratory study, Cairns et al. (1984) reported that 10-day LC_{50}s for aquatic benthic invertebrates (the

midge *Chironomus tentans*, and the amphipods *Gammarus lacustris* and *Hyalella azteca*) ranged from 857 to 2,296 mg/kg copper per kg of sediment. For *Daphnia magna*, the 48-hour LC_{50}s were 937 and 681 mg/kg, for sediments with f_{oc} (fraction organic carbon) values of 1.8 percent and 3.0 percent, respectively (Cairns et al. 1984).

Copper has a low potential for bioaccumulation in freshwater organisms (EPA 1987b). A muscle BCF factor of 1.80 was reported for bluegill (*Lepomis macrochirus*) exposed for 660 days (EPA 1985e). A BCF factor of 290 was reported for fathead minnows exposed for 30 days (EPA 1980a). No BCFs were reported by the EPA (1985e) for trout species. A BCF of 203 was reported for the stonefly, *Pteronarcys californica*, after 14 days of exposure (EPA 1985e). The freshwater alga, *Chlorella regularis*, had a BCF of 2,000 after 20 hours of exposure (EPA 1985e). In part, this high BCF is likely due to absorption of copper to the cell surface.

Terrestrial Wildlife Toxicity

Toxicity to copper for most mammals and birds appears to be insignificant as copper absorption is limited. It is a required element in animal diets, with deficiencies often observed (EPA 1985e).

Data are available on the toxicity of copper in wild birds. Canada geese (*Branta canadensis*) developed acute copper toxicosis after ingesting pond water containing 100 mg/L copper (as copper sulfate) (NAS 1980). The NOAEL in young turkeys exposed to copper through dies for 21 days in 50 ppm (approximately 6.25 mg/kg), and at 100 ppm reduced growth was reported (NAS 1980). Ducks showed increase growth after exposure to 100 ppm copper, but thinning of the cecal wall was also reported (NAS 1980). After 4 weeks of exposure to 324 ppm copper, young chickens had muscular dystrophy and retarded growth (NAS 1980).

8.3.8 LEAD

Phytotoxicity

Phosphorous and calcium are known to have antagonistic effects on lead toxicity (Lee et al. 1976). Lead and cadmium exchange synergistic and antagonistic effects. When lead concentrations are lower

than cadmium concentrations, the toxicity of cadmium is increased. When concentrations of lead are greater than cadmium, the toxicity of cadmium is decreased. Lead and arsenic also react synergistically (Antonovics et al. 1971).

Concentrates of lead in irrigation water ranging between 5 and 20 mg/L should be restricted, since inorganic lead salt buildup can be toxic (Geonomics 1978). Lead concentrations in plants normally range between 3 and 4 ppm. Apparently, 50 ppm is an upper tolerance limit for most vascular plants. No sublethal effects of toxicity are noticeable, but death occurs after 50 ppm (Salisbury and Ross 1969; Antonovics et al. 1971). Lead uptake is constant with increasing lead levels in the soil until a point is reached when uptake becomes unrestricted and rises abruptly. Species can rarely tolerate conditions above the level at which there is a sudden increase in lead uptake (Antonovics et al. 1971). There is no convincing evidence for terrestrial vegetation playing an important role in the biomagnification of lead through the food chain (Eisler 1988b).

Adverse effects on aquatic plants have been reported at concentrations from 500 to 63,800 μg/L, and these values are well above concentrations that would be protective of freshwater fish and invertebrates (EPA 1985f). Growth inhibition (35 to 53 percent) was reported at 500 μg/L in three species of algae (*Scenedesmus* sp., *Selenastrum* sp., and *Chlorella* sp.) (EPA 1985f).

Aquatic Toxicity

Among sensitive aquatic species, especially early life stages, dissolved lead is more toxic than total led, and deleterious effects of lead are more pronounced in soft waters with low pH over longer exposure periods. Aquatic flora readily take up lead from solution through a passive mechanism. Although lead is concentrated from water, no convincing evidence exists that it is transferred through the food chain. Lead concentrations were found to decrease markedly with increasing trophic level in a grazing aquatic food chain (Eisler 1988b).

The toxicity of lead to aquatic life was reviewed by Eisler (1988b) and Demayo et al. (1982b). Sublethal toxic effects in vertebrates include neurological effects, kidney dysfunction, and anemia (Ran and Petrocelli 1985). Davies et al. (1976) reported spinal curvatures and caudal erosion in

chronic studies with rainbow trout. Lead toxicity to aquatic animals decreases with increasing water hardness and water quality criteria have been established to reflect this relationship.

In acute assays with aquatic animals, invertebrates are more sensitive then vertebrates. A 48-hour EC_{50} of 124 μg/L is reported for amphipod (*Gammarus pseudolimnaeus*) at a water hardness of 46 mg/L as $CaCO_3$ (Sephar et al. 1978 as cited in EPA 1985f). The cladoceran *Daphnia magna* is also sensitive to lead and has an acute toxicity value (adjusted for hardness, 50 mg/L as $CaCO_3$) of 447.8 μg/L (EPA 1985f). LC_{50} values were not reported in EPA (1985f) for mayflies, stoneflies, and caddisflies. Cladocerans are the most sensitive invertebrate species tested with respect to chronic toxicity of lead. The chronic value for *Daphnia magna*, based on life cycle tests and a water hardness value of 52 mg/L (as $CaCO_3$), is 12.26 μg/L (In manuscript in EPA 1985f).

Trout are the most acutely sensitive freshwater fish species to lead, based on results reported by the EPA (1985f). The mean acute values (for hardness equalling 50 mg/L as $CaCO_3$) are 2,448 μg/L for rainbow trout and 4,820 μg/L for brook trout (EPA 1985f). Brook trout have a chronic value of 83.08 μg/L at a hardness of 44 mg/L as $CaCO_3$ (EPA 1985f).

Information on the toxicity of lead-contaminated sediments is limited. Pavlou and Eston (1983 in Pavlou 1987) reviewed proposed limits for lad in (marine) sediments and found values ranging from 40 to 50 mg/kg.

Fish accumulate lead primarily in the epidermis and intestines, and little is accumulated in muscle. The kidney was found to accumulate lead the most followed by opercular bone, gill arch, and liver in a study with rainbow trout (other tissues were not analyzed) (Goettl and Davies 1979). BCFs were reported by the EPA (1985f) for only two freshwater fish species. A whole-body BCF factor of 45 was reported for bluegills (exposure duration was not specified) (Atchison et al. 1977 as cited in EPA 1985f). A whole-body BCF of 42 was reported for brook trout exposed for 140 days; these fish were from embryo state to age 3 months. Freshwater fish accumulate alkyl-lead compounds more readily than inorganic lead forms according to Eisler (1988b). Whole-body BCFs for snails range from 738 to 1,700 (EPA 1985f). BCFs of 86 and 1,120 were reported by the EPA (1985f) for stonefly. A BCF of 499 was reported for caddisfly (EPA 1985f). Mayfly showed a BCF of 2,366 after 14 days'

exposure (EPA 1985f). BCFs for algae showed a wide variation with species, exposure concentration, and exposure duration. Values ranged from 20 to 92,000 (Eisler 1988b).

Terrestrial Wildlife Toxicity

Lead poisoning in waterfowl has been recognized for many years. It is estimated that 2 to 3 percent of all fall populations of waterfowl dies of lead poisoning. The primary source of lead for waterfowl is consumption of spent shot in heavily hunted areas as waterfowl pick up gravel and shot with food. A number of studies have been conducted on toxic effects of ducks resulting from consumption of spent lead shot (Irwin and Karsted 1972; Dieter and Finley 1979; and Koranda et al. 1979). These studies show that a single lead pellet (about 200 mg) can result in dysfunction of blood enzymes, brain tissue abnormalities, weight loss, and death. Consumption of greater quantities of lead accentuates these results. It was noted that ducks fed a high calcium dies survived high lead dosages, suggesting that calcium prevents the absorption of lead.

Lead poisoning of carnivorous birds usually results from the ingestion of lead shop in the foot items. Ingestion of food containing biologically incorporated lead contributes to the body burden of the bird but is unlikely in itself to result in clinical lead poisoning (Eisler 1988b).

Signs of lead poisoning in domestic and laboratory animals are similar to those in humans. Wildlife data are lacking in the literature. The general lead in the diet could result in reduced populations of species because of stillbirths and abortion and reduced learning ability that could cause young to be more susceptible to predators (Eisler 1988b).

For resident biota in Silver Bow Creek, trout species are among the most sensitive to lead toxicity. Chronic literature values for lead are limited, with only one value (83.08 μg/L) being reported. This value was based upon a water hardness of 44 mg/L of $CaCO_3$. The chronic AWQC for lead, 3.2 μg/L, is protective of sensitive species expected to occur in Silver Bow Creek.

8.3.9 ZINC

Phytotoxicity

Zinc is an essential micronutrient for normal plant growth. It is present in all plants in varying concentrations. Zinc is required in the product of precursors of the plant hormone, auxin. Auxin is a growth regulator; therefore, zinc deficiencies result in poor growth, morphological and physiological deformities, and chemical imbalance (Salisbury and Ross 1969). Deficiencies are more frequent than toxicities. Concentrations in the range of 10 to 35 ppm in leaves are adequate for most plants. Some (e.g., ragweed) accumulate over 4,000 ppm. Zinc deficiencies reduce the amount of nitrogen in plan tissue and increase the potassium and phosphorus content to higher than normal levels (Antonovics et al. 1971).

A linear relationship exists between the amount of zinc found in soils and its concentrations in plant tissue (Antonovics et al. 1971). However, that relationship does not extend to the concentration of zinc found in irrigation water and that in plant tissue. It is recommended that irrigation waters contain 10 mg/L zinc or less, because of the narrow margin between deficiency and toxicity (Geonomics 1978).

Studies using wetland plant species, such as bulrush, sedge, cattail, and reed, indicate relatively high zinc absorption ability. Tissue values of 50, 63, 47, and 37 mg/kg dry weight, respectively, indicate the high tolerance of these species to zinc (Seidel 1976). Hydroponic studies using eight marsh species and four different salinity levels indicated that all species studied had the ability to take up and translocate zinc (Lee et al. 1976).

Aquatic vegetation appears generally tolerant of high levels of zinc. However, zinc can function as an algicide. Concentrations of 70 mg/L are lethal to the alga, *Selenastrum*. Toxicity of zinc is governed by the metabolic rate of the organism in a linear fashion (Bartlett and Rabe 1974).

Adverse effects have been reported for freshwater plants (20 species) at concentrations ranging from 30 to greater than 200,000 μg/L (EPA 1987a). Very limited information is available on the influence of hardness on the toxicity of zinc to aquatic plants. An EC_{50} of 50.9 μg/L was reported for the

green alga *Selenastrum capricornutum* based on effects on biomass during 14 to 21 days of exposure (EPA 1987a). A 4-day EC_{50} of 7,000 μg/L was reported by Rachlin et al. (EPA 1987a) for the green alga *Chlorella saccharophila*. The 28-day EC_{50} for duckweek (*Lemma minor*) based on tissue damage and death is 67,700 μg/L (EPA 1987a). A 40-day EC_{50} of 10,000 μg/L was reported for duckweed based on growth effects (EPA 1987a).

Aquatic Toxicity

Zinc is an essential trace element for animals, and it is important to cell growth and differentiation and in the formation of a number of metalloenzymes (Rand and Petrocelli 1985; NAS 1980). The toxicity of zinc to aquatic life has been reviewed by Taylor et al. (1982). Acute toxicity to fish results from gill destruction and hypoxia (Rand and petrocelli 1985). Exposure of fish to sublethal concentrations of zinc can cause extensive edema and necrosis of liver tissue (Rand and Petrocelli 1985). Zinc toxicity decreases as water hardness increases (EPA 1987a), and water quality criteria have been developed that reflect this relationship.

Cladocerans are the most sensitive aquatic animal species tested with zinc (EPA 1987a). The genus *Ceriodaphnia* was the most sensitive of 35 genera reported; the mean acute value (hardness equalling 50 mg/L) is 93.95 μg/L. The genera *Daphnia* was the fourth most sensitive genus; the mean acute value is 299.8 μg/L. Damselflies (*Argia sp.*) were the most tolerant species tested, with an acute value of 88,960 μg/L (for hardness equalling 50 mg/L as $CaCO_3$) (EPA 1987a). Acute toxicity values for stoneflies, mayflies, and caddisflies were not reported in EPA (1987a). In chronic studies the lowest maximum allowable tissue concentration (MATC) reported for an invertebrate was 47 μg/L for *Daphnia magna* (EPA 1987b, 1987a), at a hardness of 104 mg/L as $CaCO_3$.

Trout are among the most sensitive fish tested in acute bioassays with zinc (EPA 1987a). The mean acute value (hardness equalling 50 mg/L as $CaCO_3$) for rainbow trout is 689.3 μg/L based on numerous tests reported by the EPA (1987a). The mean acute value (hardness equalling 50 mg/L as $CaCO_3$) for brook trout is 2,100 μg/L (EPA 1987a). Nehring and Goettl (1974) evaluated the toxicity of zinc to four trout species and reported 14 day LC_{50}s of 410 μg/L for rainbow trout (hardness equalling 22 to 55 mg/L as $CaCO_3$), 670 μg/L for cutthroat trout (hardness equalling 34 to 54 mg/L as $CaCO_3$), and 960 μg/L for brook trout (alkalinity equalling 34 to 54 mg/L, hardness not

measured). Based on an analysis of reported relative sensitivities, Nehring and Goettl (1974) determined the following order of sensitivity to zinc: brook trout (least sensitive) < brown trout < cutthroat trout < rainbow trout (most sensitive). Davies (1980) reported 96 hour LC_{50}s for rainbow trout (170 mm) of 105 μg/L (hardness equalling 36.7 mg/L as $CaCO_3$) and 186 μg/L (hardness equalling 39.2 mg/L as $CaCO_3$) in aerated and nonaerated tests, respectively.

The flagfish (*Jordanella floridae*) had an MATC of 36.4 μg/L (hardness equalling 44 mg/L as $CaCO_3$) and was the most sensitive of seven fish species tested (EPA 1987b). Trout are apparently not as sensitive to the chronic effects of zinc as flagfish, guppy, or fathead minnows. The chronic value (based on a life-cycle test) for brook trout is 854.7 μg/L (hardness equalling 45.9 mg/L as $CaCO_3$) (EPA 1987a). Chronic values based on early life-cycle tests with rainbow trout are 276.6 μg/L (hardness equalling 26 mg/L as $CaCO_3$) reported by Sinley et al. (EPA 1987a) and 603.0 μg/L (hardness equalling 25 mg/L as $CaCO_3$) reported by Cairns et al. (EPA 1987a).

Limited information is available on the toxicity of zinc-contaminated sediments. Pavlou and Eston (1983 as cited Pavlou 1987) reviewed proposed limits for zinc in (marine) sediments and found values of 75 to 100 mg/kg (wet weight). Birge et al. (Birge et al. 1978) reported statistically significant mortality in rainbow trout (early eyed-egg stage through 4 days posthatch) exposed to zinc-enriched sediment with a measured concentration of 121.4 mg/kg. The overlying water had 21.2 μg/L zinc. Midge larvae have shown lower survival rates, reduced size, and decreased rates of emergence after exposure to sediments contaminated with zinc, cadmium, and chromium (Francis et al. 1984). Midge larvae have also shown avoidance behavior to sediments contaminated with more than 8,330 mg/kg zinc and more than 422 mg/kg cadmium (Francis et al. 1984).

Bioaccumulation in the food chain does not appear to occur because of the formation of insoluble complexes with calcium in plant tissues. These complexes are unavailable to animals. Zinc BCFs of 51 to approximately 1,000 have been determined in freshwater fish (EPA 1987a), based on limited information. A whole body BCF of 417.3 was reported for flagfish (*Jordanella floridae*) following 100 days of exposure (EPA 1987a). Similar values were reported for guppy (*Poecilla reticulata*); whole body BCFs of 466.3 to 965.5 were determined from three tests (each of 134 days) by Pierson (EPA 1987a). A BCF (whole body) of 51 was reported for Atlantic salmon (*Salmo salar*) exposed, in freshwater, for 80 days (EPA 1987a). Nehring (EPA 1987a) reported BCFs of 1,130 for mayfly and

106 for stonefly, after 14 days exposure. BCFs for green algae of 133 and 210 were reported by Coleman et al. (EPA 1987a). Coleman et al. also reported a BCF of 144 for the freshwater plant, euglena (*Euglene virdis*).

Terrestrial Wildlife Toxicity

In general, studies conducted on Japanese quail, chickens, and turkey indicated evidence of reduced body weight at 270 ppm, 800 ppm, and 4,000 ppm, respectively (NAS 1980). The NOAEL is 1,000 ppm (approximately 123 mg/kg-body weight) for 1-day-old chickens exposed to zinc for 4 weeks, and reduced growth occurred at 1,500 ppm (NAS 1980). After 2 weeks, young Japanese quail exposed to doses as low as 125 ppm showed decreased hemoglobin levels and hematocritis, but no significant adverse effects were observed at 62.5 ppm (NAS 1980). Several effects including decreased body weight, paralysis of the legs, low hemoglobin and hematocrit concentrations, decreased pancreas, and gonad weights were observed in ducks after 60 days of exposure to 3,000 ppm zinc (the lowest concentration tested) (NAS 1980).

The chronic AWQC for zinc is 100 μg/L, at a water hardness of 100 mg $CaCO_3$/L. This criteria value would be protective of trout in Silver Bow Creek.

8.4 UNCERTAINTIES ASSOCIATED WITH THE TOXICITY ASSESSMENT

Aquatic Organisms

Toxicity of selected metals and organics to aquatic organisms has been evaluated, and applied to determine toxicity criteria based upon the NOAELs, LOAELS, and EPA AWQC (EPA 1987b). Actual exposures would occur to several COCs simultaneously, and toxic effects could be additive, synergistic, or competitive. If actual toxic effects are additive or synergistic, risks stated in terms of single chemical toxicity criteria could be underestimated. If actual effects are competitive, risks could be overestimated. However, in most cases additivity best describes the toxicity of complex chemical mixtures.

The limited number of surface water samples collected from Silver Bow Creek and its tributaries did not coincide with seasonal spawning periods. Therefore, use of these data to estimate risk to aquatic species may result in an underestimate or overestimate of risk to sensitive life stages.

The AWQC are derived from toxicity tests using a number of representative species. There is evidence for complex effects of long-term exposure, including the development of less sensitive populations in areas of chronic pollution; and conversely, increased sensitivity of some species after long-term exposure to sub-lethal concentrations of metals. Acclimination to metals could cause remaining populations to be less sensitive. In that case, AWQC would be overprotective of species likely to occur in SBC. In the event of increased sensitivity after long-term exposure, the application of AWQC would underestimate the risks. For the three COCs associated with Montana Pole, uncertainties are further decreased because aquatic toxicity data for species known or expected to reside in study area surface waters are used to evaluate risks.

Riparian Plants and Wildlife

Toxicity of metals to plants and wildlife found at the site is generally unknown. Lack of vegetation and suitable habitats discourages use of the area by most species of wildlife. The toxicity data reviewed are predominantly based on laboratory strains of birds and animals or on domesticated plants, and some uncertainty exists when such data are applied to environmental settings. Any assessment and inter-species comparison of toxicities could result in an under- or over-estimation of risk.

8.5 ECOLOGICAL RISK CHARACTERIZATION

The ecological risk evaluation is similar to human risk evaluation, in that exposure assumptions and toxicological data are combined with site data to estimate risk. However, nonhuman receptors vary greatly in physiology and behavior, and it is difficult to quantify risk. Thus, this ecological risk assessment is a qualitative discussion of potential risks and how these risks might affect biological receptors at the Montana Pole site. Risks to wildlife and vegetation in the Montana Pole site are qualitatively discussed in relation to toxicological information from the literature. Each receptor population is discussed in detail in the sections that follow.

8.5.1 RISKS TO AQUATIC LIFE

8.5.1.1 Inorganic Chemicals of Concern

Metals and arsenic found in sediments and surface water in Silver Bow Creek may be a primary reason for the lack of diversity and productivity of the reaches of Silver Bow Creek adjacent to the site. Elevated concentrations of these contaminants come from historical mining activity in the upper reaches of the Silver Bow Creek drainage. The Montana Pole wood treating site is not considered to be a source of metals contamination in the area.

A risk assessment which considered metals, arsenic and selenium as chemicals of concern for ecological receptors was recently completed for upper Silver Bow Creek, including that section adjacent to the Montana Pole site (Lower Area One (LAO) Preliminary Baseline Risk Assessment, CDM-FPC 1991). This assessment was based on much more extensive metals data than that available in the Montana Pole site data set. The assessment was able to consider potential sources of contaminants and estimate loading of metals at various points along the creek. In addition, some information was available to assess impacts at high and low creek flows and during storm water run-off events.

Rather than attempt to repeat this assessment for inorganic chemicals of concern using a more limited database, the findings of the LAO assessment are summarized, and are assumed to represent, in the absence of remediation, current and future impacts on Silver Bow Creek due to metals contamination. Since the Montana Pole site is not considered a source for inorganic contaminants in Silver Bow Creek, lack of a specific assessment of metal contamination in the creek is unlikely to underestimate ecological risks due to chemical migrating from the Montana Pole site.

The LAO risk assessment indicates that concentrations of aluminum, cadmium, copper, iron, lead and zinc in Silver Bow Creek present potential risks to aquatic life through continued exceedances of AWQC (p. 5-67, Table 5-12). In spite of the potential upstream source of organisms, the creek has apparently been devoid of life until recently. Although some algae and invertebrates are currently found in certain stretches of the creek, population density and diversity is low. The lack of any fish population is the most obvious sign that the creek is under severe stress. The document concludes

that "There is little reason to believe that future conditions will change significantly from current conditions unless remedial action is taken to remove sources of contaminants to Silver Bow Creek" (p. 5-68).

8.5.1.2 Organic Chemicals of Concern

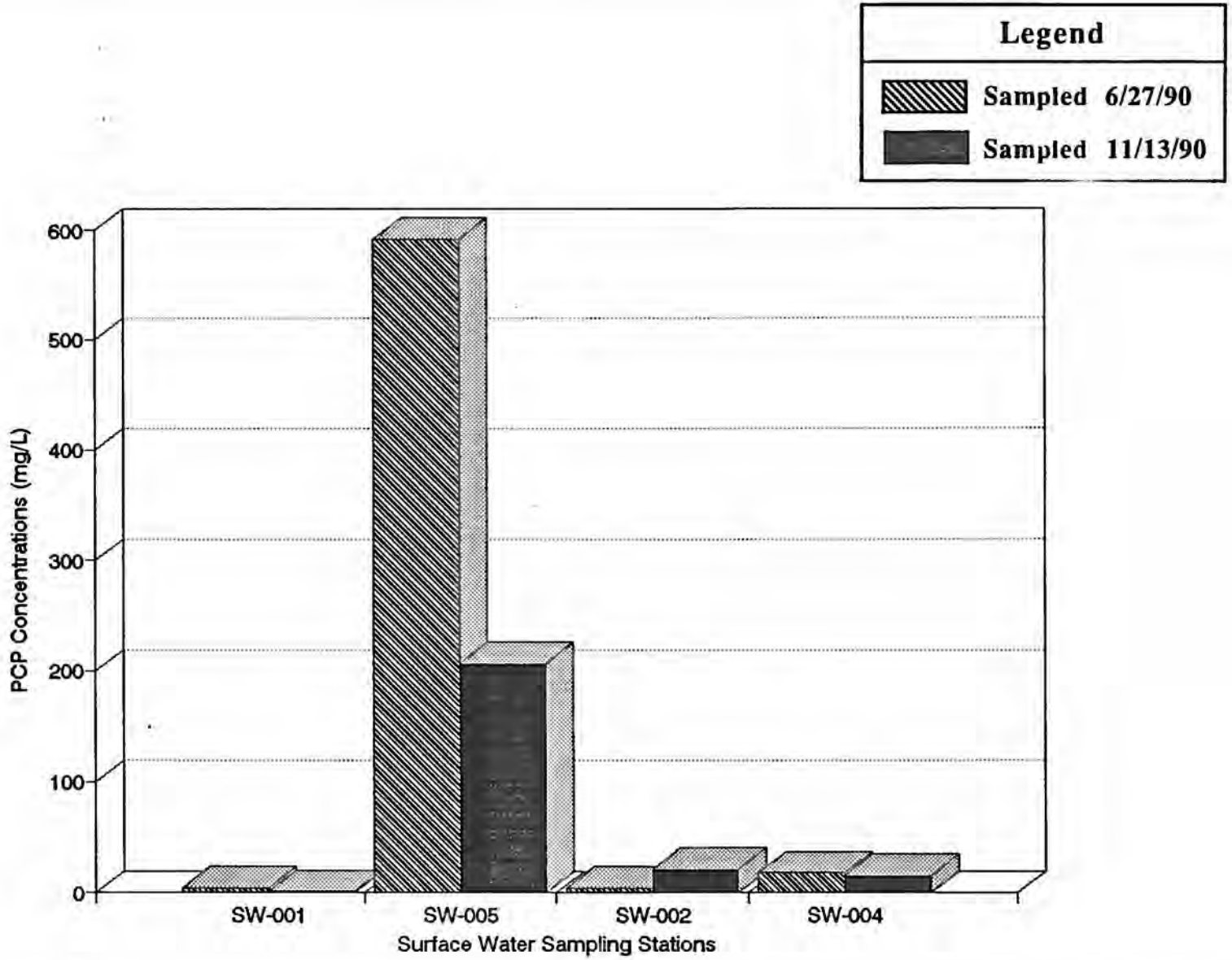
Dioxins/furans, PAHs and PCP have all been detected in surface water and/or sediments in stream reaches adjacent to the Montana Pole site. A seep where groundwater discharges into the creek can be detected visually near the location of surface water sampling station SW-005. These chemicals are currently being released to surface water, and may pose a threat to aquatic life.

The stress on the Silver Bow Creek system from inorganic contamination limits the potential receptors for exposure to organic chemicals. In particular, the lack of fish greatly shortens the aquatic food chain by eliminating the higher trophic levels in the aquatic food chain in Figure 8-3. Further, lack of food sources (aquatic plants, insects and other invertebrates, small fish) make upper Silver Bow Creek unattractive for larger animals such as migratory waterfowl or raptors. It is unlikely that such animals would spend any significant time in stretches of the creek near the Montana Pole site. Any impact of organic contamination from the Montana Pole site should be considered only a potential, especially when such impacts are due to hypothetical biomagnification of chemicals near the top of the food web.

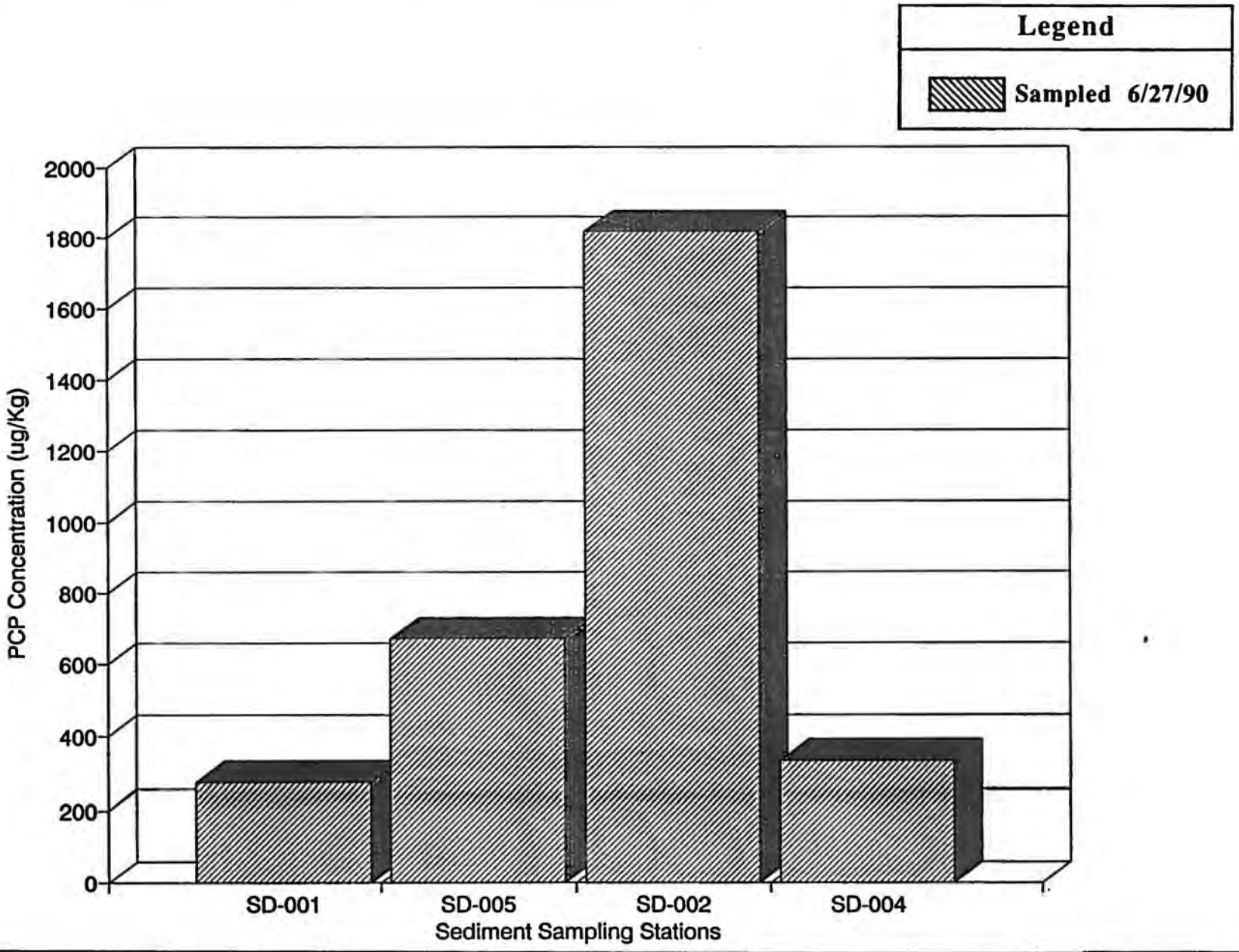
Because remedial activities are being contemplated or planned for source areas for inorganic contaminants, it is possible that the creek could become less hostile for aquatic life in the future. At such time, if contamination from the Montana Pole site was uncontrolled, potential risks associated with organic contaminants that are addressed in this assessment might be realized. Therefore, it is important to evaluate the potential importance of Montana Pole site-related organic contamination.

8.5.1.3 Area of Impact for Organic COCs

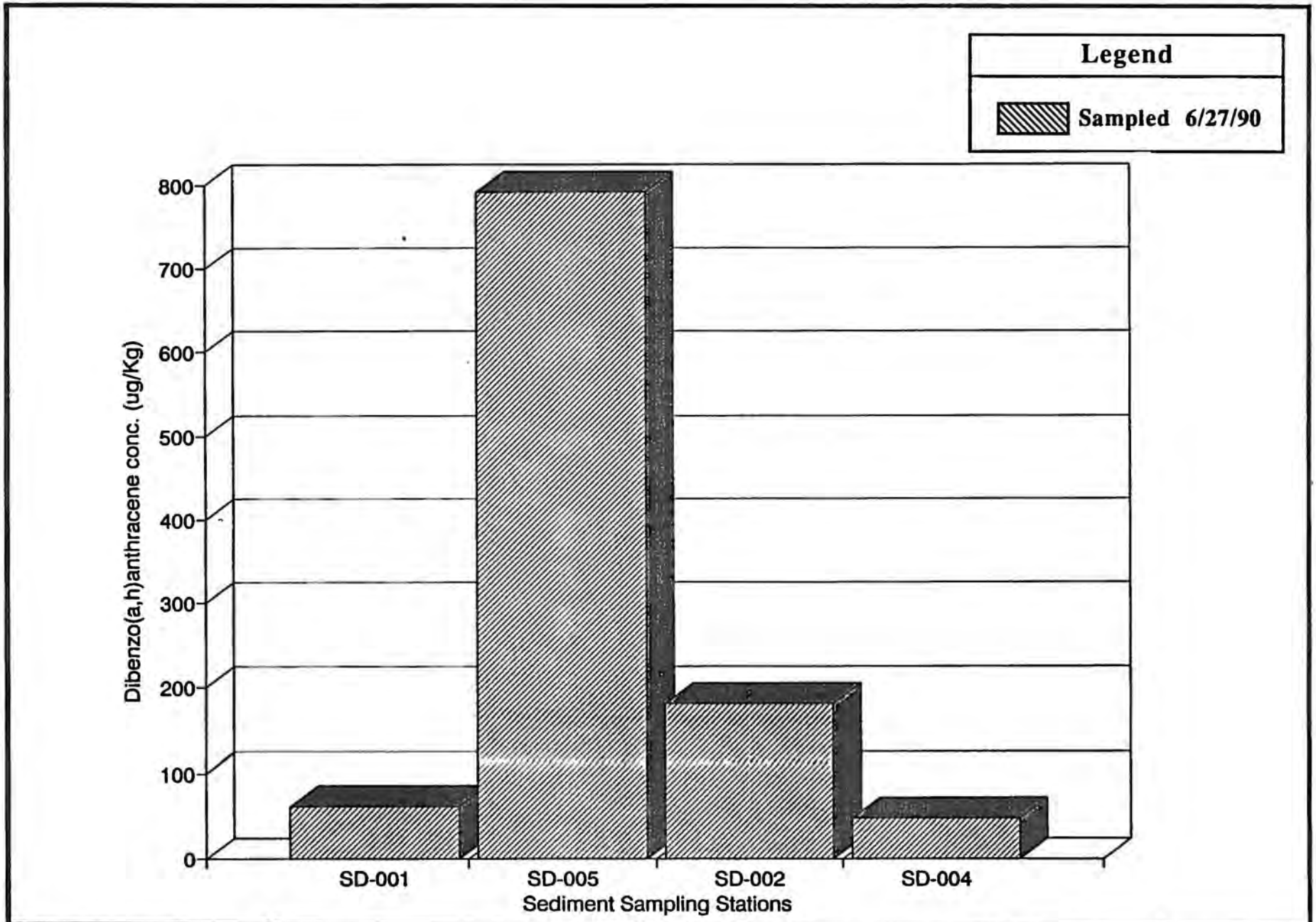
Figures 8-4 through 8-6 illustrate the distribution of PCP in surface water and PCP and dibenzo(a,h)anthracene in sediments at sampling stations above the Montana Pole site, at the site of the visible seep, and downstream of the Montana Pole site. Organic contaminants are either not



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Surface Water Concentrations for PCP in Silver Bow Creek**	Figure No. 8-4



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Sediment Concentrations for PCP in Silver Bow Creek**	Figure No. 8-5



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Sediment Concentrations for Dibenzo (a,h) - Anthracene in Silver Bow Creek**	Figure No. 8-6

detected upstream of the Montana Pole site or are detected at very low concentrations. Concentrations are clearly highest at the site of the seep adjacent to the Montana Pole site and diminish rapidly downstream.

For several other organic COCs, surface water contamination was detected only at sampling stations SW-005 and SW-004. In all cases, concentrations at SW-005 exceeded those at SW-004 by about an order of magnitude. For example, dibenzo(a,h)anthracene was detected at SW-005 at a concentration of 0.58E μg/L and at station SW-004 at a concentration of 0.03E μg/L. Similar values for benzo(a)pyrene, benzo(a)anthracene and benzo(b)flouranthene are 0.201E μg/L and 0.02E μg/L, 1.48E μg/L and 0.02E μg/L, and 3.04E μg/L and 0.02E μg/L respectively.

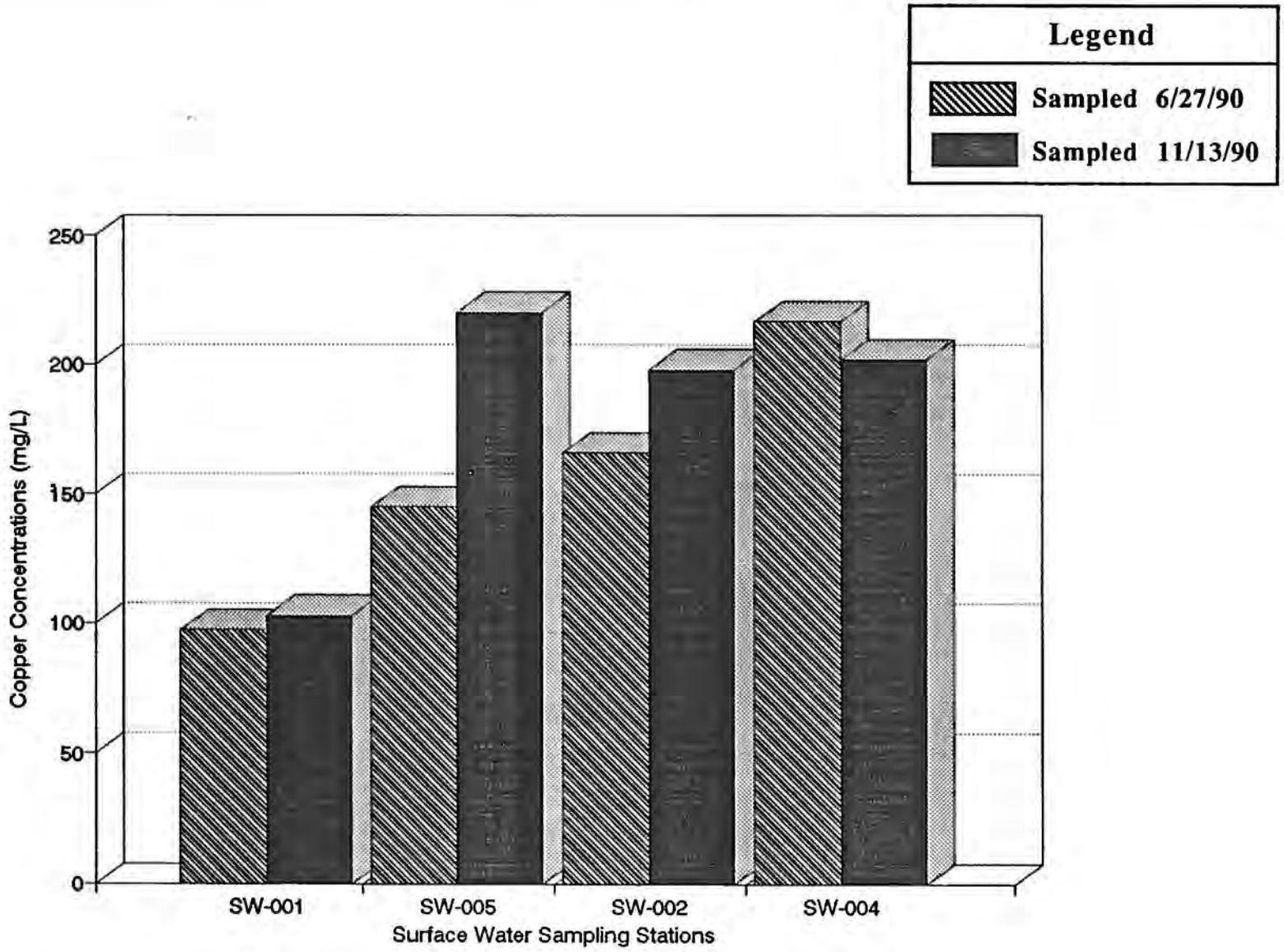
These data are consistent with current knowledge that discharge of contaminated groundwater to Silver Bow Creek occurs in the vicinity of sampling locations SW-005 and SD-005. PCP and PAH contamination in this reach of the stream can be attributed to the Montana Pole site.

Data from SW-005 and SD-005 only are available for dioxins and furans. No comparisons of upstream and downstream concentrations for these chemicals can be made. However, the existence of these compounds in groundwater and in sediments at SD-005 suggest that their distribution is likely to be similar to that of other lipophilic organic COCs.

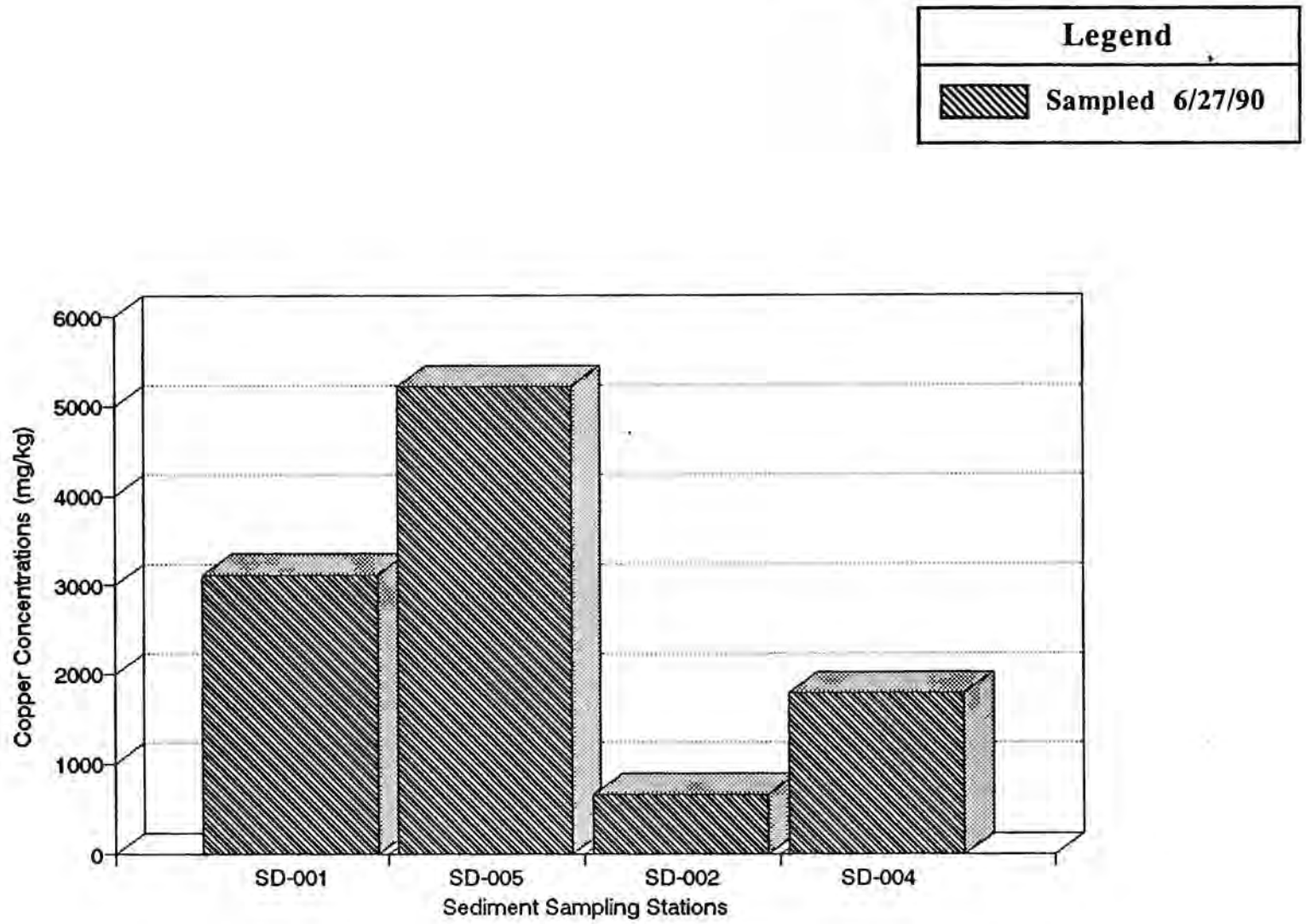
In contrast, Figures 8-7 through 8-9 depict metals concentrations in surface water and sediments along Silver Bow Creek adjacent to the site. For these chemicals, no clear pattern is evident in comparing upstream and downstream concentrations. this is expected since metal contamination comes from sources outside of and both up- and downstream of the Montana Pole site. These figures help support the conclusion that the Montana Pole site is not a source of metals contamination to Silver Bow Creek.

8.5.1.4 Summary of Risks to Aquatic Life

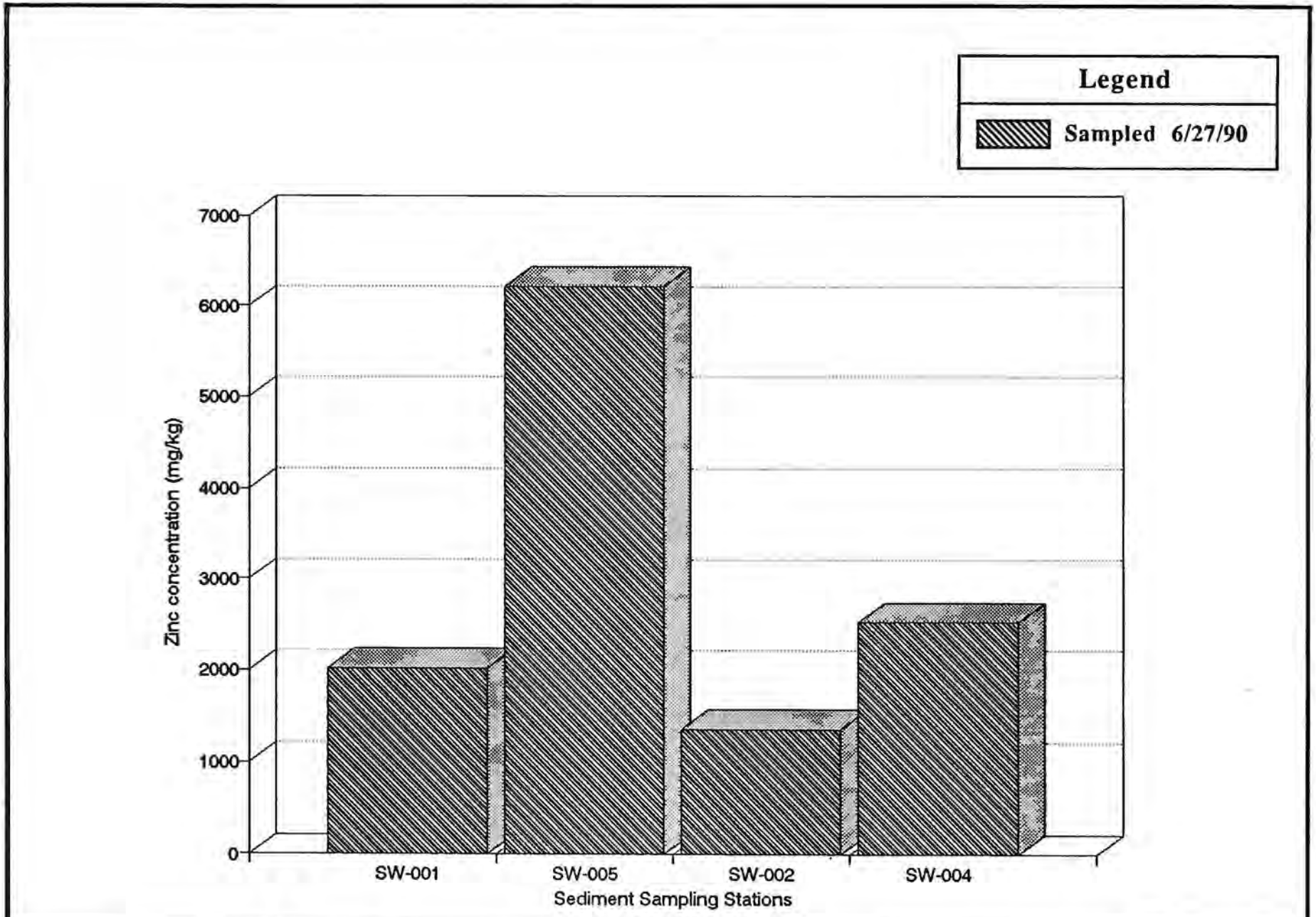
Figures 8-4 through 8-6 suggest that the current area of impact from the Montana Pole site is limited to the creek immediately adjacent to the site and for an indeterminant distance downstream. The furthest sampling station downstream is SW/SD-004, which is about 1600 feet downstream of



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Surface Water Concentrations for Copper in Silver Bow Creek**	Figure No. 8-7



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Sediment Concentrations for Copper in Silver Bow Creek**	Figure No. 8-8



CDM *Denver, Colorado*	**Montana Pole NPL Site**	**Sediment Concentrations for Zinc in Silver Bow Creek**	Figure No. 8-9

99-8

SW/SD-005. Since measurable concentrations of some organic COCs occur at this station, the area of impact currently may include sediments further downstream for the purpose of the Montana Pole RI/FS, the Silver Bow Creek investigation was limited to above the USGS station at SD/SW-004 Sediments and stormwater downstream of this location will be addressed during conduct of the Silver Bow Creek/Streamside RI/FS.

Pentachlorophenol (PCP)

Concentrations of PCP detected in surface water at SW-005, based on screening quality data, exceeded both the acute and chronic AWQC. The actual concentrations could be either over- or underestimated. Enforcement quality data from co-located sediments samples, however, are consistent with high concentrations in surface water at SW-005 (the maximum concentration of PCP in surface water, based on enforcement quality data was 16.5 μg/L). Sediment concentrations of 673 and 1,820 μg/kg were reported for SD-005 and SD-004, respectively. These concentrations are significantly elevated and are consistent with substantial amounts of PCP in the water column.

If metals contamination in Silver Bow Creek were brought under control, PCP in surface water could limit the recovery of aquatic life in the impacted stretch of the creek. Further, surface water samples collected in June and November both showed elevated PCP concentrations; therefore, PCP levels exceeding toxic levels may persist for extended periods.

PCP contamination could also limit recolonization of organisms in the reach of the creek adjacent to the Montana Pole site. Because the maximum measured level of PCP, based on screening quality data, far exceeds acute AWQC, exposures of even limited duration could result in acutely toxic effects for aquatic organisms. However, values based on screening quality data may or may not reflect actual PCP concentrations in Silver Bow Creek. The maximum concentration of PCP based on screening quality data was 591 μg/L, while a significantly lower maximum surface water concentration (16.5 μg/L) was measured using enforcement quality data. As presented in Table 8-3, 16.5 μg/L PCP is below most measurement endpoint concentrations for acute toxicity to site-specific species. However, sublethal (reproductive) effects associated with PCP exposures have been reported in *Ceriodaphnia* at 4.1 μg/L, and growth in rainbow trout is reported to be adversely affected to PCP exposure concentrations ranging from 7.4 to 19 μg/L.

Based on these data, ambient PCP concentrations in Silver Bow Creek could result in both direct and indirect adverse effects to trout. Direct effects include decreased growth in trout, which could increase predation and affect reproductive success — ultimately population density would be affected. Indirect effects include decreased populations of zooplankton that are critical components of aquatic food chains upon which trout are dependent.

It should be recalled that data from SW/SD-005 represent "worst case" conditions at the site of the major seep into Silver Bow Creek. Rapid dilution of PCP is expected in the Creek below SW-005, and some aquatic species might avoid this section of Silver Bow Creek.

To promote recovery of Silver Bow Creek along its upper reaches, control of metal contamination seems paramount. However, a short stretch of the creek near the Montana Pole site may not recover fully even if metals are remediated, unless discharge of PCP from contaminated groundwater is controlled. It would be necessary to reduce PCP levels in this reach to protect sensitive freshwater organisms such as trout and cladocerans.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAH concentrations, including lower molecular weight compounds such as anthracene, pyrene and naphthalene, are present only in low concentrations even at the area of the seep (SW/SD-005). The highest concentration reported was 12.7 μg/L for acenaphthene at SW-005. Acute and chronic toxicity values for acenaphthene and many other PAHs are not available. In fact, aquatic toxicity data for individual or total PAHs is extremely limited. Table 8-3 presents acute toxicity data (LC_{50}s) for three species that have potential to reside in Silver Bow Creek. These values suggest that acute toxicity to resident species due to PAHs in the water column are unlikely. Chronic toxicity data for aquatic organisms and PAHs are unavailable, but such data can be estimated from acute data. Acute to chronic ratios (ACRs) are commonly used to relate acute toxicity to chronic toxicity. Recommended ACRs range from 20 (EPA 1985a) to 100 (EPA 1989a), depending on chemical and acute toxicity data. Chronic toxicity data are estimated by dividing acute toxicity data by appropriate ACRs.

Acute Toxicity Value/ACR = Estimated Chronic Toxicity Value

The lowest available LC_{50} for potentially resident organisms and individual PAHs (820 μg/L, fluorene, rainbow trout) corresponds to an estimated chronic value of 8.2 to 41 μg/L, based on ACRs of 100 and 20, respectively. The use of the most conservative ACR (100) results in estimated chronic toxicity values in the range of the maximum PAH concentration measured is Silver Bow Creek. Although extrapolations from acute to chronic toxicity data and from one PAH to another should be used with caution, sublethal effects to potentially resident organisms are possible based on ambient PAH concentrations in surface water and on a conservative approach.

Dioxin/Furans

Dioxins/furans were not detected in surface water samples. Concentrations of dioxins, based on 2,3,7,8-TCDD, associated with decreased survival or sublethal effects in rainbow trout range from 0.0001 to 0.107 μg/L (Table 8-3). Concentrations this low might not be detected using common analytical techniques, and the potential for aquatic toxicity exists. The results of sediment sampling, however, do not support this potential. Highly toxic TCDD was not detected in sediment, and the concentration of other detected dioxins in sediment are sufficiently low to preclude significant water-column concentrations.

Only OCDD was detected in sediments (1.4 μg/kg). This is equivalent to 1.4 ng/kg 2,3,7,8-TCDD using the most recent toxicity equivalents. This latter value is 1000X the soil criteria presented in Table 8-2. Because intimate contact with either surface soil or with sediment could result in significant exposure in the area near SD-005, some impacts due to OCDD are hypothetically possible. It is CDM's professional judgement that any such impacts would be minimal.

8.5.2 RISKS TO TERRESTRIAL LIFE

Because organic COC concentrations appear to diminish rapidly with distance downstream from the Montana Pole site (Figures 8-4 through 8-6), potential future impacts from Montana Pole site-related chemicals are likely to be limited to a short reach of stream starting at the region of discharge of contaminated groundwater. Wildlife and/or domestic animals using the downstream portions of the creek as a drinking water source are not expected to be exposed to significant concentrations of

organic COCs now or in the future, unless discharge of contaminated groundwater significantly increases in the future.

Significant exposure of major wildlife species to surface water, sediments, and soils in the impacted reach of the creek are also unlikely. The Montana Pole site is heavily disturbed by past human activity, and is surrounded by residential housing, industrial development and an Interstate freeway. The site is unlikely to be attractive to wildlife, and larger animals (predators, deer, elk) are not expected to use the site, or the adjacent reach of the creek.

The concentrations of the three COCs in surface water, surficial soil and sediment are below available toxicity reference values for terrestrial wildlife and vegetation. Risks to terrestrial organisms from these media are expected to be minimal. Exposure to small mammals that use the site is possible, though the small size of the site suggests that any impact on local populations of such animals would be minimal. Also, because the site is small, any mammalian or avian predators that might visit the site are unlikely to take a significant portion of their prey from this area.

8.5.3 EVALUATION OF UNCERTAINTIES

In any risk assessment, it is necessary to make assumptions. The uncertainties associated with those assumptions and their impact on estimated risks helps to place risk estimates in perspective. Low confidence and limited information create high uncertainty, which indicates any derived value is less accurate and more likely to change, given more information. Low uncertainty is characterized by high confidence in a value that is more accurate, and is less likely to change as more data become available.

8.5.3.1 Potential for Underestimation of Ecological Risks

Dermal and inhalation exposure pathways were not evaluated in this ERA. Contaminated soil adhering to the organisms's skin or particulates directly inhaled might result in increased exposure and risk. Although these routes could be significant for animals whole range is limited to the Montana Pole site, insufficient exposure information makes it impossible to evaluate these pathways.

In addition, the use of toxicity values such as RTV or NOAELs could underestimate risks if receptors near the site are significantly more sensitive than the species used to establish the RTV. Thus, it is possible that risks could be underestimated using this approach.

Finally, a number of chemicals detected on site could not be addressed quantitatively in the ERA because of lack of data. These include phenolics other than PCP, which are expected in wastes from wood-treating processes. It is possible that these phenolics could contribute to overall impacts of the Montana Pole site on Silver Bow Creek. However, since the concentrations of these other phenolics are small compared to PCP concentrations, and the existing toxicologic data (e.g. for TCP) does not suggest that these phenolics will be significantly more toxic than PCP, it is not expected that risks from these phenolics will be low to non-existent.

8.5.3.2 Potential for Overestimation of Ecological Risks

The use of toxicity values could also overestimate potential risks, if ecological receptors are less sensitive than the laboratory animal used in the development of the toxicity value. In this ERA, the most sensitive species from the literature was selected, which would tend to overestimate rather than underestimate risks. Since no COCs were selected based on exceedance of a soil toxicity value, however, overestimation of risks due to these toxicity values has not occurred.

Limited water quality data, and corresponding stream flow data could contribute to overall uncertainty in risk estimates. For example, concentrations of organics could be lower during higher flows, thereby, reducing the risk to ecological receptors.

Use of the surface and sediment data from SW/SD-005 could also lead to an overestimation of risks for the entire study area. As previously discussed, these samples were taken from the area of a major seep into Silver Bow Creek. Dilution of PCP (and other COCs) would reduce concentrations substantially. However, maximum PCP concentrations, based on screening quality data, exceed chronic AWQC by over two orders of magnitude. It is therefore reasonable to conclude that a small reach of the creek could receive unacceptable contamination from the groundwater seep in this area.

8.5.4 SUMMARY

Impacts from organic chemicals of concern for Silver Bow Creek are expected to be limited to the reach of the creek adjacent to the Montana Pole site and extending for a relatively short distance downstream. Current information suggests that terrestrial wildlife using more distant reaches, or plants grown along these reaches would not receive significant exposure.

Impacts from soil contamination on the Montana Pole site are probably limited to plants and small animals that grow or live on the site. Major predators and larger birds and mammals are not likely to find the site attractive, and the small size of the site would limit potential exposures to any such animals that might visit the site.

Aquatic communities in Silver Bow Creek are currently affected by high metal concentrations that are primarily associated with historical mining activities near the Montana Pole site. Should remediation to reduce the amount of metals entering the creek occur in the Silver Bow Creek watershed the aquatic communities near and immediately downstream of the Montana Pole site could be exposed to the high levels of PCPs in the surface water and sediment.

9 Section Nine

9.0 REFERENCES

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