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BEFORE THE BOARD OF ENVIRONMENTAL QUALITY
STATE OF IDAHO

IN THE MATTER OF AIR QUALITY PERMIT TO CONSTRUCT P-2019.0047

NEZ PERCE TRIBE, IDAHO CONSERVATION LEAGUE, and SAVE THE SOUTH FORK SALMON,

Petitioner,

v.

IDAHO DEPARTMENT OF ENVIRONMENTAL QUALITY,

Respondent,

and

PERPETUA RESOURCES IDAHO, INC.

Intervenor-Respondent.

Case Docket No. 0101-22-01
OAH Case No. 23-245-01

**EXPERT DECLARATION OF THERESA LOPEZ**

I, Theresa Lopez, hereby declare and affirm as follows:

1. My name is Theresa Lopez. I am employed by Tetra Tech, Inc. as a Principal Toxicologist.

2. In addition to this Declaration, I have prepared a supporting memorandum that further elaborates on my testimony (Memorandum). A copy is attached.

**A. Qualifications and Experience**

3. My curriculum vitae is attached as Attachment C to the Memorandum.

4. I received a Master of Science degree in Public Health from the University of Colorado in 1991 and a Bachelor of Science degree in Nutritional Science with a minor in Zoology from the University of Florida in 1989. My relevant areas of expertise include chemical risk assessment, human health risk assessment, regulatory toxicology, and exposure assessments.

5. I worked extensively in the areas of public health, epidemiology, human health risk assessment, toxicology, and occupational health and safety, representing both the public and private sector for over 32 years with Tetra Tech.

6. My work included a variety of human health risk assessment projects at the federal and state levels, as well as international work. I led the investigation and assessment of chemical releases and environmentally compromised sites such as former nuclear weapons plants, active and closing naval bases, Superfund Sites, and mining, commercial, and school sites.

7. My work includes assisting in risk assessments and remediation of former mining sites with heavy metal, pesticides, petroleum compounds and other chemicals; and risk assessments for proposed mines, hazardous waste release sites, and petroleum remediation in the United States and Canada. I prepared human exposure assessments for schools and recreational settings and assessed clean-up goals and remediation strategies for closed industrial, mining,

redevelopment, and other sites. These sites often include the consideration of native and indigenous peoples. I provided exposure and dose reconstruction for Oak Ridge Associated Universities to quantify radiation doses to former nuclear weapons workers.

8. Recent relevant work includes review of human exposures, livestock exposures, plant, and food ingestion pathways for the development of water quality standards for the Navajo Nation; technical support for the Agency for Toxic Substances and Disease Registry (ATSDR) to provide scientific advise and education on public health assessment methodologies, review of toxicity data, and probabilistic risk assessment methodology, and primary experiments, for example to assess uptake measurements of metals and PFAS from soil to common food plants; and peer review of chemical assessments for the Consumer Product Safety Commission. I author and review reports for Health Canada related to Existing Substances Risk Assessment Bureau mandates. For these projects I am often designated as lead toxicologist.

**B. Scope of Review**

9. I have been retained as an expert witness by Perpetua Resources Idaho, Inc. (Perpetua) in the above-captioned case, to address issues raised in the Final Order issued by the Board of Environmental Quality on May 9, 2024 and to evaluate the risks presented by emissions of arsenic from the Stibnite Gold Project (SGP). Specifically, I have been asked to evaluate:

a. Whether the Idaho Department of Environmental Quality (DEQ) used appropriate risk assessment methodology by adjusting the maximum modeled lifetime arsenic emissions from the SGP by 16/70 to reflect the operational life of the SGP;

b. Whether, as conditioned by the Air Quality Permit to Construct (PTC) issued to Perpetua, arsenic emissions from the SGP present a greater than 1 in 100,000 cancer risk; and

c. Whether DEQ's use of a five-year rolling average production limit to demonstrate compliance with T-RACT is protective of public health and the environment.

**C. Summary of Opinions**

10. Based on my review of the permitting record in this matter, the Declarations of Norka Paden, Kevin Schilling, and Kevin Lewis, and the documents cited in, and attached to, my Memorandum, I conclude as follows:

a. I conclude that the 16/70 calculation was consistent with good scientific methodology for assessing cancer risk and consistent with the applicable rules. DEQ appropriately calculated, and considered, the exposure duration of 16 years of SGP operational life when evaluating the risk posed by arsenic emissions from the SGP. Cancer risk cannot be evaluated without taking into consideration the length of time a potential receptor will be exposed to carcinogen.

b. The Rules for the Control of Air Pollution in Idaho (Air Rules), IDAPA 58.01.01, provide a default formula for calculating cancer risk from carcinogen emissions from stationary sources. The Air Rules, specifically Section 586, adopted EPA risk assessment information and methodology, which inherently requires consideration of exposure duration.

c. The use of a 10-times adjustment to the Acceptable Ambient Concentration for Carcinogens (AACC) when a project will operate for five years or less is an example of an adjustment of exposure duration when evaluating cancer risk and does not preclude adjustment for exposure duration when a project will operate for less than 70 years.

d. I conclude that the incremental increase in cancer risk caused by arsenic emissions from the SGP is 1 in 240,000, which is less than the acceptable risk of 1

in 100,000. This conclusion is based on the maximum modeled annual concentration of arsenic predicted for SGP operations, the operational life of the SGP established by conditions in the PTC, and risk assessment methodology established by EPA.

e. I conclude that the use of a five-year rolling average production limit to demonstrate compliance with T-RACT is sufficiently protective to ensure that the ambient concentration of arsenic from the SGP will not cause a cancer risk above 1 in 100,000.

f. The PTC includes both daily production limits and limits production from the SGP to 135,000 tons per year based on a five-year rolling average. EPA's risk assessment methodology evaluates the incremental increase in cancer risk for each incremental increase in carcinogen dose over the exposure duration. Use of a five-year rolling average ensures that a receptor's total exposure to arsenic caused by the SGP will not exceed a 1 in 100,000 cancer risk. Although there may be short term increases in production, and the ambient concentration of arsenic, these short-term increases will not, alone, cause unacceptable cancer risk.

## D. Background

11. Arsenic comes in several forms and occurs naturally in soil and minerals. Arsenic can enter the atmosphere from wind-blown dust. Arsenic concentrations in soil are variable across the country, and when soil is disturbed arsenic can become airborne and be carried with dust particles. Since arsenic is naturally in the environment, everyone is exposed to some arsenic by eating food, drinking water, or breathing air. People normally ingest small amounts of arsenic from breathing air, drinking water, and eating food. Indeed, food is considered the largest source of arsenic exposure for humans.

12. EPA analyzes the long-term, or carcinogenic, health effects of arsenic through animal and worker studies. The arsenic inhalation unit risk (IUR) published by EPA in the Integration Risk Information System (IRIS) reflects many years of research and evaluation and was developed using a linear extrapolation method that assumes that any level of exposure carries a risk. Under a linear risk extrapolation method, the dose is cumulative over the duration of exposure and cancer risk increases linearly as the cumulative total dose increases. Exposure is assumed to occur over a lifetime, assumed to be 70 years. See Memorandum, Figure 1.
13. All IURs (including for arsenic) are based on an incremental lifetime risk of 1 in one million from exposure to 1 $\mu g/m^3$ of a substance continuously (24 hours day and 365 days/ year) over a lifetime of exposure (assumed to be 70 years). Importantly, this is an incremental risk that does not consider other, unrelated risks present over a lifetime.
14. DEQ adopted EPA's cancer risk values and risk assessment methodology when it promulgated the Air Rules for toxic air pollutants from stationary sources. IDAPA 58.01.01.586; Memorandum at 5-7; Memorandum, Attachment A, Idaho DEQ 1993 at 33.
15. The unit risk factor (URF) of 4.3E-03$\mu g/m^3$ for arsenic that DEQ adopted in IDAPA 58.01.01.586 is the IUR developed by EPA and reflected in the IRIS.
16. Similarly, the AACC that DEQ adopted in IDAPA 58.01.01.586 is taken from the IRIS database and is referred to by EPA as the "Air Concentration at Specific Risk Levels." The AACC is calculated by dividing the acceptable lifetime risk (1 in 1,000,000) by the URF. See Memorandum, Figure 1.

17. The AACC in Section 586 of the Air Rules reflects the ambient concentration of arsenic to which a person could be exposed 24 hours a day, 365 days a year, for a 70-year lifetime before the cancer risk level exceeds 1 in 1,000,000 (AACC = 0.00023µg/$m^3$).

18. The AACC does not represent an annual emission limit; it is the concentration that a person may be exposed to for a lifetime continuously, with an incremental risk of cancer of 1 in 1,000,000. *See* Idaho DEQ 1993 at 32.

19. Both the URF and AACC are derived using a linear risk extrapolation method, meaning that cancer risk from arsenic exposure increases linearly as the dose or exposure duration increases. See Memorandum at 4. Therefore, although the URF and AACC are based on lifetime exposure, the ultimate determinate of cancer risk is the cumulative dose, regardless of the timeframe over which that dose is delivered.

## E. Opinions

***DEQ Appropriately Adjusted the Exposure Duration of SGP to 16 Years Consistent with Permit Conditions Limiting the Project Life.***

20. When considering carcinogenic exposure, a critical factor to determine the cancer risk is the exposure duration. Although the AACC assumes a 70-year constant exposure, where the exposure duration is less than 70 years, the exposure duration must be adjusted for an accurate risk calculation. In the case of the SGP, the maximum exposure duration is 16 years. Therefore, the cancer risk from the ambient concentration of arsenic caused by the SGP cannot be compared to the AACC (that assumes 70 years) without adjusting for exposure duration.

21. Comparing unadjusted emissions to the AACC would result in an over-estimation of cancer risk because the dose will not continue for the 70-year lifetime exposure assumed

in the development of the AACC. For an "apples to apples" comparison, evaluation of SGP emissions must be adjusted for the shorter exposure duration.

22. EPA provides the following formula to compare the exposure concentration from a project that operates for less than 70 years to the lifetime exposure concentration that would cause a specified cancer risk (shaded notes are presented by author for clarification):

**Cair-adj=Cair x ET x 1day/24 hours x EF x ED/AT**

Where:
**$C_{air-adj}$** = Air Concentration adjusted for exposure
**$C_{air}$** = Concentration of contaminant in air (mg/m$^3$) (this is the modeled or measured annual air concentration)
**ET** = Exposure time (hours/day) (this is assumed to be 24 hours/day)
**EF** = Exposure frequency (days/year) (this is assumed to be 365 days/year)
**ED** = Exposure duration (years) (this is 16 years – the life of operations)
**AT** = Averaging time (days) (this is lifetime – 70 years x 365 days/year; it never changes for any carcinogen)

See Memorandum at 9-10.

23. DEQ used this formula when it determined the acceptable risk from the ambient arsenic concentration caused by SGP operations.

24. Using the highest modelled annual average arsenic concentration from the project of 0.00414 µg/m$^3$ (REC 716) the calculation of the air concentration adjusted for exposure from the SGP is as follows:

$$\left[0.00414\,\frac{\mu g}{m3}\,x\,24\,\frac{hours}{day}\,x\,\frac{1\,day}{24\,hours}\,x\,365\,\frac{days}{year}\,x\,16\,years\right] / \left[365\,\frac{days}{year}\,x\,70\,years\right] = 0.00095\,\frac{\mu g}{m3}$$

25. Using EPA methodology, as adopted by DEQ, the adjusted lifetime-exposure concentration from the SGP is 0.00095µg/m$^3$. This is the correct project-specific arsenic

concentration to compare to the T-RACT AACC of 0.0023 µg/ $m^3$ for a cancer risk of 1:100,000. *See* REC 716.

***The Cancer Risk Caused by Arsenic Emissions from the SGP is 1-in-240,000 -- Less than the Acceptable Risk of 1-in-100,000.***

26. The highest air concentration of arsenic from the project is below the acceptable lifetime risk established by the T-RACT AACC.

27. Once the ambient arsenic concentration has been adjusted from a lifetime exposure to a project specific duration, to provide an "apples-to-apples" comparison to the AACC, then that ambient concentration is used to determine the cancer risk from arsenic from SGP.

28. Calculation of the incremental increase in cancer risk from SGP arsenic emissions is as follows:

$$\frac{1}{100{,}000} T\text{–}RACT\ Risk\ x\ 0.00095 \frac{\mu g}{m3} C_{air-adj} \ /\ 0.0023 \frac{\mu g}{m3}\ T\text{–}RACT\ AACC$$
$$= 4.1E\text{–}6\ or\ \frac{1}{240{,}000} Actual\ Risk$$

29. The incremental increase in cancer risk from SGP arsenic emissions is 1 in 240,000, meaning that if 240,000 people are exposed to the maximum ambient arsenic air concentration from the SGP continuously over 16 years, then one excess cancer would probabilistically be expected to develop over a lifetime. This is less than 1 excess cancer in 100,000 people, which is the acceptable risk limit in the T-RACT AACC for SGP.

30. DEQ utilized the formulas described in paragraphs 24 and 28 to compare the risk of exposure to arsenic emissions from the SGP. This analysis is consistent with EPA methodology and provides an "apples to apples" comparison of risk between SGP emissions of 16 years and the AACC concentration that assumes exposure over 70 years.

31. DEQ's assessment ensured that the PTC, and the conditions imposed on SGP, would ensure that the cancer risk from SGP emissions would be less than 1-in-100,000.

32. Section 210.15 of the Air Rules is an example of the proper adjustment for exposure duration and does not preclude this adjustment in other circumstances for an "apples to apples" comparison, where the project life is limited to less than 70 years.

33. In its analysis, DEQ analogized the SGP exposure duration adjustment to the short-term adjustment factor for sources that operate for 5 years or less in Section 210.15 of the Air Rules. The Final Order expressed concern that the Air Rules do not explicitly allow for an adjustment for sources that will operate for more than 5 years but less than 70 years.

34. Section 210.15 of the Air Rules is an example of an appropriate adjustment for exposure duration. This is consistent with EPA's methods for risk assessment and reliance on the URFs and AACCs. Sections 210.02 and 210.03 allow DEQ to develop analyses using project specific information, such as exposure duration. In my opinion these rules cannot be read to impede DEQ's use of EPA's methods and scientific practices to perform an appropriate risk assessment under the Air Rules.

35. Adjusting exposure duration is necessary and proper from a scientific and toxicological standpoint to apply the AACCs accurately. Omitting this adjustment is contrary to common scientific practice and the intent of the Air Rules as reflect DEQ documents. Memorandum Attachment A, References, Idaho DEQ 1992 and Idaho DEQ 1993.

36. This is particularly true when comparing project specific risk to the AACC because the AACC, and the URF upon which it is based, assumes a 70 year (lifetime) exposure.

37. The AACCs are not annual average air concentration limits or standards.

38. Treating the AACC as an annual emission limitation or annual limit on ambient exposure concentration is inconsistent with the purpose and development of the AACC. The

AACC is an ambient concentration that if emitted over time (default 70 years) could result in a risk of cancer response.

39. Treating the AACC as an annual emissions limitation significantly lowers acceptable risk. If the T-RACT AACC of 0.0023 µg/m$^3$ for arsenic were based on a one-year exposure, then the risk represented by that value would be about 1-in-7,000,000.

40. The URF and the AACC derived from it represent the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a contaminant over a lifetime at an air concentration of 1 µg/m$^3$.

41. In other words, the AACC and T-RACT AACC represent the chemical-specific airborne concentration at which the risk to an individual if exposed all day, every day for a lifetime is 1-in-1,000,000 (AACC) or 1-in-100,000 (T-RACT AACC). Any other interpretation defies fundamental toxicological science used by the EPA in the development of IURs (URFs).

42. The AACC and T-RACT AACC do not represent the risk of a cancer response following a single exposure.

***The Use of a Five-Year Rolling Average Production Limit to Demonstrate Compliance with T-RACT is Sufficiently Protective to Ensure that the Ambient Concentration of Arsenic from the SGP Will Not Cause a Cancer Risk Above 1-in-100,000.***

43. Finally, use of a 5-year rolling average production limit is as protective as an annual production limit, for purposes of determining an acceptable cancer risk.

44. Cancer risk increases from cumulative long-term exposure, not short-term annual emissions. Cancer risk is assessed based on total cumulative dose over the exposure duration, as demonstrated in the Memorandum, Figure 1, column D.

45. As shown in Figure 1, a total dose of 0.16 $\mu g/m^3$ of arsenic would cause an increased cancer risk of 1-in-100,000. The risk does not change if this total cumulative dose occurs in 1 year, 5 years, 16 years, or 70 years. Therefore, the 5-year rolling average is as protective as an annual production limit.
46. The 5-year average production limit ensures that if production is above the limit in one year, then future production must be reduced below the limit. This ensures that the cumulative ambient arsenic concentration does not exceed the acceptable ambient concentration level for the defined risk.
47. The 5-year average production limit in the PTC ensures that the ambient arsenic concentration from the SGP over the project life (exposure duration of 16 years) results in an acceptable cancer risk under the Air Rules.

I declare under penalty of perjury under the laws of the United States that the foregoing is true and correct to the best of my knowledge, information, and belief.

DATED: August 30, 2024.

Theresa K. Lopez

Theresa Lopez, TetraTech

## CERTIFICATE OF SERVICE

I hereby certify that on August 30, 2024, a true and correct copy of the **EXPERT DECLARATION OF THERESA LOPEZ** was served on the following:

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/s/ Wade C. Foster
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**Memorandum in Support of Declaration: DEQ's Review of Risk for the Stibnite Gold Project**

TETRA TECH, INC.

| | |
|---|---|
| Author: | Theresa Lopez MSPH<br>Tetra Tech, Inc. |
| Date: | August 30, 2024 |
| Subject: | Memorandum in Support of Declaration: DEQ's Review of Risk for the Stibnite Gold Project |

## I. INTRODUCTION

This memorandum and my opinions address certain of the issues remanded by the Idaho Board of Environmental Quality (Board) in *In re Matter of Air Quality Permit to Construct* P-2019.0047 regarding the Idaho Department of Environmental Quality's (DEQ) issuance on June 17, 2022 of a permit to construct (PTC) to Perpetua Resources Idaho, Inc. for the Stibnite Gold Project (SGP). This memorandum concludes that, as issued, the PTC for the SGP is protective of public health and establishes conditions that ensure a less than 1 in 100,000 cancer risk from worst-case exposure to arsenic, applying established toxicological principles. This memorandum confirms that the DEQ properly interpreted and applied the toxic air pollutant (TAPs) rules (including the ambient TAP carcinogenic increments and the acceptable risk represented by Sections 161, 210.12 and 586 of the Rules for the Control of Air Pollution in Idaho, IDAPA 58.01.01 (IDAPA or the Air Rules)) consistent with scientific methods and EPA guidelines.

## II. STATEMENT OF QUALIFICATIONS

Attached hereto as Attachment C is a copy of my curriculum vitae.

## III. DOCUMENTS REVIEWED

To form the opinions expressed in this memorandum, I reviewed the following documents:

- Board's Final Order
- PTC, issued June 17, 2022
- Statement of Basis (SOB) for the PTC issued June 17, 2022
- References listed in Attachment A
- Expert Declaration of Kevin Lewis (Filed Aug. 30, 2024)
- Expert Declaration of Kevin Schilling (Filed Aug. 13, 2024)
- Expert Declaration of Norka Paden (Filed Aug. 13, 2024)

## IV. OVERVIEW OF ISSUES REMANDED BY THE BOARD AND ADDRESSED BY THIS MEMORANDUM

In its *Final Order* dated May 9, 2024, the Board remanded this case to the Hearing Officer for the development of further evidence related to DEQ's risk assessment for the Acceptable Ambient Concentration for Carcinogens (AACC) for arsenic as set forth in Section 586 of the Air Rules. Specifically, the Board directed that on remand, the Hearing Officer address the following:

- DEQ's application of the project exposure duration of 16 years when assessing compliance with the AACC. Specifically, the Board indicated that "DEQ did not provide sufficient evidence in the form of an expert opinion from a toxicologist or other qualified expert regarding the cancer risk associated with the 16/70 adjustment."
- DEQ's use of a five-year rolling average for T-RACT that the Board concluded was not supported by sufficient evidence.
- The T-RACT analysis limiting the non-West End Pit production limit.

This memorandum supports my expert opinion on the first two issues.

- This memorandum explains the role of exposure duration, including the current state of scientific methods for assessing risk from inhalation exposure and U.S. Environmental Protection Agency (EPA) directives on the use of toxicity values and risk assessment.
- This memorandum addresses the use by DEQ of a project specific adjustment factor of 16/70 to account for the operational life of the SGP.
- This memorandum addresses the use of a short-term adjustment factor, provided for in IDAPA 210.15 of the Air Rules, allowing a 10-times adjustment to the AACC for short-term projects. This is an example of the use of exposure duration in risk assessment, and adjustment for exposure duration in other risk assessments is also appropriate.
- This memorandum clarifies that AACCs are not annual average air concentration limits or standards.
- This memorandum explains why the use of a 5-year rolling average production limit is equally protective as an annual production limit.

## V. TOXICOLOGY, THE TAPs RULES, AND ARSENIC

Toxicology is the study of the adverse effects (called "responses") of exposures to chemicals (as well as other compounds) on biological systems (called "doses"). In toxicology, dose-response is critical to characterizing risk of experiencing adverse effects. A dose-response assessment is a mathematical relationship between the extent of a biological response in an organism (if any) to a substance at varying levels of exposure (i.e., concentration) for varying periods of time (i.e., the duration of the exposure). The goal of a dose-response assessment is to define an exposure threshold above which a substance may cause adverse effects. In other words, the goal is to define the rate at which adverse effects may be experienced as the dose (concentration and exposure) increases.

EPA uses established dose-response assessments for various chemicals to characterize both non-cancer and cancer risks. For carcinogens, these assessments result in inhalation unit risk (IUR) values and are set forth in the EPA Integrated Risk Information System (IRIS). EPA uses the IUR to develop ambient concentrations that reflect cancer risk levels from continuous exposure over a 70-year lifetime. These concentrations can be found in the EPA IRIS.

Based on my review of the relevant documents, in my opinion, the foundation of the TAPs rules is consistent with the scientific principles underlying dose-response assessments that are established by EPA. IDAPA 586 reflects EPA risk assessment methodology that prompts adjustment of the exposure duration to assess the dose-response risk assessment properly. DEQ appropriately

considered exposure duration when applying the TAPs rules to the PTC, consistent with the EPA assessments on which they are based. In this PTC, DEQ applied the Air Rules consistent with the best available scientific practices in establishing cancer risk levels, and evaluating the environmental and health risks of exposure to arsenic from the SGP:

A. DEQ interpreted and applied the TAPs Rules consistent with EPA's risk assessment methodology including accepted scientific principles governing toxicity values, exposure evaluations, and cancer risk.
B. Based on EPA methods and DEQ policies and methodologies, DEQ appropriately adjusted the exposure duration to 16 years from the assumed 70 years reflecting conditions in the PTC that limit the project life to 16 years; this adjustment was necessary for a scientifically defensible "apples to apples" comparison with the AACC.
C. The highest air concentration projections modelled for arsenic emissions from SGP are below the acceptable lifetime risk established by the T-RACT AACC in IDAPA 586 and 210.12, and therefore, the PTC authorizes risks that are acceptable for human health and the environment.
D. IDAPA 210.15 allows for a 10-times adjustment to the AACC when a project will operate for 5 years or less. This is an example of applying exposure duration when evaluating cancer risk. The Air Rules do not limit DEQ's analyses that follow EPA methodology and scientific principles for exposure duration adjustment in risk assessment for permits issued under the Air Rules.
E. Idaho's AACCs are not annual average air concentration limits or standards.

As described in the *Final Order*, the primary TAP of concern from the SGP is Arsenic. Arsenic comes in several forms, occurs naturally in soil and minerals, and volcanic eruptions are a source of arsenic. Arsenic can enter the atmosphere from wind-blown dust. Arsenic concentrations in soil are variable, as some areas having higher arsenic levels than others. When soil is disturbed arsenic can become airborne and carried with dust particles. Since arsenic is naturally in the environment, everyone is exposed to some arsenic by eating food, drinking water, or breathing air. People normally ingest small amounts of arsenic from breathing air, drinking water, and eating food. "Of these, food is usually the largest source of arsenic. The predominant dietary source of arsenic is seafood, followed by rice/rice cereal, mushrooms, and poultry." (ATSDR 2007)

Impacts of arsenic exposure are also well-studied. Arsenic presents risks of both noncarcinogenic (acute) and carcinogenic (long-term) health effects. Long-term, or carcinogenic, health effects are evaluated through animal or worker studies. EPA states that risk assessment is a scientific process that depends on 3 factors: (2024 - About Risk Assessment | US EPA)

1. **How much of a stressor is present** in an environmental medium (e.g., soil, water, air) over what geographic area,
2. **How much contact (exposure)** a person has with that environmental medium, and
3. **How it affects** the health of humans (e.g., **toxicity)**

EPA published toxicity values for both carcinogenic and noncarcinogenic health effects of arsenic (EPA 1984). These toxicity values are the result of many years of research and evaluation. The

carcinogenic toxicity values for arsenic via inhalation were developed by EPA using a linear extrapolation method that assumes that any level of exposure carries a risk. EPA stated: "It is assumed, unless evidence exists to the contrary, that if a carcinogenic response occurs at the dose levels used in a study, then responses at all lower doses will occur with an incidence that can be determined by an appropriate extrapolation model." (EPA 1984). The linear model used by EPA incorporated 4 different studies and resulted in the calculation of an IUR of 4.3Ex $10^{-3}$ per µg/$m^3$ for arsenic. According to EPA, the mode provides a plausible estimate of the upper limit of risk; the true risk could be considerably lower. The figure below is one of several created by EPA; a combined figure for all studies from which the IUR was calculated is not available. (EPA 1984).

Figure 1

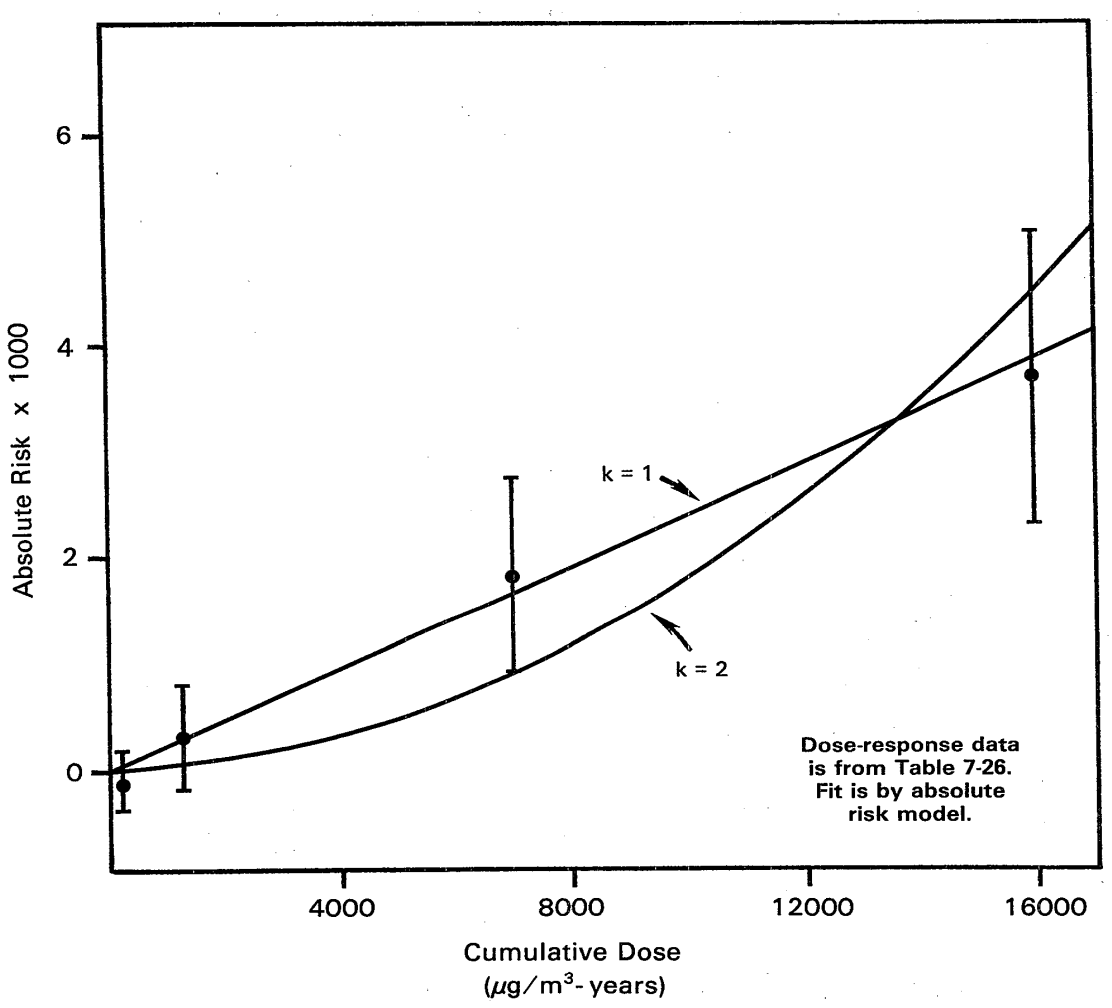


**Figure 7-5. Absolute risks and 90% confidence limits for data of Higgins (1982).**

The figure highlights EPA's scientific bases for risk assessment using the IUR for arsenic. The dose is cumulative. The exposure is assumed to occur over years. The IUR provides a measure of risk per unit exposure over a lifetime, assumed to be 70 years.

All IURs are based on an incremental lifetime risk of 1 in one million from exposure to 1 µg/m$^3$ of a substance continuously, every day over a lifetime of exposure (assumed to be 70 years). Importantly, this is an incremental risk considering unrelated risks present over a lifetime.[1]

## VI. SUPPORT FOR OPINIONS INCLUDED IN DECLARATION

### A. DEQ interpreted and applied the Air Rules to the SGP consistent with EPA's risk assessment methodology including accepted scientific principles governing toxicity values, exposure evaluations, and cancer risk.

DEQ adopted the EPA's values for assessment of risk in the TAPs rules. Correspondingly, DEQ adopted EPA's risk assessment methodology and underlying scientific principles governing those adopted values and risks. To interpret the Air Rules any other way would be to defy the science, toxicology, and application of these values established by EPA.

IDAPA 586 explicitly includes both a Unit Risk Factor (URF) and an Acceptable Ambient Concentration for Carcinogens (AACC) for each listed compound, including arsenic. Neither the URF (4.3E-03/µg/m$^3$) nor the AACC (2.3E-04 µg/m$^3$) for arsenic were developed by DEQ. EPA developed these values and published them in the IRIS (EPA 2024b), which is a database maintained by EPA (and only EPA). DEQ adopted these values into the TAPs Rules in 1993-1994. (Idaho DEQ 1992, Appendix 6; Idaho DEQ 1993)

DEQ adopted the URF directly from EPA's IUR, and the URF reflects the *cancer risk* based on continuous exposure to a concentration of 1 µg/m$^3$ of a particular compound over a lifetime. (Idaho DEQ 1992, Appendix 6; Idaho DEQ 1993). Separately, the AACC reflects that *ambient concentration* that would result in a 1 in 1 million risk of developing cancer assuming a constant exposure to that ambient concentration over a 70-year lifetime.

As noted above, the IUR represents the incremental increased cancer risk from a lifetime (70 years) inhalation exposure to a constant (24 hours/ day and 365 days/ year) dose concentration of 1 µg/m$^3$. In other words, the exposure duration is assumed within the IUR and the cancer risk assumes constant, lifetime (70 years) exposure. Because the IUR estimates cancer risk based on continuous

[1] The National Institute of Health (Cancer Statistics - NCI) states that approximately 40.5% of men and women in the US will be diagnosed with cancer at some point during their lifetimes (based on 2017–2019 data); that is roughly a risk of 4 persons in every 10 persons developing cancer over a lifetime. Risks of 1 in one million or one in 1 hundred thousand are very low. To put that risk in perspective, the National Oceanic and Atmospheric Association estimates that the chances of getting struck by lightning is much higher—1 in 15,300 in a lifetime (How Dangerous is Lightning? (weather.gov)

exposure to 1 µg/m$^3$ of a chemical over a lifetime (70 years), the IUR does not represent the lifetime risk of developing cancer after a single exposure.[2]

The URF in IDAPA 586 and the EPA IUR for arsenic are presented in Figure 1. EPA considers a lifetime to be 70 years. EPA recognizes that exposure duration is not always this long.

The AACC for each chemical is based on and derived from the EPA URF and a lifetime (70 years) exposure duration. The AACC is the identified risk divided by the URF. The AACC reflects the ambient concentration of arsenic to which a person could be exposed 24 hours a day, 365 days a year, for a 70-year lifetime before the identified cancer risk level is exceeded. The AACC value appears in EPA's IRIS database. EPA calls this value the "Air Concentration at Specific Risk Levels," and these levels assume a lifetime of 70 years and a constant exposure for 70 years.

Figure 1 illustrates: (A) risk level: (B) how the AACC is derived, (C) the AACC concentration, and (D) the ambient air concentration of arsenic that an individual would need to be exposed to for a full year to equal a full lifetime (70-year) exposure at a cancer risk expressed in the AACC (1 in 1,000,000).

**Figure 2:**
Inhalation Unit Risk — 4.3E-3 per (µg/m$^3$)
Extrapolation Method —absolute-risk linear model

| A | B | C | D |
|---|---|---|---|
| **Risk Level** | **How AACC is derived *(risk level /URF)*** | **AACC Concentration *(risk level/URF)*** | **Annual Air Concentration equating to a 70-year constant exposure** |
| **E-4 (1 in 10,000)** | 0.0001 / 4.3E-3 | 2.3E-2 µg/m$^3$ | 1.6 µg/m$^3$ (0.023 x 70 years) |
| **E-5 (1 in 100,000)** | 0.00001 / 4.3E-3 | 2.3E-3 µg/m$^3$ | 0.16 µg/m$^3$ (0.0023 x 70 years) |
| **E-6 (1 in 1,000,000)** | 0.000001 /4.3E-3 | 2.3E-4 µg/m$^3$ | 0.016 µg/m$^3$ (0.00023 x 70 years) |

Source: Arsenic IUR from EPA [*Integrated Risk Information System (*Arsenic, inorganic (CASRN 7440-38-2) | IRIS | US EPA*), modified by adding Column B to illustrate how the AACC is derived and Column D showing the Annual Air Concentration equating to a 70-year constant exposure.]*

As illustrated, the risk in Column A may manifest when the annual air concentration shown in Column D is emitted 24 hours per day, 7 days per week, for 365 days in one year. One year of constant exposure at the levels in Column D is to equal the constant exposure of the AACC concentration in Column C, over 70 years.

DEQ applied the TAPs rules to the SGP consistent with EPA's risk assessment methodology including accepted scientific principles governing toxicity values, exposure evaluations, and cancer risk. The

[2] EPA IRIS defines Inhalation Unit Risk (called URF in the IDAPA 586 table) as: Inhalation unit risk (IUR) is an estimate of the increased cancer risk from inhalation exposure to a concentration of 1 µg/m$^3$ for a lifetime. The IUR can be multiplied by an estimate of lifetime exposure (in µg/m$^3$) to estimate the lifetime cancer risk.

worst case SGP annual air concentration is 0.00414 µg/m$^3$ (page 305 of 506 in the SOB dated 6/17/2022) and is significantly lower than the annual air concentrations in Column D. This means that if arsenic were emitted from SGP at the worst-case level (0.00414 µg/m$^3$) constantly for a single year, the dose would not equal the dose of the AACC level (Column C) over 70 years. The Board's concern that SGP could emit the equivalent of 70-year emissions in one year is not possible. I confirmed this conclusion applying EPA methods and common toxicology analyses.

DEQ also applied the TAPs rules to the SGP consistent with the agency's principles reflected in Idaho DEQ 1992 that explained the bases for the Board's adoption of the TAPs rules. The statements explain that EPA risk assessment methodology formed the foundation of the TAPs rules.[3] Specifically, the document directs use of refined modeling, consistent with guidance documents from EPA at the time, updated guidance on the use of URFs, and adjustments for exposure duration in the assessment of risk.

The statements explain the basis of the Board's adoption of the TAPS rules are consistent with DEQ's calculation of an exposure concentration, indicate that the AACCs are screening levels, allow for refined modeling as needed, and reference EPA as the source of URFs (and the AACCs) with the indication that their use should be consistent with EPA risk assessment guidance. The 1992 Meeting Packet refers to the AACCs (as well as all other TAPs values) as "screening levels." Specifically:

> The DEQ list of regulated air toxics is quite large in scope. However, this was not done to burden industry. Quite the contrary. It was done in order, to the maximum extent possible, provide industry with a ready reference to acceptable screening levels of these various compounds. This enables new sources to review their processes and see if controls, may be needed early on in the planning process, there by maximizing start up budget planning. In addition, it allows existing sources to review their emissions and see how they compare to the screening levels that DEQ considers reasonably protective of human health and the environment.

Appendix 6 describes that the exposure must be 24 hours/day for 70 years to interpret the risk associated with a URF or AACC properly. The appendix describes that the URFs in IDAPA 586 are the same as EPA's IURs, noting that the URF is the probability of developing excess cancers over a 70-year lifetime exposure to 1 µg/m$^3$ of a carcinogen expressed in terms of a screening emission level or an acceptable ambient concentration for a carcinogenic TAP. (Idaho DEQ 1992, Appendix 6).

Appendix 7 describes that URFs are applied to determine the risk to off-site receptors and that refined modeling is used to evaluate the risks. According to DEQ's statements in Appendix 7, refined modeling can include adjusting model parameters such as emission, topography, meteorology, or receptors. (Idaho DEQ 1992, Appendix 7).

[3] This packet was first introduced in this proceeding when the Idaho Conservation League (ICL) submitted a declaration that included reference to a 1992 document titled "Meeting Packet: Idaho Department of Environmental Quality, Monday July 13, 1992" (Idaho DEQ 1992).

These adjustments are made in compliance with IDAPA 210.02 as described in a relevant rulemaking document (Idaho DEQ 1993, Comment 108, page 30).

DEQ's approach to implementing the TAP rules is rooted in rulemaking documents that express its intentions and authority to follow well established risk assessment methodologies adopted by EPA.

**B. Based on methods consistent with EPA and DEQ policies and methodologies, DEQ appropriately adjusted the exposure duration to 16 years (16/70) consistent with conditions in the PTC limiting project life because this adjustment was necessary for a scientifically defensible "apples to apples" comparison with the AACC.**

The values set forth in IDAPA 586 and applied by DEQ are based on EPA toxicological determinations and derived from EPA's IRIS database. DEQ's application of the AACCs to comply with the Air Rules conforms to EPA guidance for evaluating risk, including accounting for the appropriate exposure duration. As detailed in this section, DEQ appropriately conducted its analysis following EPA methodology by comparing modeled concentrations from the SGP to the arsenic AACC.

One of the critical considerations in evaluating risk is exposure duration. EPA's definition for IUR reads (emphasis added):

> Inhalation unit risk (IUR) is an estimate of the increased cancer risk from inhalation exposure to a concentration of 1 µg/m$^3$ for a lifetime. The IUR can be multiplied by an estimate of lifetime exposure (in µg/m$^3$) to estimate the lifetime cancer risk.[4]

The AACCs set forth in IDAPA 586 assume an estimate of lifetime cancer risk based on 70 years of constant exposure and a 70-year lifetime. The assumed exposure is 24 hours /day and 365 days /year for 70 years. In other words, at a specific risk level, say 1 in 1,000,000, a person can be exposed to the AACC level (0.00023 µg/m$^3$) of arsenic every day, 24 hours/day, for 70 years and will have a 1 in 1,000,000 risk of developing cancer. A project that is permitted to operate for 70 years or more would properly assume a 70-year lifetime exposure when comparing modeled concentrations against the AACC.

If 70 years is not the exposure duration, then adjustments to the exposure duration are necessary to appropriately evaluate the cancer risk, as directed by EPA and as intended by the Air Rules (Idaho DEQ 1993, Comment 108, p.30)[5]. For a project like SGP with a 16-year project life, the analysis is adjusted to reflect the actual exposure duration for an "apples to apples" comparison with the AACC.

In this case, the AACC (0.00023 µg/m$^3$) is based on an assumed 70-year exposure. The SGP exposure duration is shorter (16 years), so the cancer risk cannot be determined accurately by comparing the modeled exposure concentration (0.00414 µg/m$^3$) to the AACC. Comparing those emissions to the AACC would result in an over-estimation of cancer risk because the dose will not continue for the 70-year lifetime exposure assumed in the development of the AACC. For an "apples to apples" comparison, evaluation of SGP emissions must take into account the shorter exposure duration.

[4] https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system

[5] IDHW believes that the revised [IDAPA] Section 210. 02 Quantification of Emissions Rates and Ambient Concentrations, which allows site specific data gives adequate flexibility in compliance with toxic standards.

EPA guidance provides methodologies to assess risk, taking into consideration the exposure to an air concentration based on a formula for "air concentration adjusted for exposure." EPA guidance reflects this approach through a calculation methodology called the "adjusted air concentration" ($\mathbf{C_{air\text{-}adj}}$), which considers "the timeframe over which inhalation exposure occurs." (EPA 2009, EPA 2024a) (Exposure Assessment Tools by Routes - Inhalation | US EPA).

According to EPA, and as undertaken by DEQ, the exposure concentration must be adjusted for exposure duration to accurately reflect the exposure, associated dose, and resultant risk of cancer response. This adjustment allows for a correct ("apples to apples") comparison of exposure concentration to the AACC. This adjustment allows DEQ to evaluate whether the risk from actual exposure over the life of the project is acceptable under EPA methods and the AACCs.

DEQ followed this EPA methodology to assess the risk of arsenic emissions from the SGP.[6] The approach taken by DEQ for the SGP is set forth below (shaded notes are presented by author clarification):

**Cair-adj=Cair x ET x 1day/24 hours x EF x ED/AT**

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Where:

$\mathbf{C_{air\text{-}adj}}$ = Air Concentration adjusted for exposure
$\mathbf{C_{air}}$ = Concentration of contaminant in air ($mg/m^3$) (this is the modeled or measured annual air concentration)
**ET** = Exposure time (hours/day) (this is assumed to be 24 hours/day)
**EF** = Exposure frequency (days/year) (this is assumed to be 365 days/year)
**ED** = Exposure duration (years) (this is 16 years – the life of operations)
**AT** = Averaging time (days) (this is lifetime – 70 years x 365 days/year; it never changes for any carcinogen)

EPA specifies that the first variable, concentration in air ($\mathbf{C_{air}}$) is either a measured or modeled value based on an annual average concentration. *For the SGP, DEQ used the highest modelled concentration after considering 14 different scenarios (0.00414 $\mu g/m^3$)*[7]

The temporal parameters in the equation include the following:

- Exposure time (**ET**) and exposure frequency (**EF**), which refer to the frequency with which the exposure occurs and might be provided in hours per day and days per year, respectively. *For the SGP DEQ used 24 hours/day for ET and 365 days/year for EF for the SGP review.*

[6] EPA established three calculations to assess risk properly. For completeness the additional EPA calculation methods for are presented in Attachment B.
[7] See Kevin Lewis Declaration

- Exposure duration (**ED**) is the amount of time that an individual or population is exposed to the contaminant being evaluated and is typically given in years. *For the SGP, DEQ used the 16-year life of mine ED reflected in enforceable PTC conditions.*
- Averaging time (**AT**) is the amount of time over which exposure is averaged and is equal to ED for assessing non-cancer risks. For chronic assessments (e.g., cancer), potential lifetime average daily dose (LADD) is calculated in which lifetime (LT, in days) is substituted for AT. *For the SGP, DEQ used 70 years, the default value, for the SGP review.*

Inclusion of a project-specific exposure duration in the calculation of the exposure concentration is essential for an accurate assessment of potential risks and for an appropriate comparison to the AACC to determine compliance with the Air Rules.

Using the highest modelled annual average arsenic concentration from the project of 0.00414 µg/m$^3$ (page 305 of 506 in the SOB dated 6/17/2022)[8] the calculation of the air concentration adjusted for exposure from the SGP is as follows:

$C_{air\text{-}adj}$ (Air Concentration adjusted for exposure) =

$$\left[ \quad C_{air} \quad x \quad ET \quad x \quad {}^{1}/_{24} \quad x \quad EF \quad x \quad ED \quad \right] / \left[ \quad AT \quad \right] = \quad C_{air-adj}$$

$$\left[0.00414 \frac{\mu g}{m3} x\ 24 \frac{hours}{day} x\ \frac{1\ day}{24\ hours}\ x\ 365 \frac{days}{year}\ x\ 16\ years\right] / \left[365 \frac{days}{year} x\ 70\ years\right] = 0.00095 \frac{\mu g}{m3}$$

In this analysis, the numerator (ET x EF x ED = 24 hours/day x 1 day/24 hours x 365 days/year x 16 years) reflects the exposure duration and the denominator of the equation (AT = 365 days/year x 70 years) reflects the 70-year averaging time for carcinogens. The resulting arsenic air concentration from the SGP adjusted for exposure risk review is 0.00095 µg/ m$^3$.

This approach follows the underlying rulemaking documents, including the authority noted in IDAPA 210.02. (Idaho DEQ 1993, Comment 108, p.30). DEQ appropriately adjusted the SGP evaluation to use 16 years (the actual exposure duration) because there can be no exposure to emissions beyond the life of mine. DEQ evaluated the highest annual air concentration modelled to determine the risk of a maximum 16-year exposure over a 70-year lifetime (modelled concentration x Exposure Duration (16 years)/70-year lifetime x URF. DEQ's approach followed EPA scientific principles, accepted risk assessment method, and foundational DEQ rulemaking documents.

**C. The highest air concentration projections modelled for arsenic emissions from SGP are below the acceptable lifetime risk established by the T-RACT AACC listed in IDAPA 210.12 and 586, and therefore the risk from the SGP as permitted is protective of human health and the environment.**

The calculated air concentration adjusted for exposure is 0.00095 µg/ m$^3$ -- this is the proper project-specific arsenic concentration to compare to the T-RACT AACC of 0.0023 µg/ m$^3$ for a cancer risk of 1:100,000 (see page 305 of 506 of the 6/17/2022 SOB).

This method is reflected in the permitting reviews to determine that the arsenic emissions risk was acceptable. Comparison to the T-RACT AACC is as follows:

[8] See Kevin Lewis declaration

$$\frac{1}{100{,}000} T\text{–}RACT\ Risk\ x\ 0.00095 \frac{\mu g}{m3} C_{air-adj} \ /\ 0.0023 \frac{\mu g}{m3}\ T\text{–}RACT\ AACC =$$

$$= 4.1E\text{–}6\ or\ \frac{1}{240{,}000} Actual\ Risk$$

This acceptable method provides an "apples to apples" comparison of SGP arsenic air concentrations to the AACC adjusted for T-RACT. This is the mathematical equivalent of 16-year emission rate of arsenic, plus 54 years of zero arsenic emissions, divided by 70 years to produce an annual exposure concentration that can be compared to the AACC.

DEQ's use of EPA toxicity values and risk assessment methodology in evaluating emissions from the SGP is consistent with IDAPA 586 because the same EPA toxicity values and risk assessment methodology formed the basis of the AACCs.

**D. IDAPA 210.15 allows for a 10-times adjustment to the AACC when a project will operate for 5 years or less and is an example of DEQ's use of EPA's methods to achieve "apples to apples" analyses to compare with the AACC values.**

IDAPA 210.15 provides that "[f]or short term sources, the applicant may utilize a short-term adjustment factor of ten (10). For a carcinogen, multiply either the applicable acceptable ambient concentration (AACC) or the screening emission rate, but not both, by ten (10), to demonstrate preconsturction compliance."

The *Final Order* held that the Air Rules do not explicitly authorize an adjustment for sources that will operate for more than 5, but less than 70 years. *Final Order* at 21. The Air Rules and IDAPA 210.15 should not be read to impede adjustment for exposure duration when a project will operate for less than 70 years. (Idaho DEQ 1993, Comment 108, p.30). An exposure duration is necessary and proper from a scientific and toxicological standpoint to apply the AACCs accurately. Omitting this adjustment is contrary to common scientific practice and the intent of the Air Rules as reflect DEQ documents (Idaho DEQ 1992 and Idaho DEQ 1993). Specifically the refernce from Petitioners reads:

> For short term sources (usually less than five years in duration), such as remediation projects, a probability of greater than one in a million risk (over 70 years) will generally be acceptable to account for the decreased term of exposure. It is not acceptable however, for exposed individuals to receive a full 70 year exposure during the life of a short term project. (Idaho DEQ 1992).

Unpacking this content may be helpful. First, the accepted scientific approach to determine risk is to calculate a lifetime average daily dose that includes the air concentration for the duration of exposure. In other words, to compare "apples to apples" an evaluator must adjust the default exposure duration (70 years) to the actual of exposure duration (16 years for SGP) then divide by 70 years to account for a lifetime-based URF. This is the proper method to determine "a probability of greater than one in a million risk (over 70 years)" as described.

Next, to expose individuals to "a full 70-year exposure" means that the AACC value is multiplied by 70 to derive the full exposure. See Figure 1 above. For arsenic emissions, the calculations follow:

- At a risk of 1E-6, using the AACC of 0.00023 µg/m$^3$ arsenic air concentration
  - 0.00023 µg/m$^3$ x 70 years = 0.016 µg/m$^3$
  - Exposure to 0.016 µg/m$^3$ air concentration every day for one year would expose an individual to the full 70-year exposure in one year.
- At a risk of 1E-5, using the T-RACT AACC of 0.0023 µg/m$^3$ air concentration
  - 0.0023 µg/m$^3$ x 70 years = 0.16 µg/m$^3$
  - Exposure to 0.16 µg/m$^3$ air concentration every day for one year would expose an individual to the full 70- year exposure in one year.

The AACC is not a one-year exposure limit and such an interpretation ignores both the definition of the URF and the definition of the AACC.

**E. Idaho's AACCs are not annual average air concentration limits or standards.**

The AACCs are distinct from annual average air concentrations and are not limits or standards. (Idaho DEQ 1993, Comment 31, page 10; Comment 111, page 32). One is an apple, the other an orange. The reference to an annual average concentration represents the longest period for which air concentrations are projected by air emission models, specifically AERMOD. Models only project concentration estimates over certain time periods of prediction (hourly, daily, monthly) within one year or over an annual duration. Models do not address exposure duration.[9]

The AACCs (derived from EPA's URFs) reflect exposure duration (i.e., 70-year lifetime) and present an acceptable constant dose of a contaminant over a known averaging time (70 years is the default averaging time for carcinogens). The AACC is the concentration that a person may be exposed to for a lifetime continuously, with an incremental risk of cancer of 1 in 1,000,000. The T-RACT AACC is the concentration that a person may be exposed to for a lifetime continuously, with an incremental increased risk of cancer of 1 in 100,000. The AACCs are not one-year limits. Compliance with IDAPA 586 is not based on comparing annual air concentrations from the model to an AACC. (Idaho DEQ 1993, Comment 111, page 32).[10]

Comparing an annual average concentration from a model to the AACC is not a risk evaluation and does not demonstrate acceptable risk. Nor is the annual average concentration predicted from modeling the same as an exposure concentration.[11]

DEQ understood and applied these distinct parameters properly in the PTC reviews for the SGP. For the SGP PTC, DEQ reviewed maximum daily emissions under 14 modeling scenarios that were each modeled for 365 days/year to produce an annual average arsenic concentration for each of the 14 scenarios. DEQ then used the highest annual average concentration—0.00414 µg/m$^3$—to conduct its risk evaluation calculations following EPA and DEQ guidance referred to above to compare to the AACC. This conservative approach assumed that the highest annual concentration modelled is

[9] See Kevin Lewis Declaration
[10] In the design of this rule, the AAC relates to a stack emission rate. In the most prescribed sense, the AAC would be assured by a permit ted emission limit. This emission limit then might be guaranteed by some ample control technology design or perhaps an operational limit prescribed in the permit.
[11] See Kevin Lewis Declaration

emitted for all the years of operation at SGP. To demonstrate compliance with the arsenic AACC, DEQ adjusted the exposure duration as directed by EPA (methodology explained above) and the DEQ guidance referred to above. Based on the modeling and scientific risk evaluation methods, DEQ concluded that the maximum arsenic emissions from SGP would cause an increased cancer risk of less than 1 in 100,000. This use of the annual average concentration in DEQ's AACC risk evaluation followed appropriate scientific methods.

Treating the AACC as an annual emission limitation or annual limit on ambient exposure concentration would result in significantly lower acceptable risk from TAPS emissions than envisioned by the Air Rules. If the T-RACT AACC of 0.0023 µg/m$^3$ were based on a one-year exposure, then the risk represented by that value would be about 1 in 7,000,000. Using the EPA guidance for URFs, and assuming continuous exposure to the T-RACT AACC concentration for an exposure duration of 1 year, the $CA_{air\text{-}adj}$ is:

0.0023 µg/m$^3$ x 24 hrs/day x 365 days/yr x 1 yr / (70 yrs x 365 days/yr x 24 hrs/day) = 0.000033 µg/m$^3$

And the associated risk of developing cancer over a 70-year lifetime is:

0.0043 (µg/m$^3$) x 0.000033 µg/m$^3$ = 1.4 E-7 (about 1 in 7,000,000)

The AACC (and the URF it is based on) represent the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a contaminant over a lifetime at an air concentration of 1 µg/m$^3$. In other words, the AACC and T-RACT AACC represent the risk to an individual if exposed all day, every day for a lifetime to a concentration of 1 µg/m$^3$. The AACC and T-RACT AACC do not represent the risk of a cancer response following a single exposure. The latter interpretation defies fundamental toxicological science used by the EPA in the development of IURs (URFs).

**F. The 5-year rolling average production limit will ensure that the ambient arsenic concentration caused by the SGP does not cause unacceptable cancer risk.**

The *Final Order* expresses concern that the use of a 5-year rolling average production limit will allow for smoothing out of peak ambient concentrations of ambient arsenic and may not be equally protective of ambient air as the AACC limits set forth in IDAPA 586.

First, the AACC is not a limit based on my reading of the relevant documents cited in this memorandum, in particular Idaho DEQ 1992 and Idaho DEQ 1993. Second, in my opinion, for purposes of evaluating cancer risk, use of a 5-year rolling average production limit is as protective as an annual production limit.

As described earlier in the report, risk assessment evaluates the probable response to a particular dose of a carcinogen over time. Risks increase from cumulative long-term exposure, not short-term annual emissions. Risks are assessed based on total cumulative dose, as demonstrated in Figure 1, column D. As shown in Figure 1, a total cumulative dose of 0.16 µg/m$^3$ of arsenic would cause an increased cancer risk of 1 in 100,000. However, whether this dose occurs in 1 year, 5 years, 16 years, or 70 years, the risk would not change.

The 5-year average production limit ensures that if production is above the limit in one year, then future production must be reduced below the limit. This ensures that the cumulative ambient arsenic concentration does not exceed the acceptable ambient concentration level for the defined risk. The 5-year average production limit in the PTC ensures that the ambient arsenic concentration from the SGP over the project life (exposure duration of 16 years) results in an acceptable cancer risk under the Air Rules.

## VII. CONCLUSIONS

Based on my review of the modeled emissions from the SGP, the Air Rules, and EPA risk assessment methodology, and relevant explanatory documents, arsenic emissions from the SGP will result in an increased cancer risk of less than 1 in 100,000. DEQ appropriately applied a project specific adjustment factor to account for exposure duration, consistent with established EPA risk assessment methodology. The permit issued to SGP, including the five-year rolling average production limit, represents acceptable risk confirmed by methods that follow EPA's approaches for using URF, IUR values in risk assessments.

**ATTACHMENT A**
References

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**ATTACHMENT B**

*For completeness the additional EPA calculation methods for are presented here.*

**Method 2: Calculate the risk associated with the air concentration**

The second option for correct use of the URF in the evaluation of project risk is to forward-calculate a risk from the modelled concentrations and using the IUR (URF) as shown in the "Equation 6" from EPA 2009 (below).

The EPA screening levels, associated risk calculator and several guidance documents published by EPA rely on the unit risk factor (again, called IUR by EPA and called URF in the Air Rules). For arsenic, the IUR is 0.0043/(µg/m$^3$)(from EPA 2024b and shown in IDAPA 58.01.01.586 as the URF). Exposure for inhalation is determined by the following equation for carcinogenic chemicals (EPA 2009), which necessarily relies upon an exposure time, frequency and duration to estimate dose. Exposure concentration (EC) is the parameter that is to be evaluated by use of the URF; Concentration in Air (CA) is the estimated contaminant concentration that is predicted by the model. The exposure concentration is the equivalent to the adjusted air concentration described in Method 1.

**From EPA 2009, Risk Assessment Guidance for Superfund, Part F. Page 14**

**EC = (CA x ET x EF x ED)/AT** **(Equation 6)**

Where:
EC (µg/m$^3$) = exposure concentration;
CA (µg/m$^3$) = contaminant concentration in air;
ET (hours/day) = exposure time;
EF (days/year) = exposure frequency; ED
(years) = exposure duration; and
AT (lifetime in years x 365 days/year x 24 hours/day) = averaging time

The EC is then multiplied by the URF to calculate incremental lifetime risk.

Using the SGP highest modeled air concentration for arsenic of 0.00414 µg/m$^3$:

Exposure Concentration (µg/ m$^3$) =

$$[\quad CA \quad x \quad ET \quad x \quad EF \quad x \quad ED \quad ] / [\quad AT \quad ] = \quad EC$$

$$\left[0.00414 \frac{\mu g}{m3} x\ 24 \frac{hours}{day}\ x\ 365 \frac{days}{year}\ x\ 16\ years\right] / \left[70\ years\ x\ 365 \frac{days}{year}\ x\ 24 \frac{hours}{day}\right] = 0.0095 \frac{\mu g}{m3}$$

Then, the Exposure Concentration (EC) x IUR (or URF) estimates the risk:

$$0.00095 \frac{\mu g}{m3} EC\ x\ 0.0043 \left(\frac{\mu g}{m3}\right)^{-1} IUR = 4.1E{-}6\ or\ \frac{1}{240{,}000} Risk$$

**Method 3: Adjust the AACC to reflect the Project Duration**

The same calculation can be re-arranged to estimate a screening concentration in air, as shown in Table 4 from EPA 2009 (below).

**TABLE 4**

**RECOMMENDED PROCEDURE FOR CALCULATING RISK-BASED SCREENING CONCENTRATIONS FOR CONTAMINANTS IN AIR**

| | Cancer Risk-Based | Hazard-Based[1] |
|---|---|---|
| **Step 1: Select Target Levels** | Select target cancer risk (e.g., 1 x $10^{-6}$). | Select target HQ (e.g., 1). |
| **Step 2: Identify Toxicity Value**[2] | Identify inhalation cancer potency value (e.g., IUR). If none exists, proceed with hazard-based screening level calculation. | Identify inhalation reference value (e.g., RfC) to match exposure scenario (acute, subchronic, or chronic). If none exist, proceed with cancer screening level calculation. |
| **Step 3: Calculate CA** | Using target cancer risk from Step 1 along with the receptor- and scenario-specific exposure parameter values, calculate CA; the following equation is recommended: CA = (AT x Target Risk)/(IUR x ET x EF x ED) | Using target HQ from Step 1 along with the receptor- and scenario-specific exposure parameter values, calculate CA; the following equation is recommended: CA = (AT x Target HQ x RfC x 1000 µg/mg)/(ET x EF x ED) |
| **Step 4: Select Screening Concentration** | Select minimum of predicted cancer risk- and hazard-based values as screening concentrations.[3] Repeat for each receptor/scenario combination of interest. | |

[1] Hazard-based screening concentrations are typically derived from reference values such as RfCs. These values may be available for non-cancer effects but may include cancer, if a nonlinear MOA is thought to operate for a chemical. [2] If no inhalation toxicity value is available for a chemical, contact STSC for further direction on how to proceed.

[3] Screening levels estimated from the equations presented in Step 3 could yield concentrations that exceed the maximum possible vapor concentration for a chemical. In such cases, it may be useful to calculate the maximum possible vapor concentration of the pure contaminant at the temperature of interest, using the following formula: $C_{max}$ = S x H x $10^3$ L/$m^3$, where S = solubility at 25° C (or temperature of interest) and H (unitless) = Henry's Law Constant at 25° C (or temperature of interest). This equation is based on an established relationship (see, for example, Schwartzenbach et al. 1993), that allows the Henry's Law Constant to be estimated as the ratio of a compound's vapor pressure and aqueous solubility for compounds that are slightly to moderately soluble in water. When the dimensionless Henry's Law constant, H, is used, the relationship described above can be used to calculate the vapor concentration of a saturated solution of a given compound, assuming equilibrium between the vapor and aqueous phases.

Note: MOA = Mode of Action, which refers to the way in which a chemical exerts a health effect.

In Step 3 of this table AT is averaging time as described in "Equation 6" from EPA 2009 or 70 years x 365 days for carcinogens (note - for noncarcinogens only, AT is equal to the Exposure Duration) (source: EPA RSLs 2024c, EPA Risk Assessment Guidance for Superfund 2009).

As shown in Step 3 the third option for assessing risk is to adjust the screening value (here, the AACC) to reflect the project-specific exposure duration. The default exposure duration assumed in the AACC is 70 years. The AACC values represent a concentration in air (CA) for a 24 hour/day, 365 day/year, and for 70-years of exposure (using both a lifetime exposure duration and lifetime for averaging time). At a T-RACT risk of 1:100,000 the calculation of the T-RACT AACC is as follows, using the equation in Step 3 of EPA guidance (2009):

$$[\ AT\ x\ \text{Target Risk}]\ /\left[\ \text{IUR}\ x\ \text{ET}\left(\frac{\text{hours}}{\text{day}}\right) x\ {}^{1\ day}/_{24\ hours}\ x\ \text{EF}\ x\ \text{ED}\right] = CA$$

$$specifically$$

$$\left[70\ years\ x\ 365\frac{days}{year}\ x\ {}^{1}/_{100{,}000}\right] / \left[4.3E{-}3/\frac{\mu g}{m3}\ x\ 24\frac{hours}{day}\ x\ \frac{1\ day}{24\ hours}\ x\ 365\ \frac{days}{year}\ x\ 70\ years\right] =$$

$$= 0.0023\frac{\mu g}{m3}\ Screening\ concentration, CA_{70-year\ ED}$$

The adjustment used by DEQ for the exposure duration reflects the 16-year life of the mine (i.e., there can be no operations related exposure after operations cease). Accordingly, the appropriate AACC for this project is:

$$[\ AT\ x\ \text{Target Risk}]/\left[\ \text{IUR}\ x\ \text{ET}\ x\ {}^{1\ day}/_{24\ hours}\ x\ \text{EF}\ x\ \text{ED}\ \right] = CA$$

specifically,

$$\left[70\ years\ x\ 365\frac{days}{year}\ x\ {}^{1}/_{100{,}000}\right] / \left[4.3E{-}3/\frac{\mu g}{m3}\ x\ 24\frac{hours}{day}\ x\ \frac{1\ day}{24\ hours}\ x\ 365\ \frac{days}{year}\ x\ 16\ years\right] =$$

$$= 0.0102\frac{\mu g}{m3} Screening\ concentration, CA_{16-year\ ED}$$

The adjusted AACC of 0.0102 µg/m$^3$ is used to compare the highest annual average concentration from the modeling without further adjustment to determine the risk. In this case it would be:

$$\frac{1}{100{,}000} Target\ Risk\ x\ 0.00414\frac{\mu g}{m3} C_{air}\ /\ 0.0102\frac{\mu g}{m3}\ CA_{16-year\ ED} =$$

$$= 4.1E{-}6\ or\ \frac{1}{240{,}000} Actual\ Risk$$

Using Method 1 (presented in the memorandum) or Method 2 or 3 presented here, the same estimate of lifetime risk is obtained, which for this project is 4.1E-6.

**ATTACHMENT C**

TETRA TECH

# Theresa K. Lopez, MSPH
## Principal Toxicologist

## EXPERIENCE SUMMARY

Ms. Lopez has worked extensively in public health, epidemiology, human health risk assessment, toxicology, and occupational health and safety, representing both the public and private sector for over 30 years. She has been a task manager for a variety of human health risk assessment projects, including developing a disease burden reduction model for the optimized placement of infrastructure. She has led investigation and assessment of chemical releases and environmentally compromised sites such as former nuclear weapons plants, active and closing naval bases, and mining, commercial, and school sites. Ms. Lopez has worked extensively for federal, State, Provincial, and private clients. Her work includes assisting in remediation of former mining sites in the United States (US) and Canada with heavy metal, pesticides, TPH and other chemicals of concern; and risk assessments for proposed mines, hazardous waste release sites, and TPH remediation in the US and Canada. She has prepared human exposure assessments for schools and recreational settings, and assessed clean-up goals and remediation strategies for closed industrial, mining, redevelopment, and other sites. These sites often include the consideration of native and indigenous peoples.

In addition, she has provided exposure and dose reconstruction for Oak Ridge Associated Universities to quantify radiation doses to former nuclear weapons workers. She provided the exposure assessment information and mathematical evaluation for the development of a computer-based mouthing exposure tool for Health Canada which involved comparative evaluation of equations selected from a variety of countries and multi-national interest groups (including Japan, European Chemicals Agency, US, and UK). Currently, Ms. Lopez is performing the human health risk assessments involving both radionuclide and chemical impacts, modeling of indirect exposure pathways, TPH evaluations, and exposure assessments for private clients and US and international regulatory agencies, as well as providing scientific peer reviews and authoring reports for Health Canada related to the Existing Substances Risk Assessment Bureau mandates.

## EMPLOYMENT HISTORY

Tetra Tech, Inc., Senior Toxicologist, 1997–present

PRC Environmental Management (NKA Tetra Tech EMI), Senior Toxicologist/Toxicology Group Leader, 1992–1997

University of Colorado Health Science Center, Student/Professional research Assistant, 1989-1992.

## EDUCATION

MS, Public Health, University of Colorado, 1991

BS, Nutritional Science (Zoology minor), University of Florida, 1989

## AREAS OF EXPERTISE

- Human Health Risk Assessment
- Chemical Risk Assessment
- Radiological Risk Assessment
- Regulatory toxicology
- Risk-based Remediation
- Public Health
- Epidemiology
- Guidance Development
- Exposure Assessments

## TRAINING/CERTIFICATIONS

- Qualified Person, Site Assessment Chapter and Corrective Action Plan Chapter, Saskatchewan
- FEMA Emergency Response Training IS-00005.a, 230.d, and00029

## PROFESSIONAL AFFILIATIONS

Society for Risk Analysis

Society for Environmental Toxicology and Chemistry

## RELEVANT EXPERIENCE

**Peer Review of Chemical Assessments, Consumer Product Safety Commission (2022-present)**. Program manager and lead toxicologist for peer review of chemical and nanomaterial risk assessments developed for and by the Consumer Product Safety Commission. Current project is the review of class-based assessment for organic flame retardants. The peer review will include response to charge questions as well as commentary on the approach and case study to evaluate scientifically defensibility, thoroughness, and accuracy.

**Navajo EPA Ambient Water Quality Standards**. **(8/2018 – present)**. Provided a review of existing ambient water standards and calculated health-based standards for the Navajo Nation based on human exposures, livestock exposures, plants, and food ingestion pathways. The report will be the basis of Navajo EPA water standards for surface water bodies and specifically the San Juan River within the Navajo Nation boundaries.

**Technical Support Services, Agency for Toxic Substances and Disease Registry** (**ATSDR) (US-Nationwide). (12/2016-present).** Managing a contract with ATSDR to provide ad-hoc scientific advice on a variety of public health assessment issues, including chemical toxicity, public health, environmental risk assessment and education and

outreach activities as needed. Projects to date have involved being a subject matter expert in public health assessments methodology, review of minimal risk levels and toxicity data used by ATSDR, and reviewing EPA databases (including the TRI) for potential uses in public health evaluation and outreach as well as providing technical support for ATSDR's soilSHOP outreach (a lead poisoning prevention program). The most current project involves measuring uptake of metals and PFAS from soil to common food plants.

**Arizona Department of Environmental Quality. Phoenix AZ. (11/2019 – present).** Providing toxicology and risk assessment review of reports submitted to ADEQ for approval under the Voluntary Cleanup Program, as well as conducting site-specific risk assessments for historic mining sites to facilitate remediation. Reviewing a variety of submitted reports for former industrial facilities and release sites throughout Arizona.

**Toxicological Literature Reviews and Summaries, Health Canada. Ottawa, Ontario**. **(1/2015-present)** Provided literature search and summary for various chemicals and exposures. A specific example include a literature review and data summary for 36 organic flame retardants and 45 salicylates. A literature search was conducted for each chemical in these two efforts, and summaries of material ranged from a few pages to over 120 pages per chemical depending on the amount of literature identified. Both carcinogenic and noncarcinogenic endpoints were included in the search, and endpoints included acute toxicity (single exposure), short-term toxicity (2 to 89 days), subchronic toxicity (90 days to 1 year), chronic toxicity (more than 90 days), carcinogenicity, reproductive and development toxicity, genotoxicity, endocrine effects, immune system, sensitization, and irritation. In addition, a data extraction project for ultrafine particles was also undertaken, reviewing health outcomes for human exposure to ultrafine particles in over 130 epidemiological papers.

**Peer Review of Screening Assessment Reports (SARs), Health Canada. Ottawa Ontario. (1/2015 – present)** Providing peer review of screening assessment reports for a variety of chemicals in consumer products for the Existing Substances Risk Assessment Bureau under the Chemicals Management Plan. Reviews include a literature search, review of identified literature, qualitative and quantitative exposure assessments, toxicity assessment, calculation of margins of exposure for each exposure pathway, and recommendations on the methodology used to assess the chemicals. Over 50 reviews have been conducted to date.

**Peer Review of New Assessment Methodologies, State of the Science Reports, and Occupational Exposure Guidelines, Health Canada, Ottawa, Ontario (4/2017-present).** Providing peer review of new assessment methodology reports (including *in silico* learning and transcriptomics), State of the Science (including PFAS compounds and Vitamin A) , and Occupational Exposure Guidelines (STOT RE for carbon black) for Health Canada under the Chemicals Management Plan and Hazardous Products Regulations.

**Review of Proposed Drinking Water Guidelines, Health Canada, Ottawa, Ontario (8/2021- 12/ 2023).** Providing peer review of six drinking water guidelines, focused on health-based standards and toxicological concerns, for several inorganic and organic chemicals under the Chemical Management Plan for the Water and Air Quality Bureau (WAQB). The reviews included verification of toxicological information, calculation of a guideline, and other technical issues relevant to water guideline development.

**Exposure Assessment Studies, Health Canada, Ottawa, Ontario 4/2017-present).** Provided peer review of exposure assessment reports, including internal backgrounders on body weight, drinking water intake rates, inhalation rates, and soil/dust intake rates, dermal surface area, and breast milk and formula intake. Collected literature and provided detailed reports for Health Canada on vapor recovery systems and air quality, chemical exposure in nail salon settings. Created and implemented consumer product surveys for hand sanitizer, automobile products, certain arts and crafts products, paint remover and essential oils. The hand sanitizer project was published while the paint remover and essential oil surveys are currently in progress. The automobile products and arts and craft product surveys remained internal to Health Canada but follow-up work may be planned.

**UNICEF WASH Program, Pacific Island Countries. (4/2024-present).** Assessed current disease burden rates for Solomon Islands including diarrheal disease and other water-related illness to determine economic benefits from placement of water and sanitation infrastructure improvements. The public health evaluation identified areas with potentially the highest rates of diarrheal disease and the reduction in disease burden that can be achieved by providing chlorinated water supplies and sanitation systems. The model that was created also can estimate disease reduction through reduced flooding and drainage improvements. A mathematical model was developed for this effort that included weighting factors to account for poverty and age, and different percentages for infrastructure improvements, to estimate disease and mortality reduction from the provision of disinfected water and sanitation infrastructure.

**Relative Risk Ranking Model for Sanitary System Overflow Diversions, Metro Vancouver. (3/2024 – present).** A model was developed for the relative risk to humans and ecological receptors from 5 options for based on options developed for consideration. This evaluation considered several inputs to evaluate potential risks to receptors from the overflow to conveyances, surface water, and eventually the bays surrounding Metro Vancouver. This is was not a quantitative risk assessment but rather a relative ranking of the risks associated with each option. Inputs to the model included overflow volume, distance to conveyance from overflow, distance to receiving body, dilution, chemical/bacterial measurements, and percent paved area surrounding conveyance. Special status ecological receptors in the areas of interest were identified, as well as human uses of the land and communities of special consideration. A ranked score was created for each option, under the broad categories of logistical, ecological risk, human health, and chemical inputs.

**Public Health Assessment and Disease Burden Reduction, Millennial Challenge Corporation, Timor-Leste. (1/2020 - 9/2022).** Assessed current disease burden rates for Dili, Timor Leste, including diarrheal disease and other water-related illness to determine areas that will achieve greatest disease burden reduction from proposed infrastructure improvements. The public health evaluation identified areas with potentially the highest rates of diarrheal disease and the reduction in disease burden that can be achieved by providing chlorinated water supplies, sanitation systems, and drainage improvements to reduce flooding. These improvements may be implemented individually or together, depending on the area. A mathematical model was developed for this effort that included weighting factors to account for poverty, age, and gender differences in the population.

**Development of Biomonitoring Equivalents for Glyphosate and Ethylene Thiourea (1/2021-12/2023).** In partnership with Summit Toxicology, gathered biomonitoring data, regulatory information, and adsorption, distribution, metabolism and excretion (ADME) data for these two pesticides. Data were used to develop biomonitoring equivalents and manuscripts have been developed documenting the results (one has been published and the other has been submitted).

**City of Campbell River, British Columbia, Canada. (1/2023-present).** Performing human health and ecological risk assessment for soil and groundwater with PFOS and PFOA impacts from firefighting training at an airport. Exposure pathways include drinking water, soil contact, and impacts to off-site freshwater bodies.

**Human Health and Ecological Risk Assessment, Duck Lake, British Columbia. (4/2018-11/2018).** Conducted a human health and ecological risk assessment of a former railway right-of-way using. The intent of the risk assessment was to document any potential risks associated with legacy polycyclic aromatic hydrocarbon residue from Site use as a railway, and the community plans to redevelop the right-of-way into a multi-use recreational trail. Site was submitted for closure and redevelopment in November 2018 based on the risk assessment findings.

**Human Health and Ecological Risk Assessment, Aitchelitz, BC. (7/2021-7/2023).** Conducting a human health and ecological risk assessment of a former industrial operations area. The intent of the risk assessment was to document any potential risks associated with current and legacy buried wastes and petroleum releases from the area, including any discharge of groundwater to the Aitchelitz Creek and use of groundwater as a drinking water source. As well, ecological effects in the Aitchelitz Creek were evaluated, particularly for endangered species.

**Human Health and Ecological Risk Assessment, Kitsumkalum, BC. (3/2018 – 3/2019**). Conducted a human health and ecological risk assessment of a former industrial and woodmill operations area. The intent of the risk assessment was to document any potential risks associated with legacy buried wood waste and petroleum releases from the area, including any discharge of groundwater to the Kitsumkalum River and use of groundwater as a drinking water source. The community planned to redevelop a portion of the Site for industrial re-use, while a portion was being considered for residential redevelopment. The Site was submitted for closure and redevelopment in March 2019 based on the risk assessment findings.

**Human Health and Ecological Risk Assessment, Wei Wai Kum First Nation (WWK), Myra Fall Mine (MFM) and Indigenous Services Canada (ISC), Campbell River BC**. **(2021-2022)**. Providing human health and ecological risk assessment for Wei Wai Kum First Nations for this former port area located on Vancouver Island. Chemicals of potential concern include metals and PAHs, with a particular concern regarding marine life in the area and food chain exposures.

**Evaluation of Human Toxicity Data for PFAS and PCBs to Determine Applicability to Marine Mammals, British Columbia Ministry of Environment and Climate Change (BMOECC).** (**9/2020-3/2021).** Vancouver, British Columbia. Provided BCMOECC and federal agencies with a methodology and example of how data developed for the assessment of human toxicity could be extrapolated for use in protecting the endangered

Southern Resident Killer Whale population, as well as other marine mammals. Specific chemicals of interest were PCBs and PFAS.

**San Juan River Risk Assessment, Utah Department of Environmental Quality. (2/2017 – 3/2018).** Task leader for the human health and agricultural risk assessments of the San Juan River. Evaluating impacts of heavy metals (including arsenic) from the Gold King Mine release to residents, ranchers, farmers, and other users of the San Juan River water. Providing data evaluation including comparisons to background values, assessment of data from tributaries to the San Juan River, as well calculating exposure point concentrations. Providing assessment of current and future risks, including consideration of regulatory levels as well as site- and receptor-specific exposure parameters to fully evaluate potential long-term impacts of the release on receptors and the eventual deposition of impacted sediment in Lake Powell.

**Exposure Assessment for Nanoform Cerium Oxide Silver, Titanium Dioxide and Zinc Oxide, National Industrial Chemicals Notification and Assessment Scheme, Australian Government Department of Health and Ageing. (11/2012 – 6/2013).** Provided an exposure assessment of the public to nanoform cerium oxide, silver, titanium dioxide, and zinc oxide present in consumer products. Assessed both qualitative and quantitative measures of exposure, based on a range of nanomaterial content and use of consumer products. For each nanomaterial, provided current information on commercial uses, sources, synthesis methods, characterization, potential exposure routes, toxicokinetics, and environmental fate and transport, as well as addressing data gaps where information was not found. Developed exposure scenarios for each of the four nanomaterials, including exposure to workers, the public (consumers), and the environment, and provided estimates of exposure to the extent possible. Identified data gaps that introduced uncertainty into the estimates or prevented a quantitative estimate of exposure, including that the toxicity of nanomaterials may be related to the number of particles rather than mass of particles present, and that measures of exposure based on mass of material may therefore underestimate potential toxic effects.

**Use of Biomonitoring Data and Biomonitoring Equivalents in Risk Assessment, Health Canada, Ottawa, Ontario. (1/2015-7/2015).** Prepared a report for Health Canada regarding the current use and application of biomonitoring data and biomonitoring equivalents (BE) in regulatory settings as well as considerations for varying types of pharmacokinetic data used to interpret biomonitoring data for various matrices. Authored chapters defining BEs as the translation of screening exposure guidelines or criteria to chemical- and biological media-specific concentrations that can be used interpret biomonitoring results for specific chemicals in a health risk context. Provided an overview of its application in regulatory risk assessment as well as considerations for varying pharmacokinetic data in both animals and humans, and described international efforts in biomonitoring of several chemicals, including perfluorinated compounds, mercury, lead, 2,4-D, and phthalates. In addition, the report contains a discussion of how pharmacokinetic data can be used to interpret biomonitoring data for regulatory risk assessment of industrial chemicals under the Chemicals Management Plan.

**Peer Review of Personal Care Products (PCP) Module III, Assessment of Exposures to Chemicals in Personal Care Products for the Hair. Health Canada, Ottawa, Ontario. (12/2014-4/2015).** Provided a peer review for the Existing Substances Risk Assessment Bureau (ESRAB) - Healthy Environments and Consumer Safety Branch (HECSB) of the proposed Health Canada methodology for assessing consumer exposure to chemicals in hair care products, including an evaluation of recommended default exposure parameters for all receptors and a literature review to determine if additional applicable data were available for use in the effort.

**Peer Review of OECD Documents, Health Canada Risk Assessment Bureau, Ottawa, Ontario. (8/12-8/13).** Performed peer reviews of regulatory and advisory health summary documents for Health Canada for the Organization for Economic Co-Operation and Development (OECD) Cooperative Chemicals Assessment Meeting No. 4 (CoCAM 4). Peer review included verification of health effects data reported in IUCLID Dossier, Screening Information Data Set (SIDS) Initial Assessment Reports, and SIDS Initial Assessment Profiles documents.

**Mouthing Exposure in Young Children: In Silico Tool. Health Canada, Ottawa, Ontario. (11/2013-7/2014).** Designed exposure assessment and mathematical models for 26 separate equations to assess chemical exposure to children through mouthing of various consumer products. Provided a variety of algorithms that may be used with default or user-specified parameters to assess mouthing exposures in children from birth to 8 years of age. Identified mathematical approaches and modeling tools from a targeted search of the scientific literature, as well as various government sources, using on-line search engines and established medical information search tools and included a discussion of the circumstances and modulating factors surrounding an attempt at assessing object-to-mouth exposures in children as well as limitations to assessing such exposure. Also authored the user's guide to accompany the computer-based model that included description of mathematical similarities and differences in assessing exposure to non-food substances through direct mouthing, hand-to-mouth behavior, and swallowing as

well as compiling exposure data to provide users with a range of parameter values to assess chemical intake.

**Human Health and Ecological Risk Assessment, Cantung Mine site, Northwest Territories, Canada.** ( **3/2017-present).** Lead human health and ecological risk assessor for this closed tungsten mine. Providing a detailed quantitative assessment of potential human exposure to metals, petroleum hydrocarbons, and other chemicals of concern at this site, including exposures through hunting, fishing, and gathering of plants as traditional food sources given the remote location and First Nations traditional land uses. Provided assessment of direct exposures including soil ingestion, particulate inhalation, dermal contact with impacted soil and tailings; and ingestion and incidental contact with surface water. Provided a detailed background comparison for metals in soils that will be used to guide soil remediation.

**Risk Assessment, Public Works and Government Services Canada, Tulita NWT and Fort Reliance, NWT, and Yellowknife, NWT. (9/2017-3/2018).** Currently performing human health and ecological risk assessment for petroleum hydrocarbon releases at three sites in Canada related to previous spills and releases at Royal Mounted Canadian Police detachments and Crown-owned residences. Provided methodology and risk calculations to evaluate indoor air exposure, soil exposure, groundwater impacts, and ecological receptor exposure.

**Confidential Maryland Redevelopment Site, Baltimore Maryland. (3/2022 – 7/2022).** Conducted a multimedia, Site-Specific Risk Assessment for redevelopment of a former electrical manufacturing facility located in Baltimore, Maryland. Using soil, groundwater and soil vapor data, evaluated the potential risk to future residential, commercial, and recreational receptors for Chemicals of Potential Concern (COPCs) included PCBs, chlorinated compounds, petroleum compounds, metals (including lead), and PAHs. Working collaboratively with the Maryland Department of the Environment, the risk assessment was completed in accordance with voluntary cleanup guidance and provided a health-protective path forward to redevelopment.

**Risk Assessment, Beazer, L.A. Paint Site, Vernon, California**. **(5/2014 – present).** Performing a human health risk assessment for vapor intrusion to indoor air from soil gas and groundwater data at historical release site, including paint manufacturing process solvents and petroleum products. Providing risk calculations and correlation of soil, soil gas, and groundwater data to provide multiple lines of evidence to assess potential risks from site reuse and redevelopment. Working cooperatively with both State and local regulators involved in this risk assessment process to address concerns and obtain site closure.

**Indoor Air Risk Assessment, Lockheed Martin Corporation, Palo Alto, California. (9/2016-10/2021).** Risk assessor for ongoing monitoring and assessment of soil vapor plume for solvent release site for Lockheed Martin Corporation (LMC) in Palo Alto, California. Provide updates of indoor air modeling and risk assessment every 6 months using soil gas data collected from 3 impacted areas**.**

**Risk and Dose Assessment, American Jewish University, Simi Valley, California. (2/2016-5/2018).** Performed a review of historic data and risk assessments conducted for the site. Evaluated the potential for the AJU campus, which is downgradient of the Santa Susana Field Laboratory (SSFL), to receive storm water discharge, groundwater, and air-dispersed particulates from former operations at the SSFL. Reviewed AJU and SSFL data related to release of metals, dioxins, perchlorate, polycyclic aromatic hydrocarbons, and TCE, as well as radionuclides. Evaluated historic data and recently collected data to determine if any risks currently exist to patrons of the AJU campus. Conducted site evaluation involving tracing potential migration pathways of contaminants from origin to the campus, and review of soil, sediment, surface water, groundwater, and food data, as well as gamma survey results. Evaluation of impacts to the campus relied upon establishing background and ambient conditions that could influence sampling results, including the effects of forest fires and releases from other facilities in the surrounding urban areas. Exposure evaluation included delineating areas of specific use on the campus, as well as the types of activities, exposure durations, and populations that have access to the areas. Currently providing technical and scientific support as needed.

**Human Health Criteria Development using 2000 Human Health Criteria Methodology for U.S. EPA. (Nationwide). (2/2015-5/2018)**Supported EPA in updating the human health ambient water quality criteria for 94 chemicals published in late 2014 and researched bioaccumulation factors (BAFs) using EPA's 2000 *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* and its 2003 *Technical Support Document Volume 2: Development of National Bioaccumulation Factors*. Conducted a systematic review of peer-reviewed available literature and peer-reviewed public toxicology databases to obtain data to update the BAFs, as well as conducting research of peer reviewed sources to update the relative source contribution (RSC) values used in the 2014 criteria. Also helped provide exposure information to support relative source contribution values using the decision tree in EPA's 2000 Methodology and is currently assisting in producing BAFs and AWQC for 24 metals (including arsenic) and persistent organic pollutants.

**Policy Briefings and Update to Human Health Ambient Water Quality Criteria Methodology Document for U.S. EPA (Nationwide). (9/14-1/15).** Assisted in reviewing toxicological information, exposure factors, relative source contributions, and bioaccumulation potential of the contaminants included in this project. Provided technical input to the methodology recommendations that included recommendations for using human surveillance data as an input in the development of human health criteria, identification of exposure factors, and determination of exposure factors to appropriately assess minority and low-income populations engaged in subsistence fishing practices. Reviewed the use of NHANES data for determining fish consumption rates for this project and provided technical review of several issue papers and briefings discussing options for calculating different exposure factors, use of probabilistic methods for deriving criteria, and developing technically defensible approaches for filling critical data gaps.

**Computer Program to Evaluate Risks from Residential Indoor Water Use, Agency for Toxic Substances and Disease Registry (ATSDR). (6/15-8/17).** Assisted in providing exposure and risk assessment support in the development of a desk top computer application to assess potential risks associated with volatile organics and other compounds from the use of contaminated groundwater for showering, bathing, and other household residential uses. Provided input to the assessment of inhalation and dermal exposures, as well as consideration of background exposure concentrations in ambient air. Performed detailed QA/QC of the model through numerous test runs of each exposure scenario for various chemicals and concentrations. Reviewed EPA databases, including the TRI, to determine if a component could be added to this program to incorporate anthropogenic sources to address background concentrations.

**Human Health and Ecological Risk Assessment, Treasury Metals, Goliath Mine, Ontario. (6/2014-6/2016).** Provided oversight of the Screening Level Risk Assessment (SLRA) for a Proposed Mine Site in Ontario in support of the Environmental Impact Statement, which included a preliminary human health risk assessment and assessment of ecological health risks associated with potential exposures to dust, tailings, waste rock, and mine effluent in the operational and post-closure phases of the proposed mine. Performed the human health risk assessment using guidance from Health Canada, and relied upon site assessment and chemical of concern selection performed using guidance from Environment Canada's Federal Contaminated Sites Action Plan (FCSAP) and Canadian Council of Ministers of the Environment (CCME) as well as Ontario's Provincial Water Quality Objectives (PWQO) and CCME's Water Quality Guidelines. Performed a country foods risk assessment to determine risks from exposures through food items harvested from the proposed mining site during both the operational period and post-closure phases, focusing on mercury and lead. Also attended three public meetings on behalf of the mine proponent to discuss human health risk concerns with the community and First Nations members.

**Human Health Risk and Dose Assessment Review, United States Forest Service, Sundance, Wyoming. (1/2014-6/2015).** Provided a review of the radiological dose from rare earth mining at this proposed mine site. Performed radiological and nonradiological human health risk assessment for the proposed mine based on the Plan of Operations and baseline studies to date, including soil, ore, air, and water data. Provided calculations and predicted risks and hazards from the estimated heavy metal releases from mining operations combined with ore concentrations, rare earth recovery rates, transportation concerns, and radiological dose. Assigned estimated risks to both the operational and post-closure site conditions as determined in the Plan of Operations as part of this predictive risk assessment. The reports are under consideration as part of the Environmental Impact Statement (EIS) process and permitting processes related to mining and radiological material handling.

**Human Health Risk Assessment of Metals and Radionuclides, United States Forest Service, Riley Pass, South Dakota.). (4/2015 – 1/2018).** Reevaluated 2006 risk assessment and risk-based cleanup goals for arsenic, molybdenum, copper, radium-226, uranium, and thorium at a former uranium mine. The risk-based cleanup goals focus on site-specific receptors as determined by the U.S. Forest Service and land use patterns of Native Americans for the site. In addition, reevaluated food ingestion pathways related to hunting and ranching in developing updated risk-based goals. Currently, providing risk assessment input to the clean-up actions being performed at the Site.

**Risk-based Remediation Goal Calculations, State of Montana, Grant Kohrs Remediation Site. (12/2014-2/2015).** Calculated site-specific remediation concentrations for soil to remediate heavy metals, including arsenic, from former mining releases at a currently used recreational area. Evaluation and calculations involved identifying site-specific exposure parameters for different areas of the site based on usage patterns of different occupational and recreational receptors, and were used by the State to guide remediation of the site.

**Human Health Risk Assessment, Ross Adams Uranium Mine, Newmont Mining, Prince of Wales Island, Alaska. (8/2009 – 6/2016).** Responsible for human health screening evaluation of data from inactive mines, mine roads, shoreline area, and surrounding marine area. Environmental media of interest include soil, surface water,

stream sediment, and marine sediment. Determined the background dataset and selected chemicals of potential concern (COPCs) for different media and locations throughout the mine area; the selected COPCs (uranium, thorium, arsenic, and lead) formed the basis of both the human health and ecological risk evaluations. Included food ingestion pathways related to hunting and gathering activities in the risk assessment for both terrestrial and marine foods. Evaluated historical data to provide recommendations for future sampling and analysis, as well as to define the conceptual site model to be used to assess human health risks.

**Human Health Risk Assessment of Lead Exposures, Confidential Client, Former Mining Site, Colorado. (2011).** Conducted a risk assessment for school-aged children exposed to lead in soils from historical mining activity. Results of the risk assessment determined the acceptability of continuing to use the site for a school, as well as the need for any alteration in plans to renovate and expand the existing school grounds.

**Human Health Risk assessment in Support of Permitting, Sheep Mountain Uranium Mine, Titan Uranium, Jeffrey City, Wyoming. (2011).** Responsible for input regarding impacts to human health in application for NRC license, including portions of the Environmental Report regarding public and occupational health impacts from the preferred alternative. Assisted health physicists in incorporating information from their evaluations into appropriate sections of the Technical Report and Environmental Report that were submitted to the NRC.

**Environmental Sampling QAPP, Cameco, Wyoming. (2011).** Prepared a draft Quality Assurance Project Plan (QAPP) for the Smith Ranch-Highland Uranium Project (SR-HUP) facility that included environmental sample collection and laboratory procedures for environmental samples. Report included data quality assurance procedures, quality control specifications, and other technical activities that assure that the data collected at SR-HUP meet appropriate data objectives and protocol per NRC guidance.

**Screening Level Evaluation of an Off-site Relocation of the Large Tailings Pile, Homestake Mining Company, Grants Site. (2012).** Compiled and evaluated of costs and risks associated with moving a large tailings pile from the Site to an off-site location. The purpose of the report was to explore costs, efforts, and regulatory requirements to be met under the alternatives of continuing with the current remediation and reclamation strategy or a hypothetical move of the tailings pile to a location outside of the San Andres Aquifer basin and the Village of Milan. For costing purposes, a distance of 30 miles for relocation was chosen in this evaluation. Costs, impacts, and potential risks of the off-site removal were evaluated to assist in long-term remediation decisions.

**TENORM Dose Assessment, Waste Management of Colorado. (9/2017-5/2018).** Provide radiation dose assessment for 2 landfills and one solidification plant in Colorado. Used site-specific inputs to provide dose assessment for current workers and future hypothetical resident farmers at the Sites, and to support alternative disposal limits for technologically enhanced naturally occurring radioactive material (TENORM).

**Human Health Risk Assessment, American Bumper and Plating, Missoula, Montana. ( 2/2015-2/2016).** Responsible for calculating risks and hazard indices associated with residual metal impacts in soils for potential current and future receptors. Evaluation included site-specific exposure pathways of soil ingestion, dermal contact, and inhalation of particulates by adult and child receptors, as well as an evaluation of the acceptability of lead concentration in soil for each of the four areas of the Site that were sampled and, in some cases, remediated.

**Review of Subsurface Vapor Intrusion Model and Risk Assessment, Lockheed Martin Corporation, Torrance, California. (2011).** On behalf of LMC, conducted an internal review of a human health risk assessment for the site related to subsurface vapor intrusion from a large, heterogeneous groundwater plume in several hydrogeologic formations. Identified potential issues surrounding the vapor intrusion modeling used, as well recommending a revision of the selection of chemicals of concern. Identified potential issues for LMC from calculating risks associated with non-detected chemicals and recommended a different approach to assessing non-detect data.

**Human Health Risk Assessment, Department of Toxic Substances Control, Los Angeles, California. (2/2010 – 5/2010).** Assisted in conducting a human health risk assessment for Chatsworth Park South, a park owned by the City of Los Angeles that had residual contamination from former skeet shooting activities. Assessment included metals related to dissolved lead pellets and PAHs from clay pigeons in shallow soil. The risk assessment will be used to guide remediation activities to re-open the facility.

**Human Health Risk Assessments, Los Angeles Unified School District, Los Angeles, California. (2005-2010).** Task leader for conducting risk assessments for proposed school sites in the greater Los Angeles Area. Environmental contamination assessed varied by site, but included petroleum products, pesticides, and heavy metals. In general, soil was the medium of interest and both direct and indirect exposure pathways were of concern. Provided risk assessments that focused on school-based and residential receptors to conform to California

guidance. In some cases, investigations were required at existing schools; in these cases, provided an evaluation of indoor exposure to impacted air or dust to current receptors. Data from these projects was shared with Department of Toxic Substances Control to establish a regional background concentration of arsenic in soil, which is still in regulatory use in the greater Los Angeles area.

**Radiation Dose Reconstruction, Oak Ridge Associated Universities, Various Sites. (2003-2009).** Reviewed historical documents and estimated potential occupational medical radiation exposures at former nuclear weapons facilities as part of the U.S. Department of Labor's Energy Employees Occupational Illness Compensation Program Act (EEOICPA). Duties included site research of former nuclear facilities and reconstruction of occupational medical practices at each facility that would contribute to worker radiation exposures. Dose reconstruction project at former nuclear weapon facilities was conducted as part of CDC's National Institute for Occupational Safety and Health program to facilitate their response to concerns of DOE workers and contractors requesting compensation for radiation-related cancers resulting from work-related exposure.

**Human Health Risk Assessments, Naval Air Station Moffett, Naval Air Station Alameda, Naval Air Station Lemoore, Naval Air Weapons Station China Lake, Naval Auxiliary Landing Field Crows Landing, Naval Fuel Depot Point Molate, and Naval Station Treasure Island, California**. **(1996-2000)**. Task Leader for Risk Assessment of propellants, explosives, jet fuel, diesel, and gasoline releases to soil and groundwater and the potential to migrate to surface waters. Integrated ASTM Risk based Corrective Action (RBCA) processes and the California RWQCB's guidance documents, developed methods for screening potentially hazardous components. Method proved to be cost effective by allowing use of all data collected and decreasing the cost of confirmation sampling. Successfully negotiated site specific approach to evaluating volatilization into buildings, resulting in lower site specific air concentrations and cost savings by avoiding expensive air sampling. Site specific environmental conditions were used to demonstrate an increased rate of degradation of the components.

**Human Health Risk Assessments, Naval Air Station Lemoore, Naval Air Station Alameda, Naval Air Weapons Station China Lake, Naval Air Station Moffett and Naval Auxiliary Landing Field Crow's Landing, California**. **(1996-2000)**. Task Manager for oversight of human health risk assessments and statistical programs, including evaluation of exposure scenarios and pathways; validation of toxicity values and calculated risks; negotiation of appropriate exposure areas based on current and future land use plans for the base and surrounding communities; statistical evaluation of several data acts; selection of chemicals of concern from combined data sets; and estimation of risks using several data aggregation methods. Evaluated exposure concerns including migration of contaminants from soil to groundwater and surface water. Chemicals of concern included chlorinated pesticides, herbicides, volatile organic chemicals, semi-volatile organic chemicals, metals (including arsenic), TPH, dioxins, furans, and perfluorinated compounds. Provided risk analyses of many different environmentally contaminated areas, including landfills, pesticide/herbicide storage, mixing, and application areas, industrial complexes, artillery ranges, and areas impacted by runoff or groundwater infiltration from these sites. Developed confirmation sampling plans for several sites based on grid sampling patterns and statistical methodology, as well as deriving risk-based concentrations for remediation and compliance studies involving PCBs, pesticides, and explosives.

**Technical Support for Human Health Risk Assessments, U.S. Environmental Protection Agency**. **(1992-1996)**. Provided technical support on epidemiology, public health, and statistical issues to EPA's RCRA/CERCLA program as part of a technical enforcement support (TES) contract. Conducted technical reviews of human health risk assessment documents submitted to EPA to evaluate compliance with CERCLA guidelines. Also reviewed work plans, technical memoranda, and risk assessments submitted to EPA regarding RFETS. Provided oversight for RFETS, and assisted EPA in the development of Monte Carlo simulations for the site. Acted as EPA's representative in negotiating all phases of risk assessments for Rocky Flats, including sampling procedures, data evaluation, exposure scenarios and pathways, chemical intake assumptions and risk characterization. RFETS risk assessments included radionuclides and nonradioactive chemicals such as heavy metals, polycyclic aromatic hydrocarbons, and solvents. Also provided technical oversight in EPA Region VIII include F.E. Warren Air Force Base in Wyoming; Tooele Army Depot in Utah; and Hill Air Force Base in Utah. Project Manager and provided technical oversight for human health and ecological risk assessments at Amoco Mandan Refinery, a RCRA site in EPA Region VIII.

## PUBLICATIONS

### Peer-Reviewed Journal Articles

Hays, S., C. Kirman, J. Flippin**, T.K. Lopez.** Biomonitoring Equivalents for Ethylene Thiourea. Regulatory Toxicology and Pharmacology 150 (2024) 105618.

Hays, S., C. Kirman, J. Flippin**, T.K. Lopez.** Biomonitoring Equivalents for Glyphosate. Regulatory Toxicology and Pharmacology 144 (2023) 105481.

**Lopez T.K.,** K. Jones, A. Roseberry-Lincoln, A. Zidek, L. MacKinnon, L. Marro**.** Adult and children's use of Hand sanitizer during a pandemic - an observational study. Journal of Exposure Science and Environmental Epidemiology, September 24, 2022. DOI 10.1038/s41370-022-00479-w.

**Lopez, T.K.**, J.A. Marshall, S.M. Shetterly, J. Baxter, R.F. Hamman, 1995. Ethnic Differences in Micronutrient Intake in a Rural Biethnic Population. American Journal of Preventive Medicine, 11:301 305.

Marshall J.A., **T.K. Lopez**, S.M. Shetterly, J. Baxter, and R.F. Hamman, 1995. Association of Education Level with Atherogenic Diets in Rural Biethnic Population. American Journal of Preventive Medicine, 11:294 300.

Baxter J., R.F. Hamman, **T.K. Lopez**, et al., 1993. Excess Incidence of Known Non Insulin Dependent Diabetes Mellitus (NIDDM) in Hispanics Compared with Non Hispanic Whites in the San Luis Valley, Colorado. Ethnicity and Disease, 3(1):11 21.

### Conference Presentations

Session Lead and Presenter, 2024. "The Intersection of Human Health and Environmental Risk Assessment: A One- Health Perspective." Society for Environmental Toxicology and Chemistry. Fort Worth, Texas, October 20-24, 2024.

Poster Presentation, 2022. Society for Risk Analysis Annual Meeting, Tampa, Florida, December 4-8, 2022. "**Optimization of Infrastructure Placement Using a Novel Disease Burden Reduction Model Using Public Health Data to Save Lives through Informed Engineering Design."**

Platform Presentation, 2019. Society for Environmental Toxicology and Chemistry, Helsinki, Finland. May 29, 2019, **"Applying a "One Health" Approach to the Assessment of PFAS: Opportunity to Prevent Unintended Consequences."**

Poster Presentation, 2018. Society for Environmental Toxicology and Chemistry, Sacramento, CA, November 6, 2018. **"Applying a "One Health" Approach to the Assessment of PFAS."**

Poster Presentation, 2017. Society for Risk Analysis Annual Meeting, Arlington, VA, December 11-13, 2017. **"Alternate Approach to Deriving Metals Bioaccumulation Factors in Fish."**