

Exhibit 463

**Expert Report of Judy S. LaKind, Ph.D.
In the Matter of *Amsler v. United States***

Prepared by
Judy S. LaKind, Ph.D.
President
LaKind Associates, LLC

Prepared for
United States Department of Justice
950 Pennsylvania Avenue NW
Washington, DC 20530

8 April 2025

Judy S.
LaKind

Digitally signed by Judy S. LaKind
DN: cn=Judy S. LaKind, o=LaKind
Associates, LLC, ou=President,
email=lakindassoc@gmail.com, c=US
Date: 2025.04.04 13:10:16 -04'00'

Judy S. LaKind, Ph.D.

TABLE OF CONTENTS

ACRONYMS/ABBREVIATIONS	6
1. QUALIFICATIONS.....	8
2. CASE OVERVIEW	10
2.1 Summary of opinion	10
3. METHODOLOGY.....	13
4. BACKGROUND ON CHEMICAL EXPOSURE ASSESSMENT	14
5. CONCEPTS AND TERMINOLOGY FOR THE EXPOSURE ASSESSMENT IN THIS REPORT..	19
5.1 Concepts	19
5.2 Terminology	20
6. CHEMICAL CONCENTRATION INFORMATION FOR CAMP LEJEUNE.....	23
6.1 Background on available chemical concentration data for water at Camp Lejeune.....	23
6.2 Water concentration data relevant to Ms. Amsler	24
7. DERMAL AND INHALATION EXPOSURE – THE SHOWER MODEL.....	30
7.1 SHOWER model: Background.....	30
7.2 SHOWER model - Residential: Methodology and parameters	31
7.3 SHOWER model: Parameter default values.....	33
7.4 SHOWER model: Plaintiff-specific modifications	40
7.5 Opinion: Dermal and inhalation exposures at Camp Lejeune	43
8. INGESTION ROUTE OF EXPOSURE – THE PHAST MODEL.....	45
8.1 PHAST model: Background	45
8.2 PHAST model: Methodology and parameters	45
8.3 PHAST model: Parameter default values.....	46
8.4 Opinion: Exposure via water ingestion at Camp Lejeune.....	49
9. SWIMMING POOL-RELATED CHEMICAL CONCENTRATIONS	51
9.1 Models for estimating swimming-related exposure	51
9.1.1 PHAST Surface water model for swimming.....	51
9.1.2 US EPA SWIMODEL model.....	54
9.1.3 Comparison of the PHAST Surface water model and the EPA SWIMODEL	56

9.2 SWIMODEL: Plaintiff- and chemical-specific modifications.....	57
9.3 Opinion: Vapor chemical concentrations at indoor pools at Camp Lejeune	58
10. CONSERVATIVE NATURE OF SELECTED MODEL INPUTS.....	60
10.1 Drinking water	60
10.2 Showering/bathing	61
10.3 Time away from residence	61
10.4 Appliances	61
10.5 Residence type.....	62
10.6 Exposure via indoor pools at Camp Lejeune	62
11. REBUTTAL TO EXPERT REPORT BY DR. REYNOLDS	63
11.1 General differences in approaches	63
11.1.1 <i>Exposure route differences</i>	63
11.1.2 <i>“Cumulative consumption” versus daily intake</i>	63
11.1.3 <i>Water ingestion rates</i>	64
11.2 Differences specific to Ms. Amsler.....	65
11.2.1 <i>Dates on Base</i>	65
11.2.2 <i>Water systems</i>	66
12. CONCLUSIONS.....	67
13. REFERENCES.....	70
APPENDIX 1: Curriculum Vitae for Judy S. LaKind, Ph.D.	76
APPENDIX 2: SHOWER model factors and options for modifications.....	112
APPENDIX 3: ATSDR SHOWER model evaluation and sensitivity analysis.....	116
APPENDIX 4: Supporting information	119
Appendix 4.1 Effect of utilities on PCE average daily exposure: SHOWER model results (partial reports)	120
Appendix 4.2 Effect of bathroom door open or closed on air concentrations of PCE (partial reports)	124
Appendix 4.3 Effect of outdoor air concentrations on indoor air concentrations of PCE (partial reports)	128

Appendix 4.4 Effect of shower duration on indoor air concentrations of PCE (partial reports)	134
APPENDIX 5: PHAST model parameters and options for modifications.....	139
APPENDIX 6: SWIMODEL: Hypothetical scenario reports	141

List of Figures

Figure 1. The 4-step risk assessment process (US EPA 2022)	14
Figure 2. Routes of exposure: chemicals in water.....	15
Figure 3. Layout of a house used by the SHOWER model based on selected input options (e.g., one bathroom, shower/tub combination).	36

List of Tables

Table 1. Monthly mean modeled water concentrations ($\mu\text{g/L}$) of PCE, TCE, DCE, VC, and benzene at Hadnot Point and PCE, DCE, TCE, and VC at Tarawa Terrace from May 1966 - June 1967.....	25
Table 2. Overall estimated mean concentrations ($\mu\text{g/L}$) of PCE, TCE, DCE, VC, and benzene at Hadnot Point and Tarawa Terrace over the time-period May 1966 - June 1967. These concentration data were used to estimate chemical exposures via drinking water (both locations) and residential dermal and inhalation exposures (Hadnot Point only) in this Report.	27
Table 3. Average water concentrations for Hadnot Point and Tarawa Terrace source water for two time-periods (Fall 1965-Summer 1966 and October 1965-June 1967). Values in bold were used to assess air chemical concentrations at an on-Base swimming pool (see Section 9).....	28
Table 4. Default body weights for the ATSDR SHOWER model.	34
Table 5. Range of average daily exposure PCE concentrations for four people in a residence with the clothes washer, dish washer, kitchen sink, and utility sink operating each day (“on” or default setting) and off.....	38
Table 6. Average daily exposures ($\mu\text{g/m}^3$) with the bathroom door open or closed (hypothetical PCE water concentration of 1 $\mu\text{g/L}$; four-person household).....	38
Table 7. SHOWER model results for highest exposed adult varying the ambient outdoor air concentrations of PCE (see Appendix 4.3 for supporting information).	40
Table 8. Average daily exposure PCE air concentrations for the fourth person to shower (in a four-person residence with all four people showering the same number of minutes).	41
Table 9. Average daily exposure concentrations for the sixth person (6 - < 11 years old) using a bath at Hadnot Point, Camp Lejeune (May 1966 - June 1967).	43

Table 10. Average daily exposures to chemicals from residential inhalation and dermal contact with water at Hadnot Point, Camp Lejeune (May 1966 - June 1967).	44
Table 11. Drinking water ingestion rates in the ATSDR PHAST drinking water ingestion model used in this Report.	47
Table 12. Default water ingestion rates from the US EPA’s Exposure Factors Handbook update. Reproduced from Table 3-1 in US EPA (2019).	48
Table 13. Daily intakes of chemicals from drinking water at Hadnot Point and Tarawa Terrace, Camp Lejeune (onsite from May 1966 - June 1967).	50
Table 14. PHAST model default incidental water ingestion intake rates associated with swimming.....	53
Table 15. PHAST model default total age-specific surface area associated with swimming.	53
Table 16. Henry’s law constants (atm·m ³ /mole) for chemicals of interest. Data source: US EPA Regional Screening Level data.....	58
Table 17. Vapor chemical concentrations in indoor pools at Hadnot Point (October 1965 – June 1967) and Tarawa Terrace (October 1965 – August 1966), Camp Lejeune. See Section 6.2 for an explanation of different time-periods.	59

ACRONYMS/ABBREVIATIONS

ADD: Average Daily Dose
AT: averaging time
ATSDR: Agency for Toxic Substances and Disease Registry
BW: body weight (kg)
C: contaminant air concentration ($\mu\text{g}/\text{m}^3$)
CASRN: Chemical Abstracts Service Registry Number
 cm^2 : square centimeter
CTE: central tendency exposure
 C_{vp} : vapor concentration
D: age-specific dose ($\text{mg}/\text{kg}\text{-day}$)
DAD: dermal absorbed dose ($\mu\text{g}/\text{kg}/\text{day}$)
 DA_{event} : absorbed dose per event ($\mu\text{g}/\text{cm}^2/\text{event}$)
DCE: *trans*-1,2-dichloroethylene
ED: exposure duration (year)
EDG: Exposure Data Guidance
EF (intermediate or chronic): exposure factor (unitless) = $(F \times \text{ED})/\text{AT}$
EPC: exposure point concentration, contaminant concentration (mg/L)
EV: event frequency
F: exposure frequency ($\text{day}/\text{week} \times \text{week}/\text{year}$)
ft: feet
hr: hour
ID: inhalation dose ($\mu\text{g}/\text{kg}/\text{day}$)
IR: intake rate of water (L/day) or air (m^3/day)
kg: kilogram
 K_{ow} : octanol-water partition coefficient
Kp: permeability coefficient (cm/hr)
L/min: liters air breathed per minute
L: liter
LADD: Lifetime Average Daily Dose
 m^3 : cubic meter
 $\text{mg}/\text{kg}\text{-day}$: milligram chemical per kilogram body weight per day
mg: milligram
N: number
NATA: National Air Toxics Assessment
NHANES: National Health and Nutrition Examination Survey
PCE: perchloroethylene
PHAST: Public Health Assessment Site Tool
RME: reasonable maximum exposure
SA: dermal surface area (cm^2)

SHOWER: Shower and Household Water-use Exposure
SWIMODEL: Swimmer Exposure Assessment Model
TCE: trichloroethylene
µg/L: microgram per liter
US DOJ: United States Department of Justice
US EPA: United States Environmental Protection Agency
US: United States
v: version
VC: vinyl chloride
WTP: water treatment plant

1. QUALIFICATIONS

I am Judy S. LaKind, MS, Ph.D. I am President of LaKind Associates, LLC, a human health risk science firm specializing in exposure science and the evaluation of scientific data for regulatory decision-making. I have over 30 years of experience in the fields of exposure science and risk assessment. I have expertise in assessing child and adult exposures to environmental chemicals, risk assessment and the implications of uncertainty in the risk assessment process, evaluation of data quality, use of environmental epidemiology research in public health decision-making, weighing potential risks and benefits related to chemical use, and systematic review. I am an adjunct Associate Professor in the Department of Epidemiology and Public Health, University of Maryland School of Medicine. I am also a Fellow by Courtesy, Department of Applied Mathematics and Statistics, The Johns Hopkins University.

I have a B.A. from The Johns Hopkins University, an MS from University of Wisconsin, Madison in geology and a Ph.D. from The Johns Hopkins University in environmental engineering. My dissertation research was on the kinetics of reductive dissolution of iron oxyhydroxides by phenolic compounds. In 1988, I was a scientist at the US Environmental Protection Agency (US EPA) where one of my main activities was reviewing environmental impact assessments produced under the National Environmental Policy Act. I was a scientist at consulting firms from 1988 to 1998 during which time my work focused on the conduct of exposure and risk assessments (e.g., field, computational, and communication aspects). From 1998 until the present, I have been a self-employed scientist specializing in exposure science, assessment of human health risks, biomonitoring, scientific analysis for regulatory support, and state-of-the-science and systematic reviews. I have extensive experience in speaking and publishing on exposure- and risk-related issues, including children's exposures to environmental chemicals, the implications of uncertainty in the risk assessment process, data quality, use of environmental epidemiology research in public health decision-making, weighing potential risks and benefits related to chemical use, the presence of environmental chemicals in human milk, and time-dependence and distributional analysis of exposure. I have evaluated the use of human health risk assessments in the development of water quality criteria and have critically analyzed the environmental fate, behavior, and bioavailability of pollutants in the context of setting regulatory criteria. I have developed risk assessments for a variety of urban industrial sites, military bases, and firing ranges, and have utilized state-of-the-science models for estimating blood lead levels in adults and children.

I have taught or co-taught courses on aquatic chemistry (Johns Hopkins University) and risk assessment (Johns Hopkins University, the University of Maryland School of Law and the University of Maryland, Baltimore County). I also co-taught a short course on biomonitoring and have developed an on-line course for continuing medical education credit on chemical exposures and health effects.

From 2008 to 2009, I served as Environmental Health Advisor to the Maryland Department of the Environment, Science Services Administration. One of my many activities was to develop standard operating procedures for developing risk-based fish consumption advisories.

I am a past President of the International Society of Exposure Science and served on the Executive Committee of the Exposure Specialty Section of the Society of Toxicology. I am also a member of the American Chemical Society, Environmental Division and the Society for Risk Analysis. I was a founding member of the International Society for Children's Health and the Environment (2009-2015). I am a former member of the Health Effects Institute Energy Research Committee. I previously served on the Board of the Coalition Against Childhood Lead Poisoning (with a term as president). I was also a member of Maryland's Children's Environmental Health and Protection Advisory Council, the Maryland Lead Poisoning Prevention Commission, the Maryland Pesticide Reporting and Information Workgroup, the Maryland Department of Health and Mental Hygiene Cancer Cluster Advisory Committee, the Health and Environmental Sciences Institute (HESI) RISK21 Advisory Board, and the World Health Organization (WHO) Survey Coordinating Committee for the WHO Global Survey of Human Milk for Persistent Organic Pollutants. I also served on the Institute of Medicine Committee on Blue Water Navy Vietnam Veterans and Agent Orange Exposure and the US Environmental Protection Agency Science Advisory Board Panel on Perchlorate - Approaches for Deriving Maximum Contaminant Level Goals for Drinking Water.

I have published over 100 papers in the peer-reviewed literature, and these have been cited over 5,600 times (h-index = 41). I serve on the editorial boards of *Environment International* (where I am Insights Editor) and the *Journal of Environmental Exposure Assessment*. I am a past editorial board member of the *International Journal of Environmental Research and Public Health* and the *Journal of Toxicology and Environmental Health* and past Associate Editor for the *Journal of Exposure Science and Environmental Epidemiology*. I have conducted peer review of manuscripts and reports for numerous scientific journals and governmental agencies.

My curriculum vitae is attached to this Report as Appendix 1.

I am compensated at a rate of \$575 per hour for my time consulting on these matters, preparing this Report, and, if called upon to do so, providing testimony in this case. I have not previously testified as an expert witness. The Materials Considered Appendix lists all the materials I considered in the preparation of this Report.

2. CASE OVERVIEW

This Report was prepared at the request of the United States Department of Justice (US DOJ). As part of my engagement in this case, I have been asked to review materials relevant to the *Amsler v. United States* case and to develop opinions regarding Ms. Amsler's exposure to five chemicals in treated water used by people at Marine Corps Base Camp Lejeune, North Carolina (referred to in this Report as "Camp Lejeune" or "Base"): perchloroethylene (PCE, tetrachloroethylene, CASRN: 127-18-4), trichloroethylene (TCE, CASRN: 79-01-6), *trans*-1,2-dichloroethylene (DCE, CASRN: 156-60-5), vinyl chloride (VC, CASRN: 75-01-4), and benzene (CASRN: 71-43-2). These five chemicals are referred to in this Report as "chemicals of interest." My overall opinion is based on results from the modeling of exposures.

2.1 Summary of opinion

In this Report, I use three models to estimate Ms. Amsler's past exposures to the Agency for Toxic Substances and Disease Registry's (ATSDR) modeled monthly concentration estimates of PCE, TCE, DCE, VC, and benzene in water at Camp Lejeune: one for the dermal/inhalation routes of exposure, one for the oral route of exposure (water ingestion), and one for air concentrations at swimming pools. Based on my review and analysis of the information produced in this case, as well as my exposure and risk assessment education, training, and experience, I have formed the following opinion. My opinion herein is held to a reasonable degree of scientific certainty considering my use of ATSDR's modeled chemical concentrations in water. I reserve the right to modify or supplement my opinion if additional information is made available to me, including information from reports and testimony of other experts in this matter.

SUMMARY OF OPINION

People living and working at Camp Lejeune from the 1950's to the 1980's may have been exposed to PCE, TCE, DCE, VC, and/or benzene due to the presence of these chemicals in finished water at Camp Lejeune. Finished water is "[w]ater that has passed through a water treatment plant. All the treatment processes are completed or finished. This water is the product from the water treatment plant and is ready to be delivered to consumers" (<https://owp.csus.edu/glossary/finished-water.php>). In this Report, either "water" or "finished water" is used to indicate the water used in residences and swimming pools or for drinking water at Camp Lejeune.

Note that in this Report, I use mean monthly chemical concentration estimates modeled by ATSDR, who state that their modeled data are for finished water at Camp Lejeune (Maslia et al. 2007, 2013). In Dr. Alexandros Spiliotopoulos' Expert Report (2024, pgs. 68-69), he states that "For Hadnot Point, as with Tarawa Terrace, ATSDR assumed concentrations of contaminants in the influent to the WTP [water treatment plant] were equal to the concentrations of contaminants in the 'finished water' that was delivered to consumers...This assumption is incorrect, as treatment of the influent to the treatment plant resulted in evaporative and other losses, reducing

contaminant concentrations in the ‘finished’ water.” Based on this opinion, the concentrations of chemicals of interest used in this Report, derived from ATSDR modeling, would be an overestimate of actual chemical concentrations in water used by people at Camp Lejeune¹.

The routes of exposure for people living and working at Camp Lejeune could have included:

- Ingestion (for example, drinking the water, using the water for cooking, drinking small amounts of water while swimming)
- Inhalation (breathing the chemicals that volatilized from the finished water during activities such as showering, bathing, swimming, or having/using appliances such as washing machines)
- Skin contact (dermal exposure from contacting the finished water during activities such as showering, bathing, hand washing, or swimming)

There were very few measurements made of chemicals in the water at Camp Lejeune during the overall time-period of interest (1953-1987; <https://www.navy.mil/Camp-Lejeune-Justice-Act-Claims/Claim-Eligibility/>); measurements of the chemicals of interest in the water began in the 1980’s (Maslia et al. 2007, 2013). However, ATSDR estimated mean monthly water concentrations for the time-period of interest (Maslia et al. 2007, 2013). The US DOJ requested that I rely on ATSDR’s mean monthly chemical concentration data for estimating exposures at Camp Lejeune as these are the values reported in the Expert Report of Morris L. Maslia, P.E. (2024).

Similarly, no measurements of chemicals in indoor air at Camp Lejeune were identified for the time-period of interest. Therefore, a model that can estimate indoor air concentrations based on chemical concentrations in water was used in this Report. Note that in this Report, the modeled indoor air concentrations are from use of finished water in the residence and not from vapor intrusion; the potential presence of chemicals in indoor air from vapor intrusion is not addressed in this Report.

Finally, I did not identify detailed contemporaneous documentation related to daily behaviors and activities for people on Base decades ago. Information from various sources - including Ms. Amsler’s deposition - was used to describe behaviors and activities leading to likely contact with chemicals in water and air.

These information sources were used in conjunction with various exposure models to estimate exposures to people at Camp Lejeune (see Section 5.1 for additional information). The exposure models used in this Report were developed by the US Environmental Protection Agency

¹ Drs. Hennessey and Spiliotopoulos explain in their Expert Reports that ATSDR’s modeled exposure estimates are unreliable and likely biased high as a result of several conservative assumptions used in ATSDR’s modeling due to limited historical data available about the start and the extent of contaminant source releases, as well as the absence of concentration data prior to 1980 (Expert Reports of Drs. Hennessey [2024, pgs. 5-35 – 5-38] and Spiliotopoulos [2024, pgs. 36-45, 70-87]).

(US EPA) and ATSDR and the underlying approaches (described in Sections 7, 8, and 9) are well-established and have been used in assessments of ingestion, inhalation, and dermal exposures for many years by regulatory agencies, consultants, and academicians. The models were employed to estimate ranges of possible exposures that reflect the time that Ms. Amsler was on Base and her general likely behaviors and activities on Base. The method used to assess exposures at swimming pools relies on a model (Henry's Law) that is a well-established approach to estimating the amount of a chemical in air using the amount of the chemical in water. Henry's law, formulated in the early 1800's, describes the relationship between the amount of a chemical in water to the amount in air.

Using these existing data and models, I was able to draw conclusions about Ms. Amsler's likely exposures to PCE, TCE, DCE, VC, and benzene to a reasonable degree of scientific certainty, considering my use of ATSDR's modeled chemical concentrations in water, as detailed in this Report.

It is important to note that, where possible and scientifically supportable, conservative assumptions were used for determining model inputs. Conservative assumptions are those that tend to produce higher estimates of exposure. They are used to avoid underestimating exposures. In other words, conservative assumptions produce “[a]n estimate that tends to err on the side of caution or gives a 'worst case scenario'” and are “[o]ften used in risk assessment to ensure that as much risk as possible is taken into account” (https://www.efsa.europa.eu/en/glossary/conservative-assumption#:~:text=Description:,possible%20is%20taken%20into%20account.)).

Specific aspects of this Report that contribute to the conservative nature of the exposure estimates are described throughout the Report and summarized in Section 10.

Therefore, Ms. Amsler's actual exposures are unlikely to be higher than the exposure estimates produced by these models. These exposure estimates can be used in risk assessments to determine whether people who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in similar areas, and engaged in similar activities had an increased risk of disease (this is addressed in the Expert Report of Dr. Lisa Bailey for Karen Amsler).

3. METHODOLOGY

The opinions in this Report are based on my training and experience in exposure science and on a review of documents available as of the date of this Report. Specific documents that I have reviewed are presented in the Materials Considered Appendix. In addition, there are numerous documents that I have reviewed in my professional history that are not referenced specifically, but that have supported my understanding of this case.

I have reviewed the Expert Reports of Dr. Remy Hennet (2024) and Dr. Spiliotopoulos (2024) regarding information related to groundwater, contaminant fate and transport, and water distribution modeling for Camp Lejeune; Ms. Amsler's deposition transcript; and certain records of the Plaintiff. I have also reviewed the ATSDR's water modeling reports for Camp Lejeune and housing and other drawings for Camp Lejeune.

The specific activities I performed for my evaluation are briefly stated below:

- I reviewed the Plaintiff's deposition transcript and related documents (these documents are included in the Materials Considered Appendix).
- I reviewed the ATSDR's estimated monthly mean concentrations in finished water from the Hadnot Point and Tarawa Terrace water systems, specifically modeled concentrations for TCE, PCE, DCE, VC, and benzene.
- I applied an exposure science method to conduct a residential exposure assessment for dermal contact with – and inhalation of – chemicals of interest for a population with residential parameters (e.g., time at Camp Lejeune, shower/bath duration) substantially similar to Ms. Amsler using the ATSDR's Shower and Household Water-use Exposure (SHOWER) model.
- I applied a standard exposure science method to conduct a drinking water exposure assessment for people with parameters similar to Ms. Amsler (e.g., time at Camp Lejeune, drinking water consumption rates) using the ATSDR Public Health Assessment Site Tool (PHAST) for drinking water ingestion.
- I applied an exposure science method to conduct an assessment of indoor swimming pool vapor concentrations for people with parameters similar to Ms. Amsler (e.g., time at Camp Lejeune) using the US EPA SWIMODEL (Swimmer Exposure Assessment Model).

The following sections provide more information about methodologies for conducting exposure assessments and specifically for conducting exposure assessments for people living or working at Camp Lejeune.

4. BACKGROUND ON CHEMICAL EXPOSURE ASSESSMENT

The chemical risk assessment approach currently in use was initially put forth several decades ago (NRC 1983). The purpose was to provide a structure for estimating the possible health effects of chemical exposures to humans. Risk assessment is comprised of four basic elements as shown in **Figure 1** (US EPA 2022).

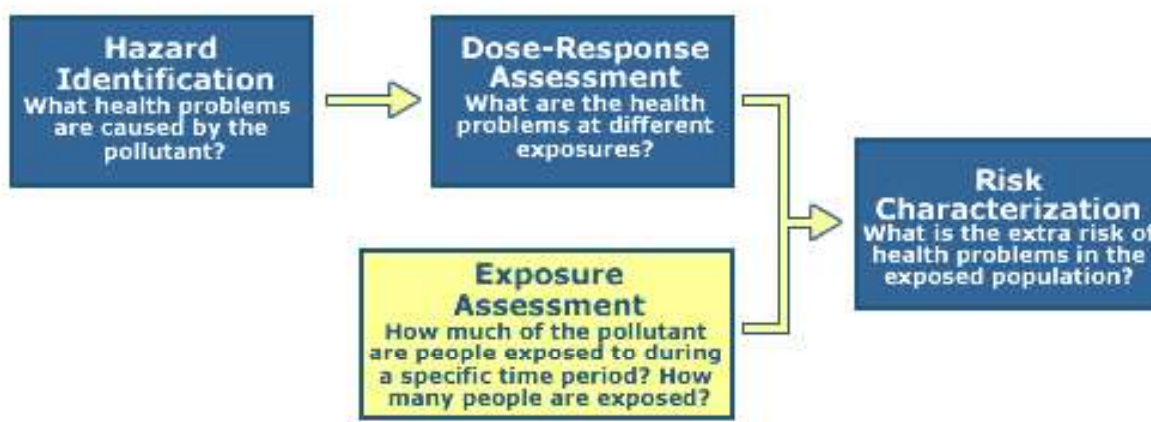


Figure 1. The 4-step risk assessment process (US EPA 2022)

One of the four basic elements is exposure assessment, defined as "[t]he process of estimating or measuring the magnitude, frequency, and duration of exposure to an agent, along with the number and characteristics of the population exposed. Ideally, it describes the sources, routes, pathways, and uncertainty in the assessment" (IPCS 2004, pg. 101).

Simply put, in conducting an exposure assessment, we seek to understand how much of a chemical people are exposed to during a specific time-period (e.g., a period of days, weeks, months, or years). When combined with information about a chemical's toxicity, the health risks associated with exposure to one or more chemicals can be assessed, or "characterized." Therefore, the assessment of human exposure is an essential component of any risk assessment.

When considering exposure to chemicals from water, three routes of exposure are evaluated: oral, inhalation, and dermal (**Figure 2**).

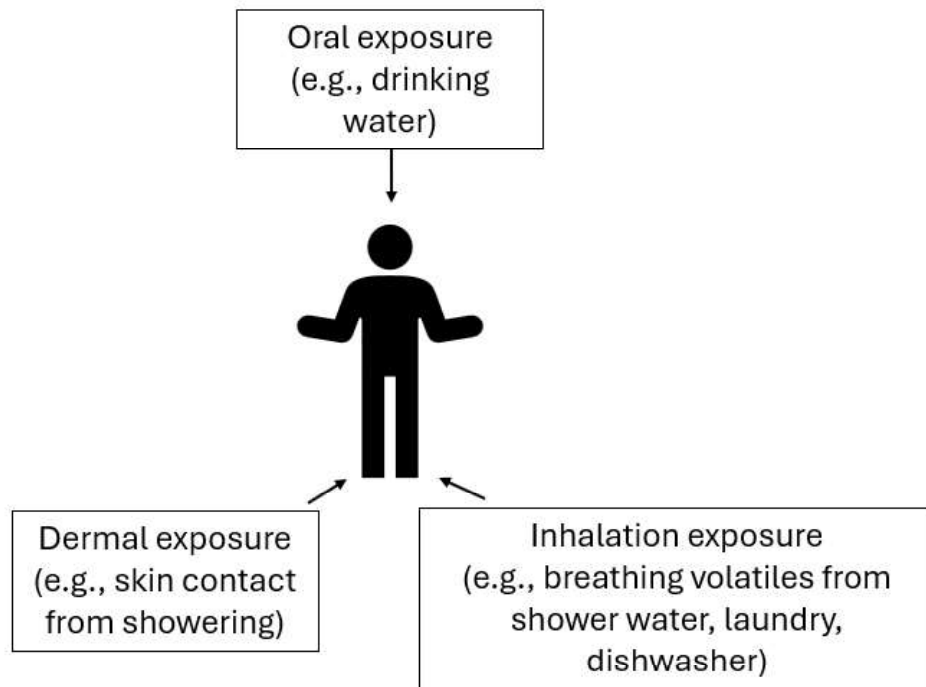


Figure 2. Routes of exposure: chemicals in water

To assess human exposures to chemicals, one needs information on chemical concentrations in environmental media such as water and air, on human behaviors, and on aspects of the environment in which people reside. These can include data on the duration of exposure (e.g., how many years a person comes into contact with the air or water), the frequency of exposure (e.g., how many days per week, hours per day), the volume of water consumed (how many liters per day), and many other factors, as well. The exposure assessor obtains site- and population-specific information where possible. When this information is not available, exposure assessors rely on information obtained from sources such as general population studies, governmental data, and scientific literature. We then make determinations regarding how to use that information to conduct site-specific exposure assessments.

The types of information described in the preceding paragraph are used as inputs to models to derive quantitative estimates of exposure. These estimates are generally expressed in units of milligram chemical per kilogram body weight per day, or mg/kg-day. The quantitative estimates describe how much of a chemical enters the body per day. A model can be a simple equation requiring at most a hand calculator or can be very complex.

In this Report, various parameters needed to estimate past human exposures to chemicals at Camp Lejeune are described and numerical values are assigned to these parameters. These

parameters are more fully described in Sections 7, 8, and 9 but can include, for example, the number of minutes spent showering or bathing each day, the size of the bathroom and living quarters, and the volume of daily water consumption.

It is important to recognize that model inputs are derived from different sources and can include well-supported site-specific values, “default” values, and values based on best professional judgment. Well-supported site-specific data are generally the preferred source of information for an exposure assessment. Examples could include information collected at – or close to – the time that a plaintiff was on Base. The information could be obtained from interviews or diaries, for example, and could be related to activities such as daily shower durations or exact amounts of daily water consumption. Unfortunately, in studies of past exposures, it is often the case that these kinds of data are not available.

A standard practice for assigning a parameter value in the case of missing or limited information is to use a default value (ATSDR 2022a; Health Canada 1999; US EPA 2011, pg. 1-16). The European Food Safety Authority describes the use of default values as follows (EFSA 2012; pg. 2): “A number of assumptions and default values are usually applied at the various steps of the risk assessment process. These can...compensate for the absence of data, in which case the risk assessor may have to refer to default values to be able to perform the assessment. These default values should be scientifically justified and, where possible, be based on existing data and represent typical values for the missing parameter.”

For the exposure assessments in this Report, various default values are used. These values are often based on data from the published literature for the general population (e.g., body weights; body surface area) or other representative types of data. For certain parameters, both average values and more conservative (e.g., 95th percentiles) values are used in the models (see Section 5.2).

Some default values in this Report were obtained from the US EPA Exposure Factors Handbook (US EPA 2011). The Handbook “...has become a key source of exposure factor information and has served to promote consistency among risk assessments conducted by the [Environmental Protection] Agency and others. It provides a unique synthesis of exposure factor data for the US population that is unavailable in any other single source. It has been cited in numerous EPA Reports and peer-reviewed publications... The Exposure Factors Handbook has also been widely used by researchers outside the United States” (Phillips and Moya 2011, pg. 13). Most of the Exposure Factors Handbook data come from studies of the general population (e.g., the National Health and Nutrition Examination Survey [NHANES]) or from studies on sample populations that focus on specific groups (e.g., children). The Exposure Factors Handbook was reviewed internally by individuals within the US EPA and also underwent peer review by an external panel of experts. Thus, the default values from this source are scientifically well-supported and appropriate for use in exposure assessment. Default values from the Exposure Factors Handbook can also be supplemented with site-specific information, if available.

The most recent complete compilation of default exposure values is the 2011 Exposure Factors Handbook (US EPA 2011). Since that time, EPA has updated certain chapters and made them available online (<https://www.epa.gov/expobox/about-exposure-factors-handbook>).

In the absence of well-supported site- and plaintiff-specific data or default values, another approach to addressing missing or limited data is to use professional judgment. Professional judgment is an accepted aspect of risk assessment. For example, in the US EPA's Guidelines for Carcinogen Risk Assessment (2005, pgs. 2-51), EPA notes that "Choosing a descriptor [for weight of evidence for carcinogenic potential] is a matter of judgment and cannot be reduced to a formula." Further, the US EPA (1992a, pg. 92) has stated that "professional judgment comes into play in virtually every aspect of the exposure assessment process, from defining the appropriate exposure scenarios, to selecting the proper environmental fate models, to determining representative environmental conditions, etc...". As noted by the US Army Corps of Engineers (2010, pg.1-5): "...there will be unavoidable data gaps and uncertainties where scientific and professional judgment is needed to predict or infer certain outcomes under certain scientific principles (Federal Focus Inc. 1994). The application of such judgment requires that the risk assessor provide the rationale or basis for the judgment."

Use of professional judgment is not unique to risk assessment but is used in various scientific disciplines. For example, professional judgment has been described as "one of the most important aspects of evidence-based practice" in psychology (Wilczynski 2017, pg. 65): "Good professional judgment is based on accessing all relevant information about the best available evidence and the clients (target/stakeholder/ leader) as well as the context, so the best clinical decision is made." In the field of biology, "[i]t has long been recognized that there are relatively few absolutes in biology, and that any interpretation of observed phenomena must be tempered by sound scientific judgment" (Weed 2007, pg. 138.) As noted by Weed (2007, pg. 139), "science would not be science without judgment."

For the exposure assessments in this Report, values derived from professional judgment are based on a combination of (i) information derived from plaintiff depositions, (ii) military and other expert Reports, (iii) the peer-reviewed published literature, and (iv) experience and education. While the information from these sources may not be specific to the plaintiff or to Camp Lejeune, for it to be used, it should be considered relevant to one or both. Where necessary and scientifically supportable, values based on professional judgment were selected to be able to derive both typical and conservative (in other words, designed to avoid under-estimating) estimates of exposure.

In summary, exposure assessment is an essential component of risk assessment and methods for estimating human exposures to chemicals have been used by exposure and risk assessors for several decades. Despite advances in exposure assessment methods, uncertainties and limitations are an inherent part of the exposure assessment process. Exposure assessments require assumptions because site-specific information is often unavailable, and individuals may

not be able to accurately recall (or may not know) exposure-related information. Further, exposure varies from day to day (e.g., shower duration, amount of water consumed, water sources and concentrations, etc.) and, in particular for retrospective assessments, data describing this variability are generally not available. Because of this, where possible and where scientifically supportable, I have chosen to utilize values and assumptions for the exposure assessment in this Report that would tend to overestimate exposure (i.e., provide conservative exposure estimates).

5. CONCEPTS AND TERMINOLOGY FOR THE EXPOSURE ASSESSMENT IN THIS REPORT

As with any scientific discipline, exposure science is replete with concepts and terminology that may be unfamiliar to those who are not experts in the field. I describe here several concepts and words/phrases that are used throughout the Report and that may be unfamiliar to the reader.

5.1 Concepts

Plaintiff activities and behaviors: The exposure assessment in this Report is intended to capture exposures experienced by people residing and/or working at Camp Lejeune during a time-period specific to the Plaintiff's actual time on Base. A necessary component of this assessment is an understanding of a plaintiff's activities and behaviors (e.g., amount of water consumed, time spent in the shower or bath). The exposure assessment in this Report is not a perfectly accurate representation of exposure to a specific individual because Plaintiff-specific information on activities and behaviors necessary to develop such a representation is not available. For example, no contemporaneous documentation (e.g., diaries) describing day-to-day activities was identified. However, exposures can still be assessed by making assumptions derived from information from depositions, other sources of information related to the United States population, the military in general, Camp Lejeune specifically, and my best professional judgment. These various sources of information are used to gain a better understanding of data uncertainties (e.g., lack of data from the time-period of interest, uncertain recall) and variability (e.g., spatial and temporal changes in a person's activities and other factors) for the exposure parameters used in the exposure assessment.

Models: Two types of models are referenced in this Report: models used to estimate concentrations of chemical of interest in the water at Camp Lejeune and models used to estimate plaintiff exposures.

- The first type of model (i.e., models used to estimate chemical concentrations in water) is referred to as water modeling, which ATSDR describes as a "...scientific method that helps ATSDR estimate past water-system conditions that no longer exist today" (https://www.atsdr.cdc.gov/camp-lejeune/php/water-modeling/meetings-faq.html?CDC_AAref_Val=https://www.atsdr.cdc.gov/sites/lejeune/water-modeling-meetings-and-faqs.html). In this Report, I use the results from ATSDR models to describe concentrations of chemicals of interest in water from the Hadnot Point and Tarawa Terrace water systems. The US DOJ requested that I rely on ATSDR's mean monthly chemical concentration data for estimating exposures at Camp Lejeune as these are the values reported in the Expert Report of Morris L. Maslia, P.E. (2024). Details regarding water modeling are provided in a separate Expert Report by Dr. Spiliotopoulos (2024) and are not described here.
- The second type of model (i.e., models used to estimate plaintiff exposures) is central to this Report. Three different exposure models are used. These models: (i) estimate

human exposures to chemicals from consumption of drinking water, (ii) estimate human exposures to chemicals from inhalation of volatiles from water and dermal contact with water in residential settings, and (iii) estimate chemical concentrations in air at swimming pools.

The basic models (i.e., equations) for estimating inhalation, dermal, and oral exposures to chemicals are well-established and have been used by various agencies, consultants, and academicians (e.g., ATSDR 2023a; Baier-Anderson et al. 2006; Chowdhury 2015; EarthCon 2019; Health Canada 2021; Huerta et al. 2023; Khan et al. 2024; Lowe and Jamall 1994; Oregon Department of Environmental Quality 2010; Ramirez-Andreotta et al. 2013; Salhotra 2011; USEPA 1989, 1992b, 2009). The model used to estimate chemical concentrations in air at swimming pools is based on a well-established approach to estimating the amount of a chemical in air from the amount of the chemical in water (Henry's Law, formulated in the early 1800's) (Sander 2023).

For the models used to estimate human exposures to chemicals of interest, it is important to note that the estimates are for a single 24-hour period. The process of converting a one-day exposure to an estimate of long-term exposure - and the results of that process for individual plaintiffs - are described in a separate Expert Report (Expert Report of Dr. Lisa Bailey for Karen Amsler).

Exposure pathways: The water at Camp Lejeune was used for a variety of purposes including drinking, use for food preparation, appliance use such as laundry and dishwashing, and showering and bathing as well as various occupational, recreational, and cleaning purposes. For use as drinking water, in this Report I consider the total amount of water that may have been consumed over the course of a 24-hour period. For dermal and inhalation contact, I consider exposures to the chemicals of interest over a 24-hour period from using water in a residence (e.g., showering, sink use) and laundry/dishwashing/kitchen-related activities. I recognize that other on-Base activities could have resulted in dermal or inhalation exposures. For example, these exposures could have occurred during mess hall activities, swimming, or car washing. In the case of the use of the Base swimming pool(s), for plaintiffs who specifically noted the use of a pool, indoor air concentrations were modeled and results included in the plaintiff Report. Ms. Amsler reported that she swam recreationally on Base (Karen Amsler April 16, 2024 Deposition Transcript, pgs. 97-98). Therefore, I modeled swimming pool air concentrations of chemicals of interest for the time Ms. Amsler was at Camp Lejeune. For outdoor activities such as car washing, in my professional judgment, inhalation exposures would be minimal due to dilution with the surrounding air. Dermal exposure would similarly likely be low due to off-gassing of volatile chemicals and minimal amounts of exposed skin surface area.

5.2 Terminology

Dose: This is the amount of a chemical that is taken into a person's body. Dose is usually estimated for a certain amount of time (for example, how much of a chemical enters the body in a day). The amount that enters the body is also adjusted for the body weight of the person (i.e., the

amount of a chemical that enters the body for each kilogram of body weight). Thus, the units to describe dose are milligram of a chemical per kilogram body weight per day, or mg/kg-day.

Extent of exposure: In the human exposure models used in this Report, there are options to assess two types of exposure: central tendency exposure (CTE) and reasonable maximum exposure (RME). These are defined by ATSDR as follows (<https://www.atsdr.cdc.gov/pha-guidance/resources/ATSDR-EDG-Body-Weight-508.pdf>):

Central Tendency Exposure (CTE): CTE refers to people who have average or typical intake factors.

Reasonable Maximum Exposure (RME): RME refers to people at the upper end of the exposure distribution (approximately the 95th percentile). The RME scenario assesses exposures that are higher than average but still within a realistic exposure range.

The model used to estimate exposure to chemicals of interest via drinking the water produces both CTE and RME results and these are included in this Report. The model used to estimate residential exposure to chemicals via inhalation of chemicals volatilized from the water and from water contact with skin has the option of assessing CTE and RME results, but for this Report, site-specific values are used.

Intake rate: Intake rate is defined by ATSDR (https://www.atsdr.cdc.gov/pha-guidance/glossary/index.html#l_definitions) as: “The amount of a contaminated medium to which a person is exposed during a specified period of time. The amount of water, soil, and food ingested on a daily basis; the amount of air inhaled; or the amount of water or soil that a person may contact through dermal exposures are all examples of intake rates.” If the medium is water, then the drinking water intake rate is expressed in units of liters per day (L/day). If the medium is air, then the air inhalation intake rate is expressed in units of cubic meters of air per day (m³/day). Intake rates refer to the medium (e.g., air, water) as opposed to dose which refers to intake of the chemical of interest.

Exposure Factor: The Exposure Factor, or EF, is “[a]n expression of how often (frequency) and how long (duration) a person may be contacting a substance in the environment. In many instances, the exposure factor (EF) will equal 1, representing a daily exposure to the contaminant. However, some exposures may occur on an intermittent or irregular basis. For these exposures, an EF can be used to average the dose over the exposure interval” (ATSDR 2018, pg. 4). The equation for EF (unitless) is $(F [\text{frequency}] \times ED [\text{exposure duration}]) / AT [\text{averaging time}]$. In this Report, I estimate exposures for a single day, and do not consider frequency, duration, or averaging time. These parameters are addressed in a separate expert witness report (Expert Report of Dr. Lisa Bailey for Karen Amsler). For a single day exposure, the parameter EF reduces to a value of 1.

Oral exposure: Oral – or ingestion - exposure occurs from consumption of contaminants in, for example, food or water. In this Report, I estimate the Plaintiff’s oral exposures from ingestion of finished water.

Dermal exposure: This Report includes consideration of dermal exposure, or exposure from skin contact with the chemicals of interest in the water. The primary equations for estimating dermal exposure are provided in a later chapter of this Report. These equations are more complex than the equations for exposure via water ingestion or for inhalation of volatiles from the air. This is because dermal exposure assessment requires information not only on the amount of skin contact that occurs, but also on the extent to which the chemical is absorbed by the skin. The reader is referred to the references in the relevant chapters in this Report for information on additional equations and equation parameters. For dermal exposure, the dose is described as the dermally absorbed dose, or the dose of the chemical absorbed through the skin and into the body (ATSDR 2023a). This dose can be converted to what is referred to as an “administered dose.” For the chemicals of interest in this Report, the dermally absorbed dose and the administered dose are equivalent. As stated by ATSDR (2023a, pg. 7): “For most chemicals, the absorbed dermal dose is the same as the oral administered dose because we assume 100% of the chemical is absorbed through the GI tract, thus [the gastrointestinal absorption factor] equals 1. Therefore, no adjustment from absorbed dermal dose to administered oral dose is needed for VOCs [volatile organic compounds], SVOCs [semi-volatile organic compounds], pesticides, PAHs [polycyclic aromatic hydrocarbons], and PCBs [polychlorinated biphenyls]. For these chemicals the absorbed dose calculated from dermal uptake is also an administered dose.”

Inhalation exposure associated with finished water: While the available ATSDR estimated mean monthly chemical concentration data are for water, the chemicals of interest are volatile, meaning that they can evaporate from the water and enter the air. Therefore, this Report includes an assessment of inhalation of air containing chemicals that have volatilized from the finished water. The concentrations in air are modeled with approaches described in later sections of this Report.

6. CHEMICAL CONCENTRATION INFORMATION FOR CAMP LEJEUNE

In the following sections of this Report, I describe three models that I used to estimate Ms. Amsler's past exposures to PCE, TCE, DCE, VC, and benzene in water at Camp Lejeune: one for the dermal/inhalation routes of exposure (SHOWER model, Section 7), one for the oral route of exposure (PHAST, Section 8), and one for air concentrations at swimming pools (SWIMODEL, Section 9). In Sections 7, 8, and 9, I describe the models themselves as well as the available information used to select values for the model parameters. Finally, I describe the results from each of these models. Where Plaintiff-specific information was available, this is shown in **bold font**.

The exposure models in this Report require information on concentrations of the chemicals of interest in water. In the following sections, I describe the sources of the water concentration data at Camp Lejeune (Section 6.1) and the water concentrations of PCE, TCE, DCE, VC, and benzene used in this Report (Section 6.2).

6.1 Background on available chemical concentration data for water at Camp Lejeune

Chemical concentrations in water (and in air from volatilization of chemicals from water to air) can be determined from measuring those chemicals in samples of the water. In the case of past exposures for which few or no measurements of chemicals were obtained, models can be used to estimate water concentrations. Modeling the chemical concentrations in water is often the only approach that can yield the information needed to conduct an exposure assessment.

There are a limited number of historical measurements of PCE, TCE, DCE, VC, and benzene in the water in the impacted areas of Camp Lejeune (Maslia et al. 2016) and these measurements were not made until the 1980's. Reconstructions (or modeling) of estimated mean monthly water concentrations of these chemicals were done by ATSDR. ATSDR modeled monthly average concentrations of PCE, TCE, DCE, VC, and benzene for the years of interest (1953-1987). They provided the results in publicly available reports (Maslia et al. 2007, 2013). These reports include modeled monthly mean concentrations of the chemicals of interest in the areas of Camp Lejeune served by the Tarawa Terrace and Hadnot Point water systems (the water systems that are the focus of this Report).

ATSDR reconstructed monthly mean concentration values (Maslia et al. 2016) for finished water from January 1952 to May 1996 for Hadnot Point (Maslia et al. 2013) and from January 1952 to February 1987 for Tarawa Terrace (Maslia et al. 2007). I relied on estimated mean monthly concentrations of PCE, TCE, DCE, VC, and benzene (benzene for Hadnot Point only) in water for Hadnot Point² and Tarawa Terrace extracted and compiled into Excel spreadsheets by S.S. Papadopoulos & Associates, Inc. It is my understanding that the data were extracted from the ATSDR Reports (Maslia et al. 2013, Appendix A7 and Maslia et al. 2007, Appendix A2,

² The Maslia et al. (2013) report refers to this as the Hadnot Point–Holcomb Boulevard study area. For detailed information on the locations of interest, see the Expert Report by Dr. Spiliotopoulos (2024).

respectively). These compiled data were used as the basis for the analyses in this Report. Reconstructed concentration minima for all chemicals were equal to 0 µg/L (micrograms per liter). While chemical concentrations in the water could have varied from day to day, only monthly average modeled concentrations were available; these were used as the basis for determining overall average water concentrations for the time the Plaintiff spent on Base.

According to the Expert Report of Dr. Spiliotopoulos (pgs. 68-69): “For Hadnot Point, as with Tarawa Terrace, ATSDR assumed concentrations of contaminants in the influent to the WTP were equal to the concentrations of contaminants in the ‘finished water’ that was delivered to consumers...This assumption is incorrect, as treatment of the influent to the treatment plant resulted in evaporative and other losses, reducing contaminant concentrations in the ‘finished’ water.” *Based on the information in this Expert Report, the ATSDR concentrations described in this Report, as well as the associated estimates of Plaintiff exposure, would be overly conservative (too high).*

6.2 Water concentration data relevant to Ms. Amsler

Assumptions for assessment of plaintiff-specific time on Base are:

- (i) If a plaintiff was on Base for part of the calendar month, I assumed that the plaintiff was there for the entire month (the exception to this was if the plaintiff was only on Base for one day for that month).
- (ii) Plaintiffs may have been off-Base for part of their time at Camp Lejeune (e.g., leave, weekends away, time spent on parts of the Base where water was not impacted). Unless they were off Base for at least one calendar month (e.g., January 1 to January 31) and the exact dates were known, it was assumed that they were on Base and exposed to the chemicals of interest for the entire time-period.

Water source(s) and time-periods for residential dermal and inhalation exposure: **Ms. Amsler moved on Base from Jacksonville, NC around May 1966 (Karen Amsler April 16, 2024 Deposition Transcript, pg. 96) and left Camp Lejeune in June 1967 when her father was deployed to Vietnam (Karen Amsler April 16, 2024 Deposition Transcript, pgs. 94-95). Ms. Amsler did not recall the exact month that her family moved from Jacksonville to Camp Lejeune (“May or June of 1966” [Karen Amsler April 16, 2024 Deposition Transcript, pg. 96]). For the purposes of this Report, I have assumed that their time on Base began in May 1966 (00284_AMSLER_0000007920-7922; dates of residence on Base from May 25, 1966 – June 5, 1967).**

Ms. Amsler’s family lived in a house at 2517 St. Mary’s Drive, Camp Lejeune, NC, located on Paradise Point, which prior to 1972 was served by the Hadnot Point water system (https://www.atsdr.cdc.gov/camp-lejeune/faq/?CDC_AAref_Val=https://www.atsdr.cdc.gov/sites/lejeune/faq_water.html).

Ms. Amsler also attended school on Base at the Stone Street Elementary School during the 1966-1967 school year (Karen Amsler April 16, 2024 Deposition Transcript pgs. 97-98; 00284_AMSLER_0000007966-0000007970). The Stone Street Elementary School was located in the Hadnot Point area and was supplied by the Hadnot Point water system (Expert Report of Dr. Jay Brigham, Table 4, pgs. 89-91).

I therefore used Hadnot Point water concentrations for the time-period May 1966 - June 1967 to assess residential dermal and inhalation exposures associated with Ms. Amsler's time living on Base.

Water source(s) and time-periods for exposure via water ingestion: **Based on Ms. Amsler's place of residence and her school location, exposure to water from the Hadnot Point water system via ingestion is likely because, as noted above, Ms. Amsler both lived and attended school on Base in the Hadnot Point area. However, I assumed she may have also visited and consumed water at Tarawa Terrace.** I have, therefore, modeled her exposure via drinking water consumption using data for both the Hadnot Point and Tarawa Terrace water systems for the time-period May 1966 - June 1967.

The monthly mean modeled values for Hadnot Point and Tarawa Terrace used for estimating the overall mean water concentrations for Ms. Amsler for exposure via drinking and residential dermal and inhalation exposures are shown in **Table 1**. To estimate residential dermal and inhalation exposures and drinking water exposures for those at Camp Lejeune during this time-period, the overall mean value for each chemical at each location is used (**Table 2**). This is consistent with ATSDR's use of a three-year rolling average for estimating exposures in its Camp Lejeune Public Health Assessment (ATSDR 2017). Estimation of the average dose is also consistent with the risk assessment paradigm that includes the use of an Average Daily Dose (ADD) or Lifetime Average Daily Dose (LADD) (US EPA 1992a). Further, the US DOJ requested that I rely on ATSDR's mean monthly chemical concentration data for estimating exposures at Camp Lejeune as these are the values reported in the Expert Report of Morris L. Maslia, P.E. (2024).

Table 1. Monthly mean modeled water concentrations ($\mu\text{g/L}$) of PCE, TCE, DCE, VC, and benzene at Hadnot Point and PCE, DCE, TCE, and VC at Tarawa Terrace from May 1966 - June 1967.

Hadnot Point	Water concentrations ($\mu\text{g/L}$)				
Month/Year	PCE	TCE	DCE	VC	Benzene
May-66	0	21	0	0	1
Jun-66	0	21	0	0	1
Jul-66	0	21	0	0	1
Aug-66	0	26	0	0	1
Sep-66	0	23	0	0	1
Oct-66	0	25	0	0	1

Nov-66	0	26	0	0	1
Dec-66	0	26	0	0	1
Jan-67	0	25	0	0	1
Feb-67	0	26	0	0	1
Mar-67	0	23	0	0	1
Apr-67	0	30	0	0	1
May-67	0	24	0	0	1
Jun-67	0	24	0	0	1
Tarawa Terrace*	Water concentrations (µg/L)				
Month/Year	PCE	DCE	TCE	VC	
May 1966	52.70	6.38	2.15	3.27	
June 1966	52.63	6.35	2.15	3.25	
July 1966	52.54	6.33	2.14	3.23	
Aug 1966	52.46	6.30	2.14	3.21	
Sept 1966	52.38	6.27	2.13	3.20	
Oct 1966	52.28	6.25	2.13	3.18	
Nov 1966	52.20	6.22	2.12	3.16	
Dec 1966	52.11	6.19	2.12	3.14	
Jan 1967	52.02	6.17	2.11	3.13	
Feb 1967	51.90	6.14	2.11	3.11	
Mar 1967	51.76	6.11	2.10	3.09	
Apr 1967	51.61	6.08	2.09	3.07	
May 1967	51.43	6.04	2.08	3.05	
June 1967	51.23	6.00	2.07	3.03	

*Benzene was not included for Tarawa Terrace as it was not included in the modeled water results (Maslia et al. 2007).

Table 2. Overall estimated mean concentrations (µg/L) of PCE, TCE, DCE, VC, and benzene at Hadnot Point and Tarawa Terrace over the time-period May 1966 - June 1967. These concentration data were used to estimate chemical exposures via drinking water (both locations) and residential dermal and inhalation exposures (Hadnot Point only) in this Report.

Hadnot Point (µg/L)					Tarawa Terrace (µg/L)			
PCE	TCE	DCE	VC	Benzene	PCE*	DCE	TCE	VC
0.0	24.4	0.0	0.0	1.0	52.1	6.2	2.1	3.2

*The Tarawa Terrace value for PCE is based on the results using the TechFlowMP model. The modeled values using the TechFlowMP model are lower than those generated using the MT3DMS model; the reasons for this are given in Jang and Aral (2008), pg. G-14. Because TCE, VC, and DCE were modeled using the TechFlowMP model only, for consistency, values for all four chemicals at Tarawa Terrace generated with that model are used in this Report.

Water source(s) and time-periods for exposure to chemicals in swimming pool air: **Before living on Base, the family lived off-Base in Jacksonville, NC from October 1965 through April 1966 (Karen Amsler April 16, 2024 Deposition Transcript, pg. 89-91, 96). Ms. Amsler also attended school at an off-base kindergarten for the 1965-1966 school year (Karen Amsler April 16, 2024 Deposition Transcript, pg. 95; 00284_AMSLER_0000007969-7970). While they were living off-Base, according to Ms. Amsler, she visited the Base. Though she did not offer details about her visits to the Base, Ms. Amsler did state: "...I'm sure my mother would shop on base and she would drag us all out there. Go swimming or whatever, do other activities" (Karen Amsler April 16, 2024 Deposition Transcript, pg. 97). She recalled occasionally using an on-Base pool during the time-period Fall of 1965 to Summer of 1966 (Karen Amsler April 16, 2024 Deposition Transcript, pg. 98). She could not recall where the pool was located and no other information specific to the pool was provided (Karen Amsler April 16, 2024 Deposition Transcript, pgs. 97-98).**

It is unclear from Ms. Amsler's testimony whether or not she continued to use a Base pool after moving on Base in May of 1966. Therefore, in order to select the time-period during which Ms. Amsler may have used an on-Base pool, I examined mean chemical concentrations in water from Hadnot Point and Tarawa Terrace for two different time-periods: (i) the period that Ms. Amsler recalled using the pool (Fall 1965-Summer 1966), or (ii) the entire period that Ms. Amsler lived near or on Base (October 1965-June 1967). For each water source, I used the time-period with the higher (more conservative) water concentrations as the basis for estimating pool air chemical concentrations (Section 9). Note that I assumed that the pools Ms. Amsler used were located in places where water sources were either Hadnot Point or Tarawa Terrace, rather than a location served by water not impacted by the chemicals of interest; this is a conservative assumption.

I estimated average water concentrations for both of these time-periods using the data from the same Excel spreadsheets by S.S. Papadopoulos & Associates, Inc. described above. These are shown in **Table 3**.

Table 3. Average water concentrations for Hadnot Point and Tarawa Terrace source water for two time-periods (Fall 1965-Summer 1966 and October 1965-June 1967). Values in bold were used to assess air chemical concentrations at an on-Base swimming pool (see Section 9).

	PCE (µg/L)	TCE (µg/L)	DCE (µg/L)	VC (µg/L)	Benzene (µg/L)
Hadnot Point					
October 1965-June 1967	0.0	23.6	0.0	0.0	1.0
October 1965 – August 1966	0.0	22.2	0.0	0.0	0.9
Tarawa Terrace					
October 1965-June 1967	52.4	2.1	6.3	3.2	NA
October 1965 – August 1966	52.8	2.2	6.4	3.3	NA

NA = Not applicable; Benzene was not included for Tarawa Terrace as it was not included in the modeled water results (Maslia et al. 2007).

PCE, DCE, and VC concentrations for water at Hadnot Point for this time-period were zero (**Table 2**).

For Hadnot Point, the average water concentrations were slightly higher for the longer time-period (October 1965-June 1967; **Table 3**). For Tarawa Terrace, the average water concentrations were slightly higher for the shorter time-period (October 1965-August 1966; **Table 3**). For both potential water sources, I used the time-periods associated with the more conservative (higher) concentration results (bold values, **Table 3**). While the Hadnot Point time-period used in this Report differs from Ms. Amsler’s recollection of dates for swimming, I selected the more conservative values (bold) in order to avoid underestimating exposures and also to recognize that Ms. Amsler was not specifically asked whether she used a pool on Base while she resided on Base.

A description of the uncertainties in the ATSDR mean monthly concentration data is outside of the scope of this Report, but information is available on this topic in the Expert Reports by Dr. Hennes (2024) and Dr. Spiliotopoulos (2024).

According to the Expert Report of Dr. Spiliotopoulos (pgs. 68-69): “For Hadnot Point, as with Tarawa Terrace, ATSDR assumed concentrations of contaminants in the influent to the WTP were equal to the concentrations of contaminants in the ‘finished water’ that was delivered to consumers...This assumption is incorrect, as treatment of the influent to the treatment plant resulted in evaporative and other losses, reducing contaminant concentrations in the ‘finished’

water.” *Based on this expert opinion, the chemical concentrations used in this Report as well as the associated estimates of Plaintiff exposure would be overly conservative (too high).*

7. DERMAL AND INHALATION EXPOSURE – THE SHOWER MODEL

7.1 SHOWER model: Background

Due to the volatile nature of PCE, TCE, DCE, VC, and benzene, inhalation of these chemicals deriving from water can occur during showering and bathing and via the use of appliances that use water (e.g., washing machines, dishwashers). In addition, dermal exposure to PCE, TCE, DCE, VC, and benzene can occur during showering and bathing or during faucet use.

While the basic models for estimating inhalation and dermal exposures to chemicals are well-established, addressing the time-varying concentrations of these chemicals in a residence is complex. ATSDR's SHOWER model addresses this complexity. The model, first released (version 1.0) in May 2018, includes the basic components of the models described by the US EPA (US EPA 1989) and used for decades but adds model components that allow for a rapid evaluation of inhalation and dermal exposures from volatile chemicals in household water (ATSDR 2022b). It was developed with the following objectives (list taken directly from ATSDR 2022c):

- providing an easy-to-navigate platform that requires minimal input to obtain results,
- providing standardized scenarios based on characteristic parameter values,
- allowing users to develop custom scenarios for site-specific simulations, and
- allowing users to evaluate the effects of changing model parameters on model outputs.
- simulating the most common water sources that contribute to indoor exposure,
- evaluating exposure from water use in bathrooms and the main house in addition to exposures from showering,
- evaluating exposure throughout the day and night,
- accounting for non-exposure when persons are away from the home, and
- accounting for exposure contributions from water use by all household members.

To run the SHOWER model, the user needs a chemical name and chemical concentration in the finished water to obtain estimates of household daily air concentrations, dermal doses, and inhalation doses (ATSDR 2022b). Since the release of version 1.0, ATSDR has released additional versions that give the model user more flexibility in terms of the behaviors of people in the household (e.g., number and timing of showers) and the layout of the modeled residence (e.g., number of bathrooms, size of the house, aspects of appliances in the residence) and improve the underlying model equations (ATSDR 2022c).

SHOWER model v2.0, released in February 2020, had several changes including the ability to evaluate the sensitivity of simulation results to changes in model parameters. It also expanded upon the functionality of the first model by, for example, allowing the user to customize several model parameters (e.g., number of bathrooms, activity sequence and duration for each household member, size and layout of the house, and household appliance parameters) (ATSDR 2022c).

Version 3.0, released in May 2022 (ATSDR 2022c), allowed estimation of both central tendency (or “typical”) exposure (CTE) and reasonable maximum exposure (RME) results for households with 1, 2, 3, and 4 persons. In addition, the effects of contaminant saturation in air were incorporated into the SHOWER model’s governing equations and an export function was added permitting the import and analysis of SHOWER model data within PHAST.

Version 4.0.0 (v4) was released 26 September 2024. The main change is that v4 “...adds the ability to simulate inhalation and dermal exposures from contaminated water in public showers and bathrooms” (e-mail from PHAST, CDC, 26 September 2024). Specifically, it “...includes default exposure scenarios for gyms, offices, schools, daycares, and dorms or barracks” (e-mail, 26 September 2024, David Mellard, ATSDR/OAD/OCDAPS). There was also “...a change in the equations for calculating chemical volatility” resulting in higher inhalation concentrations compared to Version 3.0, although for volatile chemicals the difference is considered by ATSDR to be minimal (< 5%) (e-mail, 26 September 2024, David Mellard, ATSDR/OAD/OCDAPS). Version 4.0.1 (v4.0.1) was released on 19 November 2024. In terms of relevance to the modeling for this Report, the new version produces model reports with more information on facility visits, corrects a report bug in v4 regarding peak times, and includes revised algorithms for assigning activity patterns in the public shower and bathroom scenarios and for determining the number of people who shower in scenarios with small facilities where only a low percentage of people take showers (e-mail, 19 November 2024, David Mellard, ATSDR/OAD/OCDAPS).

I note here that other models that estimate indoor air concentrations have various limitations regarding their utility for assessing indoor air human exposures at Camp Lejeune. For example, various models have been developed to estimate indoor air concentrations but do not include a component that estimates human exposures (NRC 1981). Also, some available models were designed for a different purpose (e.g., assessing the effect of use of indoor stoves on air quality [WHO 2020]).

The ATSDR model relies on standard inhalation and dermal exposure equations used by exposure scientists for many years, estimates time-varying indoor air/water concentrations and human exposures, allows for various modifications to better represent site-specific features of the indoor environmental and human behaviors, and has undergone some evaluation.

7.2 SHOWER model - Residential: Methodology and parameters

In this Report, SHOWER Model v4.0.1 was used to estimate human exposures to PCE, TCE, DCE, VC, and benzene at Camp Lejeune via residential inhalation and dermal contact. Detailed descriptions of the model algorithms and parameters were given for SHOWER Model version 3.0 in ATSDR (2022c). A technical document for SHOWER v4.0.1 was not available at the time of the preparation of this Report; I assume that the information in ATSDR (2022c) on the algorithms used to estimate inhalation and dermal exposures apply to this latest version. The information in this section describes the SHOWER model developed for residential house scenarios.

Inhalation route of exposure: The SHOWER model predicts air concentrations as a function of time in various house compartments including showers, bathrooms, and the main house. The air concentration of a chemical within a compartment increases as water is used and decreases after cessation of water use and ventilation to other rooms and outdoor air. The equations describing these processes are given in ATSDR (2022c).

The air concentrations are used in the following equation in the SHOWER model to calculate doses via inhalation:

$$ID=(C\times IR\times EF)/BW$$

Where:

ID = inhalation dose (µg/kg/day)

C = contaminant air concentration (µg/m³)

IR = intake rate (m³/day)

BW = body weight (kg)

EF = exposure factor (equal to 1)

Dermal route of exposure: The SHOWER model predicts water concentrations as a function of time for each appliance, where the concentration entering the appliance is input by the user (in this Report, these would be the average concentrations shown in **Table 2**). The concentration in the outflow is time-dependent and affected by the amount of the chemical that has volatilized (entered the air). The equations describing these processes are given in ATSDR (2022c).

The SHOWER model uses the following equation to estimate dermal dose (ATSDR 2022c; US EPA 2004):

$$DAD=(DA_{event}\times SA\times EV\times EF)/BW$$

Where:

DAD = dermal absorbed dose (µg/kg/day)

DA_{event} = absorbed dose per event (µg/cm²/event)

SA = skin surface area available for contact with water (cm²)

BW = body weight (kg)

EV = event frequency (events/day)

EF = exposure factor

Appendix B of ATSDR (2022c) shows the equations used by the model to calculate the average chemical concentrations in water for the dermal exposure equations.

The default exposure factor in the SHOWER model is set to 1 because the model assumes that the activities leading to exposure (e.g., showering, bathing, handwashing) occur daily. For organic compounds such as the chemicals of interest at Camp Lejeune, the equation used to estimate *DA_{event}* includes the chemical concentration in the water and other factors, and depends on the

time required for the chemical to reach steady state when passing through the skin compared to the duration of the human activity.³

The SHOWER model includes numerous default values that are applied to the model parameters, unless the user specifies that alternative values are to be used. Several key parameters and default values are described in the following section (Section 7.3). Appendix 2 shows the SHOWER model parameters and options for modifications.

A single run of the SHOWER model represents modeled exposure for a single 24-hour day. Time spent away from the residence does not contribute to exposure.

ATSDR has provided information on its evaluation of the SHOWER model (Appendix 3) and also has noted (ATSDR 2022b, pg. B3) that an uncertainty related to modeling the dermal permeability coefficient for certain halogenated chemicals, including PCE, TCE, DCE, and VC, can result in an underestimate of dermal doses. I did not identify any information on the extent of underestimation.

7.3 SHOWER model: Parameter default values

The SHOWER model requires information on parameters that describe population characteristics and features related to the residential environment. In the following sections, I describe several of these parameters. The default values and their bases are discussed. Many of these default values were used in the modeling for this Report. As noted above, default values are often used when site- and situation-specific information is not available. The following section (7.4) includes descriptions of parameter values that were modified based on site-specific information.

Values for most of the parameters required for the SHOWER model are unlikely to be known to individuals who resided on the Base. For example, it is not reasonable to expect people to know – let alone remember from their time on Base decades ago – their daily breathing rate (liters per minute), total body surface area, or hand surface area. Because those who resided on the Base are unlikely to reasonably know the information for these parameters, it is appropriate and necessary to use default values.

Population characteristics

Number of people in the residence

The SHOWER model default is for a 4-person household. A modification to the default is described in Section 7.4.

³ Detailed information on the equations and parameters used to estimate DA_{event} can be found in ATSDR 2022c.

Body weight

The SHOWER model default values for age-specific body weights are derived from the US EPA Exposure Factors Handbook (described above). The SHOWER model runs for this Report utilize the default SHOWER model body weight values shown in **Table 4**. For those on Base during their mid-teen years (ages 16, 17, 18 years of age), two different approaches to modeling exposure were employed. First, the body weight for 16 - < 21-year-olds (**Table 4**) was used, since mid-teens fall within this age range. In addition, separate models were run for mid-teens using the adult body weight in **Table 4**. This was done in recognition that the weights given in **Table 4** are based on national averages but some older teens can have weights more closely resembling adults (e.g., the 75th percentile for body weights for 16 - < 21-year-olds is 80.6 kg [US EPA 2011]). Those ages 19 and older are considered adults for the purposes of the modeling in this Report and the body weight for an adult (**Table 4**) is used. This is in recognition of the fact that a 19-year-old is at the high end of the 16 - < 21-year range and body weights are likely more closely approximated by an adult weight than a mid-teen weight.

Based on Ms. Amsler's birth month and year (May 1960; 00284_Amsler_NARA_0000000160), she would have been 6-7 years old when she lived on Base (May 1966 - June 1967). I therefore used the body weight for the 6 to < 11 years age group for dermal and inhalation exposures.

Table 4. Default body weights for the ATSDR SHOWER model.

Exposure group	Body weight (kg)
Birth to < 1 year	7.8
1 to < 2 years	11.4
2 to < 6 years	17.4
6 to < 11 years	31.8
11 to < 16 years	56.8
16 to < 21 years	71.6
Adult	80
Pregnant/breastfeeding women	73

kg=kilogram

As noted above, the SHOWER model utilizes data from the US EPA Exposure Factors Handbook (US EPA 2011) to obtain its default body weight data. The US EPA values as shown in the Exposure Factors Handbook are identical to the ATSDR SHOWER default values when the age groupings are the same. However, in some cases, ATSDR presented the age ranges differently from the US EPA (although the underlying weight data used by both ATSDR and the US EPA are the same). So, for example, the body weight value for ages birth to < 1 year in the SHOWER model is approximately the time-weighted average of the US EPA's values for birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to < 11 months (Table 8-1, US EPA 2011). The body weight for pregnant women of 73 kg (**Table 4** above) is equivalent to the 50th percentile estimated body weights of pregnant women derived from NHANES (1999–2006) (US EPA 2011, Table 8-29).

Breathing rate and body surface area

Age-based default values (Table C2, ATSDR 2022c) for breathing rate and surface area were used in this Report. For Ms. Amsler, values for 6 - <11-year-olds were used.

Shower behavior

The SHOWER model provides estimates of exposures to chemicals associated with showering. The SHOWER model uses the following default shower durations for a 4-person household (ATSDR 2022c):

CTE scenario: 7-minute showers for all people in the household

RME scenario: 10-minute showers for everyone except the last person to shower; 15 minutes for the last person to shower

The CTE scenario default value for a shower duration of 7 minutes is similar to the average reported shower duration of 7.8 minutes per shower for North America (DeOreo et al. 2016). The default RME value more closely approximates the recommended mean values for showering time for adults in the US EPA Exposure Factors Handbook (2011; Table 16-1): 18 minutes for 6 < 11-year olds, 20 minutes for 16 - < 21-year-olds and 17 minutes for adults 18 - < 65 years of age. Information on shower/bath durations used in this Report can be found in Section 7.4 below.

The SHOWER model also allows the user to select (i) the time of day the shower(s) occurred, (ii) the time spent in the bathroom after showering, and (iii) the time between the bathroom stay and the next shower. For these parameters, model defaults were used because no site-specific information was available. For the time of day of showering, the morning default setting is such that all showers occur before the individual of interest leaves the residence. Thus, in cases where the default values are used, the modeled person would be exposed to volatilized chemicals from all morning showers. Unless I identified information to the contrary, I used the default assumption of morning showers.

Ms. Amsler reported taking a daily bath or shower but could not recall which (Karen Amsler April 16, 2024 Deposition Transcript, pg. 99). No information was identified on

shower/bath duration or on other family members' bathing/showering habits. The approach used in this Report is described in Section 7.4.

Time away from residence

The SHOWER model default is that people remain in the residence for the entire 24-hour day. See Section 7.4 for the values used for the Plaintiff-specific model runs for this Report.

Residence-related features

The SHOWER model equations are based on a scenario in which individuals reside in a building that resembles a home; the layout of an example house with selected locations of utilities and bathroom/shower configuration is shown in **Figure 3**. Model defaults for several aspects of this house layout are described here. Site- and Plaintiff-specific modifications to these defaults are described in the following section (7.4).

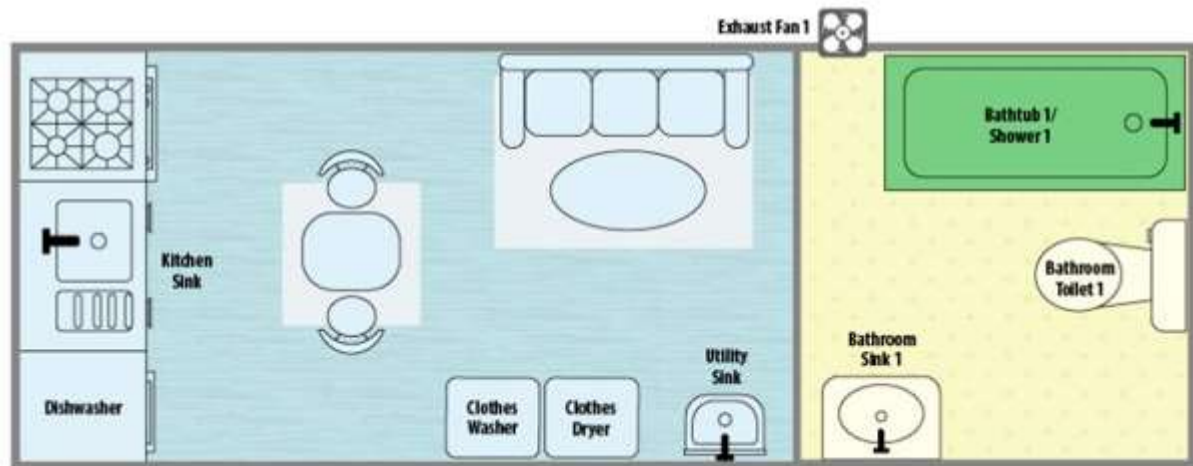


Figure 3. Layout of a house used by the SHOWER model based on selected input options (e.g., one bathroom, shower/tub combination).

House features and appliances

The SHOWER model is based on a residence that contains bathrooms and appliances (kitchen sink, utility sink, clothes washer, dish washer). The model utilizes several house-related parameters with modifiable values (see Appendix 2). These include:

- number of bathrooms with showers (one or two),
- shower/bathtub layout,
- clothes washer location,

- exhaust fan location (bathroom or shower),
- area volumes (house, bathroom, shower),
- whether the exhaust fan is on or off when the bathroom is occupied,
- whether the bathroom door is open or closed while the bathroom is occupied.

Further, the model includes several modifiable factors related to household appliances in the main house (e.g., kitchen sink flow rate and duration per use, dishwasher volume per cycle and cycle duration and start time, utility sink volume use per person), the clothes washer (location, cycle volume and duration, start time), bathroom compartment parameters (sink flow rate and duration per use, toilet volume per flush), exhaust fan parameters (location and flow rate), bathtub parameters (location and volume), and a shower parameter (flow rate).

Based on a review of plaintiff depositions, some residences on Base had certain of the appliances available in the SHOWER model while others did not. In some cases, certain appliances were in the residence but the locations relative to other parts of the residential space were not given. There is also no site-specific information related to such parameters as kitchen sink flow rate and duration per use, utility sink volume per person, dishwasher volume per cycle, cycle duration and start time, clothes washer parameters (location, volume per cycle, cycle duration), bathroom compartment parameters (sink flow rate, duration per use, toilet volume per flush, exhaust fan parameters [location and flow rate], bathtub volume, and shower flow rate). Because I did not identify any information on these site-specific parameters, I used model default values.

In this Report, I assumed that residences would include appliances that utilize water, and that these appliances could contribute to overall human exposures to PCE, TCE, DCE, VC, and benzene. These appliances include a dishwasher, washing machine, utility sink, and kitchen sink. The inclusion of the use of these appliances would yield conservative results for those living in residences without these appliances. The daily default dishwasher cycle duration is 145 minutes (default start time 9:00 pm) and the default clothes washer cycle duration is 75 minutes (default start time 7:00 pm) (these times overlap with when the modeled person is in the residence). The chemical releases for the kitchen sink and utility sink are not set at specific times; they are averaged over the time-period during which people are assumed to be awake in the residence (ATSDR 2022c). To demonstrate the effect of having these four appliances operating in the residence, I ran the SHOWER model twice for water with a hypothetical PCE concentration of 1 µg/L: once with appliances “on” and appliance-related values set to default values, and once with appliances turned “off” (all other parameters are in the default setting; four-person household with morning showers). The difference in average daily PCE exposures between the two scenarios is shown in **Table 5** (see Appendix 4.1 for supporting information). As expected, modeled exposures are higher with appliances “on.”

Table 5. Range of average daily exposure PCE concentrations for four people in a residence with the clothes washer, dish washer, kitchen sink, and utility sink operating each day (“on” or default setting) and off.

PCE average daily exposure ($\mu\text{g}/\text{m}^3$)	
Appliances “on”	Appliances “off”
0.17-0.33	0.13 - 0.30

No information on appliances in Ms. Amsler’s home was identified. In this Report, I have assumed that appliances were used in her household (model default), which could potentially yield overly conservative results (i.e., overestimate exposure).

Bathroom door and ventilation

The default setting for the SHOWER model is a closed bathroom door when the bathrooms are occupied and ventilation turned off. **No information on the status of the bathroom door or ventilation in Ms. Amsler’s home was identified.** For the models used in this Report, I assumed that the door was closed and that there was no ventilation. These, again, are conservative assumptions that are unlikely to result in underestimates of exposure.

To demonstrate the impact of the bathroom door being open or closed, the SHOWER model was run assuming a hypothetical water PCE concentration of $1 \mu\text{g}/\text{L}$ in a four-person household with all default settings for all parameters. The average daily exposures with the bathroom door closed while the bathroom is occupied are about 6% higher than with it open (**Table 6**). Thus, while the difference in model results under these two scenarios is small (see Appendix 4.2 for supporting information), the closed-door scenario results in a higher estimate of exposure.

Table 6. Average daily exposures ($\mu\text{g}/\text{m}^3$) with the bathroom door open or closed (hypothetical PCE water concentration of $1 \mu\text{g}/\text{L}$; four-person household).

	Bathroom door open	Bathroom door closed
Range of average daily exposures ($\mu\text{g}/\text{m}^3$)	0.16-0.31	0.17-0.33

Residence size

The SHOWER model default values for the compartments in the residence are: total house = 339.802 m^3 , total bathroom = 9.061 m^3 , and shower = 2.039 m^3 . The default values for the bathroom and shower volume are used in this Report and the total house volume was modified (see Section 7.4).

Outdoor air concentration

To estimate indoor air concentrations of the chemicals of interest in residences at Camp Lejeune, the SHOWER model includes an input for outdoor ambient air concentrations of these chemicals. According to ATSDR (2022, pg. A12c), “If a contaminant’s outdoor air concentration is unknown, the SHOWER model assumes a default concentration of zero.” I am unaware of outdoor air concentration data for the chemicals of interest specific to Camp Lejeune in the time-period during which Ms. Amsler was on Base. Therefore, I used the default value of 0 $\mu\text{g}/\text{m}^3$.

Modification of the outdoor air concentration can have an effect on the modeled exposure results. Here I consider the effect of modifying the ATSDR default value of 0 $\mu\text{g}/\text{m}^3$ by using a modeled ambient outdoor air concentration for PCE (with PCE selected as an exemplar chemical). I first provide information on outdoor air concentrations of PCE in the US. I then use the information from these data to examine the effect of modifying the outdoor air concentration on the results of the SHOWER model.

Outdoor ambient air concentrations of PCE from the US EPA

The US EPA has modeled ambient air concentrations of PCE in the US as part of its National Air Toxics Assessment (NATA) program (<https://www.epa.gov/national-air-toxics-assessment>).⁴ Data for specific chemicals can be found at: <https://www.epa.gov/national-air-toxics-assessment/2014-nata-assessment-results#pollutant>. Databases from two years were identified: 2014 and 1996. Neither of these databases provides data relevant to the time-period of interest for the exposure assessment in this Report nor are the data specific to Camp Lejeune. Therefore, it would be inappropriate to use these data for the exposure assessments in this Report. The information can, however, be used as the basis for providing bounds when seeking to understand the effects of modifying the outdoor air concentrations in the SHOWER model.

The NATA database of ambient air PCE concentrations did not include North Carolina in the most recent 2014 database. The overall concentration for PCE in ambient air in the US was 0.02 $\mu\text{g}/\text{m}^3$. The range of air concentrations for all of the states for which concentrations were reported is 0 – 1.74 $\mu\text{g}/\text{m}^3$. For comparison purposes, NATA data from 1996 (<https://archive.epa.gov/airtoxics/nata/web/html/tablconc.html>) are summarized here as well. The mean nationwide concentration for PCE was 0.323 $\mu\text{g}/\text{m}^3$ and the 95th percentile was 0.78 $\mu\text{g}/\text{m}^3$. This database also included data for North Carolina: the mean concentration for PCE (statewide) was 0.218 $\mu\text{g}/\text{m}^3$ and the 95th percentile was 0.371 $\mu\text{g}/\text{m}^3$.

⁴ Ambient air concentrations of hazardous air pollutants are estimated using an air dispersion model in combination with information on emissions from stationary (large facilities) and mobile sources. (https://www.epa.gov/sites/default/files/2015-05/documents/aceappendixb_nationalairtoxicsassessment.pdf)

Effect of modifying outdoor ambient air concentrations of PCE on exposure estimates

The potential effect of modifying the outdoor ambient air concentrations of PCE on SHOWER model results is illustrated here. A hypothetical water PCE concentration of 10 µg/L was used for the model runs and the residence was modeled with four individuals. For all other inputs, default values were used.

For outdoor air concentrations, two different values were used as model inputs: the ATSDR default value of 0 µg/m³ and a value of 0.2 µg/m³ which approximates the mean concentration shown above for North Carolina. **Table 7** includes the results of these different model runs. Only results for the average daily inhalation doses are shown as dermal doses remain unchanged with these modifications to outdoor air concentrations.

Table 7. SHOWER model results for highest exposed adult varying the ambient outdoor air concentrations of PCE (see Appendix 4.3 for supporting information).

Outdoor air PCE concentration (µg/m ³)	Average daily inhalation dose – PCE (µg/kg/day)
0	0.71
0.2	0.75

By increasing the outdoor air concentrations of PCE from 0 µg/m³ (model default) to 0.2 µg/m³, human daily intakes via inhalation would increase by about 5%. This example shows that increases in outdoor air concentrations can result in increases in the results of the SHOWER model exposure estimates. The extent of the increase is dependent on the outdoor air concentration. The data for North Carolina are not for a year that is relevant to plaintiff exposure nor are they specific to Camp Lejeune. Thus, in the case of exposures at Camp Lejeune for the time-period of interest, there are no data to support a scientifically sound estimate of outdoor air concentrations for the chemicals of interest and so the default value of 0 µg/m³ is used.

7.4 SHOWER model: Plaintiff-specific modifications

In the previous section, SHOWER model parameters and default values used for exposure estimates in the Report were described. Values for the following model parameters were selected or modified for the exposure estimates in this Report: number of people in the residence, duration and timing of showers/baths, time away from the residence, and size of the residence. The values and bases for these decisions are described here.

While living on Base, Ms. Amsler's home address was 2517 St. Mary's Drive, Camp Lejeune, NC, which was located at Paradise Point (00284_AMSLER_000007920-7922) and was supplied by the Hadnot Point water system (https://www.atsdr.cdc.gov/camp-lejeune/faq/?CDC_AAref_Val=https://www.atsdr.cdc.gov/sites/lejeune/faq_water.html).

Therefore, the Hadnot Point water data were used for estimating dermal and inhalation exposures with the SHOWER model.

Number of people in the residence

I assumed that that six people live in Ms. Amsler's residence. **Ms. Amsler had three younger siblings and two parents. Her siblings were born in March 1962, November 1963, and April 1966 and so would have lived on Base during the time-period May 1966 - June 1967 (00284_AMSLER_NARA_0000000160).**

Duration and timing of showers/baths

With six people in the house, I assumed that the use of an RME shower duration of 10 minutes per shower plus 15 minutes for the last person to shower/bathe would not be reasonable (for six people, this would require over an hour of continuous showering/bathing every morning). I used the CTE value of 7 minutes per shower/bath. I assumed that Ms. Amsler would be the last of the six family members to shower/bathe (a conservative assumption). I further assumed that the first five family members would shower and Ms. Amsler would bathe; this assumption yields a slightly more conservative result for the Plaintiff than the assumption that Ms. Amsler showered. **When asked if she showered daily, Ms. Amsler replied that "[i]t might have been a bath" (Karen Amsler April 16, 2024 Deposition Transcript, pg. 99).** As an additional conservative approach, I assumed that Ms. Amsler might have taken a longer bath (20 minutes) after her family members had each taken their showers (7 minutes each).

To demonstrate the effect of shower duration on average daily air concentrations of chemicals of interest, the SHOWER model was run with a hypothetical water PCE concentration (with PCE selected as an exemplar chemical) of 1 µg/L and two different shower durations (**Table 8**) (default values are used for all other parameters). Average daily exposure concentrations for the fourth person to shower are shown in **Table 8** (see Appendix 4.4 for supporting information). There is an approximately 3-fold difference in average daily exposure between a 7-minute and 15-minute shower for the fourth person who showers (i.e., longer showers are associated with higher average daily exposures).

Table 8. Average daily exposure PCE air concentrations for the fourth person to shower (in a four-person residence with all four people showering the same number of minutes).

PCE average daily exposure (µg/m ³)	
7-minute shower	15-minute shower
0.33	0.92

Time away from the residence

The SHOWER model default value for time spent away from the residence is zero, i.e., people would remain in the residence for the entire 24 hours of the modeled day. Based on my review of plaintiff depositions (including that for Ms. Amsler), it would be unusual for people to remain in the residence for the entire day as there tend to be activities such as school or family time away from Base. It is not possible to know exactly how many hours per day, on average over the time-period spent at Camp Lejeune, that a plaintiff would have been away from a residence.

For this Report, I used an assumption of no time away from the residence. **Ms. Amsler did go to school and participate in other activities (Karen Amsler April 16, 2024 Deposition Transcript, pgs. 95, 97-99).** However, no information was identified regarding the hours she was at school or participated in away-from-home activities. Further, school would only have been in session for part of the year. I opted to make the conservative assumption that Ms. Amsler stayed at home all day. I also assumed that the other five modeled residents did not leave the residence during the day and were thus “using” water in the residence all day as described by the model defaults.

Size of the residence

The SHOWER model includes a modifiable parameter related to building volume. I have seen photographs provided by Ms. Amsler that purport to be her family’s house at Camp Lejeune (00284_AMSLER_0000008039-8041), but I did not identify any information on the specific size of Ms. Amsler’s residence. I was provided with military housing documents with example building sizes to review for this Report. For the housing size estimate for this Report, I used an example 3-bedroom, 1-bathroom residence from Tarawa Terrace (CLJA_USMC_PWD_0000173504) with the following dimensions: 36 ft 8 in by 24 ft 8 in. An approximation of the ceiling height (CLJA_USMC_PWD_0000173504) is 8 ft. Thus, the residence volume is: 440 in x 296 in x 96 in = 12,503,040 in³ = 7235.56 ft³ = 204.89 m³ (cubic meter). The size of this modeled residence (~904 ft²) is roughly two thirds of the median house size (1,500 ft²) in the US (ATSDR 2022c).

The default bathroom volume in the SHOWER model is 9.061 m³ which is equal to about 319.99 ft³. According to ATSDR (2022c), the total bathroom volume (9.061 m³) corresponds to the upper range of a typical small bathroom and the shower volume (2.039 m³) corresponds to the size of a standard small shower. I did not modify either the bathroom or shower volume.

Given the lack of information for Ms. Amsler regarding the size and configuration of her residence, the use of the SHOWER model with housing dimensions described above (based on housing in Tarawa Terrace), in my professional judgment, is a reasonable approach to estimating exposures.

7.5 Opinion: Dermal and inhalation exposures at Camp Lejeune

I used the ATSDR SHOWER model and model parameter values described in this Report to estimate chemical exposures via inhalation and dermal contact with water from the Hadnot Point water system in a residence at Camp Lejeune.

People residing at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in a similar area, and engaged in similar activities could have been exposed to the following daily exposure concentrations (**Table 9**):

Table 9. Average daily exposure concentrations for the sixth person (6 - < 11 years old) using a bath at Hadnot Point, Camp Lejeune (May 1966 - June 1967).

Chemical	Average daily exposure concentration: 7-min showers and 7-min bath ($\mu\text{g}/\text{m}^3$)	Average daily exposure concentration: 7-min showers and 20-minute bath ($\mu\text{g}/\text{m}^3$)
PCE	NA	NA
TCE	17	31
DCE	NA	NA
VC	NA	NA
Benzene	0.58	1.0

*NA = Not Applicable; concentrations for water at Hadnot Point for this time-period were zero (**Table 2**).

Average daily exposures via inhalation and dermal contact for people on Base during the time-period that Ms. Amsler was there and with the scenarios described in this Report are shown in **Table 10**. Outputs from the SHOWER model – edited for length – are provided in Attachment 1.

People who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in a similar area, and engaged in similar activities could have been exposed to the following concentrations of the chemicals of interest via residential dermal contact with water and inhalation of chemicals volatilized from the water:

- Daily average exposure estimates via inhalation for TCE are 8.1 and 15 $\mu\text{g}/\text{kg}/\text{day}$ and via dermal contact are 0.076 and 0.12 $\mu\text{g}/\text{kg}/\text{day}$.
- Daily average exposure estimates via inhalation for benzene are 0.27 and 0.48 $\mu\text{g}/\text{kg}/\text{day}$ and via dermal contact are 0.0028 and 0.0044 $\mu\text{g}/\text{kg}/\text{day}$.

Table 10. Average daily exposures to chemicals from residential inhalation and dermal contact with water at Hadnot Point, Camp Lejeune (May 1966 - June 1967).

Hadnot Point, 6 - < 11-year olds	Inhalation 7-min showers and bath (µg/kg/day)	Dermal 7-min showers and bath (µg/kg/day)	Inhalation 7-min showers and 20-min bath (µg/kg/day)	Dermal 7-min showers and 20-min bath (µg/kg/day)
PCE	NA	NA	NA	NA
TCE	8.1	0.076	15	0.12
DCE	NA	NA	NA	NA
VC	NA	NA	NA	NA
Benzene	0.27	0.0028	0.48	0.0044

NA = Not Applicable; concentrations for water at Hadnot Point for this time-period were zero (**Table 2**).

8. INGESTION ROUTE OF EXPOSURE – THE PHAST MODEL

8.1 PHAST model: Background

Due to the potential presence of PCE, TCE, DCE, VC, and benzene in water during the 1950's to the 1980's at Camp Lejeune, exposure to those chemicals via ingestion of drinking water could have occurred. The approaches and equations for estimating intake of chemicals in drinking water were established decades ago (see, for example, US EPA 1989) and continue to be used to determine human exposures. Several media and exposure routes are included in PHAST; in this section of the Report, the focus is on the model developed to estimate exposures via drinking water ingestion.

The PHAST model “is based on ATSDR’s exposure dose guidance (EDGs) documents, which identify the parameters that are used to estimate exposure, either as a dose from ingestion of water or soil, or exposure as an air concentration. The EDGs were sent to EPA for review before sending them through clearance at ATSDR. PHAST is based on these EDGs and on ATSDR’s public health assessment guidance manual (PHAGM), which describes the PHA [public health assessment] process that ATSDR follows when investigating hazardous waste sites” (personal communication, PHAST Team; e-mail; 26 September 2023).

8.2 PHAST model: Methodology and parameters

Ingestion of water occurs from drinking the water directly (either straight or from its use in preparation of drinks such as coffee and tea) and by its use in food preparation (e.g., soups). In the case of human exposures at Camp Lejeune, chemical intakes (i.e., doses) were computed using PHAST version 2.3. PHAST includes the standard equation for estimating chemical intakes via water, as follows:

$$D = (EPC \times IR \times EF) / BW$$

Where:

D = age-specific dose (mg/kg-day), where values for body weight and intake rate vary according to age

EPC = exposure point concentration, or contaminant concentration (mg/L)

IR = intake rate of contaminated water (L/day)

BW = body weight (kg)

EF (intermediate or chronic) = exposure factor (unitless) = (F x ED)/AT

Where:

F = exposure frequency (days/week x week/year)

ED = exposure duration (year)

AT = averaging time (ED x F)

The user enters the name of the chemical of interest and the water concentration. The PHAST drinking water model estimates both the CTE and RME for different age groups. PHAST provides the option to use default values or to modify values for certain parameters. Appendix 5 shows the PHAST model factors and options for modification.

8.3 PHAST model: Parameter default values

In the following sections, I describe the parameters included in the PHAST water ingestion model. The default values and the bases for these values are discussed. These default values were used in the model for this Report.

Population characteristics

Scenario

The PHAST model permits the user to select from one of four scenarios: residential, daycare, school, or occupational. In this Report, the residential scenario is used. The other scenarios include inputs that allow the user to model fewer days per week and weeks per year of exposure compared to a residential scenario, but these adjustments are addressed in a separate Expert Report (Expert Report of Dr. Lisa Bailey for Karen Amsler). Here, the estimation of dose is for a single day of exposure.

Body weight

The body weights for the population age groups are the same as those described in Section 7.3 for the SHOWER model. The default values were used for this Report.

Water ingestion rates

The default values used in the PHAST model for estimating intake of drinking water represent the average or “typical” and 95th percentile of the distribution for water intake for the general US population (ATSDR 2023b). PHAST utilizes the drinking water ingestion rates for different age groups for both CTE and RME exposures shown in **Table 11**. The default RME value provides a conservative estimate of water intake.

The range of water intake values for 6 - < 11 year olds used in this Report is appropriate for the Plaintiff. **Based on Ms. Amsler’s birth month and year (May 1960; 00284_AMSLER_NARA_000000160), she would have been 6-7 years old when she lived on Base (May 1966 - June 1967).**

Table 11. Drinking water ingestion rates in the ATSDR PHAST drinking water ingestion model used in this Report.

Exposure Group	CTE Intake Rate (L/day)	RME Intake Rate (L/day)
Birth to < 1 year	0.595	1.106
1 to < 2 years	0.245	0.658
2 to < 6 years	0.337	0.852
6 to < 11 years	0.455	1.258
11 to < 16 years	0.562	1.761
16 to < 21 years	0.722	2.214
Adult	1.313	3.229
Pregnant Women	1.158	2.935
Breastfeeding Women	1.495	3.061

ATSDR based its default water intake rates on the US EPA 2019 update to its Exposure Factors Handbook. The intakes rates in **Table 11** above and those from the Exposure Factors Handbook are not identical. I explain the reason for this here.

Since the time of publication of the 2011 Exposure Factors Handbook, the US EPA has updated certain chapters and made them available online (<https://www.epa.gov/expobox/about-exposure-factors-handbook>). The recommended default values for ingestion of water and other fluids were updated in 2019 (https://www.epa.gov/sites/default/files/2019-02/documents/efh_-_chapter_3_update.pdf). These updated values are shown in **Table 12**.

Table 12. Default water ingestion rates from the US EPA's Exposure Factors Handbook update. Reproduced from Table 3-1 in US EPA (2019).

Table 3-1. Recommended Values for Drinking Water Ingestion Rates (2-day average community water intake) ^a					
Age Group	Mean		95 th Percentile		Multiple Percentiles
	mL/day	mL/kg-day	mL/day	mL/kg-day	
Per Capita ^b					
Birth to <1 month	184	42	851 ^c	200 ^c	See Tables 3-9 and 3-13
1 to <3 months	145	25	905 ^c	164 ^c	
3 to <6 months	187	27	981 ^c	141 ^c	
6 to <12 months	269	30	988	112	
Birth to <1 year	220	29	974	137	
1 to <2 years	146	13	565	51	
2 to <3 years	205	15	778	58	
3 to <6 years	208	11	741	42	
6 to <11 years	294	10	1,071	34	
11 to <16 years	315	6	1,395	26	
16 to <21 years	436	6	1,900	28	
21 to <30 years	781	10	2,848	39	
30 to <40 years	902	11	2,967	38	
40 to <50 years	880	11	2,964	38	
50 to <60 years	956	12	2,976	37	
60 to <70 years	941	12	2,972	35	
70 to <80 years	772	10	2,273	31	
80+ years	784	11	2,122	30	
21 to <50 years	858	11	2,938	38	
50+ years	902	11	2,827	35	
All ages	711	11	2,641	37	
Consumers-Only ^d					
Birth to <1 month	581	133	938 ^c	224 ^c	See Tables 3-17 and 3-21.
1 to <3 months	785	136	1,224 ^c	267 ^c	
3 to <6 months	649	93	1,125 ^c	158 ^c	
6 to <12 months	554	62	1,104 ^c	133 ^c	
Birth to <1 year	595	79	1,106 ^c	174 ^c	
1 to <2 years	245	22	658	57	
2 to <3 years	332	24	901	67	
3 to <6 years	338	19	836	45	
6 to <11 years	455	15	1,258	41	
11 to <16 years	562	10	1,761	31	
16 to <21 years	722	10	2,214	31	
21 to <30 years	1,183	16	3,407	47	
30 to <40 years	1,277	16	3,278	44	
40 to <50 years	1,356	17	3,374	43	
50 to <60 years	1,419	18	3,388	42	
60 to <70 years	1,394	17	3,187	40	
70 to <80 years	1,214	16	2,641	37	
80+ years	1,087	16	2,250	33	
21 to <50 years	1,277	16	3,353	44	
50+ years	1,343	17	3,081	40	
All ages	1,096	17	2,972	44	

Table 3-1. Recommended Values for Drinking Water Ingestion Rates (2-Day Average Community Water Intake) ^a (Continued)					
Age Group	Mean		95 th Percentile		Multiple Percentiles
	mL/day	mL/kg-day	mL/day	mL/kg-day	
Per Capita ^b					
^a	Ingestion rates for combined direct and indirect water from community water supply. Estimates are based on the average of 2 days of water consumption reported for each NHANES respondent. If the respondent reported zero consumption on one of the 2 days and nonzero consumption on the other day, his/her average consumption would be the average of zero and nonzero consumption.				
^b	Per capita intake rates are generated by averaging consumer-only intakes over the entire population (including those individuals that reported no intake).				
^c	Estimates are less statistically reliable based on guidance published in the <i>Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII Reports: NHIS/NCHS Analytical Working Group Recommendations</i> (NCHS, 1993).				
^d	Consumer-only intake represents the quantity of water consumed only by individuals that reported consuming water during the survey period.				
FCID	= Food Commodity Intake Database.				
NCHS	= National Center for Health Statistics.				
NHIS	= National Health Interview Survey.				
Source:	U.S. EPA analysis of NHANES 2005–2010 data using the FCID Consumption Calculator at http://fcid.foodrisk.org/ .				

For cases in which ATSDR and the US EPA organized age ranges the same way (e.g., children ages 1 < 2 years), the ATSDR (**Table 11**) and the US EPA (**Table 12**; “consumers only” data) intake values are identical. However, in certain cases ATSDR utilized different age groupings than the US EPA. So, for example, for children ages 2 to < 6 years, the ATSDR CTE and RME intake values are equivalent to the time-weighted sum of the values for children ages 2 to < 3 and 3 to < 6 in the US EPA’s consumers-only data above. Similarly, the ingestion rate value for adults (21-78 years) is the time-weighted average of the US EPA age groups within that age range, as shown in **Table 12** above (these calculations are described in ATSDR 2023b, Appendix C). Therefore, even though the numbers in the Tables appear to differ, they are in fact derived from the same underlying database. The intake rates for pregnant and breastfeeding women are taken directly from the US EPA’s Exposure Factors Handbook (Table 3-3).

The water intake rates in **Table 11** represent both direct ingestion (i.e., drinking water as a beverage) and indirect ingestion (e.g., intake of water that has been added during food and drink preparation) (US EPA 2019). The intake rate values are derived from NHANES and were estimated only from those NHANES participants who reported consuming water during the NHANES survey period (US EPA 2019). The values are considered to be representative of the general population in the US (<https://perma.cc/5GFB-SHV9>).

8.4 Opinion: Exposure via water ingestion at Camp Lejeune

I used the PHAST drinking water model with parameter values described in this Report to estimate chemical exposures via drinking water from the Hadnot Point and Tarawa Terrace water systems. Daily exposures via water ingestion for people on Base during the time-period when Ms. Amsler was at Camp Lejeune, with the scenarios described in this Report, are shown in **Table 13**.

Results are provided for both Hadnot Point and Tarawa Terrace as Ms. Amsler did not specify that she only consumed water from one of these water sources. Further, results are based on the time-period during which Ms. Amsler lived on Base as it is not known whether – or the extent to which – Ms. Amsler may have consumed water from Base during the period she lived in Jacksonville, NC.

People who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in a similar area, and engaged in similar activities could have been exposed to the following concentrations of the chemicals of interest via ingestion of water, with the ranges reflecting different water sources and different likely behaviors:

- Daily exposure estimates via water ingestion for PCE range from 0.00075 to 0.0021 mg/kg/day.
- Daily exposure estimates via water ingestion for TCE range from 3.0E-05 to 0.00097 mg/kg/day.
- Daily exposure estimates via water ingestion for DCE range from 8.9E-05 to 0.00025 mg/kg/day.
- Daily exposure estimates via water ingestion for VC range from 4.6E-05 to 0.00013 mg/kg/day.
- Daily exposure estimates via water ingestion for benzene range from 1.4E-05 to 4.0E-05 mg/kg/day.

Table 13. Daily intakes of chemicals from drinking water at Hadnot Point and Tarawa Terrace, Camp Lejeune (onsite from May 1966 - June 1967).

	Default Dose CTE (mg/kg/day)	Default Dose RME (mg/kg/day)
Hadnot Point		
PCE	NA	NA
TCE	0.00035	0.00097
DCE	NA	NA
VC	NA	NA
Benzene	1.4E-05	4.0E-05
Tarawa Terrace		
PCE	0.00075	0.0021
DCE	8.9E-05	0.00025
TCE	3.0E-05	8.3E-05
VC	4.6E-05	0.00013

CTE is based on a water intake rate of 0.455 L/day. RME is based on a water intake rate of 1.258 L/day. NA = Not Applicable; concentrations for water at Hadnot Point for this time-period were zero (**Table 2**).

9. SWIMMING POOL-RELATED CHEMICAL CONCENTRATIONS

Camp Lejeune has both indoor and outdoor swimming pools. **Certain plaintiffs – including Ms. Amsler – recalled using a pool on Base (Karen Amsler April 16, 2024 Deposition Transcript, pg. 98).** Therefore, in this Report I include information on swimming pool water-related concentrations of chemicals of interest.

Due to a general lack of information on what type of pool was used (indoor versus outdoor), the source of pool water (Hadnot Point, Tarawa Terrace, other water sources), specific activities at the pool (recreation, training, etc.), or duration and frequency of pool use (e.g., hours per day), various assumptions must be made.

It is important to note that for indoor pool use, exposure routes include inhalation, dermal contact, and incidental ingestion (incidental ingestion is the unintentional ingestion of typically small amounts of water while swimming). In contrast, for outdoor pool use, only dermal and incidental ingestion routes are considered as inhalation of chemicals in outdoor pool environments is considered to be negligible (Blando and Cohn 2004). This is because “... atmospheric concentrations above the pool water surface are very low, even when their concentrations in water are high” (Prud’homme de Lodder et al. 2006, pg. 87).

9.1 Models for estimating swimming-related exposure

Two models for estimating swimming pool-related exposures were identified: the ATSDR PHAST Surface water model and the US EPA SWIMODEL. A description of both models is provided here. I also include a comparison of results from a hypothetical exposure scenario and use this as the basis for selection of one of the models for use in this Report. Specifically, the model that yields the more conservative results is *a priori* selected for use in this Report.

9.1.1 PHAST Surface water model for swimming

The ATSDR PHAST Surface water model includes different scenarios for exposure to surface water (used here to represent pool-related water contact):

- Swimming (estimates both water ingestion and dermal contact)
- Wading (estimates dermal contact only as it is not expected that water would be ingested during this activity)

The model is for outdoor water exposure and therefore does not include the inhalation route of exposure. Wading in the water would result in less dermal contact as compared to swimming as it is assumed that the person would not be completely submerged in the water (i.e., exposure would only be to head, hands, forearms, lower legs and feet). As there is limited information on the amount of time people at Camp Lejeune may have spent swimming versus wading, in order to avoid underestimating exposures, only the PHAST model for swimming is considered here.

The PHAST model equation for estimating chemical intakes via incidental ingestion of pool water is as follows:

$$D = (EPC \times IR \times t_{\text{event}} \times EV \times EF) / BW$$

Where:

D = age-specific dose (mg/kg/day)

EPC = exposure point concentration (mg/L)

IR = intake rate (L/hr)

t_{event} = event duration (hr/event)

EV = event frequency (events/day)

EF = exposure factor (unitless)

BW = body weight (kg)

The PHAST model equation for estimating chemical intakes via dermal exposure to pool water is as follows:

$$ADD = (DA_{\text{event}} \times SA \times EV \times EF) / (BW \times ABS_{GI})$$

Where:

ADD = administered dermal dose (mg/kg/day)

DA_{event} = absorbed dose per event (mg/cm²/event) (see ATSDR 2018 for equations)

SA = skin surface area available for contact (cm²)

EV = event frequency (events/day)

EF = exposure factor (unitless)

BW = body weight (kg)

ABS_{GI} = gastrointestinal absorption factor (unitless)

Note that for this model, the Administered Dermal Dose (ADD) is estimated rather than the dermal absorbed dose. These are defined as follows (ATSDR 2018):

Dermal Absorbed Dose (DAD): The amount of chemical absorbed through the skin.

Administered Dermal Dose (ADD): A dermal absorbed dose that has been adjusted to an administered dermal dose using a GI absorption factor using the following equation:

$$ADD = DAD / ABS_{GI}$$

As the PHAST model uses a value of 1 for the ABS_{GI} for all of the chemicals of interest in this Report (ATSDR 2024), the ADD is equal to the DAD.

Default exposure age groups and body weights are the same as those described above (**Table 4**) for the SHOWER model. Model inputs are required for the number of hours per swimming event, the number of events per day, the number of days per week, the number of weeks per year and the number of years that swimming or wading occurred.

The model default incidental water ingestion intake rates for CTE and RME groups are shown in **Table 14**. These values are the same as those recommended by the US EPA in its Exposure Factors Handbook for child and adult ingestion of water while swimming (US EPA 2011). The CTE values in **Table 14** are reported as mean values in the Exposure Factors Handbook. The RME values are reported in the Exposure Factors Handbook as the 97th percentile of the distribution for children and the maximum value for adults.

Table 14. PHAST model default incidental water ingestion intake rates associated with swimming.

Exposure Group	Default Intake Rates (L/hr)	
	CTE	RME
Birth to < 1 year	0.049	0.12
1 to < 2 years	0.049	0.12
2 to < 6 years	0.049	0.12
6 to < 11 years	0.049	0.12
11 to < 16 years	0.049	0.12
16 to < 21 years	0.049	0.12
Adult	0.021	0.071

The default values for skin surface area used in the PHAST model for swimming are shown in **Table 15**. The skin surface areas are derived from US EPA (2011) and ATSDR (2018; Appendix A).

Table 15. PHAST model default total age-specific surface area associated with swimming.

Exposure Group	Default Total Surface Area for Swimming (cm ²)
1 to < 2 years	5,300
2 to < 6 years	7,225

Exposure Group	Default Total Surface Area for Swimming (cm ²)
6 to < 11 years	10,800
11 to < 16 years	15,900
16 to < 21 years	18,400
Adult	19,652

9.1.2 US EPA SWIMODEL model

The SWIMODEL was developed by the US EPA. It was designed as a screening tool for conducting exposure assessments of pesticides in indoor swimming pools and spas (US EPA 2024). Others have used the SWIMODEL for chemicals in pools other than pesticides (e.g., Anchal et al. 2020; Dehghani et al. 2022; Florida Department of Health 2012; Health Canada 2025; Minh Chau et al. 2025; Pándics et al. 2018; Peng et al. 2020).

According to the US EPA (2024), the “model itself can only run on computers using the Windows XP or older operating systems.” Therefore, EPA has ... “developed a spreadsheet that includes the formulas from the model” (US EPA 2024). This spreadsheet was used for estimating pool-related exposures in this Report. The spreadsheet is available at the following URL: <https://perma.cc/7KSY-EJSY>.

Several routes of exposure are considered by the SWIMODEL. The three standard routes are considered here: incidental ingestion of pool water, dermal exposure, and inhalation of volatile chemicals from the pool water. As noted by the US EPA, the “...SWIMODEL inhalation exposure route is only applicable to indoor exposure assessment calculations” (US EPA 2003, pg. 3).

The age groups considered in SWIMODEL are: adults, children (11-<16 yrs of age), and children (6-<11 yrs of age).

For incidental ingestion, SWIMODEL includes the following equation:

$$\text{Dose (mg/kg-day)} = (\text{CW} \times \text{IR} \times \text{ET}) / \text{BW}$$

Where:

CW = concentration of the chemical in the pool water (mg/L)

IR = ingestion rate (L/hr)

ET = exposure time (hours/day)

BW = body weight (kg)

The chemical-specific parameter that must be selected by the user is the chemical concentration in water (mg/L). The default water incidental ingestion rates for non-competitive adult swimmers, children (11-<16 yrs of age), and children (6-<11 yrs of age) are: 0.025 L/hr, 0.05 L/hr, and 0.05 L/hr, respectively. The exposure time for all age groups is 1 hour per day. The default body weights for all equations in SWIMODEL for adults, children (11-<16 yrs of age), and children (6-<11 yrs of age) are 80 kg, 57 kg, and 32 kg, respectively.

The equation for dermal exposure is:

$$\text{Dose (mg/kg-day)} = (\text{Cw} \times \text{SA} \times \text{ET} \times \text{Kp} \times \text{CF}) / \text{BW}$$

Where:

Cw = concentration of the chemical in the pool water (mg/L)

SA = dermal surface area (cm²)

ET = exposure time (hr-day)

Kp = permeability coefficient (cm/hr)

CF = conversion factor (L/1000 cm³)

BW = body weight (kg)

The chemical-specific parameters that must be input by the user are the chemical concentration in the water (mg/L), the chemical's molecular weight (grams/mole), and the octanol-water partition coefficient (Kow). The molecular weight and Kow are used to estimate the Kp value. Alternatively, if a Kp value is known, this can be used in the model directly. The default dermal surface area for adults, children (11-<16 yrs of age), and children (6-<11 yrs of age) are: 19,500 cm², 15,900 cm², and 10,800 cm², respectively. The default exposure time is 1 hr/day for all age groups for non-competitive swimmers.

The equation for inhalation exposure is:

$$\text{Dose (mg/kg-day)} = (\text{Cvp} \times \text{ET} \times \text{IR}) / \text{BW}$$

Where:

Cvp = vapor concentration (mg/m³)

ET = exposure time (hr/day)

IR = inhalation rate (m³/hr)

BW = body weight (kg)

The chemical-specific parameter that must be input by the user is the Henry's Law constant (HLC, atm-m³/mol). Henry's law describes the distribution of a chemical between a liquid and gas phase and states that "the amount of dissolved gas is proportional to its partial pressure in the gas phase" (Sander 2023), or:

$$\text{HLC} = p / \text{chemical concentration in water}$$

Where p is the partial pressure of the chemical in the gas phase under equilibrium conditions.

The default inhalation rate for adults, children (11-<16 yrs of age), and children (6-<11 yrs of age) are: 1 m³/hr, 1.5 m³/hr, and 1.3 m³/hr, respectively. The default exposure time is 1 hr/day for all age groups for non-competitive swimmers.

9.1.3 Comparison of the PHAST Surface water model and the EPA SWIMODEL

To evaluate which model is best suited for the exposure estimates at Camp Lejeune, the results of the PHAST surface water/swimming model and the SWIMODEL can be compared. I decided *a priori* to use the model that yielded the more conservative results.

I used a hypothetical scenario in which an individual swims for 1 hour in water with a concentration of PCE equal to 0.1 mg/L. The ingestion rate was set to 0.025 L/hr for both models and the skin surface area was set at 19,500 cm². I utilized the US EPA Regional Screening Level (RSL) data (US EPA 2024) to obtain values for molecular weight, Henry's Law constant, and K_{ow} . The data can be found at: <https://semspub.epa.gov/work/HQ/405301.pdf> and the data sources can be found at: epa.gov/risk/regional-screening-levels-rsls-generic-tables.

PHAST surface water swimming model

As noted above, the surface water model is used to simulate swimming in surface water (here, an outdoor pool) and therefore only considers dermal contact and incidental ingestion exposure routes (no inhalation). For the hypothetical scenario considered here, the daily modeled route-specific exposures using the acute scenario for an adult swimmer (CTE) are (see Appendix 6):

Dose from incidental ingestion: 3.1×10^{-5} mg/kg-day

Dose from dermal contact: 2.1×10^{-3} mg/kg-day

SWIMODEL

The SWIMODEL was developed for assessing chemical exposures for indoor pools. The SWIMODEL includes dermal, incidental ingestion, and inhalation routes of exposure. For the hypothetical scenario considered here, the daily modeled route-specific exposures for an adult non-competitive swimmer are (see Appendix 6):

Dose from inhalation: 0.9 mg/kg-day

Dose from incidental ingestion: 3.1×10^{-5} mg/kg-day

Dose from dermal contact: 1.11×10^{-3} mg/kg-day

The SWIMODEL includes a result for inhalation exposure which is orders of magnitude greater than estimated exposures via the ingestion or dermal routes from either model. Thus, the overall

exposures estimated using the SWIMODEL are substantially greater than those from the PHAST model because of the inclusion of the inhalation pathway via indoor air.

Because the SWIMODEL yields higher estimates of exposure than the PHAST model, the SWIMODEL approach of focusing on inhalation exposure at indoor pools is more protective of plaintiffs. Therefore, while it is often not known whether a plaintiff – including Ms. Amsler - used indoor or outdoor pools, by assuming indoor pool use and using the SWIMODEL, the exposure estimate is more conservative.

Because the inhalation route of exposure in the SWIMODEL yields exposure estimates that are orders of magnitude higher than dermal and ingestion routes combined, only the inhalation route is considered in this Report. This is consistent with the results from Costa et al. (2022) and ATSDR (2017, pg. 109), who concluded that for swimming pool exposures, “[t]he dermal exposure estimates are negligible when compared to the inhalation estimates.” Separate vapor concentration estimates are made for Hadnot Point and Tarawa Terrace because most plaintiffs - including Ms. Amsler - did not specify the location of the pool(s) they used.

9.2 SWIMODEL: Plaintiff- and chemical-specific modifications

In this Report, I use the following SWIMODEL equations to estimate the indoor air concentrations of the chemicals of interest in an indoor pool environment. First, the Henry’s law constant for a given chemical (**Table 16**) is converted to a unitless Henry’s law constant:

$$H' = HLC / (R \times (T + 273))$$

Where:

H' = Henry’s law constant (unitless)

HLC = Henry’s law constant (atm-m³/mol)

R = gas constant (8.19E-05 atm³/mole-K)

T = ambient air temperature (25° C)

Then H' is used to estimate the air concentration as follows:

$$C_{vp} = C_w \times H' \times CF$$

Where:

C_{vp} = vapor concentration (mg/m³)

C_w = water concentration (mg/L)

CF = conversion factor (1000 L/m³)

Human exposure estimates are provided in a separate Expert Report (Expert Report of Dr. Lisa Bailey for Karen Amsler).

Chemical concentrations in pool water: I am not aware of any site-specific measured data on concentrations of the chemicals of interest specific to pool water at Camp Lejeune. Mean water concentrations for the Hadnot Point and Tarawa Terrace water systems shown in **Table 3** are used to estimate vapor chemical concentrations in the indoor pool facility as no pool locations were specified by Ms. Amsler. **Ms. Amsler only recalled the following for the time she lived off-Base: "...I'm sure my mother would shop on base and she would drag us all out there. Go swimming or whatever, do other activities."** (Karen Amsler April 16, 2024 Deposition Transcript, pg. 97).

Physico-chemical data for chemicals of interest: The SWIMODEL requires chemical-specific inputs as described above. For estimating vapor concentrations in the air in indoor pool facilities, values for Henry's law constants (HLC) are needed. The HLC values used in this Report are shown in **Table 16**.

Table 16. Henry's law constants ($\text{atm}\cdot\text{m}^3/\text{mole}$) for chemicals of interest. Data source: US EPA Regional Screening Level data.

Chemical	HLC ($\text{atm}\cdot\text{m}^3/\text{mole}$)
Benzene	5.6E-03
DCE	9.4E-03
PCE	1.8E-02
TCE	9.9E-03
VC	2.8E-02

9.3 Opinion: Vapor chemical concentrations at indoor pools at Camp Lejeune

I did not identify information that placed the pools in a location where the water source was known with certainty, so to be conservative, I assumed they were located in places where the water source was either the Hadnot Point or Tarawa Terrace water system. I used model parameter values described in this Report to estimate average vapor chemical concentrations at indoor pools on Base (**Table 17**).

Based on the information described in this Report, I conclude that average vapor concentrations of chemicals of interest at indoor pools at the time(s) relevant to Ms. Amsler are as follows:

- Vapor concentration at indoor swimming pools for PCE is $3.89\text{E}+01 \text{ mg}/\text{m}^3$.
- Vapor concentrations at indoor swimming pools for TCE are $8.92\text{E}-01$ and $9.57\text{E}+00 \text{ mg}/\text{m}^3$.
- Vapor concentration at indoor swimming pools for DCE is $2.46\text{E}+00 \text{ mg}/\text{m}^3$.
- Vapor concentration at indoor swimming pools for VC is $3.79\text{E}+00 \text{ mg}/\text{m}^3$.
- Vapor concentration at indoor swimming pools for benzene is $2.29\text{E}-01 \text{ mg}/\text{m}^3$.

Table 17. Vapor chemical concentrations in indoor pools at Hadnot Point (October 1965 – June 1967) and Tarawa Terrace (October 1965 – August 1966), Camp Lejeune. See Section 6.2 for an explanation of different time-periods.

Vapor concentration (mg/m ³)	
Hadnot Point	
PCE	NA
TCE	9.57E+00
DCE	NA
VC	NA
Benzene	2.29E-01
Tarawa Terrace	
PCE	3.89E+01
DCE	2.46E+00
TCE	8.92E-01
VC	3.79E+00

10. CONSERVATIVE NATURE OF SELECTED MODEL INPUTS

As noted in previous sections of this Report, there are either limited or no data on various chemical (e.g., water concentrations) and behavioral (e.g., shower duration, water consumption) aspects of plaintiffs' chemical exposure during their time on Base. Some inputs for model parameters used in this Report are based on information recalled by Ms. Amsler. However, plaintiffs may not always recall the details of their environment or behaviors from decades prior. Thus, while information from plaintiffs on their behaviors is used as guidance for selecting parameter input values, judgment is also used to ensure that the exposure estimates are *not likely* to underestimate overall exposures during a plaintiff's time on Base.

In this Report, I used model input values that in my view should provide conservative estimates of exposure (i.e., not result in underestimates of Ms. Amsler's exposures). These are described in the following sections (these were mentioned in previous sections and are reiterated in this summary).

Overall, regarding the estimates for the mean monthly chemical concentrations in water developed by ATSDR and used in this Report, according to the Expert Report of Dr. Spiliotopoulos (pgs. 68-69): "For Hadnot Point, as with Tarawa Terrace, ATSDR assumed concentrations of contaminants in the influent to the WTP were equal to the concentrations of contaminants in the 'finished water' that was delivered to consumers...This assumption is incorrect, as treatment of the influent to the treatment plant resulted in evaporative and other losses, reducing contaminant concentrations in the 'finished' water." *Therefore, the chemical concentrations used in this Report as well as the associated estimates of Plaintiff exposure would be overly conservative (too high).*

10.1 Drinking water

Chemical concentrations: The chemical concentrations were based on monthly mean concentration data for the months that Ms. Amsler reported that she was on Base. Assumptions were made that would result in conservative estimates of the number of months on Base. Specifically: (i) If a plaintiff was on Base for part of the month, I assumed that the plaintiff was there for the entire month (the exception to this was if the plaintiff was only on Base for one day for that month). (ii) Plaintiffs may have been off-Base for part of their time assigned to Camp Lejeune (e.g., leave, weekends away, time spent on parts of the Base where water was not impacted). Unless they were off Base for at least one calendar month and the exact dates were known, it was assumed that they were on Base and exposed to the chemicals of interest for the entire time-period. I recognize that while these assumptions result in conservative (longer) estimates of time on Base, they may not always yield the most conservative estimates of water concentrations.

Intake rates: It is unreasonable to expect that any individual could recall their exact water intake from their time on Base. In depositions that I reviewed, volumes of water intake were variably described using language such as "cups," "glasses," "sips," or "canteens" (and the

descriptions of the size of a canteen varied). It is also unlikely that any individual would consume the same amount of water each day, and this is borne out by deposition statements in which plaintiffs note varying water consumption, depending on outdoor temperature and activities.

The model used in this Report provides an estimate of average daily water consumption over the duration of time spent on Base. Without exact information from plaintiffs on water consumption, it is reasonable to use national estimates of daily water intake (CTE equal to 0.455 L/day and RME equal to 1.258 L/day for 6 - <11 year-olds).

To visualize this amount of water intake, it is useful to recall that there are 8 fluid ounces in a cup. Consumption of 1.258 L as drinking water in a day is equivalent to about 5 and a third 8-ounce cups of water, or about a full 8-ounce glass of water every other hour over the course of a 10-hour day.

10.2 Showering/bathing

I selected SHOWER model options of no bathroom ventilation during showering/bathing and the bathroom door being closed during showering/bathing times. While no specific information on these factors was identified in documents related to Ms. Amsler, in a private residence it is possible that the door would be left open while a child is bathing. Assuming a closed door and no ventilation during showering/bathing would likely result in conservative estimates of chemical exposure.

It is also worth noting that the SHOWER model inputs used in this Report include having five showers occurring with the target person still in the residence (i.e., they have not left the residence during the five consecutive 7-minute showers). Therefore, they are in the residence not only during their bath but also during the 35 minutes of showers being taken by others in the residence prior to their own shower.

10.3 Time away from residence

The SHOWER model default has people remaining in their residence and exposed to chemicals from the water throughout the day unless the model user specifies otherwise. While Ms. Amsler spent part of her time on Base at school and presumably participating in other activities outside of the house, given the uncertainties associated with her amount of time away from her residence, I assumed that she remained in the residence all day. This is a conservative assumption.

10.4 Appliances

As described in this Report, having the dishwasher and washing machine running during the modeled day (appliances “on” in the SHOWER model) yields higher estimates of exposure to chemicals of interest. Further, the SHOWER model default (used for this Report) includes the use of a kitchen sink (15 times per day for 0.64 minutes per use) as well as the use of a utility sink

(8.544 L/person/day). Ms. Amsler did not say whether kitchen or utility sinks were used throughout the day or whether her residence had a washing machine or dishwasher.

Therefore, the use of the default SHOWER model settings (utilities on and in use in the residence during times when people are in the residence) could yield conservative estimates of exposure.

10.5 Residence type

Windows may have been left open in Ms. Amsler's residence. This increase in ventilation is not considered in the SHOWER model. It is likely that the open windows would result in lower concentrations of chemicals in the air. This would contribute to the possible overestimates of exposure from the SHOWER model.

10.6 Exposure via indoor pools at Camp Lejeune

The route of exposure with the most substantial contribution to overall swimming-related exposure is inhalation of volatilized chemicals at an indoor pool. In this Report, I assumed that the facility was an indoor pool. If Ms. Amsler swam at an outdoor pool, then this assumption would produce conservative results.

Further, it was unclear whether Ms. Amsler used the pool only during the time she lived off Base, or whether she continued to use the pool after she moved on Base. I used the more conservative time-periods for both Hadnot Point and Tarawa Terrace (i.e., the time-period with the higher water concentrations).

In addition, due to the lack of definitive information on the location of the pool used by Ms. Amsler, exposures were estimated for water from both the Hadnot Point and Tarawa Terrace water systems rather than a location served by water not impacted by the chemicals of interest; this is a conservative assumption.

Finally, as noted by ATSDR (2017), the SWIMODEL does not account for reductions in water concentrations due to volatilization from the pool over time. In addition, the model does not take into consideration any ventilation system that may be part of the indoor pool structure. Because I assumed that the swimming pool water concentrations are equal to the source water concentrations (**Table 3**) with no consideration of loss through volatilization and ventilation, the air concentration estimates are likely to be conservative (i.e., may over-estimate exposure).

11. REBUTTAL TO EXPERT REPORT BY DR. REYNOLDS

My overall approach to estimating exposures to chemicals of interest is similar to that of Dr. Reynolds in that we both provide a range of exposure estimates for each plaintiff. However, my approach differs from Dr. Reynolds' approach in several respects (described in the following paragraphs). In my opinion, and based on my training and professional experience in assessing exposures to chemicals, my assumptions are both conservative (in other words, would be unlikely to underestimate exposure) and more reasonable, i.e., supported by the scientific literature, Ms. Amsler's records, and my training, experience, and professional judgment). My exposure estimates consequently provide a more appropriate picture of Ms. Amsler's exposure to chemicals of interest than Dr. Reynolds' estimates.

In the following sections, I describe the general differences in approach between Dr. Reynolds' report and my Reports (Section 11.1) and differences specific to Ms. Amsler (Section 11.2).

11.1 General differences in approaches

11.1.1 Exposure route differences

Dr. Reynolds' exposure estimates are based on one exposure route: consumption of drinking water. However, plaintiffs would have also been exposed via the dermal and inhalation routes of exposure. In this Report, I use models to address these routes. In addition, where relevant, I use models to assess plaintiff exposures for specific additional scenarios including swimming pools and the mess hall. These were not addressed in Dr. Reynolds' overall report. Including these other exposure routes provides a more realistic picture of plaintiffs' potential exposure based on the available evidence. My inclusion of three routes of exposure provides a more conservative (i.e., higher) estimate of exposure compared to the exposure estimate I *would* have obtained had I only included the water ingestion route of exposure (as was done by Dr. Reynolds). As discussed in the Expert Report of Dr. Lisa Bailey, including these more realistic exposure routes does not result in an unacceptable cancer risk for people who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in similar areas, and engaged in similar activities.

11.1.2 "Cumulative consumption" versus daily intake

Dr. Reynolds provided exposure results in the form of "cumulative consumption," or the total number of micrograms of a chemical consumed by each plaintiff via drinking water over their entire time at Camp Lejeune, whereas I accounted for the body weight of the plaintiff. Generally, I used age-based default values (as described in this Report) to adjust for dose.

Inclusion of an approximate body weight (e.g., adult versus child) enhances one's ability to interpret the exposure results in a risk-based context. Generally speaking, given the same amount of chemical intake, the lighter the person, the higher the dose. To use a familiar example, "...smaller people usually have a higher ratio of alcohol in their blood if they drink the same amount a heavier person drinks..."

(<https://www.stanfordchildrens.org/en/topic/default?id=understanding-alcohols-effects-1-2860>).

In using this method, I employed the approach of ATSDR in its PHAST and SHOWER models, as well as the US EPA in its Risk Assessment Guidance for Superfund (1989), and assessed average daily exposures for each plaintiff in units of mg/kg-day or µg/kg-day. Average daily exposure values are the foundation for estimating human health risks (see Expert Report of Dr. Lisa Bailey). Dr. Reynolds instead represents the exposure results in terms of cumulative consumption.

11.1.3 Water ingestion rates

The drinking water intake rates used by Dr. Reynolds differ from those used in my exposure estimates.

Default values: The default values for CTE and RME estimates in my Reports are derived from the most recent US EPA Exposure Factors Handbook (updated drinking water ingestion chapter from 2019). For example, for adults, I use values of 1.313 and 3.229 L/day for CTE and RME estimates, respectively. These values are used by ATSDR in its PHAST model.

In contrast, Dr. Reynolds used adult CTE and RME values of 1.227 and 3.092 L/day, respectively. According to Dr. Reynolds, these values are derived from the US EPA's Exposure Factors Handbook (2011). These values were updated by the US EPA in 2019 (US EPA 2019). I used the updated values, which are more conservative for adults.

Other values: For plaintiffs who were marines in training on Base, Dr. Reynolds used ATSDR values to estimate drinking water intake rates: 6 L/day for 3 days per week and 3.1 L/day for 4 days per week. The overall weighted value reported by Dr. Reynolds is 4.334 L/day⁵. However, in at least one instance (Expert Report of Dr. Reynolds, pg. 126), Dr. Reynolds assumed a plaintiff water consumption values of 6 L/day for 3 days per week and 3 L/day for 4 days per week, for an overall weighted value of 4.29 L/day. She does not provide justification for selecting one over the other.

For some plaintiffs, Dr. Reynolds relied on US Army Field Manuals for information on water intakes associated with light and heavy activity to derive additional water intake values of 5.21 L/day and 8.52 L/day.

According to ATSDR (2017), "A marine in training at Camp Lejeune consumes an estimated 6 liters of water per day for three days per week and 3 liters per day the rest of the week (ATSDR 2016). Under warm weather conditions, a marine may consume between 1 and 2 quarts of water per hour... (Bove et al. 2014a)." The value of "1 to 2 quarts of water per hour" is difficult to rely on as the number of hours is not provided. However, the estimate of 6 L/day is supportable given that

⁵ See, for example, pg. 26 of Dr. Reynolds' Expert Report. Based on my calculations, the weighted daily value should be: $(6 \times 3) + (3.1 \times 4) / 7 = (18 + 12.4) / 7 = 30.4 / 7 = 4.34$ L/day.

the information is specific to marines in training. Therefore, a value of 6 L/day is a reasonable and conservative value for water intake by a marine in training. I used a high-end value of 6 L/day to estimate drinking water intake for many of my Reports that considered adult exposures. The estimate of 6 L in a day is equal to about 25 8-ounce glasses of water (or about two full glasses every hour during a 12-hour day). I did not make assumptions regarding the number of days per week that a plaintiff engaged in heavy activity (see the Expert Report of Dr. Lisa Bailey).

Dr. Reynolds stated in her Report (pg. 5): “For some plaintiffs, specific information was available in their deposition detailing their training and consumption habits...if consumption data was given, for example, recall of refilling and drinking a specific number of canteens (estimated to hold 32 oz each) during training, or a specific amount of coffee or tea (5-10 oz cups), “bug juice” or glasses of water (12 oz cups), or other beverage made from the contaminated water sources, deposition-informed ingestion data was used in the exposure assessment.” Dr. Reynolds utilized this kind of information from the depositions to develop water intake rates that appear to be very accurate, including several significant digits (e.g., “3.54882” L/day, pg. 27).

However, in my view, Dr. Reynolds’ degree of implied accuracy is not supported by the record. As noted in this Report, in my professional opinion and based on my professional experience, it is unreasonable to expect that any individual could recall their *exact* daily water intake from their time on Base decades ago. Further, variations in water intake from one day to the next are expected as “...individual water requirements can vary greatly on a day-to-day basis because of differences in physical activity, climates, and dietary contents” (Armstrong and Johnson 2018, pgs. 1-2). Therefore, I did not assume that plaintiff-derived information on amounts of water (or water-based drinks such as coffee) are *exact* amounts consumed by a plaintiff every day for their entire time at Camp Lejeune. Rather, I used the plaintiff deposition water intake information to describe whether the use of the CTE and RME values are indicated (i.e., does the CTE/RME range of water intakes include the water consumption amounts that were generally recalled by the plaintiff?). Thus, this degree of implied accuracy in Dr. Reynold’s Report is not supported by the record.

11.2 Differences specific to Ms. Amsler

11.2.1 Dates on Base

Dr. Reynolds assumed that Ms. Amsler consumed water on Base between October 1965 and June 1967.

This differs from my assumptions about Ms. Amsler’s on-Base activities. I modeled her exposure via drinking water consumption using data for the time-period May 1966 - June 1967. Before this time, she lived off Base (see Section 6.2 in this Report). During the time she lived off Base, Ms. Amsler described visiting the Base with her mother for shopping or to occasionally use a pool (Karen Amsler April 16, 2024 Deposition Transcript, pgs. 97-98). I did not note any information suggesting that she regularly drank water during these Base visits. I therefore did not estimate

water ingestion-related exposures for the time-period Ms. Amsler lived off Base (October 1965 – April 1966). Dr. Reynolds’ assumption that Ms. Amsler drank water on Base twice per week during this time-period (Expert Report of Kelly A. Reynolds, Chart 1, pg. 63) is not supported by the record and is therefore unsubstantiated.

11.2.2 Water systems

During her time living on Base (May 1966 – June 1967), I included the possibility that Ms. Amsler could have consumed water from the Tarawa Terrace water system and so estimated daily ingestion exposures for both Hadnot Point and Tarawa Terrace water systems. Dr. Reynolds assessed only exposure associated with water consumption from the Hadnot Point water system.

12. CONCLUSIONS

People living and working at Camp Lejeune from the 1950's to the 1980's may have been exposed to PCE, TCE, DCE, VC and/or benzene due to the presence of these chemicals in finished water at Camp Lejeune.

Dr. Spiliotopoulos (Expert Report, 2024, pgs. 68-69) stated that "For Hadnot Point, as with Tarawa Terrace, ATSDR assumed concentrations of contaminants in the influent to the WTP [water treatment plant] were equal to the concentrations of contaminants in the 'finished water' that was delivered to consumers...This assumption is incorrect, as treatment of the influent to the treatment plant resulted in evaporative and other losses, reducing contaminant concentrations in the 'finished' water." *Based on this opinion, the concentrations of chemicals of interest used in this Report, derived from ATSDR modeling, would be an overestimate of chemical concentrations in water used by people at Camp Lejeune.*

The routes of exposure could have included:

- Ingestion (for example, drinking the water, using the water for cooking, drinking small amounts of water during swimming)
- Inhalation (breathing the chemicals that volatilized from the water during activities such as showering, bathing, swimming, or using appliances such as washing machines)
- Skin contact (dermal exposure from contacting the water during activities such as showering, bathing, hand washing, or swimming)

The exposure assessment in this Report is intended to capture exposures experienced by people residing and/or working at Camp Lejeune during a time-period specific to the Plaintiff's actual time on Base (with some conservative assumptions) combined with exposure-related information generally considered to be representative of people on Base. The exposure assessment in this Report is not a perfectly accurate representation of exposure to a specific individual because the information necessary to develop such a representation is not available. For example, no contemporaneous documentation (e.g., diaries) describing day-to-day activities was identified. However, exposures can still be assessed by making assumptions derived from information from depositions, other sources of information related to the United States population, the military in general, Camp Lejeune specifically, and my best professional judgment.

Using these existing data in conjunction with modeled water concentration data, I was able to draw conclusions about Ms. Amsler's likely exposures to PCE, TCE, DCE, VC, and benzene to a reasonable degree of scientific certainty, considering my use of ATSDR's modeled chemical concentrations in water, as detailed in this Report. Where possible, conservative assumptions were made for determining model inputs. Conservative assumptions are used to avoid

underestimating exposures. Therefore, Ms. Amsler's actual exposures are unlikely to be higher than the exposure estimates produced by these models. These exposure estimates can be used in risk assessments to determine whether people who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in similar areas, and engaged in similar activities had an increased risk of disease (this is addressed in the Expert Report of Dr. Lisa Bailey for Karen Amsler).

Based on the information described in this Report, I conclude that children who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in a similar area, and engaged in similar activities could have been exposed to the following concentrations of the chemicals of interest via residential dermal contact with water and inhalation of chemicals volatilized from the water:

- Daily average exposure estimates via inhalation for TCE are 8.1 and 15 $\mu\text{g/kg/day}$ and via dermal contact are 0.076 and 0.12 $\mu\text{g/kg/day}$.
- Daily average exposure estimates via inhalation for benzene are 0.27 and 0.48 $\mu\text{g/kg/day}$ and via dermal contact are 0.0028 and 0.0044 $\mu\text{g/kg/day}$.

Further, based on the information described in this Report, I conclude that children who resided at Camp Lejeune during the time-period that Ms. Amsler was there, who lived in a similar area, and who engaged in similar activities could have been exposed to the following concentrations of the chemicals of interest via ingestion of water, with the ranges reflecting different water sources and different likely behaviors:

- Daily exposure estimates via water ingestion for PCE range from 0.00075 to 0.0021 mg/kg/day .
- Daily exposure estimates via water ingestion for TCE range from 3.0E-05 to 0.00097 mg/kg/day .
- Daily exposure estimates via water ingestion for DCE range from 8.9E-05 to 0.00025 mg/kg/day .
- Daily exposure estimates via water ingestion for VC range from 4.6E-05 to 0.00013 mg/kg/day .
- Daily exposure estimates via water ingestion for benzene range from 1.4E-05 to 4.0E-05 mg/kg/day .

Finally, based on the information described in this Report, I conclude that average vapor concentrations of chemicals of interest at indoor pools at the times relevant to Ms. Amsler are as follows:

- Vapor concentration at indoor swimming pools for PCE is 3.89E+01 mg/m^3 .
- Vapor concentrations at indoor swimming pools for TCE are 8.92E-01 and 9.57E+00 mg/m^3 .
- Vapor concentration at indoor swimming pools for DCE is 2.46E+00 mg/m^3 .

- Vapor concentration at indoor swimming pools for VC is 3.79E+00 mg/m³.
- Vapor concentration at indoor swimming pools for benzene is 2.29E-01 mg/m³.

13. REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 2016. Exposure Dose Guidance for Body Weight. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, October 26.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2017. Public Health Assessment for Camp Lejeune Drinking Water. U.S. Marine Corps Base Camp Lejeune, North Carolina. January 20, 2017.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2018. Exposure Dose Guidance for Dermal and Ingestion Exposure to Surface Water. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Sept 25.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2022a. Estimating Site-Specific Ingestion and Dermal Exposure Doses. https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/epcs_and_exposure_calculations/estimating-site-specific-ingestion-and-dermal-exposure-doses.html
- Agency for Toxic Substances and Disease Registry (ATSDR). 2022b. Shower and Household Water-use Exposure (SHOWER) Model v3.0 User's Guide. September 2.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2022c. Technical Document for the Shower and Household Water-use Exposure (SHOWER) Model v3.0. May 4.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2023a. Exposure Dose Guidance for Soil/Sediment Dermal Absorption. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, July 2023.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2023b. Exposure Dose Guidance for Water Ingestion. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Jan 31.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2024. PHAST User Guide, Version 2.4.
- Anchal P, Kumari M, Gupta SK. 2020. Human health risk estimation and predictive modeling of halogenated disinfection by- products (chloroform) in swimming pool waters: a case study of Dhanbad, Jharkhand, India. J Environ Health Sci Engineer 18:1595–1605. <https://doi.org/10.1007/s40201-020-00578-6>.
- Armstrong LE, Johnson EC. 2018. Water intake, water balance, and the elusive daily water requirement. Nutrients 10(12):1928. doi: 10.3390/nu10121928.
- Baier-Anderson C, Blount BC, Lakind JS, Naiman DQ, Wilbur SB, Tan S. Estimates of exposures to perchlorate from consumption of human milk, dairy milk, and water, and comparison to current

reference dose. J Toxicol Environ Health A. 2006 Feb;69(3-4):319-30. doi: 10.1080/15287390500323420. PMID: 16407090.

Blando JD, Cohn P. 2004. Exposure and health risk from swimming in outdoor pools contaminated by trichloroethylene. Human and Ecological Risk Assessment: An International Journal 10(4):717–731. <https://doi.org/10.1080/10807030490484183>

Chowdhury S. 2015. Predicting human exposure and risk from chlorinated indoor swimming pool: a case study. Environ Monit Assess. 187(8):502. doi: 10.1007/s10661-015-4719-8.

Costa C, Assunção R, Sequeira D, Esteves F, Valdiglesias V, Laffon B, Teixeira JP, Madureira J. 2022. From trihalomethanes chronic daily intake through multiple exposure routes to cancer and non-cancer health risk assessment: Evidence from public Portuguese indoor swimming pools facilities using a probabilistic approach. Sci Total Environ 818:151790. doi: 10.1016/j.scitotenv.2021.151790.

Dehghani M, Shahsavani S, Mohammadpour A, Jafarian A, Arjmand S, Rasekhi MA, Dehghani S, Zaravar F, Derakhshan Z, Ferrante M, Oliveri Conti G. 2022. Determination of chloroform concentration and human exposure assessment in the swimming pool. Environ Res. 203:111883. doi: 10.1016/j.envres.2021.111883.

DeOreo WB, Mayer P, Dziegielewski B, Kiefer J. 2016. Residential End Uses of Water, Version 2. Water Research Foundation, Denver.

EarthCon. 2019. Human Health Risk Assessment, Kerr-McGee Chemical Corp - Navassa Superfund Site, Navassa, North Carolina. EPA ID #NCD980557805.

EFSA (European Food Safety Authority). 2012. SCIENTIFIC OPINION Guidance on selected default values to be used by the EFSA Scientific Committee, Scientific Panels and Units in the absence of actual measured data. EFSA Journal 10(3):2579. <https://efsa.onlinelibrary.wiley.com/doi/pdf/10.2903/j.efsa.2012.2579#:~:text=In%20this%20context%2C%20the%20term,and%20be%20therefore%20scientifically%20justified.>

Florida Department of Health. 2012. Health Consultation Off-site Groundwater, Surface Water, and Vapor Intrusion Flash Cleaners Pompano Beach, Broward County, Florida EPA Facility ID: FLD83111005. https://www.floridahealth.gov/environmental-health/hazardous-waste-sites/_documents/f/flashcleaners020812.pdf

Health Canada. 1999. Canadian exposure factors used in human health risk assessments Fact sheet series: Topics in risk assessment of substances under the Canadian Environmental Protection Act, 1999 (CEPA 1999). <https://www.canada.ca/content/dam/hc-sc/documents/services/chemical-substances/fact-sheets/canadian-exposure-factors-human-health-risk-assessments/fact-sheet-exposue-factors.pdf>.

Health Canada. 2021. Federal Contaminated Site Risk Assessment in Canada: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA) VERSION 3.0.

Health Canada. 2025. Additional risk characterization document – Update to the human health assessment of melamine. January. <https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/additional-rcd-update-human-health-assessment-melamine.html#toc2>

Huerta D, Schobel T, Alexander-Ozinskas A, Hild J, Lauder J, Reynolds P, Von Behren J, Meltzer D, Ramírez-Andreotta MD. 2023. Probabilistic risk assessment of residential exposure to metal(loid)s in a mining impacted community. *Sci Total Environ.* 872:162228. doi: 10.1016/j.scitotenv.2023.162228.

IPCS (International Programme on Chemical Safety). 2004. IPCS Risk Assessment Terminology. IPCS harmonization project document n. 1. <https://www.who.int/publications/i/item/9241562676>

Jang W, Aral MM. 2008. Analyses of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water at Tarawa Terrace and Vicinity, U.S. Marine Corps Base Camp Lejeune, North Carolina: Historical Reconstruction and Present-Day Conditions – Chapter G: Simulation of Three Dimensional Multispecies, Multiphase Mass Transport of Tetrachloroethylene (PCE) and associated Degradation By-Products. Atlanta, GA: Agency for Toxic Substances and Disease Registry.

Khan K, Khan MS, Younas M, Yaseen M, Al-Sehemi AG, Kavil YN, Su C, Ali N, Maryam A, Liang R. 2024. Pathways and risk analysis of arsenic and heavy metal pollution in riverine water: Application of multivariate statistics and USEPA-recommended risk assessment models. *J Contam Hydrol.* 269:104483. doi: 10.1016/j.jconhyd.2024.104483.

Lowe JA, Jamall IS. 1994. Assessing health risks associated with DDT residues in soils in California: a Proposition 65 case study. *Risk Anal.* 14(1):47-52. doi: 10.1111/j.1539-6924.1994.tb00027.x.

Maslia ML, Sautner JB, Faye RE, Suárez-Soto RJ, Aral MM, Grayman WM, Jang W, Wang J, Bove FJ, Ruckart PZ, Valenzuela C, Green JW, Krueger AL. 2007. Analyses of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water at Tarawa Terrace and Vicinity, U.S. Marine Corps Base Camp Lejeune, North Carolina: Historical Reconstruction and Present-Day Conditions. Chapter A: Summary of Findings. Appendix A2.

Maslia ML, Suárez-Soto RJ, Sautner JB, Anderson BA, Jones LE, Faye RE, Aral MM, Guan J, Jang W, Telci IT, Grayman WM, Bove FJ, Ruckart PZ, Moore SM. 2013. Analyses and Historical Reconstruction of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water Within the Service Areas of the Hadnot Point and Holcomb Boulevard Water Treatment Plants and Vicinities, U.S. Marine Corps Base Camp Lejeune, North Carolina, Chapter A: Summary and Findings. Appendix A7.

Maslia ML, Aral MM, Ruckart PZ, Bove FJ. 2016. Reconstructing Historical VOC Concentrations in Drinking Water for Epidemiological Studies at a U.S. Military Base: Summary of Results. *Water (Basel)*. 8(10):449. doi: 10.3390/w8100449. Epub 2016 Oct 13. PMID: 28868161; PMCID: PMC5580837.

Minh Chau KN, Carroll K, Li X-F. 2025. Swimming benefits outweigh risks of exposure to disinfection byproducts in pools. *Journal of Environmental Sciences* 152:527-534. <https://doi.org/10.1016/j.jes.2024.05.040>.

NRC (National Research Council). 1981. US Committee on Indoor Pollutants. *Indoor Pollutants*. Washington (DC): National Academies Press (US). VI, Monitoring and Modeling of Indoor Air Pollution. <https://www.ncbi.nlm.nih.gov/books/NBK234059/>

NRC (National Research Council). 1983. *Risk Assessment in the Federal Government: Managing the Process*. Washington, DC: The National Academies Press. <https://doi.org/10.17226/366>.

Oregon Department of Environmental Quality Environmental Cleanup Program. 2010. Human Health Risk Assessment Guidance. October. <https://www.oregon.gov/deq/filterdocs/humanhealthriskassessmentguidance.pdf>

Pándics T, Hofer Á, Dura G, Vargha M, Szigeti T, Tóth E. 2018. Health risk of swimming pool disinfection by-products: a regulatory perspective. *J Water Health*. 16(6):947-957. doi: 10.2166/wh.2018.178.

Peng F, Peng J, Li H, Li Y, Wang B, Yang Z. 2020. Health risks and predictive modeling of disinfection byproducts in swimming pools. *Environ Int*. 139:105726. doi: 10.1016/j.envint.2020.105726.

Phillips L, Moya J. The evolution of EPA's Exposure Factors Handbook and its future as an exposure assessment resource. *J Expo Sci Environ Epidemiol*. 2013 Jan-Feb;23(1):13-21. doi: 10.1038/jes.2012.77. Epub 2012 Jul 18. PMID: 22805985.

Prud'homme de Lodder LCH, Bremmer HJ, Pelgrom SMGJ, Park MVDZ, van Engelen JGM. 2006. Disinfectant Products Fact Sheet. To assess the risks for the consumer. RIVM report 320005003/2006.

Ramirez-Andreotta MD, Brusseau ML, Beamer P, Maier RM. 2013. Home gardening near a mining site in an arsenic-endemic region of Arizona: assessing arsenic exposure dose and risk via ingestion of home garden vegetables, soils, and water. *Sci Total Environ*. 454-455:373-82. doi: 10.1016/j.scitotenv.2013.02.063.

Salhotra AM. 2011. Human health risk assessment for contaminated properties. *Progress in Molecular Biology and Translational Science*, Volume 112. ISSN 1877-1173.

Sander R. 2023. Compilation of Henry's law constants (version 5.0.0) for water as solvent, *Atmos Chem Phys* 23:10901-12440. doi:10.5194/acp-23-10901-2023.

US Army Corps of Engineers CEMP-RT Washington, D.C. 20314-1000 Manual No. 200-1-4 31 December 2010 Environmental Quality Risk Assessment Handbook, Volume II: Environmental Evaluation. Department of the Army EM 200-1-4. <https://perma.cc/KV4J-AQDM>

US EPA (US Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response. EPA/540/1-89/002. https://www.epa.gov/sites/default/files/2015-09/documents/rags_a.pdf

US EPA (US Environmental Protection Agency). 1992a. Guidelines for Exposure Assessment. EPA/600/Z-92/001. May. https://www.epa.gov/sites/default/files/2014-11/documents/guidelines_exp_assessment.pdf

US EPA (US Environmental Protection Agency). 1992b. Dermal Exposure Assessment: Principles and Applications. Office of Health and Environmental Assessment, Office of Research and Development. January.

US EPA (US Environmental Protection Agency). 2000. Volatilization rates from water to indoor air. Phase II. Washington, DC: Office of Research and Development, EPA/600/R-00/096. October. <https://perma.cc/EF9E-J3ZG>

US EPA (US Environmental Protection Agency). 2003. User's Manual Swimmer Exposure Assessment Model (SWIMODEL) Version 3.0. U.S. Environmental Protection Agency Office of Pesticide Programs Antimicrobials Division. November 2003. <https://www.epa.gov/sites/default/files/2015-09/documents/swimodel-users-guide.pdf>

US EPA (US Environmental Protection Agency). 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Final. EPA/540/R/99/005 OSWER 9285.7-02EP PB99-963312. July.

US EPA (US Environmental Protection Agency). 2005. Guidelines for Carcinogen Risk Assessment Risk Assessment Forum Washington, DC. EPA/630/P-03/001F March.

US EPA (US Environmental Protection Agency). 2009. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment). Final. EPA-540-R-070-002. OSWER 9285.7-82. January.

US EPA (US Environmental Protection Agency). 2011. Exposure Factors Handbook: 2011 Edition. Washington, DC: National Center for Environmental Assessment. EPA/600/R-090/052A. <https://perma.cc/P6D7-Z7LG>

US EPA (US Environmental Protection Agency). 2019. Update for Chapter 3 of the Exposure Factors Handbook Ingestion of Water and Other Select Liquids. Washington, DC: National Center for Environmental Assessment. February. EPA/600/R-18/259F. https://www.epa.gov/sites/default/files/2019-02/documents/efh_-_chapter_3_update.pdf

US EPA (US Environmental Protection Agency). 2022. Conducting a Human Health Risk Assessment. <https://www.epa.gov/risk/conducting-human-health-risk-assessment>

US EPA (US Environmental Protection Agency). 2024. Swimmer Exposure Assessment Model (SWIMODEL). <https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/swimmer-exposure-assessment-model-swimodel>

Weed DL. 2007. The Nature and Necessity of Scientific Judgement, 15 J. L. & Pol'y. <https://brooklynworks.brooklaw.edu/jlp/vol15/iss1/6>

Wilczynski SM. 2017. Section IV. Professional Judgment. A Practical Guide to Finding Treatments That Work for People with Autism. ISBN 978-0-12-809480-8. Academic Press.

World Health Organization (WHO). 2020. Model documentation: WHO Household Multiple Emission Sources (HOMES) Model. <https://www.who.int/publications/m/item/household-multiple-mission-sources-homes-model>

APPENDIX 1: Curriculum Vitae for Judy S. LaKind, Ph.D.

Judy S. LaKind, Ph.D.

LaKind Associates, LLC

Catonsville MD USA 21228 410.788.8639 lakindassoc@gmail.com

Judy S. LaKind, Ph.D., President of LaKind Associates, LLC, and Adjunct Associate Professor, Department of Epidemiology and Public Health, University of Maryland School of Medicine is a health and environmental scientist with expertise in exposure science, assessment of human health risks, biomonitoring, scientific and technical analysis for regulatory support, and state-of-the-science and systematic reviews. She has managed a wide array of successful projects, with completion in a timely manner and within budget, and has organized and facilitated numerous workshops on a variety of scientific subjects. Dr. LaKind has spoken and published extensively on exposure- and risk-related issues, including children's exposures to environmental chemicals, the implications of uncertainty in the risk assessment process, data quality, use of environmental epidemiology research in public health decision-making, weighing potential risks and benefits related to chemical use, the presence of environmental chemicals in human milk, and time-dependence and distributional analysis of exposure. Dr. LaKind has evaluated the use of human health risk assessment in the development of water quality criteria, and has critically analyzed the environmental fate, behavior, and bioavailability of pollutants in the context of setting regulatory criteria. She has developed risk assessments for a variety of urban industrial sites, military bases, and firing ranges, and has utilized state-of-the-science models for estimating blood lead levels in adults and children.

Previously, Dr. LaKind was a geologist at the US EPA's Office of Federal Activities, where she was responsible for the evaluation of Environmental Impact Statements and legislative reports. Dr. LaKind has taught graduate level courses at The Johns Hopkins University and the University of Maryland in risk assessment and aquatic chemistry. Dr. LaKind is Insights Editor for *Environment International*. She also serves on the editorial board of the *Journal of Environmental Exposure Assessment* and is past Associate Editor for the *Journal of Exposure Science and Environmental Epidemiology* and past editorial board member of the *Journal of Toxicology and Environmental Health*.

Dr. LaKind is a Past President of the International Society of Exposure Science. She was a member of the Health Effects Institute Energy Research Committee and the Maryland Department of Health and Mental Hygiene Cancer Cluster Advisory Committee and was a Junior Councilor, Society of Toxicology's Exposure Specialty Section. She previously served on the Boards of the National Swimming Pool Foundation and the Coalition Against Childhood Lead Poisoning (with a term as president). She is a former member of Maryland's Children's Environmental Health and Protection Advisory Council, the Lead Poisoning Prevention Commission, the Maryland Pesticide Reporting and Information Workgroup, the HESI RISK21 Advisory Board, and the World Health Organization Survey Coordinating Committee for the WHO Global Survey of Human Milk for Persistent Organic Pollutants (POPs). Dr. LaKind also served on the Institute of Medicine Committee on Blue Water Navy Vietnam Veterans and Agent Orange Exposure and the US Environmental Protection Agency Science Advisory Board Panel on Perchlorate - Approaches for Deriving Maximum Contaminant Level Goals for Drinking Water.

Academic Appointments:

Fellow-by-Courtesy, The Johns Hopkins University, Department of Applied Mathematics and Statistics.
February 2013 – present.

Adjunct Associate Professor, University of Maryland School of Medicine, Department of Epidemiology and Preventive Medicine, August 2003 – August 2008; August 2009 – October 2009. February 2012 – present.

Associate Professor, University of Maryland School of Medicine, Department of Epidemiology & Public Health, September 2008 – August 2009; November 2009-February 2012.

Part Time Instructor, College of Engineering & Information Technology at University of Maryland Baltimore County, January 2010 – June 2010.

Adjunct Associate Professor, University of Maryland School of Law, May 2003 – May 2004.

Adjunct Associate Professor, Penn State College of Medicine, Department of Pediatrics, Milton S. Hershey Medical Center, 2002 – 2016.

Education:

Ph.D.; The Johns Hopkins University; Geography and Environmental Engineering; 1988
M.S.; The University of Wisconsin, Madison; Geology; 1984
B.A.; The Johns Hopkins University; Earth and Planetary Sciences; 1982

Litigation Support Training, 1994
Project Manager Training, 1995
Mid-America Toxicology Course, 1995
Risk Communication, 1995
Hershey Medical College Investigator Certification for Protecting Human Subjects, 2004
CITI Course in the Protection of Human Research Subjects, 2014
CITI Course in Institutional/Signatory Official: Human Subject Research, 2022
CITI Course in Community-Engaged and Community-Based Participatory Research, 2022
CITI Course in The Protection of Human Subjects, 2022

Experience:

Human Health Risk Assessment/Product Stewardship – Developed distributional exposure analyses for body burdens of persistent organic chemicals in breastfed infants. Conducted site-specific, health-based risk assessments for urban industrial sites, military bases, and firing ranges, with emphasis on PAHs, heavy metals (including lead), and volatile organic compounds. Developed exposure scenarios, with appropriate assumptions and parameters, for on-site and off-site exposure pathways, including recreational scenarios. These assessments included determination of receptors-of-concern and the development of site-specific conceptual site models as per U.S. EPA criteria. Prepared risk assessments under Maryland's Voluntary Cleanup Program. Utilized state-of-the-science models for predicting blood lead levels in adults and children. Evaluated and utilized model developed by the American Water Works Association to predict disinfection by-product formation resulting from chlorination of drinking water for zebra mussel control. Managed the

development of technical papers which utilized innovative methodologies to correlate reductions of atmospheric concentrations of lead, carbon monoxide, ozone, and air toxics with improvements in human health. Performed literature research, prepared manuscripts and comments for the USEPA, and provided litigation and regulatory support in evaluation of toxicity and environmental impacts of ethylene glycol (EG), propylene glycol (PG), and EG and PG de-icing and anti-icing formulations.

Systematic Review: Published multiple medium- and chemical-specific systematic and critical reviews. Invited member of the Risk Of Bias In Non-randomized Studies of Exposures (ROBINS-E) Working Group and participated in the GRADE Guidance for Modelled Data Working Group. Developed instrument for assessing study quality as part of systematic review (Biomonitoring, Environmental Epidemiology, and Short-Lived Chemicals - BEES-C – instrument); approach is now used by the US Environmental Protection Agency.

Project Management – Over 30 years of project management experience with teams of scientists from both inside and outside the US; focus on team communication and meeting client expectations regarding deliverables, deadlines, and budget.

Scientific workshop/expert panel development - Developed, coordinated, and facilitated numerous expert panels and workshops on a wide range of topics including environmental chemicals in breast milk, interpretation and communication of biomonitoring data, neurodevelopmental function testing, exposure to disinfection byproducts in swimming pool environments and associated health effects, biomonitoring of chemicals with short physiologic half-lives, and disease cluster methodologies.

Criteria Development - Determined scientific issues associated with the use of bioconcentration factors for regulating hydrophobic organic chemicals (HOCs), including dioxin. Developed an alternative risk assessment formula for HOC criteria determination.

Litigation Support - Provided litigation support for pulp and paper industry counsel on issues associated with aquatic organism accumulation of dioxin. Provided seminars to pulp and paper industry counsel on dioxin bioaccumulation. Provided litigation support for chemical industry on relative toxicity and environmental fate of a group of widely used compounds. Completed Litigation Support training course.

Regulatory Review - As an invited member of the Washington State Department of Health/Department of Ecology Sediment Scientific Review Board, provided scientific evaluation of proposed method for development of marine sediment chemical criteria relative to human health. Provided regulatory review, update, and analysis of: Clean Water Act 304(l) listing and approval/disapproval process; EPA pulp and paper mill guidance documents; and states' development of dioxin water quality criteria, for the pulp and paper industry. Critiqued bioaccumulation section of EPA's Great Lakes Water Quality Initiative. Analyzed scientific basis for proposed particulate matter standard.

Lead - Former member of the Coalition Against Childhood Lead Poisoning (with a term as president) and the Maryland Lead Poisoning Prevention Commission. Managed and conducted risk assessments for sites with lead contamination. Evaluated potential for human health risks associated with lead exposure to soil, water, and air, at firing ranges, and at residential, urban, and industrial sites. Utilized state-of-the-science models for predicting blood lead levels in both adults and children and has explored the utility of these models for assessing blood lead levels in people exposed to lead-contaminated media on an episodic basis. Made

presentations to the public and media on risks associated with exposure to lead and created risk communication documentation on childhood lead poisoning prevention, used by the Kennedy-Krieger Institute's Lead Poisoning Prevention Program and the Baltimore City Department of Health. Technical editor of HUD's Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing.

Document Review and Analysis - Conducted Record of Decision search and analysis for development of remediation strategy for mitigation of subsurface migration of DNAPL. Performed scientific review, analysis, and critique of a wide range of documents including: Environmental Impact statements associated with Federal Energy Regulatory Commission hydroelectric power projects, natural gas pipeline siting, dredging projects; legislative reports on the Arctic National Wildlife Refuge and offshore oil exploration near the Georges Bank; risk assessments on formaldehyde air emissions from a particleboard plant and aquatic organism contamination in the Sacramento River; Endangerment Assessment and RI/FS of sawmill and landfill Superfund site.

Risk Communication - Gave presentations to public and media on risks associated with exposure to lead. Created risk communication information on childhood lead poisoning prevention, including *Derek the Dinosaur's Coloring Book About Lead*, used by the Kennedy-Krieger Institute's Lead Poisoning Prevention Program and the Baltimore City Department of Health. Coloring book was also used by Lead Safe St. Louis where it was translated into Spanish, Bosnian, Somali, Dari, and Vietnamese. Assisted in the development of a decision support document and white paper outlining the health risks and benefits associated with continued use of MTBE in the U.S. Assisted in the development of a Risk Primer for a major trade association.

Teaching - University of Maryland School of Law: Environmental Law and Science. The Johns Hopkins University: graduate-level courses on aquatic chemistry and environmental risk assessment. University of Maryland Baltimore County: upper-level course on human health risk assessment.

Professional Affiliations:

American Public Health Association (APHA) (1999-2015)
Maryland Public Health Association (Board member, 2008-2009)
American Chemical Society, Environmental Division (ACS)
Int. Society for Children's Health and the Environment (ISCHE), Founding member (2009-2015)
International Society of Exposure Science (ISES)
Society for Risk Analysis (SRA)
Society of Toxicology (SOT)
SOT Exposure Specialty Section, founding member (2017-present)

Selected Publications:

Macey K, Lange R, Apel P, Poddalgoda D, Calafat AM, Kolossa-Gehring M, LaKind JS, Melnyk LJ, Nakayama SF, St-Amand A. 2025. Human biomonitoring health-based guidance values: A case study of the HB2GV Dashboard and DEHP. *International Journal of Hygiene and Environmental Health*. Vol 263. <https://doi.org/10.1016/j.ijheh.2024.114490>

Rule AM, Wagner FA, Negi N, Tajouh-Daghuie CJ, Rosman L, Naiman J, Lange SS, Clougherty JE, Vorhees D, LaKind JS. Principles and elements for creating and sustaining successful public-private partnerships (PPPs) for environmental community monitoring programs: Results from a scoping review and interviews. *Environmental Justice* doi.org/10.1089/env.2024.002

Higgins JPT, Morgan RL, Rooney AA, Taylor KW, Thayer KA, Silva RA, Lemeris C, Akl EA, Bateson TF, Berkman ND, Glenn BS, Hróbjartsson A, LaKind JS, McAleenan A, Meerpohl JJ, Nachman RM, Obbagy JE, O'Connor A, Radke EG, Savović J, Schünemann HJ, Shea B, Tilling K, Verbeek J, Viswanathan M, Sterne JAC. 2024. A tool to assess risk of bias in non-randomized follow-up studies of exposure effects (ROBINS-E). *Environment International* 186:108602.

Ebelt S, Baxter L, Erickson HS, Henneman LRF, Lange S, Luben TJ, Neidell M, Rule AM, Russell AG, Wendt Hess J, Burns CJ, LaKind JS, Goodman JE. 2023. Air pollution accountability research: Moving from a chain to a web. *Global Epidemiology* 100128. ISSN 2590-1133 <https://doi.org/10.1016/j.gloepi.2023.100128>.

LaKind JS, Burns CJ, Johnson GT, Lange SS. 2023. Epidemiology for risk assessment: US EPA guidance and the Matrix. *Hygiene and Environmental Health Advances* 106:100059. <https://doi.org/10.1016/j.heha.2023.100059>

LaKind JS. 2023. Invited Perspective. PFAS and infant nutrition: Why aren't we monitoring? *Environmental Health Perspectives* 131(3): <https://doi.org/10.1289/EHP12134>.

LaKind JS, Naiman J, Verner M-A, Lévêque L, Fenton S. 2023. Per- and polyfluoroalkyl substances (PFAS) in breast milk and infant formula: A global issue. *Environmental Research* 219:115042.

Nakayama SF, St-Amand A, Pollock T, Ashley-Martin J, Bamai YA, Barr DB, Bessems J, Calafat A, Castaño A, Covaci A, Duca RC, Faure S, Galea KS, Hays S, Hopf NB, Ito Y, Jeddi MZ, Kolossa-Gehring M, Kumar E, LaKind JS, López ME, Louro H, Makris KC, Melnyk L, Naiman J, Nassif J, Noisel N, Quirós-Alcalá L, Rafiee A, Rambaud L, Silva MJ, Ueyama J, Verner M-A, Waras MN, Werry K. 2023. Interpreting Biomonitoring Data: Introducing the i-HBM Working Group's Guidance Value Dashboard. *International Journal of Hygiene and Environmental Health* 247:114046.

Wilson AM, Mussio I, Chilton S, Gerald LB, Jones RA, Drews FA, LaKind JS, Beamer PI. 2022. A novel application of risk-risk tradeoffs in occupational health: Nurses' occupational asthma and infection risk perceptions related to cleaning and disinfection during COVID-19. *International Journal of Environmental Research and Public Health* 19(23):16092 <https://doi.org/10.3390/ijerph192316092>

LaKind JS, Burns CJ, Donald R, Mattison DR. 2022. Commentary: Systematic reviews and observational epidemiology: the more things change... *Global Epidemiology* 4:100088.

LaKind JS, Burns CJ, Naiman DQ. 2,4-D and NHANES: Sources of exposure and identification of data gaps. *Hygiene and Environmental Health Advances* 4:100023.

- Burns CJ, LaKind JS, Naiman J, Boon D, Clougherty JE, Rule AM, Zidek A. Research on COVID-19 and air pollution: A path towards advancing the science. *Environmental Research* 212, Part A:113240. <https://www.sciencedirect.com/science/article/pii/S0013935122005679?via%3Dihub>
- LaKind JS, Verner M-A, Rogers R, Goeden H, Naiman DQ, Marchitti S, Lehmann G, Hines E, Fenton SE. 2022. Current breast milk PFAS levels in North America: After all this time why don't we know more? *Environmental Health Perspectives* 130(2): 25002. <https://doi.org/10.1289/EHP10359>
- Burns CJ, LaKind JS. 2022. Elements to increase translation in pyrethroid epidemiology research: a review. *Science of the Total Environment* 813:152568 doi: 10.1016/j.scitotenv.2021.152568.
- Burns CJ, LaKind JS. 2021. Using the Matrix to bridge the epidemiology/risk assessment gap: A case study of 2,4-D. *Critical Reviews in Toxicology* 51(7):591-599. doi: 10.1080/10408444.2021.1997911
- LaKind JS, Burns CJ, Pottenger LH, Naiman DQ, Goodman JE, Marchitti SA. 2021. Does ozone inhalation cause adverse metabolic effects in humans? A systematic review. *Critical Reviews in Toxicology* 51(6):467-508. DOI: 10.1080/10408444.2021.1965086
- Verner M-A, Salame H, Housand C, Birnbaum LS, Bouchard M, Chevrier J, Aylward L, Naiman DQ, LaKind JS. 2020. How many urine samples are needed to accurately assess exposure to non-persistent chemicals? The Biomarker Reliability Assessment Tool (BRAT) for scientists, research sponsors and risk managers. *International Journal of Environmental Research and Public Health* 17:9102; doi:10.3390/ijerph17239102.
- Brozek J, Canelo C, Akl E, Bowen J, Bucher J, Chiu W, Cronin M, Djulbegovic B, Falavigna M, Guyatt G, Gordon A, Hilton Boon M, Hutubessy R, Joore M, Katikireddi S, LaKind J, Langendam M, Manja V, Magnuson K, Mathioudakis A, Meerpohl J, Mertz D, Mezencev R, Morgan R, Morgano GP, Mustafa R, Naidoo B, O'Flaherty M, Patlewicz G, Riva J, Posso M, Ringborg A, Rooney A, Schlosser P, Schwartz L, Shemilt I, Tarride J-E, Thayer K, Tsaioun K, Vale L, Wambaugh J, Wignall JA, Williams AR, Xie F, Zhang Y, Schünemann H. 2020. GRADE Guidelines 30: The GRADE Approach to Assessing the Certainty of Evidence from Models – an Overview in the Context of Health Decision-making. *Journal of Clinical Epidemiology* S0895-4356(20)31103-3.
- Goodman M, Li J, Flanders WD, Mahood D, Anthony LG, Zhang Q, LaKind JS. 2020. Epidemiology of PCBs and neurodevelopment: Systematic assessment of multiplicity and completeness of reporting. *Global Epidemiology* 2:100040.
- LaKind JS. 2020. Foreward. Total Exposure Health: An Introduction. Editors: Phillips KA, Yamamoto DP, Racz L. CRC Press.
- LaKind JS, Naiman J, Burns CJ. Translation of exposure and epidemiology for risk assessment: A shifting paradigm. 2020. *International Journal of Environmental Research and Public Health* 17(12):4220; <https://doi.org/10.3390/ijerph17124220>.
- LaKind JS, Burns CJ, Erickson H, Graham SE, Jenkins S, Johnson GT. 2020. Bridging the epidemiology risk assessment gap: An NO₂ case study of the Matrix. *Global Epidemiology* 2:100017.
- LaKind JS, Goodman M. 2019. Methodological evaluation of human research on asthmagenicity and occupational cleaning: A case study of quaternary ammonium compounds (“quats”). *Allergy, Asthma & Clinical Immunology* 15:69.

Burns CJ, LaKind JS, Mattison DR, Alcala CS, Branch F, Castillo J, Clark A, Clougherty JE, Darney SP, Erickson H, Goodman M, Greiner M, Jurek AM, Miller A, Rooney AA, Zidek A. 2019. A Matrix for bridging the epidemiology and risk assessment gap. *Global Epidemiology* 1: 100005.

LaKind JS, Pollock T, Naiman DQ, Kim S, Nagasawa A, Clarke J. 2019. Factors affecting interpretation of national biomonitoring data from multiple countries: BPA as a case study. *Environmental Research* 173:318-329. PMID: 30951958

LaKind JS, O'Mahony C, Armstrong T, Tibaldi R, Blount BC, Naiman DQ. 2019. ExpoQual: Evaluating measured and modeled human exposure data. *Environmental Research* 171:302–312.

LaKind JS, Idri F, Naiman DQ, Verner M-A. 2019. Biomonitoring and nonpersistent chemicals – understanding and addressing variability and exposure misclassification. *Current Environmental Health Reports* 6(1):16-21.

LaKind JS, Davis M, Lehmann GM, Hines E, Marchitti SA, Alcala C, Lorber M. 2019. Infant dietary exposures to environmental chemicals and infant/child health: A critical assessment of the literature. *Environmental Health Perspectives* 126(9):96002. Highlighted in: Arnold C. 2019. Baby steps forward: Recommendations for better understanding environmental chemicals in breast milk and infant formula. *Environmental Health Perspectives* <https://doi.org/10.1289/EHP4804>

Lehmann GM, LaKind JS, Davis M, Hines E, Marchitti SA, Alcala C, Lorber M. 2019. Environmental chemicals in breast milk and formula: Exposure and risk assessment implications. *Environmental Health Perspectives* 126(9):96001. Highlighted in: Arnold C. 2019. Baby steps forward: Recommendations for better understanding environmental chemicals in breast milk and infant formula. *Environmental Health Perspectives* <https://doi.org/10.1289/EHP4804>

Metwally DE, Chain K, Stefanak MP, Alwis U, Blount BC, LaKind JS, Bearer CF. 2018. Urinary metabolites of volatile organic compounds of infants in the neonatal intensive care unit. *Pediatric Research* 83(6):1158-1164.

Goodman M, Naiman DQ, LaKind JS. 2018. Systematic review of the literature on triclosan and health outcomes in humans. *Critical Reviews in Toxicology* 48(1):1-51.

LaKind JS, Burns CJ, Naiman DQ, O'Mahony C, Vilone G, Burns AJ, Naiman JS. 2017. Critical and systematic evaluation of data for estimating human exposures to 2,4-dichlorophenoxyacetic acid (2,4-D) - quality and generalizability. *Journal of Toxicology and Environmental Health, Part B*. 20(8):423-446.

LaKind JS, Anthony LG, Goodman M. 2017. Review of reviews on exposures to synthetic organic chemicals and children's neurodevelopment: Methodological and interpretation challenges. *Journal of Toxicology and Environmental Health, Part B* 20(8):390-422.

Vincent MJ, Bernstein JA, Basketter D, LaKind JS, Dotson GS, Maier A. 2017. Chemical-induced asthma and the role of clinical, toxicological, exposure and epidemiological research in regulatory and hazard characterization approaches. *Regulatory Toxicology & Pharmacology* 90: 126-132.

LaKind JS, Overpeck J, Breyse PN, Backer L, Richardson S, Sobus J, Sapkota A, Upperman CR, Jiang C, Beard CB, Brunkard JM, Bell J, Harris R, Chretien J-P, Peltier RE, Chew GL, Blount B. 2016. Exposure

science in an age of rapidly changing climate: Challenges and opportunities. *Journal of Exposure Science and Environmental Epidemiology* 26: 529-538.

Beck NB, Becker RA, Erraguntla N, Farland WH, Grant RL, Gray G, Kirman C, LaKind JS, Lewis RJ, Nance P, Pottenger LH, Santos SL, Shirley S, Simon T, Dourson ML. 2016. Approaches for describing and communicating overall uncertainty in toxicity characterizations: U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) as a case study. *Environment International* 89–90:110–128.

Weldon RH, LaKind JS. 2015. Biomonitoring of dioxins and furans: Levels and trends in humans. In: *The Handbook of Environmental Chemistry*. ISSN 1867-979X. Springer:Berlin Heidelberg. 23 pp. doi: 10.1007/698_2015_433. http://link.springer.com/chapter/10.1007/698_2015_433

LaKind JS, Goodman M, Makris SL, Mattison DR. 2015. Improving concordance in environmental epidemiology: A three-part proposal. *Journal of Toxicology & Environmental Health, Part B*. 18(2):105-120.

LaKind JS, Naiman, DQ. 2015. Temporal trends in bisphenol A exposure in the United States from 2003–2012 and factors associated with BPA exposure: Spot samples and urine dilution complicate data interpretation. *Environmental Research* 142:84–95.

LaKind JS, Goodman M, Barr DB, Weisel CP, Schoeters G. 2015. Lessons learned from the application of BEES-C: Systematic assessment of study quality of epidemiologic research on BPA, neurodevelopment, and respiratory health. *Environment International* 80:41-71.

LaKind JS, Sobus JR, Goodman M, Barr DB, Fürst P, Albertini RJ, Arbuckle TE, Schoeters G, Tan Y-M, Teeguarden J, Tornero-Velez R, Weisel CP. 2014. A proposal for assessing study quality: Biomonitoring, Environmental Epidemiology, and Short-Lived Chemicals (BEES-C) Instrument. *Environment International* 73C:195-207.

Mattison DR, Karyakina N, Goodman M, LaKind JS. 2014. Pharmacokinetics of selected exogenous and endogenous estrogens: A review of the data and identification of knowledge gaps. *Critical Reviews in Toxicology* 44(8):696-724.

Lehmann GM, Verner MA, Luukinen B, Henning C, Assimon SA, LaKind JS, McLanahan ED, Phillips LJ, Davis MH, Powers CM, Hines EP, Haddad S, Longnecker MP, Poulsen MT, Farrer DG, Marchitti SA, Tan YM, Swartout JC, Sagiv SK, Welsh C, Campbell JL Jr, Foster WG, Yang RS, Fenton SE, Tornero-Velez R, Francis BM, Barnett JB, El-Masri HA, Simmons JE. 2014. Improving the risk assessment of lipophilic persistent environmental chemicals in breast milk. *Critical Reviews in Toxicology* 44(7):600-17.

LaKind JS, Goodman M, Mattison DR. 2014. Bisphenol A and indicators of obesity, glucose metabolism/type 2 diabetes and cardiovascular disease: A systematic review of epidemiologic research. *Critical Reviews in Toxicology* 44(2):121–150.

Goodman M, LaKind JS, Mattison DR. 2014. Do phthalates act as obesogens in humans? A systematic review of the epidemiology literature. *Critical Reviews in Toxicology* 44(2):151–175.

Marchitti SA, Hines EP, LaKind JS, Berlin CM Jr., Fenton SE, Kenneke JF. 2013. Environmental Chemicals in Breast Milk. Reference Module in Earth Systems and Environmental Sciences. Elsevier. <http://dx.doi.org/10.1016/B978-0-12-409548-9.02139-4>

Goodman M, LaKind JS, Fagliano JA, Lash TL, Wiemels JL, Winn DM, Patel C, VanEenwyk J, Kohler BA, Schisterman EF, Albert P, Mattison DR. 2014. Cancer cluster investigations: Review of the past and proposals for the future. *International Journal of Environmental Research and Public Health* 11:1479-1499; doi:10.3390/ijerph110201479.

Marchitti SA, LaKind JS, Naiman DQ, Berlin CM, Kenneke JF. 2013. Improving infant exposure and health risk estimates: Using serum data to predict polybrominated diphenyl ether concentrations in breast milk. *Environmental Science & Technology* 47:4787–4795.

LaKind JS, Goodman M, Naiman DQ. 2012. Use of NHANES data to link chemical exposures to chronic diseases: a cautionary tale. *PLoS ONE* 7(12):e51086. doi:10.1371/journal.pone.0051086

LaKind J. 2013. Can coatings for foods and beverages: Issues and options. *International Journal of Technology, Policy and Management*. 13(1):80-95.

Dórea JG, Fenton SE, LaKind JS, Berlin CM Jr. 2012. Researching chemicals in human milk can be conducted without discouraging breastfeeding. *Bosnian Journal of Basic Medical Sciences* 12(2):137-138.

Goodman M, Naiman JS, Goodman D, LaKind JS. 2013. Response to Condon et al. comments on “Cancer clusters in the USA: What do the last twenty years of state and federal investigations tell us?” *Critical Reviews in Toxicology* 43(1)75-76.

Goodman M, Naiman JS, Goodman D, LaKind JS. Cancer clusters in the US – What do the last twenty years of state and federal investigations tell us? 2012. *Critical Reviews in Toxicology* 42(6):474-490. *Number 50 of “Most read articles in Critical Reviews in Toxicology”

LaKind JS, Levesque J, Dumas P, Bryan S, Clarke J, Naiman DQ. 2012. Comparing United States and Canadian population exposures from national biomonitoring surveys: Bisphenol A intake as a case study. *Journal of Exposure Science and Environmental Epidemiology* 22:219-226.

Youngstrom E, Kenworthy L, Lipkin PH, Goodman M, Squibb K, Mattison DR, Anthony LG, Makris SL, Bale A, Raffaele KC, LaKind JS. 2011. A proposal to facilitate weight-of-evidence assessments: Harmonization of neurodevelopmental environmental epidemiology studies (HONEES). *Neurotoxicology and Teratology*. 33:354–359.

LaKind JS, Berlin CM Jr., Fenton SE. 2011. Environmental Chemicals in Breast Milk. In: Nriagu, J.O. (ed.) *Encyclopedia of Environmental Health*, volume 2, pp. 347–356 Burlington: Elsevier.

Blount BC, Backer LC, Aylward LL, Hays SM, LaKind JS. 2011. Human Exposure Assessment for DBPs: Factors Influencing Blood Trihalomethane Levels. In: Nriagu, J.O. (ed.) *Encyclopedia of Environmental Health*, volume 3, pp. 100–107 Burlington: Elsevier.

LaKind JS, Naiman DQ. 2011. Daily intake of bisphenol A (BPA) and potential sources of exposure – 2005-2006 NHANES. *Journal of Exposure Science and Environmental Epidemiology* 21:272-279.

Blount BC, McElprang DO, Chambers DM, Waterhouse MG, Squibb KS, LaKind JS. 2010. Methodology for collecting, storing, and analyzing human milk for volatile organic compounds. *Journal of Environmental Monitoring* 12:1265-1273.

Goodman M, Youngstrom E, Gutermuth Anthony L, Kenworthy L, Lipkin PH, Squibb K, Mattison DR, LaKind JS. 2010. Using systematic reviews and meta-analyses to support regulatory decision-making for neurotoxicants: Lessons learned from a case study of PCBs. *Environmental Health Perspectives* 118:727-734. doi:10.1289/ehp.0901835.

LaKind JS, Richardson SD, Blount BC. The good, the bad, and the volatile – Can we have both healthy pools and healthy people? 2010. *Environmental Science & Technology* 44:3205–3210.

Youngstrom E, LaKind JS, Kenworthy L, Lipkin PH, Goodman M, Squibb K, Mattison DR, Anthony BJ, Gutermuth Anthony L. 2010. Advancing the selection of neurodevelopmental measures in epidemiological studies of environmental chemical exposure and health effects. *International Journal of Environmental Research and Public Health* 7:229-268.

LaKind JS, Birnbaum LS. 2010. Out of the frying pan AND out of the fire: the indispensable role of exposure science in assessing replacement chemicals. *Journal of Exposure Science and Environmental Epidemiology* 20:115–116.

LaKind JS, Naiman DQ, Hays SM, Aylward LL, Blount BC. 2010. Public health interpretation of trihalomethane blood levels in the U.S.: NHANES 1999-2004. *Journal of Exposure Science and Environmental Epidemiology* 20(3):255-262. Featured article.

LaKind JS, Berlin CM Jr., Sjödin A, Turner W, Wang RY, Needham LL, Paul IM, Stokes JL, Naiman DQ, Patterson DG Jr. 2009. Do human milk concentrations of persistent organic chemicals really decline during lactation? Chemical concentrations during lactation and milk/serum partitioning. *Environmental Health Perspectives* 117(10):1625–1631.

LaKind JS, Berlin CM Jr., Mattison DR. 2009. Response to Geraghty et al. Letter to the Editor. *Breastfeeding Medicine* 4(2):127.

LaKind JS, Fenton SE, Dórea JG. 2009. Human milk biomonitoring of phthalates: Expanding our understanding of infant exposure is compatible with supporting breastfeeding. Letter to the editor. *Environment International* 35:994-995.

LaKind JS, Hays SM, Aylward LL, Naiman DQ. 2009. Perspective on serum dioxin levels in the United States: An evaluation of the NHANES data. *Journal of Exposure Science and Environmental Epidemiology* 19:435-441.

Weisel CP, Richardson SD, Nemery B, Aggazzotti G, Baraldi E, Blatchley ER III, Blount BC, Carlsen K-H, Eggleston PA, Frimmel FH, Goodman M, Gordon G, Grinshpun S, Heederik DJJ, Kogevinas M, LaKind JS, Nieuwenhuijsen MJ, Piper FC, Sattar SA. 2009. Conclusions and key research recommendations from the Workshop on Advancing the Science: Childhood Asthma and Environmental Exposures at Swimming Pools. *Environmental Health Perspectives* 117:500–507. doi:10.1289/ehp.11513. Science Selections: Widening the Pool of Factors Studies Needed to Assess Asthma–Swimming Link p. A162.

LaKind JS, Berlin CM Jr., Mattison DR. 2008. The heart of the matter on breast milk and environmental chemicals: Essential points for health care providers and new parents. *Breastfeeding Medicine* 4(3):251-259. New York Times summary: Despite Worries Over Toxins, Breast-Feeding Still Best for Infants. <http://www.nytimes.com/2008/12/20/health/research/20breast.html?scp=6&sq=lakind&st=cse>

LaKind JS, Naiman DQ. 2008. Bisphenol A (BPA) Daily intakes in the United States: Estimates from the 2003-2004 NHANES urinary BPA data. *Journal of Exposure Science and Environmental Epidemiology* 18:608–615. Featured Article.

Aylward LL, LaKind JS, Hays SM. 2008. Derivation of biomonitoring equivalent (BE) values for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds: a screening tool for interpretation of biomonitoring data in a risk assessment context. *Journal of Toxicology and Environmental Health A* 71(22):1499-1508.

Aylward LL, LaKind JS, Hays SM. 2008. Biomonitoring Equivalents (BEs) for interpretation of whole blood biomonitoring data for trihalomethanes. *Regulatory Toxicology and Pharmacology* 51(3, Suppl 1):S68-S77.

Hays SM, Aylward LL, LaKind JS, Bartels MJ, Barton HA, Boogaard PJ, Brunk C, DiZio S, Dourson M, Goldstein DA, Lipscomb J, Kilpatrick ME, Krewski D, Krishnan K, Nordberg M, Okino M, Tan Y-M, Viau C, Yager JW. 2008. Guidelines for the Derivation of Biomonitoring Equivalents: Report from the Biomonitoring Equivalents Expert Workshop. *Regulatory Toxicology and Pharmacology* 51(3, Suppl 1):S4-S15.

LaKind JS, Aylward LL, Brunk C, DiZio S, Dourson M, Goldstein DA, Kilpatrick ME, Krewski D, Bartels M, Barton HA, Boogaard PJ, Lipscomb J, Krishnan K, Nordberg M, Okino M, Tan Y-M, Viau C, Yager JW, Hays SM. 2008. Guidelines for the Communication of Biomonitoring Equivalents: Report from the Biomonitoring Equivalents Expert Workshop. *Regulatory Toxicology and Pharmacology* 51(3, Suppl 1):S16-S26.

Hays SM, Aylward LL, LaKind JS. 2008. Introduction to the Biomonitoring Equivalents Pilot Project: Development of guidelines for the derivation and communication of Biomonitoring Equivalents. *Regulatory Toxicology and Pharmacology* 51(3, Suppl 1):S1-S2.

LaKind JS, Berlin CM Jr., Stokes JL, Naiman DQ, Paul IM, Patterson DG Jr., Jones RS, Niehüser S., Park A, Wang RY, Needham LL, Lorber MN, Sjödin A. 2008. Lifestyle and polybrominated diphenyl ethers (PBDEs) in human milk in the United States: A pilot study. *Toxicological & Environmental Chemistry* 70(9):61-63.

LaKind JS, Barraj L, Tran N, Aylward LL. 2008. Guest Commentary: Environmental chemicals in people: Challenges in interpreting biomonitoring information. *Journal of Environmental Health* 70(9):61-64.

LaKind JS, Holgate ST, Ownby DR, Mansur AH, Helms PJ, Pyatt D, Hays SM. 2008. Authors' response to 'Critical review of Clara cell protein: sound science?' *Biomarkers* 13(3):244-245.

LaKind JS, Holgate ST, Ownby DR, Mansur AH, Helms PJ, Pyatt D, Hays SM. 2007. A critical review of the use of Clara Cell secretory protein CC16 as a biomarker of exposure and effect. *Biomarkers* http://www.informaworld.com/smpp/title~content=t713693137~db=all~tab=issueslist~branches=12_v1212:445-467.

LaKind JS, Wilkins AA, Bates MN. 2007. Breast biomonitoring and environmental chemicals: Use of breast tissues and fluids in breast cancer etiologic research. *Journal of Exposure Science and Environmental Epidemiology* 17:525-540.

LaKind JS. 2007. Recent global trends and physiologic origins of dioxins and furans in human milk. *Journal of Exposure Science and Environmental Epidemiology* 17:510–524. Featured Article.

Needham LL, Naiman DQ, Patterson DG, Jr., LaKind JS. 2007. Assigning concentration values for dioxin and furan congeners in human serum when measurements are below limits of detection: an observational approach. *Chemosphere* 67:439–447.

Amler RW, Barone S Jr., Belger A, Berlin CM Jr., Cox C, Frank H, Goodman M, Harry J, Hooper SR, Ladda R, LaKind JS, Lipkin PH, Lipsitt LP, Lorber MN, Myers G, Mason AM, Needham LL, Sonawane B, Wachs TD, Yager JW. 2006. Hershey Medical Center Technical Workshop Report: Optimizing the design and interpretation of epidemiologic studies for assessing neurodevelopmental effects from *in utero* chemical exposure. *NeuroToxicology* 27(5):861-874.

Baier-Anderson C, Blount B, LaKind JS, Naiman DQ, Wilbur SB, Tan S. 2006. Estimates of exposure to perchlorate from consumption of human milk, dairy milk, and water and comparison to current reference dose. *Journal of Toxicology and Environmental Health* 69(4):319-330.

Sjödin A, LaKind JS, Patterson D, Needham LL, Wang R, Berlin CM Jr., Paul IM, Stokes J. 2005. Current concentrations and changes in concentrations of PBDEs, persistent pesticides, and PCBs in human milk. *Organohalogen Compounds* 67:1745-1748.

Bates MN, Hamilton JW, LaKind JS, Langenberg P, O'Malley M, Snodgrass W. 2005. Biomonitoring Workshop Report: Biomonitoring study design, interpretation, and communication - Lessons learned and path forward. *Environmental Health Perspectives* 113:1615–1621.

LaKind JS, Brent RL, Dourson ML, Kacew S, Koren G, Sonawane B, Tarzian AJ, Uhl K. 2005. Human milk biomonitoring data: interpretation and risk assessment issues. *Journal of Toxicology and Environmental Health* 68(20):1713-1770.

Fenton SE, Condon M, Ettinger A, Mason A, McDiarmid M, Qian Z, Selevan SG, LaKind JS. 2005. Collection and use of exposure data from human milk biomonitoring in the United States. *Journal of Toxicology and Environmental Health* 68(20):1691-1712.

Berlin CM Jr., LaKind JS, Fenton SE, Wang RY, Bates MN, Brent RL, Condon M, Crase BL, Dourson ML, Ettinger AS, Foos B, Fürst P, Giacoia GP, Goldstein DA, Haynes SG, Hench KD, Kacew S, Koren G, Lawrence RA, Mason A, McDiarmid MA, Moy G, Needham LL, Paul IM, Pugh LC, Qian Z, Salamone L, Selevan SG, Sonawane B, Tarzian AJ, Tully MR, Uhl K. 2005. Conclusions and recommendations of the Expert Panel: Technical Workshop on Human Milk Surveillance and Biomonitoring for Environmental Chemicals in the United States. *Journal of Toxicology and Environmental Health* 68(20):1825-1831.

Berlin CM Jr, Crase BL, Fürst P, Moy G, Needham LL, Pugh L, Tully MR, LaKind JS. 2005. Methodologic considerations for improving and facilitating human milk research. *Journal of Toxicology and Environmental Health* 68(20):1803-1824.

LaKind JS, Berlin CM Jr., Bates MN. 2005. Overview: Technical Workshop on Human Milk Surveillance and Biomonitoring for Environmental Chemicals in The United States. *Journal of Toxicology and Environmental Health* 68(20):1683-1690.

- LaKind JS, Wilkins AA, Berlin CM. 2004. Environmental chemicals in human milk: a review of concentrations, determinants, infant exposures and health, and guidance for future research. *Toxicology and Applied Pharmacology* 198:184-208.
- Berlin CM, LaKind JS, Selevan SG. 2003. Human Milk Monitoring for Environmental Chemicals: Guidance for Future Research. *ABM News and Views*. 9(17):23-24.
- LaKind JS, Bates MN, Wilkins AA. 2003. How useful is measurement of environmental chemicals in human milk in investigations of breast cancer etiology? *Organohalogen Compounds* 65:346-349. Presented at Dioxin2003, Boston, MA. August.
- LaKind JS. 2003. Interpreting and Communicating Health and Risk Information on Chemicals in Breast Milk: DDT as a Case Study. In: *Reviews in Food and Nutrition*, Volume 1. V.R. Preedy and R.R. Watson, eds. Taylor & Francis. Pp. 230-242.
- Borgert CJ, LaKind JS, Witorsch RJ. 2003. A critical review of potential methods for comparing hormonal activity of endogenous and exogenous chemicals in human milk and infant formula. *Environmental Health Perspectives* 111(8):1020-1036.
- Aylward LL, Hays SM, LaKind JS, Ryan JJ. 2003. Partitioning of persistent lipophilic compounds including dioxins between human milk lipid and blood lipid: An initial assessment. *Journal of Toxicology and Environmental Health* 66(1):1-5.
- Berlin CM, Kacew M, Lawrence R, LaKind JS, Campbell R. 2002. Criteria for chemical selection for programs on human milk surveillance and research for environmental chemicals. *Journal of Toxicology and Environmental Health* 65(22):1839-1852.
- LaKind JS, Berlin CM. 2002. Technical Workshop on Human Milk Surveillance and Research on Environmental Chemicals in the United States: An Overview. *Journal of Toxicology and Environmental Health* 65(22):1829-1838.
- LaKind JS, Birnbach N, Borgert CJ, Sonawane BR, Tully MR, Friedman L. 2002. Human milk surveillance and research of environmental chemicals: Concepts for consideration in interpreting and presenting study results. *Journal of Toxicology and Environmental Health* 65(22):1909-1928.
- Berlin CM, LaKind JS, Sonawane BR, Kacew S, Borgert CJ, Bates MN, Birnbach N, Campbell R, Dermer A, Dewey KG, Ellerbee SM, Fürst P, Giacoia GP, Gartner L, Groer M, Haynes SG, Humenick SS, Lawrence RA, Lorber M, Lovelady C, Mason A, Needham LL, Picciano MF, Plautz J, Ryan JJ, Selevan SG, Sumaya CV, Tully MR, Uhl K, Vesell E, Wilson JT. 2002. Conclusions, research needs and recommendations of the expert panel: technical workshop on human milk surveillance and research for environmental chemicals in the United States. *Journal of Toxicology and Environmental Health* 65(22):1929-1935.
- LaKind JS, Berlin C, Naiman DQ. 2001. Infant exposure to chemicals in breast milk in the United States: What we need to learn from a breast milk monitoring program. *Environmental Health Perspectives* 109:75-88.
- LaKind JS, Berlin CM. 2000. PDBEs in breast milk: Where do we go from here? Presented at Dioxin2000, Monterey, California, August 13-17, 2000. *Organohalogen Compounds* 47:241-244.

- Pandak CA, Mason AM, LaKind JS. 2000. Tools for improving community health: Community health assessment and asset evaluation. *Environmental Regulation and Permitting*. 9(3):15-23.
- LaKind JS, Berlin CM, Naiman DQ, Park CN. 2000. Characterization of dose distributions of selected breast milk contaminants to nursing infants: DDE and TCDD. *Organohalogen Compounds* 48:223-226.
- LaKind JS, Berlin C, Park C, Naiman DQ, Gudka NJ. 2000. Methodology for characterizing distributions of incremental body burdens of 2,3,7,8-TCDD and DDE from breast milk in North American nursing infants. *Journal of Toxicology and Environmental Health, Part A* 59:605-639.
- Peddicord RK, LaKind JS. 2000. Ecological and human health risks at an outdoor firing range. *Environmental Toxicology & Chemistry* 19(10):2602-2613.
- LaKind JS, Filser JG. 1999. Dietary exposure to PCBs and dioxins. *Environmental Health Perspectives* 107(10):A9.
- LaKind JS, McKenna EA, Hubner RP, Tardiff RG. 1999. A review of the comparative mammalian toxicity of ethylene glycol and propylene glycol. *Critical Reviews in Toxicology* 29(4):331-365.
- LaKind JS, Ginevan ME, Naiman DQ, James AC, Jenkins RA, Dourson ML, Felter SP, Graves CG, Tardiff RG. 1999. Distribution of exposure concentrations and doses for constituents of environmental tobacco smoke. *Risk Analysis: An International Journal* 19(3):375-390.
- LaKind JS, Jenkins RA, Naiman DQ, Ginevan ME, Graves CG, Tardiff RG. 1999. Use of environmental tobacco smoke (ETS) constituents as markers for ETS exposure. *Risk Analysis: An International Journal* 19(3):359-376.
- LaKind JS, Graves CG, Ginevan ME, Jenkins RA, Naiman DQ, Tardiff RG. 1999. Exposure to environmental tobacco smoke in the workplace and the impact of away-from-work exposure. *Risk Analysis: An International Journal* 19(3):349-358.
- LaKind JS. 1998. Comparison of three models for predicting blood lead levels in children: Episodic exposures to lead. *Journal of Exposure Analysis and Environmental Epidemiology* 8(3):399-406.
- LaKind JS. 1998. Forward to Special Issue on Environmental Risk Assessment: Issues and Methods. *International Journal of Environment and Pollution* 9(1):1-2.
- Bartell SM, LaKind JS, Moore JA, Anderson P. 1998. Bioaccumulation of hydrophobic organic chemicals by aquatic organisms: A workshop summary. *International Journal of Environment and Pollution* 9(1):3-25.
- LaKind JS. 1994. Sediment dioxin levels as the basis for risk assessment and human health criteria. *International Journal of Environment and Pollution* 3(4):226-232.
- LaKind JS, Naiman DQ. 1993. Comparison of predicted and observed dioxin levels in fish: Implications for risk assessment. *Risk: Issues in Health & Safety* 4(3):253-262.
- Rifkin E, LaKind JS. 1991. Dioxin bioaccumulation: Key to a sound risk assessment methodology. *Journal of Toxicology and Environmental Health* 33:103-112.
- LaKind J. 1991. Bioconcentration – letter to the editor. *Environmental Science & Technology* 25(1):6.

LaKind JS, Rifkin E. 1990. Current method for setting dioxin limits in water requires reexamination. *Environmental Science & Technology*. 24:963-965.

LaKind JS, Stone AT. 1989. Reductive Dissolution of Goethite by Phenolic Reductants. *Geochimica Cosmochimica Acta*. 53:961-971

LaKind JS, Stone AT. 1988. Reductive Dissolution of Goethite by Substituted Phenols. *EOS* 69(16):369.

LaKind JS, Stone AT. 1986. Reductive Dissolution of Goethite and Hematite by Substituted Phenols. *EOS* 67(44):948.

Gieskes JM, Elderfield H, Lawrence JR, LaKind J. 1984. Interstitial Water Studies, Leg 78a, *Initial Reports of the Deep Sea Drilling Project*. LXXVIII:377-384.

Gieskes JM, Sirocky FX, LaKind JS. 1984. Interstitial Water Studies, Leg 73, *Initial Reports of the Deep Sea Drilling Project*. LXXIII:539-541.

Gieskes JM, Sirocky FX, LaKind JS. 1983. Interstitial Water Studies, Leg 72, *Initial Reports of the Deep Sea Drilling Project*. LXXII:391-394.

Selected Presentations:

LaKind JS. (with A.M. Rule and F. Wagner). 2024. Creating and Sustaining Successful Public-Private Partnerships (PPPs) for Environmental Monitoring Programs: Principles and Elements. Webinar. 25 July.

Keynote speaker. 2023. Epidemiology and risk assessment: Reflections on working together to improve public health. International Conference on Using Epidemiological Studies in Health Risk Assessments: Relevance, Reliability and Causality. Berlin, Germany. 9 November.

Invited lecturer. 2023. Everything you wanted to know about consulting* - *but were afraid to ask. Lecture, Applied Mathematics and Statistics, The Johns Hopkins University. 15 February. 14 September.

Invited speaker. 2022. “Forever Chemicals” (PFAS) in Breast Milk and Infant Formula: A Global Issue. International Clean-up Conference. Adelaide, Australia. 12 September.

Invited speaker. 2022. PFAS and breast milk: What we don’t know, what we should know. 3rd National PFAS Meeting: Highly Fluorinated Compounds – Environmental Justice and Scientific Discovery. Wilmington, NC. 16 June.

Invited speaker. 2022. PFAS in breast milk in the US and Canada: Mom/infant exposure data gaps. Health Canada Environmental Health Science and Research Bureau. 25 May.

Invited speaker. 2022. Chemical exposures and health effects: Exposure assessment and interpreting epidemiology research. Center for Food Safety and Applied Nutrition (CFSAN). Division of Risk and Decision Analysis. U.S. Food and Drug Administration. 25 March.

Invited speaker. 2022. Epidemiology and exposure assessment: What toxicologists need to know (or remember). The Toxicology Forum—2022 Virtual Winter Meeting. 25 January.

LaKind JS. 2021. Current breast milk PFAS levels in the US and Canada: After all this time why don't we know more? International Society for Exposure Science Annual Meeting (virtual). 1 September.

LaKind JS. 2020. The Matrix: Bridging the gap between epidemiology and risk assessment. International Society for Exposure Science Annual Meeting. Webinar. 22 September.

LaKind JS, Burns CJ. 2020. The Matrix: Bridging the gap between epidemiology and risk assessment. Series of invited webinars (e.g., US EPA OPPP/OPPT, 9 September; Environmental and Occupational Health Sciences (EOHS) Research Seminar Series at The University of Texas Health Science Center at Houston, School of Public Health, 11 September; Johns Hopkins Bloomberg School of Public Health Current Topics in Epidemiology seminar series, 30 September; Department of Environmental and Occupational Health, Dornsife School of Public Health, Drexel University, 9 November).

LaKind JS. 2020. Environmental Chemicals in Breast Milk and Formula: Exposure and Risk Assessment Implications. The Society for Birth Defects Research & Prevention Virtual 60th Annual Meeting. 30 June.

LaKind JS, Burns CJ. 2020. Epidemiology, exposure and risk assessment. Texas Commission on Environmental Quality. Webinar. 18 June.

LaKind JS. 2019. Exposure Data Quality Assessments: Why and How? Society for Risk Analysis Annual Conference. Arlington, VA. 11 December.

LaKind JS, Burns CJ. 2019. The Matrix: Bridging the gap between epidemiology and risk assessment. Health Canada. Ottawa, Canada. 4 November.

Invited speaker. 2019. Biomonitoring and epidemiology research on personal care products: We're not in Kansas anymore. Personal Care Products Council Annual Safety Seminar. Philadelphia, PA. 30 October.

Invited lecture (with Dr. Heidi S. Erickson and Dr. Carol Burns). 2019. The University of Texas Medical Branch at Galveston/ Chronic Disease Epidemiology Course. 23 April.

Invited lecture. 2019. Conflicts of Interest and Environmental Research. Bioethics, Honors College of Florida Atlantic University. Jupiter, FL. 20 March.

LaKind JS, Burns CJ. 2019. Evidence-based environmental decisions: Bridging the gap between epidemiology and risk assessment. SOT RASS/ISES Webinar. 13 February.

LaKind JS. 2018. Exposure data quality assessments: ExpoQual. International Society of Exposure Science/International Society of Environmental Epidemiology. Ottawa, Canada. 28 August.

Invited speaker. 2018. How to assess and interpret biomonitoring data once you have it.

Workshop on the Feasibility of Addressing Environmental Exposure Questions Using Department of Defense Biorepositories. The National Academies of Sciences, Engineering and Medicine. Washington, DC. 15 June.

Invited speaker. 2018. Chemical exposures and human health: What can we take away from epidemiology research? Occupational Medicine, Clinical Public Health & Epidemiology Army Public Health Center. Aberdeen Proving Ground, MD. 6 June.

Invited speaker. 2018. Evidence-based environmental decision-making: Problems and progress. Bundesinstitut für Risikobewertung. Berlin, Germany. 24 May.

Invited speaker. 2018. Exposure data quality and environmental epidemiology: Implications for systematic reviews and weight of evidence. Environmental Health Science and Research Bureau (EHSRB) Seminar Series. Health Canada. 21 February. Ottawa, Canada.

Invited speaker. 2018. Exposure data quality in environmental epidemiology: Bad habits and remedies. Université de Montréal Public Health Research Institute. 20 February. Montreal, Canada.

Invited speaker. 2017. Exposure data in environmental epidemiology: limitations and quality assessments. European Food Safety Authority Scientific Conference on the Use of Epidemiological findings in Regulatory Pesticide Risk Assessment. 21 November. Parma Italy.

LaKind JS. 2017. Critical and systematic evaluation of 2,4-dichlorophenoxyacetic acid (2,4-D) exposure data: quality and generalizability for human assessments. International Society of Exposure Science Annual Meeting. 18 September. Durham NC.

LaKind JS. 2017. Transparent and systematic reviews of exposure data in environmental epidemiology: Approaches and case studies. International Society of Exposure Science Annual Meeting. 17 September. Durham NC.

LaKind JS. 2017. Evaluating strengths and limitation of the exposure data using the Biomonitoring, Environmental Epidemiology, and Short-Lived Chemicals (BEES-C) Instrument: Implications for science and policy. American College of Epidemiology Annual Conference. 25 September. New Orleans, LA.

Invited speaker. 2017. Chemical exposures and health effects: What we know and what we don't know from epidemiology research. Mid-Atlantic Regional Conference in Occupational and Environmental Medicine. 23 September. Baltimore, MD.

Invited speaker. 2017. Chemical exposures and health effects: What we know and what we don't know from epidemiology research. Occupational and Environmental Residency Program, Johns Hopkins Bloomberg School of Public Health. 18 September. Baltimore, MD.

LaKind JS. 2017. Human exposure to 2,4-D: What do the data tell us? American Chemical Society 254th Annual Meeting. 21 August. Washington DC.

Invited speaker. 2016. Quality matters in environmental epidemiology: The exposure data we collect versus the data we need. Grand Rounds, University of Maryland School of Medicine. 17 November. Baltimore, MD.

Invited speaker. 2016. Can coating complexities. Workshop - Identifying and Evaluating Alternative Materials: The Case of BPA-Free Can Linings. 4 November. UC Berkeley. Berkeley, CA. <https://www.youtube.com/watch?v=UqNXi1qNXHQ>

Invited speaker. 2016. Biomonitoring and environmental epidemiology: Implications for personal care products. Personal Care Products Council Safety Workshop. 26 October. Alexandria, VA.

LaKind JS. 2016. Assessing Biomonitoring Data Quality: The Biomonitoring, Environmental Epidemiology, and Short-Lived Chemicals (BEES-C) Instrument. International Society of Exposure Science Annual Meeting. 12 October. Utrecht, The Netherlands.

LaKind JS. 2016. Harmonization, transparency, and access: Why we need these in environmental epidemiology [exposure science]. International Society of Exposure Science Annual Meeting. 10 October. Utrecht, The Netherlands.

Invited speaker. 2016. Cleaning, environmental exposures and respiratory health effects: Issues, challenges and opportunities. 17 June. Advancing the Science Webinar Series. Sponsored by the American Cleaning Institute (ACI), in collaboration with the Toxicology Excellence for Risk Assessment (TERA) Center, University of Cincinnati and Endorsed by the Society of Toxicology.

Invited speaker. 2016. Environmental Epidemiology: The importance of exposure assessment. CropLife America and RISE Spring Conference. 14 April. Arlington, Virginia.

LaKind JS. 2016. Quality Matters in Environmental Epidemiology: The data we collect versus the data we need. 14 March. Society of Toxicology. New Orleans, LA.

Invited speaker. 2016. Biomonitoring and temporality in environmental epidemiology: The data we collect versus the data we need. U.S. Environmental Protection Agency. Temporal Exposure Issues for Environmental Pollutants: Health Effects and Methodologies for Estimating Risk. 27–29 January. Research Triangle Park, NC

LaKind JS. 2015. Biomonitoring Data in Cumulative Risk Assessment: The Biomonitoring, Environmental Epidemiology, and Short-Lived Chemicals (BEES-C) Instrument. Society for Risk Analysis. 9 December. Arlington, Virginia.

LaKind JS, Naiman DQ. 2015. Temporal trends in BPA exposure in the US from 2003–2012 and factors associated with BPA exposure: Spot samples and urine dilution complicate data interpretation. International Society for Exposure Science. 19 October. Henderson, Nevada.

Invited speaker/panelist. 2015. Exposure Science and Environmental Epidemiology: Problems and Proposed Solutions. ICCA-LRI & US EPA Workshop. What Will Work? Application of New Approaches for Chemical Safety Assessment. June 16-17. New Orleans, Louisiana.

Invited poster presentation. 2015. Issues with quality and harmony in environmental epidemiology: PCBs, BPA and phthalates. ICCA-LRI & US EPA Workshop. What Will Work? Application of New Approaches for Chemical Safety Assessment. June 16-17. New Orleans, Louisiana.

Invited speaker. 2015. Institute of Medicine Workshop on the Interplay between Environmental Exposures and Obesity. March 2-3. Research Triangle Park, NC.

Invited speaker. 2014. The need for more robust data in environmental epidemiology: BPA as a case study. Toxicology Forum. July 9. Aspen, Colorado.

Invited panelist. 2014. What Is Safe? Integrating Multi-Disciplinary Approaches for Decision Making about the Human Health and Environmental Impacts of Chemicals. ICCA-LRI & JRC Workshop. June 17-18, Lugano, Switzerland.

Speaker. 2014. PCBs and related chemicals in breast milk: What do the data mean for mothers, infants, doctors, regulators and others? Society of Toxicology Annual Meeting. 26 March. Phoenix, Arizona.

Invited speaker. 2013. Endocrine disruptors and obesity, diabetes and heart disease: What does epidemiological research tell us? 15th Cefic-LRI Annual Workshop. 21 November. Brussels, Belgium.

Invited speaker. 2013. Uncertainties in Epidemiology: The Example of Bisphenol A. 2013 Center for Advancing Risk Assessment Science And Policy Workshop. 6 November. Washington DC.

Invited speaker. 2013. Urine and Pool Water: Exposure and Health. World Aquatic Health Conference. 18 October. Indianapolis, Indiana.

Invited speaker. 2013. Cancer Clusters in the USA: What Do the Last 20 Years of State and Federal Investigations Tell Us? DHMH Workgroup on Cancer Clusters and Environmental Causes of Cancer. September 10, Baltimore, Maryland.

Invited speaker/panelist. 2013. What is Normal? Biomarkers of Exposure & Effect. ICCA-LRI & NCATS Workshop: What Is Normal? Implications for Chemical Safety Assessment. June 11-12, Santa Fe, New Mexico.

Guest lecturer. 2013. Human Health Risk Assessment Primer. University of Maryland, College Park. 30 April.

Invited speaker. 2012. 21st Century Solutions for 20th Century Problems: Lessons from 4 decades of environmental epidemiology research. CropLife America & RISE. Spring Conference. Arlington, Virginia. 5 April.

Invited speaker. 2011. Endocrine disruption and risk assessment: The controversial case of bisphenol A. Grand Rounds. Division of Endocrinology, Diabetes and Nutrition, University of Maryland School of Medicine. 31 October.

LaKind JS, Levesque J, Dumas P, Bryan S, Clarke J, Naiman DQ. 2011. Can We Compare United States and Canadian Population Exposures from National Biomonitoring Surveys? Bisphenol A (BPA) as a Case Study. International Society for Exposure Science. Baltimore, Maryland. 27 October.

Invited speaker. 2011. Swimming and asthma: What does the current research say? ACI Asthma Science Forum. Arlington, VA. 10 May.

Invited speaker. 2010. Are the kids alright? Strengthening regulatory decision-making in the uncertain world of children's health research. 12th Cefic LRI Annual Workshop. Brussels, Belgium. 18 November.

Guest Lecturer. 2010. Human Health Risk Assessment Primer. University of Maryland, College Park. 8 November.

Speaker. 2010. The Good, the Bad, and the Volatile: Can We Have Both Healthy Pools and Healthy People? World Aquatic Health Conference. Colorado Springs, CO. 8 October.

Invited speaker. 2010. A Multidisciplinary Approach to Advancing the Science of Neurodevelopmental Testing in Cohorts of Infants and Young Children. Teratology Society's 50th Annual Meeting. Louisville, Kentucky. Joint TS/Neurobehavioral Teratology Society Symposium on Advancing Neurodevelopmental Evaluation in Children. June 29. Citation: LaKind JS, Youngstrom E, Goodman M, Squibb K, Lipkin PH, Anthony LG, Kenworthy L, Mattison D. 2010. A multidisciplinary approach to advancing the science of neurodevelopmental testing in cohorts of infants and young children. *NBTS 34 Neurotoxicology and Teratology* 32:505.

Kenworthy L, Anthony LG, Goodman M, LaKind JS, Lipkin PH, Mattison D, Squibb K, Youngstrom E. 2010. Getting the biggest bang for your buck: Choosing neurodevelopmental tests that maximize power. *NBTS35 Neurotoxicology and Teratology* 32:506.

Anthony LG, Youngstrom E, Kenworthy L, LaKind JS, Goodman M, Squibb K, Lipkin PH, Mattison D. 2010. Threats to study validity: The Flynn Effect, examiner drift, confounders, lost in translation, and other important considerations. *NBTS36 Neurotoxicology and Teratology* 32:506.

Invited speaker. 2010. Environmental fate of chemicals: Bring babies into the food web. University of Maryland Baltimore County. 10 March.

Invited participant/speaker. 2009. Human milk biomonitoring: data interpretation and risk assessment issues. International Atomic Energy Agency. Vienna, Austria. 16 February.

Invited speaker. 2008. Grand Rounds. Environmental chemicals and breastfeeding infants. The Johns Hopkins School of Medicine. February 6. Baltimore, Maryland.

LaKind JS, Squibb KS, McElprang DO, Blount BK. Methodologic pilot study of volatile organic compounds (VOCs) in human milk. 2007. 17th Annual Conference of the International Society for Exposure Analysis. October. Durham, North Carolina.

LaKind JS, Aylward LL, Brunk C, DiZio S, Dourson M, Goldstein DA, Kilpatrick ME, Krewski D, Bartels M, Barton HA, Boogaard PJ, Lipscomb J, Krishnan K, Nordberg M, Okino M, Tan Y-M, Viau C, Yager JW, Hays SM. 2007. Guidelines for the Communication of Biomonitoring Equivalents: Report from the Biomonitoring Equivalents Expert Workshop. 17th Annual Conference of the International Society for Exposure Analysis. October. Durham, North Carolina.

Speaker. 2007. Workshop on Childhood Asthma and Environmental Exposures at Indoor Swimming Pools. Advancing the Science. Fourth Annual World Aquatic Health™ Conference. 3 October. Cincinnati, Ohio.

LaKind JS, Berlin CM Jr., Stokes JL, Naiman DQ, Paul IM, Patterson DG Jr., Jones RS, Niehüser S, Park A, Wang RY, Needham LL, Lorber MN, Sjödin A. 2007. Lifestyle and polybrominated diphenyl ethers (PBDEs) in human milk in the United States: A pilot study. 17th Annual Conference of the International Society for Exposure Analysis. October. Durham, NC.

Invited speaker. 2007. Environmental chemicals and breastfeeding infants (update). La Leche League

International's 50th Anniversary Conference. July 23. Chicago.

Invited speaker. 2006. Women's & Children's Health and the Environment. Talking about Environmental Chemicals in Human Milk: Why "Breast is Best." April 24. Baltimore, Maryland.

Invited speaker. 2006. Grand Rounds. What is in mother's milk and what does it mean? Environmental chemicals and breastfeeding infants. Children's Hospital at Sinai. February 14. Baltimore, Maryland.

LaKind JS, Berlin CM Jr. 2005. Workshop on Human Milk Surveillance and Biomonitoring for Environmental Chemicals in the United States. 15th Annual International Society of Exposure Analysis Annual Meeting. November. Tucson, Arizona.

Invited speaker. 2005. Grand Rounds. Interpretation and communication of information from biomonitoring studies. What physicians should know. Maryland General Hospital. October 10. Baltimore, Maryland.

Invited speaker. 2005. Biomonitoring Panel Report: Biomonitoring study design, interpretation, and communication. International Society of Regulatory Toxicology and Pharmacology Workshop: Understanding Human Biomonitoring. June 16. Sacramento, California.

Invited speaker. 2005. What is in mother's milk and what does it mean? Environmental chemicals and breastfeeding infants. Pediatric Academic Societies' Annual Meeting, Perinatal Nutrition and Metabolism Club. May 16. Washington, DC. Invited speaker. 2005. Interpretation and communication of information from biomonitoring studies. Ethics & Sustainability Dialogue Group. May 12. Alexandria, Virginia.

Invited speaker. 2004. Breast Feeding Promotion Task Force. June 7. Baltimore, Maryland.

Invited speaker. 2004. What is in mother's milk and what does it mean? A discourse on environmental chemicals and breastfeeding infants. Institute of Pharmacology and Toxicology, Section of Developmental and Environmental Toxicology, University of Zurich, April 22, Lausanne, Switzerland; World Health Organization, April 26, Geneva, Switzerland.

LaKind JS, Susten A, Mistry K. 2003. Uses and interpretation of human biomonitoring data. Society for Risk Analysis Annual Meeting. December 10. Baltimore, Maryland.

Invited speaker. 2003. Environmental chemicals in human milk. Sixth National Environmental Public Health Conference. December 4. Atlanta, Georgia.

LaKind JS, Bates MN, Wilkins AA. 2003. How useful is measurement of environmental chemicals in human milk in investigations of breast cancer etiology? Dioxin2003. August. Boston, MA.

Invited speaker. 2003. Department of Health and Human Services, Office on Women's Health. Workshop on Breast Cancer and the Environment. June 26. Washington, DC.

Invited speaker. 2003. Chemicals and Risk: What You Should Know, What Patients May Ask. Grand Rounds, Hershey Medical Center, Penn State College of Medicine. April 8. Hershey, Pennsylvania.

LaKind JS, Susten A, Mistry K. 2003. Society for Risk Analysis Annual Meeting. Uses and Interpretation of Human Biomonitoring Data. December 10. Baltimore, Maryland.

Invited speaker. 2003. US Environmental Protection Agency's Children's Health Protection Advisory Committee. Research and surveillance of environmental chemicals in human milk. March 19. Washington, DC.

Invited speaker. 2002. The Johns Hopkins University Bloomberg School of Public Health Education and Research Center Lecture Series. Environmental Chemicals in Human Milk. 2 December. Baltimore, Maryland.

Invited speaker. 2002. US Environmental Protection Agency Children's Health and Protection Advisory Council Science and Regulatory Work Group. 15 October. Washington, DC. Invited speaker. 2002. Breast milk monitoring for environmental chemicals in the U.S. Summary Expert Panel Workshop, Hershey, PA. Workshop on Chemicals and Drugs in Breast Milk. National Institutes of Health. April 24. Bethesda, Maryland.

Pittinger CA, LaKind JS. 2001. Weighing ecological risks and societal benefits: Pharmaceuticals and personal care products in the environment. 22nd Annual Society of Environmental Toxicology and Chemistry Meeting. November 15. Baltimore, Maryland.

Invited speaker. 2001. Protocol for breast milk monitoring for environmental chemicals. Toxic Chemicals in Breast Milk: A National Workshop to Assess Hazards to Children's Health of Chemical Contaminants in Breast Milk. Center for Children's Health and the Environment, Mt Sinai School of Medicine. October 5. New York City, New York.

LaKind JS, Berlin CM. 2001. Developing a protocol for breast milk monitoring for environmental chemicals: Workshop overview. International Society of Exposure Analysis Annual Meeting. November 4-8. Charleston, South Carolina.

LaKind JS, Berlin CM, Naiman DQ. 2001. Infant exposure to chemicals in breast milk in the United States: What we need to learn from a breast milk monitoring program. Presented at the Children's Environmental Health II: A Global Forum for Action. September 8. Washington, DC.

LaKind JS, Berlin CM. 2000. PDBEs in breast milk: Where do we go from here? Dioxin2000. August 13-17. Monterey, California.

LaKind JS, Berlin CM, Naiman DQ, Park CN. Characterization of dose distributions of selected breast milk contaminants to nursing infants: DDE and TCDD. American Public Health Association Annual Meeting, November, 1999; Society for Risk Analysis Annual Meeting, December, 1999; and Dioxin2000, Monterey, California, August 13-17, 2000.

Invited speaker. 1998. Principles of toxicology. School Nurse Institute. August 5. Towson, Maryland.

Invited speaker. 1998. Alchemy, risk assessment, and other phenomena. Lawrence University Science Colloquium. April 17. Appleton, Wisconsin.

Invited speaker. 1997. Managing risk in the face of scientific uncertainty. The Center for Technology, Environment, and Development (CENTED). Clark University. September 26. Worcester, Massachusetts.

Williams LG, Fendick E, LaKind JS, Stern B, Strand JA, Tardiff RG. 1995. Risk-based water quality criteria for treated mine-tailings effluent. Second World Congress of the Society of Environmental Toxicology and Chemistry.

Invited speaker. 1994. Comparison of human health risk assessment modeled data with observed data: Dioxin and lead. University of Guelph Department of Statistics. Guelph, Canada.

Invited speaker. 1993. Morgan State University Chemistry Department. Lecture on aquatic chemistry concepts and environmental and regulatory applications.

Invited speaker. 1992. Contradictions between Predictions and the Real World. National Association of Health Professionals Annual Conference. Norfolk, VA.

LaKind JS, Naiman DQ. 1991. Comparison of predicted and observed dioxin levels in fish: Implications for risk assessment. Society for Risk Analysis Annual Meeting.

LaKind JS, Rifkin E. 1991. A coordinated approach to dioxin regulation. Presented at Dioxin: National Conference on Establishing Multimedia Controls. May, 1991. Washington, DC.

Invited speaker. 1991. Use of the BCF in criteria development for hydrophobic compounds. Virginia Water Pollution Control Association Annual Conference.

LaKind JS, Rifkin E. 1990. Current method for setting dioxin limits in water requires reexamination. Dioxin and PCBs: National Conference on Approaches to Address Human Health Risks and Aquatic Life Impacts. May 10-11, 1990. Washington, DC.

LaKind JS, Rifkin E. 1990. Alternative approach for developing criteria for hydrophobic substances. 11th Annual Meeting of the Society of Environmental Toxicology and Chemistry.

LaKind JS, Stone AT. 1988. Reductive dissolution of goethite by substituted phenols. Annual Meeting of the American Geophysical Union.

LaKind JS, Stone AT. 1986. Reductive dissolution of goethite and hematite by substituted phenols. Annual Meeting of the American Geophysical Union.

LaKind JS, Brown PE. 1984. Characterization of the gold-bearing fluid at Red Lake, Ontario. Annual Meeting of the Geological Association of Canada- Mineralogical Association of Canada.

Professional Activities/Recognition:

Special Issue Guest Editor (with J. Domino). 2024. *Journal of Environmental Exposure Assessment*. Guest editorial: Domingo JL, LaKind JS. Environmental chemicals in breast milk and infant formula: measurements, interpretation, and communication. *J. Environ. Expo. Assess.* 2024, 3, 25. <http://dx.doi.org/10.20517/jeea.2024.49>.

Insights Editor (founder). 2024 - present. *Environment International*.

Special Issues Editor. 2023-2024. *Environment International*.

Member. 2022 – 2024. Justice, Equity and Risk Specialty Group, Society for Risk Analysis.

Society of Toxicology. Junior Councilor, SOT Exposure Specialty Section. 2022-2023.

Mentor. 2021 – present. The Johns Hopkins University Mentoring Program.

Invited panelist. National Academies Committee on Guidance on PFAS Testing and Health Outcomes Information Gathering Session. 2021.

Member, Peer Consultation on Biomonitoring Data and Reverse Dosimetry to Estimate Chemical Exposures. 2021. FDA/CFSAN/Versar.

Member, Technical Organizing Committee. 2021. International Society of Exposure Science Annual Meeting. ISES. 2020 - 2022. Ethics Committee.

EPA Grant Review Panel. 2020.

Steering Committee, 2020-present. i-HBM (International Human Biomonitoring) Working Group, ISES.

Session chair. 2020. Epidemiology, Exposure Science, and Risk Assessment: We need each other. International Society of Exposure Science. 22 September.

Member, HESI Assembly. 2019-2020.

Member, 2019 - 2020. Core Science Panel of the Beyond Science and Decisions Workshop Series.

Special issue editor. 2019. International Journal of Environmental Research and Public Health. Special Issue: Environmental Health Study with Remote Sensing Technologies: Exposure Assessment and Health Outcomes.

Appointed member. Health Effects-Energy Research Committee. December 18, 2017-2023.

ISES Committee member, Diversity, General Scientific Meetings Committees. January -December 2019.

ISES Vice Chair, Finance Committee, January-December 2019.

ISES Past President. January-December 2019.

ISES President. 2017-2018.

Session co-chair. 2018. Society Presidents' Call for Discussion: Intersection of Epi, Exposure and Decision-Making: Data Quality for Public Health Protection. International Society of Exposure Science/International Society of Environmental Epidemiology. Ottawa, Canada. 29 August.

Session co-chair. 2018. Exploring Current Worker Exposure Tools and Their Capability to Support Risk Evaluations of Chemicals under Amended TSCA. International Society of Exposure Science/International Society of Environmental Epidemiology. Ottawa, Canada. 28 August.

Session co-chair. 2018. Strengthening Exposure Assessment in Environmental Epidemiology: Problem Identification and Suggestions for Path Forward International Society of Exposure Science/International Society of Environmental Epidemiology. Ottawa, Canada. 28 August.

Invited member. 2018. Organizing Committee of the Conference on Uncertainty in Risk Analysis, 2019, Berlin, Germany.

Invited member. 2018. Technical Advisory Board, Total Exposure Health Conference and Workshop "Total Exposure Health: Bridging Exposure Science and Precision Medicine".

ISES Committees. ex officio member, all Committees, 2017-2018.

Founder, ISES Newsletter, 2017. Editorial Board, ISES Newsletter, 2017-2019.

Invited member. 2017. HESI Epidemiology “Best Practices” Project.

Session co-chair. 2017. International Society of Exposure Science Annual Meeting. 18 September. Durham NC. Exposure Assessment and Epidemiology for Regulatory Decision Making- Challenges and Opportunities (with June Yan). Durham, NC. 18 October.

Session co-chair. 2017. International Society of Exposure Science Annual Meeting. 2,4-D – A Case Study of Decades of Exposure Science; A Discussion of Quality, Quantity, and Harmonization (with Carol Burns). Durham, NC. 19 October.

Session Organizer. 2017. 2,4-D Human Exposure Data: Lessons from Decades of Study. American Chemical Society 254th Annual Meeting. Washington DC. 21 August.

Invited reviewer. 2017. Research-Practice Grants. Gulf Research Program of the National Academies of Sciences, Engineering, and Medicine. Washington DC. 12 September.

Invited reviewer. 2017. Minnesota Department of Health (MDH) revised health-based values for water. PFOS and PFOA.

Invited member. 2017. GRADE Guidance for Modelled Data Working Group. Hamilton, Ontario. 15-16 May.

Invited member. 2017. Risk Of Bias In Non-randomized Studies of Exposures (ROBINS-E) Working Group. Bristol, UK. 30-31 January.

HESI RISK21 Science Advisory Board. 2017-2020.

2017 SOT Regulatory and Safety Evaluation Specialty Section Award: Best Paper Contributing to the Field of Regulatory and Safety Evaluation in Toxicology. Beck et al. Approaches for describing and communicating overall uncertainty in toxicity characterizations: U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) as a case study. *Environment International* 89–90:110–128.

Member, Technical Organizing Committee. 2017. International Society of Exposure Science Annual Meeting.

Reviewer. 2017. Using 21st Century Science to Improve Risk-Related Evaluations. The National Academies Press.

Symposium Chair (with M. Mortensen). 2016. Biomonitoring: The Genie is out of the Bottle: Challenges in Data Quality and Interpretation. International Society of Exposure Science. Utrecht, The Netherlands. 12 October.

Symposium Chair (with D. Mattison). 2016. Harmonization, access, transparency: improving environmental epidemiology for public health decision-making. International Society of Exposure Science. Utrecht, The Netherlands. 10 October.

Invited member. 2016. National Institutes of Health Working Group - Risk Of Bias In Non-randomized Studies of Exposures. 2016.

Invited member. Epidemiology and Risk Assessment Expert Panel. 8 April 2016.

Invited member. EPA Expert Workshop on Aggregate Exposure Pathway: A Conceptual Framework to Advance Exposure Science Research and Complete the Source-to-Outcome Continuum for Risk Assessment. May 9-11, 2016. Research Triangle Park, North Carolina.

Invited member, Maryland Department of Health and Mental Hygiene (DHMH) Cancer Cluster Advisory Committee. 2016.

Membership Committee, Society for Risk Analysis. 2016.

President-Elect, International Society of Exposure Science. 2016.

Member, Technical Organizing Committee. 2016 International Society of Exposure Science Annual Meeting.

EPA Scientific and Technological Achievement Award (STAA) Level III for 2015 for: Providing Critical Models and Information Needed for Exposure and Risk Assessments of Environmental Chemicals in Infants.

Invited member, Review Panel, National Cancer Institute Laboratory of Metabolism (LM) of the NCI Intramural Program. September 16-18, 2015. Bethesda MD.

Jury member, ISES representative. 2015 LRI Innovative Science Award.

Invited participant. 2015. Institute of Medicine's Roundtable on Environmental Health Sciences, Research, and Medicine Workshop: The Interplay between Environmental Exposures and Obesity. March 2-3, Research Triangle Park, NC.

Co-Chair (with Dr. Benjamin Blount, CDC), 2015 Annual Meeting, International Society of Exposure Science. Henderson, NV. 18-22 October.

Founder, ISES Women's Networking Event. 2014.

Member, Diversity Committee. 2015 - present. International Society of Exposure Science.

Member, Nominations Committee. 2014 - present. International Society of Exposure Science.

Member, General Scientific Meetings Committee. 2014 - present. International Society of Exposure Science.

External Peer Reviewer. 2013. America's Children and the Environment. Third Edition. Environmental Protection Agency. EPA 240-R-13-001.

Grant Proposal Review. Health Canada's Chemicals Management Monitoring and Surveillance Fund. 2013.

Appointed member. Maryland Pesticide Reporting and Information Workgroup. June 2013.

Grant Proposal Review. Research Foundation - Flanders (Fonds Wetenschappelijk Onderzoek - Vlaanderen, FWO). April 2013.

Facilitator, Best Practices for Obtaining, Interpreting and Using Human Biomonitoring Data in Epidemiology and Risk Assessment: Chemicals with Short Biological Half-Lives. April 10-12, 2013. Baltimore, MD.

Facilitator, Advancing Cancer Cluster Assessments: Starting the Dialogue. April 3-5, 2013. Baltimore, MD.

Editorial Board. 2013. *Environment International*. February 2013- present.

Scientific Program Committee, 2013. Environmental Health Conference, Basel, Switzerland. 19-23 August. Joint conference of the International Society of Environmental Epidemiology (ISEE), International Society of Exposure Sciences (ISES) and International Society of Indoor Air Quality (ISIAQ).

Councilor, International Society of Exposure Science. 1 January 2013 – 31 December 2015.

Board of Directors, National Swimming Pool Foundation. 1 November 2012 – 28 October 2015.

Invited participant. 2012. Expert Workshop on Approaches to Improving the Risk Assessment of Persistent, Bioaccumulative and Toxic (PBT) Chemicals in Breast Milk. Environmental Protection Agency, Research Triangle Park, North Carolina. October 24-26.

Discussion Leader. 2012. Swimming Pools: Chemistry and Respiratory Effects, Gordon Research Conference, Drinking Water Disinfection Byproducts. Mount Holyoke College, August 5-10.

Panel member. 2012. US Environmental Protection Agency Science Advisory Board Panel on Perchlorate - Approaches for Deriving Maximum Contaminant Level Goals for Drinking Water.

Invited participant. Experts panel on exposure to swimming pool disinfection by-products and asthma and allergy effects. Porto, Portugal. 15 March 2011.

Mentor. 2011 - present. International Society of Exposure Science Mentor Program.

Facilitator, Children's Environmental Health & Protection Advisory Council: Feasibility of Biomonitoring in Maryland: An Open Meeting & Discussion. 1 April 2011. Laurel, MD.

Grant Proposal Review. Health Canada's Chemicals Management Monitoring and Surveillance Fund. 2011.

Grant Proposal Review. Health Canada's Chemicals Management Plan Monitoring & Surveillance Fund. 2011.

Grant Proposal Review. Human and Social Sciences, Epidemiology and Public Health, National Cancer Institute, France. 2011.

Institute of Medicine Committee on Blue Water Navy Vietnam Veterans and Agent Orange Exposure. May 2010 - 2011.

Graduate Council, UMBC. Associate member. April 2010 – present.

Grant Proposal Review: NIEHS. Superfund Basic Research and Training Program. October 2009.

Environmental Health Advisor, Maryland Department of the Environment Science Services Administration. June 2008-June 2009.

Grant Proposal Peer Review: NIEHS R21. Research to Action: Assessing and Addressing Community Exposures to Environmental Contaminants. July 2009.

Grant Proposal Peer Review: AAAS Research Competitiveness Service; Washington State's Life Sciences Discovery Fund. 2009.

Society of Toxicology Risk Assessment Specialty Section 2008 Top Ten Publications Advancing the Science of Risk Assessment awarded to Hays, S.M., Aylward, L.L., LaKind, J.S., et al. 2008. Guidelines for the Derivation of Biomonitoring Equivalents: Report from the Biomonitoring Equivalents Expert Workshop. *Regulatory Toxicology and Pharmacology* 51(3, Suppl 1):S4-S15.

Society of Toxicology Risk Assessment Specialty Section 2008 Top Ten Publications Demonstrating an Application of Risk Assessment awarded to Aylward LL, LaKind JS, et al., *J Toxicol Environ Health A* 71(22):1499-1508.

Board of Directors, U.S. – Montenegro Business Council. January -September, 2009.

Project Committee. 2008. *Maryland's Children and the Environment*. August. <http://www.dhmh.state.md.us/reports/pdf/MDChildrenEnv08.pdf>

Associate Editor. *Journal of Exposure Science and Environmental Epidemiology* 2008-2014.

Aquatics International Power 25. 2008. http://www.aquaticsintl.com/2008/feb/0802_power.html

Workshop Facilitator. 2007. Workshop on Childhood Asthma and Environmental Exposures at Indoor Swimming Pools. Advancing the Science. 21-24 August. Leuven, Belgium.

Associate Editor. 2006. Environmental and Neurodevelopmental Disorders. Special Issue of *NeuroToxicology*, vol 27, Issue 5.

Invited participant. 2006. WHO Consultation to Develop a Strategy to Estimate the Global Burden of Foodborne Diseases. 25-27 September. Geneva, Switzerland.

Workshop Co-Instructor (D. Barr, A. Calafat, L. Needham). 2005. Exposure Assessment for Environmental Chemicals Using Biomonitoring. International Society for Exposure Analysis. Tucson, Arizona. November, 2005.

Symposium Chair (with B. Blount). 2005. Environmental Chemicals in Human Milk. International Society for Exposure Analysis. Tucson, Arizona. November, 2005.

Organizing Committee. 2005. Twenty-Second International Neurotoxicology Conference. Environment and Neurodevelopmental Disorders. Research Triangle Park, NC. 11-14 September.

Workshop Steering Committee and Organizer. 2005. Hershey Medical Center Technical Workshop: Optimizing the Design and Interpretation of Epidemiologic Studies for Assessing Neurodevelopmental Effects from In Utero Chemical Exposure. Research Triangle Park, NC. 14 September, 2005.

Session Co-chair (with L.L. Needham). Body Burden and Dietary Intake, Dioxin 2005. Toronto, Canada. August, 2005.

Invited Participant: International Biomonitoring Workshop, ILSI Health and Environmental Sciences Institute, Research Triangle Park, NC, September, 2004.

Member, World Health Organization Survey Coordinating Committee for the WHO Global Survey of Human Milk for Persistent Organic Pollutants (POPs). Since 2004.

Workshop Organizer (with C.M. Berlin): Second Technical Workshop on Human Milk Surveillance and Biomonitoring Research on Environmental Chemicals in the United States. Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, 24-26 September 2004.

Symposium Chair. 2003. Society for Risk Analysis Annual Meeting. Uses and Interpretation of Human Biomonitoring Data. Baltimore, MD. December 7-10.

Technical Program Committee. 2003. Dioxin2003, Boston, MA. Session Chair: Public Health Decision-Making and Resource Allocation: Dioxin and Other PBTs as a Case Study.

Guest Editor. 2002, 2005. *Journal of Toxicology and Environmental Health*, issues on the Technical Workshop on Human Milk Surveillance and Research on Environmental Chemicals in the United States.

Workshop Organizer (with C.M. Berlin): Technical Workshop on Human Milk Surveillance and Research on Environmental Chemicals in the United States. Milton S. Hershey Medical Center, Pennsylvania State University College of Medicine, 15-17 February 2002.

Appointed Member: Maryland's Children's Environmental Health and Protection Advisory Council, December 2000 – July 2008.

Appointed Member: Maryland Lead Poisoning Prevention Commission, January 2000 – February 2002.

Invited Award Selection Panel Member: USEPA Science Achievement Award in Water Quality. 1998.

Guest Editor: *International Journal of Environment and Pollution*. Special Issue on Environmental Risk Assessment: Issues and Methods. Vol. 9, No. 1. 1998.

Session Organizer and Chair: Emerging EPA Guidance: Implications for the Pulp and Paper Industry. Annual TAPPI Environmental Division Conference, May 5-7, 1997.

TAPPI, Technical Program Committee Member. 1996 - 1997.

Technical Editor: Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing (1995 Edition). US Department of Housing and Urban Development.

Symposium Chair: Society for Risk Analysis Annual Meeting. Organized session on *Predicting Blood Lead Levels: Models and Applications*. December, 1994.

Invited Participant: Alliance for the Chesapeake Bay Roundtable on the Toxics Reduction Strategy of the Chesapeake Bay Program. Baltimore, May, 1994.

Invited Participant: Washington State Departments of Health and Ecology Sediment Scientific Review Board. Seattle, 1993.

Participant: Scientific Working Conference on Bioaccumulation of Hydrophobic Organic Chemicals. Institute for Evaluating Health Risks, Washington DC, June 1992.

Editorial Board: *Journal of Toxicology and Environmental Health*. 1992-2024.

Editorial Board: *Environmental Toxicology and Chemistry*. 1996-1998.

Peer Reviewer: *Environmental Health Perspectives, Journal of Exposure Science and Environmental Epidemiology, Chemosphere, Risk Analysis: An International Journal, Public Health Reports, Environmental Research, Journal of Pediatric Gastroenterology and Nutrition: An International Journal of Clinical, Experimental and Developmental Investigation, Toxicology and Applied Pharmacology, Integrated Environmental Assessment and Management, Reproductive Toxicology, Food and Chemical Toxicology, Environment International, Environmental Pollution, Reviews on Environmental Health, Toxicology and Industrial Health, Critical Reviews in Toxicology, International Journal of Hygiene and Environmental Health*

Member of Board of Directors, Advisory Board, and past President: Baltimore Coalition Against Childhood Lead Poisoning, Inc., Coalition for a Lead Safe Environment. 1992-1994.

Guest Editor: *Journal of Toxicology and Environmental Health*, 1991.

American Chemical Society Graduate Student Award in Environmental Chemistry. 1987.

On-line media:

ROBINS-E Development Group (Higgins J, Morgan R, Rooney A, Taylor K, Thayer K, Silva R, Lemeris C, Akl A, Arroyave W, Bateson T, Berkman N, Demers P, Forastiere F, Glenn B, Hróbjartsson A, Kirrane E, LaKind J, Luben T, Lunn R, McAleenan A, McGuinness L, Meerpohl J, Mehta S, Nachman R, Obbagy J, O'Connor A, Radke E, Savović J, Schubauer-Berigan M, Schwingl P, Schunemann H, Shea B, Steenland K, Stewart T, Straif K, Tilling K, Verbeek V, Vermeulen R, Viswanathan M, Zahm S, Sterne J). Risk Of Bias In Non-randomized Studies - of Exposure (ROBINS-E). Launch version, 1 June 2022. Available from: <https://www.riskofbias.info/welcome/robins-e-tool>.

LaKind JS. 2018. Webinar: Chemical exposures and health effects: What we know and what we don't know from epidemiology research. CME through Accreditation Council for Continuing Medical Education (ACCME). Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Education and Research Center for Occupational Safety and Health. <https://www.jhsph.edu/research/centers-and-institutes/johns-hopkins-education-and-research-center-for-occupational-safety-and-health/ce/ChemicalEpiCME>

LaKind JS. 2106. Webinar: Environmental Contributions to Asthma Prevalence: Assessing the Link between Exposure and Disease. Advancing the Science Webinar Series: Chemical-Induced Asthma. University of Cincinnati College of Medicine. 17 June.

LaKind JS. 2013. Soapbox Science, Nature.com Guest blog. Environmental chemicals in our bodies – we know they are in there, but what does it mean? <http://blogs.nature.com/soapboxscience/2013/01/02/environmental-chemicals-in-our-bodies-we-know-they-are-in-there-but-what-does-it-mean> 2 January.

Exposure science video for the International Society of Exposure Science. “Get connected - join the International Society of Exposure Science!!” <https://www.youtube.com/watch?v=Qcx65X5Davo>

Research/Grants:

Investigator: Pilot Study on Concentrations of PBDEs in Human Milk (with Drs. C. M. Berlin, Jr. and I. Paul, Milton S. Hershey Medical Center, Penn State College of Medicine, and Dr. D. Patterson, Centers for Disease Control and Prevention). Cooperative Agreement CR-83150601-0 from the US Environmental Protection Agency. 2003.

Investigator: Partitioning and Elimination Kinetics Study of Human Milk and Blood (with Drs. C. M. Berlin, Jr. and I. Paul, Milton S. Hershey Medical Center, Penn State College of Medicine, and Drs. A. Sjödin and D. Patterson, Centers for Disease Control and Prevention). 2004.

Investigator: Human Milk Biomonitoring For Environmental Chemical (Volatile Organic Compound) Exposures (with Dr. K Squibb, University of Maryland School of Medicine and Dr. B. Blount, Centers for Disease Control and Prevention). 2005.

Principle Investigator. Review of Neurodevelopmental Function Tests in Children (with Drs. Eric Youngstrom, Michael Goodman, Katherine Squibb, Paul H. Lipkin, Laura Gutermuth Anthony, Lauren Kenworthy, Donald R. Mattison). Cefic/LRI Research Grant. 2009.

Principle Investigator. Development of Guidelines for Addressing Contamination and Associated Toxicity in Freshwater/Marine/Estuarine Sediments. Maryland Department of the Environment. 2009-2010.

Principle Investigator. Critical review of epidemiological evidence for the potential association between endocrine active chemicals and obesity, diabetes and cardiovascular disease (with Drs. Donald Mattison, Michael Goodman). Cefic/LRI Research Grant. 2013.

Principle Investigator. Exploring the Design Elements for Successful Public-Private Partnerships (PPPs) for Community Environmental Monitoring Programs (with Drs. Ana Rule and Fernando Wagner). Foundation for Chemistry Research and Initiatives Research Grant. 2022.

MPI (with Dana Boyd Barr [Emory] and Daniel Q. Naiman [Johns Hopkins]). Does NHANES underestimate true population-based exposures to pesticides? Exploring bias in NHANES human biomonitoring data." NIEHS RO3. 2023.

Selected Co-Authored Reports/Articles:

LaKind JS, Naiman J. 2022. White Paper: Review of the PFAS Personal Intervention Literature, Appendix E. In: National Academies of Sciences, Engineering, and Medicine 2022. Guidance on PFAS Exposure, Testing, and Clinical Follow-Up. Washington, DC: The National Academies Press. <https://doi.org/10.17226/26156>.

HEI Energy Research Committee. Rosofsky A, Dunn-Norman S, Ebelt S, Hornberger G, Hu H, LaKind JS, Russell AG, Thorne PS, Adelsheim LA, Vorhees DJ. 2022. Recommendations for epidemiologic research to inform environmental health policy for unconventional oil and gas development.

HEI Energy Research Committee. 2020. Human Exposure to Unconventional Oil and Gas Development: A Literature Survey for Research Planning (FINAL COMMUNICATION). Communication 1. June 2020.

HEI Energy Research Committee. 2019. Potential Human Health Effects Associated with Unconventional Oil and Gas Development: A Systematic Review of the Epidemiology Literature (FINAL REPORT). Special Report 1. September 2019

Environmental Protection Perchlorate Advisory Panel. 2013. SAB Advice on Approaches to Derive a Maximum Contaminant Level Goal for Perchlorate Final Report.

IOM Committee. 2011. Blue Water Navy Vietnam Veterans and Agent Orange Exposure. The National Academies Press. Washington DC.

LaKind JS, Blatchley ER. 2011. The ABCs of DBPs. Aquatics International. February.

http://www.aquaticsintl.com/2011/feb/1102_tech.html

University of Maryland. 2009. Standard Operating Procedures for Fish and Shellfish Collection and Analysis. For: Maryland Department of the Environment Science Services Administration. 22 May.

University of Maryland. 2009. Technical Support Document for Establishing Fish and Shellfish Consumption Advisories in Maryland. For: Maryland Department of the Environment Science Services Administration. 23 March.

LaKind Associates, LLC (with Dr. E.J. Bouwer). 2003. Investigation of the Removal of Formaldehyde and Phenol by Funeral Home Septic Systems. Prepared for the National Funeral Directors Association. May 2003.

LaKind Associates, LLC and ENVIRON International Corporation. 2002. Assessment of Triclosan Residues In Breast Milk Based on Available Data: Final Report.

LaKind Associates, LLC. Human Health Risk Evaluation of the Windsor Terminal Site, Baltimore, Maryland. December, 2000.

LaKind Associates, LLC. Onsite Human Health Risk Evaluation of TCE at the Sparks, Maryland Leica, Inc. Site. October, 1999.

The Sapphire Group, Inc. Distributions of Exposures Among Workers to Selected ETS-Related Chemicals in Indoor Workplace Air Using Data from the Oak Ridge 16-City Study. March, 1998.

The Sapphire Group, Inc. Critical Review of the USEPA's Proposed Rule for National Ambient Air Quality Standards for Particulate Matter. February, 1997.

EA Engineering, Science, and Technology, Inc. Ethylene Glycol: Scientific Rationale for Continued Listing on EPA's Toxics Release Inventory (TRI). Prepared for ARCO Chemical Company, February, 1996.

EA Engineering, Science, and Technology, Inc. Comparative Toxicity and Environmental Impacts of Ethylene Glycol and Propylene Glycol: A Review. Prepared for ARCO Chemical Company, February, 1996.

EA Engineering, Science, and Technology, Inc. Decision Support Document on Health Benefits and Health and Safety Associated with the Use of Methyl Tertiary Butyl Ether (MTBE) in Gasoline. Prepared for ARCO Chemical Company, December, 1995.

EA Engineering, Science, and Technology, Inc. Report on Toxins Analysis and Assessment (Phase I). Prepared for International Paper Company, November, 1995.

EA Engineering, Science, and Technology, Inc. Phase II Site Investigation Camp Buckner Skeet and Trap Range, U.S. Military Academy, West Point, New York. Prepared for U.S. Army Corps of Engineers - Baltimore District, November, 1995.

EA Engineering, Science, and Technology, Inc. Technical Papers on MTBE and Human Health. Health Benefits Analyses. Prepared for ARCO Chemical Company, October, 1995.

EA Engineering, Science, and Technology, Inc. Human Health Risk Assessment of Manufactured Gas Plant Residuals and Other Chemicals at Baltimore Gas & Electric Company's (BGE) Spring Gardens Facility — Evaluation of the Need for Additional Offsite Information to Conduct an Offsite Risk Assessment. Prepared for Baltimore Gas & Electric Company, March, 1995.

EA Engineering, Science, and Technology, Inc. Preliminary Analysis of Health Risk for the Proposed Kensington Mine Submarine Discharge. Prepared for confidential client. 1994.

EA Engineering, Science, and Technology, Inc. Human Health Risk Assessment of Manufactured Gas Plant Residuals and Other Chemicals to Construction Workers at Baltimore Gas & Electric Company's (BGE) Spring Gardens Facility. Prepared for Baltimore Gas & Electric Company, November, 1994.

EA Engineering, Science, and Technology, Inc. Environmental Impact Analysis: Blue Mountain Sportsman's Center. Prepared for Westchester County, September, 1994.

EA Engineering, Science, and Technology, Inc. Modeled Predictions of Disinfection By-Products for the Baltimore Water Supply System After Implementation of Zebra Mussel Control. Prepared for KCI Engineers, February, 1994.

Student Mentoring:

2024-present: International Society for Exposure Science Mentor Program. Alexandra Del Favero-Campbell, Ph.D. candidate, Dalhousie University.

2024-present: mentoring Melissa Vendramini, student at Lakewood Ranch High School, Bradenton, Florida.

2021. Facilitator. International Society for Exposure Science Webinar: Top tips for Writing an Academic and Industrial Curriculum Vitae. 8 November.

2021-present. Johns Hopkins Engineering Mentoring Program.

2018-present. Dissertation committee member. Cecilia Alcala, Tulane University Ph.D. candidate. Awarded Ph.D. in 2020.

2014-2017. Doctoral defense committee member. Huan Xia, UMBC Ph.D. candidate. Awarded Ph.D. in 2017.

2012-2013 International Society for Exposure Science Mentor Program. Satori Marchitti, Ph.D., US Environmental Protection Agency, National Exposure Research Laboratory.

2012. Eric Sewell, summer intern, Johns Hopkins University Department of Applied Mathematics and Statistics.

2011-2012. International Society for Exposure Science Mentor Program. Liesel M. Seryak, Ph.D. candidate, The Ohio State University College of Public Health.

2011. Doctoral defense committee member. Piuly Paul, UMBC Ph.D. candidate.

2009. Mentor, Maryland Department of the Environment, Chunxiao Zhu, MS candidate, Department of Geography & Environmental Engineering, Johns Hopkins University.

2009. Mentor, Maryland Department of the Environment, Edward Berg, MS candidate, Department of Geography & Environmental Engineering, Johns Hopkins University.

Employment History:

Employer: LaKind Associates, LLC
Employed: June 1998 - present
Title: Founder, President

Employer: University of Maryland Baltimore County
Employed: January 2010 – May 2010
Title: Part Time Instructor, College of Engineering & Information Technology

Employer: University of Maryland School of Medicine
Employed: September 2008 – 2009
Title: Associate Professor

Employer: University of Maryland School of Medicine
Employed: July 2008 – June 2009
Title: Environmental Health Advisor, Maryland Department of the Environment

Employer: University of Maryland School of Medicine
Employed: May 2003 – present
Title: Adjunct Associate Professor

Employer: University of Maryland School of Law
Employed: May 2003 – May 2004
Title: Adjunct Associate Professor

Employer: The Sapphire Group
Employed: January 1997 - May 1998
Title: Co-founder, Vice President, and Managing Principal

Employer: EA Engineering, Science and Technology, Inc.
Employed: September 1993 - December 1996
Title: Senior Scientist

Employer: The Johns Hopkins University
Employed: September 1991 - 1994
Title: Instructor, Aquatic Chemistry

Employer: The Johns Hopkins University
Employed: September 1993 - December 1994
Title: Instructor, Environmental Risk Assessment

Employer: Self-employed, JSL Consulting
Employed: June 1991 - August 1993
Title: Environmental Consultant

Employer: Rifkin & Associates, Inc.
Employed: October 1988 - May 1991
Title: Senior Associate

Employer: U.S. Environmental Protection Agency, Office of Federal Activities
Employed: 1988
Title: Geologist

APPENDIX 2: SHOWER model factors and options for modifications

The following table shows the ATSDR SHOWER model parameters and the exposure assessment factors that can be modified. Unless otherwise specified in the main body of this Report, default values were used. The chemical information characterizes the water concentration of the chemical; the other factors describe the people and residence being modeled (ATSDR 2022c). There are also model parameters related to properties of chemicals; the values for these were not modified and these are not shown here.

Factor type	Options
Chemical information¹	
Concentration in water	User-specified
Units	User-specified (e.g., ppm, ppb)
Concentration in air ²	User-specified
Units	User-specified (e.g., ppm, µg/m ³)
Report units	ppb or µg/m ³
Household scenarios	
Number of people	From 1 to 8
Number and time of showers/baths	Morning or evening
Exhaust fan when bathroom occupied	Open or closed
Bathroom door when bathroom occupied	Open or closed
Exposure group ³	9 standard ATSDR groups
House information	
Number of bathrooms with showers	1 or 2
Shower/bath layout	Bathtub with shower or separate ⁴
Clothes washer location	Main house or bathroom ⁵
Exhaust fan location	Bathroom or shower ⁶
Area volumes (house, bathroom, shower)	Default or user-specified ⁷
Appliance parameters	
Main house compartment parameters (Kitchen sink flow rate and duration per use, utility sink volume per person, dishwasher volume per cycle, cycle duration and start time)	Default or user-specified ⁸

Clothes washer parameters (location, volume per cycle, cycle duration, start time)	Default or user-specified ⁹
Bathroom compartment parameters (sink flow rate, duration per use, toilet volume per flush)	Default or user-specified
Exhaust fan parameters (location and flow rate)	Default or user-specified
Bathtub volume	Default or user-specified
Shower flow rate	Default or user-specified
Activity Patterns	
Shower pattern for each modeled person (morning or evening; duration, time in bathroom after showering; optional bathtub setting)	Default or user-specified
Time between bathroom stay and next shower	Default or user-specified
Number of bathroom visits separate from shower	0 to 5
Kitchen sink uses	Maximum of 30
Activity start and end times (time when all morning/evening showers begin and are completed)	Default or user-specified
Time away from home for each resident	Default or user-specified.

¹ The sources for the chemical properties (e.g., molecular weight, f values, permeability coefficients) are described in ATSDR (2022c).

²If outdoor air concentration is not known, the default value is zero.

³A customized group can be added if total body surface area, hand surface area, body weight, daily breathing rate and shower and bathroom breathing rate are known.

⁴Bathtub with shower used in this Report as there was no indication in depositions of availability of separate bathtubs.

⁵Main house location used in this Report.

⁶Default of bathroom used in this Report; no other information available.

⁷See main Report for information on user-specified inputs.

⁸Defaults used in the Report; default start time of 9:00 pm would be conservative as people are modeled to be back in residence by that time.

⁹ Defaults used in the Report; default start time of 7:00 pm would be conservative as people are generally modeled to be back in residence by that time.

APPENDIX 3: ATSDR SHOWER model evaluation and sensitivity analysis

ATSDR utilized an EPA study (EPA 2000) to verify the SHOWER model. The EPA study provided sufficient experimental data on three chemicals (acetone, ethyl acetate, toluene) to verify shower air concentrations, but not for simulated air concentrations in the bathroom or the main house. In EPA's experiment, each of the following parameters was varied: temperature, showerhead flow rate, shower spray setting, and air exchange rate. ATSDR concluded that "Across all times and experiments, the average percent error \pm one standard deviation for each chemical was $12.6\% \pm 44.8\%$ for acetone, $0.81\% \pm 36.29\%$ for ethyl acetate, and $-15.0\% \pm 22.9\%$ for toluene. In general, the SHOWER model simulated concentrations were fairly accurate for ethyl acetate, were slightly above the measured concentrations for acetone, and were slightly below the measured concentrations for toluene. Overall, the simulated concentrations from the ATSDR SHOWER model are in good agreement with EPA's experimental results" (ATSDR 2022c, p. 20).

ATSDR conducted a sensitivity analysis of the SHOWER model v2.0.

The following is taken directly from the ATSDR Technical Manual for SHOWER v3.0 (pages D1-D2):

"ATSDR performed a sensitivity analysis on the model to determine the relative impact of changes to individual model parameters on the model-calculated inhalation concentrations and dermal doses. ATSDR performed the sensitivity analysis in SHOWER model v2.0 using a four-person household, four morning showers custom scenario which had the same parameters as those used in the SHOWER model v2.0 default scenario. ATSDR used chlorobenzene at a water concentration of 100 $\mu\text{g/L}$ as the contaminant in all sensitivity analysis simulations.

To run the sensitivity analysis, ATSDR changed the value of individual model parameters one by one while keeping all other parameters constant. ATSDR evaluated changes to each parameter in 10% increments within a range of $\pm 50\%$ of the original parameter value. ATSDR also evaluated the impact of binary (on/off, open/closed, etc.) parameters within the model individually. For each parameter change, ATSDR ran the SHOWER model and recorded the average daily exposure concentration and administered dermal dose for each age group and evaluated the percent difference in each output value from the default scenario's output. Table D1 shows example results from the sensitivity analysis for four model parameters after a 50% increase in each parameter value.

Table D1. Sample sensitivity analysis results for four parameters Parameter	SHOWER Model v2.0 Default Value	Value Increased by 50%	Inhalation Concentration Percent Difference	Adult Dermal Dose Percent Difference
Shower duration (all persons)	8 min	12 min	62.3%	17.5%
Shower flow rate	7.6 L/min	11.4 L/min	36.3%	0.0%

Bathroom volume	5 ft x 8 ft x 8 ft	5 ft x 12 ft x 8 ft	-11.7%	0.0%
Main house air exchange rate	0.45 ACH	0.675 ACH	-9.5%	0.0%

The sensitivity analysis showed that the SHOWER model's inhalation and dermal results were most sensitive to changes in human activity parameters, particularly shower duration. Inhalation concentrations were also sensitive to shower flow rate, bathroom volume, and bathroom air exchange rate; and dermal doses were also sensitive to parameters related to kitchen and bathroom sink usage."

APPENDIX 4: Supporting information

ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	Appliances on
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	0.17
2	—	Showering in Shower #1 at 6:35 a.m.	0.22
3	—	Showering in Shower #1 at 6:48 a.m.	0.28
4	X	Showering in Shower #1 at 7:01 a.m.	0.33

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	Appliances off
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Modified custom scenario
Basis for modified scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4-Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	0.13
2	—	Showering in Shower #1 at 6:35 a.m.	0.19
3	—	Showering in Shower #1 at 6:48 a.m.	0.24
4	X	Showering in Shower #1 at 7:01 a.m.	0.30

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Appendix 4.2 Effect of bathroom door open or closed on air concentrations of PCE (partial reports)



ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	Door closed
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4

Information	Report Setting
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4-Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	0.17
2	—	Showering in Shower #1 at 6:35 a.m.	0.22
3	—	Showering in Shower #1 at 6:48 a.m.	0.28
4	X	Showering in Shower #1 at 7:01 a.m.	0.33

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	Door open
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Modified custom scenario
Basis for modified scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4-Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	0.16
2	—	Showering in Shower #1 at 6:35 a.m.	0.22
3	—	Showering in Shower #1 at 6:48 a.m.	0.27
4	X	Showering in Shower #1 at 7:01 a.m.	0.31

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Appendix 4.3 Effect of outdoor air concentrations on indoor air concentrations of PCE
(partial reports)



ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	—
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	10 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person

Information	Report Setting
Most highly exposed person:	4
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4-Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	1.7
2	—	Showering in Shower #1 at 6:35 a.m.	2.2
3	—	Showering in Shower #1 at 6:48 a.m.	2.8
4	X	Showering in Shower #1 at 7:01 a.m.	3.3

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 4). This contact occurs from showering or bathing and from hand washing during the day. According to EPA guidance, dermal doses for this halogenated compound may be underestimated (see the SHOWER model technical document or EPA's RAGS, Part E for further details).

Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation
Adult	0.71

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day; NC = not calculated



ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	—
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	10 µg/L
Outdoor air concentration:	0.2 µg/m ³
Number of persons in household:	4
Household scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	1.9
2	—	Showering in Shower #1 at 6:35 a.m.	2.4
3	—	Showering in Shower #1 at 6:48 a.m.	3.0
4	X	Showering in Shower #1 at 7:01 a.m.	3.5

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

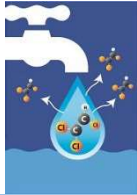
Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 4). This contact occurs from showering or bathing and from hand washing during the day. According to EPA guidance, dermal doses for this halogenated compound may be underestimated (see the SHOWER model technical document or EPA's RAGS, Part E for further details). Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation
Adult	0.75

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day; NC = not calculated



ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	—
Address:	—
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
Synonym:	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person
Most highly exposed person:	4

Information	Report Setting
Target person main activity:	7-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4).



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 6:22 a.m.	0.17
2	—	Showering in Shower #1 at 6:35 a.m.	0.22
3	—	Showering in Shower #1 at 6:48 a.m.	0.28
4	X	Showering in Shower #1 at 7:01 a.m.	0.33

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air



ATSDR SHOWER Model Report

Custom 4-Person Household

Four morning showers

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	
Address:	
Application:	Version 4.0.0
CASRN:	127-18-4
Contaminant:	Tetrachloroethylene
Synonym:	1,1,2,2-perchloroethylene
	1,1,2,2-tetrachloroethylene
	PCE
	PERC
	Perchloroethylene
	Tetrachloroethene
Model Input Information	
Chemical name:	Tetrachloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	4
Household scenario:	Modified custom scenario
Basis for modified scenario:	Four morning showers
Number of bathrooms in house:	1
Target person:	Most highly exposed person

Information	Report Setting
Most highly exposed person:	4
Target person main activity:	15-minute morning shower

Scenario Description

This report is for a custom 4-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 4). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

4-Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 4-person household, including the most highly exposed person (person 4). The air concentration estimated by the model is independent of who is showering and thus applies to both children and adults.



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Pers on	Targ et Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 5:50 a.m.	0.34
2	—	Showering in Shower #1 at 6:11 a.m.	0.56
3	—	Showering in Shower #1 at 6:32 a.m.	0.75
4	X	Showering in Shower #1 at 6:53 a.m.	0.92

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

APPENDIX 5: PHAST model parameters and options for modifications

The following table includes the inputs to the ATSDR PHAST model for drinking water ingestion and the exposure assessment factors that can be modified.

Factor type	Options
Chemical information	
Concentration in water	User-specified
Units	User-specified (e.g., ppm, ppb)
Type	User-specified (e.g., arithmetic mean, geometric mean)
Exposure groups and body weights	
Exposure group	Residential, daycare, school, or occupational
Age groups and body weights	Default and/or customized
Intake rates	
Drinking water intake rate	Default (CTE, RME) or and/or site-specific intake rates

APPENDIX 6: SWIMODEL: Hypothetical scenario reports

SWIMODEL hypothetical example

Swimmer Dermal and Ingestion Exposures to PCE

				Source
Log Kp = -2.72 + (0.71 x log Kow) - 0.0061 x MW			Units	SwiModel, 2003
Kow				
Log Kow	3.40			SIDS
MW = Molecular Weight	170		grams/mol	
Log Kp	-1.34			
Kp = Permeability coefficient	4.54E-02		cm/hr	
Conversion Factor Used for Dermal Exposures	0.001		L/1000 cm ³	
Concentration in Water	0.1		mg/liter	
Body Weight, Adult	80		kg	2011 EFH
Body Weight, Child 11 to <16	57		kg	2011 EFH
Body Weight, Child 6 to <11	32		kg	2011 EFH
Dermal Surface Area, Adult	19500		cm ²	2011 EFH
Dermal Surface Area, Child 11 to <16	15900		cm ²	2011 EFH
Dermal Surface Area, Child 6 to <11	10800		cm ²	2011 EFH
	Competitive	Non-Competitive		
Exposure Time, Adult	3	1	Hours/day	SwiModel, 2003
Exposure Time, Child 11 to <16	2	1	Hours/day	SwiModel, 2003
Exposure Time, Child 6 to <11	1	1	Hours/day	SwiModel, 2003
Ingestion Rate, Adult	0.0125	0.025	Liters/Hour	SwiModel, 2003
Ingestion Rate, Child 11 to <16	0.025	0.05	Liters/Hour	SwiModel, 2003
Ingestion Rate, Child 6 to <11	0.05	0.05	Liters/Hour	SwiModel, 2003
	Competitive Swimmers	Non-Competitive Swimmers		
	Dose	Dose		
	(mg/kg/day)	(mg/kg/day)		
Dermal				
Adult	3.3E-03	1.11E-03		

Child 11 to <16	2.5E-03	1.27E-03
Child 6 to <11	1.5E-03	1.53E-03
Ingestion		
Adult	4.7E-05	3.1E-05
Child 11 to <16	8.8E-05	8.77E-05
Child 6 to <11	1.6E-04	1.56E-04
Combined		
Adult	3.4E-03	1.1E-03
Child 11 to <16	2.6E-03	1.35E-03
Child 6 to <11	1.7E-03	1.69E-03

**Swimmer Inhalation Exposures to TCE - hypothetical
(Using Henry's Law which is the preferred
method)**

H' = HLC/(R x (T+273))				USE RSL unitless H'
Henry's law constant	HLC		Units	
Gas Constant	R	8.19E-05	atm-m3/mol	
Ambient air temp	T	25	at-m3/mole-K	
Conversion (K= C+273)		273	C	
Henry's Law constant	H'	7.20E-01	unitless	
Cvp = Cw x H' x 1000 L/m3				
Water conc	Cw	0.1	mg/liter	
Henry's Law Constant	H'	7.20E-01	unitless	
Conversion factor	CF	1000	liter/M3	
Vapor Conc	Cvp	7.20E+01	mg/m3	
PDR = Cvp x ET x IR / BW				
Exposure time (hr/day)	ET	Competitive	Non-comp	
adult		3.0		SWIMMODEL 2003
children (11-<16yrs of age)		2.0		SWIMMODEL 2003
children (6-<11yrs of age)		1.0		SWIMMODEL 2003
adult			1.0	NHAPS Study

children (11-<16yrs of age)	1.0	NHAPS Study
children (6-<11yrs of age)	1.0	NHAPS Study

Inhalation rate (m³/hr)	IR			
adult		3.2	1	EFH, 2011
children (11-<16yrs of age)		2.9	1.5	EFH, 2011
children (6-<11yrs of age)		2.5	1.3	EFH, 2011
body weight (kg)	BW			
all adults		80	80	EFH, 2011
children (11-<16yrs of age)		57	57	EFH, 2011
children (6-<11yrs of age)		32	32	EFH, 2011

	<i>PD</i>		
Adult dose (mg/kg/day)	<i>R</i>	8.6E+00	9.0E-01
	<i>PD</i>		
Child (11-<16) dose (mg/kg/day)	<i>R</i>	7.3E+00	1.9E+00
	<i>PD</i>		
Child (6-<11) dose (mg/kg/day)	<i>R</i>	5.6E+00	2.9E+00

Contents

Site-specific Input Parameters and Equations.....	145
Site-specific Surface Water Swimming Results for Acute Duration Exposures.....	147
Tetrachloroethylene	147
<i>Surface Water Dermal Only Acute</i>	148
Tetrachloroethylene	148

Site-specific Input Parameters and Equations

PHAST Report, v2.5.1.0, January 16, 2025

Site-specific Exposure Factors

Durat ion Category	Event Duration (hours/ev ent)	Event Frequenc y (events/d ay)	Da ys per Week	We eks per Year	Ye ars	Expos ure Group Specific EF _{noncancer}	Expos ure Group Specific* EF _{cancer}
Acute	1	1	-	-	-	1	-

Abbreviations: EF = exposure factor; NC = not calculated

Site-specific Exposure Parameters

Exposure Group	Body Weight (kg)	Exposure Duration (years)	CTE Intake Rate (L/hr)	RME Intake Rate (L/hr)	Custom Intake Rate (L/hr)	Combined Skin Surface Area (cm ²)	Notes
Adult	80	-	-	-	0.025	19,500	-

Abbreviations: cm² = centimeters square skin; CTE = central tendency exposure (typical); kg = kilograms; L/hr = liters per hour; RME = reasonable maximum exposure (higher)

Contaminant Information

Contaminant Name	Entered Concentration	EPC Type	Converted Concentration*	AB S _{GI}	DA _{event}
Tetrachloroethylene	0.1 mg/L	Arithmetic mean	0.1 mg/L	1	8.71E-06 mg/cm ² /event

Abbreviations: ABS_{GI} = gastrointestinal absorption factor; DA_{event} = absorbed dose per event; EPC = exposure point concentration; mg/cm²/event = milligrams per centimeter squared per event; mg/L = milligram chemical per liter water; mg/L = milligrams per liter

* Contaminant concentration converted to standard unit for calculating exposure.



Site-specific Surface Water Swimming Results for Acute Duration Exposures

PHAST Report, v2.5.1.0, January 16, 2025

Surface Water Ingestion Only Acute

Tetrachloroethylene

Table 2. Swimming: Site-specific ingestion only exposure doses for acute exposure to tetrachloroethylene in surface water at 0.1 mg/L

Exposure Group	Dose (mg/kg/day)
Adult	3.1E-05

Source: [list reference of environmental data]


Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water

* The calculations in this table were generated using ATSDR's PHAST v2.5.1.0.

Surface Water Dermal Only Acute

Tetrachloroethylene

Table 3. Swimming: Site-specific dermal only exposure doses for acute exposure to tetrachloroethylene in surface water at 0.1 mg/L

	Dose (mg/kg/day)
Exposure Group	
Adult	0.0021

Source: [[list reference of environmental data](#)]

Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water

* The calculations in this table were generated using ATSDR's PHAST v2.5.1.0.



ATSDR SHOWER Model Report

Custom 6-Person Household

Six morning showers

Contents

Site and Model Input Information	2
Scenario Description	3
6-Person Household Results - Tables	3
6-Person Household Results – Figures	6
Model Parameters	8
References	18

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	AMSLER HP RESIDENTIAL
Address:	—
Application:	Version 4.0.1
CASRN:	71-43-2
Contaminant:	Benzene
Synonym:	—
Model Input Information	
Chemical name:	Benzene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	6
Household scenario:	Modified custom scenario
Basis for modified scenario:	Six morning showers
Number of bathrooms in house:	1
Target person:	6
Most highly exposed person:	6
Target person main activity:	7-minute morning tub bath

Scenario Description

This report is for a custom 6-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 6). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

6–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 6-person household, including the most highly exposed person (person 6). The air concentration estimated by the model is independent of who is showering and thus applies to both children and adults.



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Person	Target Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 5:56 a.m.	0.31
2	—	Showering in Shower #1 at 6:09 a.m.	0.38
3	—	Showering in Shower #1 at 6:22 a.m.	0.43
4	—	Showering in Shower #1 at 6:35 a.m.	0.48
5	—	Showering in Shower #1 at 6:48 a.m.	0.53
6	X	Tub bath in Shower #1 at 7:01 a.m.	0.58

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 6). This contact occurs from showering or bathing and from hand washing during the day. Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation	Dermal
6 to < 11 years	0.27	0.0028

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day

Peak and Percentage Exposure By Location (Table 3 and 4)

This SHOWER model scenario consists of three compartments: a shower stall, the bathroom, and the main house. The next two tables show the amount of time the target person spends bathing (shower or bath) and the amount of time they spend in the bathroom afterwards. It also provides information on the amount of time the target person spends in the main house throughout the rest of the day. When the target person visits the bathroom during other parts of the day, those bathroom times are included in the main house exposure time. Table 3 shows the average exposure concentration that the target person experiences in each location, and Table 4 shows the percent of exposure that the target person experiences in each location.

The exposure from bathing (shower or bath) and being in the bathroom afterwards can be much higher (but for shorter periods) than the exposure from being in the main house. Knowledge of this brief exposure to high levels in the shower and bathroom compartments might be useful when evaluating whether harmful effects might be possible from acute exposure to high concentrations. This acute exposure to high levels might be particularly important for irritant chemicals, such as formaldehyde, 2-butanone, and acetone. Some irritants, however, cannot be run using the model because parameters are lacking. Health assessors should evaluate this acute exposure duration if the acute EMEG is exceeded. More information about evaluating acute exposure can be found in ATSDR's Guidance for Evaluating Inhalation and Dermal Exposure Using the SHOWER Model (ATSDR 2024a). **Health assessors should consult with the Associate Director of Science (ADS) when evaluating brief exposure to high levels.**



Table 3. Exposure time and average exposure concentration by location for the target person

Location	Exposure Time (min)	Average Exposure Concentration ($\mu\text{g}/\text{m}^3$)
Tub bath	7	50
Bathroom after tub bath	5	7.3
Main house with additional bathroom visits	1,428	0.31
Away from house	0	0
Average daily exposure	1,440	0.58
Main house (all day)	1,440	0.18

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; min = minute



Table 4. Exposure time and percent of total exposure by location for the target person

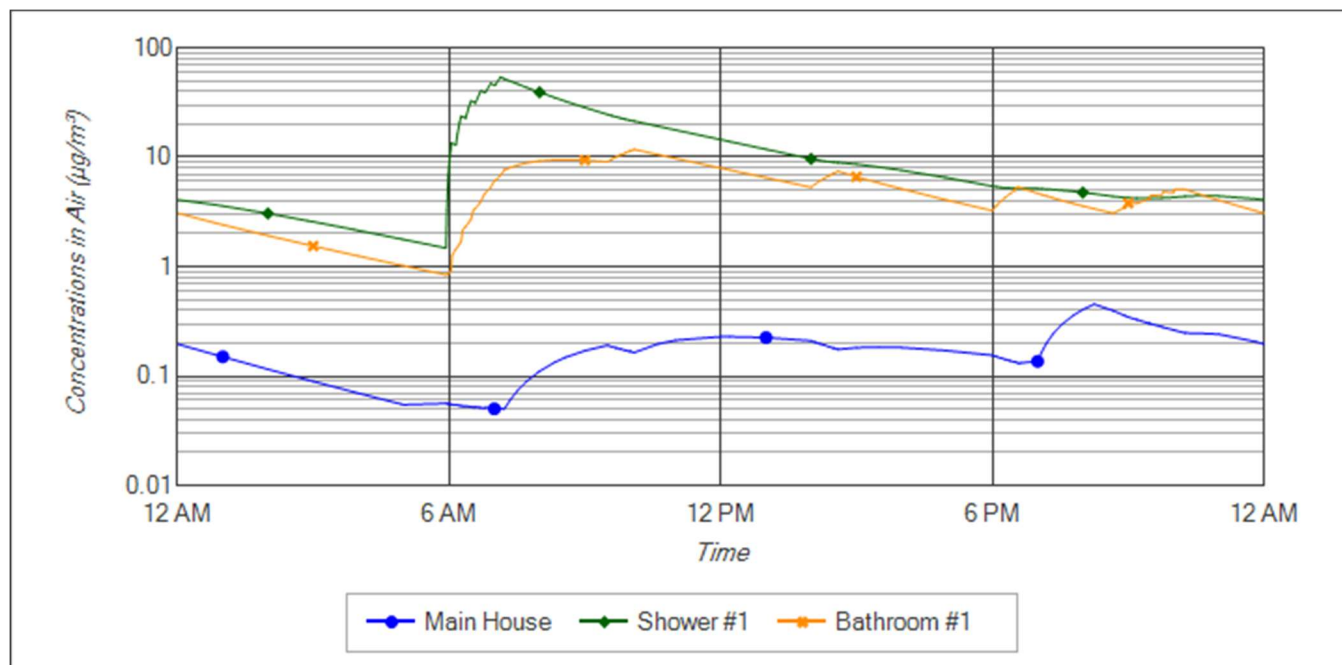
Location	Exposure Time (min)	Percent Exposure (%)
Tub bath	7	42
Bathroom after tub bath	5	4.4
Main house with additional bathroom visits	1,428	54
Away from house	0	0
Average daily exposure	1,440	100
Main house (all day)	1,440	100

Abbreviations: min = minute; % = percent

6-Person Household Results – Figures

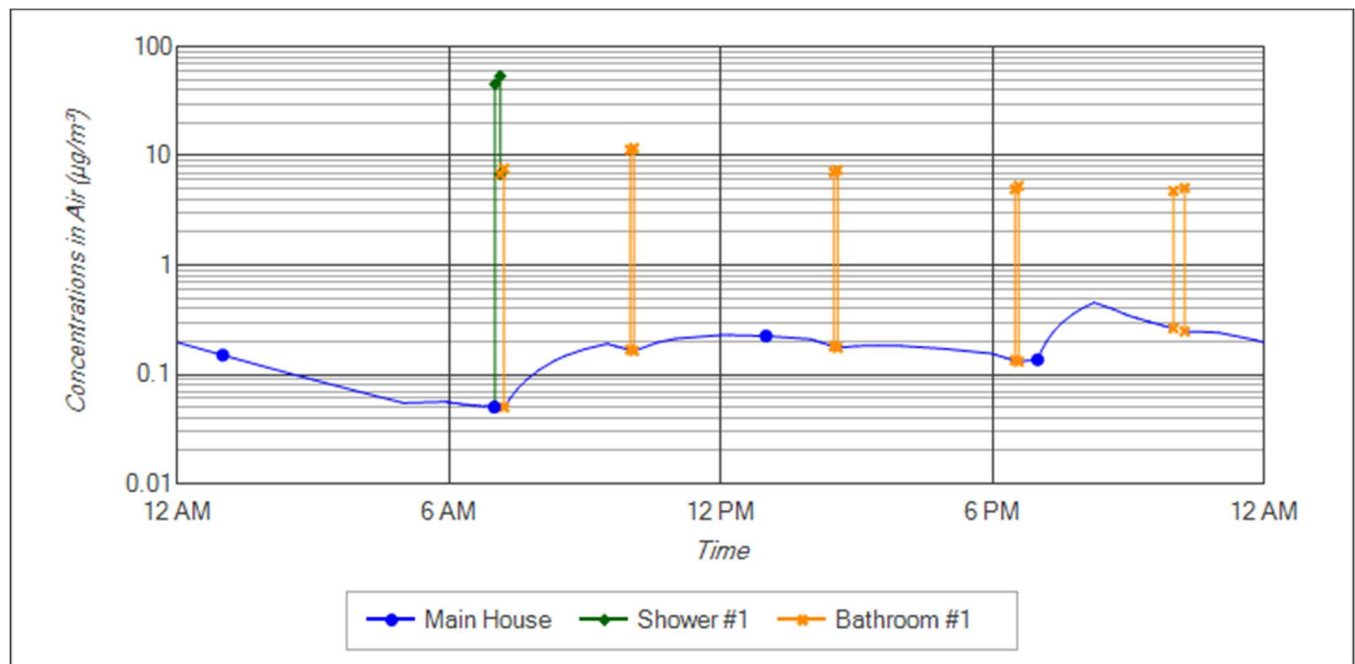
Using a log scale for concentration, Figure 1 shows the calculated chemical air concentrations in the three compartments throughout the day predicted using the SHOWER Model. In general, the chemical air concentrations in the bathroom and shower compartments increase when a person showers or uses other bathroom appliances (e.g. bathroom sink, toilet) and slowly decrease afterwards throughout the day. Chemical air concentrations in the main house compartment rise and fall depending upon other sources in the house and movement of contaminated air from the bathroom compartment through the main house and to the outdoor air.

Figure 1. Calculated air concentrations in the shower, bathroom, and main house compartments for a 6-person household



Using a log scale, Figure 2 shows the contaminant air concentrations the target person (person 6) is exposed to as they move between compartments throughout the day. The contaminant air concentrations shown in Figure 2 are used to calculate the average daily exposure concentration and dermal doses for the target person (person 6) shown in Tables 1 and 2. The contaminant air concentrations are also used to calculate the doses and statistics for the target person shown in Tables 2, 3, and 4.

Figure 2. Calculated exposure air concentrations in different compartments throughout the day for the target person selected in this scenario (person 6)



Model Parameters

The following tables and figures present the parameter values that were used to run this household scenario. These tables are provided as reference and generally are not reported in your public health documents.

In Table 5, the term f value refers to the percentage of a chemical that will be released from a water source (e.g., shower water) to air. Chemical f values are both chemical- and appliance-specific, such that the same chemical will have different f values for different appliances. More information about f values and their derivation can be found in the SHOWER model technical document (ATSDR 2024b).



Table 5. Chemical properties

Parameter	Value
$\mu\text{g}/\text{m}^3$ to ppb conversion factor	$1 \mu\text{g}/\text{m}^3 = 0.313 \text{ ppb}$
Inhalation Parameters	
Shower f value	0.4878
Bathroom sink f value	0.2324
Bathtub f value	0.3639
Toilet f value	0.232
Clothes washer f value	0.697
Dishwasher f value	0.6975
Kitchen sink f value	0.2324
Utility sink f value	0.2324
Henry's law constant	0.2307
Dermal Parameters	
Chemical type	Organic
Molecular weight (MW)	78.12 g/mol
Dermal permeability coefficient (K_p)	0.0149 cm/hr
Fraction absorbed through skin (FA)	1
Fraction absorbed in gastrointestinal tract (ABS_{GI})	1
Permeability coefficient ratio (B)	0.051
Lag time per event (τ_{event})	0.29 hr/event
Time to reach steady state (t^*)	0.69 hr

Abbreviations: cm/hr = centimeters per hour; g/mol = grams chemical per mole; hr = hours; hr/event = hours per event; $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; ppb = parts chemical per billion parts air



Table 6. Mean parameters used to calculate inhalation and dermal doses

Exposure Group	Body Weight (kg)	Daily Breathing Rate (L/min)	Shower and Bathroom Breathing Rate (L/min)	Total Body Surface Area (cm ²)	Hand Surface Area (cm ²)
6 to < 11 years	31.8	8.33	11.00	1.1E+4	510

Abbreviations: kg = kilograms body weight; L/min = liters air breathed per minute; cm² = square centimeters



Table 7. House information

Parameter	Value
Exhaust fan when bathrooms are occupied?	Off
Bathroom door when bathrooms are occupied?	Closed
Appliance Locations	
Shower/bathtub #1 layout	Bathtub with shower
Exhaust fan #1 location	Bathroom #1
Clothes washer location	Main house
Area Volumes	
Total house volume	204.89 m³
Total bathroom #1 volume	9.061 m ³
Shower #1 volume	2.039 m ³
Air Exchange Rates	
Main house/outdoor air exchange rate	0.45 ACH
Bathroom #1/main house air exchange rate	0.45 ACH
Shower/bathroom #1 air exchange rate	0.45 ACH

Abbreviations: ACH = air changes per hour; m³ = cubic meters air



Table 8. Appliance parameters

Appliance	Parameter	Value
Main House		
Kitchen sink	Flow rate	3.347 L/min
Kitchen sink	Average duration per use	0.64 min
Kitchen sink	Maximum kitchen sink uses	15 uses/person/day
Utility sink	Maximum volume use per person	8.544 L/person/day
Dishwasher	Volume per cycle	23.1 L/load
Dishwasher	Average cycle duration	145 min
Clothes washer	Volume per cycle	117 L/load
Clothes washer	Average cycle duration	75 min
Bathroom #1		
Bathroom sink	Flow rate	3.347 L/min
Bathroom sink	Average duration per use	0.64 min
Toilet	Volume per flush	8.7 L/flush
Exhaust fan	Flow rate	1,416 L/min
Shower #1		
Shower	Flow rate	7.6 L/min
Bathtub	Volume	76.47 L

Abbreviations: uses/person/day = appliance uses per person per day; L/min = liters per minute; L = liters water; L/flush = liters water per flush; L/load = liters water per load; L/person/day = liters water used per person per day; min = minute



Table 9. Clothes washer and dishwasher use schedule

Clothes Washer	Dishwasher
7:00 p.m.	9:00 p.m.



Table 10. Timing and duration of showers and bathroom stays

Person	Activity	Location	Activity Start Time	Activity Duration (min)	Bathroom Stay after Activity (min)	Person Helping with Tub Bath
1	Showering	Shower #1	5:56 a.m.	7	5	NA
2	Showering	Shower #1	6:09 a.m.	7	5	NA
3	Showering	Shower #1	6:22 a.m.	7	5	NA
4	Showering	Shower #1	6:35 a.m.	7	5	NA
5	Showering	Shower #1	6:48 a.m.	7	5	NA
6	Tub bath	Shower #1	7:01 a.m.	7	5	NA

Abbreviations: min = minute; NA = not applicable



Table 11. Other activity parameters

Parameter	Value
Time between bathroom stays and next shower/tub bath	1 min
Bathroom visits separate from shower/tub bath	4 visits per person per day
Time when all morning showers/tub baths are complete	7:13 a.m.

Abbreviations: min = minute

Tables 12a-12f show the activity pattern throughout the day for each person in the household. Depending upon the pattern selected, each person starts out in the main house compartment and then moves between compartments at various times during the day.



Table 12a. Human activity pattern throughout the day for person 1 in this scenario

Person	Start Time	Location	Appliance Start Times
1	12:00 a.m.	Main house	—
1	5:56 a.m.	Shower #1	Shower #1: 5:56 a.m.
1	6:03 a.m.	Bathroom #1	Toilet #1: 6:03 a.m.; Bathroom sink #1: 6:04 a.m.
1	6:08 a.m.	Main house	—

Person	Start Time	Location	Appliance Start Times
1	9:30 a.m.	Bathroom #1	Toilet #1: 9:30 a.m.; Bathroom sink #1: 9:31 a.m.
1	9:35 a.m.	Main house	—
1	2:00 p.m.	Bathroom #1	Toilet #1: 2:00 p.m.; Bathroom sink #1: 2:01 p.m.
1	2:05 p.m.	Main house	—
1	6:00 p.m.	Bathroom #1	Toilet #1: 6:00 p.m.; Bathroom sink #1: 6:01 p.m.
1	6:05 p.m.	Main house	—
1	8:40 p.m.	Bathroom #1	Toilet #1: 8:40 p.m.; Bathroom sink #1: 8:41 p.m.
1	8:55 p.m.	Main house	—



Table 12b. Human activity pattern throughout the day for person 2 in this scenario

Person	Start Time	Location	Appliance Start Times
2	12:00 a.m.	Main house	—
2	6:09 a.m.	Shower #1	Shower #1: 6:09 a.m.
2	6:16 a.m.	Bathroom #1	Toilet #1: 6:16 a.m.; Bathroom sink #1: 6:17 a.m.
2	6:21 a.m.	Main house	—
2	9:36 a.m.	Bathroom #1	Toilet #1: 9:36 a.m.; Bathroom sink #1: 9:37 a.m.
2	9:41 a.m.	Main house	—
2	2:06 p.m.	Bathroom #1	Toilet #1: 2:06 p.m.; Bathroom sink #1: 2:07 p.m.
2	2:11 p.m.	Main house	—
2	6:06 p.m.	Bathroom #1	Toilet #1: 6:06 p.m.; Bathroom sink #1: 6:07 p.m.
2	6:11 p.m.	Main house	—
2	8:56 p.m.	Bathroom #1	Toilet #1: 8:56 p.m.; Bathroom sink #1: 8:57 p.m.
2	9:11 p.m.	Main house	—



Table 12c. Human activity pattern throughout the day for person 3 in this scenario

Person	Start Time	Location	Appliance Start Times
3	12:00 a.m.	Main house	—
3	6:22 a.m.	Shower #1	Shower #1: 6:22 a.m.
3	6:29 a.m.	Bathroom #1	Toilet #1: 6:29 a.m.; Bathroom sink #1: 6:30 a.m.
3	6:34 a.m.	Main house	—
3	9:42 a.m.	Bathroom #1	Toilet #1: 9:42 a.m.; Bathroom sink #1: 9:43 a.m.
3	9:47 a.m.	Main house	—
3	2:12 p.m.	Bathroom #1	Toilet #1: 2:12 p.m.; Bathroom sink #1: 2:13 p.m.
3	2:17 p.m.	Main house	—
3	6:12 p.m.	Bathroom #1	Toilet #1: 6:12 p.m.; Bathroom sink #1: 6:13 p.m.
3	6:17 p.m.	Main house	—
3	9:12 p.m.	Bathroom #1	Toilet #1: 9:12 p.m.; Bathroom sink #1: 9:13 p.m.
3	9:27 p.m.	Main house	—



Table 12d. Human activity pattern throughout the day for person 4 in this scenario

Person	Start Time	Location	Appliance Start Times
4	12:00 a.m.	Main house	—
4	6:35 a.m.	Shower #1	Shower #1: 6:35 a.m.
4	6:42 a.m.	Bathroom #1	Toilet #1: 6:42 a.m.; Bathroom sink #1: 6:43 a.m.
4	6:47 a.m.	Main house	—
4	9:48 a.m.	Bathroom #1	Toilet #1: 9:48 a.m.; Bathroom sink #1: 9:49 a.m.
4	9:53 a.m.	Main house	—
4	2:18 p.m.	Bathroom #1	Toilet #1: 2:18 p.m.; Bathroom sink #1: 2:19 p.m.
4	2:23 p.m.	Main house	—
4	6:18 p.m.	Bathroom #1	Toilet #1: 6:18 p.m.; Bathroom sink #1: 6:19 p.m.
4	6:23 p.m.	Main house	—
4	9:28 p.m.	Bathroom #1	Toilet #1: 9:28 p.m.; Bathroom sink #1: 9:29 p.m.
4	9:43 p.m.	Main house	—



Table 12e. Human activity pattern throughout the day for person 5 in this scenario

Person	Start Time	Location	Appliance Start Times
5	12:00 a.m.	Main house	—
5	6:48 a.m.	Shower #1	Shower #1: 6:48 a.m.
5	6:55 a.m.	Bathroom #1	Toilet #1: 6:55 a.m.; Bathroom sink #1: 6:56 a.m.
5	7:00 a.m.	Main house	—
5	9:54 a.m.	Bathroom #1	Toilet #1: 9:54 a.m.; Bathroom sink #1: 9:55 a.m.
5	9:59 a.m.	Main house	—
5	2:24 p.m.	Bathroom #1	Toilet #1: 2:24 p.m.; Bathroom sink #1: 2:25 p.m.
5	2:29 p.m.	Main house	—
5	6:24 p.m.	Bathroom #1	Toilet #1: 6:24 p.m.; Bathroom sink #1: 6:25 p.m.
5	6:29 p.m.	Main house	—
5	9:44 p.m.	Bathroom #1	Toilet #1: 9:44 p.m.; Bathroom sink #1: 9:45 p.m.
5	9:59 p.m.	Main house	—



Table 12f. Human activity pattern throughout the day for person 6 in this scenario

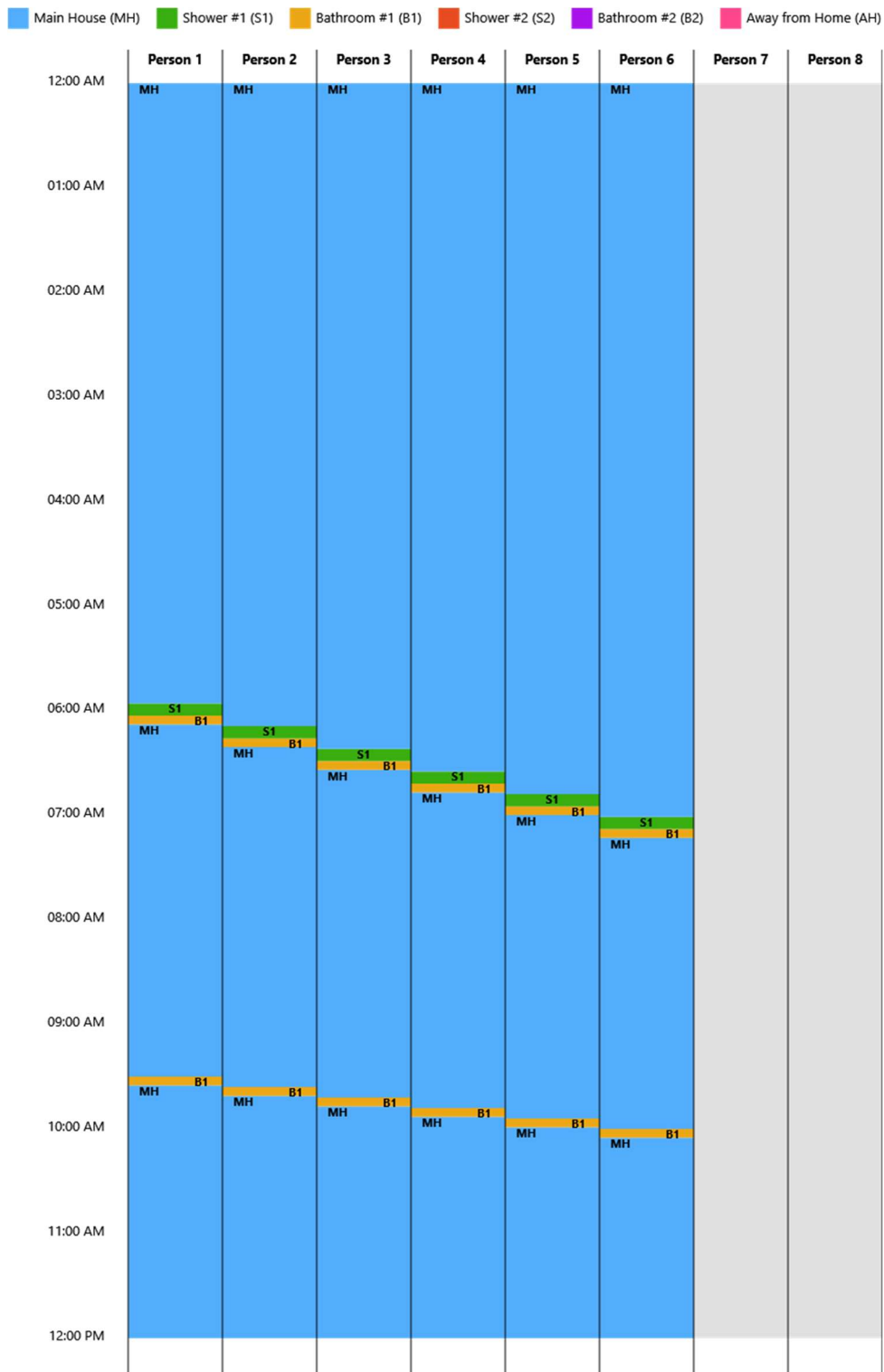
Person	Start Time	Location	Appliance Start Times
6	12:00 a.m.	Main house	—
6	7:01 a.m.	Shower #1	Bath tub #1: 7:01 a.m.
6	7:08 a.m.	Bathroom #1	Toilet #1: 7:08 a.m.; Bathroom sink #1: 7:09 a.m.
6	7:13 a.m.	Main house	—
6	10:00 a.m.	Bathroom #1	Toilet #1: 10:00 a.m.; Bathroom sink #1: 10:01 a.m.
6	10:05 a.m.	Main house	—
6	2:30 p.m.	Bathroom #1	Toilet #1: 2:30 p.m.; Bathroom sink #1: 2:31 p.m.
6	2:35 p.m.	Main house	—
6	6:30 p.m.	Bathroom #1	Toilet #1: 6:30 p.m.; Bathroom sink #1: 6:31 p.m.

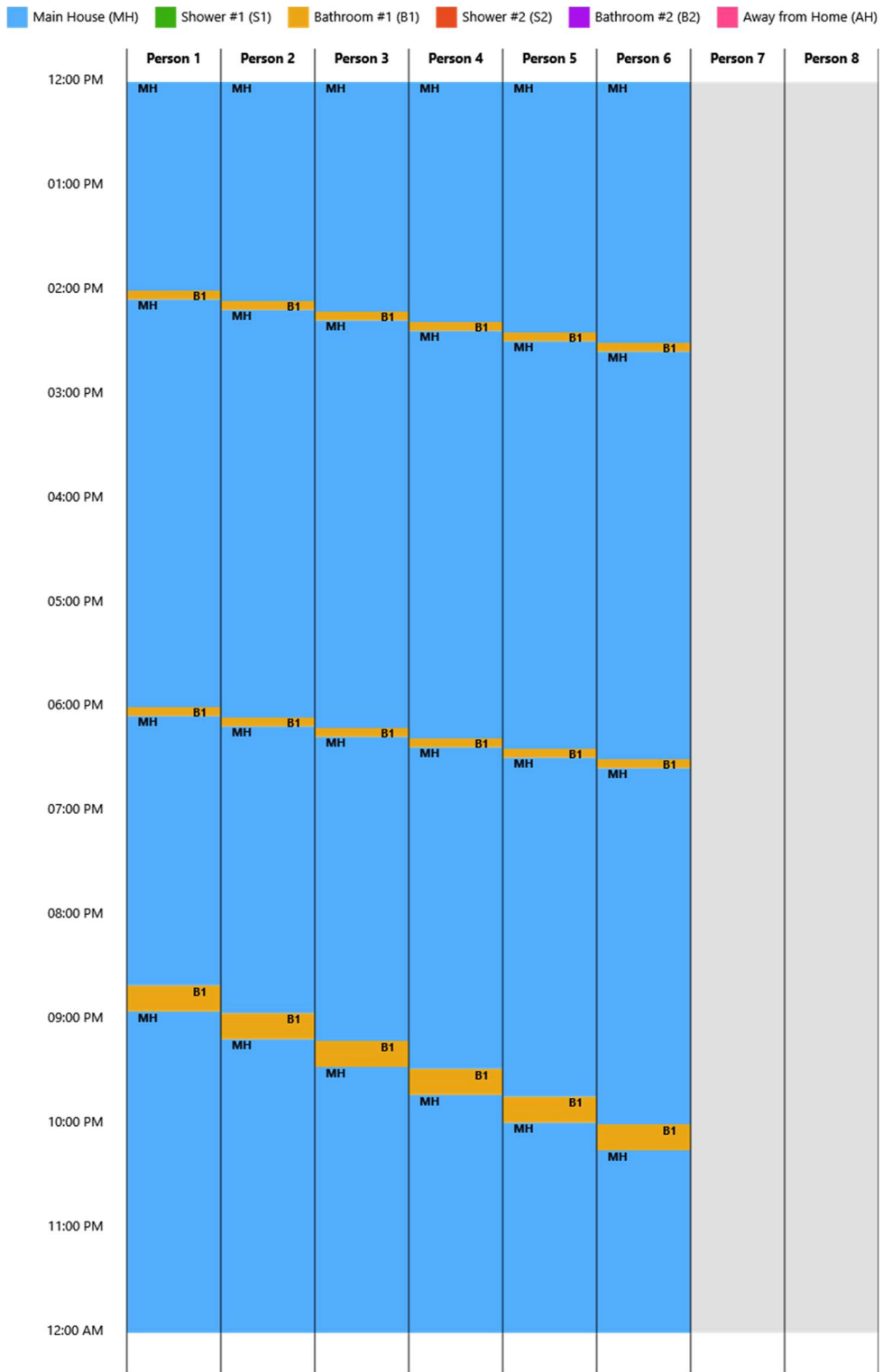
Person	Start Time	Location	Appliance Start Times
6	6:35 p.m.	Main house	—
6	10:00 p.m.	Bathroom #1	Toilet #1: 10:00 p.m.; Bathroom sink #1: 10:01 p.m.
6	10:15 p.m.	Main house	—

Figure 3 uses color blocks and text symbols to show the compartment location of each person in the house throughout the day. The top edge of each block represents the time when the person enters the compartment and the bottom edge represents the time when the person exits the compartment. Text symbols (e.g., S1, B1, MH) also denote start times in each location. In addition, tables 12a-12f show the precise time a person enters a compartment.

Depending upon the custom scenario, each person starts in the main house and moves through the bathroom or shower several times during the day, ending the day in the main house. The target person in this scenario is person 6 because they have the highest exposure.

Figure 3. Human Activity Patterns.





References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024a. Guidance for Evaluating Inhalation and Dermal Exposure Using the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024b. Technical Document for the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.



ATSDR SHOWER Model Report

Custom 6-Person Household

Six morning showers

Contents

Site and Model Input Information	2
Scenario Description	3
6-Person Household Results - Tables	3
6-Person Household Results – Figures	6
Model Parameters	8
References	18

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	AMSLER HP RESIDENTIAL
Address:	—
Application:	Version 4.0.1
CASRN:	79-01-6
Contaminant:	Trichloroethylene
Synonym:	1,1,2-trichloroethylene
	TCE
	Trichloroethene
Model Input Information	
Chemical name:	Trichloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	24.4 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	6
Household scenario:	Modified custom scenario
Basis for modified scenario:	Six morning showers
Number of bathrooms in house:	1
Target person:	6
Most highly exposed person:	6
Target person main activity:	7-minute morning tub bath

Scenario Description

This report is for a custom 6-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 6). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

6–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 6-person household, including the most highly exposed person (person 6). The air concentration estimated by the model is independent of who is showering and thus applies to both children and adults.



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Person	Target Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 5:56 a.m.	8.9
2	—	Showering in Shower #1 at 6:09 a.m.	11
3	—	Showering in Shower #1 at 6:22 a.m.	13
4	—	Showering in Shower #1 at 6:35 a.m.	15
5	—	Showering in Shower #1 at 6:48 a.m.	16
6	X	Tub bath in Shower #1 at 7:01 a.m.	17

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 6). This contact occurs from showering or bathing and from hand washing during the day. According to EPA guidance, dermal doses for this halogenated compound may be underestimated (see the SHOWER model technical document or EPA's RAGS, Part E for further details). Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation	Dermal
6 to < 11 years	8.1	0.076

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day

Peak and Percentage Exposure By Location (Table 3 and 4)

This SHOWER model scenario consists of three compartments: a shower stall, the bathroom, and the main house. The next two tables show the amount of time the target person spends bathing (shower or bath) and the amount of time they spend in the bathroom afterwards. It also provides information on the amount of time the target person spends in the main house throughout the rest of the day. When the target person visits the bathroom during other parts of the day, those bathroom times are included in the main house exposure time. Table 3 shows the average exposure concentration that the target person experiences in each location, and Table 4 shows the percent of exposure that the target person experiences in each location.

The exposure from bathing (shower or bath) and being in the bathroom afterwards can be much higher (but for shorter periods) than the exposure from being in the main house. Knowledge of this brief exposure to high levels in the shower and bathroom compartments might be useful when evaluating whether harmful effects might be possible from acute exposure to high concentrations. This acute exposure to high levels might be particularly important for irritant chemicals, such as formaldehyde, 2-butanone, and acetone. Some irritants, however, cannot be run using the model because parameters are lacking. Health assessors should evaluate this acute exposure duration if the acute EMEG is exceeded. More information about evaluating acute exposure can be found in ATSDR's Guidance for Evaluating Inhalation and Dermal Exposure Using the SHOWER Model (ATSDR 2024a). **Health assessors should consult with the Associate Director of Science (ADS) when evaluating brief exposure to high levels.**



Table 3. Exposure time and average exposure concentration by location for the target person

Location	Exposure Time (min)	Average Exposure Concentration ($\mu\text{g}/\text{m}^3$)
Tub bath	7	1,650
Bathroom after tub bath	5	223
Main house with additional bathroom visits	1,428	8.6
Away from house	0	0
Average daily exposure	1,440	17
Main house (all day)	1,440	4.8

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; min = minute



Table 4. Exposure time and percent of total exposure by location for the target person

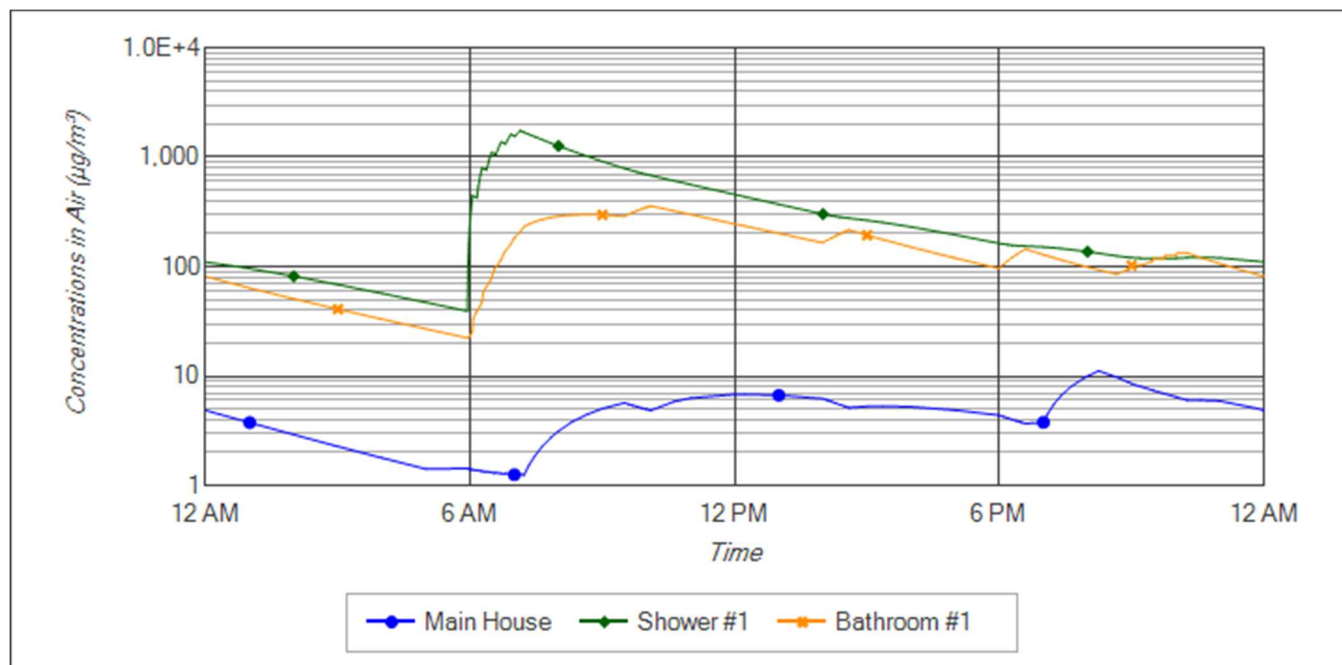
Location	Exposure Time (min)	Percent Exposure (%)
Tub bath	7	46
Bathroom after tub bath	5	4.5
Main house with additional bathroom visits	1,428	49
Away from house	0	0
Average daily exposure	1,440	100
Main house (all day)	1,440	100

Abbreviations: min = minute; % = percent

6–Person Household Results – Figures

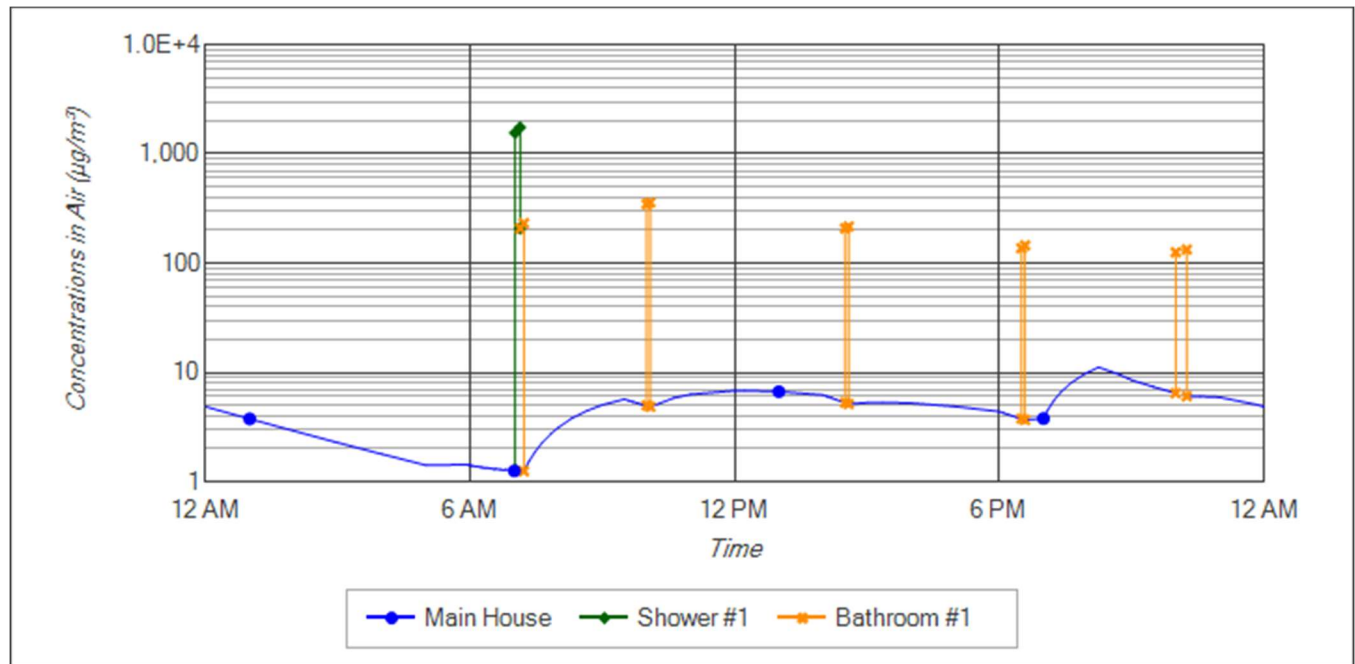
Using a log scale for concentration, Figure 1 shows the calculated chemical air concentrations in the three compartments throughout the day predicted using the SHOWER Model. In general, the chemical air concentrations in the bathroom and shower compartments increase when a person showers or uses other bathroom appliances (e.g. bathroom sink, toilet) and slowly decrease afterwards throughout the day. Chemical air concentrations in the main house compartment rise and fall depending upon other sources in the house and movement of contaminated air from the bathroom compartment through the main house and to the outdoor air.

Figure 1. Calculated air concentrations in the shower, bathroom, and main house compartments for a 6-person household



Using a log scale, Figure 2 shows the contaminant air concentrations the target person (person 6) is exposed to as they move between compartments throughout the day. The contaminant air concentrations shown in Figure 2 are used to calculate the average daily exposure concentration and dermal doses for the target person (person 6) shown in Tables 1 and 2. The contaminant air concentrations are also used to calculate the doses and statistics for the target person shown in Tables 2, 3, and 4.

Figure 2. Calculated exposure air concentrations in different compartments throughout the day for the target person selected in this scenario (person 6)



Model Parameters

The following tables and figures present the parameter values that were used to run this household scenario. These tables are provided as reference and generally are not reported in your public health documents.

In Table 5, the term f value refers to the percentage of a chemical that will be released from a water source (e.g., shower water) to air. Chemical f values are both chemical- and appliance-specific, such that the same chemical will have different f values for different appliances. More information about f values and their derivation can be found in the SHOWER model technical document (ATSDR 2024b).



Table 5. Chemical properties

Parameter	Value
$\mu\text{g}/\text{m}^3$ to ppb conversion factor	$1 \mu\text{g}/\text{m}^3 = 0.186 \text{ ppb}$
Inhalation Parameters	
Shower f value	0.67
Bathroom sink f value	0.2229
Bathtub f value	0.3491
Toilet f value	0.2226
Clothes washer f value	0.6686
Dishwasher f value	0.6689
Kitchen sink f value	0.2229
Utility sink f value	0.2229
Henry's law constant	0.4121
Dermal Parameters	
Chemical type	Organic
Molecular weight (MW)	131.3889 g/mol
Dermal permeability coefficient (K_p)	0.0116 cm/hr
Fraction absorbed through skin (FA)	1
Fraction absorbed in gastrointestinal tract (ABS_{GI})	1
Permeability coefficient ratio (B)	0.051
Lag time per event (τ_{event})	0.57 hr/event
Time to reach steady state (t^*)	1.4 hr

Abbreviations: cm/hr = centimeters per hour; g/mol = grams chemical per mole; hr = hours; hr/event = hours per event; $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; ppb = parts chemical per billion parts air



Table 6. Mean parameters used to calculate inhalation and dermal doses

Exposure Group	Body Weight (kg)	Daily Breathing Rate (L/min)	Shower and Bathroom Breathing Rate (L/min)	Total Body Surface Area (cm ²)	Hand Surface Area (cm ²)
6 to < 11 years	31.8	8.33	11.00	1.1E+4	510

Abbreviations: kg = kilograms body weight; L/min = liters air breathed per minute; cm² = square centimeters



Table 7. House information

Parameter	Value
Exhaust fan when bathrooms are occupied?	Off
Bathroom door when bathrooms are occupied?	Closed
Appliance Locations	
Shower/bathtub #1 layout	Bathtub with shower
Exhaust fan #1 location	Bathroom #1
Clothes washer location	Main house
Area Volumes	
Total house volume	204.89 m³
Total bathroom #1 volume	9.061 m ³
Shower #1 volume	2.039 m ³
Air Exchange Rates	
Main house/outdoor air exchange rate	0.45 ACH
Bathroom #1/main house air exchange rate	0.45 ACH
Shower/bathroom #1 air exchange rate	0.45 ACH

Abbreviations: ACH = air changes per hour; m³ = cubic meters air

**Table 8. Appliance parameters**

Appliance	Parameter	Value
Main House		
Kitchen sink	Flow rate	3.347 L/min
Kitchen sink	Average duration per use	0.64 min
Kitchen sink	Maximum kitchen sink uses	15 uses/person/day
Utility sink	Maximum volume use per person	8.544 L/person/day
Dishwasher	Volume per cycle	23.1 L/load
Dishwasher	Average cycle duration	145 min
Clothes washer	Volume per cycle	117 L/load
Clothes washer	Average cycle duration	75 min
Bathroom #1		
Bathroom sink	Flow rate	3.347 L/min
Bathroom sink	Average duration per use	0.64 min
Toilet	Volume per flush	8.7 L/flush
Exhaust fan	Flow rate	1,416 L/min
Shower #1		
Shower	Flow rate	7.6 L/min
Bathtub	Volume	76.47 L

Abbreviations: uses/person/day = appliance uses per person per day; L/min = liters per minute; L = liters water; L/flush = liters water per flush; L/load = liters water per load; L/person/day = liters water used per person per day; min = minute

**Table 9. Clothes washer and dishwasher use schedule**

Clothes Washer	Dishwasher
7:00 p.m.	9:00 p.m.



Table 10. Timing and duration of showers and bathroom stays

Person	Activity	Location	Activity Start Time	Activity Duration (min)	Bathroom Stay after Activity (min)	Person Helping with Tub Bath
1	Showering	Shower #1	5:56 a.m.	7	5	NA
2	Showering	Shower #1	6:09 a.m.	7	5	NA
3	Showering	Shower #1	6:22 a.m.	7	5	NA
4	Showering	Shower #1	6:35 a.m.	7	5	NA
5	Showering	Shower #1	6:48 a.m.	7	5	NA
6	Tub bath	Shower #1	7:01 a.m.	7	5	NA

Abbreviations: min = minute; NA = not applicable



Table 11. Other activity parameters

Parameter	Value
Time between bathroom stays and next shower/tub bath	1 min
Bathroom visits separate from shower/tub bath	4 visits per person per day
Time when all morning showers/tub baths are complete	7:13 a.m.

Abbreviations: min = minute

Tables 12a-12f show the activity pattern throughout the day for each person in the household. Depending upon the pattern selected, each person starts out in the main house compartment and then moves between compartments at various times during the day.



Table 12a. Human activity pattern throughout the day for person 1 in this scenario

Person	Start Time	Location	Appliance Start Times
1	12:00 a.m.	Main house	—
1	5:56 a.m.	Shower #1	Shower #1: 5:56 a.m.
1	6:03 a.m.	Bathroom #1	Toilet #1: 6:03 a.m.; Bathroom sink #1: 6:04 a.m.
1	6:08 a.m.	Main house	—

Person	Start Time	Location	Appliance Start Times
1	9:30 a.m.	Bathroom #1	Toilet #1: 9:30 a.m.; Bathroom sink #1: 9:31 a.m.
1	9:35 a.m.	Main house	—
1	2:00 p.m.	Bathroom #1	Toilet #1: 2:00 p.m.; Bathroom sink #1: 2:01 p.m.
1	2:05 p.m.	Main house	—
1	6:00 p.m.	Bathroom #1	Toilet #1: 6:00 p.m.; Bathroom sink #1: 6:01 p.m.
1	6:05 p.m.	Main house	—
1	8:40 p.m.	Bathroom #1	Toilet #1: 8:40 p.m.; Bathroom sink #1: 8:41 p.m.
1	8:55 p.m.	Main house	—



Table 12b. Human activity pattern throughout the day for person 2 in this scenario

Person	Start Time	Location	Appliance Start Times
2	12:00 a.m.	Main house	—
2	6:09 a.m.	Shower #1	Shower #1: 6:09 a.m.
2	6:16 a.m.	Bathroom #1	Toilet #1: 6:16 a.m.; Bathroom sink #1: 6:17 a.m.
2	6:21 a.m.	Main house	—
2	9:36 a.m.	Bathroom #1	Toilet #1: 9:36 a.m.; Bathroom sink #1: 9:37 a.m.
2	9:41 a.m.	Main house	—
2	2:06 p.m.	Bathroom #1	Toilet #1: 2:06 p.m.; Bathroom sink #1: 2:07 p.m.
2	2:11 p.m.	Main house	—
2	6:06 p.m.	Bathroom #1	Toilet #1: 6:06 p.m.; Bathroom sink #1: 6:07 p.m.
2	6:11 p.m.	Main house	—
2	8:56 p.m.	Bathroom #1	Toilet #1: 8:56 p.m.; Bathroom sink #1: 8:57 p.m.
2	9:11 p.m.	Main house	—



Table 12c. Human activity pattern throughout the day for person 3 in this scenario

Person	Start Time	Location	Appliance Start Times
3	12:00 a.m.	Main house	—
3	6:22 a.m.	Shower #1	Shower #1: 6:22 a.m.
3	6:29 a.m.	Bathroom #1	Toilet #1: 6:29 a.m.; Bathroom sink #1: 6:30 a.m.
3	6:34 a.m.	Main house	—
3	9:42 a.m.	Bathroom #1	Toilet #1: 9:42 a.m.; Bathroom sink #1: 9:43 a.m.
3	9:47 a.m.	Main house	—
3	2:12 p.m.	Bathroom #1	Toilet #1: 2:12 p.m.; Bathroom sink #1: 2:13 p.m.
3	2:17 p.m.	Main house	—
3	6:12 p.m.	Bathroom #1	Toilet #1: 6:12 p.m.; Bathroom sink #1: 6:13 p.m.
3	6:17 p.m.	Main house	—
3	9:12 p.m.	Bathroom #1	Toilet #1: 9:12 p.m.; Bathroom sink #1: 9:13 p.m.
3	9:27 p.m.	Main house	—



Table 12d. Human activity pattern throughout the day for person 4 in this scenario

Person	Start Time	Location	Appliance Start Times
4	12:00 a.m.	Main house	—
4	6:35 a.m.	Shower #1	Shower #1: 6:35 a.m.
4	6:42 a.m.	Bathroom #1	Toilet #1: 6:42 a.m.; Bathroom sink #1: 6:43 a.m.
4	6:47 a.m.	Main house	—
4	9:48 a.m.	Bathroom #1	Toilet #1: 9:48 a.m.; Bathroom sink #1: 9:49 a.m.
4	9:53 a.m.	Main house	—
4	2:18 p.m.	Bathroom #1	Toilet #1: 2:18 p.m.; Bathroom sink #1: 2:19 p.m.
4	2:23 p.m.	Main house	—
4	6:18 p.m.	Bathroom #1	Toilet #1: 6:18 p.m.; Bathroom sink #1: 6:19 p.m.
4	6:23 p.m.	Main house	—
4	9:28 p.m.	Bathroom #1	Toilet #1: 9:28 p.m.; Bathroom sink #1: 9:29 p.m.
4	9:43 p.m.	Main house	—



Table 12e. Human activity pattern throughout the day for person 5 in this scenario

Person	Start Time	Location	Appliance Start Times
5	12:00 a.m.	Main house	—
5	6:48 a.m.	Shower #1	Shower #1: 6:48 a.m.
5	6:55 a.m.	Bathroom #1	Toilet #1: 6:55 a.m.; Bathroom sink #1: 6:56 a.m.
5	7:00 a.m.	Main house	—
5	9:54 a.m.	Bathroom #1	Toilet #1: 9:54 a.m.; Bathroom sink #1: 9:55 a.m.
5	9:59 a.m.	Main house	—
5	2:24 p.m.	Bathroom #1	Toilet #1: 2:24 p.m.; Bathroom sink #1: 2:25 p.m.
5	2:29 p.m.	Main house	—
5	6:24 p.m.	Bathroom #1	Toilet #1: 6:24 p.m.; Bathroom sink #1: 6:25 p.m.
5	6:29 p.m.	Main house	—
5	9:44 p.m.	Bathroom #1	Toilet #1: 9:44 p.m.; Bathroom sink #1: 9:45 p.m.
5	9:59 p.m.	Main house	—



Table 12f. Human activity pattern throughout the day for person 6 in this scenario

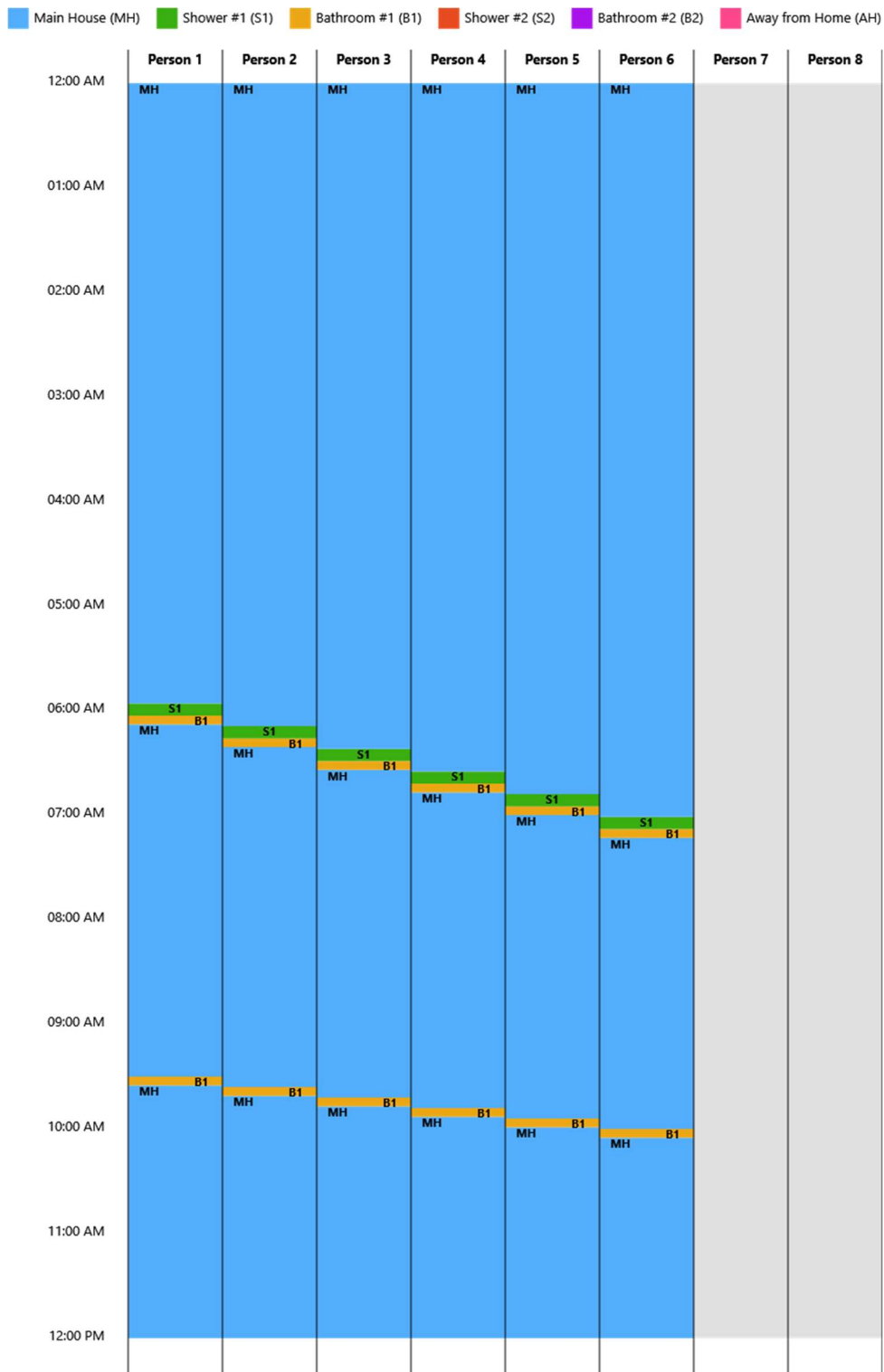
Person	Start Time	Location	Appliance Start Times
6	12:00 a.m.	Main house	—
6	7:01 a.m.	Shower #1	Bath tub #1: 7:01 a.m.
6	7:08 a.m.	Bathroom #1	Toilet #1: 7:08 a.m.; Bathroom sink #1: 7:09 a.m.
6	7:13 a.m.	Main house	—
6	10:00 a.m.	Bathroom #1	Toilet #1: 10:00 a.m.; Bathroom sink #1: 10:01 a.m.
6	10:05 a.m.	Main house	—
6	2:30 p.m.	Bathroom #1	Toilet #1: 2:30 p.m.; Bathroom sink #1: 2:31 p.m.
6	2:35 p.m.	Main house	—
6	6:30 p.m.	Bathroom #1	Toilet #1: 6:30 p.m.; Bathroom sink #1: 6:31 p.m.

Person	Start Time	Location	Appliance Start Times
6	6:35 p.m.	Main house	—
6	10:00 p.m.	Bathroom #1	Toilet #1: 10:00 p.m.; Bathroom sink #1: 10:01 p.m.
6	10:15 p.m.	Main house	—

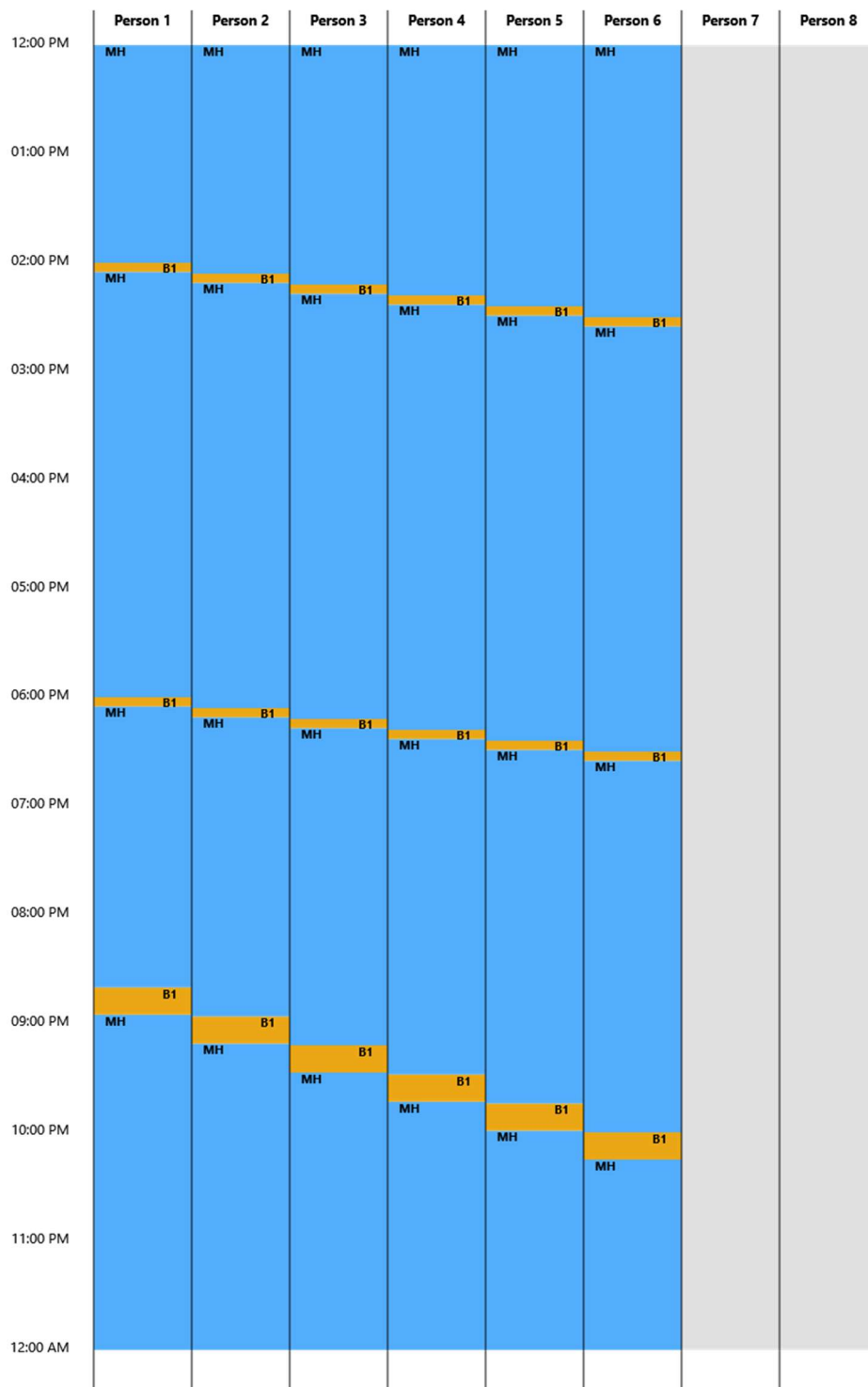
Figure 3 uses color blocks and text symbols to show the compartment location of each person in the house throughout the day. The top edge of each block represents the time when the person enters the compartment and the bottom edge represents the time when the person exits the compartment. Text symbols (e.g., S1, B1, MH) also denote start times in each location. In addition, tables 12a-12f show the precise time a person enters a compartment.

Depending upon the custom scenario, each person starts in the main house and moves through the bathroom or shower several times during the day, ending the day in the main house. The target person in this scenario is person 6 because they have the highest exposure.

Figure 3. Human Activity Patterns.



■ Main House (MH)
 ■ Shower #1 (S1)
 ■ Bathroom #1 (B1)
 ■ Shower #2 (S2)
 ■ Bathroom #2 (B2)
 ■ Away from Home (AH)



References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024a. Guidance for Evaluating Inhalation and Dermal Exposure Using the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024b. Technical Document for the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.



ATSDR SHOWER Model Report

Custom 6-Person Household

Six morning showers

Contents

Site and Model Input Information	2
Scenario Description	3
6-Person Household Results - Tables	3
6-Person Household Results – Figures	6
Model Parameters	8
References	18

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	AMSLER HP 20 MIN BATH
Address:	—
Application:	Version 4.0.1
CASRN:	71-43-2
Contaminant:	Benzene
Synonym:	—
Model Input Information	
Chemical name:	Benzene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	1 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	6
Household scenario:	Modified custom scenario
Basis for modified scenario:	Six morning showers
Number of bathrooms in house:	1
Target person:	6
Most highly exposed person:	6
Target person main activity:	20-minute morning tub bath

Scenario Description

This report is for a custom 6-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 6). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

6–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 6-person household, including the most highly exposed person (person 6). The air concentration estimated by the model is independent of who is showering and thus applies to both children and adults.



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Person	Target Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 5:43 a.m.	0.31
2	—	Showering in Shower #1 at 5:56 a.m.	0.38
3	—	Showering in Shower #1 at 6:09 a.m.	0.43
4	—	Showering in Shower #1 at 6:22 a.m.	0.48
5	—	Showering in Shower #1 at 6:35 a.m.	0.53
6	X	Tub bath in Shower #1 at 6:48 a.m.	1.0

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 6). This contact occurs from showering or bathing and from hand washing during the day. Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation	Dermal
6 to < 11 years	0.48	0.0044

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day

Peak and Percentage Exposure By Location (Table 3 and 4)

This SHOWER model scenario consists of three compartments: a shower stall, the bathroom, and the main house. The next two tables show the amount of time the target person spends bathing (shower or bath) and the amount of time they spend in the bathroom afterwards. It also provides information on the amount of time the target person spends in the main house throughout the rest of the day. When the target person visits the bathroom during other parts of the day, those bathroom times are included in the main house exposure time. Table 3 shows the average exposure concentration that the target person experiences in each location, and Table 4 shows the percent of exposure that the target person experiences in each location.

The exposure from bathing (shower or bath) and being in the bathroom afterwards can be much higher (but for shorter periods) than the exposure from being in the main house. Knowledge of this brief exposure to high levels in the shower and bathroom compartments might be useful when evaluating whether harmful effects might be possible from acute exposure to high concentrations. This acute exposure to high levels might be particularly important for irritant chemicals, such as formaldehyde, 2-butanone, and acetone. Some irritants, however, cannot be run using the model because parameters are lacking. Health assessors should evaluate this acute exposure duration if the acute EMEG is exceeded. More information about evaluating acute exposure can be found in ATSDR's Guidance for Evaluating Inhalation and Dermal Exposure Using the SHOWER Model (ATSDR 2024a). **Health assessors should consult with the Associate Director of Science (ADS) when evaluating brief exposure to high levels.**



Table 3. Exposure time and average exposure concentration by location for the target person

Location	Exposure Time (min)	Average Exposure Concentration ($\mu\text{g}/\text{m}^3$)
Tub bath	20	48
Bathroom after tub bath	5	8.5
Main house with additional bathroom visits	1,415	0.31
Away from house	0	0
Average daily exposure	1,440	1.0
Main house (all day)	1,440	0.18

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; min = minute



Table 4. Exposure time and percent of total exposure by location for the target person

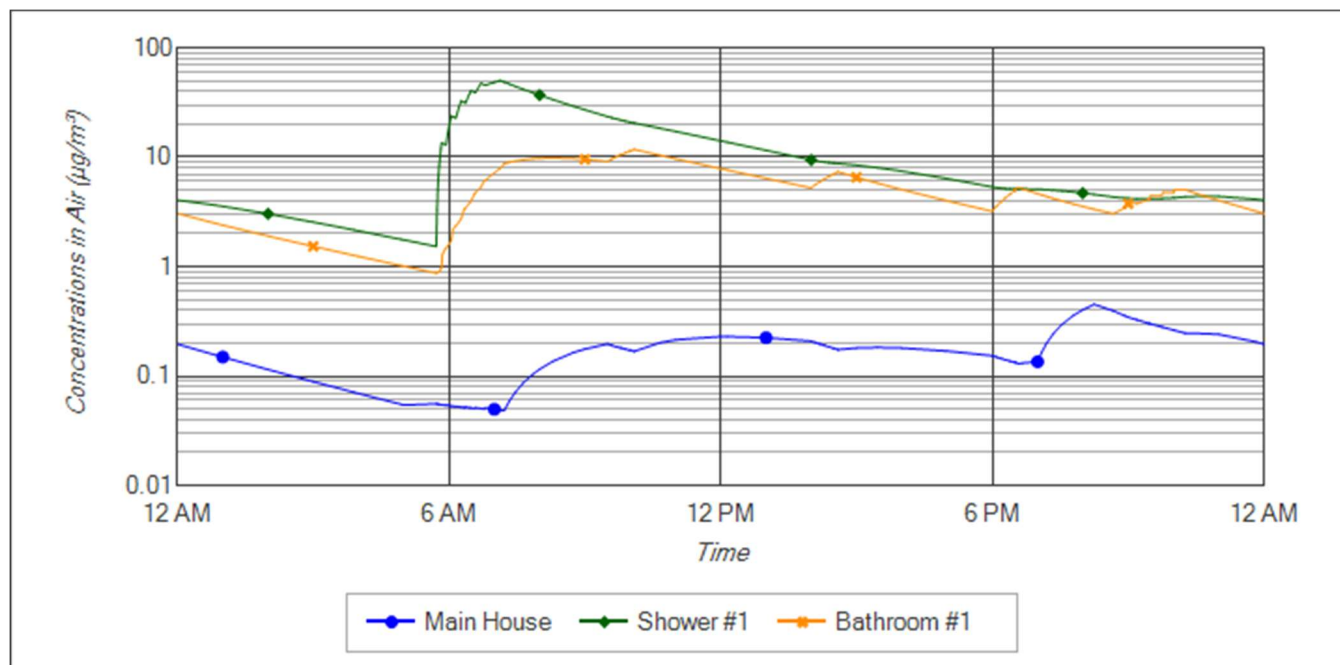
Location	Exposure Time (min)	Percent Exposure (%)
Tub bath	20	66
Bathroom after tub bath	5	2.9
Main house with additional bathroom visits	1,415	31
Away from house	0	0
Average daily exposure	1,440	100
Main house (all day)	1,440	100

Abbreviations: min = minute; % = percent

6-Person Household Results – Figures

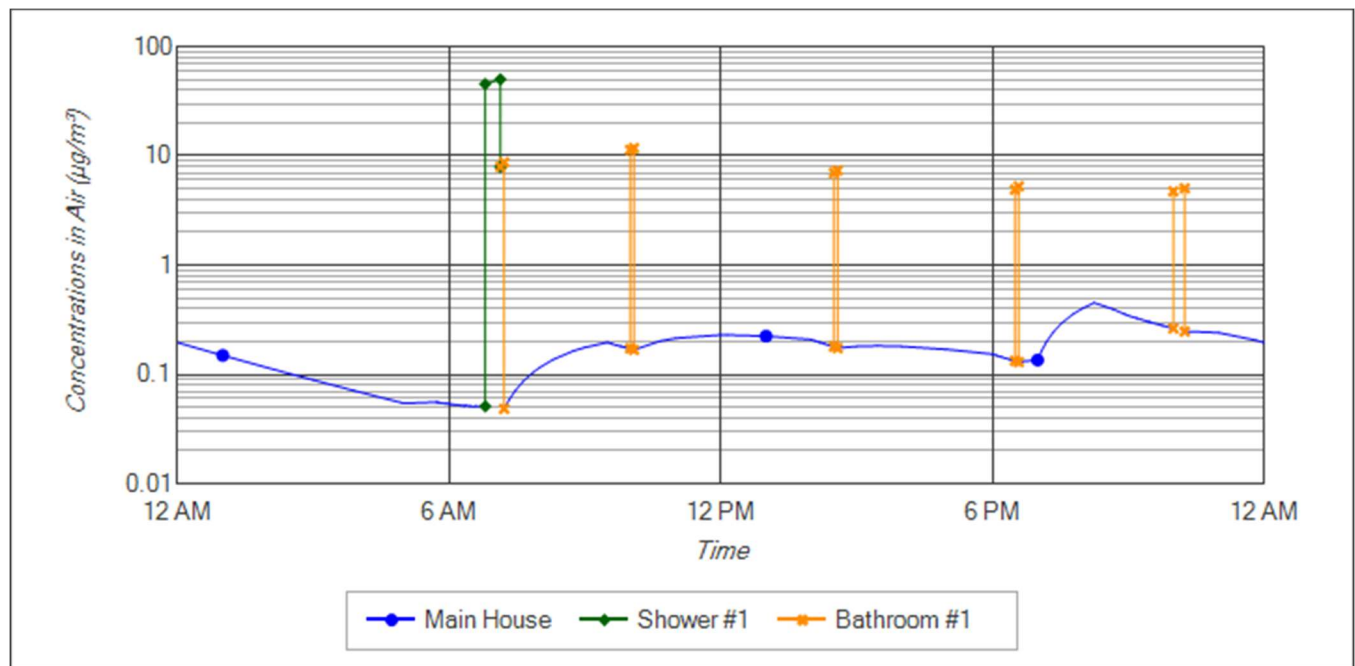
Using a log scale for concentration, Figure 1 shows the calculated chemical air concentrations in the three compartments throughout the day predicted using the SHOWER Model. In general, the chemical air concentrations in the bathroom and shower compartments increase when a person showers or uses other bathroom appliances (e.g. bathroom sink, toilet) and slowly decrease afterwards throughout the day. Chemical air concentrations in the main house compartment rise and fall depending upon other sources in the house and movement of contaminated air from the bathroom compartment through the main house and to the outdoor air.

Figure 1. Calculated air concentrations in the shower, bathroom, and main house compartments for a 6-person household



Using a log scale, Figure 2 shows the contaminant air concentrations the target person (person 6) is exposed to as they move between compartments throughout the day. The contaminant air concentrations shown in Figure 2 are used to calculate the average daily exposure concentration and dermal doses for the target person (person 6) shown in Tables 1 and 2. The contaminant air concentrations are also used to calculate the doses and statistics for the target person shown in Tables 2, 3, and 4.

Figure 2. Calculated exposure air concentrations in different compartments throughout the day for the target person selected in this scenario (person 6)



Model Parameters

The following tables and figures present the parameter values that were used to run this household scenario. These tables are provided as reference and generally are not reported in your public health documents.

In Table 5, the term *f* value refers to the percentage of a chemical that will be released from a water source (e.g., shower water) to air. Chemical *f* values are both chemical- and appliance-specific, such that the same chemical will have different *f* values for different appliances. More information about *f* values and their derivation can be found in the SHOWER model technical document (ATSDR 2024b).



Table 5. Chemical properties

Parameter	Value
$\mu\text{g}/\text{m}^3$ to ppb conversion factor	$1 \mu\text{g}/\text{m}^3 = 0.313 \text{ ppb}$
Inhalation Parameters	
Shower <i>f</i> value	0.4878
Bathroom sink <i>f</i> value	0.2324
Bathtub <i>f</i> value	0.3639
Toilet <i>f</i> value	0.232
Clothes washer <i>f</i> value	0.697
Dishwasher <i>f</i> value	0.6975
Kitchen sink <i>f</i> value	0.2324
Utility sink <i>f</i> value	0.2324
Henry's law constant	0.2307
Dermal Parameters	
Chemical type	Organic
Molecular weight (MW)	78.12 g/mol
Dermal permeability coefficient (K_p)	0.0149 cm/hr
Fraction absorbed through skin (FA)	1
Fraction absorbed in gastrointestinal tract (ABS_{GI})	1
Permeability coefficient ratio (B)	0.051
Lag time per event (τ_{event})	0.29 hr/event
Time to reach steady state (t^*)	0.69 hr

Abbreviations: cm/hr = centimeters per hour; g/mol = grams chemical per mole; hr = hours; hr/event = hours per event; $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; ppb = parts chemical per billion parts air



Table 6. Mean parameters used to calculate inhalation and dermal doses

Exposure Group	Body Weight (kg)	Daily Breathing Rate (L/min)	Shower and Bathroom Breathing Rate (L/min)	Total Body Surface Area (cm ²)	Hand Surface Area (cm ²)
6 to < 11 years	31.8	8.33	11.00	1.1E+4	510

Abbreviations: kg = kilograms body weight; L/min = liters air breathed per minute; cm² = square centimeters



Table 7. House information

Parameter	Value
Exhaust fan when bathrooms are occupied?	Off
Bathroom door when bathrooms are occupied?	Closed
Appliance Locations	
Shower/bathtub #1 layout	Bathtub with shower
Exhaust fan #1 location	Bathroom #1
Clothes washer location	Main house
Area Volumes	
Total house volume	204.89 m³
Total bathroom #1 volume	9.061 m ³
Shower #1 volume	2.039 m ³
Air Exchange Rates	
Main house/outdoor air exchange rate	0.45 ACH
Bathroom #1/main house air exchange rate	0.45 ACH
Shower/bathroom #1 air exchange rate	0.45 ACH

Abbreviations: ACH = air changes per hour; m³ = cubic meters air

**Table 8. Appliance parameters**

Appliance	Parameter	Value
Main House		
Kitchen sink	Flow rate	3.347 L/min
Kitchen sink	Average duration per use	0.64 min
Kitchen sink	Maximum kitchen sink uses	15 uses/person/day
Utility sink	Maximum volume use per person	8.544 L/person/day
Dishwasher	Volume per cycle	23.1 L/load
Dishwasher	Average cycle duration	145 min
Clothes washer	Volume per cycle	117 L/load
Clothes washer	Average cycle duration	75 min
Bathroom #1		
Bathroom sink	Flow rate	3.347 L/min
Bathroom sink	Average duration per use	0.64 min
Toilet	Volume per flush	8.7 L/flush
Exhaust fan	Flow rate	1,416 L/min
Shower #1		
Shower	Flow rate	7.6 L/min
Bathtub	Volume	76.47 L

Abbreviations: uses/person/day = appliance uses per person per day; L/min = liters per minute; L = liters water; L/flush = liters water per flush; L/load = liters water per load; L/person/day = liters water used per person per day; min = minute

**Table 9. Clothes washer and dishwasher use schedule**

Clothes Washer	Dishwasher
7:00 p.m.	9:00 p.m.



Table 10. Timing and duration of showers and bathroom stays

Person	Activity	Location	Activity Start Time	Activity Duration (min)	Bathroom Stay after Activity (min)	Person Helping with Tub Bath
1	Showering	Shower #1	5:43 a.m.	7	5	NA
2	Showering	Shower #1	5:56 a.m.	7	5	NA
3	Showering	Shower #1	6:09 a.m.	7	5	NA
4	Showering	Shower #1	6:22 a.m.	7	5	NA
5	Showering	Shower #1	6:35 a.m.	7	5	NA
6	Tub bath	Shower #1	6:48 a.m.	20	5	NA

Abbreviations: min = minute; NA = not applicable



Table 11. Other activity parameters

Parameter	Value
Time between bathroom stays and next shower/tub bath	1 min
Bathroom visits separate from shower/tub bath	4 visits per person per day
Time when all morning showers/tub baths are complete	7:13 a.m.

Abbreviations: min = minute

Tables 12a-12f show the activity pattern throughout the day for each person in the household. Depending upon the pattern selected, each person starts out in the main house compartment and then moves between compartments at various times during the day.



Table 12a. Human activity pattern throughout the day for person 1 in this scenario

Person	Start Time	Location	Appliance Start Times
1	12:00 a.m.	Main house	—
1	5:43 a.m.	Shower #1	Shower #1: 5:43 a.m.
1	5:50 a.m.	Bathroom #1	Toilet #1: 5:50 a.m.; Bathroom sink #1: 5:51 a.m.
1	5:55 a.m.	Main house	—

Person	Start Time	Location	Appliance Start Times
1	9:30 a.m.	Bathroom #1	Toilet #1: 9:30 a.m.; Bathroom sink #1: 9:31 a.m.
1	9:35 a.m.	Main house	—
1	2:00 p.m.	Bathroom #1	Toilet #1: 2:00 p.m.; Bathroom sink #1: 2:01 p.m.
1	2:05 p.m.	Main house	—
1	6:00 p.m.	Bathroom #1	Toilet #1: 6:00 p.m.; Bathroom sink #1: 6:01 p.m.
1	6:05 p.m.	Main house	—
1	8:40 p.m.	Bathroom #1	Toilet #1: 8:40 p.m.; Bathroom sink #1: 8:41 p.m.
1	8:55 p.m.	Main house	—



Table 12b. Human activity pattern throughout the day for person 2 in this scenario

Person	Start Time	Location	Appliance Start Times
2	12:00 a.m.	Main house	—
2	5:56 a.m.	Shower #1	Shower #1: 5:56 a.m.
2	6:03 a.m.	Bathroom #1	Toilet #1: 6:03 a.m.; Bathroom sink #1: 6:04 a.m.
2	6:08 a.m.	Main house	—
2	9:36 a.m.	Bathroom #1	Toilet #1: 9:36 a.m.; Bathroom sink #1: 9:37 a.m.
2	9:41 a.m.	Main house	—
2	2:06 p.m.	Bathroom #1	Toilet #1: 2:06 p.m.; Bathroom sink #1: 2:07 p.m.
2	2:11 p.m.	Main house	—
2	6:06 p.m.	Bathroom #1	Toilet #1: 6:06 p.m.; Bathroom sink #1: 6:07 p.m.
2	6:11 p.m.	Main house	—
2	8:56 p.m.	Bathroom #1	Toilet #1: 8:56 p.m.; Bathroom sink #1: 8:57 p.m.
2	9:11 p.m.	Main house	—



Table 12c. Human activity pattern throughout the day for person 3 in this scenario

Person	Start Time	Location	Appliance Start Times
3	12:00 a.m.	Main house	—
3	6:09 a.m.	Shower #1	Shower #1: 6:09 a.m.
3	6:16 a.m.	Bathroom #1	Toilet #1: 6:16 a.m.; Bathroom sink #1: 6:17 a.m.
3	6:21 a.m.	Main house	—
3	9:42 a.m.	Bathroom #1	Toilet #1: 9:42 a.m.; Bathroom sink #1: 9:43 a.m.
3	9:47 a.m.	Main house	—
3	2:12 p.m.	Bathroom #1	Toilet #1: 2:12 p.m.; Bathroom sink #1: 2:13 p.m.
3	2:17 p.m.	Main house	—
3	6:12 p.m.	Bathroom #1	Toilet #1: 6:12 p.m.; Bathroom sink #1: 6:13 p.m.
3	6:17 p.m.	Main house	—
3	9:12 p.m.	Bathroom #1	Toilet #1: 9:12 p.m.; Bathroom sink #1: 9:13 p.m.
3	9:27 p.m.	Main house	—



Table 12d. Human activity pattern throughout the day for person 4 in this scenario

Person	Start Time	Location	Appliance Start Times
4	12:00 a.m.	Main house	—
4	6:22 a.m.	Shower #1	Shower #1: 6:22 a.m.
4	6:29 a.m.	Bathroom #1	Toilet #1: 6:29 a.m.; Bathroom sink #1: 6:30 a.m.
4	6:34 a.m.	Main house	—
4	9:48 a.m.	Bathroom #1	Toilet #1: 9:48 a.m.; Bathroom sink #1: 9:49 a.m.
4	9:53 a.m.	Main house	—
4	2:18 p.m.	Bathroom #1	Toilet #1: 2:18 p.m.; Bathroom sink #1: 2:19 p.m.
4	2:23 p.m.	Main house	—
4	6:18 p.m.	Bathroom #1	Toilet #1: 6:18 p.m.; Bathroom sink #1: 6:19 p.m.
4	6:23 p.m.	Main house	—
4	9:28 p.m.	Bathroom #1	Toilet #1: 9:28 p.m.; Bathroom sink #1: 9:29 p.m.
4	9:43 p.m.	Main house	—



Table 12e. Human activity pattern throughout the day for person 5 in this scenario

Person	Start Time	Location	Appliance Start Times
5	12:00 a.m.	Main house	—
5	6:35 a.m.	Shower #1	Shower #1: 6:35 a.m.
5	6:42 a.m.	Bathroom #1	Toilet #1: 6:42 a.m.; Bathroom sink #1: 6:43 a.m.
5	6:47 a.m.	Main house	—
5	9:54 a.m.	Bathroom #1	Toilet #1: 9:54 a.m.; Bathroom sink #1: 9:55 a.m.
5	9:59 a.m.	Main house	—
5	2:24 p.m.	Bathroom #1	Toilet #1: 2:24 p.m.; Bathroom sink #1: 2:25 p.m.
5	2:29 p.m.	Main house	—
5	6:24 p.m.	Bathroom #1	Toilet #1: 6:24 p.m.; Bathroom sink #1: 6:25 p.m.
5	6:29 p.m.	Main house	—
5	9:44 p.m.	Bathroom #1	Toilet #1: 9:44 p.m.; Bathroom sink #1: 9:45 p.m.
5	9:59 p.m.	Main house	—



Table 12f. Human activity pattern throughout the day for person 6 in this scenario

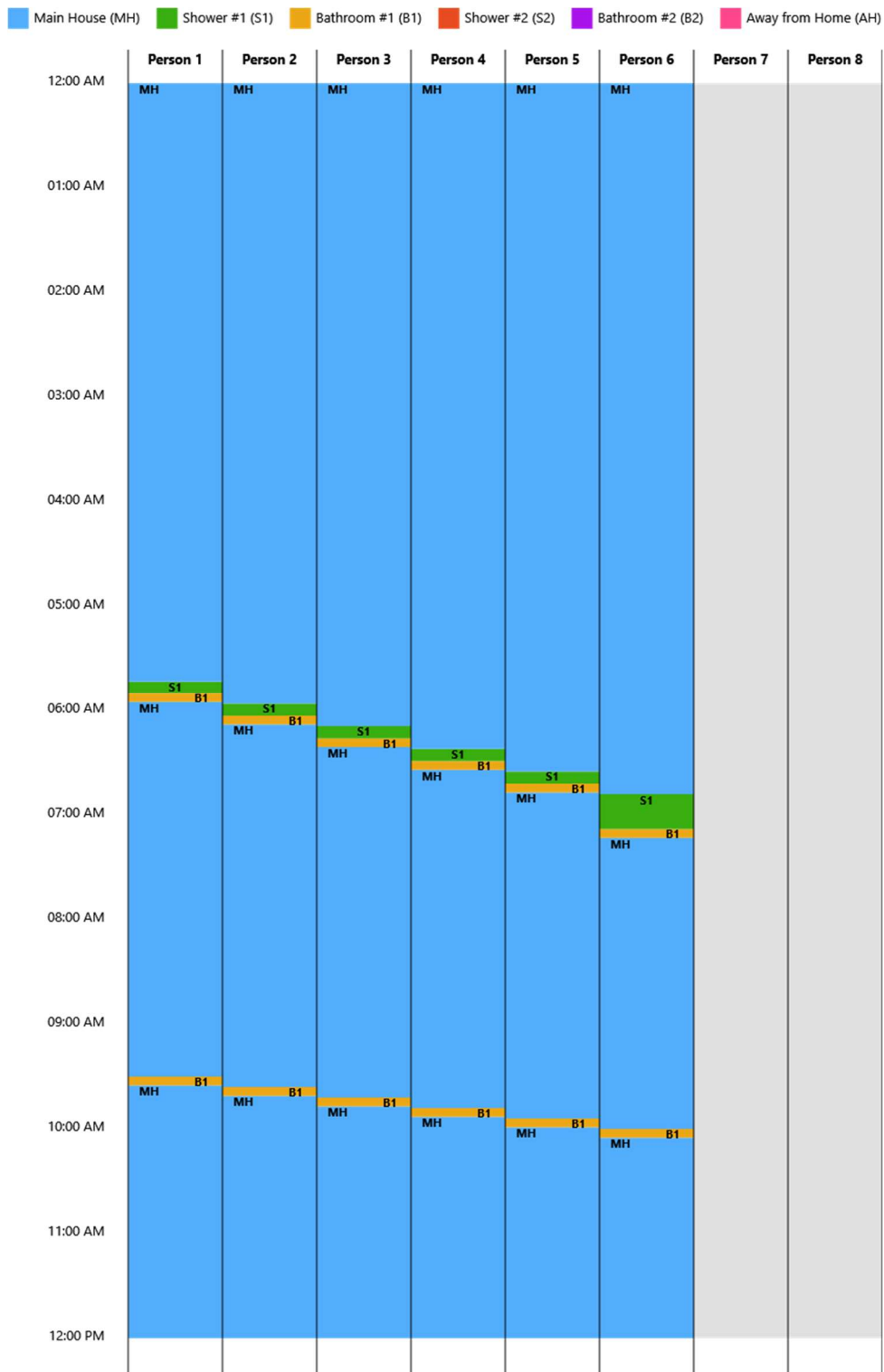
Person	Start Time	Location	Appliance Start Times
6	12:00 a.m.	Main house	—
6	6:48 a.m.	Shower #1	Bath tub #1: 6:48 a.m.
6	7:08 a.m.	Bathroom #1	Toilet #1: 7:08 a.m.; Bathroom sink #1: 7:09 a.m.
6	7:13 a.m.	Main house	—
6	10:00 a.m.	Bathroom #1	Toilet #1: 10:00 a.m.; Bathroom sink #1: 10:01 a.m.
6	10:05 a.m.	Main house	—
6	2:30 p.m.	Bathroom #1	Toilet #1: 2:30 p.m.; Bathroom sink #1: 2:31 p.m.
6	2:35 p.m.	Main house	—
6	6:30 p.m.	Bathroom #1	Toilet #1: 6:30 p.m.; Bathroom sink #1: 6:31 p.m.

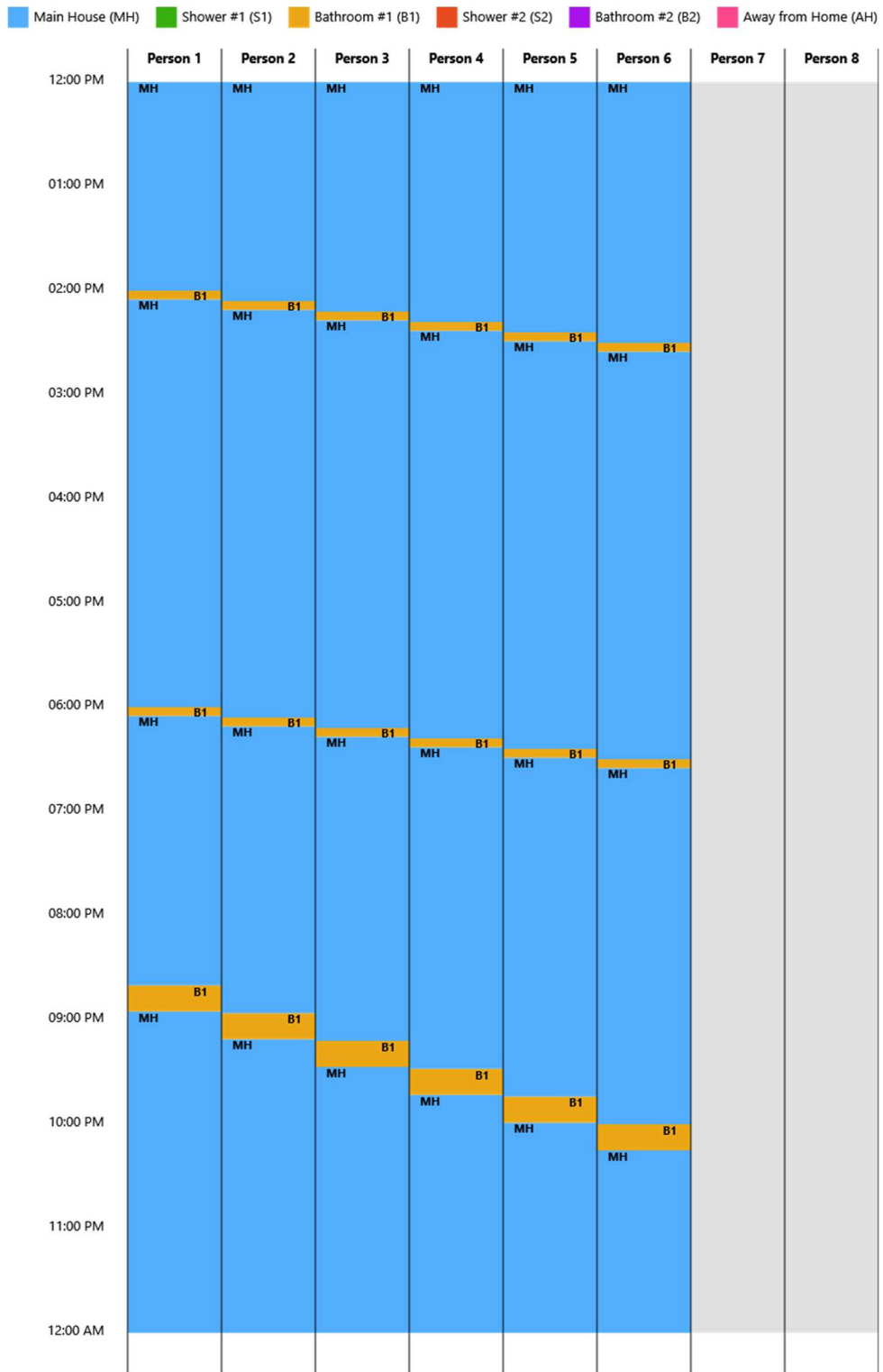
Person	Start Time	Location	Appliance Start Times
6	6:35 p.m.	Main house	—
6	10:00 p.m.	Bathroom #1	Toilet #1: 10:00 p.m.; Bathroom sink #1: 10:01 p.m.
6	10:15 p.m.	Main house	—

Figure 3 uses color blocks and text symbols to show the compartment location of each person in the house throughout the day. The top edge of each block represents the time when the person enters the compartment and the bottom edge represents the time when the person exits the compartment. Text symbols (e.g., S1, B1, MH) also denote start times in each location. In addition, tables 12a-12f show the precise time a person enters a compartment.

Depending upon the custom scenario, each person starts in the main house and moves through the bathroom or shower several times during the day, ending the day in the main house. The target person in this scenario is person 6 because they have the highest exposure.

Figure 3. Human Activity Patterns.





References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024a. Guidance for Evaluating Inhalation and Dermal Exposure Using the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024b. Technical Document for the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.



ATSDR SHOWER Model Report

Custom 6-Person Household

Six morning showers

Contents

Site and Model Input Information	2
Scenario Description	3
6-Person Household Results - Tables	3
6-Person Household Results – Figures	6
Model Parameters	8
References	18

Site and Model Input Information

Information	Report Setting
Site Information	
Site name:	AMSLER HP 20 MIN BATH
Address:	—
Application:	Version 4.0.1
CASRN:	79-01-6
Contaminant:	Trichloroethylene
Synonym:	1,1,2-trichloroethylene
	TCE
	Trichloroethene
Model Input Information	
Chemical name:	Trichloroethylene
Chemical properties:	Default
Exposure routes available:	Inhalation and Dermal
Water concentration:	24.4 µg/L
Outdoor air concentration:	0 µg/m ³
Number of persons in household:	6
Household scenario:	Modified custom scenario
Basis for modified scenario:	Six morning showers
Number of bathrooms in house:	1
Target person:	6
Most highly exposed person:	6
Target person main activity:	20-minute morning tub bath

Scenario Description

This report is for a custom 6-person household scenario. It provides information about the scenario and the most highly exposed person in the house (person 6). Parameter values in this custom scenario that differ from the default parameters for the standard scenario are highlighted in **red**.

6–Person Household Results - Tables

Individual Household Members (Table 1)

Table 1 shows the average daily exposure concentration for each person in the 6-person household, including the most highly exposed person (person 6). The air concentration estimated by the model is independent of who is showering and thus applies to both children and adults.



Table 1. Average daily exposure concentration in $\mu\text{g}/\text{m}^3$ for each person in the house

Person	Target Person	Main Activities	Average Daily Exposure Concentration
1	—	Showering in Shower #1 at 5:43 a.m.	8.9
2	—	Showering in Shower #1 at 5:56 a.m.	11
3	—	Showering in Shower #1 at 6:09 a.m.	13
4	—	Showering in Shower #1 at 6:22 a.m.	15
5	—	Showering in Shower #1 at 6:35 a.m.	16
6	X	Tub bath in Shower #1 at 6:48 a.m.	31

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air

Inhalation and Dermal Doses (Table 2)

Table 2 presents the dermal doses from contact with water only for the most highly exposed person in this scenario (person 6). This contact occurs from showering or bathing and from hand washing during the day. According to EPA guidance, dermal doses for this halogenated compound may be underestimated (see the SHOWER model technical document or EPA's RAGS, Part E for further details). Table 2 also shows the inhaled dose in $\mu\text{g}/\text{kg}/\text{day}$, which is derived from the average daily exposure concentration and age-specific breathing rates for the most highly exposed person in Table 1.



Table 2. Average daily inhalation dose and administered dermal dose in $\mu\text{g}/\text{kg}/\text{day}$ for the target person

Exposure Group	Inhalation	Dermal
6 to < 11 years	15	0.12

Abbreviations: $\mu\text{g}/\text{kg}/\text{day}$ = micrograms chemical per kilograms body weight per day

Peak and Percentage Exposure By Location (Table 3 and 4)

This SHOWER model scenario consists of three compartments: a shower stall, the bathroom, and the main house. The next two tables show the amount of time the target person spends bathing (shower or bath) and the amount of time they spend in the bathroom afterwards. It also provides information on the amount of time the target person spends in the main house throughout the rest of the day. When the target person visits the bathroom during other parts of the day, those bathroom times are included in the main house exposure time. Table 3 shows the average exposure concentration that the target person experiences in each location, and Table 4 shows the percent of exposure that the target person experiences in each location.

The exposure from bathing (shower or bath) and being in the bathroom afterwards can be much higher (but for shorter periods) than the exposure from being in the main house. Knowledge of this brief exposure to high levels in the shower and bathroom compartments might be useful when evaluating whether harmful effects might be possible from acute exposure to high concentrations. This acute exposure to high levels might be particularly important for irritant chemicals, such as formaldehyde, 2-butanone, and acetone. Some irritants, however, cannot be run using the model because parameters are lacking. Health assessors should evaluate this acute exposure duration if the acute EMEG is exceeded. More information about evaluating acute exposure can be found in ATSDR's Guidance for Evaluating Inhalation and Dermal Exposure Using the SHOWER Model (ATSDR 2024a). **Health assessors should consult with the Associate Director of Science (ADS) when evaluating brief exposure to high levels.**



Table 3. Exposure time and average exposure concentration by location for the target person

Location	Exposure Time (min)	Average Exposure Concentration ($\mu\text{g}/\text{m}^3$)
Tub bath	20	1,586
Bathroom after tub bath	5	260
Main house with additional bathroom visits	1,415	8.6
Away from house	0	0
Average daily exposure	1,440	31
Main house (all day)	1,440	4.8

Abbreviations: $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; min = minute



Table 4. Exposure time and percent of total exposure by location for the target person

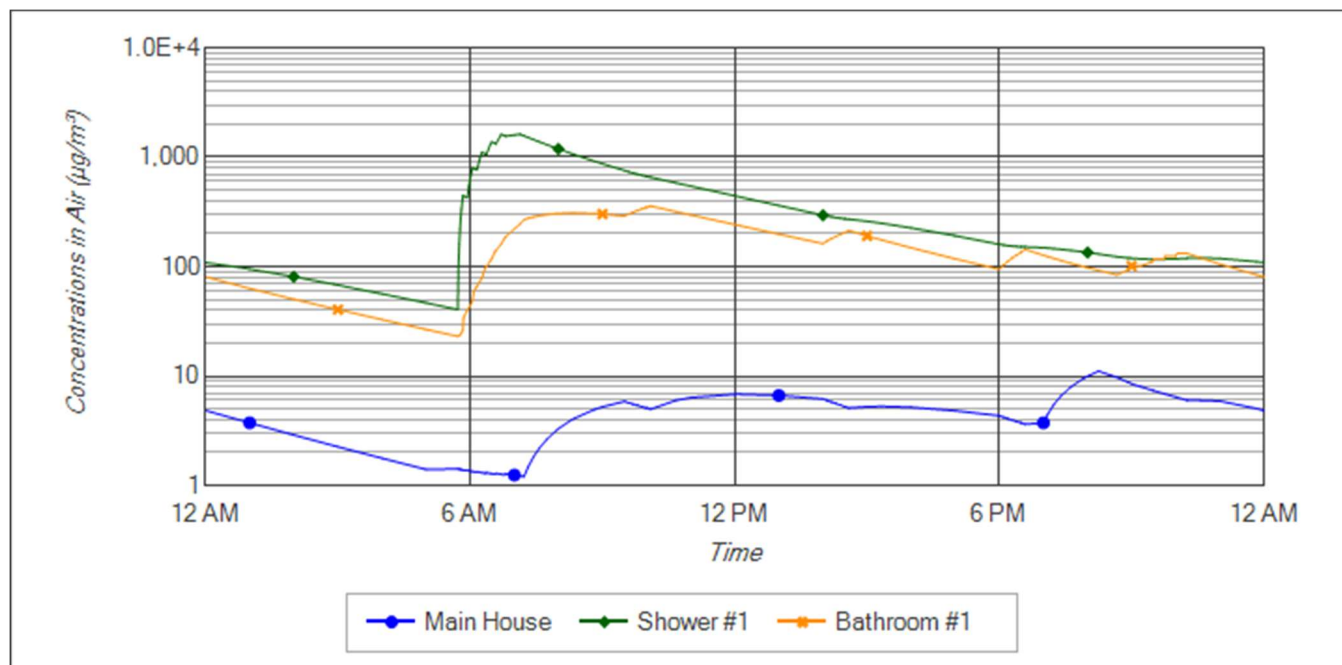
Location	Exposure Time (min)	Percent Exposure (%)
Tub bath	20	70
Bathroom after tub bath	5	2.9
Main house with additional bathroom visits	1,415	27
Away from house	0	0
Average daily exposure	1,440	100
Main house (all day)	1,440	100

Abbreviations: min = minute; % = percent

6–Person Household Results – Figures

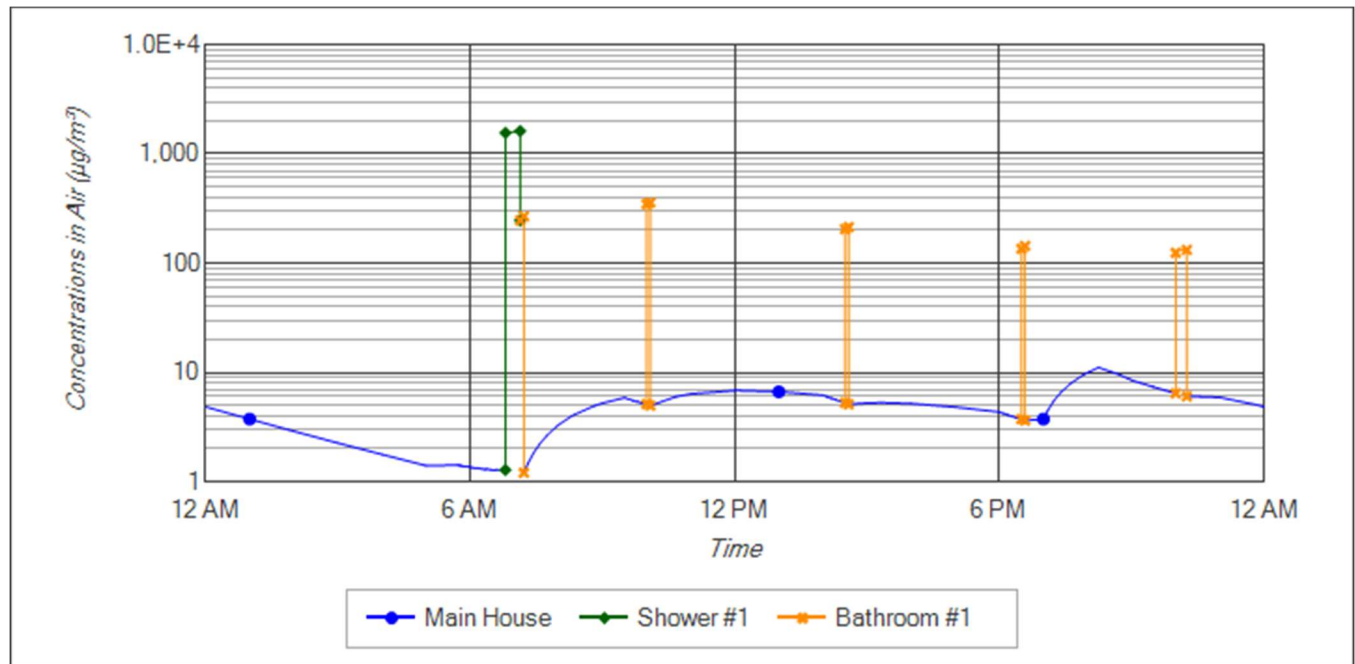
Using a log scale for concentration, Figure 1 shows the calculated chemical air concentrations in the three compartments throughout the day predicted using the SHOWER Model. In general, the chemical air concentrations in the bathroom and shower compartments increase when a person showers or uses other bathroom appliances (e.g. bathroom sink, toilet) and slowly decrease afterwards throughout the day. Chemical air concentrations in the main house compartment rise and fall depending upon other sources in the house and movement of contaminated air from the bathroom compartment through the main house and to the outdoor air.

Figure 1. Calculated air concentrations in the shower, bathroom, and main house compartments for a 6-person household



Using a log scale, Figure 2 shows the contaminant air concentrations the target person (person 6) is exposed to as they move between compartments throughout the day. The contaminant air concentrations shown in Figure 2 are used to calculate the average daily exposure concentration and dermal doses for the target person (person 6) shown in Tables 1 and 2. The contaminant air concentrations are also used to calculate the doses and statistics for the target person shown in Tables 2, 3, and 4.

Figure 2. Calculated exposure air concentrations in different compartments throughout the day for the target person selected in this scenario (person 6)



Model Parameters

The following tables and figures present the parameter values that were used to run this household scenario. These tables are provided as reference and generally are not reported in your public health documents.

In Table 5, the term *f* value refers to the percentage of a chemical that will be released from a water source (e.g., shower water) to air. Chemical *f* values are both chemical- and appliance-specific, such that the same chemical will have different *f* values for different appliances. More information about *f* values and their derivation can be found in the SHOWER model technical document (ATSDR 2024b).



Table 5. Chemical properties

Parameter	Value
$\mu\text{g}/\text{m}^3$ to ppb conversion factor	$1 \mu\text{g}/\text{m}^3 = 0.186 \text{ ppb}$
Inhalation Parameters	
Shower <i>f</i> value	0.67
Bathroom sink <i>f</i> value	0.2229
Bathtub <i>f</i> value	0.3491
Toilet <i>f</i> value	0.2226
Clothes washer <i>f</i> value	0.6686
Dishwasher <i>f</i> value	0.6689
Kitchen sink <i>f</i> value	0.2229
Utility sink <i>f</i> value	0.2229
Henry's law constant	0.4121
Dermal Parameters	
Chemical type	Organic
Molecular weight (MW)	131.3889 g/mol
Dermal permeability coefficient (K_p)	0.0116 cm/hr
Fraction absorbed through skin (FA)	1
Fraction absorbed in gastrointestinal tract (ABS_{GI})	1
Permeability coefficient ratio (B)	0.051
Lag time per event (τ_{event})	0.57 hr/event
Time to reach steady state (t^*)	1.4 hr

Abbreviations: cm/hr = centimeters per hour; g/mol = grams chemical per mole; hr = hours; hr/event = hours per event; $\mu\text{g}/\text{m}^3$ = micrograms chemical per cubic meter air; ppb = parts chemical per billion parts air

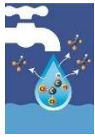


Table 6. Mean parameters used to calculate inhalation and dermal doses

Exposure Group	Body Weight (kg)	Daily Breathing Rate (L/min)	Shower and Bathroom Breathing Rate (L/min)	Total Body Surface Area (cm ²)	Hand Surface Area (cm ²)
11 to < 16 years	56.8	10.56	13.00	1.6E+4	720

Abbreviations: kg = kilograms body weight; L/min = liters air breathed per minute; cm² = square centimeters



Table 7. House information

Parameter	Value
Exhaust fan when bathrooms are occupied?	Off
Bathroom door when bathrooms are occupied?	Closed
Appliance Locations	
Shower/bathtub #1 layout	Bathtub with shower
Exhaust fan #1 location	Bathroom #1
Clothes washer location	Main house
Area Volumes	
Total house volume	204.89 m³
Total bathroom #1 volume	9.061 m ³
Shower #1 volume	2.039 m ³
Air Exchange Rates	
Main house/outdoor air exchange rate	0.45 ACH
Bathroom #1/main house air exchange rate	0.45 ACH
Shower/bathroom #1 air exchange rate	0.45 ACH

Abbreviations: ACH = air changes per hour; m³ = cubic meters air



Table 8. Appliance parameters

Appliance	Parameter	Value
Main House		
Kitchen sink	Flow rate	3.347 L/min
Kitchen sink	Average duration per use	0.64 min
Kitchen sink	Maximum kitchen sink uses	15 uses/person/day
Utility sink	Maximum volume use per person	8.544 L/person/day
Dishwasher	Volume per cycle	23.1 L/load
Dishwasher	Average cycle duration	145 min
Clothes washer	Volume per cycle	117 L/load
Clothes washer	Average cycle duration	75 min
Bathroom #1		
Bathroom sink	Flow rate	3.347 L/min
Bathroom sink	Average duration per use	0.64 min
Toilet	Volume per flush	8.7 L/flush
Exhaust fan	Flow rate	1,416 L/min
Shower #1		
Shower	Flow rate	7.6 L/min
Bathtub	Volume	76.47 L

Abbreviations: uses/person/day = appliance uses per person per day; L/min = liters per minute; L = liters water; L/flush = liters water per flush; L/load = liters water per load; L/person/day = liters water used per person per day; min = minute



Table 9. Clothes washer and dishwasher use schedule

Clothes Washer	Dishwasher
7:00 p.m.	9:00 p.m.



Table 10. Timing and duration of showers and bathroom stays

Person	Activity	Location	Activity Start Time	Activity Duration (min)	Bathroom Stay after Activity (min)	Person Helping with Tub Bath
1	Showering	Shower #1	5:43 a.m.	7	5	NA
2	Showering	Shower #1	5:56 a.m.	7	5	NA
3	Showering	Shower #1	6:09 a.m.	7	5	NA
4	Showering	Shower #1	6:22 a.m.	7	5	NA
5	Showering	Shower #1	6:35 a.m.	7	5	NA
6	Tub bath	Shower #1	6:48 a.m.	20	5	NA

Abbreviations: min = minute; NA = not applicable



Table 11. Other activity parameters

Parameter	Value
Time between bathroom stays and next shower/tub bath	1 min
Bathroom visits separate from shower/tub bath	4 visits per person per day
Time when all morning showers/tub baths are complete	7:13 a.m.

Abbreviations: min = minute

Tables 12a-12f show the activity pattern throughout the day for each person in the household. Depending upon the pattern selected, each person starts out in the main house compartment and then moves between compartments at various times during the day.



Table 12a. Human activity pattern throughout the day for person 1 in this scenario

Person	Start Time	Location	Appliance Start Times
1	12:00 a.m.	Main house	—
1	5:43 a.m.	Shower #1	Shower #1: 5:43 a.m.
1	5:50 a.m.	Bathroom #1	Toilet #1: 5:50 a.m.; Bathroom sink #1: 5:51 a.m.
1	5:55 a.m.	Main house	—

Person	Start Time	Location	Appliance Start Times
1	9:30 a.m.	Bathroom #1	Toilet #1: 9:30 a.m.; Bathroom sink #1: 9:31 a.m.
1	9:35 a.m.	Main house	—
1	2:00 p.m.	Bathroom #1	Toilet #1: 2:00 p.m.; Bathroom sink #1: 2:01 p.m.
1	2:05 p.m.	Main house	—
1	6:00 p.m.	Bathroom #1	Toilet #1: 6:00 p.m.; Bathroom sink #1: 6:01 p.m.
1	6:05 p.m.	Main house	—
1	8:40 p.m.	Bathroom #1	Toilet #1: 8:40 p.m.; Bathroom sink #1: 8:41 p.m.
1	8:55 p.m.	Main house	—



Table 12b. Human activity pattern throughout the day for person 2 in this scenario

Person	Start Time	Location	Appliance Start Times
2	12:00 a.m.	Main house	—
2	5:56 a.m.	Shower #1	Shower #1: 5:56 a.m.
2	6:03 a.m.	Bathroom #1	Toilet #1: 6:03 a.m.; Bathroom sink #1: 6:04 a.m.
2	6:08 a.m.	Main house	—
2	9:36 a.m.	Bathroom #1	Toilet #1: 9:36 a.m.; Bathroom sink #1: 9:37 a.m.
2	9:41 a.m.	Main house	—
2	2:06 p.m.	Bathroom #1	Toilet #1: 2:06 p.m.; Bathroom sink #1: 2:07 p.m.
2	2:11 p.m.	Main house	—
2	6:06 p.m.	Bathroom #1	Toilet #1: 6:06 p.m.; Bathroom sink #1: 6:07 p.m.
2	6:11 p.m.	Main house	—
2	8:56 p.m.	Bathroom #1	Toilet #1: 8:56 p.m.; Bathroom sink #1: 8:57 p.m.
2	9:11 p.m.	Main house	—



Table 12c. Human activity pattern throughout the day for person 3 in this scenario

Person	Start Time	Location	Appliance Start Times
3	12:00 a.m.	Main house	—
3	6:09 a.m.	Shower #1	Shower #1: 6:09 a.m.
3	6:16 a.m.	Bathroom #1	Toilet #1: 6:16 a.m.; Bathroom sink #1: 6:17 a.m.
3	6:21 a.m.	Main house	—
3	9:42 a.m.	Bathroom #1	Toilet #1: 9:42 a.m.; Bathroom sink #1: 9:43 a.m.
3	9:47 a.m.	Main house	—
3	2:12 p.m.	Bathroom #1	Toilet #1: 2:12 p.m.; Bathroom sink #1: 2:13 p.m.
3	2:17 p.m.	Main house	—
3	6:12 p.m.	Bathroom #1	Toilet #1: 6:12 p.m.; Bathroom sink #1: 6:13 p.m.
3	6:17 p.m.	Main house	—
3	9:12 p.m.	Bathroom #1	Toilet #1: 9:12 p.m.; Bathroom sink #1: 9:13 p.m.
3	9:27 p.m.	Main house	—



Table 12d. Human activity pattern throughout the day for person 4 in this scenario

Person	Start Time	Location	Appliance Start Times
4	12:00 a.m.	Main house	—
4	6:22 a.m.	Shower #1	Shower #1: 6:22 a.m.
4	6:29 a.m.	Bathroom #1	Toilet #1: 6:29 a.m.; Bathroom sink #1: 6:30 a.m.
4	6:34 a.m.	Main house	—
4	9:48 a.m.	Bathroom #1	Toilet #1: 9:48 a.m.; Bathroom sink #1: 9:49 a.m.
4	9:53 a.m.	Main house	—
4	2:18 p.m.	Bathroom #1	Toilet #1: 2:18 p.m.; Bathroom sink #1: 2:19 p.m.
4	2:23 p.m.	Main house	—
4	6:18 p.m.	Bathroom #1	Toilet #1: 6:18 p.m.; Bathroom sink #1: 6:19 p.m.
4	6:23 p.m.	Main house	—
4	9:28 p.m.	Bathroom #1	Toilet #1: 9:28 p.m.; Bathroom sink #1: 9:29 p.m.
4	9:43 p.m.	Main house	—



Table 12e. Human activity pattern throughout the day for person 5 in this scenario

Person	Start Time	Location	Appliance Start Times
5	12:00 a.m.	Main house	—
5	6:35 a.m.	Shower #1	Shower #1: 6:35 a.m.
5	6:42 a.m.	Bathroom #1	Toilet #1: 6:42 a.m.; Bathroom sink #1: 6:43 a.m.
5	6:47 a.m.	Main house	—
5	9:54 a.m.	Bathroom #1	Toilet #1: 9:54 a.m.; Bathroom sink #1: 9:55 a.m.
5	9:59 a.m.	Main house	—
5	2:24 p.m.	Bathroom #1	Toilet #1: 2:24 p.m.; Bathroom sink #1: 2:25 p.m.
5	2:29 p.m.	Main house	—
5	6:24 p.m.	Bathroom #1	Toilet #1: 6:24 p.m.; Bathroom sink #1: 6:25 p.m.
5	6:29 p.m.	Main house	—
5	9:44 p.m.	Bathroom #1	Toilet #1: 9:44 p.m.; Bathroom sink #1: 9:45 p.m.
5	9:59 p.m.	Main house	—



Table 12f. Human activity pattern throughout the day for person 6 in this scenario

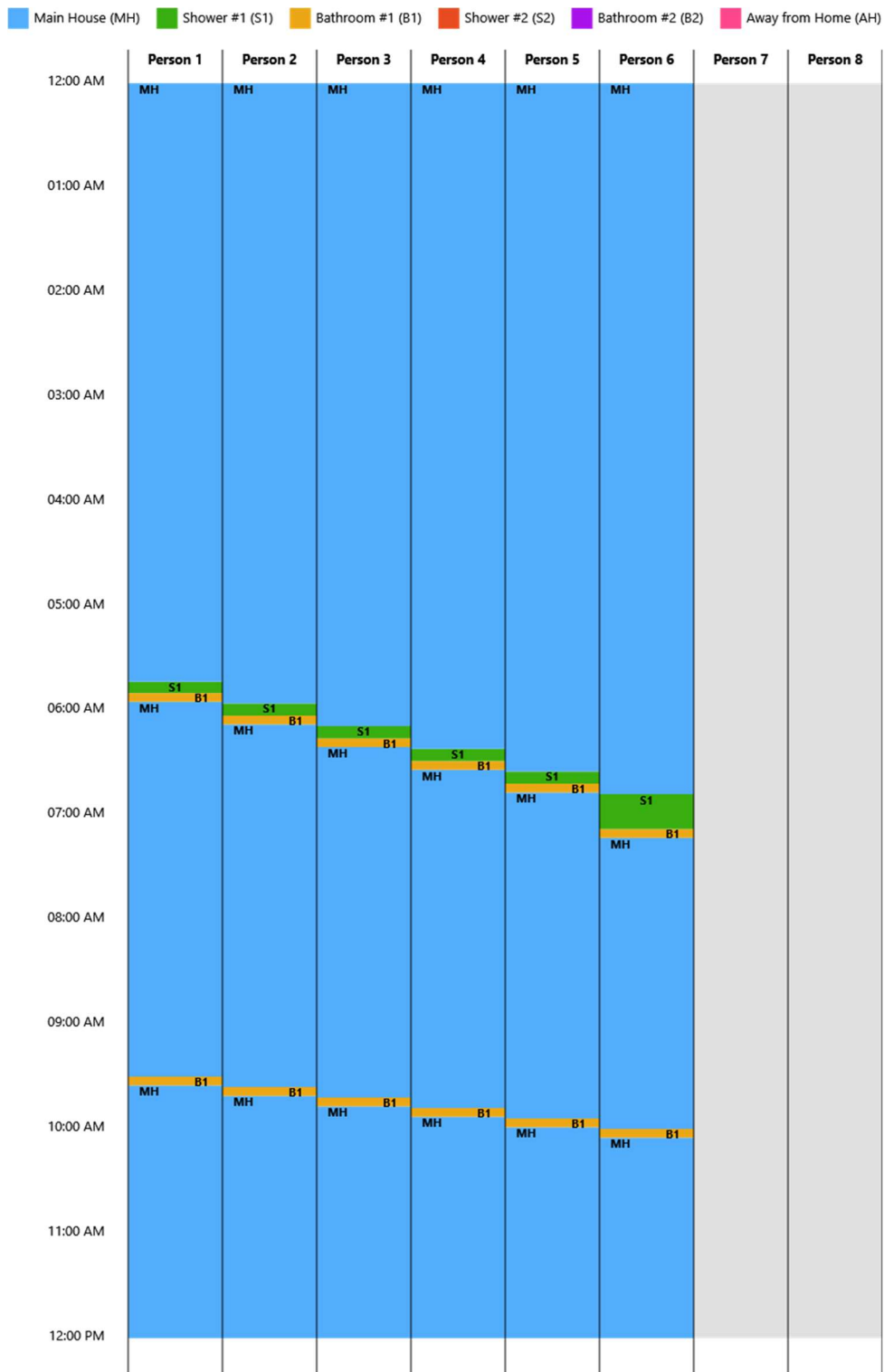
Person	Start Time	Location	Appliance Start Times
6	12:00 a.m.	Main house	—
6	6:48 a.m.	Shower #1	Bath tub #1: 6:48 a.m.
6	7:08 a.m.	Bathroom #1	Toilet #1: 7:08 a.m.; Bathroom sink #1: 7:09 a.m.
6	7:13 a.m.	Main house	—
6	10:00 a.m.	Bathroom #1	Toilet #1: 10:00 a.m.; Bathroom sink #1: 10:01 a.m.
6	10:05 a.m.	Main house	—
6	2:30 p.m.	Bathroom #1	Toilet #1: 2:30 p.m.; Bathroom sink #1: 2:31 p.m.
6	2:35 p.m.	Main house	—
6	6:30 p.m.	Bathroom #1	Toilet #1: 6:30 p.m.; Bathroom sink #1: 6:31 p.m.

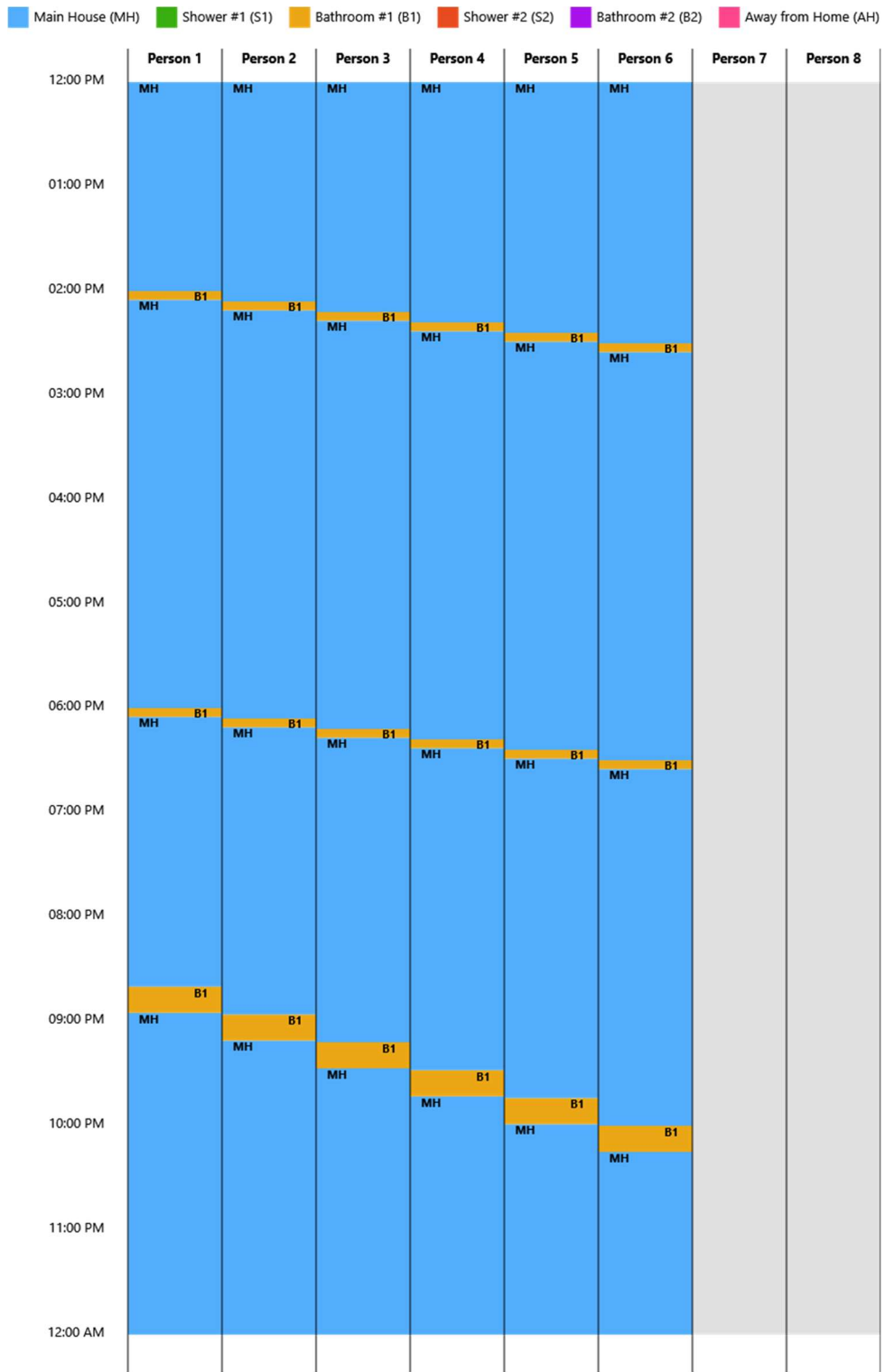
Person	Start Time	Location	Appliance Start Times
6	6:35 p.m.	Main house	—
6	10:00 p.m.	Bathroom #1	Toilet #1: 10:00 p.m.; Bathroom sink #1: 10:01 p.m.
6	10:15 p.m.	Main house	—

Figure 3 uses color blocks and text symbols to show the compartment location of each person in the house throughout the day. The top edge of each block represents the time when the person enters the compartment and the bottom edge represents the time when the person exits the compartment. Text symbols (e.g., S1, B1, MH) also denote start times in each location. In addition, tables 12a-12f show the precise time a person enters a compartment.

Depending upon the custom scenario, each person starts in the main house and moves through the bathroom or shower several times during the day, ending the day in the main house. The target person in this scenario is person 6 because they have the highest exposure.

Figure 3. Human Activity Patterns.





References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024a. Guidance for Evaluating Inhalation and Dermal Exposure Using the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2024b. Technical Document for the Shower and Household Water-use Exposure (SHOWER) Model v4.0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.