Health Consultation

Palestine Bioavailability Study
Arsenic and Vanadium Soil Action Levels

Palestine, Anderson County, Texas

EPA No. TXN000605670

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Prepared by

Texas Department of State Health Services
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

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Purpose and Statement of Issues

In response to recommendations made in previous health consultations, the U.S. Environmental Protection Agency Region 6 (EPA-6) contracted with the College of Veterinary Medicine at the University of Missouri, Columbia (CVMUM) to assess the relative bioavailability of arsenic and vanadium in soil from the Palestine area. Using data from this study, scientists from EPA-6 proposed residential soil action levels of 155 mg/kg for arsenic and 975 mg/kg for vanadium. The EPA has asked the Texas Department of State Health Services (DSHS) and the Agency for Toxic Substances and Disease Registry (ATSDR) for an independent assessment to evaluate whether the proposed residential surface soil action levels for arsenic and vanadium would be protective of public health (Note: Appendix A lists abbreviations and acronyms used in this report).

Background

Site Description and History

The area in question, a mixed industrial/residential area, is located in the southeastern portion of Palestine, Anderson County, Texas. Although the exact source and extent of the contamination have not been determined, historically, two major industrial facilities operated in the area: the Palestine Light, Heat, and Power Company and the former George M. Dilley and Son, Founders and Machinists Shop (also known as the Palestine Foundry).

The Palestine Light, Heat, and Power Company site is a one-acre site that was formerly a town coal gas operation. The site consists of several waste piles (railroad ties, concrete blocks, and rock piles) from the adjacent railroad system on the east side of the property. The Palestine Foundry site is north of the Palestine Light, Heat, and Power Company and east of the railroad tracks on South May Street. It was the first large industry in Palestine and operated from 1873 to 1949. Historically, several buildings and a smoke stack may have existed on the property; only two buildings currently remain and the site is heavily vegetated [1].

Both properties are accessible to children as well as adults. Residential homes are located across South May Street from the Palestine Foundry. The fence along the road does not prevent access to the foundry property. The foundry office building is dilapidated and thereby poses a physical hazard.

In previous consults [2, 3], it was determined that there may be human exposure to elevated levels of arsenic, vanadium, and lead in the soil and that selected residences and daycare facilities warranted further attention [3].

Discussion

In determining the health risks that may be associated with contaminants found in various media, the concentration of each contaminant is compared to its health-based assessment comparison (HAC) value for non-cancer and cancer endpoints. These values are guidelines that specify

levels of chemicals in specific environmental media (soil, air, and water) that are considered safe for human contact with respect to identified human endpoints. Non-cancer screening values are generally based on ATSDR's minimal risk levels (MRLs)[1] and EPA's reference doses (RfDs)[2]. Both of these are based on the assumption that there is an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects. Thus, MRLs and RfDs are estimates of daily exposures to contaminants that are unlikely to cause adverse non-cancer health effects even if exposure occurs for a lifetime. The cancer risk comparison values used in this consultation are based on EPA's chemical-specific cancer slope factors (CSFs)[3].

Knowing how much of the contaminant is actually absorbed into the body (bioavailability) is important in determining the potential risks associated with the contaminant [4]. The bioavailability of any contaminant is both soil- and contaminant-specific; thus, it is not possible to predict bioavailability without considering these factors. In the absence of bioavailability data, and in order to be protective of public health, it is customary to assume that a contaminant is 100% bioavailable; all the contaminant that enters the body is absorbed into the blood. In reality the rate and extent of absorption of a chemical from an ingested media such as soil is much less than 100%.

CVMUM determined the bioavailability of the contaminants at this site by feeding site specific soil to juvenile swine. Swine were given oral doses of sodium arsenate, vanadyl sulfate, or the Palestine soil; and the relative bioavailability of arsenic and vanadium was assessed by comparing the absorption of arsenic or vanadium from the reference material to that of the test soil [4]. Results of this study indicated that the relative bioavailability of arsenic and vanadium in Palestine soil was approximately 15% and 8%, respectively [4].

Using the results of the CVMUM bioavailability study, EPA revised the action levels for arsenic and vanadium in Palestine soil to 155 and 975 mg/kg, respectively [5]. DSHS was asked to conduct an independent assessment to evaluate whether these revised action levels for residential soil would be protective of public health.

Arsenic

Arsenic is a naturally occurring element that is widely distributed throughout the earth's crust and may be found in air, water, and soil [6]. Arsenic exists as inorganic arsenic, organic arsenic, and arsine gas. Generally, organic arsenic is less toxic than inorganic arsenic, with some forms of organic arsenic being virtually non-toxic. Inorganic arsenic occurs naturally in soil, and children may be exposed to arsenic by eating soil or by direct skin contact with soil or water

[1] An MRL is a contaminant specific exposure dose below which adverse health effects in the people most sensitive to chemical-induced effects would not be expected. MRLs generally are based on the most sensitive chemical-induced end point considered to be of relevance to humans.
[2] An RfD is an estimate (with a level of uncertainty from 10 to 1000 times below the level of harmful effects) of a daily exposure to the human population (including sensitive groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.
[3] A CSF is an estimate of excess lifetime risk of one cancer in one million (1×10^{-6}) people exposed over a lifetime (70 years).

containing arsenic [6]. Children and adults normally take in small amounts of arsenic through inhalation of air and ingestion of food and water, with food being the largest source of arsenic. Fish and seafood contain the highest concentrations of arsenic; however, most of this is in the less toxic organic form of arsenic [6].

A chronic oral MRL of 0.0003 mg/kg/day for inorganic arsenic was derived by dividing the identified chronic No Observable Adverse Effect Levels (NOAEL) of 0.0008 mg/kg/day (obtained from human epidemiologic studies) by an uncertainty factor of three to account for the lack of data on reproductive toxicity and to account for some uncertainty as to whether the NOAEL accounts for all sensitive individuals [6]. The Lowest Observed Adverse Effect Levels (LOAEL) associated with these epidemiologic studies was 0.014 mg/kg/day, where exposure to arsenic above this level resulted in hyperpigmentation of the skin, keratosis (patches of hardened skin), and possible vascular complications [6]. The chronic oral MRL was used to evaluate the potential public health significance of exposure to arsenic.

A provisional acute oral MRL of 0.005 mg/kg/day for inorganic arsenic was derived by dividing the LOAEL of 0.05 mg/kg/day (based upon gastrointestinal effects and facial edema in humans) by an uncertainty factor of 10 for extrapolation from a LOAEL to a NOAEL [6]. The acute oral MRL was used to evaluate the potential public health significance of acute exposure to arsenic due to pica behavior.

Inorganic arsenic is a known carcinogen [6]. The most characteristic effect of long-term exposure to inorganic arsenic is a pattern of skin changes including darkening of the skin and the appearance of small “corns” or “warts” on the palms, soles, and torso. A small number of these corns may ultimately develop into skin cancer. Liver, bladder, kidney, and lung cancer also have been associated with exposure to arsenic [6]. While there are certainly both cases of and deaths due to these types of cancer in Palestine, Texas, it would be impossible to determine if any one of these cancers was caused by exposure to arsenic b[0]ecause there is nothing that indisputably links these cancers to arsenic exposure and there are other causes of cancer. The incidence and mortality of these cancer types in Palestine are similar to what would be expected based on state rates [2].

Vanadium

Vanadium is a naturally occurring element in the earth that may be released to air, groundwater, surface water, or soil when rocks or soil containing vanadium are broken down by wind or water erosion [7]. People are exposed to vanadium daily in food, water, and air, with food being the largest source of vanadium. Determining the public health implications of vanadium in soil is difficult. The toxic effects of vanadium are greater when vanadium is inhaled as compared to when it is taken orally [7]. Protein and other trace elements in the diet may have an affect on vanadium toxicity and the toxic effects also may vary by species. Humans who have been exposed to relatively large doses for up to five months only reported minor complaints at the higher doses; whereas, in animals numerous effects such as weight loss, dehydration, depressed growth, cardiac irregularities, and loss of renal function have been reported [7]. Whether vanadium is essential to the diet is controversial. There is *in vivo* evidence that vanadium may be

needed for normal iodine and/or thyroid function and other evidence that it may have some effect on glucose metabolism. Although a variety of inconsistent deficiency symptoms have been reported in animals, no specific function for vanadium has been identified for humans [7].

An intermediate oral MRL of 0.003 mg/kg/day for vanadium was derived by dividing the NOAEL of 0.3 mg/kg/day (based upon renal effects in rats) by uncertainty factors of 10 for human variability and 10 for interspecies variability [7]. The intermediate oral MRL was used to evaluate the potential public health significance of exposure to vanadium.

Although no acute oral MRL has been derived for vanadium, an acute LOAEL of 8.4 mg/kg/day based upon developmental effects in rats is available [7]. As no other advisories are available, this acute LOAEL was used to evaluate the potential public health significance of acute exposure to vanadium due to pica behavior.

No human studies are available regarding the carcinogenicity of vanadium [7]. However, no increase in tumor frequency was noted in rats and mice chronically exposed to 0.5 to 4.1 mg-vanadium/kg-body weight as vanadyl sulfate in drinking water [8]. Currently, vanadium is not classified as a human carcinogen.

Non-Carcinogenic Effects

We used the proposed actions level of 155 mg/kg and 975 mg/kg for arsenic and vanadium, respectively, to evaluate the exposure that children might receive from incidental ingestion of surface soil. Children were considered because they constitute the sub-population most likely to experience the highest levels of exposure to contaminants in soil (due to play activities and normal hand-to-mouth exposure) and because their dose relative to body weight is higher than that of adults.

We applied the bioavailability factors reported by CVMUM to the proposed action levels and used standard assumptions for body weight (16 kg, child) and incidental soil ingestion (200 mg per day, child) to estimate exposure. Other assumptions that were used are listed in Tables 1-8. For non-cancer endpoints, wherever possible, we compared estimated exposures either to known effect levels in humans or to documented NOAEL and/or LOAEL in humans or animals.

In addition to incidental ingestion of small quantities of soil, a sizable portion of the child population periodically may ingest more than 1.0 g[4] of soil per day (pica behavior) [9]. While an individual child may exhibit pica behavior infrequently it has been estimated that about 62% of children will ingest >1.0 g of soil on 1-2 days/year. Additionally, 42% of children will ingest >5 g of soil and 33% will ingest >10 g of soil on 1-2 days per year. Since ingesting large amounts of soil potentially could result in acute intoxication we also evaluated the proposed action levels with respect to periodic pica behavior [9].

[4] 1 g is equal to 1,000 mg (about the same size as a pack of artificial sweetener)

Assuming a maximum soil arsenic concentration of 155 mg/kg, the estimated daily dose that a child would receive from the ingestion of soil (0.0003 mg/kg/day, Tables 1-2) is equal to the chronic oral MRL of 0.0003 mg/kg/day. Therefore, with respect to non-carcinogenic health effects, the proposed action level of 155 mg/kg for arsenic in soil is adequate to protect children.

Assuming a maximum soil vanadium concentration of 975 mg/kg, the estimated daily dose that a child would receive from the ingestion of soil (0.0009 mg/kg/day, Tables 3-4) is below the intermediate oral MRL of 0.003 mg/kg/day. Therefore, with respect to non-carcinogenic health effects, the proposed action level of 975 mg/kg for vanadium in soil is adequate to protect children.

Short-Term or Sporadic Pica Behavior

To explore the potential public health significance of pica behavior at this site, we considered the scenario of a 16 kg child ingesting 5,000 mg of soil per day for 14 days. At a soil arsenic concentration of 155 mg/kg and a bioavailability factor of 15%, the daily dose of absorbed arsenic during pica events would be approximately 0.007 mg/kg/day (Table 5). This estimated exposure dose is slightly higher than the acute oral MRL for arsenic which would indicate some degree of risk; however, as it is seven times lower that the dose reported to cause gastrointestinal effects in humans the likelihood that it would result in adverse health effects is low. At a soil vanadium concentration of 975 mg/kg and a bioavailability factor of 8%, the daily dose of absorbed vanadium during pica events would be approximately 0.02 mg/kg/day (Table 6), 420 times below the acute LOAEL for less serious effects in animals. We would not expect to see children exhibiting signs or symptoms of acute toxicity either from arsenic or vanadium as a result of short-term, sporadic pica behavior.

Carcinogenic Effects

Arsenic is a known carcinogen, and oral cancer slope factor and inhalation unit risk factors have been developed by EPA to estimate the excess lifetime risk for developing cancer. Vanadium has not been classified as a human carcinogen, and thus no slope factors or risk factors have been developed for vanadium.

Using EPA's cancer slope factor for arsenic, based on an 30 year exposure scenario (a 70 kg adult ingesting 100 mg of soil per day) we estimated the excess lifetime risk of developing cancer from the incidental ingestion of soil to be 2.0×10^{-5} (Tables 7-8). Qualitatively, we would interpret this as no apparent increased lifetime risk for developing cancer.

There is evidence that suggests some carcinogens, especially ones with a mutagenic mode of action, exhibit a greater effect in early-life versus later-life exposures [10]. The EPA has developed guidance on assessing the potential for early-life exposure to contribute to the appearance of cancer later in life. In this, cancer risks are calculated by combining age-dependent adjustment factors and exposure estimates [10]. The lifetime risk of developing cancer is based upon the risk during three time periods: risk during the first two years of life (10-fold adjustment representing to toxicokinetic and toxicodynamic differences between

children and adults), risk for ages 2 through <16 (3-fold adjustment to account for developmental changes occurring prior to middle adolescence), and risk for ages 16 until 70 years (average life expectancy, no adjustment factor for adult exposures) [10].

The risk for cancer due to exposure to arsenic over a lifetime was calculated using EPA methodology [10]. Body weight (10 kg infant, 40 kg child/middle adolescent, and 70 kg adult), soil intake rate (200 mg/day infant, 150 mg/day child/middle adolescent, and 100 mg/day adult), and age-dependent adjustment factors were used to calculate cancer risk over a lifetime of exposure (4.8×10^{-5}). Qualitatively, we would interpret this as no apparent increased lifetime risk for developing cancer.

Public Health Implications

Based on the above assumptions we would not expect the proposed action levels to pose an appreciable risk either to children or adults. However, in this consultation, we only evaluated the potential health effects associated with ingestion of soil. In some communities, backyard vegetable gardens are common and may provide an additional source of exposure, particularly during the growing season. In such situations it would be prudent to also consider the ingestion of home-grown vegetables and the incidental inhalation of dust while people are working in their gardens when proposing an action level. Thus, if there is reason to believe that vegetable gardening is occurring in Palestine, the proposed action level for arsenic may need to be revised. Using default exposure assumptions for inhalation of dust and ingestion of home-grown vegetables (Tables 1-8) we estimate that a soil arsenic concentration of 131 mg/kg would be a suitable action level. For vanadium the proposed action level of 975 mg/kg would still be adequate even when considering other sources of exposure. The estimated daily dose of vanadium from all sources is 0.001 mg/kg/day, well below the intermediate oral MRL for vanadium. The absorption of either arsenic or vanadium through the skin via dermal contact with soil was too low to consider[5].

Based on data presented in previous health consultations [2, 3], the maximum arsenic and vanadium concentrations detected in surface soils (0-1 inch) in Palestine (85.4 mg/kg and 325 mg/kg, respectively) were well below proposed action levels.

Child Health Considerations

In communities faced with air, water, or food contamination, children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. Sufficient exposure levels during critical growth stages can sustain permanent damage to the developing body systems of children. Children are dependent on adults for access to housing,

[5] Cadavers were used to assess the uptake of arsenic from soil via dermal absorption. Following 24 hours of exposure to soil containing arsenic, only 0.43% of the arsenic passed through the skin and 0.33% of the arsenic remained in the skin after washing [6].

for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

In this consult, we evaluated the exposure children might receive from accidental ingestion of soil to evaluate whether the proposed residential surface soil action levels for arsenic and vanadium would be protective of public health. We used children because they constitute the sub-population most likely to experience the highest levels of arsenic and vanadium in soil and because their dose of arsenic and vanadium relative to body weight is higher than that of adults.

Conclusions

Based on available information we have concluded that:

1. Considering exposure to arsenic and vanadium via accidental ingestion of surface soil, the proposed soil action levels would pose no apparent public health hazard.

2. The soil action level of 131 mg/kg may be more appropriate for yards where vegetable gardening occurs; however, based on available data all the levels measured in Palestine were well below this level.

Recommendations

Not applicable

Public Health Action Plan

Not applicable

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14. US Environmental Protection Agency. Exposure Factors Handbook. August 1997.
15. Justification for the Approach Used to Calculate the Above and Belowground Vegetable Ingestion Rate for TRRP.

Certification

This public health consultation was prepared by the Texas Department of State Health Services (DSHS) under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the public health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

Technical Project Officer, SPS, SSAB, DHAC, ATSDR

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

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Appendix A: Acronyms and Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
CSF	Cancer Slope Factor
CVMUM	College of Veterinary Medicine at the University of Missouri, Columbia
DSHS	Texas Department of State Health Services
EFH	Exposure Factors Handbook
EPA	U.S. Environmental Protection Agency
EPA-6	U.S. Environmental Protection Agency Region 6
g	gram
HAC	health-based assessment comparison
kg	kilogram
kg/m^3	kilogram per cubic meter
LOAEL	Lowest Observable Adverse Effect Level
m^3/day	cubic meter per day
mg	milligram
mg/day	milligram per day
mg/kg	milligram per kilogram
mg/kg/day	milligram per kilogram per day
MRL	Minimal Risk Level
NOAEL	No Observable Adverse Effect Level
PHAGM	Public Health Assessment Guidance Manual
RAGS	Risk Assessment Guidance for Superfund
RfD	Reference Dose
TAC	Texas Administrative Code
TRRP	Texas Risk Reduction Program

Appendix B: Tables

Table 1. Summary of non-carcinogenic estimated daily doses for children exposed to arsenic via various pathways. Refer to Table 2 for calculations of estimated exposure doses.

Exposure Pathway	Estimated Daily Dose (mg/kg/day)
Soil Ingestion Exposure Dose	**0.0003**
Inhalation Exposure Dose	0.00000002
Aboveground Vegetables Exposure Dose	0.00006
Belowground Vegetables Exposure Dose	0.00002
Total Daily Dose from all Pathways	**0.0004**

Table 2. Non-carcinogenic estimated daily doses for children exposed to arsenic via various pathways.

Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.95890411
F=frequency of exposure (day/year)	350 [a]
ED=exposure duration (year)	6 [b]
AT=averaging time (ED*365 day/year)	2190
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.0003
C=contaminant concentration (mg/kg)	155 [c]
RBAF=relative bioavailability factor (unitless)	15% [d]
IR=intake rate of soil (mg/day)	200 [b]
EF=exposure factor (unitless)	0.95890411
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]
Inhalation Exposure Dose	**D=(C*SPL*IR*EF)/BW**
D=exposure dose (mg/kg/day)	0.00000002
C=contaminant concentration (mg/kg)	155 [c]
SPL=suspended particulate level (kg/m^3)	2.16E-10 [e]
IR=intake rate (m^3/day)	8.08 [f]
EF=exposure factor (unitless)	0.95890411
BW=body weight (kg)	16 [b]
Aboveground Vegetables	**D=(C*CF*PC*CF*IR*EF*PH)/BW**
D=exposure dose (mg/kg/day)	0.00006
C=contaminant concentration (mg/kg)	155 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.01 [g]
CF=conversion factor (g dry weight/g as consumed)	17.4% [h]
IR=intake rate (g as consumed/day)	41.87
EF=exposure factor (unitless)	0.95890411
PH=% of food homegrown (%)	8.2% [i]
BW=body weight (kg)	16 [b]
Belowground Vegetables	**D=(C*CF*PC*CF*IR*EF*PH)/BW**
D=exposure dose (mg/kg/day)	0.00002
C=contaminant concentration (mg/kg)	155 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.008 [g]
CF=conversion factor (g dry weight/g as consumed)	22.2% [h]
IR=intake rate (g as consumed/day)	29.36
EF=exposure factor (unitless)	0.95890411
PH=% of food homegrown (%)	4.3% [i]
BW=body weight (kg)	16 [b]

[a] TRRP standard default, 30 TAC 350.74(a) [11]
[b] PHAGM standard default, child 1-6 years old [12]
[c] proposed action level [5]
[d] site-specific study [4]
[e] 1/Particulate Emission Factor, EPA RAGS [13]
[f] EFH Table 5-23, weighted average, child 1-6 years old [14]
[g] TRRP standard default, 30 TAC 350.73(e) [10]
[h] [15]
[i] EFH Table 13-71, total population [14]

Table 3. Summary of non-carcinogenic estimated daily doses for children exposed to vanadium via various pathways. Refer to Table 4 for calculations of estimated exposure doses.

Exposure Pathway	Estimated Daily Dose (mg/kg/day)
Soil Ingestion Exposure Dose	**0.0009**
Inhalation Exposure Dose	0.0000001
Aboveground Vegetables Exposure Dose	0.0001
Belowground Vegetables Exposure Dose	0.00005
Total Daily Dose from all Pathways	**0.001**

Table 4. Non-carcinogenic estimated daily doses for children exposed to vanadium via various pathways.

Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.95890411
F=frequency of exposure (day/year)	350 [a]
ED=exposure duration (year)	6 [b]
AT=averaging time (ED*365 day/year)	2190
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.0009
C=contaminant concentration (mg/kg)	975 [c]
RBAF=relative bioavailability factor (unitless)	8% [d]
IR=intake rate of soil (mg/day)	200 [b]
EF=exposure factor (unitless)	0.95890411
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]
Inhalation Exposure Dose	**D=(C*SPL*IR*EF)/BW**
D=exposure dose (mg/kg/day)	0.0000001
C=contaminant concentration (mg/kg)	975 [c]
SPL=suspended particulate level (kg/m^3)	2.16E-10 [e]
IR=intake rate (m^3/day)	8.08 [f]
EF=exposure factor (unitless)	0.95890411
BW=body weight (kg)	16 [b]
Aboveground Vegetables	**D=(C*CF*PC*CF*IR*EF*PH)/BW**
D=exposure dose (mg/kg/day)	0.0001
C=contaminant concentration (mg/kg)	975 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.0036 [g]
CF=conversion factor (g as consumed/g dry weight)	17.4% [h]
IR=intake rate (g as consumed/day)	41.87
EF=exposure factor (unitless)	0.95890411
PH=% of food homegrown (%)	8.2% [i]
BW=body weight (kg)	16 [b]
Belowground Vegetables	**D=(C*CF*PC*CF*IR*EF*PH)/BW**
D=exposure dose (mg/kg/day)	0.00005
C=contaminant concentration (mg/kg)	975 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.003 [g]
CF=conversion factor (g dry weight/g as consumed)	22.2% [h]
IR=intake rate (g as consumed/day)	29.36
EF=exposure factor (unitless)	0.95890411
PH=% of food homegrown (%)	4.3% [i]
BW=body weight (kg)	16 [b]

[a] TRRP standard default, 30 TAC 350.74(a) [11]
[b] PHAGM standard default, child 1-6 years old [12]
[c] proposed action level [5]
[d] site-specific study [4]
[e] 1/Particulate Emission Factor, EPA RAGS [13]
[f] EFH Table 5-23, weighted average, child 1-6 years old [14]
[g] TRRP standard default, 30 TAC 350.73(e) [11]
[h] [15]
[i] EFH Table 13-71, total population [14]

Table 5. Non-carcinogenic estimated daily doses of arsenic resulting from short-term or sporadic pica behavior in children.

2 days out of 365 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.005479452
F=frequency of exposure (day/year)	2
ED=exposure duration (year)	1
AT=averaging time (ED*365 day/year)	365
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.00004
C=contaminant concentration (mg/kg)	155 [c]
RBAF=relative bioavailability factor (unitless)	15% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	0.005479452
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

2 days out of 14 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.142857143
F=frequency of exposure (day/year)	2
ED=exposure duration (year)	1
AT=averaging time (day)	14
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.001
C=contaminant concentration (mg/kg)	155 [c]
RBAF=relative bioavailability factor (unitless)	15% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	0.142857143
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

14 days out of 14 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	1
F=frequency of exposure (day/year)	14
ED=exposure duration (year)	1
AT=averaging time (day)	14
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.007
C=contaminant concentration (mg/kg)	155 [c]
RBAF=relative bioavailability factor (unitless)	15% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	1
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

[b] PHAGM standard default, child 1-6 years old [12]
[c] proposed action level [5]
[d] site-specific study [4]
[j] PHAGM standard default, pica child, acute exposure situations [12]

Table 6. Non-carcinogenic estimated daily doses of vanadium resulting from short-term or sporadic pica behavior in children.

2 days out of 365 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.005479452
F=frequency of exposure (day/year)	2
ED=exposure duration (year)	1
AT=averaging time (ED*365 day/year)	365
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.0001
C=contaminant concentration (mg/kg)	975 [c]
RBAF=relative bioavailability factor (unitless)	8% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	0.005479452
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

2 days out of 14 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.142857143
F=frequency of exposure (day/year)	2
ED=exposure duration (year)	1
AT=averaging time (day)	14
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.003
C=contaminant concentration (mg/kg)	975 [c]
RBAF=relative bioavailability factor (unitless)	8% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	0.142857143
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

14 days out of 14 days	
Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	1
F=frequency of exposure (day/year)	14
ED=exposure duration (year)	1
AT=averaging time (day)	14
Soil Ingestion Exposure Dose	**D=(C*RBAF*IR*EF*CF)/BW**
D=exposure dose (mg/kg/day)	0.02
C=contaminant concentration (mg/kg)	975 [c]
RBAF=relative bioavailability factor (unitless)	8% [d]
IR=intake rate of soil (mg/day)	5000 [j]
EF=exposure factor (unitless)	1
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	16 [b]

[b] PHAGM standard default, child 1-6 years old [12]
[c] proposed action level [5]
[d] site-specific study [4]
[j] PHAGM standard default, pica child, acute exposure situations [12]

Table 7. Summary of estimated lifetime excess cancer risk from exposure to arsenic via various pathways. Refer to Table 8 for calculations of cancer risk.

Exposure Pathway	Estimated Cancer Risk
Soil Ingestion Cancer Risk	**2.0E-05**
Inhalation Cancer Risk	5.9E-08
Aboveground Vegetables Cancer Risk	2.1E-05
Belowground Vegetables Cancer Risk	7.8E-06
Total Cancer Risk from all pathways	**5.0E-05**

Table 8. Estimated lifetime excess cancer risk from exposure to arsenic via various pathways.

Exposure Factor	**EF=(F*ED)/AT**
EF=exposure factor (unitless)	0.410958904
F=frequency of exposure (day/year)	350 [k]
ED=exposure duration (year)	30 [k]
AT=averaging time (ED*365 day/year)	25550
Soil Ingestion Cancer Risk	**CR=((C*RBAF*IR*EF*CF)/BW)*CSF**
CR=excess lifetime cancer risk (unitless)	2.0E-05
C=contaminant concentration (mg/kg)	155 [c]
RBAF=relative bioavailability factor (unitless)	15% [d]
IR=intake rate of soil (mg/day)	100 [l]
EF=exposure factor (unitless)	0.410958904
CF=conversion factor (10^{-6} kg soil/mg soil)	0.000001
BW=body weight (kg)	70 [l]
CSF=cancer slope factor $(mg/kg/day)^{-1}$	1.5
Inhalation Cancer Risk	**CR=(C*SPL*IUR*CF*EF)**
CR=excess lifetime cancer risk (unitless)	5.9E-08
C=contaminant concentration (mg/kg)	155 [c]
SPL=suspended particulate level (kg/m^3)	2.16E-10 [e]
IUR=inhalation unit risk $(ug/m3)^{-1}$	0.0043
CF=conversion factor (10^3 ug arsenic/mg arsenic)	1000
EF=exposure factor (unitless)	0.410958904
Aboveground Vegetables	**CR=((C*CF*PC*CF*IR*EF*PH)/BW)*CSF**
CR=excess lifetime cancer risk (unitless)	2.1E-05
C=contaminant concentration (mg/kg)	155 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.01 [g]
CF=conversion factor (g dry weight/g as consumed)	17.4% [h]
IR=intake rate (g as consumed/day)	109.32
EF=exposure factor (unitless)	0.410958904
PH=% of food homegrown (%)	8.2% [i]
BW=body weight (kg)	70 [l]
CSF=cancer slope factor $(mg/kg/day)^{-1}$	1.5
Belowground Vegetables	**CR=((C*CF*PC*CF*IR*EF*PH)/BW)*CSF**
CR=excess lifetime cancer risk (unitless)	7.8E-06
C=contaminant concentration (mg/kg)	155 [c]
CF=conversion factor (10^{-3} kg soil/g soil)	0.001
PC=partition coefficient soil to vegetable (g soil/g dry weight)	0.008 [g]
CF=conversion factor (g dry weight/g as consumed)	22.2% [h]
IR=intake rate (g as consumed/day)	74.7
EF=exposure factor (unitless)	0.410958904
PH=% of food homegrown (%)	4.3% [i]
BW=body weight (kg)	70 [l]
CSF=cancer slope factor $(mg/kg/day)^{-1}$	1.5

[c] proposed action level [5]
[d] site-specific study [4]
[e] 1/Particulate Emission Factor, EPA RAGS [13]
[g] TRRP standard default, 30 TAC 350.73(e) [11]
[h] [15]
[i] EFH Table 13-71, total population [14]
[k] PHAGM standard default, cancer risk [12]
[l] PHAGM standard default, adult [12]