Health Consultation

KANTISHNA GRAVEL SOURCE

DENALI NATIONAL PARK, ALASKA

DECEMBER 17, 2008

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

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Prepared By:

Alaska Department of Health and Social Services
Division of Public Health, Epidemiology Section
Environmental Public Health Program
Under a Cooperative Agreement with the
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Background and Statement of Health Issues

On April 11, 2008 the Alaska Department of Health and Social Services (ADHSS) was asked by the Alaska Department of Environmental Conservation (ADEC) to evaluate the Kantishna Gravel Source Risk Assessment (1), as prepared by the US Army Corps of Engineers (USACE) for the National Park Service (NPS). The current analysis uses information from the three sampling events of the Area of Concern that was completed by the NPS and USACE between 2005 and 2007. The NPS wanted to evaluate the potential for contaminants in the proposed borrow source (DU6) to cause adverse health effects and asked the ADHSS to evaluate the data. Furthermore, a NPS representative expressed concern that the USACE risk assessment did not address any potential residents in the Kantishna area. This health consultation addresses that concern and evaluates health risk from exposure to Decision Unit (DU) soils.

The issue of concern involves the proposed use of mine tailings from the Kantishna area as material for maintenance of the Denali National Park Road (DNPR). The proposed gravel source, labeled Decision Unit number 6 or DU6, are six mine tailings piles from gold placer mining in the Kantishna area. Kantishna began gold mining in 1906 and increased production in the mid to late 1930s with continued activity to 1985 at the Kantishna Hills placer deposits and in Moose Creek (2, 3). The Kantishna area also had occurrences, mines or prospects of the following metals/metalloids: antimony, arsenic, barium, beryllium, bismuth, copper, iron, lead, manganese, mercury, molybdenum, silver, thorium, tin, titanium, tungsten, uranium, vanadium, and zinc (3).

Discussion

The Area of Concern (AOC) is the last 20 miles of unpaved road within Denali National Park where the proposed gravel (DU6 with dot symbol on creek) is to be utilized (Fig. 1). In 2007, the AOC was divided into Decision Units (DUs) for sampling purposes and a site investigation was conducted by USACE. The AOC includes Wonder Lake Campground and a ranger station in DU3. In DU1 there is National Park Service housing, Kantishna lodges, and a community with an airstrip. A portion of the AOC parallels the McKinley River and has numerous creeks with Wonder Lake within close proximity.

Mercury may be present in gold and silver ores and is often a by-product of processing (4). In the Kantishna district there were over 400 patented and unpatented mining claims in the Denali National Park (2) and the Merinser Prospect which had mercury as a major ore (3). Unpatented claims may not have records; therefore, it is unknown how many of these claims also encountered mercury ores. Additionally, throughout the western United States, miners used mercury (quicksilver) to recover gold in placer (alluvial) and hardrock (lode) mines. The recovery process often lost 30% of the original amount of mercury and frequently contaminated surrounding soils (5). Mills processed ores from mines and may have used mercury in processing operations. Several mills were constructed in the Kantishna area (2) including Red Top Mine's Banjo Mill on a tributary of Eureka Creek and Little Annie Mill near the Kantishna air strip (Fig. 1, DU1 area).

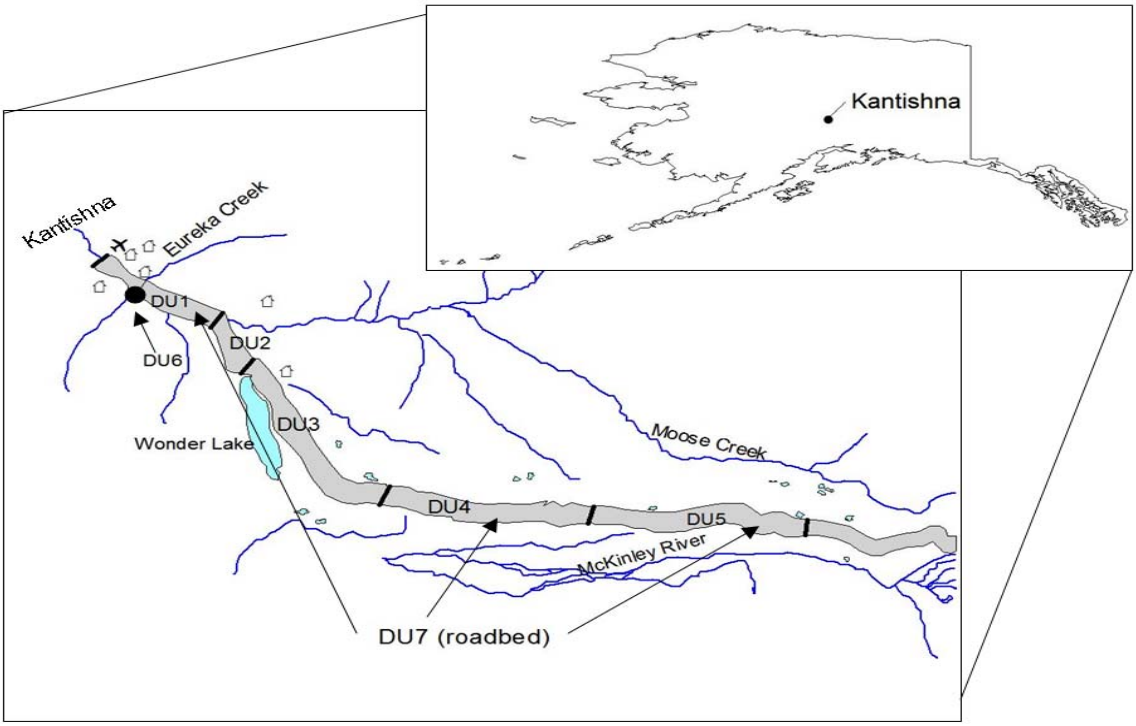

Fig. 1. Area of detail map for Kantishna, Alaska.
Denali National Park Road (DNPR) labeled with Decision Units (DUs).

Identifying Contaminants of Concern

Data Prior to 2007

In 2005, the NPS collected 19 soil samples from the borrow source (tailings pile) area and analyzed these for levels of antimony, arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver (Table 1). Other elements were not tested for in these Kantishna soil samples. Selenium (< 2.1 mg/kg) and silver (< 0.13 mg/kg) were not detected in any of the samples. However, this sampling indicated high concentrations of antimony, arsenic and chromium relative to EPA guidelines (6). It should be noted that this borrow source sampling may not be directly comparable to the currently proposed borrow source (DU6) sampling that occurred in 2007.

In 2006, the NPS collected six soil samples in areas that they predicted would have background concentrations of metals (Table 1). The background soil samples were analyzed for the same nine compounds that were tested for in 2005. Based on the background results the NPS and USACE decided to remove mercury, barium, cadmium, lead, selenium, and silver from future analysis. The USACE identified chromium, antimony and arsenic as potential chemicals of concern because levels were above ADEC cleanup levels (6). The USACE identified appropriate chemicals of concern (arsenic, antimony, and chromium) based on EPA cleanup levels (6).

The Alaska Department of Environmental Conservation (ADEC) set a concentration of 1.4 mg/kg of mercury as the soil cleanup level for mercury migration from soil into groundwater (7).

Background mercury concentrations from the 2006 soil samples ranged from 0.067 mg/kg to non-detectable (< 0.04 mg/kg). Surface soils, from various countries, range between 0.02 and 0.625 mg/kg concentration of mercury (8). The background Kantishna mercury concentrations (from the 2006 sampling) were near the lower end of the range of soil mercury reported worldwide and lower than the ADEC mercury soil cleanup level. There would be no additional adverse health effects from exposure to Kantishna soils than what would happen from natural background concentrations, provided that the background area chosen was representative of the Kantishna area.

Data from 2007

In 2007, the NPS and USACE utilized multi-increment sampling (9) of the Area of Concern except for in DU6. In a very general way, multi-increment sampling obtains single samples (≥ 50) of within an area and then mixes them together and analyzes one sample which is to represent the entire area of the gathered samples. The NPS and USACE deviated from the ADEC multi-increment sampling protocol in the following manner:

1. Multi-increment sampling is not suggested for metals. ADEC guidelines indicate that multi-increment sampling is to be utilized for petroleum hydrocarbon contamination only (9).
2. ADEC was not notified prior to initiating the planning process, detailed in the multi-increment guidance (9).
3. The borrow source (DU6) was mixed and then sampled which is different then stratifying and vertically sampling, as the guidelines suggest (9).
4. No triplicate multi-increment sample was collected in DU3, DU4, or DU5 and this is needed in order to characterize these similar areas at a 10% rate (9).

The maximum arsenic levels ranged from 19 to 300 mg/kg in Kantishna Decision Unit (DU) soils (Table 5). These arsenic concentrations are higher than the US national average (7.2 mg/kg) but within the range (> 27,000 mg/kg) for soils in mining areas (10).

Antimony ranged from 1.4 to 34 mg/kg in Decision Unit soils (Table 5). Antimony naturally occurs at low levels and U.S. soils have antimony concentrations of 1 to 8.8 mg/kg. Contaminated sites such as a National Priorities Listed (Superfund) site may exceed 25,000 mg/kg (11) in antimony concentrations. Contaminated mining areas average 111 mg/kg of antimony within 2 km of these sites (11). The samples in the Kantishna Decision Units were at least ten times lower than either of the contaminated site concentrations. Antimony levels were well below the Reference Dose Media Evaluation Guide (RMEG) Comparison Value of 300 mg/kg for adults in all Decision Units (Table 5).

Two forms of chromium are known to exist. One form is trivalent (Cr^{3+}) while the other is hexavalent (Cr^{6+}) chromium. Trivalent chromium is considered relatively non-toxic. On the other hand, hexavalent chromium is known to cause lung fibrosis and cancer in humans (12). Hexavalent chromium is considered a human carcinogen by EPA, the National Toxicology Program and the International Agency for Research on Cancer but only for inhalation of the substance. The data is reported for total chromium (Table 5) which measures both the trivalent and hexavalent forms. The maximum total chromium levels ranged from 9.9 to 20 mg/kg in Decision Unit soils. Background chromium concentrations in the U.S. range from 1 - 2,000

mg/kg, with an average of 37 mg/kg (14). The Kantishna soil samples are lower than the US national average. The chromium in the borrow source appears to have decreased by half from the 2005 (Table 1) and 2007 (Table 5) soil sampling events (within 2 years). This suggests that the observed concentration differences may have been because of the extended hold time in the 2007 samples or environmental factors (i.e. organics in soils) may have influenced these results.

The total chromium (Cr^{3+}, Cr^{6+}) concentrations in Kantishna soils are known from the USACE sampling and laboratory analysis occurring in 2007. However, specific levels of each chromium analyte (i.e. Cr^{3+} and Cr^{6+}) are unknown in the Kantishna samples because the hexavalent chromium samples were held too long (hold time exceeded) to be analyzed. It is important to analyze samples for chromium within the time frame specified by a method to avoid reduction-oxidation reactions that may significantly change the proportion of chromium species ($Cr^{6+,}$ Cr^{3+}) within a given sample. Hexavalent chromium can oxidize to trivalent chromium from the amount of acidity (pH), bacteria in soils and/or the presence of other elements (e.g. manganese) or organics in soils (15).

There is some also concern over the methods used to analyze the chromium samples. Literature indicates that hexavalent chromium in soil samples should be analyzed by the following methods: solid waste (SW) 7195 followed by SW7190/SW7191 or SW3060 followed by SW6010 (15). Use of another hexavalent chromium methodology such as that (SW7195/SW6010B) employed in this study would need to be validated by the laboratory through quality assurance and quality control procedures. Laboratory validation for chromium analyses could not be determined by the data collected.

The NPS did not analyze for mercury in soil samples from 2007 and this included the borrow source (DU6) and other Decision Units. Unfortunately, borrow source (DU6) mercury concentrations (collected in 2005) are invalid because sample hold times were exceeded by a 24 day time period. Decisions on whether to keep an element for assessment should not have been made based on invalid samples or low background concentrations, especially given the likelihood that mercury may have been used in ore processing or may have been present in Kantishna gold and silver ores. The proposed borrow source (DU6) is a tailings pile (leftover, unwanted materials) from mining and may indeed have mercury content; without valid analysis it is unknown whether DU6 poses a health hazard from mercury.

A contaminant of concern is a substance that is present at levels that might cause harmful (adverse) health effects. A contaminant of concern is identified by screening of the site-specific concentrations (levels) to environmental guideline (EMEGs, RMEGs, CREGs; see glossary, in back, for details) Comparison Values (CVs). CVs are chemical concentrations that are below levels that are known or anticipated to result in adverse health effects to humans. CVs are conservative because they incorporate safety factors as a precaution for each area of scientific uncertainty, resulting in more robust protection of human health. Contaminants found at levels below CVs are not considered a public health risk, and no adverse health effects are expected from exposure.

When the concentration of a chemical exceeds the environmental Comparison Value (CV) or if no CV exists, the substance is further evaluated by calculating doses and comparing to health

based guidelines. Health guidelines include, but are not limited to, Minimal Risk Levels (MRLs) or the Environmental Protection Agency (EPA) reference dose (RfD). If a MRL or RfD does not exist then the No Observable Adverse Effect Level (NOAEL) or Lowest Observable Adverse Effect Level (LOAEL) is used for comparison to the site-specific dose.

The ADHSS and the Agency for Toxic Substances and Disease Registry (ATSDR) screening process utilizes the maximum contaminant concentration when making comparisons, to represent a worst case scenario for exposure. Large volumes of data are rapidly screened using this method, but the method does not necessarily indicate potential adverse health effects.

Once the contaminants of concern are identified, this health consultation evaluates the USACE risk assessment and interprets the public health significance of the Kantishna soil samples (the proposed borrow source and other DUs), taken in 2007. The health consultation identifies exposure pathways (see back of document for methods) and compares calculated doses with available toxicological information to ascertain if exposure may cause an adverse health effect.

Based on the soil results, ADHSS identified the contaminants of concern as arsenic, antimony, chromium, and mercury in the Kantishna area soils (Table 1) for the following reasons: Antimony and arsenic concentrations were above the child Comparison Value (20 mg/kg) in both background and borrow source soil samples, though not above the adult Comparison Value (200 mg/kg). Mercury is included as a contaminant of concern because it has no established ATSDR environmental or health Comparison Values and not enough sampling data were collected for public health decision-making. The borrow source (DU6) are piles of gold mine tailings, and mercury would be an expected contaminant in these piles and the surrounding area because of historical use in gold mining. Chromium is included as a contaminant of concern due to analytical concerns.

Completed Exposure Pathways

There are no known year-round residents in Kantishna, though lodge caretakers may be present during the winter off-season. At any one time during the summer, there are approximately 135 residents with the potential of 265 more people when the area lodges are full (1). Thus people may be exposed to metals and other compounds in the Kantishna area. The potentially exposed population with completed pathways of exposure would consist of drivers and visitors going to Kantishna, road and brush maintenance crews, Denali backcountry employees, rangers and seasonal residents (≤ 26 weeks/year) and their children. In the past few summers, seasonal residents, of Kantishna included at least two children under the age of 5. It is unknown whether these children exhibit pica behavior, which occurs in 16-60% of children under the age of 6 (17). These people are potentially exposed by incidental soil ingestion and through dermal (skin) contact with surface (≤ 3”) soils elevated with arsenic and antimony (Table 2).

Past populations of up to 2,000 people consisted of miners and their families (2). The Decision Unit 1 (DU1) area (Fig. 1) was heavily mined and families lived in Kantishna and the surrounding area; these people were likely exposed to elevated metal concentrations in the Kantishna area. A report in 1990 recommended that the NPS buy all mining claims within the Denali National Park (DNP) so land use may potentially decrease as claims are bought in the

area (2). However recent demand for a greaterr number of vehicles allowed in the DNP may increase the number of people exposed to Kantishna soils in the future.

Other Potential Pathways

The exposure to mercury through incidental soil ingestion is a potential exposure pathway (Table 3) because of the lack of sampling data.

Another potential pathway involves traffic along the Area of Concern which extends for 20 miles along an unpaved portion of the Denali National Park Road (DNPR). Vehicular traffic on the un-paved portion of the DNPR causes fine solid particles (dust) to become suspended in the air (Fig. 2). Due to precipitation and vehicular traffic patterns the majority of dust is generated from late spring through early fall. Contaminated dust, once suspended in air, may be inhaled and yet another potential pathway for human exposure to chemicals. Traffic on the DNPR is currently limited to 10,512 trips annually (1) though community meetings occurring in 2008 are discussing a change in traffic volume. Nonetheless many of these trips do not include the last twenty miles of road. Since the Denali National Park restricts road traffic on the DNPR and few vehicles drive the entire road, there are a limited number of people coming in contact with Decision Unit dust.

Fig. 2. Dust from vehicle traffic on the unpaved portion of the Denali National Park Road

Elevated levels of arsenic are in portions of Kantishna roadbed soils thus dusts may also have elevated concentrations of arsenic. Therefore the risks associated with the inhalation of dusts in the Kantishna area should be explored. However, the method used by the USACE for calculating inhalation exposures has limited applicability in public health risk assessment because it may over- or under-estimate inhalation concentrations based on the amount observed in soil. The USACE utilized standard and acceptable methods for calculating inhalation (dust exposures) as defined by the EPA (16) though the applicability of an internal Federal Highways Administration (18) email for obtaining concentration conversion factors or particulate emission factors for air is questionable. Because no air samples were taken, the dust pathway was not adequately quantified in the USACE study; contaminant exposures may also be occurring

through this route (Table 3). The ADHSS uses the ATSDR guidelines when judging if contaminant exposures exist. ATSDR requires air sampling or modeled data to obtain the concentration of contaminants in the air. No air sampling was undertaken for the Kantishna risk assessment; therefore, the inhalation pathway would be considered an indeterminate public health hazard.

More sampling and data collection would be necessary to assess the total risk of health effects from contaminants in the Kantishna area. It is likely that the most important pathway for worker exposure will be through inhalation of dusts from vehicular traffic and/or gravel production with mine tailings. The dust inhalation route was not well researched in the sampling plan and subsequent risk assessment. At minimum, a study utilizing air sample monitoring stations or personnel monitoring along the Area of Concern should have been undertaken. The monitors would have potentially addressed the amount of dust inhaled and amount of current road-bed contaminants in the dust. Critical information is lacking on the concentration of mercury, arsenic, antimony, and chromium in Kantishna Decision Unit dusts. This information is important to assess whether the inhalation of dusts may cause a public health hazard, especially to workers exposed to these dusts for long durations.

Health Considerations

Inhaling, ingesting or absorbing arsenic, antimony, and/or chromium from soil, dust, water or subsistence foods are potential pathways of exposure for these compounds. Each of these metals may cause skin and eye irritations as well as more severe health effects. The potential pathways of exposure for mercury are through inhalation or ingestion of the substance. Exposure to high levels of mercury may result in neurological effects or organ damage (8).

Risk of adverse health effects from a contaminant is greater with increased time of exposure, frequency of exposure, and increasing contaminant concentration. The USACE risk assessment was not quite accurate for the Exposure Duration for the Wonder Lake Grader Operator (WLGO) grading job and given a "†" symbol in Table 4. The WLGO-grading description states that four days in every three week period the person is exposed (4 days/3 weeks =1.33 days/week) not 0.75 days/week. This minor detail does not substantially change the results in analyzing if the WLGO grading exposure exceeds Minimal Risk Levels or acceptable measures of Excess Lifetime Cancer Risks (ELCRs). Additionally, the risk assessment for the Wonder Lake Laborer had a typographical error in that 40 days/year were to be listed for the frequency and not 25. Again, the magnitude of this error does not significantly change the risk exposure results. Furthermore, combining the WLGO-roading and grading jobs together seems practical because the risk assessment mentions that they were performed by the same individual.

Potential adverse health effects are not addressed for sensitive individuals when a Lowest Observable Adverse Effect Level or No Observable Adverse Effect Level is used to assess whether there is risk from a contaminant. In these instances and specifically for arsenic and antimony exposures, highly sensitive individuals such as those with a chronic respiratory or cardiovascular disease, kidney dysfunction, immuno-deficiency or chemical sensitivity are not assessed. This health consultation does not assess any past exposures because data for that has not been obtained.

Toxicological Evaluation

Arsenic Ingestion Pathway

Decision Unit 1 (DU1) was observed to have a maximum concentration of 300 mg/kg arsenic (Table 5). Arsenic concentrations above 200 mg/kg in soil are higher than the chronic Environmental Media Evaluation Guide Comparison Value for adults. Arsenic levels were also above the chronic Environmental Media Evaluation Guide Comparison Value for children (20 mg/kg) in all Decision Units (DUs) except DU4 (Table 2). Thus, the ingestion and dermal contact pathways for arsenic, in soil, required further evaluation for adults and children. The proposed borrow source, DU6 (70 mg/kg arsenic), is below the chronic Environmental Media Evaluation Guide Comparison Value for adults (< 200 mg/kg). However, the DU6 arsenic concentration was chosen for analysis because this is the proposed borrow source material for the Area of Concern.

Through incidental soil ingestion, workers may potentially absorb non-carcinogenic doses of arsenic ranging from 0.00003 to 0.0002 mg/kg-day, based on the maximum observed concentration of 300 mg/kg (Table 6). Only the Resident Lodge Owner has an arsenic dose (0.0002 mg/kg-day) in the same range as the chronic Minimal Risk Level (0.0003 mg/kg-day) for non-carcinogenic effects (Table 6; footnote section).

The non-carcinogenic oral doses for children exposed to arsenic in Kantishna soils were between 0.0004 mg/kg/day and 0.04 mg/kg/day (Table 6) and these are within or above the acute and chronic Minimal Risk Level (0.0003 mg/kg/day) for arsenic. Pica children have the highest arsenic doses (0.01 mg/kg/day and 0.04 mg/kg/day) depending on soil concentrations.

For workers, carcinogenic arsenic doses (Table 7) calculated from incidental soil ingestion were 0.0009 – 0.2 µg/kg-day using the maximum (70 years) exposure duration. Of the workers (adults), the Wonder Lake Grader Operator who undertakes grading had the lowest dose and the Resident Lodge Owner was observed to have the highest carcinogenic arsenic dose.

Carcinogenic doses from arsenic exposure for children are listed in Table 7. Doses for non-pica children were 0.00003 - 0.00015 mg/kg-day. Children with pica behavior had potential carcinogenic arsenic doses of 0.00086 – 0.00370 mg/kg-day.

The lowest observable arsenic Cancer Effect Level (CEL) for people is 1.1 µg/kg-day or 0.0011 mg/kg-day (10). Most of the adult arsenic carcinogenic doses were a minimum of 100 times lower than the CEL in humans. The Resident Lodge Owner and non-pica children had carcinogenic arsenic doses within a factor of 10 of the CEL.

The theoretical Excess Lifetime Cancer Risk (ELCR) is calculated for worker and child exposures (Table 8) and it should be noted that these estimates are for excess cancers that *might* result in addition to those normally expected in an unexposed population. Based on the ELCR analysis a child exhibiting pica behavior has the highest theoretical risk for excess cancer (6 in 1,000) from the highest arsenic exposure (300 mg/kg in DU1). Pica children were also observed to have a 1 in 1,000 theoretical Excess Lifetime Cancer Risk when exposed to the arsenic levels (70 mg/kg) in DU6. Non-pica children and all other workers were observed to have theoretical

Excess Lifetime Cancer Risks at or below 2 in 10,000 people (Table 8) which would be considered an acceptable level by the EPA.

The proposed borrow source, DU6, has maximum arsenic concentrations of 70 mg/kg and an average arsenic concentration of 66 mg/kg (over 3 times the Comparison Value used for children) but does not produce an unacceptable Excess Lifetime Cancer Risk (5 in 100,000) for normal (non-pica) children.

Individuals (adults and children) may absorb 0.0003 to 0.04 mg/kg-day of arsenic through soil ingestion when considering the site-specific exposure scenarios. Exposures to Kantishna soils, and the arsenic within, occur on an intermediate time scale (15-364 days) but no oral intermediate Minimal Risk Level exists for arsenic in soil. Furthermore, oral intermediate studies have not observed a No Observable Adverse Effect Level (NOAEL) for arsenic in humans (10). Chronic human arsenic studies indicate NOAELs between 0.0004 and 0.0008 mg/kg-day. Based on these NOAELs, non-pica children exposed to DU6 (proposed borrow source) arsenic would not be considered at risk for adverse non-carcinogenic health effects. However, pica children exposed to DU6 and all children exposed to DU1 arsenic concentrations may experience adverse non-carcinogenic health effects. Parents should be wary and limit the amount of time children spend in area soils because inorganic arsenic may influence a child's intellect and also increase the chance of developing cancer. Parents should not encourage hand-to mouth behaviors in children, especially in areas with current or prior mining activities. Additionally, parents should encourage hand washing before meals.

The calculation of arsenic doses may over-estimate the true exposure because other factors should be considered when assessing this data. For instance, the calculations rely on the assumption of chronic exposure to arsenic. Chronic exposures occur on a regular basis throughout a year. People do not live in the Kantishna area during the off-(winter) season (26 - 39 weeks) so they would not be exposed to the Kantishna arsenic during this time period. Due to the seasonal nature of residency in Kantishna, arsenic soil exposures would not be daily over the course of a lifetime therefore adverse health effects would not be expected. Another factor to consider is that arsenic is excreted from the body on a regular basis and stored arsenic would naturally leave the body as exposure decreases. Furthermore it is unlikely that individuals would fully absorb the arsenic in soil as gastrointestinal absorption is noted to be approximately 3-50% of the dose (10). For children, specifically, the dose may be over-estimated because calculations used 6 years as the Exposure Duration (ED) and 168 days/year as the Frequency (F). It may be likely that children are not playing in Kantishna soils for 168 days per year for 6 years.

Exposure to arsenic from Kantishna Decision Unit soils is unlikely to cause non-carcinogenic or carcinogenic health effects in adults. However, pica children and children exposed at the highest arsenic concentration (300 mg/kg in DU1) may have increased cancer risk. Currently DU6 is composed of at least four if not seven discrete tailings piles that are within the DU1 area; a health hazard exists if pica children are allowed to play on these tailings piles. Since it is unlikely that parents would let their children play on a roadbed, no hazard exists for children exposed to DU6 gravel that is placed on the roadbed.

Arsenic Dermal Pathway

The absorption of a contaminant through the skin is a dermal contact pathway. Dermal doses for Resident Lodge Owner (RLO) and children are listed in Table 9; these individuals were chosen because they have the highest risk of potential adverse health effects based on the duration of exposure and previous calculations. Soil concentrations that exceeded an adult or child environmental Comparison Value were selected for dose calculations. The Resident Lodge Owner was observed to have a dermal dose of 0.034 μg/kg-day with children having a higher dose of 0.217 μg/kg-day when exposed to Decision Unit 1 (DU1) soil concentrations at 300 mg/kg (Table 9). A child's dermal dose from the exposure to DU6 soil concentrations of 70 mg arsenic/kg is expected to be 0.051 μg/kg-day.

No studies were found that determined non-carcinogenic health effects from dermal exposure to arsenic in humans (10). A single intermediate duration study occurred in mice dermally exposed to 6,000 μg arsenic/kg-day and this produced less serious skin and cell changes (10). A No Observable Adverse Health Effect Level can be calculated for humans from the mouse study. Division of the dose observed in mice by 1,000 will account for the uncertainty between species, sensitive individuals, and the effect level. The estimated human No Observable Adverse Effect Level would be 6 μg/kg-day and this dose is a minimum of 10 times higher than the doses (0.004 - 0.217 μg/kg-day) that are observed for children dermally exposed to soils in DU1. For adults, the calculated No Observable Adverse Effect Level is 100 to 1000 times higher than the worker doses (0.003-0.034 μg/kg-day). No adverse non-carcinogenic health effects are expected from dermal absorption of arsenic for either adults or children.

No studies were found that have associated cancer in humans with dermal exposure to arsenic (10). Comparisons of the site-specific carcinogenic doses are not possible because only three studies have been performed on animals and none observed a Cancer Effect Level from dermal exposure to arsenic. Nonetheless, the propensity of dermal exposure to cause excess cancers was calculated. The arsenic Excess Lifetime Cancer Rate (ELCR) was calculated for the worker and children with the highest potential risk, based on exposure duration and with the highest observed concentrations in Decision Unit 1 and 6 (Table 9). The ELCRs indicated that an additional 5 to 51 individuals per million people *may* theoretically develop cancer from dermal arsenic exposure in the Kantishna Decision Units as opposed to the rate observed in an unexposed population. The EPA believes ELCRs between 10,000 and 1,000,000 to be acceptable measures of excess cancer risk. Resident Lodge Owners and children had the highest (but still well below 1 in 10,000) ELCRs; however, it should be remembered that this analysis is an over-estimation of the actual risk. It would be expected that the risk from dermal arsenic exposure is much lower based on people's habits, the amount of arsenic absorbed, and the amount of time of the exposure.

Arsenic Combined Pathways

Exposures to contaminants through multiple pathways may be combined if the contaminant targets the same organ. Ingestion of arsenic effects all organ systems but with small to no effects to the kidneys (10). Arsenic exposure may be associated with lung and skin cancers and increasing evidence suggests bladder cancer may also result (10). Arsenic is considered a human carcinogen by EPA, the National Toxicology Program and the International Agency for Research on Cancer. The skin is affected by ingestion and dermal absorption of arsenic. Absorption of

arsenic through the skin (a dermal exposure) may result in skin color changes and skin ulcerations whereas the ingestion of arsenic may cause skin color changes and skin lesions (warts, corns). It is justified to sum the ingested and dermal non-carcinogenic doses of arsenic because both of these pathways may affect the same organ (skin). Individuals have the potential of receiving 0.000106 - 0.04 mg/kg-day arsenic from Decision Unit 1 exposure (Table 10).

The total arsenic dose is compared with the chronic (no intermediate duration) arsenic oral Minimal Risk Level (0.0003 mg/kg-day) to observe if health effects are possible from the combined pathways. Resident Lodge Owners (RLO) and children (with and without pica behavior) were observed to have total arsenic doses at or above the chronic oral Minimal Risk Level.

The theoretical Excess Lifetime Cancer Risk (ELCR) values from oral (ingestion) and dermal exposure pathways were combined (Table 10) because studies are lacking on whether cancer can occur from dermal exposure. ELCRs ranged from 1 in 100,000 (Kantishna Air Transport Driver) to 6 in 1,000 (pica child) individuals that *may* theoretically develop cancer from Kantishna DU1 exposure. The largest contribution to cancer risk was from oral exposure.

Antimony Ingestion Pathway

The Kantishna soil concentrations are below the adult Comparison Value (300 mg/kg) so adverse health effects are unlikely from antimony exposure in these soils. However, to allay any possible concerns from the incidental ingestion of antimony the amount of exposure for the Resident Lodge Owner was calculated because this individual was not previously assessed (Table 6). The Kantishna Air Transport Driver's exposure was calculated because this individual has the longest duration (years) and amount of time (days) exposed to Decision Unit soils. Worker antimony doses were between 0.006 and 0.02 μg/kg-day (Table 6). As listed in Table 6, the non-carcinogenic antimony doses for children are between 0.0001 and 0.005 mg/kg-day.

No Minimal Risk Level exists for antimony in soils but there is a single acute Lowest Observable Adverse Effect Level (LOAEL) reported for antimony exposures in humans. The LOAEL of 0.529 mg/kg-day (= 529 μg/kg-day) caused individuals to vomit from a single dose of potassium-antimony-tartrate in water (11). The potential doses of antimony from Kantishna soils are 100 to 1,000 times lower than the acute LOAEL for children and 10,000 to 100,000 times lower than the acute LOAEL for workers.

In an intermediate duration (15 - 364 days) study, rats were observed to have less serious cardiovascular effects (LOAEL) and decreased maternal weight gain from an oral dose of 74.8 μg/kg-day (11). Using the rat LOAEL and dividing by 1,000 to account for uncertainty, the No Observable Adverse Effect Level for humans would be 0.0748 μg/kg-day. Worker doses are a minimum of 10,000 times lower than the calculated no observable effect level and children's doses are also below the no observable effect level.

Antimony is not considered a carcinogen so there are no calculations for carcinogenic doses or Excess Lifetime Cancer Risk for this contaminant. From the calculated doses and literature review an adverse health effect is unlikely from the ingestion of antimony in the Kantishna area for both adults and children.

The absorption of antimony is over-estimated in this health consultation. Intestinal absorption of antimony appears to be less than 10% (11) indicating an absorption factor (AF) equal to 0.1. To be conservative, this health consultation utilizes an AF of 1 which over-estimates the doses by a factor of 10.

Antimony Dermal Pathway

The concentration of antimony in Decision Unit soils was not greater than the adult environmental Comparison Value (300 mg/kg). However dermal doses for workers were calculated because of later combined risk and Hazard Index assessments. Both the Resident Lodge Owner and Kantishna Air Transport Driver, whom have the longest exposures, were observed to have an antimony dose of 0.0001 μg/kg-day (Table 9).

As observed from Table 5, the maximum concentrations in DU2 (34 mg/kg) and DU6 (21 mg/kg) are greater than the Reference Dose Media Evaluation Guide for children (20 mg/kg). The highest observed concentration of antimony (34 mg/kg in DU2) resulted in a very small dermal dose (0.0008 μg/kg-day; Table 9) for children. No Minimal Risk Level and no adequate health guidelines exist for antimony. The antimony toxicological profile reports a dermal No Observable Adverse Effect Level (NOAEL) in rabbits for a 5% solution of inorganic antimony; this solution is equivalent to 50,000 ppm or 50,000 mg/L. The highest observed concentration in soil is 34 mg/kg and this is 1,000 times lower than the concentration used for the NOAEL experiment. Children and adults are unlikely to have adverse health effects from dermal exposure to antimony in any of the Decision Units.

Antimony Combined Pathways

Exposure to high levels of antimony may cause lung and heart problems, stomach pain, diarrhea, vomiting and stomach ulcers (11). The oral and dermal routes of antimony exposure affect different organ systems so adding the two pathways together for a cumulative total dose should not be done (Table 10). Ingestion of antimony predominately impacts the gastrointestinal organs (causing vomiting) in humans. Rats are noted to have blood and liver changes from ingestion of antimony. However, the dermal route of antimony exposure is known to irritate skin and eyes and these are different organ systems than the gastrointestinal system.

Arsenic and Antimony Combined Health Risk

It is not known if there is an arsenic and antimony interaction that may cause greater health effects (10, 11). Lead, cadmium, zinc and selenium interact with arsenic to change toxic effects. Nutritional deficiencies (lower levels of choline, methionine, or protein) and ethanol also interact with arsenic and change the degree of adverse health effects (10) expected relative to effects from arsenic exposure alone. A worst case scenario of an interactive effect between arsenic and antimony is assumed to protect health. The Hazard Index (HI) is calculated to determine if arsenic and antimony, in combination, has the potential to lead to an additive or synergistic health effect.

The Hazard Index (HI) is dependent on the chemical specific Minimal Risk Level (MRL). The HI for arsenic exposure in Decision Unit soils was adapted because arsenic has no intermediate duration oral MRL. A MRL based on chronic arsenic exposure should be more conservative of health and therefore the chronic oral MRL of 0.0003 mg/kg-day was used in the HI calculations.

Antimony also has no established Minimal Risk Level. For the use in this assessment, a MRL was calculated using the Lowest Observable Adverse Effect Level (LOAEL, 0.529 mg/kg-day) from a human acute exposure study (11). The LOAEL was divided by 100 to obtain a MRL of 0.00529 mg/kg-day. The 100 value was arrived at by multiplying 10, to extrapolate from the LOAEL to the No Observable Adverse Effect Level, by 10 (to account for sensitive individuals).

Total Hazard Indexes (HIs) ranged from 0.355 for the Kantishna Air Transport Driver (KATD) to 135 for pica children (Table 10). The HI is below one for the Resident Lodge Owner (RLO) and KATD (Table 11). However, the HI is above one for children indicating that a hazard exists from contaminant exposures in the Kantishna soils. The contaminant pathway of greatest concern is arsenic exposure from soil ingestion (HI >1). A Hazard Index greater than one will often occur for chemicals that have a non-carcinogenic dose higher than the Minimal Risk Level. The non-carcinogenic arsenic doses for children are also higher than the lowest observed concentration for ingestion with no adverse effects (NOAEL of 0.0004 mg/kg-day) for chronic exposure to arsenic in humans. The HI evaluation further supports that arsenic ingested by children from DU1 soils may cause adverse health effects.

Chromium Ingestion Pathway

The discussion on chromium, in Kantishna soils, is limited to the total chromium results and the assumption that 100% of the total chromium is in the more toxic, hexavalent chromium form. The Kantishna DU soils ranged from 9.9 to 20 mg/kg. These levels are not expected to cause adverse health effects in adults or children as they do not exceed the environmental Comparison Values (2,000 mg/kg adults; 200 mg/kg children) for hexavalent chromium (Table 2).

No cancer studies have been undertaken in people exposed orally to chromium (12) and animal studies indicate very little risk for cancer from oral exposure to chromium. Hexavalent chromium was not observed to cause cancer in a chronic, multi-generational study of rats exposed (9 mg/kg-day) in drinking water (12). The level of chromium in Kantishna Decision Unit soils are not at levels expected to cause non-carcinogenic or carcinogenic adverse health effects when ingested.

Chromium Dermal Pathway

Chromium dermal exposures were not calculated because chromium was below the hexavalent chromium soil Comparison Value. Dermal exposures from soil contaminants are commonly a minor contributor to the overall exposure dose relative to ingestion and inhalation pathways.

Chromium is associated with cancer only through inhalation exposures (12). Inhalation exposures were not calculated in this consultation because of a lack of data. Until chromium air concentrations are obtained, chromium is an indeterminate public health hazard for inhalation exposures.

Child Health Considerations

Children differ in the routes that contaminants are taken up and eliminated from the body then what is observed in adults. For instance, children often engage in hand-to-mouth behaviors thereby increasing the possibility of ingesting a contaminant in greater amounts then an adult. A "pica" child is one that repeatedly eats non-nutritive substances including, but not limited to, soil. Pica behavior occurs most commonly in children under the age of six with between 16-60% of children exhibiting the behavior (17). Pica children are at risk of increased potential adverse health effects from contaminants because of the behavior. If exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Additionally, children are dependent on adults for access to housing, medical care and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health. ADHSS took the special conditions involving into account when developing this health consultation.

Conclusions

The National Parks Service (NPS) would like to use mine tailings labeled Decision Unit 6 (DU6) as a source of gravel for road maintenance in the Denali National Park System. Additionally, since the Kantishna area has been heavily mined the NPS wanted to know if there is a risk to human health in the Kantishna area. Arsenic, antimony, chromium (tri- and hexa-valent), and mercury were the potential contaminants of concern in these mine tailings and also near Kantishna. The Alaska Department of Health and Social Services notes the following conclusions in regards to the contaminants of concern and the USACE risk assessment:

- A public health hazard exists for children and pica children that ingest arsenic in Kantishna DU1 soils. Potential health effects may be non-carcinogenic (skin or eye irritations) or carcinogenic.
- Ingesting or having skin contact (<168 days/year) with DU3, DU4, DU5, and DU7 soils presents no apparent health hazard for workers, adults or children exposed to arsenic, antimony, or chromium in these soils.
- The proposed borrow source, DU6, poses no apparent worker or child (without pica behavior) health hazard from soil ingestion or dermal contact pathways for the evaluated contaminants (arsenic, antimony, chromium).
- If DU6 is used for roading, then arsenic concentrations would be decreased in DU1 and DU2 roadbeds, but only if the DU6 layer remains intact upon the roadbed. Additionally, the placement of DU6 as gravel on DU3 through DU5 and DU7 roadbeds presents no apparent public health hazard from ingestion and dermal contact with arsenic, antimony, or chromium in this soil.
- Mercury is a possible contaminant in the Kantishna area. Due to a lack of data and unreliable test results an indeterminate public health hazards exist in the Kantishna area for mercury exposure through soil ingestion or the inhalation of contaminated dusts.
- Calculated doses for soil ingestion and dermal contact pathways are approximately equal (within the same order of magnitude) to those calculated by the USACE. Even with typographical and calculation errors, the qualitative findings from the USACE

risk assessment for the worker ingestion and dermal contact exposures to DU6 arsenic and antimony concentrations would be applicable.

Recommendations

General and/or Related to Decision Unit (DU) 6:

- Each Decision Unit (including DU6) should have soil samples taken and analyzed for mercury without exceeding holding times.
- For a more thorough assessment, NPS or USACE should conduct air sampling to evaluate possible contaminant exposure from dust inhalation.
- Workers are encouraged to minimize exposure to Kantishna soils by using personal protective equipment when crushing gravel and when working in the DU1 and DU2 areas.
- Prudent public health practice encourages people to wash thoroughly after working, gardening or otherwise coming in contact with Kantishna area soils.

The following recommendation is for the Kantishna area (DU1, DU2, DU3) and is not concerned with DU6 as a gravel source:

- It is recommended that children not play in the soils in the Kantishna DU1 area but rather in areas that have wood chips or vegetation (grass/moss) on top of soils.

Public Health Action Plan

This health consultation will be shared with the National Park Service and published on the Agency for Toxic Substances and Disease Registry (ATSDR) website. After the report is distributed, the Alaska Department of Health and Social Services (ADHSS) will be responsive to any health education needs that may arise. In particular, the ADHSS will coordinate with employers of the seasonal workers (e.g. NPS, lodge owners) to distribute appropriate fact sheets, posters, or other educational materials on childhood pica behavior to their employees at the park. The goal of this effort will be to educate seasonal workers who bring their children to the site for extended periods of potential health concerns from pica behavior, and to minimize exposure through proper cleaning activities (e.g. hand washing).

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Certification

This Health Consultation (Kantishna Gravel Source) was prepared by the Alaska Department of Health and Social Services under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodology and procedures existing at the time the health consultation were initiated. Editorial review was completed by the Cooperative Agreement partner.

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The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.

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Methods

Identifying Exposure Pathways

A substance is a contaminant if it is present where it does not belong. For human health effects to be possible, contaminant pathways must have the following:

1. A source of contamination, for instance a metal deposit (mined or undisturbed),
2. A way (air, water, biota) of traveling from one place to another,
3. A point where the contaminant comes in contact with a human,
4. A route of exposure (such as ingestion, inhalation, or absorption through skin), and
5. People that may be exposed to the contaminant.

If a pathway meets all of the above criteria, it is labeled as a "complete" exposure pathway (Table 2). Should information be lacking or insufficient for one of the criteria, the exposure pathway is labeled "potential". In this health consultation, pathways are labeled as potential when data are missing or absent (Table 3). A pathway is eliminated when no known exposure exists because there is no exposed population, protective measures have been undertaken, or contamination does not exist.

Dose Calculations

In order to calculate a dose, assumptions are made based upon the best available science or site-specific data (Table 4). The number of days per year (or F = frequency) that a person is exposed is estimated as well as the exposure duration (ED) which is the amount of time, in years, that a person is exposed to the contaminant. These measures are then placed into an equation, along with values for the chemical concentration and other factors (i.e. body weight, BW) important to calculating a dose (Table 4). Two types of health effects doses are possible. Health effect doses may either be non-carcinogenic or carcinogenic.

Non-carcinogenic and carcinogenic doses differ because of the value of the exposure factor (EF). The exposure factor (EF) is calculated by dividing the product of the frequency (F) and exposure duration (ED) by the averaging time (EF = F * ED/AT). A non-carcinogenic dose assumes a person is exposed to a contaminant only during the time period specified by the exposure duration. For instance, a Kantishna Air Transport Driver is only exposed to the road contaminants for a maximum of 15 years (ED_{max}) and would only have an averaging time (AT) equal to 15 years multiplied by 365 days/year or 5,475 days. On the other hand, a carcinogenic dose has the same exposure duration and frequency but the averaging time is 70 years. The averaging time for a carcinogenic dose assumes a person is exposed to the contaminant every day in a 70 year lifetime for a total of 25,550 days. Differences in averaging time between a non-carcinogenic and carcinogenic dose calculation would therefore be evident, and site-specific, in the numerical value of the exposure factor. For example, with a frequency of 78 days and an exposure duration of 5 years the non-carcinogenic exposure factor would be 0.21 (78 days*15 years/[15 years * 365 days/year]) versus a carcinogenic exposure factor of 0.05 (78 days*15 years/[70 years*365 days]).

Non-carcinogenic exposures may be further evaluated by combining multiple pathways and different contaminants. Normally, contaminants that have effects on the same system are the only exposures that are combined. In order to evaluate this interactive effect, the Hazard Index (HI) is calculated for each chemical and compared to a value of one. The HI is calculated by dividing the non-carcinogenic dose by the Minimal Risk Level. The resultant HI values are then added together to get a total HI for the combined chemicals. The use of the HI is strictly for the

interpretation on whether combined chemicals or multiple pathways may cause adverse health effects. A HI greater than one would indicate that the sum of the doses may result in greater adverse health effects when exposures occur together versus when exposures occur by themselves. HI numbers less than one indicate that it is highly unlikely that greater health effects would occur from the combination of chemicals.

If a chemical is a known carcinogen and its concentration exceeds the Cancer Risk Evaluation Guide (CREG) Comparison Value then a carcinogenic dose is calculated and compared to doses observed to result in cancer in experimental studies (also known as a cancer effect level or CEL). To further place the risk of carcinogen exposure in perspective an estimate of the theoretical Excess Lifetime Cancer Risk (ELCR) is calculated for carcinogenic contaminants.

Table 1. Metal concentrations and Comparison Values (CVs) from Kantishna soil samples taken from borrow sources (2005) and background (2006) areas

Element	Concentration (mg/kg) in		Comparison Value. CV (mg/kg)		Further Consideration Needed
	Borrow Source (2005)	Background (2006)	Concentration, C (mg/kg)	Source	
Mercury (Hg)	HE	nd (0.04) - 0.067	1.4	ADEC cleanup level	Yes
Arsenic (As)	27 - 170	14 - 110	20 200 10 0.5	child chronic EMEG adult chronic EMEG pica acute EMEG CREG	Yes
Antimony (Sb)	nd (0.53) - 140†	nd (6.0) - 33	20 300	child chronic EMEG adult chronic EMEG	Yes
Barium (Ba)	29 - 100	16 - 78	30,000 400,000 1,000	child chronic EMEG adult chronic EMEG pica int EMEG	No
Cadmium (Cd)	nd (0.048) - 1.6	nd (0.4) - 0.85	10 100	child chronic EMEG adult chronic EMEG	No
Total Chromium (Cr^{3+}, Cr^{6+})	15 - 44	11 - 33	200* 2,000*	child RMEG adult RMEG	No
Lead (Pb)	10 - 30	9.9 - 88	400	EPA cleanup level	No
Selenium (Se)	nd (2.0)	nd (5.4)	300 4,000	child chronic EMEG adult chronic EMEG	No
Silver (Ag)	nd (0.12)	6.1 - 7.9	300 4,000	child RMEG adult RMEG	No

EMEG- Environmental Media Evaluation Guide; CREG- Cancer Risk Evaluation Guide; RMEG- Reference Dose Media Evaluation Guide (ATSDR); * RMEG for chromium is for soluble salts of the hexavalent species (Cr^{6+}) only; No other CV reported for chromium; † USACE reported 48 mg/kg as the highest concentration range when sample DPKG 17 was 140 mg/kg; nd = non-detect; number in () is lowest method reporting limit for chemical on this group of samples; HE - hold time exceeded; int = intermediate; ADEC = Alaska Dept Environmental Conservation

Table 2. Completed exposure pathways

Pathway Name	Source	Media (Way)	Point	Route	Time Frame of Exposure	Estimated Number of People Exposed	Contaminant of Concern †
Soil Ingestion	Contaminants from nature and or mining activities	Tailings pile erosion; soils	DU1-DU6 and potentially surrounding areas	Incidental ingestion from road work, gardening, playing	Past Present Future	≤ 2,000 ≤ 400 ≤ 400	Arsenic, Antimony
Dermal Contact	Contaminants from nature and or mining activities	Dust from contaminated rock crushing; soils	Roadway and surrounding area	Absorption through skin when soiled during road work, gardening, playing	Past Present Future	≤ 2,000 ≤ 400 ≤ 400	Arsenic, Antimony

† The contaminants listed on this table are above soil screening Comparison Values. Other contaminants may exist in Kantishna soils that are or may not be a concern for health effects.

Table 3. Potential exposure pathways

Pathway Name	Source	Media (Way)	Point	Route	Time Frame of Exposure	Estimated Number of People Exposed	Contaminant of Concern †
Soil Ingestion	Contaminants from nature and or mining activities	Tailings pile erosion	DU1-DU6 and potentially surrounding areas	Incidental ingestion from road work, gardening, playing	Past Present Future	≤ 2,000 ≤ 400 ≤ 400	Unknown; Potentially mercury
Dust Inhalation	Contaminants from nature and or mining activities	Erosion of contaminated soils	Roadway and potentially surrounding area	Inhalation when near roadway, driving, or working	Past Present Future	≤ 2,000 ≤ 400 ≤ 400	Unknown; Potentially arsenic, antimony, mercury

† Other contaminants may exist in Kantishna soils that may or may not be a concern for health effects.

Table 4. Representative worker exposure timeframes for Kantishna

Job	Days/Wk	Weeks	Frequency (F) days/yr	Avg Exposure Duration (ED) yr	Maximum Exposure Duration (ED_{max}) yr	Note
Resident-Lodge Owner (RLO)-Caretaker	-	-	168	35	70	Assumes RLO at Kantishna from May 1 – Oct 15 and returns yearly for a lifetime (ED_{max})
Kantishna Air Transport Driver (KATD)	6	13	78	5	15	
Wonder Lake Grader Operator, roading (WLGO, roading)	4	17	68	5	10	
WGLO, grading	1.33†	17	23†	5	10	† 4 days/3 weeks = 1.33 d/wk, 1.33 d/wk* 17 wks = 23 d/yr EF
Wonder Lake Laborer (WLL)	2.5	16	40†	5	10	† EF = 2.5 d/wk*16 wk = 40 d

† indicates change from USACE risk assessment; EF = F*ED/AT where EF = Exposure Factor, F = Frequency (days/yr); ED = Exposure Duration (time exposed to contaminant); AT = Averaging Time (days for duration of exposure); AT = number of years of exposure*days/year,

Table 5. Comparison of contaminants in Kantishna samples from 2007 with ATSDR Comparison Values (CVs)

Metal	Maximum sample concentration (mg/kg)							ATSDR CV (mg/kg)		
	DU1	DU2	DU3	DU4	DU5	DU6 §	DU7 road bed	EMEG	CREG	RMEG
Arsenic	300	140	23	19	23	70	35	200[a,10] 20[c]	0.5[4]	200[a, 10] 20[c]
Antimony	17	34	2.4	1.4	1.5	21	6.6	-	-	300[a, 11] 20[c]
Chromium, total	9.9	12	18	15	17	9.9	20	-	-	-
Chromium, hexavalent*	HE	HE	HE	HE	HE	HE	HE	-		2,000[a,12] 200[c]

HE = Hold time exceeded, results invalid; a = adult; c = child; 4, 10-12 = see references; CREG- Cancer Risk Evaluation Guide; EMEG- Environmental Media Evaluation Guide; RMEG- Reference Dose Media Evaluation Guide; * hexavalent chromium is a human carcinogen for inhalation exposures; oral exposure is not classifiable for human carcinogenicity; § Proposed borrow source

Table 6. Representative non-carcinogenic contaminant dose calculations from soil ingestion

Individual	Arsenic C_{max} (Location) mg/kg	Antimony C_{max} (Location) mg/kg	F d/yr	ED_{max} yr	Time (AT) max days	Non-carcinogenic Dose (mg/kg-day) Arsenic max	Non-carcinogenic Dose (mg/kg-day) Antimony max
Resident Lodge Owner (RLO)	300 (DU1)	17 (DU1)	168	70	25,550	0.0002	0.00002
Kantishna Air Transport Driver	300 (DU1)	34 (DU2)	78	15	5,475	0.00009	0.00001
Wonder Lake Grader Operator, roading	300 (DU1)	-	68	10	3,650	0.00008	-
Wonder Lake Grader Operator, grading	300 (DU1)	-	23	10	3,650	0.00003	-
Wonder Lake Laborer	300 (DU1)	-	40	10	3,650	0.00005	-
Kantishna Air Transport Driver	70 (DU6)	21 (DU6)	78	15	5,475	0.00002	0.000006
Wonder Lake Grader Operator, roading	70 (DU6)	-	68	10	3,650	0.00002	-
Wonder Lake Grader Operator, grading	70 (DU6)	-	23	10	3,650	0.000006	-
Wonder Lake Laborer	70 (DU6)	-	40	10	3,650	0.00001	-
Child	300 (DU1)	34 (DU2)	168	6	2,190	0.002	0.0002
Pica Child	300 (DU1)	34 (DU2)	168	6	2,190	0.04	0.005
Child	70 (DU6)	21 (DU6)	168	6	2,190	0.0004	0.0001
Pica Child	70 (DU6)	21 (DU6)	168	6	2,190	0.01	0.003

Dose = (C*IR*AF*F*ED*CF)/(BW*AT) where C_{max}= maximum concentration observed in DUs (mg/kg); IR = Intake Rate of contaminated soil per day (mg/day), 100 mg/d for adult, 200 mg/d for child or 5,000 mg/d for pica child (13); EF = Exposure Factor and EF = F*ED/AT; F = Frequency (days/yr) ED = Exposure Duration (time exposed to contaminant); AT = Averaging Time (days for duration of exposure) and AT = years of exposure*365 days/year; AF = Bioavailability Factor (bioavailability of the contaminant), 1 (13); CF = Conversion Factor, 0.0000001 kg/mg (to standardize units); BW = Body Weight (kg), 70 kg-adult and 16 kg-child (13); Child EMEG CV for arsenic = 20 mg/kg; RMEG for antimony = 20 mg/kg; No intermediate arsenic Minimal Risk Level (MRL). Arsenic MRL = 0.0003 mg/kg-day (chronic [≥ 365 d] exposure); No MRL for antimony. LOAEL for rats exposed to antimony is 0.0748 mg /kg-day.

Table 7. Representative carcinogenic doses for arsenic from soil ingestion

Individual	Arsenic C_{max} (Location) mg/kg	F d/yr	Exposure Duration, ED avg(max) yr	Time, AT days	Carcinogenic Dose (mg/kg-day) Arsenic ED_{avg}	Arsenic ED_{max}
Resident Lodge Owner (RLO)	**300** (DU1)	**168**	**35(70)**	25,550	0.0001	**0.0002**
Kantishna Air Transport Driver	70 (DU6)	78	5(15)	25,550	0.000002	0.000005
Wonder Lake Grader Operator, roading	70 (DU6)	68	5(10)	25,550	0.000001	0.000003
Wonder Lake Grader Operator, grading	70 (DU6)	23	5(10)	25,550	0.0000005	0.0000009
Wonder Lake Laborer	70 (DU6)	40	5(10)	25,550	0.0000008	0.000002
Child	300 (DU1)	168	-(6)	25,550	-	0.00015
Pica Child	300 (DU1)	168	-(6)	25,550	-	0.0037
Child	70 (DU6)	168	-(6)	25,550	-	0.00003
Pica Child	70 (DU6)	168	-(6)	25,550	-	0.00086

Dose = (C*IR*AF*F*ED*CF)/(BW*AT) where C_{max}= maximum concentration observed in DUs (mg/kg); IR = Intake Rate of contaminated soil per day (mg/day), 100 mg/d adult, 200 mg/d for child or 5,000 mg/d for pica child (13); EF = Exposure Factor and EF = F*ED/AT; F = Frequency (days/yr); ED = Exposure Duration (time exposed to contaminant); AT = Averaging Time (days for duration of exposure) and AT = years of exposure*365 days/year where 70 yr*365 d/yr = 25,550 days (13); AF = Bioavailability Factor (bioavailability of the contaminant), 1 (13); CF = Conversion Factor, 0.000001 kg/mg (to standardize units); BW = Body Weight , 70 kg for adult and 16 kg-child (13); Cancer Effect Level (CEL) for humans = 1.1 (water) - 40 (ingestion) µg/kg-day.

Representative calculation (bold blocks) for carcinogenic dose of a Resident Lodge Owner at 300 mg/kg and using maximum exposure duration and time:

$$\text{Dose} = \frac{300\ \text{mg/kg}*100\ \text{mg/d}*1*168\ \text{d/yr}*70\ \text{yr}*0.000001\ \text{k/mg}}{(70\ \text{kg}*25{,}550\ \text{d})} = 0.0002\ \text{mg/kg-day}$$

Convert from mg/kg-day to µg/kg-day = 0.0002 mg/kg-day*1000 µg/mg = 0.2 mg/kg-day

Table 8. Representative theoretical Excess Lifetime Cancer Risk (ELCR) for arsenic in ingested soil

Individual	Carcinogenic Dose (mg/kg-day)		ELCR	ELCR
	Arsenic ED_{avg}	Arsenic ED_{max}	ED_{avg}	ED_{max}
Resident Lodge Owner	NC	0.0002	NC	0.0003
Kantishna Air Transport Driver	0.000002	0.000005	0.000002	0.000007
Wonder Lake Grader Operator, roading	0.000001	0.000003	0.000002	0.000004
Wonder Lake Grader Operator, grading	0.0000005	0.0000009	0.0000007	0.000001
Wonder Lake Grader Operator, Total	0.000002	0.000004	0.000003	0.000005
Child	-	0.00015	-	0.00022
Pica Child	-	0.0037	-	0.00555
Child	-	0.00003	-	0.00005
Pica Child	-	0.00086	-	0.00129

ELCR = Carcinogenic dose *Cancer Slope Factor (CSF); CSF for arsenic = 1.5 mg/kg-day (4); NC = Not Calculated.

Table 9. Dermal contact dose calculations for workers and children

Individual	Arsenic C_{max} (Location)	Antimony C_{max} (Location)	AF	F	ED max	Non-carcinogenic Dose µg/kg-day		Carcinogenic Dose µg/kg-day	ELCR
	mg/kg			d/yr	yr	Arsenic	Antimony	Arsenic	
Resident Lodge Owner (RLO)	300 (DU1)	17 (DU1)	0.1	168	70	0.034	0.0001	0.034	0.000051
Kantishna Air Transport Driver	300 (DU1)	34 (DU2)	0.1	78	15	0.016	0.0001	0.003	0.000005
Child	300 (DU1)	34 (DU2)	0.3	168	6	0.217	0.0008	0.019	0.000029
Child	**70** (DU6)	21 (DU6)	0.3	**168**	**6**	**0.051**	0.0005	0.004	0.000006

D = (C*A*AF*F*ED*CF)/(BW*AT) where D = Dose (mg/kg/day); C_{max}= maximum concentration observed in DUs (mg/kg); A = Total soil adhered (mg); A = SA*soil adherence factor; SA = Surface Area (cm^2) where SA_{adult} = 5700 cm^2; SA_{child} = 2800 cm^2 and assumes adult and child (1 – 6 years old) with short-sleeve shirt and shorts; shoes for adults and no shoes for children (15); Soil adherence factor, arsenic = 0.03 (14); Antimony soil adherence factor = 0.001 (16). A therefore equals 171 mg for adult arsenic exposures, 84 for child arsenic exposures and 5.7 mg for adult antimony exposures and 2.8 mg for child antimony exposures; AF = Bioavailability Factor (bioavailability of the contaminant) and based on RLO as a groundskeeper; children playing in- and out-doors (16; exhibit 3-3; 95th percentile);F = Frequency (days/yr); ED = Exposure Duration (time in years exposed to contaminant); CF = Conversion Factor 0.001 µg-kg/mg^2 (to standardize units); BW = Body Weight 70 for adults and 16 for children (Kg); AT = Averaging Time (days for duration of exposure); AT = years of exposure*365 days/year where AT = ED_{max}*365 d/y (non-carcinogenic) or AT =70 y*365 d/y =25,550 d (carcinogenic); ELCR = Arsenic Carcinogenic Dose (in mg/kg-day) *1.5 (mg/kg-day)

Example calculations (bold blocks) for child at 70 mg/kg arsenic concentration:

A = 2800 cm^2 * 0.03 mg/cm^2 = 84 mg

$$\text{Dose} = \frac{70\ \text{mg/kg} * 84\ \text{mg} * 0.3 * 168\ \text{d/yr} * 6\ \text{yr} * 0.001\ \mu\text{g-kg/mg}^2}{16\ \text{kg} * 2190\ \text{d}} = 0.05\ \mu\text{g/kg-day}$$

Table 10. Hazard Index (HI) and cumulative arsenic Excess Lifetime Cancer Risk (ELCR) for all pathways

Individual	Pathway	Arsenic Maximum $Dose_{non\text{-}carcinogenic}$ (mg/kg-day)	Antimony Maximum $Dose_{non\text{-}carcinogenic}$ (mg/kg-day)	Arsenic + Antimony HI_{max}	Arsenic ELCR
Resident Lodge Owner (RLO)	Soil ingestion	**0.00020**	**0.00002**	**0.673**	0.00030
	Dermal absorption	0.000034	0.0000001	0.113	0.00003
RLO Total		**0.000234**	**-**	**0.784**	**0.00033**
Kantishna Air Transport Driver (KATD)	Soil ingestion	0.00009	0.00001	0.302	0.000007
	Dermal absorption	0.000016	0.0000001	0.053	0.000003
KATD Total		**0.000106**	**-**	**0.355**	**0.00001**
Child	Soil ingestion	0.002	0.0002	6.704	0.00022
	Dermal absorption	0.000217	0.0000008	0.723	0.00003
Child Total		**0.002217**	**-**	**7.428**	**0.00025**
Pica Child	Soil ingestion	0.04	0.005	134.279	0.00555
	Dermal absorption*	0.000217	0.000008	0.723	0.00003
Pica Child Total		**0.040217**	**-**	**135.002**	**0.00558**

* Dermal absorption pathway dose and ELCR for pica child is the same as a child; - is not applicable; the two pathways are observed to target different organs for antimony toxicity; HI = sum of non-carcinogenic doses = $Dose_{As}/MRL_{Arsenic} + Dose_{Sb}/MRL_{Antimony}$‡; $MRL_{Arsenic}$ = 0.0003 mg/kg-day (chronic); ‡ No MRL for antimony; Antimony MRL calculated from LOAEL of 0.529 divided by 100 (for uncertainty) = 0.00529 mg/kg-day

Representative Hazard Index (HI) calculation (bold blocks) for RLO and the soil ingestion pathway:
HI = (0.00020/0.0003) + (0.00002/0.00529) = 0.67 + 0.00378 = 0.67378

Table 11. Risk of non-carcinogenic health effects from arsenic soil ingestion for vacationing children in Kantishna DU1. **See caution

Individual	Arsenic C_{max} mg/kg	Intake Rate (IR) mg/kg	F† d/yr	Exposure Duration (ED) yr	Time (AT) days	$Arsenic_{max}$ Non-carcinogenic Dose mg/kg-day	Minimal Risk Level (MRL) mg/kg-day	Non-carcinogenic Risk?
Child	300	200	7	1	365	0.00007	0.005	No
Pica Child	300	5000	7	1	365	0.002	0.005	No
Child	300	200	14	1	365	0.0001	0.005	No
Pica Child	300	5000	14	1	365	0.004	0.005	No
Child	300	200	21	1	365	0.0002	0.0003	No
Pica Child	300	5000	21	1	365	0.0054	0.0003	Yes
Child	300	200	7	2	730	0.00007	0.005	No
Pica Child	300	5000	7	2	730	0.002	0.005	No
Child	300	200	14	2	730	0.0001	0.005	No
Pica Child	300	5000	14	2	730	0.004	0.005	No
Child	300	200	21	2	730	0.0002	0.0003	No
Pica Child	300	5000	21	2	730	0.0054	0.0003	Yes
Child	300	200	7	3	1095	0.00007	0.005	No
Pica Child	300	5000	7	3	1095	0.002	0.005	No
Child	300	200	14	3	1095	0.0001	0.005	No
Pica Child	300	5000	14	3	1095	0.004	0.005	No
Child	300	200	21	3	1095	0.0002	0.0003	No
Pica Child	300	5000	21	3	1095	0.0054	0.0003	Yes
Child	300	200	7	4	1460	0.00007	0.005	No
Pica Child	300	5000	7	4	1460	0.002	0.005	No
Child	300	200	14	4	1460	0.0001	0.005	No
Pica Child	300	5000	14	4	1460	0.004	0.005	No
Child	300	200	21	4	1460	0.0002	0.0003	No
Pica Child	300	5000	21	4	1460	0.0054	0.0003	Yes
Child	300	200	7	5	1825	0.00007	0.005	No
Pica Child	300	5000	7	5	1825	0.002	0.005	No
Child	300	200	14	5	1825	0.0001	0.005	No
Pica Child	300	5000	14	5	1825	0.004	0.005	No
Child	300	200	21	5	1825	0.0002	0.0003	No
Pica Child	300	5000	21	5	1825	0.0054	0.0003	Yes
Child	300	200	7	6	2190	0.00007	0.005	No
Pica Child	300	5000	7	6	2190	0.002	0.005	No
Child	300	200	14	6	2190	0.0001	0.005	No

Table 11 (Cont'd). Risk of non-carcinogenic health effects from arsenic soil ingestion for vacationing children in Kantishna DU1. **See caution

Individual	Arsenic C_{max} mg/kg	Intake Rate (IR) mg/kg	F† d/yr	Exposure Duration (ED) yr	Time (AT) days	$Arsenic_{max}$ Non-carcinogenic Dose mg/kg-day	Minimal Risk Level (MRL) mg/kg-day	Non-carcinogenic Risk?
Pica Child	300	5000	14	6	2190	0.004	0.005	No
Child	300	200	21	6	2190	0.0002	0.0003	No
Pica Child	300	5000	21	6	2190	0.0054	0.0003	Yes

F = Frequency; conservative (3 days considered max) estimate of the amount of time that a child may vacation in the Kantishna area in any year between the ages of 1 and 6 (T. Scholten, 2008); ED is the number of years a child returns to vacation in Kantishna DU1 area; AT = ED*365 d/yr; IRs, AF = 1, BW = 16 kg (ATSDR, 2007); Dose = (C*IR*AF*F*ED*CF)/(BW*T); CF = 0.000001 kg/mg; Acute (to 14 days) MRL arsenic = 0.005 mg/kg-day; no intermediate MRL for arsenic, chronic MRL is 0.0003 mg/kg-day; F† below 7 days would have even less risk of non-carcinogenic health effects; See Table 7 for representative calculations.

Time period where children and children with pica behavior are likely not to have risk of non-carcinogenic health effects from arsenic exposure in DU1 soils

**Caution: The following tables should be used with caution because Table 11 allows for a longer frequency (number of days) that a child may be exposed without effects whereas Table 12 does not. Tables 11 & 12 should be used in coordination for health risk. An example: If a child is expected to visit Kantishna for 3 years, Table 12 would indicate that the maximum amount of time a child should spend there would be 4 days. That is 4 days for a period of 3 years or 4 days/year. Do not misinterpret the tables. A child should not visit 14 days in the first year and 7 days in the second year if the child is returning for a third year. Another words, by three years of return vacations to the Kantishna DU1 area a child would be limited to a 4 day stay (in every year) and this would be further restricted to 3 days by year five. Please note that exposure duration (ED) would be equal to the number of times a child is expected to return to the Kantishna area for vacation between the ages of 1 and 6 years.

It is unknown the type of behavior a child that may vacation in Kantishna may exhibit. With that being the case, then the maximum amount of time a child might spend in Kantishna would be less than 14 days if they return every year from 1-6 years of age. Please note, however, the Excess Lifetime Cancer Rate (ELCR) for pica children vacationing for 14 days in the Kantishna area is slightly increased (1.5 excess cancers per 10,000 people) after only 2 years of vacations in the area and this may not be acceptable to some parents (Table 12).

Table 12. Arsenic carcinogenic dose calculations and Excess Lifetime Cancer Risk (ELCR) for vacationing children from Kantishna DU1 soil ingestion. **See caution

Individual	Arsenic C_{max} mg/kg	Intake Rate (IR) mg/kg	F d/yr	Exposure Duration (ED) yr	Time (AT) days	Arsenic Carcinogenic Dose mg/kg-day	ELCR ED_{max}	Increased ELCR?
Child	300	200	3	1	25550	0.0000004	0.000001	No
Pica Child	300	5000	3	1	25550	0.000011	0.00002	No
Child	300	200	4	1	25550	0.0000006	0.000001	No
Pica Child	300	5000	4	1	25550	0.000015	0.00002	No
Child	300	200	7	1	25550	0.0000010	0.000002	No
Pica Child	300	5000	7	1	25550	0.000026	0.00004	No
Child	300	200	14	1	25550	0.0000021	0.000003	No
Pica Child	300	5000	14	1	25550	0.000051	0.00008	No
Child	300	200	21	1	25550	0.0000031	0.000005	No
Pica Child	300	5000	21	1	25550	0.000077	0.00012	Yes
Child	300	200	3	2	25550	0.0000009	0.000001	No
Pica Child	300	5000	3	2	25550	0.000022	0.00003	No
Child	300	200	4	2	25550	0.0000012	0.000002	No
Pica Child	300	5000	4	2	25550	0.000029	0.00004	No
Child	300	200	7	2	25550	0.0000021	0.000003	No
Pica Child	300	5000	7	2	25550	0.000051	0.00008	No
Child	300	200	14	2	25550	0.0000041	0.000006	No
Pica Child	300	5000	14	2	25550	0.000103	0.00015	Yes
Child	300	200	3	3	25550	0.0000013	0.000002	No
Pica Child	300	5000	3	3	25550	0.000033	0.00005	No
Child	300	200	4	3	25550	0.0000018	0.000003	No
Pica Child	300	5000	4	3	25550	0.000044	0.00007	No
Child	300	200	7	3	25550	0.0000031	0.000005	No
Pica Child	300	5000	7	3	25550	0.000077	0.00012	Yes
Child	300	200	3	4	25550	0.0000018	0.000003	No
Pica Child	300	5000	3	4	25550	0.000044	0.00007	No
Child	300	200	4	4	25550	0.0000023	0.000004	No
Pica Child	300	5000	4	4	25550	0.000059	0.00009	No
Child	300	200	7	4	25550	0.0000041	0.000006	No
Pica Child	300	5000	7	4	25550	0.000103	0.00015	Yes
Child	300	200	3	5	25550	0.0000022	0.000003	No
Pica Child	300	5000	3	5	25550	0.000055	0.00008	No
Child	300	200	4	5	25550	0.0000029	0.000004	No
Pica Child	300	5000	4	5	25550	0.000073	0.00011	Yes

Table 12 (Cont'd). Arsenic carcinogenic dose calculations and Excess Lifetime Cancer Risk (ELCR) for vacationing children from Kantishna DU1 soil ingestion. **See caution

Individual	Arsenic C_{max} mg/kg	Intake Rate (IR) mg/kg	F d/yr	Exposure Duration (ED) yr	Time (AT) days	Arsenic Carcinogenic Dose mg/kg-day	ELCR ED_{max}	Increased ELCR?
Child	300	200	3	6	25550	0.000003	0.000004	No
Pica Child	300	5000	3	6	25550	0.000066	0.00010	No
Child	300	200	4	6	25550	0.000004	0.00001	No
Pica Child	300	5000	4	6	25550	0.000088	0.00013	Yes

Dose = (C*IR*AF*F*ED*CF)/(BW*AT); CF =0.000001 kg/mg; ELCR = Dose*1.5 (arsenic cancer slope factor); AT = 25,550 days; ED = 6 yr; Assumes a child (1 - 6 yrs old) with daily soil ingestion for F days per year; IRs, AF = 1; BW = 16 kg (13). See Table 7 for representative calculations.

ATSDR and EPA policy designates an upper limit of acceptable cancer risk levels (ELCR) at 0.0001 (1 excess cancer per 10,000 people) though this is up to parent. "Excess" in terms of above normal background cancer rates.

**Caution: It is unknown the type of behavior a child that may vacation in Kantishna may exhibit. With that being the case, then the maximum amount of time a child might spend in Kantishna would be 3 days, if they return for 5 or 6 years during childhood (1-6 years old).

Glossary

Alaska Department of Environmental Conservation (ADEC)	Alaska state government agency tasked with conserving, improving and protecting Alaska's natural resources and the environment.
Alaska Department of Health and Social Services (ADHSS)	Alaska state government agency with the mission to promote and protect the health and well-being of all Alaskans.
Agency for Toxic Substances and Disease Registry (ATSDR)	The principal federal public health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services.
Area of Concern (AOC)	The area of contamination and sometimes surrounding areas based on the ability of contaminants to be transported by environmental factors or media.
Averaging Time (AT)	The total number of days that a person is exposed to a contaminant or the years exposed to a contaminant (ED) multiplied by the number of days in a year (365). AT = ED (years)* 365 (days/year)
Cancer Risk Evaluation Guide (CREG)	The concentration of a chemical in air, soil or water that is expected to cause no more than one excess cancer in a million persons exposed over a lifetime. The CREG is a *Comparison Value* used to select contaminants of potential health concern and is based on the *cancer slope factor* (CSF).
Cancer Effect Level (CEL)	The lowest dose level observed to produce a significant increase in the incidence of cancer or tumors (as shown in human epidemiologic or experimental animal studies).
Cancer Slope Factor (CSF)	A number assigned to a cancer-causing chemical that is used to estimate its ability to cause cancer in humans.
Carcinogen	Any substance that causes cancer.
Comparison value (CV)	Concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people.
Contaminant	A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.
Dermal Contact	Contact with the skin (see route of exposure).

Dose (for chemicals that are not radioactive)	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. An "exposure dose" is how much of a substance is encountered in the environment but it is not necessarily the amount that is absorbed.
Environmental Media Evaluation Guide (EMEG)	A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The EMEG is a *Comparison Value* used to select contaminants of potential health concern and is based on ATSDR's *Minimal Risk Level* (MRL).
Environmental Protection Agency (EPA)	United States Environmental Protection Agency. The mission of the Environmental Protection Agency is to protect human health and the environment.
Excess Lifetime Cancer Risk (ELCR)	A theoretical risk for developing cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be acute (14 days or less), intermediate (15-364 days) or chronic (365 days or more).
Exposure Duration (ED)	The amount of time, in years, that a person is exposed to a contaminant.
Frequency (F)	The number of days per year that a person is exposed to a contaminant.
Hazard Index (HI)	The sum of the quotients of the estimated dose of a chemical divided by its MRL or comparable value. $HI = Dose_1/MRL_1 + Dose_2/MRL_2 + \ldots Dose_n/MRL_n$
Ingestion	The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].
Ingestion Rate (IR)	The amount of an environmental medium that could be ingested typically on a daily basis.
Inhalation	The act of breathing.
Inorganic	Compounds made of non-carbon elements such as salts and metals.
Lowest Observed Adverse Effect Level (LOAEL)	The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
Media	Soil, water, air, plants, animals, or any other part of the environment that can contain contaminants.

Minimal Risk Level (MRL)	An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), non-cancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic).
National Park Service (NPS)	A federal agency involved in caring for national parks, a network of nearly 400 natural, cultural and recreational sites across the nation. The NPS also preserves and enhances important local heritage and close-to-home recreational opportunities.
No apparent public health hazard	A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.
No Observed Adverse Effect Level (NOAEL)	The highest dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals for the organ system studied.
Reference Dose (RfD)	An amount of chemical that can be ingested daily over the course of a lifetime and not cause serious adverse health effects. RfDs are calculated and published by EPA.
Organic	Compounds containing carbon.
Reference Dose Media Evaluation Guide (RMEG)	A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The RMEG is a *Comparison Value* used to select contaminants of potential health concern and is based on EPA's oral reference dose (RfD).
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
Surface Water	Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs.
United States Army Corp of Engineers (USACE)	A federal agency involved in engineering and environmental matters. Its mission is to provide quality, responsive engineering services to the nation.