



ATSDR

Health Consultation



Analysis of Ambient Air concentrations

Bristol Quarry Landfill

Bristol, Virginia

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and Disease Registry

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A health consultation is written documentation of analysis and health conclusions from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. To prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; collection and analysis of additional environmental samples to better characterize exposures; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partners which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Analysis of Ambient Air Concentrations
Bristol Quarry Landfill
Bristol, Virginia

EPA FACILITY ID:

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry (ATSDR)
Office of Community Health Hazard Assessment

Atlanta, GA 30341

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About ATSDR

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency of the U.S. Department of Health and Human Services (HHS). ATSDR works with other agencies and tribal, state, and local governments to study possible health risks in communities where people could come in contact with dangerous chemicals. For more information about ATSDR, visit the ATSDR website at www.atsdr.cdc.gov/.



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Acronyms and Abbreviations

95UCL	95 th percentile upper confidence limit	HQ	Hazard quotient
ATSDR	Agency for Toxic Substances and Disease Registry	H ₂ S	Hydrogen sulfide
COC	Contaminant of concern	IARC	International Agency for Research on Cancer
ELCR	Excess lifetime cancer risk	IUR	Inhalation unit risk
ETLF	Elevated temperature landfill	LOAEL	Lowest observed adverse effect level
EPC	Exposure point concentration	LOD	Limit of detection
CREG	Cancer risk evaluation guide	MOE	Margin of exposure
CV	Comparison value	MRL	Minimal risk level
1,2-DCP	1,2-dichloropropane	NAAQS	EPA's National Ambient Air Quality Standard
DTSC	California Division of Toxic Substance Control	NOAEL	No observed adverse effect Level
EDB	1,2-Dibromoethane	RfC	Reference concentration
EMEG	Environmental media evaluation guide	RfD	Reference dose
EPA	U.S. Environmental Protection Agency	RSL	Regional screening level
EPC	Exposure Point Concentration	SO ₂	Sulfur dioxide
ESL	Effect screening level	1,2,4-TCB	1,2,4-trichlorobenzene
HHS	U.S Department of Health and Human Services	TCE	Trichloroethylene
HI	Hazard index	TCEQ	Texas Commission on Environmental Quality
		TIC	Tentatively identified compound

VADEQ	Virginia Department of Environmental Quality	VOC	Volatile organic compounds
		WHO	World Health Organization

1. Summary

Residents in the community surrounding the Bristol Quarry Landfill have expressed numerous odor complaints to local, state, and federal agencies since 2020. The Virginia Department of Environmental Quality (VADEQ) has been working with the landfill operator (City of Bristol, Virginia) to address ongoing problems at the landfill that are believed to be a primary source of odors in nearby communities. The Agency for Toxic Substances and Disease Registry (ATSDR) received a May 25, 2021 petition request for public health assessment activities related to these air quality, odor, and exposure concerns in Bristol, Virginia and Bristol, Tennessee, which was accepted October 8, 2021. Between December 2020 and April 2022, the City of Bristol, VADEQ, and the U.S Environmental Protection Agency (EPA) conducted air quality sampling, collecting over 65 samples for volatile organic compound (VOC) analyses (i.e., EPA Method TO-15). In June, July, and October 2021, EPA conducted continuous air monitoring at targeted locations in Virginia and Tennessee for ammonia, methyl mercaptan, hydrogen sulfide (H₂S), sulfur dioxide (SO₂), and total VOCs. ATSDR evaluated these data to determine whether harmful health effects may have occurred from exposures to these chemicals. This document describes the ATSDR process for public health assessment activities, defines the completed exposure pathways, presents the assessment of the environmental exposure data, and provides conclusions and recommendations based on the evaluation.

ATSDR acknowledges the substantial progress that Bristol, VA, has made to reduce the potential for exposures to emissions from the Bristol Quarry Landfill. The city has already fully implemented most of the recommendations included in our report. The landfill stopped accepting waste in 2022 and has made a series of engineering improvements that would further reduce emissions from the landfill. Bristol has maintained an air quality monitoring program since May 2023. While ATSDR did not analyze the results from that program as part of this report, Bristol has indicated that they show that emissions are consistently much lower in recent years than those that were measured in 2021 and 2022. The progress made thus far suggests that any health risks posed to the public by the landfill have been adequately addressed.

Conclusion #1

Exposures of a year or more (chronic-duration) to benzene at the concentrations measured immediately west and southwest of the landfill may result in harmful non-cancer health effects (e.g., decreased B-cell counts) and increased risk of developing leukemia. Exposures to benzene at the detected levels for less than a year are not expected to result in harmful health effects.

Basis for Decision

- The estimated maximum benzene chronic-duration (i.e.,

greater than one year) exposure concentration of 10.2 ppb (based on the 95th percentile upper confidence limit [95UCL] on the mean of all samples from Locations 8, 9 and 10) is three times higher than the chronic exposure duration Minimal Risk Level (MRL) of 3 ppb.

- The concentration of benzene where studies observed chronic exposure duration toxicity to lab animals (30 ppb) is three times the estimated maximum benzene exposure to residents around Bristol Landfill. Considering potential uncertainties in experimental measurement (i.e., the toxicology database), susceptibility, and environmental sampling measurements, a toxicity level only 3 times higher than the estimated exposure concentration raises concerns that exposures may harm people's health.
- Estimated cancer risk from twenty-five years of exposure to benzene at 10.2 ppb is slightly below 1 in 10,000. ATSDR considers this to be an elevated cancer risk. This calculated excess risk from benzene exposure for the most highly exposed residents raises the potential for a small increase above the already existing risk of cancer from all causes.
- While benzene contributed more to the cancer risk than other contaminants, exposure to other contaminants may have contributed to the overall cancer risk for residents around the Landfill.
- Based on the weight of evidence and estimates of short-term benzene exposures, acute (i.e., up to two weeks) and intermediate (i.e., 2 to 52 weeks) duration benzene exposures are not expected to result in harmful health effects.

Conclusion #2

Short-term exposures (e.g., for 5 minutes) to the detected levels of SO₂ may result in harmful health effects in Bristol communities. SO₂ exposures at the 5-minute average detected concentrations may result in airway resistance, or bronchoconstriction. Bronchoconstriction can make it difficult to breathe for individuals, and especially children, with asthma and other respiratory conditions, particularly when exercising or doing rigorous outdoor activities.

Basis for Decision

- Maximum 5-minute SO₂ concentrations in Bristol were above 100 ppb. A study of exercising asthmatic adults found SO₂ exposures for 5 minutes at 100 to 250 ppb results in severe bronchoconstriction.
- Given that they breathe more air for their body weight, are more active than adults, and are more likely to have experienced asthma attacks in the last year, children may be more likely to experience harmful health effects from these short term SO₂ exposures. There is not enough research on SO₂ exposures in children to know whether they are more sensitive than adults.
- The air quality monitoring equipment used to obtain SO₂ levels has a detection limit (100 ppb) that is significantly higher than ATSDR's acute exposure duration MRL (10 ppb). As a result, it is not possible to determine how often SO₂ concentrations near the landfill were high enough to be a concern, particularly for children and other sensitive individuals.

Conclusion #3

Tentatively-identified compounds (TICs) detected by TO-15 sampling and the measured concentrations of methyl mercaptan support resident concerns regarding nuisance odor events in the Bristol Area. These nuisance odor events may cause negative health effects to residents, but the risks for health effects from these odors are challenging to quantify using available data. ATSDR cannot determine whether methyl mercaptan or TIC exposures at the detected levels will cause health effects.

Basis for Decision

- Exposure to foul-smelling odors can result in reduced quality of life and some individuals may experience health symptoms, such as nausea, headaches, and fatigue. These symptoms may be due to the stress of these odor events and/or sensitivity to specific chemicals in the outdoor air.
- Methyl mercaptan was present at concentrations that could contribute to the perception of foul-smelling odors by residents and could thus contribute to the resulting symptoms associated with those odors. However, ATSDR is unable to identify toxicological data to suggest direct

physical health effects from exposures to methyl mercaptan at these concentrations.

- Multiple aldehydes, including 2-butenal (a sensitizer), were TICs in multiple samples collected in 2021 and 2022. Aldehydes can cause irritation of the eyes and respiratory tract upon contact.
- More than 15 other known irritants were TICs in multiple samples collected in 2021 and 2022 in Bristol community locations. These irritants are known to affect the nose, throat, eyes, lung, and skin upon contact.
- The concentrations of TICs could not be determined, and many of the compounds lack scientific studies on their effects. Therefore, the potential for health effects from these compounds could not be assessed.

Conclusion #4

- Other than benzene, methyl mercaptan, and SO₂, inhalation exposures to chemicals sampled and monitored are not expected to result in non-cancer health effects.

Basis for Decision

- Except for benzene, SO₂, and methyl mercaptan, sampled and monitored chemicals, including those listed as potential contaminants of concern (1,2-dibromoethane, 1,2-dichloroethane, carbon tetrachloride, chloroform, hexachlorobutadiene, TCE, ammonia, and H₂S), did not exceed their respective non-cancer health-based screening levels. Maximum concentrations below non-cancer, health-based screening levels are not expected to result in harmful health effects for any individuals, including children and other vulnerable subpopulations.
- Upon close review and based on additional sampling activities conducted in 2022, ATSDR concluded that the acrolein results collected prior to 2022 were not representative of the sampled ambient air and could not be used for health assessment. Additional steps beyond the standard field and laboratory methods are required to ensure the representativeness of acrolein data gathered by EPA Method TO-15. VADEQ and EPA took additional acrolein samples that represented outdoor air in Bristol in 2022.

These verified data showed acrolein concentrations below levels of public health concern.

Next Steps

ATSDR recommends future monitoring of ambient air to determine whether actions being taken at emission sources are reducing community exposures to benzene and sulfur dioxide. Specifically, monitoring is recommended near Locations 8, 9 and 10 (See Appendix B).

Although the landfill has closed and stopped accepting all waste since ATSDR accepted this petition, there may still be a potential for exposures. As such, ATSDR has several recommendations to improve air quality surrounding the landfill. ATSDR recommends that the landfill operators notify the community of onsite activities that are expected to result in increased emissions. ATSDR recommends that the landfill operators continue to investigate the onsite engineering controls, and improve them as needed, potentially including the following:

- enhancing the landfill gas collection system with improved piping and well installations that collect more vapors/emissions,
- improving the leachate control and collection system, and other systems where odorous compounds are most often produced,
- addressing issues with the liner, including along the edges of the landfill where landfill gas and vapors may be released.

ATSDR encourages VADEQ and EPA continue to closely oversee landfill activities, enforce landfill permit regulations, and publicly available the response and oversight activities they have conducted at the landfill, and the solutions being implemented to resolve ongoing issues.

ATSDR recommends VADEQ, the City of Bristol and local/state health agencies conduct timely responses to nuisance odor complaints and that VADEQ or the City of Bristol consider

maintaining and posting an odor complaint log that documents the frequency, duration, and characteristics of odor complaints, the weather conditions (e.g., wind speed and direction) at the time of the complaint, and the location of the complaints reported. These data can be utilized by the landfill to improve their operations and reduce/adjust activities that result in odor events.

ATSDR recommends community members continue to communicate with their local and state agencies regarding odorous events and exposure concerns, including reporting nuisance odor events that may originate from the landfill. ATSDR recommends community members that are sensitive to some of the air contaminants detected in the community take steps to reduce their exposures during noxious odor events. These steps might include closing windows and staying inside the home until the odorous event concludes; and, installing and safely operating air filtration units in the home to reduce indoor air levels of noxious odors.

ATSDR **does not** recommend the use of ozone generating air filtration/treatment systems in residential buildings.

ATSDR will, upon request, continue to provide technical assistance to state and federal agencies regarding monitoring and assessment of community exposures related to air quality in the Bristol area. ATSDR will make this report public on its website and will distribute the document to interested parties, including residents, and state, federal, and local health and environmental agencies. ATSDR will conduct public outreach, including presenting the findings and recommendations of this document in a public meeting. ATSDR will continue to be available to answer questions and provide health education to community members regarding ambient air quality concerns in the Bristol area related to landfill emissions.

There are several limitations to ATSDR's conclusions. From December 2020 through April 2022, ambient air samples were collected in Bristol. These samples provide some understanding of the contaminants that are present in outdoor air. However, it is not practical to gather enough data to characterize exposures in all areas of Bristol for all times of the day and year and for multiple years. The following list provides the primary data limitations recognized for this environmental health assessment:

- Intermediate- and chronic- duration exposure concentrations remain uncertain in Bristol areas. Over sixty time-weighted samples were analyzed for volatile organic compounds and continuous air monitoring was conducted for more than six weeks. However, seasonal weather variability, daily weather fluctuations, such as temperature inversions, and activities conducted at source locations and close to the sampling equipment will impact ambient air concentrations. Although these days of variability may not have been captured by sampling and monitoring, ATSDR's approach is considered health-protective because ATSDR used the maximum detected concentrations in the data set when there are insufficient data to better determine exposure concentrations over time. By using this health-protective approach, ATSDR's conclusions may overestimate the risk of harmful effects.
- Exposures to chemicals that were not assessed are of unknown public health significance. Some common landfill gas chemicals, such as amines, aldehydes, and reduced sulfur compounds, were not targeted for assessment in the Bristol area. These classes of chemicals can present challenges to both field and laboratory personnel not accustomed to these methods and the data produced by the available methods may not be reliable or representative of actual exposures.
- Continuous monitoring technology has detection limits higher than some health-based screening levels, including for ammonia, SO₂, and methyl mercaptan. These chemicals were detected from intermittent peak concentrations above the instrument's limit of detection, but the average concentrations of these chemicals over time are unknown in Bristol ambient air.
- The TICs correspond to signals detected for which the lab did not have identification standards available. Therefore, we cannot be sure of their identity and cannot quantify how much of these compounds were present.
- Some areas of Bristol were not assessed by ambient air monitoring, and it is not certain what the airborne chemical concentrations were in these areas. However, based on our understanding of prevailing local wind patterns and by using geographic information from citizen's reported odor complaints over several years, ATSDR believes the communities that are most impacted by the Landfill's emissions were assessed for air quality.
- Non-landfill sources of emissions were noted but could not be quantified, such as

emissions from a nearby bus depot, a major recycling processor, lawn equipment, etc.

- ATSDR assumed that exposure durations were 25 years for a maximum, health-protective approach. The landfill was open and actively accepting waste from 1998 to 2022 (approximately 24 years). In addition, a sidewall odor mitigation system was installed in June 2023 that is expected to have reduced emissions (Bristol 2025). Other engineering controls have been installed to reduce emissions from the landfill in the time since the data were collected (City of Bristol 2025). Given that emissions from the landfill are expected to be much lower after the sidewall system became operational in June 2023, 25 years was selected as the longest amount of time that residents could have been exposed to the levels of pollutants that were measured between 2020 and 2022.

2. Background

This health consultation has been prepared in response to a May 25, 2021, petition to assess exposures to air contaminants in communities near the Bristol City Landfill, located less than ¼ mile north of the Virginia and Tennessee border. The Agency for Toxic Substances and Disease Registry (ATSDR) assessed the petition request and concluded that relevant exposure data would become available in 2021 to assess inhalation exposures in the community. This consultation provides ATSDR's assessment of data collected by both the Environmental Protection Agency (EPA) and the Virginia Department of Environmental Quality (VADEQ) from December 2020 through April 2022 and includes recommendations to reduce unhealthy exposures and improve the quality of life in the impacted communities in and around the Cities of Bristol, Virginia, and Tennessee. ATSDR intends to make this consultation publicly available by posting the report online and sharing the report with the local community, and state and federal stakeholders.

2.1 Statement of Issues and Purpose

For more than four years, community members in both Virginia and Tennessee have placed numerous odor complaints and voiced health concerns related to the Bristol City Landfill (Landfill) to both the City of Bristol, VA, and VADEQ employees. As of early 2021, odor complaints had increased considerably in frequency. While conducting site visits since 2021, local, state, and federal employees have acknowledged the presence of noxious odors in the communities surrounding the Landfill. Based on an evaluation of the exposure pathways, descriptions of odor complaints, and assessment of analytical data (further discussed in this report), ATSDR acknowledges that a completed inhalation exposure pathway is present from landfill emissions that expose residents in the community to harmful contaminants in air. Although landfill emissions are the focus of this document, ATSDR cannot exclude the possibility that other sources of air emissions (i.e., buses/trucks, industrial emissions, etc.) can contribute to overall ambient air quality. ATSDR accepted this petition to assess the health implications of exposures to contaminants identified by air sampling and continuous air monitoring conducted between December 2020 and April 2022 in the surrounding communities.

2.2 Site Description and Timeline

The Landfill is a municipal solid waste (MSW) landfill managed by the City of Bristol, VA, and located north of the Tennessee border on the eastern edge of the City of Bristol (see [Figure 1](#)

below). The Landfill began operations in 1998. Due to the Landfill's location and shape within an old stone quarry, there are issues that result in excess liquid collection. These issues stem from both from the leachate produced from the solid waste and from rainwater collecting in the quarry. The Landfill is filled with waste to a level below the rim of the quarry presenting surface water infiltration and capping complications for the operators. Older, inactive landfill cells are located to the east of the quarry portion of the Landfill. Additional concerns for the Landfill include,

- elevated benzene concentrations in leachate and on-site air samples (unknown source);
- an ongoing reaction at depth in portions of the Landfill waste, resulting in elevated temperatures and possibly increased odorous and toxic emissions (cause unknown); and,
- inconsistent landfill gas collection for energy use or flaring on the Landfill property.

Figure 1. Bristol Quarry Landfill Location Map

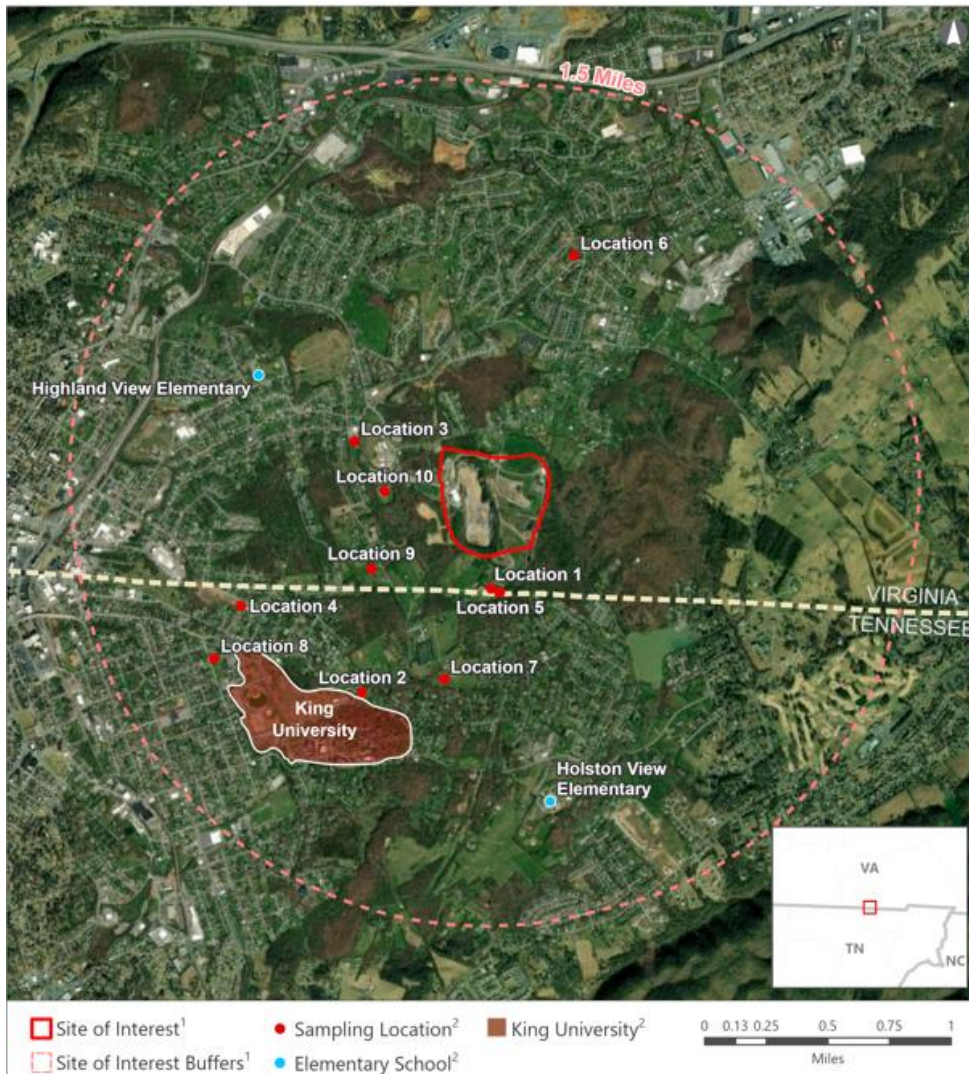


Table 1. Timeline of Bristol Landfill Concerns

Date/Year	Event
April 2021	Landfill operator installs horizontal piping to dewater the landfill to reduce noxious odor emissions.
May 2021	ATSDR receives a petition to evaluate public health exposures surrounding the Landfill.
August 10, 2021	Bristol City Landfill operators informed the community they had acquired additional funding to improve the landfill gas collection systems (i.e., more gas collection wells and piping), increase daily cover activities, install an odor “neutralizer” system, and add additional staff to conduct these activities.
End of 2021	City of Bristol doubled the number of gas collection wells at the Landfill.
January 2022	City of Bristol concluded these measures had not reduced the malodor issue in neighboring communities.
January 3, 2022	City of Bristol, VA requested additional technical assistance from the EPA and VADEQ for Landfill operations and more specifically to address the malodor issues in Virginia and Tennessee.
February 2022	EPA and VADEQ increased their efforts to provide technical support to the Landfill operator regarding landfill engineering controls.
April 25, 2022	An expert panel convened by the VADEQ provided conclusions (see below) and recommendations regarding the Landfill.
May 2022	Tennessee City of Bristol filed a lawsuit in federal court against the Landfill operator (Bristol City, Virginia). The lawsuit placed specific demands and timelines on the Landfill operator, including cessation of waste collection for landfilling and other steps to mitigate releases to the environment.
June 10, 2022	The City of Bristol, Virginia has agreed to the terms demanded by Bristol, Tennessee.
September 9, 2022	Landfill stops accepting additional waste.
April 14, 2023	Bristol, TN and Bristol, VA jointly submitted a Consent Order. This Order outlines requirements that prevent additional trash or waste from disposal in Bristol Landfill. The order also defines plans to close the Landfill upon completion of remedial efforts. Air monitoring will continue during these efforts.
June 14, 2023	A sidewall odor mitigation system becomes operational, reducing the emissions of gasses from the landfill.

The April 2022 panel concluded that:

- The Landfill is exhibiting early signs of an Elevated Temperature Landfill (ETLF) which is linked to production and release of odors. ETLFs are primarily characterized by temperatures in excess of 55°C (131°F) over a broad area for a sustained period of time*

and an atypical accumulation of heat. ETLFs are characterized by low methane content in the landfill gas, high leachate production rates, leachate with elevated concentrations of organic compounds, production of odoriferous gas, rapid settlement, and self-propagating reactions that generate heat. This condition has the potential to worsen unless prompt (immediate) action is taken. (Virginia Tech 2022).

In response to the Landfill exhibiting early signs of an ETLF, the panel recommended a number of steps to mitigate the impacts to the community. The panel suggested that the Landfill operator, “strongly consider a cessation of waste disposal operations at the Landfill due to incompatibility of operations with the necessary odor mitigation and ETLF remedial strategy. Short-term waste filling operations to shape the surface of the Landfill for the placement of the interim geomembrane cover must be carefully coordinated with engineers working on remedial actions” (Virginia Tech 2022).

In September 2023, the Landfill stopped accepting new waste, which was expected to reduce emissions. An April 2023 Consent Order required the City of Bristol, VA to implement a series of steps to further reduce emissions, in accordance with the expert panel’s recommendations. These steps included installing a series of engineering controls to prevent emissions. Importantly, Bristol installed a sidewall odor mitigation system, which became operational in June 2023 (City of Bristol 2025). The City of Bristol has committed to closing the landfill by the end of 2026 as part of this process.

3. Community Description and Concerns

3.1 Community Demographics

Residential areas are located to the north, west, and south of the Bristol Landfill. The nearest residential properties are located to the south within 600 feet of the Landfill boundary. Sensitive receptor locations include the Highlands Juvenile Detention Center (identified as “Location 10” in [Figure 1](#) in this report), and two nearby elementary schools (Holsten View and Highland View Elementary Schools). Industrial parcels are located along the western edge of the landfill, while forested areas are located to the immediate southwest and east of the Landfill. Community members have reported malodors in all directions surrounding the landfill, but the malodor complaints are concentrated in the west, south, and further to the southwest of the site.

Appendix B, [Figure B-1](#) provides demographic information for residents living within 1.5 miles of the Landfill. According to the 2020 U.S. census, 14,203 people occupy 6,778 residential housing units within a 1.5-mile radius of the Landfill. Several commercial and industrial parcels are located around the perimeter of the Landfill where individuals may also be exposed to contaminants released into the ambient air from the Landfill. Within a 1.5-mile radius of the Landfill, approximately 87% of the population is white, 6% of the population is African American, and less than 7% of the population is composed of other races. Approximately 46% of the surrounding population is composed of potentially sensitive groups (i.e., children under age 6, adults over age 65, and women of child-bearing age). This figure does not include individuals with chronic respiratory or cardiopulmonary disease who may also be especially sensitive to contaminants in air.

According to the US Census 2023 American Community Survey 5-Year Estimates, the neighborhood that includes the landfill (census tract 203) has economic characteristics that could make its residents more susceptible to health effects from the Landfill. Residents of this neighborhood had an estimated household income of \$31,299, well below the state's household income of \$90,974. An estimated 27.5% of this neighborhood's residents lived in poverty, a much higher percentage than the 10.5% of Virginia residents who live below the poverty line, and more than the 18.3% of residents of the entire City of Bristol, VA living in poverty.

While demographic data on the residents of the Highland Juvenile Detention Center (Location 10), approximately 1000 feet from the landfill, are not available, they represent a particularly vulnerable population. Children are generally more vulnerable to harmful health effects related to landfill emissions. Additionally, detainees do not have the option to relocate, purchase air filters, or otherwise make adjustments that could reduce their exposures.

3.2 Community Concerns

Community residents have expressed concerns with odors and environmental exposures related to their health. People living in Bristol VA and TN have expressed concerns that breathing landfill gases originating from the Landfill could harm their health.

Following is a summary of the health effects people who have reported landfill gas exposures to ATSDR have noted:

- Headaches and migraines,

- Nausea and throwing up,
- Chest pain and chest tightness,
- COPD and asthma attacks triggered by odors,
- Stomach cramping,
- Arms and hands tingling,
- Burning eyes, sinuses, and throat,
- Dizziness,
- Brain fog or feeling mentally “foggy,” and,
- Loss of sense of smell.

People have reported that landfill odors negatively affect every part of their lives. They don't go outside when the odors are strong. Their sleep is disrupted when odors are strong in their houses, and some people can't work the following day due to lack of sleep. Some also report symptoms such as tingling in hands and arms that start during the night and continue into the following day. Some people have voluntarily evacuated their homes when odors are very strong. Others don't have this option due to lack of transportation or other factors.

People have reported that their pet's health has been impacted and that some pets have died potentially due to exposure to landfill gas contaminants.

4. Sampling Data

ATSDR assessed air quality data for residential areas likely to be impacted by the Landfill to evaluate the health implications of inhalation exposures to ambient air contaminants. This section discusses the air monitoring data collected, the process for selecting community monitoring locations, the assessment of the data for its quality and representativeness, and an overview of the statistical methods used for evaluating the monitoring data collected in 2021.

4.1 Continuous Air Monitoring

Air monitoring data, collected in June, July, and October 2021 by EPA, were used to quantify common malodorous air contaminants emitted from landfills. Continuous monitoring equipment included Honeywell Analytics Single-Point Monitor (SPM) Flex instruments for H₂S, with a 1 part per billion (ppb) lower detection limit; ammonia, with a 10 ppb lower detection limit; and SO₂, with a 100 ppb lower detection limit. A MultiRAE Pro was used for methyl mercaptan monitoring with a lower detection limit of 100 ppb. Continuous monitoring equipment logged contaminant

concentrations every 2–3 seconds from nine distinct locations in communities surrounding the landfill; however, not all locations included continuous monitoring for all listed contaminants. H₂S, total volatile organic compounds (VOCs), and ammonia were monitored at six locations for six weeks in June and July 2021. Additional monitoring for H₂S, total VOCs, ammonia, methyl mercaptan, and SO₂ was conducted for 2 additional weeks in October 2021. Except for total VOCs, ATSDR evaluated these monitoring data to identify maximum acute- and chronic-duration exposure point concentrations (EPCs) for health assessment. Because total VOCs monitoring cannot identify the particular VOC present, these data were not used for health assessment. In total, continuous air monitoring was conducted for approximately eight weeks and those data are included in this health consultation.

4.2 Air Sampling

Air samples were collected and analyzed following EPA Method TO-15 for VOCs (EPA 1999). Air samples were collected by the City of Bristol and by VADEQ between December 2020 and February 2022. EPA also collected additional samples in January and February 2022. Samples collected in February 2022, were collocated with VADEQ TO-15 sampling for data quality assurance purposes (further discussed in next section). In total, 68 samples were collected for TO-15 analysis. These data are assessed in this health consultation ([Table 2](#) and Appendix C, Data Tables).

Table 2. Ambient Air Sampling Summary

Sampling Period	Sampled for	Number of VOC Samples Collected	Location Description
December 2020	VADEQ	3	Community
January 2021	City of Bristol	2	Community
March 2021	City of Bristol	1	Landfill fence line
April 2021	VADEQ	4	Community
May 2021	City of Bristol	1	Landfill fence line
June 2021	VADEQ	8	Community
June 2021	City of Bristol	1	Landfill fence line
July 2021	VADEQ	6	Community
September 2021	VADEQ	1	Community
October 2021	VADEQ	6	Community
December 2021	VADEQ	2	Community
December 2021	City of Bristol	1	Landfill fence line

Sampling Period	Sampled for	Number of VOC Samples Collected	Location Description
January 2022	City of Bristol	1	Landfill fence line
January 2022	EPA	8	Community
February 2022	City of Bristol	1	Landfill fence line
February 2022	EPA	5	Community
February 2022	VADEQ	4	Community
April 2022	EPA	13	Community

Note: VOC = volatile organic compound

4.3 Assessment of Data Quality and Representativeness

The primary goal in assessing the quality of environmental data is to determine whether the data accurately reflect contaminant concentrations in the air sampled. To ensure data represent the environmental media sampled, approved field and laboratory methods that have been tested and proven to produce accurate and precise results must be followed. Also, a sufficient data set that produces representative data must be collected. Unapproved methods can lead to uncertainty in the results, and insufficient sampling information can lead to incorrect assumptions about contaminant exposure concentrations. ATSDR found that the majority of sampling and handling procedures and laboratory analyses followed standard protocols that ensure the data represent environmental contaminant levels. ATSDR also found that the quantity of samples and locations are likely to have captured worst-case and more-common air quality exposure concentrations in the impacted Bristol communities. However, ATSDR did identify two specific concerns regarding data usability and representativeness. These concerns and ATSDR's recommendations to address these concerns were provided to the EPA and VADEQ in 2021. Both agencies agreed that additional sampling was needed to address these issues. Additional sampling at select locations, including some samples that were collected side-by-side, resolved these concerns.

While ATSDR accepts the majority of data reported by VADEQ, EPA, and the City of Bristol, one field sampling procedure (grab sampling) limited the usability of some of the TO-15 data. Based on the grab sample results collected in December 2020 and early 2021, ATSDR requested additional sampling to better define chronic-duration exposures at two specific locations in the Bristol area (Location 9 and the Highlands Juvenile Detention Center, identified as Location 10). In follow up, additional rounds of sampling were conducted by VADEQ and EPA in early 2022 to better define exposures in those areas (see bullet 1. [Instantaneous/Grab vs. Time-Weighted Sampling Results \(EPA Method TO-15\)](#), below for more detail).

Due to concerns with cleaning procedures for field sampling equipment and laboratory analytical issues for one analyte, acrolein, ATSDR has determined the results are not representative of ambient air concentrations. Therefore, acrolein data collected by VADEQ have not been used for assessing exposures in this report (see bullet 2. Acrolein, below for more detail).

1. Instantaneous/Grab vs. Time-Weighted Sampling Results (EPA Method TO-15)—To best represent human exposures to ambient air contaminants, samples should be collected over an extended period of time, as this procedure better represents human inhalation exposures and is more analogous to the toxicological literature used in determining health-based screening levels. For human health assessment, samples should be collected in the range of hours to days depending on the scenario being assessed. Some of the TO-15 samples collected in 2020 and early 2021 were “instantaneous,” or grab, samples collected for only a few minutes. These data may represent sub-chronic, or short-term, exposures, but they do not represent human inhalation exposures, which occur over time. Instantaneous sample results can be overly influenced by plumes of peak contaminant concentrations passing by the sampler for a very brief period of time, such as from a passing vehicle or a nearby lawn mower. Alternately, if the plume does not pass by the sampler during an instantaneous sample, the sample results could underestimate the actual exposure. Results for one instantaneous sample collected near the Highlands Juvenile Detention Center included the highest benzene concentration recorded in the Bristol data set. Results in other instantaneous samples were also higher than those identified in the overall data set. These samples collected instantaneously are not used for assessing chronic-duration exposures. Based on these peak detected concentrations, ATSDR requested and EPA/VADEQ conduct additional sampling at select locations. These additional data are added to the overall data set and are used to identify chronic-duration EPCs. **In summary, instantaneous sample results are valid for assessing peak short-term exposures, but they are not used by ATSDR for intermediate- or chronic-duration exposure assessment.**
2. Acrolein – Gathering representative acrolein data by EPA Method TO-15 is challenging and requires additional procedures to ensure the data represent the air that was sampled (EPA 2010c). Unless additional steps are taken, acrolein results may be biased high due to “growth” in the sample container following sample collection. EPA recommends a number of steps to ensure the sample containers are clean before use, including heating the containers to higher temperatures than typically required for TO-15 analyses, and verifying each sample container is clean before use. Also, laboratories can conduct testing in coordination with other laboratories to determine whether their acrolein results are biased or accurate.

Bristol ambient air samples collected and analyzed by VADEQ have shown consistently elevated acrolein concentrations above background levels and screening values. ATSDR has learned that VADEQ has not been able to employ the additional recommended cleaning procedures and that their state laboratory has had an ongoing problem with biased-high acrolein results. For these reasons, EPA and VADEQ agreed to simultaneously collect TO-15 samples in the same location in 2022. EPA and VADEQ used their own equipment and laboratories for analyses of these replicated samples. These analyses found that VADEQ acrolein measurements are biased high. Acrolein was not detected above the detection limit, which was well below the CV, in EPA samples. The VADEQ samples that were collected side-by-side showed acrolein concentrations above health-based screening values.

ATSDR was unable to use the acrolein data collected by VADEQ for human health assessment because:

- There are known problems with the analysis of acrolein by method TO-15,
- VADEQ was unable to conduct additional equipment cleaning procedures as recommended for confirming acrolein data, and,
- confirmation sampling showed a high bias in VADEQ results as compared to EPA samples in the same location collected in February 2022.

Samples collected by EPA in 2022 provide a limited, valid, and representative acrolein data set for ATSDR's assessment of Bristol ambient air quality in residential areas.

5. Scientific Evaluations

5.1 Exposure Pathway Analysis

To evaluate whether contaminants in ambient air will affect human health, ATSDR must evaluate the site's exposure pathways. ATSDR determines whether the exposure pathways are complete, potential, or eliminated before assessing whether exposures will result in harmful health effects. This section discusses the elements of a completed exposure pathway, identifies the ATSDR method for determining potential contaminants of concern (COCs), and presents information about the health-based screening values selected for comparison to the contaminant concentrations detected in Bristol ambient air.

In order for residents to be exposed to chemical contaminants, they must come into direct contact with the contaminants through a completed exposure pathway. A completed exposure pathway consists of five main parts:

1. A **Source** of contamination (e.g., a chemical release from a landfill),
2. A method of environmental **Transport** (air, water, soil, sediment, etc.), which allows the contaminants to move from the source area to a point where it comes into contact with people,
3. A **Point of Exposure** where people come into physical contact with the contaminants,
4. A **Route of Exposure** (ingestion, inhalation, or skin contact), which is how contaminants enter into people's bodies, and,
5. A **Population at Risk**, i.e., people likely to come into contact with contaminants.

For residents in the Bristol communities located near the Bristol Landfill, and particularly those communities located to the immediate west and southwest of the Landfill, a completed exposure pathway exists. That is, there are chemicals released from the Landfill (Source) that migrate through ambient air (Transport) to residential communities (Point of Exposure) where residents (Population at Risk) inhale contaminants throughout the day (Route of Exposure).

Physical contact with a contaminant alone does not necessarily result in harmful health effects. A contaminant's ability to affect a person's health depends on:

- How much of the contaminant a person is exposed to (dose),
- How long a person is exposed (duration),
- How often a person is exposed (frequency), and,
- The toxicity of the contaminant (how contaminants interact with the body to make people sick).

Other factors affecting a contaminant's likelihood of causing harmful health effects upon contact include an individual's personal habits, diet, age, sex, current health status, and past exposures to toxic chemicals (e.g., occupation, hobbies, do-it-yourself projects).

After determining that both a completed exposure pathways exists and that there are data of sufficient quality and representativeness to conduct an assessment, the human health evaluation takes place in two steps:

- **Step 1, Screening:** Maximum contaminant concentrations are compared to screening values to determine which contaminants exceed health-based comparison levels and require further

evaluation. Descriptive statistics are generated for monitoring data to compare to health-based screening values specific to the sampling time frames. For example, 24-hour time frames were compared to acute health-based comparison values, and overall data sets for specific locations were compared to chronic health-based comparison values.

- **Step 2, Evaluation of Health Implications:** Detailed evaluations of measured data for contaminants that exceed screening values are completed.

These two steps are the basis of the environmental public health assessment process for this site. The public health assessment process varies from site to site based on site conditions, including environmental media types and other factors, and may have additional steps involved to calculate exposure doses. Because the only environmental media and exposure route at this site is air and inhalation, ATSDR does not need to calculate exposure doses.

The next section describes the process for identifying potential COCs followed by a summary of the health-based screening values identified for this assessment.

5.1.1. Overview for identifying contaminants of concern and evaluating risk

Some general observations regarding outdoor air quality are important to understand as a baseline for comparison prior to reviewing the Bristol ambient air data. First, ATSDR notes that outdoor air in populated areas throughout the United States will contain trace amounts of numerous contaminants. Therefore, ATSDR expects that TO-15 air samples collected in Bristol area communities contain many different common air contaminants. It is the concentrations of air contaminants that are most relevant for evaluating public health implications. Second, measured air contaminant levels in the Bristol area, as with air contaminants anywhere, will reflect contributions from many emission sources—some close by and related to industry and road traffic, others from distant sources. ATSDR evaluates exposures to the contaminants detected in the ambient air where people are exposed regardless of source.

TO-15 laboratory data packages and tabulated continuous air contaminant data sets were provided to ATSDR by EPA and VADEQ. These data were assessed for their quality and representativeness and then compared to the most health-protective (lowest) screening levels from ATSDR, EPA, or other agencies to identify potential COCs. Contaminants detected above health-based screening values are considered COCs. COCs are further evaluated in the discussion section of this health consultation. Contaminants with maximum concentrations below their

respective health-based screening value were eliminated from further health assessment and are not expected to be of health concern for any individuals in the Bristol community.

5.1.2. Defining Comparison Values (CV) and Screening Values

ATSDR develops minimal risk levels (MRLs) based on scientific literature that evaluates exposure to specific chemicals and their associated health effects in human or animal studies (ATSDR 2024c). Using the same studies, ATSDR develops media-specific comparison values (CVs), more generically known as health-based screening values, using health-protective exposure assumptions. As a result, ambient air concentrations lower than their corresponding screening value are unlikely to cause harmful health effects. Because screening values are often much lower than harmful health effect levels identified in animal toxicological and human epidemiological studies, ambient air contaminant concentrations greater than screening values are not necessarily levels of contamination that would present a public health hazard. Rather, contaminants with air concentrations higher than screening values, including CVs, require further evaluation.

To select the contaminants requiring further evaluation, ATSDR considered its own CVs first, then EPA risk-based screening levels (e.g., reference concentrations or RfCs), and finally, those screening levels published by other agencies, including state agencies (EPA 2021). Screening values were identified for both short-term (up to two weeks), intermediate (more than two weeks but less than one year) and chronic (over one year) exposure durations. In this process, ATSDR assesses exposures for both cancer and non-cancer health effects. In our evaluation, the air sampling results were compared to the relevant screening values. The screening values that ATSDR used included ATSDR Cancer Risk Evaluation Guidelines (CREGs), ATSDR environmental media evaluation guidelines (EMEGs), and ATSDR reference dose media evaluation guidelines (RMEGs). CREGs are based on EPA cancer slope factors (CSFs). EMEGs are based on ATSDR minimal risk levels (MRLs), and RMEGs are based on U.S. EPA Regional Screening Levels (RSLs) and RfCs. When ATSDR and EPA values were not available, ATSDR used screening values from other organizations who have derived health-based comparison values, including California's Division of Toxic Substance Control (DTSC 2018) screening values, Effect Screening Levels (ESLs) from Texas Commission on Environmental Quality (TCEQ 2022), and World Health Organization (WHO). These CVs include,

- ATSDR CREGs: estimates of the concentrations of a carcinogen at which there is an elevated risk for one case of cancer in one million people exposed over a lifetime.

- ATSDR inhalation MRLs/EMEGs: estimates of the concentrations of pollutants that anyone could be exposed to without experiencing harmful non-cancer health effects, based on chronic, intermediate, and acute exposures (those occurring longer than 365 days, from between 14–365 days, and 14 days of exposure or less, respectively.)
- U.S. EPA reference concentrations (RfCs): estimates of the concentrations of pollutants that anyone could be exposed to for a lifetime without experiencing health effects. RfCs are for inhalational exposures and based on contaminant specific non-cancer health effects.
- U.S. EPA RSLs: risk-based numbers that are available for multiple exposure pathways and for chemicals with both carcinogenic and noncarcinogenic effects. The RSLs used in this analysis correspond to either the risk of one excess cancer per million exposed people (1 in 1,000,000) for carcinogens or a Hazard Quotient (HQ) of 1 for non-carcinogens.
- DTSC screening levels (DTSC SLs): chemical-specific air concentrations developed by the California DTSC for use in the human health risk assessment process at hazardous waste sites and permitted facilities.
- Effect Screening Levels (ESLs): chemical-specific air concentrations developed by the TCEQ to protect human health and welfare. Exposure to an air concentration at or below the ESL is not likely to cause health effects in the general public, including sensitive subgroups such as children, the elderly, pregnant women, and people with preexisting health conditions.
- World Health Organization Air Quality Guidelines (WHO 2021): defined by WHO as “evidence-informed, non-binding recommendations for protecting public health from the harmful effects of air pollutants by eliminating or reducing exposure to hazardous air pollutants and by guiding national and local authorities in their risk management decisions.”

The underlying premise in this approach is that ATSDR uses screening values to focus on the subset of contaminants having the greatest potential to contribute to harmful health effects, while assuming, based on the literature, that the pollutants that have not been found above health-based comparison values do not reach levels of health concern. The contaminants requiring further evaluation (i.e., those contaminants with concentrations above health-based screening values) are discussed in the next section.

5.2. Outdoor Air Analysis

5.2.1. Screening Analysis

ATSDR screened the monitoring data and sampling results to identify maximum concentrations that exceed health-based screening levels (i.e., ATSDR CVs, EPA RSLs, etc.). Maximum concentrations of nine contaminants detected by TO-15 exceeded ATSDR health-based comparison values (see [Table 3](#) below, and Appendix C, Public Health Assessment Site Tool Screening Table). Each of these nine contaminants are carcinogens exceeding their respective ATSDR cancer risk evaluation guideline (CREG). Only one contaminant, benzene, exceeded its non-cancer, health-based screening value. Sixty chemicals analyzed by TO-15 were below health-based screening values and are not of public health significance at the detected levels. For reference, [Table 3](#) provides typical U.S urban background ranges for the TO-15 COCs detected in ambient air in Bristol area communities.

Continuously-monitored air contaminants (H₂S, methyl mercaptan, ammonia, and SO₂) are discussed in more detail below. Maximum exposure concentrations for methyl mercaptan and SO₂ exceeded health-based screening values. Higher air concentrations were also observed over short durations for H₂S and ammonia at community-located monitoring locations, although their time-weighted concentrations did not exceed health-based screening values. Because each of these four contaminants have exceeded their odor thresholds and all contribute to the landfill odor concerns expressed by the community, they are considered COCs and are further evaluated in this document in the Discussion section.

Table 3. Potential Contaminants of Concern (COCs) and Background Levels in U.S. Urban Air – parts per billion (ppb)

Contaminant	Background Level in U.S. [†]	Maximum Bristol Air Concentration	Screening Value	Screening Value Source
1,2-Dibromoethane (EDB)	0.0026	0.01	0.00022	CREG
1,2-Dichloroethane (1,2-DCA)	0.1-1.5	0.05	0.0095	CREG
1,3-butadiene	0.04-0.9 [‡]	0.37 (Grab)	0.015	CREG
1,4 Dioxane	0.1	0.4	0.055	CREG
Benzene (short-term)	0.12-0.58	44.8 (Grab)	9	aEMEG
Benzene (long-term)	0.12-0.58	10.2 [§]	0.043/3	CREG/cEMEG
Carbon tetrachloride	0.2-0.6	0.08	0.026	CREG
Chloroform	0.02-0.05	0.22	0.0089	CREG

Contaminant	Background Level in U.S. [†]	Maximum Bristol Air Concentration	Screening Value	Screening Value Source
Hexachlorobutadiene	0.002-0.003	0.02	0.0043	CREG
Trichloroethylene (TCE)	0.06-0.6	0.19	0.040	CREG
Ammonia	1-5	16.8	100	cEMEG
Hydrogen Sulfide (H ₂ S)	0-1	16.2	1.4/20/70	RMEG/iEMEG/aEMEG
Methyl mercaptan	4*	49.5	0.5	Texas ESL
Sulfur Dioxide (SO ₂)	0-14**	42.4	10	aEMEG

Notes: all values in parts per billion;

† = Levels identified in ATSDR Toxicological Profiles or National Institutes of Health (NIH) Reports on Carcinogens, except where noted;

§ = chronic benzene exposure point concentration is the 95th upper confidence limit based on 26 samples collected at 3 locations with highest benzene levels (Location 8, 9, and 10);

‡ = <https://cfpub.epa.gov/roe/documents/benzeneconcentrations.pdf>

* = level found in ambient air, not urban specific;

** = 1-hour max upper range from 90th percentile of U.S. northeast region values, available at: <https://www.epa.gov/air-trends/sulfur-dioxide-trends>;

ppb = parts per billion; EDB = ethylene dibromide; CREG = cancer risk screening guideline; iEMEG = intermediate environmental media evaluation guideline; aEMEG = acute environmental media evaluation guideline; cEMEG = chronic environmental media evaluation guideline; RMEG = ATSDR chronic exposure media evaluation guideline based on the EPA reference concentration; ESL = Texas long term, health-based Effect Screening Level (Texas ESLs have not been fully evaluated by ATSDR).

5.2.2. Evaluation of Breathing Air

In this section, the potential health implications from acute and chronic exposures to each COC is discussed. Additional information, including the common uses of these chemicals, their background levels in ambient air in the United States, and primary information from the toxicology literature is provided in Appendix D.

5.2.2.1. Background Air Concentrations and Cancer Risk Estimates

When assessing chronic inhalation exposures to chemicals in air, it is important to note what the typical, or common, background levels of contaminants are in ambient air in the United States. Background levels of a number of chemicals in urban air in the United States can exceed ATSDR cancer risk evaluation guidelines (CREG). CREGs represent concentrations unlikely to increase cancer rates in an exposed population above one excess cancer in one million individuals exposed daily over a 78-year lifetime (i.e., <1E-6 cancer risk). For a comparison of carcinogen inhalation exposure risk to background U.S. cancer rates, the American Cancer Society estimates that approximately one in three men and one in two women will be diagnosed with cancer in

their lifetimes (ACS 2024). Cancer toxicity factors are derived from cancer effect levels identified in occupational or laboratory animal studies where exposure levels are higher than background ambient chemical levels.

ATSDR calculated estimated cancer risks to determine whether exposure to a contaminant contributes to an elevated risk of developing cancer. These calculated cancer risks represent the risk of getting cancer resulting from 25 years of exposure to a carcinogen. The calculated excess cancer risk estimates are designed to be protective of health, as they use reasonable upper bound exposure estimates and are likely an overestimate of the potential risk. The actual cancer risks from landfill gas exposure may be lower than the calculated estimate. Excess cancer risk estimates form the basis for determining whether harmful health effects are possible and as a guide in making public health conclusions and recommendations, not to predict the number of cancers in a community. The ATSDR calculated excess cancer risk estimates from exposure are not actual cancer cases, do not represent the actual cases of cancer in communities, and are not estimates of an individual's cancer risk. Therefore, ATSDR cannot determine an individual's risk of developing cancer. An individual's lifetime risk of developing cancer depends on many factors other than exposure to landfill gasses. Any risk of developing cancer from exposure is in addition to an individual's risk from other factors.

Four of the nine chemicals exceeding their CREG values in Bristol ambient air data also exceed typical U.S. ambient air concentrations in urban environments (benzene, chloroform, 1,4-dioxane, and hexachlorobutadiene). [Table 3](#) above shows the maximum detected Bristol ambient air concentrations of the COCs that exceeded CREG values, compared to concentrations commonly detected in urban air. [Table 4](#) below compares the cancer risks from chronic exposures to both U.S. background contaminant levels and the detected levels of carcinogens in Bristol ambient air. Note that air concentration units presented in Table 4 were converted from parts per billion (ppb) to micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) in order to calculate cancer risks. It's also important to note that the background cancer risks that are presented for the U.S. are for a lifetime of exposure, while Bristol cancer risks represent 25 years of exposure.

Table 4. Comparison of Bristol Cancer Risks and Background Levels in U.S. Urban Ambient Air

Contaminant	IUR ($\mu\text{g}/\text{m}^3$) ⁻¹	U.S. Urban Air Background Levels ($\mu\text{g}/\text{m}^3$) [†]	Bristol Chronic EPC [‡] ($\mu\text{g}/\text{m}^3$)	Bristol Cancer Risk	How do Bristol Contaminant Concentrations Compare to U.S. Background Levels?
Benzene	0.0000078	0.4-1.9	33.2	8.3E-5	Higher
Chloroform	0.000023	0.1-0.25	1.09	8.0E-6	Higher
Hexachlorobutadiene	0.000022	0.02-0.03	0.022	1.6E-6	Within Range
1,2-Dibromoethane (EDB)	0.0006	0.02	0.08	1.5E-05	Higher
1,3-Butadiene	0.00003	0.08-1.88	0.23	2.2E-06	Within Range
1,4-Dioxane	0.000005	0.4	2	3.2E-06	Higher
Carbon Tetrachloride	0.000006	1.3-3.9	0.51	9.8E-07	Lower
TCE	0.0000041	0.3-3.3	1.04	1.0E-06	Within Range
1,2-Dichloroethane	0.000026	0.41-6.19	0.12	3.4E-06	Lower

Notes:

† = Levels identified in ATSDR Toxicological Profiles or National Institutes of Health (NIH) Reports on Carcinogens;

‡ = 95UCL from Locations 8, 9, and 10 used for maximum benzene chronic EPC, all other EPCs are maximum detected values;

COC = contaminant of concern; ELCR = IUR x air exposure point concentration (EPC); $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; IUR = inhalation unit risk

5.2.2.2 Exposure Point Concentrations and Exposure Calculations

To conduct a comparison to health-based screening levels, ATSDR must identify an exposure point concentration (EPC) based on the available data. EPCs are determined for specific time frames:

- To assess acute, or short term, inhalation exposures for continuous monitoring data, the maximum of the rolling averages in the data set were used to represent acute EPCs. The rolling averages for each compound were calculated over a duration based on the duration used in the corresponding literature. ATSDR also assessed acute exposures through instantaneous TO-15 sample results to estimate maximum short-term exposure concentrations for contaminants that were not monitored over longer time periods.
- To assess chronic, or long-term, inhalation exposures, the data are assessed using statistical methods to determine representative chronic EPCs. If there is an insufficient quantity of samples for statistical evaluation (i.e., less than 8 samples from the same exposure area), the maximum detected concentrations are used for chronic exposure assessment. For this data set, the maximum time-weighted average concentrations from

continuous monitoring were used for chronic exposure assessment, except for benzene. For benzene, the 95th upper confidence limit (95UCL) on the average concentration was identified by using all non-instantaneous benzene results from locations 8, 9, and 10 (the locations with the highest benzene results, located to the west and southwest of the Landfill).

Continuous monitoring data were analyzed statistically to better understand exposures over the sampling period as well as spatially to better understand source contributions to detected contaminants in ambient air at the ten community-located monitoring stations. The data were collected from June 8 through July 22, 2021, and October 18 through October 29, 2021 at Bristol, VA and were tested for total VOCs, H₂S, methyl mercaptan, SO₂, and ammonia. The statistical program “R” (version 4.4.3) was used to evaluate the data for this assessment (<http://www.r-project.org/>). Means were calculated as rolling averages¹ based on the time frame of the EPC and then were screened against health-based screening levels to identify potential COCs for further analysis. Because of the large amount of non-detects with the data (averaging about 92%), samples that were not detected were substituted with the limit of detection (LOD) to be more health protective in our estimation. Because of the large number of samples contained in the dataset, discrete time averages were first calculated with a statistical package for air quality data known as the “openair” package to smooth the data without removing any data, before calculating a rolling average matching the time frame of the MRL.

5.2.2.3 Location Selection and Timing

Monitoring and sampling locations were selected by EPA in consultation with VADEQ, ATSDR, and community members. In addition to logistical considerations (e.g., access to open space, electricity, and security), the locations selected for sampling and monitoring were based on dominant wind direction, a log of odor complaints indicating areas where the majority of complaints originated, and proximity to the landfill (see wind rose information in Appendix B). A total of ten locations were selected for monitoring and sampling within a one-mile radius of the Landfill (see Figure 2, Appendix B).

Additional “grab samples,” were collected in June, July, and October 2021 by EPA, to assess methyl mercaptan, total amines, and total VOCs in ambient air. These grab samples were collected at many community locations during brief stops by EPA while traveling through the site

¹ A rolling average continuously updates the average of a data set to include all the data in the set for a specific time period with start time rolling forward incrementally (e.g., January 1 to March 1, January 2 to March 2, etc.)

area. The grab sample data are snapshots of air quality with limitations to their use in health assessment; however, these data can indicate the presence of common landfill gases and odorous compounds in the nearby communities.

The timing of TO-15 grab air sample collection by VADEQ in June, July, and October 2021, was based on the continuous air monitoring results collected by EPA. Continuous air monitoring data were observed remotely (i.e., secure EPA website) and in real time by EPA, VADEQ and ATSDR, allowing for air sample collection as soon as elevations or trends in the monitoring data were identified.

Sampling in December 2021 and early 2022 was conducted to address data quality and representativeness concerns raised during review of the initial 2021 data set by the site team (see *Assessment of Data Quality and Representativeness* section above for additional information).

5.2.2.4 Health Evaluations from TO-15 Sampling

EPA air sampling Method TO-15 provides quality-assured results through standard field protocols and laboratory analyses. Each sample was analyzed by the laboratory for over fifty targeted VOCs. Those results best represent ambient air exposure concentrations at each sampling location. Nine COCs were identified in the 68 community-located sample results collected for laboratory analyses. Health implications from exposure to these COCs are discussed below.

5.2.2.5 Benzene Evaluation

ATSDR has derived inhalation MRLs for acute-, intermediate-, and chronic-duration exposures to benzene. MRLs are concentrations of contaminants below which exposure would not cause a non-cancer health effect. MRLs are created to be protective of even the most vulnerable populations, such as children and elderly people. For benzene, these MRLs include:

- 9 ppb for acute-duration inhalation exposure (14 days or less), based on altered immune-associated processes from short-duration exposures in mice (Rozen et al. 1984).
- 6 ppb for intermediate-duration inhalation exposure (15 to 364 days), based on immunodepressive effects in mice (Rosenthal and Snyder 1987).

- 3 ppb for chronic-duration inhalation exposures (>365 days), based on effects of blood elements (hematotoxicity), particularly decreased B-cell counts in humans (Lan et al. 2004a).

The following summary provides additional human studies relevant to benzene inhalation exposure from the ATSDR Benzene Toxicological Profile (ATSDR 2007). To assess residential exposures, health assessors must consider the differences in exposure scenarios and the potential for health effects in children, older individuals, and individuals with underlying medical conditions. For example, residential exposures can occur continuously through day and night, extending through weekends (i.e., 24 hours/day, 7 days/week). These differences in exposure scenarios and exposed populations must be considered when using occupational studies to assess residential exposures. Relevant information from worker studies is listed below; however, these studies focused on the occupational population, typically defined as being healthy, of working age (18 to 65 years of age for 8 to 10 hours per day and 250 days per year), and aware of their potential workplace exposures (ATSDR 2007):

- Inhalation exposures to benzene in excess of regulated workplace limits (an average exposure level of 1,000 ppb during an 8-hour shift) for several months to several years can decrease the relative numbers of circulating blood cells (ATSDR 2007, Qu et al. 2002 and 2003).
- An abnormal decrease in white blood cells was observed in workers exposed to 690–140,000 ppb (average of 6,000 ppb) benzene for more than 1 year (Xia et al. 1995).
- One study found no significant correlations between benzene exposure and the prevalence of abnormal blood cell levels among 200 workers exposed to benzene at estimated concentrations ranging from 10 to 1,400 ppb, relative to the prevalence of abnormal blood cell values obtained from 268 unexposed workers in the same plant (Collins et al. 1991).
- Another study found no significant correlation between exposures to benzene at 550 ppb for an 8-hour shift and blood cell diseases among a group of 387 workers exposed for more than 5 years at this exposure level (Collins et al. 1997).
- The lowest observed adverse effect level (LOAEL) in a long-term occupational study is 570 ± 240 ppb benzene. In this study reduced white blood cell and platelet counts were observed in workers exposed to benzene as compared to workers in the same manufacturing facility not exposed to benzene (Lan et al. 2004a).
- The LOAEL in another short-term occupational study is 60,000 ppb benzene. In this study workers were exposed from 1 to 21 days, 2.5 to 8 hours per day. Among the health effects observed were mucous membrane irritation, reduced white blood cell levels, anemia, skin irritation, dizziness, nausea, and headache (Midzenski et al. 1992).

By comparing the levels of benzene exposure in the Bristol area to the inhalation MRLs and effect levels referenced in the toxicology literature, including the ATSDR benzene Toxicological Profile, we can draw some conclusions as to the potential for harmful health effects in residents living near the Landfill.

Benzene was detected in all air samples collected in the Bristol area. Ambient air benzene concentrations in Bristol community locations ranged from 0.02 ppb to 44.8 ppb; benzene levels were typically higher at monitoring locations 8, 9 and 10 than the other seven Locations (see [Table 5](#)). ATSDR assessed the data set to identify acute-, intermediate- and chronic-duration EPCs. The maximum benzene concentrations detected in community locations included 27 ppb (averaged over 18 hour sample time) and 44.8 ppb (averaged over 5 minutes - grab sample collection time) near the Highlands Juvenile Detention Center (identified as Location 10). The other maximum benzene concentrations at a community location included 26.6 ppb at Location 9 and 21.8 ppb (averaged over 17 hours and 5 minutes, respectively) at the intersection of Booher Road and Willow Oak Court (close to Location 9 and included in Location 9 data set). The maximum Highlands Juvenile Detention Center grab sample of 44.8 ppb and the Booher Road/Willow Oak Court grab sample of 21.8 ppb were collected in December 2020 for the Bristol Solid Waste Management Facility, while the maximum time-weighted benzene concentration of 27 ppb was collected by EPA on January 25, 2022, at Location 10. Though time-weighted air samples are preferred to grab samples (i.e., instantaneous) for health assessment, grab samples collected over a period of seconds to minutes may represent a peak exposure at time of sampling and can be relevant when assessing acute duration exposures for some chemicals. ATSDR's acute duration MRL is used to assess exposures lasting up to 2 weeks in duration.

Table 5. Summary of Benzene Air Sampling Results

Location	Number of Samples	Range of Benzene Concentrations (in ppb)	Sampling Date Range
1	5	0.36 - 3.21	6/15/21 - 7/16/21
2	3	0.08 - 1.89	6/20/21 - 6/29/21
3	2	1.34 - 4.55	6/18/21 - 12/15/21
4	2	0.16 - 3.91	7/8/21 - 7/15/21
5	1	0.11	7/8/2021
6	3	0.22 - 0.37	7/6/21 - 9/30/21
7	5	0.16 - 3.97	10/1/21 - 2/8/22
8	11 (2 side-by-side)	0.02 - 17.7	10/20/21 - 4/20/22
9 (grab)	1	21.76	12/28/2020
9 (TWA)	11 (1 side-by-side)	1.8 - 26.6	12/28/20 - 4/20/22
10 (grab)	1	44.8	12/28/2020

Location	Number of Samples	Range of Benzene Concentrations (in ppb)	Sampling Date Range
10 (TWA)	7	0.5 - 27	12/28/20 – 4/20/22

Notes: ppb = parts per billion

ATSDR recognizes that locations on the landfill property had benzene concentrations at much higher concentrations than those found in the community; however, those are not community exposure point locations and are not directly relevant to community benzene exposure assessment. To assess acute benzene exposures in the Bristol area, ATSDR used maximum results from Location 10 grab (i.e., the maximum grab sample of 44.8 ppb) and the maximum time-weighted benzene concentration of 27 ppb. To assess intermediate and chronic-duration exposures, respectively, for their harmful health implications, including cancer risks, ATSDR evaluated the entire data set to determine the most representative benzene exposure concentration. ATSDR also recognizes that its use of the maximum detected benzene concentration from a grab sample may not be representative of exposure concentrations over time, whether short or chronic time frames. The maximum time-weighted sample result of 26.6 ppb was detected during EPA sampling on January 25, 2022; however, the next highest time-weighted sampling result was 17.7 ppb (Location 8, also from January 25, 2022, sampling event). On January 25, 2022, the operator was conducting pipe/well cleaning activities at the Landfill that released benzene to the ambient air. These operations are expected at certain times of the year; however, they are not continuous and the detected levels of benzene in the community on January 25, 2022, are not expected to be present over long periods of time. In fact, on January 26, 2022 (the day after pipe cleaning at the Landfill), the EPA collected benzene samples from both Locations 8 and 9 and benzene concentrations were lower, at 0.02 and 1.8 ppb, respectively. This observation indicates that the January 25, 2022 time-weighted sampling result for benzene may have been biased high.

To determine a representative worst-case intermediate (greater than 2 weeks but less than 1 year) and chronic (more than a year of exposure) benzene exposure concentration from the available data set, ATSDR used the 26 time-weighted benzene results (samples collected between 18 and 28 hours) with 3 duplicate samples from the 3 Locations with the highest concentrations in the overall benzene data set (Locations 8, 9, and 10). From the benzene data collected at these three locations, which are all located generally to the west and southwest of the Landfill, a 95th percentile upper confidence limit (95UCL) was 10.2 ppb (see Appendix C for benzene 95UCL information). The 95UCL of 10.2 ppb predicts that 95% of the time the true mean is less than or

equal to 10.2 ppb. Benzene concentrations to the north and south of the Landfill were lower (range 0.08 to 4.55 ppb) than those detected to the southwest.

Acute-Duration Benzene Exposures

As noted above, the acute-duration MRL (9 ppb) was exceeded eight times out of 68 samples collected by VADEQ, the City of Bristol, and EPA. Samples from only three of the 10 sampling locations (Locations 8, 9, and 10) were above the acute MRL (9 ppb). Note that Booher Road and Willow Oak Court intersection is in close proximity to Location 9 and the Highlands Juvenile Detention Center property is the location of Location 10:

- Location 8 included one 17-hour duration sample result of 17.7 ppb; 10 samples at Location 8 were below the acute benzene MRL;
- Location 9 included a grab sample result of 21.76 ppb and four time-weighted samples with benzene ranging from 9.53 to 26.6 ppb (all collected over 17 to 28 hour time frames); eight samples were below the acute benzene MRL of 9 ppb; and,
- Location 10 (Highlands Juvenile Detention Center property) included a grab sample (collected over a period of minutes) result of 44.8 ppb and one time-weighted sample (18 hour sample time) result of 27 ppb; six samples were below the acute benzene MRL.

For Locations 1 through 7, maximum benzene concentrations were below the acute MRL, and effects from acute-duration (less than 2 weeks) benzene exposures (i.e., south, north and northwest of the Landfill) are not expected based on the available data.

To assess acute exposure at Locations 8, 9, and 10, ATSDR looked at the acute MRL and associated studies. The acute-duration inhalation MRL is based on the Rozen et al. (1984) study that exposed mice to benzene at 0 ppb; 10,200 ppb; 31,000 ppb; 100,000 ppb; or 301,000 ppb for 6 hours per day for 6 consecutive days. Exposures at 10,200 ppb resulted in no adverse effect on erythrocytes; however, the researchers observed that exposures at 10,200 ppb (LOAEL) depressed peripheral lymphocytes and mitogen-induced blastogenesis of femoral B-lymphocytes. The LOAEL for acute duration inhalation exposures is based on these immunotoxic effects in mice. To derive the acute inhalation MRL, the LOAEL (10,200 ppb) was adjusted from intermittent exposure (the study duration) to a full day of exposure ($LOAEL_{ADJ} = 2,500$ ppb). Because the benzene blood-to-gas partition coefficient for mice and humans is greater than one, no adjustment is required to determine the human equivalent concentrations (i.e., the $LOAEL_{ADJ} = LOAEL_{HEC} = 2,500$ ppb). Following these adjustments, a cumulative uncertainty factor of 300 (10 for use of a LOAEL, 10 for human variability, and 3 for extrapolation from animals to humans

using dosimetric conversion) was applied to the $LOAEL_{HEC}$ to derive the acute human MRL of 9 ppb.

To better understand the potential for harmful health effects from exposures to benzene above the acute MRL, a margin of exposure (MOE) and the weight of evidence is considered. An MOE is a ratio of the concentration of a chemical in air in a study where that chemical caused toxicity in animal studies to the estimated concentration of a chemical in air at the site. As the exposure concentration gets closer to the health effect level (i.e., lower MOE), the risk of harmful effects increases. For acute inhalation exposures to benzene, the toxicity effect level is the $LOAEL_{HEC}$ of 2,500 ppb, based on Rozen et al., (1984). The estimated maximum acute exposure concentration is 44.8 ppb, based on an instantaneously collected sample near Location 10 (Shakesville Road, immediately west of the landfill). The MOE of 52 for acute benzene exposures in this industrial area was identified by dividing the toxicity effect level (2,500 ppb) by the maximum-detected acute exposure level (44.8 ppb). The maximum time-weighted samples (27 ppb) had an MOE of 93.

ATSDR also considers the weight of evidence when determining whether health effects will occur from exposures. Additional benzene studies have reported human health effects from short-duration exposures to concentrations considerably higher than 44.8 ppb. In one short-term occupational study (Midzenski et al. 1992), a $LOAEL$ of 60,000 ppb benzene was identified with multiple observed health effects (mucous membrane irritation, reduced white blood cell levels, anemia, skin irritation, dizziness, nausea, and headache). In this study workers were exposed from 1 to 21 days, 2.5 to 8 hours per day. To better assess exposures in Bristol, the Midzenski et al. (1992) $LOAEL$ of 60,000 ppb is adjusted from intermittent to continuous exposure (i.e., dividing by 3 to 10 times), yielding a $LOAEL_{ADJ}$ range of 20,000 to 6,000 ppb. In another study, Collins et al. (1991) found no significant correlations between benzene exposure and the prevalence of abnormal hematological values among 200 workers exposed to benzene at estimated concentrations ranging from 10 to 1,400 ppb, relative to the prevalence of abnormal hematological values obtained from 268 unexposed workers in the same plant. In a more recent evaluation, Collins et al. (1997) found no significant correlation between exposure to benzene at an 8-hour TWA of 550 ppb and prevalence of clinically-defined lymphopenia (or other measures of hematotoxicity including mean corpuscular volume [MCV], and counts of white blood cells, red blood cells, hemoglobin, and platelets) among a group of 387 workers exposed for up to or greater than 5 years.

The maximum ambient air concentration in Bristol (44.8 ppb) was an instantaneous sample

collected over a period of minutes. The highest time-weighted sample of 27 ppb benzene was collected from the same area. A total of 8 out of 68 samples collected by VADEQ, the City of Bristol and EPA had benzene concentrations exceeding the acute MRL of 9 ppb. Maximum acute-duration benzene exposure concentrations (27 to 44.8 ppb) were between one and two orders of magnitude below the toxic effect level and multiple studies have not observed health effects at these exposure levels. **Based on the weight of evidence, ATSDR concludes that harmful effects from short-term exposures to benzene at or below the maximum detected benzene concentration of 44.8 ppb are not expected.**

Intermediate-Duration Benzene Exposures

The intermediate duration benzene MRL of 6 ppb was used to evaluate intermediate exposures. The MRL is based on mice exposed to benzene at three concentrations (10,000 ppb; 30,000 ppb; and 100,000 ppb) for 6 hours/day, 5 days/week for 20 exposure days (Rosenthal and Snyder 1987). Rosenthal and Snyder (1987) found that mice exposed to 10,000 ppb (LOAEL) benzene had a decreased capacity to mount an immune response to a foreign antigen (i.e., significantly delayed splenic lymphocyte reaction to foreign antigens). To determine the intermediate MRL, the LOAEL (10,000 ppb) was adjusted to account for intermittent exposures and to identify the human equivalent concentration ($LOAEL_{HEC} = 1,800$ ppb). A cumulative uncertainty factor of 300 was applied to the $LOAEL_{HEC}$ (10x each for use of a LOAEL and for human variability, and 3x for extrapolation from animals to humans using dosimetric conversion) to derive the intermediate-duration MRL of 6 ppb.

None of the samples collected from location 1 through 7 exceeded the intermediate MRL. Less than half of the benzene results collected from Locations 8, 9 and 10 exceeded the intermediate MRL of 6 ppb. The most health protective estimate of the maximum intermediate-duration benzene exposure concentration is the 95UCL of 10.2 ppb.

To better understand the potential for harmful effects from benzene exposures above the intermediate MRL, the MOE is calculated, and a weight of evidence approach is considered. For intermediate exposures, the effect level of 1,800 ppb ($LOAEL_{HEC}$) used for the MRL derivation is divided by the intermediate duration benzene exposure concentration of 10.2 ppb, yielding an MOE of 176. If the MOE is within 1 to 2 orders of magnitude, then a weight of evidence evaluation is recommended. Several occupational studies have assessed intermediate inhalation exposures to benzene, yet none of these studies found health effects below 570 ± 240 ppb benzene:

- Cody et al. (1993) studied workers during their first year of employment and found significantly lower white blood and red blood cell counts in employees exposed to benzene levels greater than the median (LOAEL estimated at 40,000 to 54,000 ppb), compared to those with lower estimated exposure levels.
- Yin et al. (1987) found serious health effects (aplastic anemia) among workers exposed to 29,000 ppb between 3.5 months and 19 years. This study revealed effects, ranging from mild to severe, of benzene exposure in factory workers in China. Of the 528,729 workers, 95% were exposed to mixtures of benzene, toluene, and xylene, while 5% (26,319 workers) were exposed to benzene alone at 20 to 264,000 ppb in air in 95% of the workstations. Over half of the workstations had levels of benzene in the air of less than 13 ppm; about 1% had levels of 13,000 and 264,000 ppb. Benzene toxicity, as indicated by leukopenia, aplastic anemia, and leukemia, was seen in 0.94% of the workers exposed to benzene and 0.44% of the workers exposed to the mixtures. Similar toxicity was found in employees of 28 of the 141 shoe factories studied (124 cases in 2,740 employees) (Yin et al. 1987). A positive correlation was observed for prevalence of adverse benzene effects and benzene concentration in data from these 28 shoe factories. The authors determined that the affected people were exposed to 29,000 ppb benzene (LOAEL). The main limitation of this study is the lack of information on the duration of exposure.

Based on an MOE that shows maximum Bristol-area, intermediate-duration benzene exposures (10.2 ppb) are 2 orders of magnitude from the effect level, and the weight of evidence which shows that harmful effects from intermediate exposures have not been observed below 570 ± 240 ppb, ATSDR concludes that harmful health effects from intermediate-duration exposures are not expected.

Chronic-Duration Benzene Exposures

The chronic-duration MRL of 3 ppb is based on a 6.1 ± 2.9 year occupational study where workers exposed to benzene had statistically-significant decreased counts of B-lymphocytes as compared to control populations in the same industry where measurable concentrations of benzene were not found. In this study, the group identified as those exposed to <1,000 ppb benzene had significantly decreased counts of all types of white blood cells and platelets. The mean 1-month exposure level in this group was 570 ± 240 ppb benzene (ATSDR 2007). Mean 1-month benzene exposure levels in the control group were estimated to be less than 40 ppb benzene. The highest magnitude of hematological effect in this study was noted in decreased B-cell counts for the highest exposure group, and this was selected as the critical effect for benchmark dose modeling. Using the benchmark dose modeling approach, the point of departure of 100 ppb for the 8-hour

exposure study was selected and adjusted for continuous exposure (24 hours) to 30 ppb. To account for human variability in response to exposures, the adjusted point of departure of 30 ppb was divided by 10 to produce the chronic-duration inhalation MRL of 3 ppb, which represents the level below which harmful health effects are not expected in any individuals chronically exposed to that level of benzene for over a year or more.

Location 8, Location 9, and Location 10 each had multiple time-weighted samples with benzene concentrations exceeding the chronic duration inhalation MRL of 3 ppb. Benzene also exceeded the MRL one time each at Location 1 (3.21 ppb), Location 3 (4.55 ppb), Location 4 (3.91 ppb), and Location 7 (3.91 ppb). Locations 2, 5, and 6 did not have benzene concentrations above the chronic MRL of 3 ppb. As discussed above, because the ambient air data are limited to only a few 24-hour samples for each location over the entire year of sampling, ATSDR cannot identify a chronic benzene exposure level for any single location with certainty.

Consequently, ATSDR used data from the three Locations with the maximum detected time-weighted benzene concentrations (Locations 8, 9, and 10) to determine the best estimate of the worst-case chronic benzene exposure concentration in the Bristol area. These sampling locations cover an area directly west and to the southwest of the Landfill. The chronic benzene exposure point concentration of 10.2 ppb was identified by statistically assessing the quantity, distribution, and range of benzene results from the three selected locations. This assessment of 26 time-weighted samples produced an upper confidence limit around the true mean, or a 95UCL, of 10.2 ppb.

The estimated chronic exposure concentration (10.2 ppb) exceeded the chronic non-cancer screening value of 3 ppb. While individuals will not spend 24 hours per day outdoors, indoor air benzene concentrations are unknown in the Bristol area and may be lower or higher than the chronic exposure concentration identified in outdoor ambient air. To understand the potential for health effects from benzene exposures above the chronic MRL, an MOE was calculated. The chronic LOAEL of 100 ppb (Lan et al. 2004a) was adjusted from intermittent to continuous exposure to give the toxicity effect level of 30 ppb for the general population (ATSDR 2007). The toxicity effect level of 30 ppb is divided by the chronic exposure concentration of 10.2 ppb yielding an MOE of 3. An MOE that is less than an order of magnitude (i.e., 10x) suggests the potential for harmful health effects to area residents.

Based on the health effect studies referenced above and a narrow MOE (< 3), ATSDR concludes that harmful non-cancer health effects (i.e., decreased B-cell counts) from continuously

inhaling benzene at 10.2 ppb or higher for a year or more may occur for individuals residing near the Landfill.

Cancer Risk Assessment for Benzene

The EPA has identified an inhalation unit risk (IUR) range for benzene of 2.2E-6 to 7.8E-6 per $\mu\text{g}/\text{m}^3$ (EPA 2000) based on the development of leukemia. ATSDR uses the upper end of the benzene inhalation unit risk range to estimate cancer risk. To calculate the cancer risk from chronic exposure to benzene in the Bristol area, the IUR ($0.0000078 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (10.2 ppb, which equals $33.2 \mu\text{g}/\text{m}^3$), resulting in an estimated cancer risk of less than 1 excess cancer in 10,000 individuals exposed to benzene at 10.2 ppb for 25 years ($8.3\text{E}-05$). Twenty-five years of continuous, daily exposure to benzene at the concentrations observed near the Landfill could increase the risk of developing leukemia.

Based on twenty-five years of exposure to the chronic benzene EPC of 10.2 ppb, less than 1 excess case of leukemia may occur among every 10,000 individuals. This is higher than the expected rate of cancer from exposure to background levels of benzene exposure for U.S. residents. Therefore, there is a concern for excess cancer from exposure to benzene among the most-exposed residents living near the Landfill.

In summary, ATSDR concludes that exposure to benzene in the Bristol area for a year or less is not expected to result in harmful health effects; however, long term exposure to benzene (i.e., greater than a year) at the detected levels (i.e., 10.2 ppb), particularly to the west and southwest of the Landfill, may result in harmful non-cancer health effects for some individuals. There is also a slightly elevated risk of developing leukemia from chronic (25 years) benzene exposures at the levels measured in areas west and southwest of the Landfill.

5.2.2.6 Chloroform Evaluation

The maximum detected chloroform concentration in Bristol ambient air (0.22 ppb, Location 1) did not exceed non-cancer screening levels (acute, intermediate, or chronic duration). Harmful non-cancer health effects are not expected from exposures to chloroform at the concentrations detected in Bristol area ambient air. However, the maximum detection (0.22 ppb) and all other chloroform detections, which ranged from 0.02 and 0.05 ppb, exceeded the ATSDR CREG of 0.0089 ppb.

The EPA has identified an IUR for chloroform of 2.3×10^{-5} per $\mu\text{g}/\text{m}^3$ based on quantitative inhalation risk estimates from a gavage study (i.e., oral/ingestion) of male rats, which showed increased rates of kidney tumors as doses increased (IRIS 2001). Human equivalent doses were identified to determine tumor incidence rates by exposure dose. To calculate the cancer risk from chronic exposure to chloroform in the Bristol area, the IUR ($0.000023 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (0.22 ppb, which equals $1.09 \mu\text{g}/\text{m}^3$).

Based on twenty-five years of exposure to the chronic chloroform EPC of 0.22 ppb, less than 1 excess cancer is expected in every 100,000 individuals exposed ($8.0\text{E}-06$). Although this estimate is slightly higher than the estimated cancer risk from exposure to background levels of chloroform in the U.S., ATSDR does not find this elevated cancer risk to be a concern. **Harmful non-cancer health effects are not expected from exposures to chloroform in the Bristol area ambient air and cancer risk is considered low.**

5.2.2.7 Hexachlorobutadiene Evaluation

Only one community-located ambient air sample, collected in October 2021 at Location 9, had a detectable level of hexachlorobutadiene (0.02 ppb). ATSDR and EPA have not derived non-cancer screening values (i.e., MRL or RfC) for hexachlorobutadiene inhalation exposures. However, the California Division of Toxic Substances Control (DTSC) has identified 0.38 ppb as their noncancer screening level. The singular detection of hexachlorobutadiene was below the DTSC noncancer screening value; therefore, noncancer health effects are not expected from exposures to hexachlorobutadiene.

The EPA has identified an IUR for hexachlorobutadiene of 2.2×10^{-5} per $\mu\text{g}/\text{m}^3$ due to development of renal tubular adenomas and adenocarcinomas in Sprague-Dawley rats (IRIS 1987). To calculate the cancer risk from chronic exposure to the maximum singular detection of hexachlorobutadiene in the Bristol area, the IUR ($0.000022 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC of 0.02 ppb, which equals $0.22 \mu\text{g}/\text{m}^3$.

Based on twenty-five years of exposure to the maximum detected hexachlorobutadiene concentration of 0.02 ppb, less than two excess cancers are expected in every 1,000,000 individuals exposed ($1.6\text{E}-06$). ATSDR does not find this cancer risk to be of concern. **Harmful non-cancer health effects are not expected from exposures to hexachlorobutadiene in the Bristol area ambient air and the cancer risk is considered low.**

5.2.2.8 1,2-Dibromoethane (EDB) Evaluation

The maximum EDB concentration detected in Bristol ambient air (0.01 ppb) is below the EPA RfC of 1.1 ppb. Though analyzed in all TO-15 samples, EDB was detected only twice: once each at Locations 1 and 2. Continuous exposure to EDB at the maximum detected concentration is not expected to result in harmful non-cancer effects.

The International Agency for Research on Cancer (IARC), EPA, and NTP have concluded that EDB is likely carcinogenic to humans (EPA 2004; IARC 1999; and NTP 2021). To estimate the cancer risk from daily exposure over twenty-five years, the EPA (2004) has identified an inhalation unit risk (IUR) for EDB of $0.0006 (\mu\text{g}/\text{m}^3)^{-1}$, based on the dose-response data in animal studies identifying the development of nasal tumors, hemangiosarcomas and mesotheliomas (EPA 2004). The IUR ($0.0006 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic exposure point concentration (EPC) of 0.01 ppb, which equals $0.08 \mu\text{g}/\text{m}^3$, to produce the cancer risk for the exposed population.

Between one and two excess cancers are estimated in every 100,000 individuals exposed daily over twenty-five years to the maximum EDB concentration detected in Bristol ambient air ($1.5\text{E}-05$). However, EDB was only detected twice in a total of 68 samples over a 1.5-year period. Although this estimate is slightly higher than estimated cancer risk ranges from exposures to background levels of EDB in the U.S., ATSDR does not find this cancer risk to be of concern.

Inhalation exposures to the detected levels of EDB in the Bristol area are not expected to result in harmful non-cancer health effects and the cancer risk is considered low.

5.2.2.9 1,3-Butadiene Evaluation

1,3-Butadiene was rarely detected in ambient air samples, and it was only detected at two locations where sampling occurred (Locations 2 and 8). Due to species differences in the metabolism of 1,3-butadiene and the lack of chemical-specific data to adjust for these differences, ATSDR has not derived inhalation MRLs. ATSDR did not derive MRLs due to the potential for overestimating the risk to humans (ATSDR 2012a). However, the EPA, the Texas Commission on Environmental Quality (TCEQ) and Kirman and Grant (2012) have derived chronic risk assessment values, all based on ovarian atrophy toxicity in mice (ATSDR 2012a):

- EPA identifies 0.9 ppb as their reference concentration (RfC),
- TCEQ identifies 15.4 ppb as their chronic reference value, and,

- Kirman and Grant identify 200 ppb as their reference value.

The maximum concentrations detected in Bristol ambient air samples (0.11 ppb from a 24-hour TO-15 sample; 0.37 ppb from a short-term grab sample) are below all of the health-based non-cancer screening values identified above. Harmful non-cancer health effects are not expected from inhalation exposures to 1,3-butadiene at the levels detected in the Bristol area.

The EPA has identified an IUR for 1,3-butadiene based on leukemia incidence data (EPA 2002). To calculate the cancer risk from chronic exposure to 1,3-butadiene at the maximum level detected in the data set, the IUR ($0.00003 \text{ } (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (0.11 ppb, which equals $0.23 \text{ } \mu\text{g}/\text{m}^3$).

Based on twenty-five years of exposure to the maximum 1,3-butadiene ambient air concentration, approximately two excess cancers are expected in every 1,000,000 individuals exposed ($2.2\text{E-}06$). ATSDR does not find this cancer risk to be of concern. **Inhalation exposures to the detected levels of 1,3-butadiene in the Bristol area are not expected to result in harmful non-cancer health effects and the cancer risk is considered low.**

5.2.2.10 1,4-Dioxane Evaluation

1,4-Dioxane was detected four times in Bristol air samples. Three samples with 1,4-dioxane were collected on January 25, 2022, from Locations 8, 9 and 10, with concentrations ranging from 0.2 to 0.4 ppb. 1,4-Dioxane was detected only one other time, at Location 9 on April 12, 2022 at 0.2 ppb. None of the detections of 1,4-dioxane exceed ATSDR non-cancer health-based screening values (i.e., ATSDR chronic EMEG of 30 ppb); however, each detection exceeded the CREG of 0.055 ppb. Based on the few detections of 1,4-dioxane in the overall data set, the use of the maximum concentration for chronic exposure assessment may overestimate the cancer risk from 1,4-dioxane exposure in the Bristol area.

The EPA has identified an IUR for 1,4-dioxane based on the combined tumor incidence in male rats exposed via inhalation for 2 years (EPA 2013). To calculate the cancer risk from chronic exposure to 1,4-dioxane at the maximum level detected in the data set, the IUR ($0.000005 \text{ } (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (0.4 ppb, which equals $2.0 \text{ } \mu\text{g}/\text{m}^3$) to produce a cancer risk of three in 1,000,000 individuals exposed daily for twenty-five years ($3.2\text{E-}06$). ATSDR does not find this cancer risk to be of concern. Past assessments of indoor air in the U.S. observed

typical 1,4-dioxane concentrations 10 times higher than outdoor levels (i.e., 4 ppb) (ATSDR 2012b). Exposures to 1,4-dioxane in typical indoor air would result in higher cancer risks than those identified from exposures to 1,4-dioxane in Bristol ambient air. **Inhalation exposures to the detected levels of 1,4-dioxane in Bristol ambient air are not expected to result in harmful non-cancer health effects and the cancer risk is considered low.**

5.2.2.11 Carbon Tetrachloride Evaluation

The maximum carbon tetrachloride concentration detected in the Bristol data set (0.1 ppb) is below typical background ambient air levels found in U.S. urban environments and is below the lowest non-cancer health-based screening level of 16 ppb (EPA RfC). Harmful non-cancer health effects are not expected from exposure to the detected levels of carbon tetrachloride in Bristol ambient air.

The maximum non-instantaneous sample results (0.04 to 0.08 ppb range) exceed the ATSDR CREG of 0.026 ppb. EPA has identified an IUR for carbon tetrachloride of 6.0×10^{-6} per $\mu\text{g}/\text{m}^3$ based on the potential for tumor development in the endocrine system, i.e., pheochromocytoma (EPA 2010a). To calculate the cancer risk from chronic exposure to carbon tetrachloride in the Bristol area, the IUR ($0.000006 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (0.08 ppb, which equals $0.51 \mu\text{g}/\text{m}^3$).

Based on twenty-five years of exposure to the chronic carbon tetrachloride EPC of 0.08 ppb, approximately one excess cancer is expected in every 1,000,000 individuals exposed ($9.8\text{E}-07$). ATSDR does not find this cancer risk to be of concern. **Harmful non-cancer health effects are not expected from exposures to carbon tetrachloride at the concentrations detected in Bristol area ambient air and the cancer risk is considered low.**

5.2.2.12 Trichloroethylene (TCE) Evaluation

The maximum detected community-located TCE ambient air concentration of 0.19 ppb was collected for the Bristol Solid Waste Management Facility in April 2021. The majority of samples did not have detectable TCE in ambient air (5 out of 8 grab samples and 30 out of 43 non-instantaneous samples were non-detect). TCE was detected in ambient air, ranging from 0.01 to 0.19 ppb, at all locations except for Locations 6 and 9.

Community-located samples (0.01 to 0.19 ppb) did not exceed the ATSDR non-cancer health-based screening level of 0.4 ppb. Based on the available data, harmful non-cancer health effects from TCE exposures in the Bristol area are not expected.

Seven non-instantaneous samples (range of 0.05 to 0.14 ppb) had TCE concentrations that exceed ATSDR's CREG of 0.04 ppb. Cancer risk estimates are calculated below for the maximum sample concentrations.

The EPA has identified an IUR for TCE of 4.1×10^{-6} per $\mu\text{g}/\text{m}^3$ based on development of renal cell carcinomas, non-Hodgkin's lymphoma, and liver tumors (EPA 2011). To calculate the cancer risk from chronic exposure to TCE in the Bristol area, the IUR ($0.0000041 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC of 0.14 ppb, which equals $0.77 \mu\text{g}/\text{m}^3$. Based on twenty-five years of exposure to the maximum detected TCE concentrations of 0.14 ppb, one to two excess cancers are expected in every 1,000,000 individuals exposed ($1.4\text{E}-06$). ATSDR does not find this cancer risk to be of concern. **Harmful non-cancer health effects are not expected from exposures to TCE in the Bristol area ambient air and the cancer risk is considered low.**

One grab sample from the landfill property had a TCE concentration (1.27 ppb) exceeding the non-cancer screening value of 0.4 ppb. While worker exposures are expected to occur over less time per week than for residential exposures, further assessment of occupational exposures for TCE and other detected contaminants at the Landfill is recommended.

5.2.2.13 1,2-Dichloroethane (1,2-DCA) Evaluation

The maximum 1,2-DCA concentration detected (0.03 ppb) is below the ATSDR non-cancer screening value of 600 ppb; therefore, non-cancer harmful health effects are not expected. 1,2-DCA was rarely detected, and it was only detected at three of the 10 locations where non-instantaneous sampling was conducted (Locations 1, 6, and 7).

NTP has determined that 1,2-DCA may reasonably be anticipated to be a human carcinogen (NTP 1991). IARC considers 1,2-DCA as possibly carcinogenic to humans, and the EPA concludes 1,2-DCA is a probable human carcinogen (ATSDR 2022). To calculate the cancer risk, the IUR ($0.000026 (\mu\text{g}/\text{m}^3)^{-1}$) is multiplied by the chronic EPC (0.03 ppb, which equals $0.12 \mu\text{g}/\text{m}^3$).

Based on twenty-five years of exposure to the maximum 1,2-DCA concentration, approximately one excess cancer is estimated in every 1,000,000 individuals exposed over twenty-five years (1.0E-06). This cancer risk is lower than estimated cancer risks from exposures to background levels of 1,2-DCA in the U.S. ATSDR therefore does not find this cancer risk to be of concern.

Inhalation exposures to the detected levels of 1,2-DCA in the Bristol area are not expected to result in harmful non-cancer health effects and the cancer risk is considered low.

Health Implications: COCs from Continuous Monitoring Data

EPA conducted continuous air monitoring for ammonia, H₂S, SO₂, total volatile organic compounds (VOCs), and methyl mercaptan. Continuous monitoring was conducted with multiple instruments, each with different lower limits of detection, quality assurance and accuracy. Honeywell Analytics single point monitor (SPM) Flex units were used to determine H₂S, SO₂, and ammonia concentrations in ambient air. This continuous monitoring data consists of 3-second samples in two stages of 6-weeks and 2-weeks, with rate of non-detections ranging from 57% to 100%, depending on contaminant. Due to the large volume of data, ATSDR calculated a series of discrete averages from the raw data for each contaminant to make the data easier to interpret. These discrete averages used an exposure duration that was relevant to the toxicological literature.

5.2.2.14 Ammonia Evaluation

Ammonia is corrosive and can burn the skin, eyes, respiratory tract, mouth, and digestive tract upon direct contact with high enough concentrations of the substance (ATSDR 2004). ATSDR calculated 10-minute discrete averages and subsequently calculated 2-hour rolling averages to compare to the appropriate toxicological literature. The maximum 2-hour average ammonia concentrations detected in Bristol ambient (i.e., outdoor) air occurred at Locations 1 and 5, with maximum short term (2-hour) peak concentrations around 412 ppb. The acute screening value of 1,700 ppb, where corrosive health effects from direct contact may occur, was not exceeded. ATSDR also calculated the mean concentration over the sampling time frame. However, the brief time periods of exposure could not accurately represent chronic exposures.

Table 6. Ammonia Concentrations and Percent of Time Not Detected*

Location [†]	Percent Non-Detect	# of 2-hour Averages	Maximum 2-hour Average (ppb)	Acute MRL (ppb)
1 [‡]	91.1	2,846	104	1700
5 [‡]	99.7	868	412	1700
7	98.9	1,262	19.4	1700
8	86.4	1,261	22.2	1700
9	99.4	893	32.1	1700

Notes:

* = ammonia lower detection limit of 10 ppb;

† = locations 2, 3, 4, and 6 were non-detect for the entire duration of air monitoring;

‡ = Location 1 and 5 were located on same property within 250' of each other; ppb = parts per billion

Exposure to ammonia at the detected concentrations are not expected to result in harmful health effects. The concentration at which most people can begin to smell the pungent odor of ammonia in the air is 46,800 ppb (Leonardos et al. 1969), though some sensitive individuals may start to smell ammonia at concentrations above 43 ppb (AIHA 1989).

5.2.2.15 Hydrogen Sulfide (H₂S)

H₂S is an irritant that affects the eyes, nose, and throat by acting as a reducing agent. Once inhaled, H₂S will enter the bloodstream by diffusion in the lungs and can cause neurological effects such as headaches and fatigue. H₂S is oxidized in the liver before being excreted in urine. People most sensitive to the harmful effects of H₂S exposure include the very young, the elderly and those with pre-existing respiratory conditions, such as asthma and restrictive lung disease. Individuals with cardiac and nervous system conditions are also more susceptible to harmful health effects from H₂S exposures (ATSDR 2016). Most people can smell H₂S as low as 0.47 to 10 ppb.

The maximum concentrations of H₂S detected in Bristol area monitors ranged from non-detect (i.e., less than 1 ppb) to 8.53 ppb (see [Table 7](#)). ATSDR calculated 5-minute discrete averages before creating 30-minute rolling averages. To assess acute and intermediate exposures, ATSDR used the maximum 30-minute H₂S concentrations from each location. The maximum 30-minute H₂S concentration of 33.3 ppb was detected at Location 5. The maximum 30-minute concentration and all other 30-minute maximum H₂S concentrations were below the acute (70 ppb) and intermediate (20 ppb) screening values.

Table 7. Continuous Monitoring Data and Screening Values for Hydrogen Sulfide (H₂S)

Location	Percent Non-Detect	Number of 30-minute Averages	Maximum 30-minute Average (ppb)	Acute MRL
1	96.3	11,793	6.2	70 ppb
2	95.2	624	2.3	70 ppb
3	98.0	6,155	2.4	70 ppb
4	94.5	6,026	2.2	70 ppb
5	100	5,556	33.3	70 ppb
6	99.8	2,646	2.1	70 ppb
7	100	2,531	N/A	70 ppb
8	91.3	2,534	6.57	70 ppb
9	88.7	1,822	8.53	70 ppb

Notes: ppb = parts per billion, <1 = less than 1 parts per billion, RMEG = ATSDR environmental media evaluation guideline based on the EPA reference dose (RfD)

Based on the continuous monitoring data set, H₂S odors are expected to have been observed for short periods of time at all of the monitored locations, except near Location 7, where H₂S was not detected during the monitoring period. Though some individuals may not be sensitive to these odors, others may experience health symptoms (e.g., nausea, headache) that adversely affect their quality of life due to these foul odor events. For a majority of the sampling period (89% or more of the time period monitored), H₂S was not detected at any of the 9 monitoring locations (see [Table 7](#) above). **Exposures to H₂S in ambient air are not expected to result in harmful health effects; however, intermittent foul-smelling H₂S odor events have occurred in most of the areas monitored in Bristol.**

5.2.2.16 Methyl Mercaptan

Methyl mercaptan is a naturally-occurring, colorless gas that smells like rotten cabbage (ATSDR 1992). It is released from decaying organic matter, such as from marshes and as a decay product of wood in pulp mills. Little is known about the health effects of exposure to methyl mercaptan. We do not know if long-term exposure to low levels results in harmful effects. Methyl mercaptan can be smelled in air at about 1.6 ppb, and at much lower levels when present in water (ATSDR 1992). ATSDR and EPA have not set health-based screening levels for methyl mercaptan. Background levels of methyl mercaptan in U.S. air have been measured up to 4 ppb. The Texas Commission on Environmental Quality (TCEQ) has set an effects screening level of 0.5 ppb for

methyl mercaptan, based on occupational exposure standards set at 500 ppb for workers (TCEQ 2022).

EPA monitored Bristol community air for methyl mercaptan at Locations 7, 8 and 9 in October 2021. Average methyl mercaptan concentrations were 38.7, 49.5, and 6.5 ppb for Locations 7, 8, and 9, respectively. Methyl mercaptan was detected almost 60% of the monitoring time period at Location 8. Methyl mercaptan was rarely detected at Locations 7 and 9 (detected 7.3% and 6.5% of the time, respectively). Because the monitoring equipment cannot detect below 10 ppb, it is not known if methyl mercaptan concentrations below 10 ppb were present for longer periods of time during the monitoring period.

Due to limitations in the toxicology and epidemiology database, ATSDR cannot draw any conclusions regarding whether health effects may occur from inhalation exposure to methyl mercaptan in the Bristol area. Monitoring results show that methyl mercaptan concentrations were above typical background levels in the U.S. and above the TCEQ ESL of 0.5 ppb; however, concentrations never approached the occupational action levels of 500 ppb. **ATSDR concludes that nuisance odors are likely to impact residents that are sensitive to these odors and some individuals may experience health symptoms (i.e., headaches, nausea, lethargy), but ATSDR cannot determine whether methyl mercaptan exposures at the detected levels may result in harmful health effects.**

5.2.2.17 Sulfur Dioxide (SO₂)

SO₂, a colorless gas with a pungent odor, is released to the air primarily from burning fossil fuels (i.e., coal, oil). Short-term exposures to high concentrations of SO₂ (e.g., 100,000 ppb) is immediately dangerous to life and health due to the chemical's effects on the respiratory tract, including burning of the nose and throat, breathing difficulties, and severe airway obstruction (ATSDR 1998). Both short and long-term exposure to SO₂ may result in reduced lung function. There is limited information on the health effects of long-term exposure to low concentrations of SO₂. One worker study found that long term exposure to SO₂ at 0.4-3 ppm for 20 years or more resulted in lung function changes, though other chemical exposures occurred at the same time making it difficult to attribute health effects solely to SO₂ (ATSDR 1998). Shorter duration exposure assessments found that sensitive subpopulations include exercising asthmatics, who may experience health effects from SO₂ exposure at levels as low as 250 ppb. For comparison, typical outdoor levels of SO₂ range from non-detect up to 1,000 ppb.

The World Health Organization (WHO 2021) has identified 187 ppb as their SO₂ air quality guideline where health effects are not expected following up to 10 minutes of exposure at or below this concentration. The EPA National Ambient Air Quality Standard (NAAQS) has set its health-based 1-hour maximum standard at 75 ppb. The EPA NAAQS standard of 75 ppb was retained following EPA review in 2019 to provide protection to sensitive individuals (i.e., people with asthma) exposed to SO₂ for 5 to 10 minutes while breathing at elevated rates (EPA 2019). ATSDR has identified 10 ppb as the acute-exposure minimal risk level (aMRL) where individuals may be exposed continuously for up to 2 weeks without experiencing harmful health effects. The aMRL is based on a small study of adults with asthma exposed to SO₂ before and during moderate exercise. The lowest observed adverse health effects (i.e., bronchoconstriction, or specific airway resistance) in this study of adult human subjects were observed at 100 and 250 ppb SO₂ exposure concentrations. To identify the aMRL, this minimal LOAEL of 100 ppb was divided by an uncertainty factor of 3 for human variability and by 3 for the use of a minimal LOAEL. The use of 3 for human variability is included to account for the sensitivity of asthmatics and for the possibly increased sensitivity of children to SO₂ exposures.

ATSDR assessed the SO₂ data set and found a large proportion of the data collected by continuous monitoring was below the instrument’s limit of detection (100 ppb). However, even when assuming data logged as non-detect equaled zero ppb, average SO₂ concentrations still exceeded the ATSDR acute screening value of 10 ppb. [Table 8](#) provides a range of results for each location based on the use of zero and the limit of detection (100 ppb) for averaging with such a high proportion of “non-detect” values.

ATSDR calculated 1-minute average SO₂ concentrations before calculating 5-minute averages. These analyses showed the following averages, using the limit of detection of 100 ppb for non-detects:

- Location 7 maximum 5-minute average of 100 to 160 ppb;
- Location 8 maximum 5-minute average of 100 to 103 ppb; and,
- Location 9 maximum 5-minute average of 100 to 105 ppb.

Table 8. Continuous Monitoring Data and Screening Values for Sulfur Dioxide (SO₂)

Location	Percent Non-detect*	# of 5-minute Averages	Maximum 5-minute Average (ppb)	Acute MRL (in ppb)
7	60.4	12,665	160	10
8	74.6	12,684	103	10
9	96.4	9,155	105	10

Notes: ppb = parts per billion; aEMEG = ATSDR acute environmental media evaluation guideline; CV = health-based comparison value;

* =Instrument minimum limit of detection was 100 ppb

Due to the monitoring equipment's high detection limit of 100 ppb and technical challenges with laboratory analysis of sulfur compounds, including SO₂, it was not possible to determine whether SO₂ was present in ambient air at less than 100 ppb. For this reason, ATSDR presents the results in a range and assumes that average SO₂ concentrations may have been higher than those detected. For this reason, ATSDR concludes that SO₂ concentrations may have exceeded ATSDR's acute screening value of 10 ppb for more of the time than reported.

It is noted in Sheppard et al. (1981) that adult subjects exposed to SO₂ for 5 minutes at levels between 100 and 250 ppb experienced bronchoconstriction. Maximum 5-minute SO₂ levels in Bristol exceeded 100 ppb (see [Table 8](#)). Because the Sheppard et al. (1981) study did not include children, and because children may be more sensitive to SO₂, harmful health effects may occur following exposures to SO₂ at the levels detected in areas to the south (Location 7) and southwest (Locations 8 and 9) of the landfill.

Due to the high detection limits for SO₂ resulting in a possibly low bias of average SO₂ concentrations, and the exceedance of the ATSDR acute screening value at two of the three monitoring locations, ATSDR concludes that harmful health effects from short-term SO₂ inhalation exposures are possible.

5.2.2.18 Cancer Risk Assessment from Combined Chemical Exposures

To determine the cumulative cancer risks from inhalation exposures to a mixture of chemicals in ambient air, ATSDR follows a response-additive approach of adding cancer risks (i.e., individual cancer risks are summed). [Table 9](#) provides this combined cancer risk screening information. Contaminant concentration data from the three locations with the highest benzene levels, 8,9,10, all located southwest of the landfill, were combined and the 95UCL of that data was used to determine cancer risks for each contaminant, as described for benzene above, and then summed. This approach resulted in an estimated cancer risk for those three locations that approached the cancer risk range of less than one in 10,000. The maximum contaminant concentrations were used to estimate the cumulative cancer risk for the other locations. Locations 1-4 and 7 also had estimated cancer risks higher than one in 100,000, while Locations 5 and 6 had cancer risks of between one in a million and one in 100,000.

Overall, the combined cancer risk to the community from contaminants associated with the landfill is slightly elevated over background cancer risks. This represented a public health concern before the recent steps taken to mitigate emissions from the Landfill.

Table 9. Combined Excess Cancer Risk by Location

Location	Cumulative Cancer Risk
Location 1	5.5E-05
Location 2	6.2E-05
Location 3	1.7E-05
Location 4	3.5E-05
Location 5	3.1E-06
Location 6	7.2E-06
Location 7	3.5E-05
Location 8	8.3E-05
Location 9	8.3E-05
Location 10	8.3E-05

5.3 Addressing Community Concerns

Between December 2020 and January 2022, more than 11,000 individual odor concerns were recorded by Bristol area residents on the SmellMyCity.org application². Over 70% of these odor reports were logged from three Tennessee zip codes located south and southwest of the Landfill. Residents commonly characterize the odors in general terms, including “landfill/garbage/trash”, “rotten” or “rotting”, “chemical”, “burnt/burning”, “sour”, “egg”, and “sulfur” smells. Many Bristol area residents have reported upper respiratory tract and eye irritation, along with other acute symptoms that they associate with exposure to the odors they detect in ambient air. Additional symptoms have included itchy and dry nose, headaches and dizziness, nosebleeds, swollen glands, sore and burning throat, dry mouth, nausea, burning eyes, congestion, fatigue, chest tightness, anxiety, and depression. Some residents have reported pets also being affected, with some residents’ dogs vomiting during malodor events. Residents have noted that health

² Reported on Smell My City.org website for six Bristol area zip codes (24201, 24202, 37617, 37618, 37620) from January 1, 2021 through February 1, 2022

effects, particularly in their children, are severe (e.g., nose bleeds), and have resulted in visits to emergency departments for medical assistance.

Many of the symptoms listed by community members can be caused by short-term (i.e., acute duration on the order of minutes to hours) exposure to chemicals that cause irritation as soon as the chemical makes contact with people (i.e., skin, respiratory tract, eyes). Other symptoms reported by residents (e.g., headaches, nausea, lethargy) may be associated with exposure or the stress of living with such a strong and oppressive malodor. The combination of low-dose exposures to odorous/irritant chemicals and the stress from daily living with noxious odors can lead to harmful health impacts in some individuals.

In this section, irritant and odorous chemicals tentatively identified in TO-15 samples and from continuous monitoring are presented. Many chemicals identified by TO-15 sampling are qualified by the laboratory as “tentatively-identified compounds,” or TICs. TICs are those chemicals for which a laboratory standard was not used to confirm the presence or concentration of the chemical because it was not one of the chemicals targeted for analysis. Therefore, TICs and the concentration of the TICs in ambient air cannot be confirmed without additional sampling or laboratory analysis to validate the result. With no associated concentrations, ATSDR is not able to draw conclusions regarding the health effects that may result from exposures to these pollutants.

Each of the continuously monitored chemicals (H_2S , ammonia, methyl mercaptan, and SO_2) are considered irritants. Our assessment of acute and chronic exposures to the chemicals detected in ambient air above our screening values was provided earlier in this document. Briefly, ATSDR concludes that acute exposures to SO_2 may result in harmful health effects for some individuals, including children and those with asthma and other respiratory conditions. There is also a low theoretical risk of harmful non-cancer health effects for some individuals following chronic exposures to the maximum detected concentrations of benzene, though health effects at these benzene exposure concentrations have not been observed in past studies. There is insufficient information to draw conclusions about the health effects of methyl mercaptan exposures in the Bristol area. Non-cancer health effects from exposures to all other monitored and sampled chemicals are not expected.

Given the number of reported odor events, the reports of irritation when odor events are occurring, and the list of odorous and irritant chemicals, including multiple TICs, that were identified in multiple samples, ATSDR cannot rule out the potential for exposures that could result in harmful health effects in the past, present, or future. Because of these uncertainties,

ATSDR provides a discussion below of exposures to irritants and odorous chemicals detected in ambient air for the Bristol community.

Exposure to irritant chemicals may cause some individuals to have a physical reaction when exposed, even at exposure concentrations where most people will not have an adverse reaction. This is known as chemical sensitivity or chemical intolerance. Exposures to combinations of irritant chemicals may also lead to symptoms in some individuals. Due to the inherent temporal and spatial limitations of air sampling, there may have been short time periods in some areas of Bristol where irritant concentrations may have been high enough to result in health effects for some individuals; though these effects are typically transient and will resolve once the individual is removed from exposure. With the available data, ATSDR cannot rule out irritant effects for some individuals in the affected Bristol communities.

There are two types of environmental data that ATSDR has available for this assessment that could be relevant to residents' odor concerns: chemicals with ambient concentrations, and, chemicals that were tentatively identified in ambient air, but no associated concentration is available (i.e., TICs).

5.3.1 Chemicals with ambient concentrations

The assessment to determine whether exposure to chemicals with ambient air concentrations will result in harmful health effects was presented earlier in this document.

Because TO-15 samples were typically collected for a period of 8 to 30 hours, the potential for shorter duration exposures to higher concentrations than those detected during extended sample collection time periods cannot be ruled out. This data limitation is noted for all data collected by Method TO-15. While the maximum short-term exposure concentration for COCs is not known, six of the COCs identified by method TO-15 analysis are known to cause respiratory irritation (1,2-dichloroethane, 1,3-butadiene, carbon tetrachloride, chloroform, hexachlorobutadiene, and TCE)

5.3.2 TICs

Many chemicals were tentatively identified in the samples collected by the VADEQ in 2021. Some of the TICs identified in the data set are common landfill gas emissions, including aldehydes. Aldehydes were identified in at least seventeen samples as TICs in TO-15 samples ([Table 10](#)).

Some of the TICs listed in sampling results are malodorous, and some are known to cause respiratory, skin, and eye irritation. Dimethyl sulfide, with its disagreeable cabbage-like smell at low concentrations, was identified in 11 samples. One aldehyde, 2-butenal, is a sensitizer. Sensitizers are defined by the Occupational Safety and Health Agency (OSHA) as chemicals that cause a substantial proportion of exposed people or animals to develop an allergic reaction in normal tissue after repeated exposure to the chemical. Multiple TICs that are known irritants were detected in the same samples, suggesting periods of time in Bristol when plumes of irritant chemicals were present.

Table 10. Aldehydes Tentatively Identified in Community-Located Air Samples

Chemical	Common name	Irritant?	Irritant targets	Characteristic Odor
2-butenal*	Crotonaldehyde	Yes	Eyes, respiratory tract	Pungent, suffocating
Ethanal	Acetaldehyde	Yes	Eyes, respiratory tract, skin	Strong, fruity
2-methyl-propanal	Isobutyraldehyde	Yes	Throat	Pungent, floral
Butanal	Butyraldehyde	Yes	Eyes, respiratory tract, skin	Pungent
Hexanal	Hexanaldehyde	Yes	possible: eye, skin	Cut grass, fruity

Notes:

* = known sensitizer;

TIC = tentatively identified compound.

[Table 11](#) presents the other irritant chemicals identified as TICs in multiple samples of Bristol ambient air in 2021 and 2022.

Table 11. Other Irritant Chemicals Tentatively Identified in Community-Located Air Samples

Chemical	Number of Times Detected	Irritant?	Irritant targets	Characteristic Odor
Ethanol	20	Yes, at high levels	Eyes, nose, throat, lungs, skin	Wine-like
Pentane	19	Yes	Nose, throat	Mild gasoline
Acetone	17	Yes	Eyes, nose, throat, lungs	Nail polish
2-Butanone	16	Yes, mild	Eyes, respiratory tract, skin	Sharp, sweet
Alpha Pinene	14	Potential	Nose, throat, lungs	Pine forest
Isopropyl Alcohol	14	Yes	Nose, throat	Rubbing alcohol
Decane	13	Yes	Respiratory tract	Gasoline
Dimethyl Sulfide	11	Yes, strong	Respiratory tract	Cabbage-like

Chemical	Number of Times Detected	Irritant?	Irritant targets	Characteristic Odor
Nonane	8	Yes	Lungs	Gasoline
Octane	7	Yes	Nose, throat, lungs	Gasoline
Isobutane	6	Yes	Nose, throat	None
Furan	5	Yes	Nose, skin, throat, lungs, eyes	Pleasant, ethereal
2-methyl-pentane	5	Yes, mild	Eyes, throat, skin	Gasoline
Undecane	5	Yes	Nose, skin	Mild gasoline
2-methyl-1-propene	4	Yes	Eyes, nose, throat	Mild gasoline
Beta Pinene	2	Yes	Eyes, respiratory tract, skin, mucous membranes	Woody-green, pine
3-methyl-hexane	2	Yes	Eyes, respiratory tract, skin	Mild gasoline

Based on the available data, ATSDR cannot determine whether the concentrations of individual chemicals or combination of those chemicals tentatively identified in ambient air samples are causing acute irritant effects among individuals exposed in the community; however, transient, irritant effects from exposure to some of the TICs identified are plausible for some individuals in the Bristol area.

6. Conclusions

In the following sections, ATSDR provides several important conclusions based on our assessment of the ambient air quality data collected in the impacted Bristol communities near the Quarry Landfill. Based on these conclusions, several recommendations are provided to protect community members from unhealthy exposures. Important information regarding the limitations of the data set is also provided below.

Conclusion 1. Exposures of a year or more (chronic-duration) to benzene at the concentrations measured immediately west and southwest of the landfill may result in harmful non-cancer health effects (e.g., decreased B-cell counts) and increased risk of developing leukemia. Exposures to benzene at the detected levels for less than a year are not expected to result in harmful health effects.

Basis for Conclusion 1

- The estimated maximum benzene chronic (i.e., greater than one year) exposure concentration of 10.2 ppb (based on the 95th upper confidence limit [95UCL] on the mean of all samples from Locations 8, 9, and 10) is three times higher than the chronic Minimal Risk Level (MRL) of 3 ppb.
- The concentration of benzene that studies suggest will cause chronic toxicity to lab animals (30 ppb) is three times the estimated maximum benzene exposure. Considering potential uncertainties in experimental measurement (i.e., the toxicology database), susceptibility, and environmental sampling measurements, a toxicity level only 3 times higher than the estimated exposure concentration raises concerns that exposures may harm people's health.
- Estimated cancer risk from twenty-five years of exposure to benzene at 10.2 ppb is slightly less than 1 in 10,000. ATSDR considers this to be an elevated cancer risk. This calculated excess risk from benzene exposure for the most highly exposed residents raises the potential for a small increase above the already existing risk of cancer from all causes.
- While benzene contributed more to the cancer risk than other contaminants, exposure to other contaminants may have contributed to the overall cancer risk for residents around the Landfill.
- Based on the weight of evidence and estimates of short-term benzene exposures, acute (i.e., up to two weeks) and intermediate (i.e., 2 to 52 weeks) benzene exposures are not expected to result in harmful health effects.

Conclusion 2. Short-term exposures (e.g., 5 minutes) to the detected levels of SO₂ may result in harmful health effects in Bristol communities. SO₂ exposures at the detected concentrations may result in airway resistance, or bronchoconstriction. Bronchoconstriction can make it difficult to breathe for individuals, and especially children, with asthma and other respiratory conditions, particularly when exercising or doing rigorous outdoor activities.

Basis for Conclusion 2

- Maximum 5-minute SO₂ concentrations in Bristol were above 100 ppb. A study of exercising asthmatic adults found SO₂ exposures for 5 minutes at 100 to 250 ppb results in severe bronchoconstriction.
- Given that they breathe more air for their body weight, are more active than adults, and are more likely to have experienced asthma attacks in the last year, children may be more likely to experience harmful health effects from these short term SO₂ exposures (ATSDR 1998; Pate et al. 2021). There is not enough research on SO₂ exposures in children to know whether they are more sensitive than adults.

- The air quality monitoring equipment used to obtain SO₂ levels has a detection limit for SO₂ (100 ppb) that is significantly higher than ATSDR's acute MRL (10 ppb). As a result, it is not possible to determine how often SO₂ concentrations near the landfill were high enough to be a concern, particularly for children and other sensitive individuals.

Conclusion 3. Tentatively-identified compounds (TICs) detected by TO-15 sampling and the measured concentrations of methyl mercaptan support resident concerns regarding nuisance odor events in the Bristol area. These nuisance odor events may cause negative health effects to residents, but the risks for health effects from these odors cannot be calculated using available data. Exposure to foul-smelling odors can result in reduced quality of life and some individuals may experience health symptoms, such as nausea, headaches, and fatigue. These symptoms may be due to the stress of these odor events and/or sensitivity to specific chemicals in the outdoor air. ATSDR cannot determine whether methyl mercaptan or TIC exposures at the detected levels will cause health effects.

Basis for Conclusion 3

- Exposure to foul-smelling odors can result in reduced quality of life and some individuals may experience health symptoms, such as nausea, headaches, and fatigue. These symptoms may be due to the stress of these odor events and/or sensitivity to specific chemicals in the outdoor air.
- Methyl mercaptan was present at concentrations that could contribute to the perception of foul-smelling odors by residents and could thus contribute to the resulting symptoms associated with those odors. However, ATSDR is unable to identify toxicological data to suggest direct physical health effects from exposures to methyl mercaptan at these concentrations.
- Multiple aldehydes, including 2-butenal (a sensitizer), were TICs in multiple samples collected in 2021 and 2022. Aldehydes can cause irritation of the eyes and respiratory tract upon contact.
- More than 15 other known irritants were TICs in multiple samples collected in 2021 and 2022 in Bristol community locations. These irritants are known to affect the nose, throat, eyes, lungs, and skin upon contact.
- The concentrations of TICs could not be determined, and many of the compounds lack scientific studies on their effects. Therefore, the potential for health effects from these compounds could not be assessed.

Conclusion 4. Other than benzene, methyl mercaptan and SO₂, inhalation exposures to chemicals sampled and measured are not expected to result in non-cancer health effects.

Basis for Conclusion 4

- Except for benzene, SO₂, and methyl mercaptan, sampled and measured chemicals, including those listed as potential COCs (1,2-dibromoethane, 1,2-dichloroethane, carbon tetrachloride, chloroform, hexachlorobutadiene, TCE, ammonia, and H₂S), did not exceed their respective non-cancer health-based screening levels. Maximum concentrations below non-cancer, health-based screening levels are not expected to result in harmful health effects for any individuals, including children and other vulnerable subpopulations.
- Upon close review and based on additional sampling activities conducted in 2022, ATSDR concluded that the acrolein results collected prior to 2022 were not representative of the sampled ambient air and could not be used for health assessment. Additional steps beyond the standard field and laboratory method are required to ensure the representativeness of acrolein data gathered by EPA Method TO-15. VADEQ and EPA took additional acrolein samples that represented outdoor air in Bristol in 2022. These verified data showed acrolein concentrations below levels of public health concern.

DATA LIMITATIONS

From December 2020 through April 2022, ambient air samples were collected in Bristol. These samples provide some understanding of the contaminants that are present in outdoor air. However, it is not practical to gather enough data to characterize exposures in all areas of Bristol for all times of the day and year and for multiple years. The following list provides the primary data limitations recognized for this environmental health assessment:

- Intermediate and chronic exposure concentrations remain uncertain in Bristol areas. Over sixty time-weighted samples were collected for volatile organic compounds analyses and continuous air monitoring was conducted for more than six weeks. However, seasonal weather variables, daily weather fluctuations, such as temperature inversions, and activities conducted at source locations and close to the sampling equipment will impact ambient air concentrations. Although these days of variability may not have been captured by sampling and monitoring, ATSDR's approach is considered health protective because ATSDR used the maximum detected concentrations in the data set when there are insufficient data to better determine exposure concentrations over time. By using this health protective approach, ATSDR's conclusions may overestimate the risk of harmful effects from intermediate and chronic exposures.

- Exposures to chemicals that were not assessed are of unknown public health significance. Some common landfill gas chemicals, such as amines, aldehydes, and reduced sulfur compounds, were not targeted for assessment in the Bristol area. These classes of chemicals can present challenges to both field and laboratory personnel not accustomed to these methods and the data produced by the available methods may not be reliable or representative of actual exposures.
- Continuous monitoring technology has detection limits higher than some health-based screening levels, including for ammonia, SO₂ and methyl mercaptan. These chemicals were detected from intermittent peak concentrations above the instrument's limit of detection, but the average concentrations of these chemicals over time are unknown in Bristol ambient air.
- The TICs correspond to signals for which the lab did not have standards available. Therefore, we cannot be sure of their identity and cannot quantify how much of these compounds were present.
- Some areas of Bristol were not assessed by ambient air monitoring, and it is not certain what the airborne chemical concentrations were in these areas. However, based on our understanding of prevailing local wind patterns and by using geographic information from citizen's reported odor complaints over several years, ATSDR believes the communities that are most impacted by the Landfill's emissions were assessed for air quality.
- Non-landfill sources of emissions were noted but could not be quantified, such as emissions from a nearby bus depot, a major recycling processor, lawn equipment, etc.
- ATSDR assumed that exposure durations were twenty-five years for a maximum, health-protective approach. The landfill was open and actively accepting waste from 1998 to 2022 (approximately 24 years). Furthermore, a sidewall odor mitigation system was installed in 2023 that was expected to further reduce contaminant levels, as were additional steps taken to mitigate exposure since the data were collected. ATSDR selected twenty-five years, the time between when the landfill started accepting waste and the installation of the sidewall odor mitigation system as a conservative estimate of how long residents may have been exposed to the measured concentrations.

7. Recommendations and Public Health Action Plan

ATSDR recommends future monitoring of ambient air to determine whether actions being taken at emission sources are reducing community exposures to benzene and sulfur dioxide. Specifically, monitoring is recommended near Locations 8, 9 and 10 (See Appendix B).

Although the landfill has closed and stopped accepting all waste since ATSDR accepted this petition, there may still be a potential for exposures. As such, ATSDR has several recommendations to improve air quality surrounding the landfill. ATSDR recommends that the landfill operators notify the community of onsite activities that are expected to result in increased emissions. ATSDR recommends that the landfill operators continue to investigate the onsite engineering controls and improve them as needed, potentially including the following:

- continuing to enhance the landfill gas collection system with improved piping and well installations that collect more vapors/emissions,
- improving the leachate control and collection system, and other systems where odorous compounds are most often produced,
- addressing issues with the liner, including along the edges of the landfill where landfill gas and vapors may be released.

ATSDR encourages VADEQ and EPA continue to closely oversee landfill activities, enforce landfill permit regulations, and make publicly available the response and oversight activities they have conducted at the landfill and the solutions being implemented to resolve ongoing issues.

ATSDR recommends VADEQ, the City of Bristol, and local/state health agencies conduct timely responses to nuisance odor complaints and that VADEQ or the City of Bristol consider maintaining and posting an odor complaint log that documents the frequency, duration, and characteristics of odor complaints, the weather conditions (e.g., wind speed and direction) at the time of the complaint, and the location of the complaints reported. These data can be utilized by the landfill to improve their operations and reduce/adjust activities that result in odor events.

ATSDR recommends community members continue to communicate with their local and state agencies regarding odorous events and exposure concerns, including reporting nuisance odor events that may originate from the landfill. ATSDR recommends community members that are sensitive to some of the air contaminants detected in the community take steps to reduce their exposures during noxious odor events. These steps might include closing windows and staying inside the home until the odorous event concludes; and installing and safely operating air filtration units in the home to reduce indoor air levels of noxious odors.

ATSDR **does not** recommend the use of ozone generating air filtration/treatment systems in residential buildings.

ATSDR will, upon request, continue to provide technical assistance to state and federal agencies regarding monitoring and assessment of community exposures related to air quality in the Bristol area. ATSDR will make this report public on its website and will distribute the document to interested parties, including residents, and state, federal, and local health and environmental agencies. ATSDR will conduct public outreach, including presenting the findings and recommendations of this document in a public meeting. ATSDR will continue to be available to answer questions and provide technical support to community members regarding ambient air quality concerns in the Bristol area related to landfill emissions.

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Appendices

Appendix A: Brief Summary of ATSDR's Public Health Assessment (PHA) Process

ATSDR follows the PHA process to find out:

- Whether people living near a hazardous waste site are being exposed to toxic substances.
- Whether that exposure is harmful.
- What must be done to stop or reduce exposure.

The PHA process is a step-by-step consistent approach during which ATSDR:

- Establishes communication mechanisms, including [engaging communities](#) at the beginning of site activities and involves them throughout the process to respond to their health concerns.
- Collects many different kinds of [site information](#).
- Obtains, compiles, and evaluates the usability and quality of environmental and biological [sampling data](#) (and sometimes modeling data) to examine environmental contamination at a site.
- Conducts four main, sequential scientific evaluations.
 - [Exposure pathways evaluation](#) to identify past, present, and future site-specific exposure situations, and categorize them as completed, potential, or eliminated.
 - [Screening analysis](#) to compare the available sampling data to media-specific environmental screening levels (ATSDR comparison values [CVs] and non-ATSDR screening levels). This identifies potential COCs that require further evaluation for completed and potential exposure pathways.
 - [EPCs and exposure calculations](#) for contaminants flagged as requiring further evaluation in completed and potential exposure pathways. It involves calculating EPCs, using the estimated EPCs to perform exposure calculations, and determining which site-specific scenarios requires an in-depth toxicological effects analysis.
 - [In-depth toxicological effects evaluation](#), if necessary, based on the three previous scientific evaluations. This step looks more closely at contaminant-specific information in the context of site exposures. This evaluation can also help determine if there is a potential for non-cancer or cancer health effects.
- Summarizes findings and next steps, while acknowledging uncertainties and limitations.
- Provides recommendations to site-related entities, partner agencies, and communities to prevent and minimize harmful exposures.

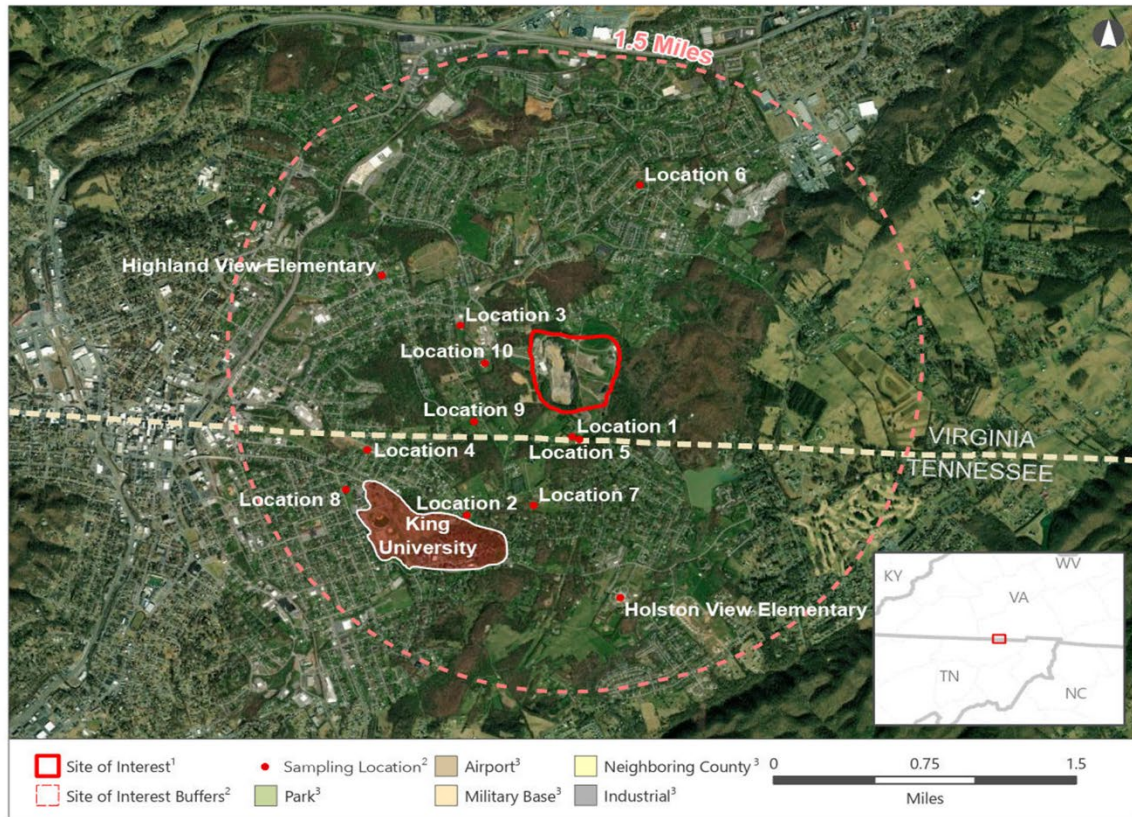
The sequence of steps can differ based on site-specific factors. For instance, health assessors might define an exposure unit before or after the screening analysis.

For more detail on the PHA process, please visit [Explanation of ATSDR's PHA Process Evaluation](#). Readers can also refer to [ATSDR's Public Health Assessment Guidance Manual](#) for all information related to the step-wise PHA process.

Appendix B. Maps and Figures

Figure 1. Bristol Quarry Landfill Location Map (Demographic Snapshot)

Bristol Landfill Odors and Air Qual. Assess.
Bristol VA and Bristol, TN



Demographic Statistics^{4,5}

Within 1.5 Miles buffer of site boundary

Measure	2010	2020	Change	Measure	2010	2020	Change
Total Population	14,328	14,203	+0%	Two or More Races	258	713	+176%
White Alone	12,957	12,414	-4%	Hispanic or Latino ⁶	190	286	+50%
Black Alone	892	822	-7%	Children Aged 6 and Younger	1,158	1,117	-3%
Am. Indian & AK Native Alone	45	32	-28%	Adults Aged 65 and Older	2,450	2,827	+15%
Asian Alone	93	111	+19%	Females Aged 15 to 44	2,853	2,593	-9%
Native Hawaiian & Other Pacific Islander Alone	4	2	-50%	Housing Units	6,611	6,778	+2%
Some Other Race Alone	80	103	+28%	Housing Units Pre-1950	1,435	1,079	-24%

Data Sources: ¹ATSDR GRASP, ²ATSDR GRASP, ³TomTom 2021Q3, ⁴US Census 2020 Demographic and Housing Characteristics. **Notes:** ⁵Calculated using area-proportion spatial analysis method, ⁶Individuals identifying origin as Hispanic or Latino may be of any race. **Coordinate System:** NAD 1983 StatePlane Virginia South FIPS 4502 Feet

Wind Roses from the Virginia Highlands Airport

Wind roses are a visual description of the relative frequency of winds from specific directions and speeds at a given location (the center point of the wind rose). The following wind roses are based on the overall wind data from the Virginia Highlands Airport from 2021 through 2022 and for specific seasons of the year over the same time frame. It is important to note that topographical features, such as hills and valleys, buildings, and other surface objects can have significant effects on wind direction and speed, especially in small geographic areas. Because the topography near the Bristol landfill is characterized by hills and valleys generally aligned northeast to southwest, dominant wind patterns may not be best represented by the nearest airport weather stations (e.g., Virginia Highlands Airport).

Figure B- 2. Meteorological Wind Rose of Virginia Highlands Airport (2021-2022)

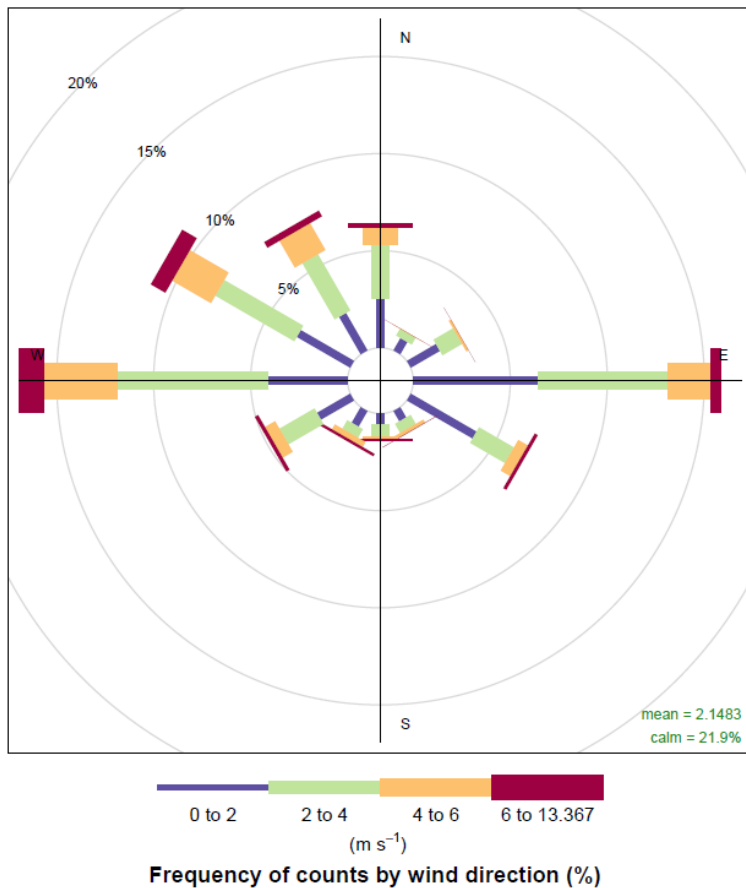
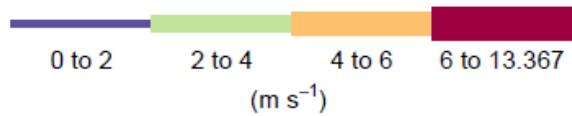
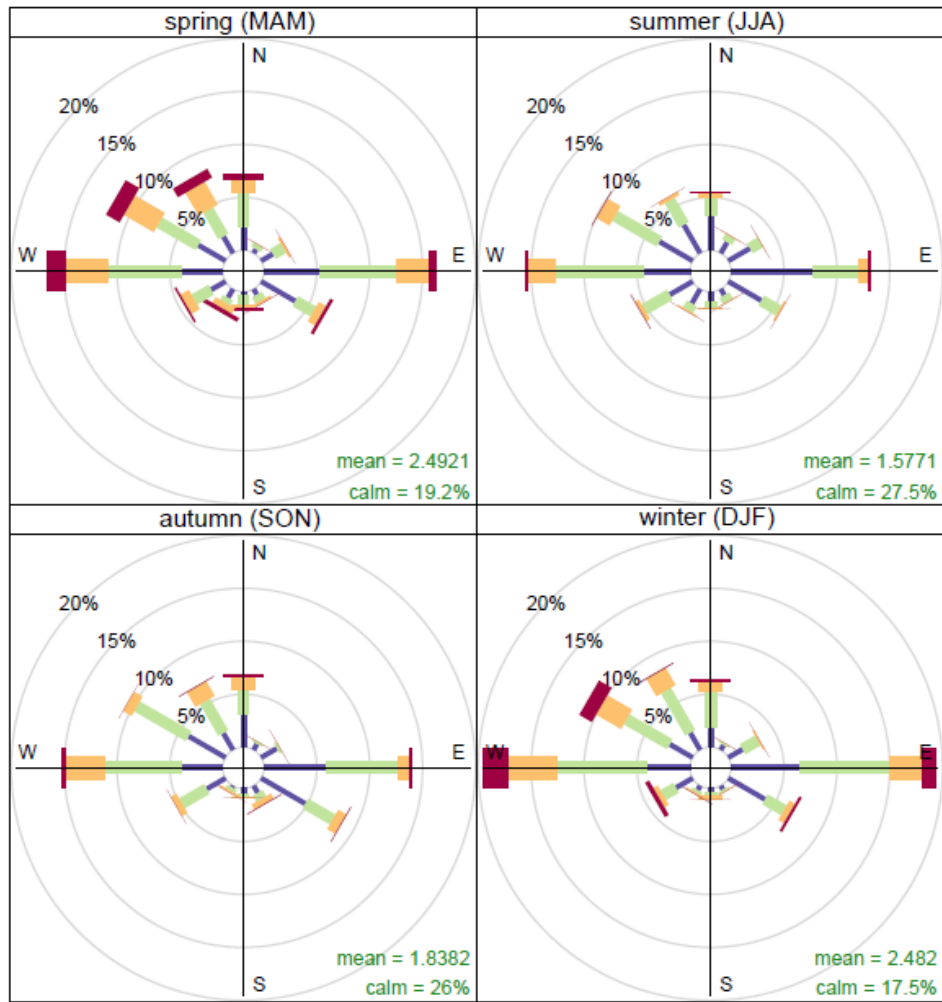


Figure B- 3. Seasonal Wind Roses Virginia Highlands Airport (2021-2022)



Frequency of counts by wind direction (%)

Appendix C. Data Tables

Public Health Assessment Site Tool (PHAST) - Screening Table

Contaminant Name	Maximum Site Concentration in Air (ppb)	ATSDR Recommended CV	ATSDR CV Type	Selected for Further Evaluation?	Alternate Comparison Value
1,2-dibromoethane	0.01	0.00022	CREG	Yes	NA
Trichloroethylene	0.19	0.040	CREG	Yes	NA
Carbon tetrachloride	0.1	0.026	CREG	Yes	NA
Hexachlorobutadiene	0.02	0.0043	CREG	Yes	NA
1,4-dioxane	0.4	0.055	CREG	Yes	NA
1,3-butadiene	0.37	0.015	CREG	Yes	NA
1,2-dichloroethane	0.05	0.0095	CREG	Yes	NA
Chloroform	0.22	0.0089	CREG	Yes	NA
Benzene	44.8	0.040	CREG	Yes	NA
Methyl methacrylate	0.09	170	RMEG	No	NA
1,2,4-trimethylbenzene	1.9	12	RMEG	No	NA
Bromomethane	0.02	1.0	Chronic EMEG/MRL	No	NA
Cyclohexane	0.27	1,700	RMEG	No	NA
1,4-dichlorobenzene	0.15	10	Chronic EMEG/MRL	No	NA
Methyl-t-butyl ether	0.02	830	RMEG	No	NA
Xylenes, total	2.39	23	RMEG	No	NA
Hexane, n-	0.62	200	RMEG	No	NA
1,1,1-trichloroethane	0.02	700	Intermediate EMEG/MRL	No	NA
Styrene	1.77	200	Chronic EMEG/MRL	No	NA
1,2-dichloropropane	0.09	0.87	RMEG	No	NA
Tetrahydrofuran	3.5	680	RMEG	No	NA
1,3,5-trimethylbenzene	0.36	12	RMEG	No	NA
Acetonitrile	7.2	36	RMEG	No	NA
Ethylbenzene	3.39	60	Chronic EMEG/MRL	No	NA
Tetrachloroethylene	0.06	0.57	CREG	No	NA
Chloromethane	0.99	30	Chronic EMEG/MRL	No	NA
Toluene	3.36	1,000	Chronic EMEG/MRL	No	NA
2-butanone	5.9	1,700	RMEG	No	NA

Contaminant Name	Maximum Site Concentration in Air (ppb)	ATSDR Recommended CV	ATSDR CV Type	Selected for Further Evaluation?	Alternate Comparison Value
Chloroethane	0.1	3,800	RMEG	No	NA
Methylene chloride	1.56	18	CREG	No	NA
1,1-dichloroethane	0.02	NA	NA	No	0.44 ppb (EPA RSL)
1,2-dichloroethene, cis-	0.03	NA	NA	No	200 ppb (TCEQ ESL)
1,2-dichlorobenzene	0.05	NA	NA	No	34 ppb (EPA RSL)
1,2,4-trichlorobenzene	0.15	NA	NA	No	0.28 ppb (EPA RSL)
Ethyl acetate	1.96	NA	NA	No	20 ppb (EPA RSL)
Acetone	3.55	NA	NA	No	2,000 ppb (TCEQ ESL)
Dichlorodifluoromethane	0.66	NA	NA	No	20 ppb (EPA RSL)
Ethanol	24.6	NA	NA	No	1,000 ppb (TCEQ ESL)
Heptane	0.42	NA	NA	No	100 (EPA RSL)
1,3-dichlorobenzene	0.125	NA	NA	No	27 (TCEQ ESL)
Chlorobenzene	0.05	NA	NA	No	10 ppb (EPA RSL)
Xylene, o-	0.84	NA	NA	No	22 ppb (EPA RSL)
Propylene	6.4	NA	NA	No	1,768 ppb (EPA RSL)
4-ethyltoluene	0.58	NA	NA	No	25 (TCEQ ESL)
1,1,2-trichloro-1,2,2-trifluoroethane	0.08	NA	NA	No	666 ppb (EPA RSL)
Isopropanol	8.6	NA	NA	No	83 ppb (EPA RSL)
Trichlorofluoromethane	0.45	NA	NA	No	1,000 ppb (TCEQ ESL)

Notes: Bold = included as contaminant of concern for health consultation, ppb = parts per billion, CV = ATSDR health-based comparison value, CREG = ATSDR cancer risk evaluation guideline, RMEG = ATSDR screening value based on EPA reference concentration, EMEG = ATSDR environmental media evaluation guideline, MRL = ATSDR minimal risk level, EPA RSL = U.S. Environmental Protection Agency Regional Screening Level, TCEQ ESL = Texas Commission on Environmental Quality long-term exposure Effect Screening Level, NA = Not available

Exposure Point Concentration (EPC) Tool for BENZENE - Gamma UCL Statistics for Uncensored Full Data Sets

Exposure Unit	1
Contaminant	benzene
CASRN	000071-43-2
Media	Air
Units	ppb
EPC Value	9.363711
EPC Type	95% UCL of the mean
EPC Quality Control Flags	NA
R Warnings and Error Flags	NA
Notes	This dataset contained 20 or more records, so the EPC was calculated using bootstrap sampling.
Number of Observations	26
Number of Detected Observations	26
Number of Distinct Detected Observations	24
Minimum Detected Value	0.02
Maximum Detected Value	27
Number of Distinct Non-detect Observations	0
Minimum Non-Detect Value	NA
Maximum Non-Detect Value	NA
Arithmetic Mean	6.935
Standard Deviation	6.947
Median	4.765
Interquartile Range	2.95-7.875 (4.925)
Best Fitting Data Distribution	NA
Mean: Model Estimated	NA
Median: Model Estimated	NA
Standard Deviation: Model Estimated	NA
Interquartile Range: Model Estimated (Q1–Q3)	NA
Normal Distribution GOF Test Statistic	NA
Lognormal Distribution GOF Test Statistic	NA
Gamma Distribution GOF Test Statistic	NA

These statistics are based upon data size, data distribution, and skewness, as predicted from the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).

Appendix D. Potential Contaminants of Concern

Appendix D provides additional information about each of the potential COCs), including their common uses, background levels in ambient air in the United States, and primary information from the toxicology literature.

1,2-Dibromoethane (EDB)

1,2-Dibromoethane, also known as ethylene dibromide (EDB) is a colorless liquid with a mild, sweet odor. The primary source of EDB released to the environment is from emissions into air from industrial processing facilities. EDB is used as an intermediate in the production of dyes, resins, gums, and waxes and as a pesticide treatment of felled logs. The most likely exposure to EDB for the general population is from inhalation of air near processing facilities or ingestion of contaminated drinking water (ATSDR 2018). Typical levels detected in urban air in the United States are estimated to be 0.0026 ppb (ATSDR 2018).

Little information on the health effects of EDB in humans is available. Case reports of acute exposures by inhalation or ingestion at very high levels (i.e., at or near lethal doses) identify the respiratory tract, gastrointestinal tract, liver, and kidney as targets of EDB (ATSDR 2018). Studies of workers showed serious effects to the male reproductive system (ATSDR 2018).

More information on the toxic effects of inhalation exposures to EDB are available from animal studies. Studies in animals provide support for the target organs observed in humans and identify additional targets. In animal studies, inhalation exposures to EDB are shown to cause weight loss or decreased weight gain, and harmful effects in the blood and the liver, and both the respiratory and gastrointestinal tracts. By all routes, including inhalation, tissue damage is observed at the point of contact, making it difficult to observe which target organ is the most sensitive in animal studies. ATSDR has not derived a minimal risk level (MRL) for inhalation exposures to EDB. However, the EPA has identified a reference concentration (RfC) of 1.1 ppb, based on nasal inflammation, what the EPA has identified as the most sensitive health endpoint from the literature (EPA 2004). The EPA notes that, in general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory

system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects).

1,2-Dichloroethane (1,2-DCA)

1,2-Dichloroethane (1,2-DCA) is a clear, manufactured liquid that is not found naturally in the environment. It evaporates quickly at room temperature and has a pleasant smell and a sweet taste. The most common use of 1,2-DCA is to make vinyl chloride, which is used to make a variety of plastic and vinyl products including polyvinyl chloride (PVC) pipes and other important construction materials, packaging materials, furniture and automobile upholstery, wall coverings, housewares, and automobile parts. 1,2-DCA is also used as a solvent and is added to leaded gasoline to remove lead. In the past, it was also found in small amounts in products that industries used to clean cloth, remove grease from metal, and break down oils, fats, waxes, resins, and rubber. In the household, 1,2-DCA was formerly a component of some cleaning solutions and pesticides; some adhesives, such as those used to glue wallpaper or carpeting; and some paint, varnish, and finish removers. Although large amounts of 1,2-DCA are produced today, most is used to make other chemical products. Low levels of 1,2-DCA have been found in the air near industries where it is made or used in manufacturing.

Humans can be exposed to low levels of 1,2-DCA through the skin or air by contact with old products made with 1,2-DCA, such as cleaning agents, pesticides, and adhesives used to glue wallpaper and carpets. Such exposure is probably not enough to cause harmful health effects (ATSDR 2022). Median daily atmospheric concentrations of 1,2-DCA are typically in the 0.01–0.1 ppb range for urban, suburban, rural, and remote sites (ATSDR 2022).

Most of what we know about health effects from exposure to 1,2-DCA come from occupational and accidental exposures to humans and from animal studies. People who were accidentally exposed to large amounts of 1,2-DCA in the air or by ingestion often developed nervous system disorders and liver and kidney disease. Lung effects were also seen after a large amount of 1,2-DCA was inhaled. In these cases of exposure, people often died from heart failure. We do not know what levels of 1,2-DCA caused these effects, but they are probably high. Studies in laboratory animals also found that breathing or swallowing large amounts of 1,2-DCA produced nervous system disorders, kidney disease, or lung effects. Reduced ability to fight infection was also seen in laboratory animals who breathed or swallowed 1,2-DCA, but we do not know if this also occurs in humans. Longer-term exposure to lower doses also caused kidney disease in animals (ATSDR 2022a).

So far, exposure to 1,2-DCA has not been associated with cancer in humans. Cancer was found in laboratory animals who were fed large doses of 1,2-DCA. When 1,2-DCA was put on the skin of laboratory animals, they developed lung tumors. We are not sure whether breathing 1,2-DCA causes cancer in animals. Because of the cancer findings in animals, the possibility of cancer in humans cannot be ruled out. The NTP has determined that 1,2-DCA may reasonably be expected to cause cancer. The IARC has determined that 1,2-DCA can possibly cause cancer in humans. Based on ingestion studies showing increased rates of hemangiosarcomas from increasing rates of 1,2-DCA ingestion, EPA has determined that 1,2-DCA is a probable human

carcinogen. Based on dose-response data from the oral exposure study, the EPA has identified an inhalation unit risk (IUR) of $0.000026 (\mu\text{g}/\text{m}^3)^{-1}$ (EPA 1987).

1,3-Butadiene

1,3-Butadiene is a colorless gas with a mild gasoline-like odor. About 60% of 1,3-butadiene is used to make man-made rubber, which is then used mostly for car and truck tires. 1,3-Butadiene is also used to make certain types of plastics such as acrylics. Large amounts of 1,3-butadiene are released into the air by industrial sources. Automobile exhaust is a constant source of 1,3-butadiene release into the air. Other sources of 1,3-butadiene include cigarette smoke and the smoke of wood fires, including forest fires, burning of rubber and plastic, and accidental or intentional release at manufacturing plants (ATSDR 2012a). The primary way you can be exposed to 1,3-butadiene is by breathing air containing it. The average amount of 1,3-butadiene in the air in cities and suburban areas of the U.S. is between 0.04 and 0.9 ppb.

Information on the toxicity of 1,3-butadiene in humans comes from primarily case reports and epidemiology studies focused on carcinogenicity. In one occupational study, workers exposed to 1,3-butadiene gas during the manufacture of rubber complained of irritation of the eyes, nasal passages, throat, and lungs. In some, coughing, fatigue, and drowsiness developed. All symptoms disappeared on removal from the gas (ATSDR 2012a). The associated exposure levels were not reported. In laboratory animals, 1,3-butadiene causes inflammation of nasal tissues, changes to lung, heart, and reproductive tissues, neurological effects, and blood changes. However, determining the relevance to human health of adverse health effects observed in laboratory animals is encumbered by large differences in the tested species metabolism of 1,3-butadiene. Studies of workers exposed to 1,3-butadiene suggest that workers may have an increased risk for cancers of the blood and lymphatic system. Laboratory animals have developed cancer in multiple body tissues after exposure to 1,3-butadiene for 13 weeks or longer. Animals appear to be most sensitive to blood and lymphatic system cancers. IARC, NTP, and EPA all classify 1,3-butadiene as a human carcinogen.

1,4-Dioxane

1,4-Dioxane is a clear liquid with a slight pleasant odor. It is used as a laboratory reagent, to manufacture other chemicals, and can be found at trace levels in some cosmetics, detergents, and shampoos (ATSDR 2012b). 1,4-Dioxane can be released into the air where it is produced or used as a solvent. Current levels of 1,4-dioxane in air are not known. In the mid-1980s, average levels of 1,4-dioxane in air samples from the United States were about 0.1 ppb for outdoor air and 1 ppb for indoor air (ATSDR 2012b).

Inhaling 1,4-dioxane for short periods of time may cause eye and nose irritation. Animal studies have shown that inhalation affects the nasal cavity, the liver and the kidneys. Laboratory rats that breathed 1,4-dioxane vapors developed cancer in the nose and abdominal cavity (ATSDR 2012b). IARC has determined that 1,4-dioxane is possibly carcinogenic to humans. The NTP considers 1,4-dioxane as reasonably anticipated to be a human carcinogen. The EPA has established that 1,4-dioxane is likely to be carcinogenic to humans

Benzene

Benzene, also known as benzol, is a colorless liquid with a sweet odor. Benzene evaporates into air very quickly and dissolves slightly in water. Benzene is highly flammable. Most people can begin to smell benzene in air at approximately 60,000 ppb and recognize it as benzene at 100,000 ppb. Benzene is found in air, water, and soil. Benzene comes from both industrial and natural sources. Various industries use benzene to make other chemicals, such as styrene (for Styrofoam® and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibers). Benzene is also used in the manufacturing of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene, which include gas emissions from volcanoes and forest fires, also contribute to the presence of benzene in the environment. Benzene is present in crude oil, gasoline, and cigarette smoke (ATSDR 2007). The major sources of benzene exposure to U.S. residents are tobacco smoke (45%), automobile exhaust and industry (20%), and other home sources (16%). Home sources include paints and gasoline stored in the home (i.e., in basements or attached garages) (Wallace 1995, Ott 1998).

Measured levels of benzene in outdoor air have ranged from 0.02 to 34 ppb. Typical or median levels of benzene in urban air in the Eastern United States are estimated to be 11 ppb or less (ATSDR 2007). The EPA estimates the average background benzene level in the U.S. is approximately 1 ppb (EPA 2010b). People living in cities or industrial areas are generally exposed to higher levels of benzene in air than those living in rural areas. Benzene levels in the home are usually higher than outdoor levels.

Most information on effects of long-term exposure to benzene are from worker studies. Worker studies assess exposures at levels much higher than exposures for the normal population (Bristol ambient air levels are much lower than those used in the occupational studies discussed below). From worker studies, we have learned that inhaling benzene at elevated concentrations (700,000–3,000,000 ppb) can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. People who breathe benzene for long periods may experience harmful effects in the tissues that form blood cells, especially the bone marrow. Blood production may return to normal after exposure to benzene stops. Excessive exposure to benzene can be harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against cancer. Benzene exposures may also be harmful to the reproductive organs. HHS, IARC and EPA have determined that benzene is carcinogenic to humans. Long-term exposure to benzene can cause cancer and has been associated with development of a particular type of leukemia called acute myeloid leukemia, or AML (ATSDR 2007).

Carbon Tetrachloride

Carbon tetrachloride is a clear liquid that evaporates very easily. Most carbon tetrachloride that escapes to the environment is therefore found as a gas. Carbon tetrachloride has a sweet odor, and most people can begin to smell it in air when the concentration reaches 10 ppm carbon tetrachloride in air (10,000 ppb). Carbon tetrachloride is a manufactured chemical and does not occur naturally in the environment. Carbon tetrachloride has been produced in large quantities to make refrigeration fluid and propellants for aerosol cans. Since many refrigerants and

aerosol propellants have been found to affect the earth's ozone layer, the production of these chemicals is being phased out. Consequently, the manufacture and use of carbon tetrachloride has declined a great deal. In the past, carbon tetrachloride was widely used as a cleaning fluid (in industry and dry-cleaning establishments as a degreasing agent, and in households as a spot remover for clothing, furniture, and carpeting). Carbon tetrachloride was also used in fire extinguishers and as a fumigant to kill insects in grain. Most of these uses were discontinued in the mid-1960s. Until 1986, carbon tetrachloride was used as a pesticide (ATSDR 2005). Concentrations in air of 0.1 ppb are common around the world, with somewhat higher levels (0.2– 0.6 ppb) often found in cities (ATSDR 2005).

Most information on the health effects of carbon tetrachloride in humans comes from cases where people have been exposed to relatively high levels of carbon tetrachloride, either only once or for a short period, for example, by accidental poisoning or by working with the chemical in a confined space without ventilation. Experiments have not been performed on the effects of long-term exposure of humans to low levels of carbon tetrachloride, so the human health effects of such exposures are not well known. The liver is especially sensitive to carbon tetrachloride since it contains a large amount of the enzymes that change the form of the chemical. Some of the breakdown products may attack cell proteins, interfering with the functions of the liver cells. Products that attack cell membranes may result in the death of the cells. The kidney is also sensitive to carbon tetrachloride. Less urine may be formed, leading to a buildup of water in the body (especially in the lungs) and buildup of waste products in the blood. Long-term breathing exposure to carbon tetrachloride worsened age-related kidney disease in rats. Fortunately, if injuries to the liver and kidney are not too severe, these effects eventually disappear after exposure stops. This is because both organs can repair damaged cells and replace dead cells. Function usually returns to normal within a few days or a few weeks after the exposure has stopped. High levels of carbon tetrachloride exposure affect the nervous system, including the brain. The immediate effects are usually signs of intoxication, including headache, dizziness, and sleepiness perhaps accompanied by nausea and vomiting (ATSDR 2005).

Studies in animals have shown that breathing carbon tetrachloride over a period of years increases the frequency of liver tumors. Mice breathing carbon tetrachloride developed tumors of the adrenal gland. Studies have not been performed to determine whether breathing carbon tetrachloride causes tumors in humans, but it should be assumed that carbon tetrachloride could produce cancer. NTP has determined that carbon tetrachloride may reasonably be anticipated to be a carcinogen. IARC has classified carbon tetrachloride as possibly carcinogenic to humans, and the EPA has determined that carbon tetrachloride is a probable human carcinogen.

Chloroform

Chloroform, also known as trichloromethane, is a colorless liquid with a pleasant, nonirritating odor and a slightly sweet taste. Most of the chloroform found in the environment comes from industry. Chloroform was one of the first inhaled anesthetics to be used during surgery, but it is not used for anesthesia today. Nearly all the chloroform made in the United States today is

used to make other chemicals, but some is sold or traded to other countries. Chloroform enters the environment from chemical companies and emissions from paper mills. It is also found in wastewater from sewage treatment plants and drinking water to which chlorine has been added. Chlorine is added to most drinking water and many waste waters to destroy bacteria. Small amounts of chloroform are formed as an unwanted product during the process of adding chlorine to water. Chloroform can enter the air directly from factories that make or use it and by evaporating from water and soil that contain it (ATSDR 1997).

The amount of chloroform normally expected to be in the air ranges from 0.02 to 0.05 ppb. However, much higher levels can be found in the vicinity of a landfill. ATSDR's toxicological profile for chloroform found that chloroform concentrations in air reached 610 ppb at a municipal landfill. Chloroform has been found in the air from all areas of the United States and in nearly all of the public drinking water supplies (ATSDR 1997).

In humans, chloroform affects the central nervous system (brain), liver, and kidneys after a person breathes air that contain large amounts of chloroform. Chloroform was used as an anesthetic during surgery for many years before its harmful effects on the liver and kidneys were recognized. Breathing about 900,000 ppb chloroform for a short time causes fatigue, dizziness, and headache. If you breathe air containing elevated levels of chloroform over a long period, the chloroform may damage your liver and kidneys. We do not know whether chloroform causes harmful reproductive effects or birth defects in people. Miscarriages occurred in rats and mice that breathed air containing elevated levels of chloroform (30,000 ppb or more) during pregnancy. Abnormal sperm were found in mice that breathed air containing elevated levels (400,000 ppb) of chloroform for a few days. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects (ATSDR 1997).

Hexachlorobutadiene

Hexachlorobutadiene is a colorless liquid with a turpentine-like odor with an odor threshold of approximately 1,000 ppb. The main source of hexachlorobutadiene in the United States is its production as a byproduct of chlorinated hydrocarbon synthesis. Atmospheric levels of hexachlorobutadiene in rural and urban air samples typically range from 0.002 to 0.011 ppb, with a mean value of 0.002–0.003 ppb. Higher levels can be detected at areas near industrial and chemical waste disposal and production sites (ATSDR 2021).

Most of what we know about the toxicity of hexachlorobutadiene primarily comes from studies in laboratory animals where a wide range of toxic endpoints were evaluated; the kidneys appear to be the most sensitive endpoint of hexachlorobutadiene exposure. Other targets include body weight gain, developmental toxicity, respiratory effects, hematological alterations, and hepatic toxicity; additionally, there is some evidence that chronic exposure can result in kidney tumors. Only two epidemiology studies, both of limited scope examining renal and hepatic effects, were identified (ATSDR 2021).

Trichloroethylene (TCE)

TCE is a colorless, volatile liquid. Liquid TCE evaporates quickly into the air. It is nonflammable and has a sweet odor. The two major uses of TCE are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. TCE has also been used as an extraction solvent for greases, oils, fats, waxes, and tars; by the textile processing industry to scour cotton, wool, and other fabrics; in dry cleaning operations; and as a component of adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners. Most of the TCE used in the United States is released into the atmosphere by evaporation, primarily from degreasing operations. Some TCE is released to the air by its evaporation from products such as adhesives, paints, and coatings; and through its evaporation from TCE-contaminated soil at landfills (ATSDR 2019). Once in the atmosphere, the estimated half-life is about 3–7 days. This relatively short half-life indicates that TCE is not a persistent atmospheric compound.

TCE was once used as an anesthetic for surgery. People who are overexposed to moderate amounts of TCE may experience headaches, dizziness, and sleepiness; large amounts of TCE may cause coma and even death. Some people who breathe high levels of TCE may develop damage to some of the nerves in the face. Other effects seen in people exposed to high levels of TCE include evidence of nervous system effects related to hearing, seeing, and balance, changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Relatively short-term exposure of animals to TCE resulted in harmful effects on the nervous system, liver, respiratory system, kidneys, blood, immune system, heart, and body weight. Long-term exposure studies in animals have mainly focused on carcinogenicity and relatively insensitive noncancer end points following oral exposure; these studies are not helpful in defining noncancer end points in humans following long-term exposure. However, depressed body weight and evidence of effects on the thymus were reported in one recent study of mice exposed to TCE via their mothers during gestation and lactation and via the drinking water for up to 12 months thereafter (ATSDR 2019).

There is strong evidence that TCE can cause kidney cancer in people and some evidence that it causes liver cancer and malignant lymphoma (a blood cancer). Lifetime exposure to TCE resulted in increased liver cancer in mice and increased kidney cancer in rats at relatively high exposure levels. There is some evidence for TCE-induced testicular cancer and leukemia in rats and lymphomas and lung tumors in mice. HHS has classified TCE as “known to be a human carcinogen” based on sufficient evidence of carcinogenicity from humans. Similarly, IARC has classified it as “carcinogenic to humans” and EPA has characterized it as “carcinogenic in humans by all routes of exposure.” These agencies concluded that there were sufficient evidence from human studies that TCE exposure can cause kidney cancer in humans. There is also some evidence of an association between TCE exposure and non-Hodgkin’s lymphoma in humans (ATSDR 2019).

Ammonia

Ammonia is a chemical that is made both by humans and by nature. The amount of ammonia manufactured every year by humans is almost equal to the amount produced by nature every year. However, when ammonia is found at a level that may cause concern, it was likely

produced either directly or indirectly by humans. Ammonia is a colorless gas with a very sharp odor. The odor of ammonia is familiar to most people because ammonia is used in smelling salts, household cleaners, and window cleaning products. Ammonia is very important to plant, animal, and human life. It is found in water, soil, and air, and is a source of much needed nitrogen for plants and animals. Most of the ammonia in the environment comes from the natural breakdown of manure, dead plants and animals. Eighty percent of all manufactured ammonia is used as fertilizer. A third of this is applied directly to soil as pure ammonia. The rest is used to make other fertilizers that contain ammonium compounds, usually ammonium salts. These fertilizers are used to provide nitrogen to plants. Ammonia is also used to manufacture synthetic fibers, plastics, and explosives. Many cleaning products also contain ammonia in the form of ammonium ions (ATSDR 2004).

Since ammonia occurs naturally in the environment, we are regularly exposed to low levels of ammonia in air, soil, and water. Ammonia exists naturally in the air at levels between 1 and 5 ppb.

Hydrogen Sulfide (H₂S)

H₂S is a flammable, colorless gas that smells like rotten eggs. People usually can smell H₂S at low concentrations in air, ranging from 0.5 to 300 parts H₂S per billion parts of air (ppb). At high concentrations, a person might lose their ability to smell it. H₂S occurs both naturally and from human-made processes. It is in the gases from volcanoes, sulfur springs, undersea vents, swamps, stagnant bodies of water, and in crude petroleum and natural gas. H₂S is also associated with municipal sewers and sewage treatment plants, swine containment and manure-handling operations, and pulp and paper operations. Other industrial sources of H₂S include petroleum refineries, natural gas plants, petrochemical plants, coke oven plants, food processing plants, and tanneries. Bacteria found in your mouth and gastrointestinal tract produce H₂S during the digestion of food containing vegetable or animal proteins. H₂S is used primarily in the production of sulfur and sulfuric acid. It can also be used to make other chemicals such as sodium sulfide and sodium hydrosulfide, which are used to make a variety of products (ATSDR 2016).

H₂S air concentrations from natural sources range between 0.11 and 0.33 ppb. In urban areas, the air concentrations are generally less than 1 ppb. H₂S remains in the atmosphere for approximately 1–42 days, depending on the season. It can change into SO₂ and sulfates in the air.

Methyl Mercaptan

Methyl mercaptan, also known as methanethiol, is a colorless gas with a smell like rotten cabbage. It is a natural substance found in the blood, brain, and other tissues of humans and other animals, and it is released from animal feces. It occurs naturally in certain foods such as some nuts (filberts) and cheese (Beaufort). Methyl mercaptan is released from decaying organic matter in marshes and is present in the natural gas of certain regions of the United States, in coal tar, and in some crude oils. Methyl mercaptan is manufactured for use in pesticides, as a jet fuel additive, in the plastics industry, and in making methionine, a nutrient that is added to

poultry feed. Methyl mercaptan is also released as a decay product of wood in pulp mills (ATSDR 1992). Methyl mercaptan is always present in your body and in your urine and feces. It can also be present in the breath of persons with liver damage. You can be exposed to methyl mercaptan in the air if you live near a natural source of this gas, such as a marsh, an underground gas pocket, or a dump site that releases it (ATSDR 1992). Methyl mercaptan has been found in environmental air at 4 ppb (ATSDR 1992).

Sulfur Dioxide (SO₂)

SO₂ is a colorless gas with a pungent odor. It is a liquid when under pressure. SO₂ dissolves in water very easily. It cannot catch fire. SO₂ in the air results primarily from activities associated with the burning of fossil fuels (coal, oil) such as at power plants or from copper smelting. In nature, SO₂ can be released to the air, for example, from volcanic eruptions. Typical outdoor concentrations of SO₂ may range from 0 to 1000 ppb (ATSDR 1998).

Appendix E. Cancer Risk Equations

Appendix E provides the equations used to calculate cancer risk for individuals exposed daily over 25 years to each of the nine carcinogenic chemicals detected in ambient air in Bristol communities.

Benzene Cancer Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.0000078 (\mu\text{g}/\text{m}^3)^{-1} \times 33.2 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.000083 = 8.3 \text{ in } 100,000 = 8.3\text{E-}05$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

1,2-Dibromoethane (EDB) Cancer Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.0006 (\mu\text{g}/\text{m}^3)^{-1} \times 0.08 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.000015 = 1.5 \text{ in } 100,000 = 1.5\text{E-}05$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

1,2-Dichloroethane (1,2-DCA) Cancer Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.000026 (\mu\text{g}/\text{m}^3)^{-1} \times 0.41 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000034 = 3.4 \text{ in } 1,000,000 = 3.4\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

1,3-Butadiene Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.00003 (\mu\text{g}/\text{m}^3)^{-1} \times 0.23 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000022 = 2.2 \text{ in } 1,000,000 = 2.2\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

1,4-Dioxane Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.000005 (\mu\text{g}/\text{m}^3)^{-1} \times 2 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000032 = 3.2 \text{ in } 1,000,000 = 3.2\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

Carbon Tetrachloride Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.000006 (\mu\text{g}/\text{m}^3)^{-1} \times 0.51 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.00000098 = 9.8 \text{ in } 10,000,000 = 9.8\text{E-}07$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

Chloroform Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.000023 (\mu\text{g}/\text{m}^3)^{-1} \times 1.09 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000080 = 8.0 \text{ in } 1,000,000 = 8.0\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ =

Hexachlorobutadiene Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.000022 (\mu\text{g}/\text{m}^3)^{-1} \times 0.22 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000016 = 1.6 \text{ in } 1,000,000 = 1.6\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration

Trichloroethylene Risk Calculation

IUR x chronic EPC x ED = Cancer Risk

$$0.0000041 (\mu\text{g}/\text{m}^3)^{-1} \times 0.77 \mu\text{g}/\text{m}^3 \times (25 \text{ years}/78 \text{ years}) = 0.0000010 = 1.0 \text{ in } 1,000,000 = 1.0\text{E-}06$$

Notes: IUR = inhalation unit risk; EPC = chronic exposure point concentration; $\mu\text{g}/\text{m}^3$ = micrograms per cubic meter; ED = exposure duration