

**Garfield County
Air Toxics Inhalation: Screening Level
Human Health Risk Assessment**

**Inhalation of Volatile Organic Compounds Measured In 2008 Air
Quality Monitoring Study**

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Executive Summary

Increased oil and gas development activity within Garfield County has generated concerns about potential impacts to public health. The Colorado Department of Public Health and Environment (CDPHE) has been providing technical assistance to the Garfield County Public Health Department (GCPHD) since 2002 to assess these potential impacts. The findings of the 2007 CDPHE health risk assessment of air toxics data suggested potential cancer and noncancer (short-term) health effects of benzene in the oil and gas development area, based on the estimated exposures at one monitoring site (Brock). Based on the 2007 results and recommendations of this previous study, the GCPHD enhanced its air quality monitoring in 2008 by analyzing for significantly more (90) organic compounds, increasing the frequency of sampling, and focusing on a smaller number of monitoring sites. The 2008 ambient air quality monitoring study findings indicated that some of the primary organic chemicals associated with petroleum and natural gas emission sources were higher in rural Garfield County than in other urban areas (e.g., Grand Junction) outside the County where measurements were available.

Based on the work completed in 2007 and 2008, the GCPHD in 2009 requested that the CDPHE determine if residents in Garfield County are being exposed to airborne concentrations of measured air toxics such as speciated non-methane organic compounds and carbonyls that may pose unacceptable health risks via inhalation. The findings of this 2009 health risk assessment will help guide risk management decision-making and future air monitoring by the GCPHD. It is important to note that health risk assessments provide predictions of hypothetical health risks, which are intended as screening tools for risk managers and cannot be used to make realistic predictions of biological effects.

This report provides an overview of EPA's risk assessment approach, potential exposure scenarios, and our conclusions regarding the potential for health effects, accounting for associated uncertainties of health risk assessment exercises. Risks were determined using data collected by the GCPHD during the 2008 air quality monitoring study. Two types of health effects were evaluated: 1) Increased risk of cancer in a lifetime; and, 2) Noncancer hazards (chronic and acute). For use in this risk assessment, GCPHD collected 24-hour air samples from four fixed monitoring sites on a weekly or bi-weekly basis over the course of 12 months. The four monitoring sites, Bell, Brock, Parachute, and Rifle, were located in close proximity (<1.5 mile) to oil and gas development activities in the rural and urban oil and gas development areas.

Conclusions

The available information suggests a potential for public health impacts across the oil and gas development areas in Garfield County because of the following:

- The estimated cumulative lifetime cancer risks for the 6 air toxics with known toxicity values are at or slightly above the high-end of EPA's acceptable cancer risk range of 1 to 100 excess cancers in a million (1E-06 to 1E-04) across all monitoring sites. These total risk estimates are based on all carcinogenic

chemicals including crotonaldehyde, which has a highly uncertain cancer toxicity value. It should be noted that the total cancer risks at all monitoring sites remain above the mid-point of EPA's acceptable cancer risk range of 1E-06 to 1E-04 even when crotonaldehyde is excluded. The major contributors to this risk are formaldehyde and benzene. The estimated risks with and without crotonaldehyde indicate a low to moderate increased risk of developing cancer during a lifetime. Overall, it is important to note that the cancer risks are likely to be underestimated in this assessment because cancer toxicity values are only available for a small number of air toxics.

- Each of the 20 individual air toxics assessed at any monitoring site have a chronic noncancer hazard estimate well below an acceptable value of one. However, when accounting for the cumulative chronic noncancer hazards for all of these 20 air toxics the chronic noncancer hazard estimate is just below the acceptable level of one across the two monitoring sites. The major contributing chemicals to the cumulative hazard estimate are acetaldehyde, formaldehyde, trimethylbenzenes, and benzene. This finding indicates a low increased risk of developing noncancer health effects (e.g., respiratory, immunological, and nervous system effects). Again, it is important to note that the noncancer hazards are likely to be underestimated in this assessment because noncancer toxicity values are not available for 65 air toxics.
- Based on the available 24-hour air monitoring data, the estimated acute noncancer hazards for benzene are well below an acceptable value of one indicating a low increased potential for acute health effects of benzene (e.g., immune system effects).
- The cumulative health impacts of 86 detected ambient air toxics cannot be determined due to the absence of toxicity values, which EPA is responsible for determining, for 65 air toxics.

Finally, this investigation is best viewed as a "snapshot" of air quality due to the uncertainties and limitations in the methods used to assess exposure and toxicity.

Recommendations

The findings of this risk assessment support the need for the following:

- Continue long-term air monitoring; increase the frequency of sampling; and include in the sampling of a complete list of contaminants associated with oil and gas development.
- Implement short-term (acute) air monitoring by collecting 1-hour air samples in order to evaluate health risks posed by intermittent peak exposures.
- Determine source apportionment including sources other than the oil and gas operations, such as stationary industrial sources and mobile traffic sources.

- Continue management of the risk posed by potential exposures to air toxics as a result of increase in oil and gas development activities (e.g., additional monitoring, sample analysis, and action as appropriate).

1 Introduction

The oil and gas industry in Garfield County has grown rapidly since 2002. Increased oil and gas development activity within Garfield County has generated concerns about the impact on public health. The Garfield County Public Health Department (GCPHD) has been monitoring air quality since 2005 in response to residents' concerns regarding the health impacts of increased oil and gas development activities. The GCPHD ambient air quality monitoring study, June 2005-May 2007, and the subsequent screening-level health risk assessment by the Colorado Department of Public Health and Environment (CDPHE) focused on 43 volatile organic compounds (VOCs) with sampling on a once per month or once per quarter basis across 14 monitoring sites (GCPD/CDPHE, 2007; CDPHE, 2007). The 2007 joint GCPHD and CDPHE air quality monitoring study indicated the influence of local sources in Garfield County based on the measured concentrations of VOCs related to oil and gas emission sources (GCPD/CDPHE, 2007). The findings of the 2007 CDPHE health risk assessment suggested a potential for cancer and noncancer (short-term) health effects of benzene across the oil and gas development area, based on the estimated exposures at the Brock monitoring site (CDPHE, 2007).

Based on the results and recommendations of these studies, GCPHD enhanced air quality monitoring in 2008 by analyzing samples for 90 speciated non-methane organic compounds (SNMOCs) and carbonyls, increasing the frequency of sampling to a weekly or bi-weekly basis, and focusing on 4 of the original 14 monitoring sites. The findings of the 2008 ambient air quality monitoring study indicated that some of the primary chemicals (e.g., light alkanes, benzene, toluene, ethylbenzene, and xylene) associated with petroleum and natural gas emission sources were higher in rural Garfield County than in other urban areas (e.g., Grand Junction) outside the County where measurements were available (GCPHD, 2009).

1.1 CDPHE Role

The CDPHE provides technical assistance to local agencies on an as needed basis. As part of this commitment, Air Pollution Control Division (APCD) and Disease Control and Environmental Epidemiology Division (DCEED) staff of the CDPHE are members of the Garfield County Air Quality Technical Workgroup, providing technical assistance to the Garfield County Public Health Department since 2002. In this capacity, APCD staff provides technical assistance regarding the ambient air quality monitoring issues and DCEED staff provides assistance regarding the health evaluation issues. In 2007, for example, APCD analyzed the 2005-2007 ambient air quality monitoring data and DCEED conducted the screening-level human health risk assessment. This report is a part of the ongoing joint APCD and DCEED assistance to the GCPHD.

1.2 Purpose

Most recently, the GCPHD requested that the CDPHE investigate the basis for the concern that residents in Garfield County are being exposed to airborne concentrations of measured air toxics such as SNMOCs and carbonyls that may pose unacceptable health risks via inhalation. The findings of this health risk assessment are designed to help guide risk management decision-making and future air monitoring and associated

analysis of the monitoring results by the GCPHD. For example, this risk assessment will help the GCPHD to set priorities for the collection of additional information to improve future health risk assessments.

1.3 Risk Assessment Approach

A human health risk assessment process attempts to understand potential public health risks from exposures to measured VOCs emitted into the air from sources of interest and any uncertainties associated with the assessment. This risk assessment is prepared in accordance with the US Environmental Protection Agency (EPA) *Residual Risk Report to Congress and the EPA Risk Assessment Reference Library*, (USEPA, 2004). According to the EPA, risk assessment is generally a four-step process consisting of hazard identification, exposure assessment, dose-response assessment, and characterization of risk based on the combination of results of the three previous steps, and the associated uncertainties.

This risk assessment represents a “snapshot” in time for characterizing health risks from exposure to only a select number of organic compounds. It does not take into account potential changes in emissions over time, as a result of increase or decrease in oil and gas development activities. It is also not designed to characterize risks sufficiently from inhalation of all types of air toxics (e.g., all VOCs, semi-VOCs, and metals). Additionally, the risk assessment is limited to inhalation risk from outdoor sources and it does not characterize risks through pathways other than inhalation of contaminated outdoor air (i.e., indirect exposure pathways). In air toxic risk assessment, the inhalation pathway is commonly assessed. However, indirect exposure pathways can be significant for chemicals which are relatively persistent in the environment.

The principal sections of this risk assessment are:

- Introduction
- Exposure Assessment
- Toxicity Assessment
- Risk Characterization
- Uncertainty Analysis
- Conclusions
- References

1.3.1 Utility of Risk Assessments

It is important to note that health risk assessments provide predictions of hypothetical health risks that are intended as screening tools for risk managers and cannot be used to make realistic predictions of biological effects. As such, risk estimates cannot be used to determine whether someone who already has cancer or any other disease is ill because of a past exposure. In addition, risk assessments only provide one of several important tools in the whole risk management process. EPA’s regulatory process also calls for consideration of non-scientific factors (e.g., economic, social, political, and legal factors) in decision-making (USEPA, 2004).

2 Exposure Assessment

The process that characterizes the route, duration, intensity, and frequency of contact with a chemical by a receptor is known as the exposure assessment. In this assessment, the receptors of interest are individuals who may reside within a monitoring area, and the principal exposure route of interest is inhalation. For this assessment, both long-term (chronic) and short-term (acute) exposures are evaluated. Chronic assessment addresses repeated exposure to relatively low levels of VOCs over a prolonged period of time, and acute assessment addresses short-term exposure to the measured maximum concentrations in a 24-hour period.

2.1 Exposure Assumptions

The following assumptions are used in this risk assessment based on the EPA methodology regarding chronic exposure at the monitoring locations:

- a person lives, works, and otherwise stays near a given monitoring location for 24 hours per day, 365 days per year, for a 70-year time period.
- the air that the person breathes, both while indoors and outdoors, contains the same concentrations of pollutants measured in the air quality monitoring study.
- air quality, as reflected by the monitoring results, is assumed to remain relatively constant over the entire 70-year lifetime of a person living in the area.

The following assumptions are used in this risk assessment based on the EPA methodology regarding acute noncancer exposure at the monitoring locations:

- a person lives, works, and otherwise stays near a given monitoring location for a time period of up to 1-day.
- the air that the person breathes, both while indoors and outdoors, contains the same concentrations of pollutants as monitored in the air quality monitoring study.

2.2 Air Monitoring

This health risk assessment utilized data from the ambient air toxics monitoring conducted by the GCPD at four fixed sites in Garfield County, Colorado from January 2008 through December 2008. The four monitoring sites were Parachute, Rifle, Bell, and Brock. Detailed site locations and air monitoring are discussed in the GCPHD 2008 air quality monitoring report (GCPHD, 2009). Overall, all four sites were located in close proximity (<1.5 mile) to oil and gas development activities in the Garfield County, with two sites (Parachute and Rifle) located in urban areas and two sites (Bell and Brock) in rural areas.

2.2.1 Data Quality

Organic sampling and analysis conducted by the GCPHD was performed using methods that have been approved and recommended by the EPA. Specifically, the speciated non-methane organic compounds were collected with whole-air Summa canisters over a 24-hour period and analyzed via gas chromatography, in accordance with EPA Method TO-12. Likewise, carbonyls were collected on DNPH-coated cartridges and analyzed by liquid chromatography in accordance with EPA Method TO-11a. These methods can be

accessed at <http://www.epa.gov/ttn/amtic/airtox.html>. The laboratory that was used for sample analyses performs analyses nationally for EPA's air toxics program. Thus, data from this study are expected to be of high quality.

The laboratory sends the data to the APCD in an Excel file on a monthly basis. The files include not only the analysis results but also the minimum detection levels and quality control check results. These 12-month data were compiled by the APCD and given to the DCEED in an Excel file for use in the risk assessment.

2.2.2 Data Robustness

For statistical data analyses, the ideal scenario would be to have sampling conducted on an every day basis. However, due to budget constraints, this typically does not occur. For the GCPHD 2008 study, sampling was conducted once every 6th day for the speciated non-methane organic compounds (approximately 60 samples per year) and once every 12th day for the carbonyls (approximately 30 samples per year). While this follows general EPA protocols, the quantity of data is less than ideal for a robust statistical analysis on a one-year basis and can lead to an increased uncertainty.

2.3 Contaminants of Potential Concern

All chemicals that were detected at least once in the study period were conservatively retained as chemicals of potential concern (COPCs) for further evaluation. Overall, 90 chemicals were analyzed in the monitoring study. Four out of 90 chemicals were never detected. These chemicals were removed from further consideration in this assessment. Thus, 86 chemicals were retained as COPCs for the risk characterization at each monitoring location (Tables A1, A1.1, A2, and A2.1). For the assessment of acute health risks, COPCs were selected by comparing the highest maximum detected concentrations with the chronic toxicity value. Only benzene and 1, 2, 4-trimethylbenzene were selected as COPCs for acute risk evaluation (Table A3). However, only benzene was evaluated further for acute health risks because no acute toxicity value was available for 1,2,4-trimethylbenzene.

2.4 Exposure Concentration

Analytical data for COPCs were processed to derive exposure concentrations. As is standard practice in conducting risk assessments, all samples reported as non-detects were assigned a value of $\frac{1}{2}$ the lowest concentration that the instrument can detect, known as the sample quantitation limit (SQL) or detection limit.

The EPA recommends that the upper 95th upper confidence limit (UCL) of the arithmetic mean concentration be used as the Exposure Point Concentration (EPC) in calculating exposure and risk. The 95 percent UCL was calculated using the EPA ProUCL version 4.0 software (available at: <http://www.epa.gov/nerlesd1/tsc/install.htm>) and the GCPHD monitoring data (compiled by the CDPHE Air Pollution Control Division). The EPC values for the 86 COPCs on a monitoring location basis are summarized in Tables A1, A1.1, A2, and A2.1.

3 Toxicity Assessment

3.1 Overview

The basic objective of a toxicity assessment is to identify the adverse health effects a chemical may cause, and how the appearance of these adverse effects depends on dose. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal), the duration of exposure (acute, intermediate, chronic or lifetime), age, sex, diet, family traits, lifestyle, and state of health.

The toxicity assessment process is usually divided into two parts: the first characterizes and quantifies the cancer effects of the chemical, while the second addresses the noncancer effects of the chemical. This two-part approach is employed because there are typically major differences in the risk assessment methods used to assess cancer and noncancer effects. For example, cancer risks are expressed as a probability of suffering an adverse effect (cancer) during a lifetime and noncancer hazards are expressed, semi-quantitatively, in terms of the hazard quotient (HQ), defined as the ratio between an individual's estimated exposure and the RfC (see below). HQs are not an estimate of the likelihood that an effect will occur, but rather an indication of whether there is potential cause for concern for adverse health effects. Both cancer risks and hazard quotients estimate risks to individuals in a population and not to a particular individual (i.e., personal risk).

For carcinogens, toxicity measurements are generally expressed as a risk per unit concentration (e.g., an inhalation unit risk (IUR) in units of risk per $\mu\text{g}/\text{m}^3$). For noncancer effects, toxicity benchmarks are generally expressed as a concentration in air (e.g., an inhalation reference concentration (RfC) in units of $\mu\text{g}/\text{m}^3$ air). The reference concentration is an exposure that is believed to be without significant risk of adverse noncancer health effects in a chronically exposed population, including sensitive individuals.

3.2 Toxicity Values

The following hierarchy was used to compile a list of cancer and noncancer toxicity values for this report. To start, inhalation values established specifically by the State of Colorado were given priority over all other sources of toxicity values. The second source, used to identify relevant toxicity values, was EPA's Air Toxics Website (<http://www.epa.gov/ttn/atw/toxsource/summary.html>). If values were not available from Colorado or the Air Toxics Website, an effort was made to fill these data gaps using (in order of preference) IRIS (EPA's Integrated Risk Information System), PPTRVs (EPA's Provisional Peer-Reviewed Toxicity Values), and other applicable secondary sources (e.g., California EPA; ATSDR). Inhalation toxicity values were available for 21 out of 86 COPCs (Table A5). Therefore, 65 COPCs with no inhalation toxicity values were omitted altogether from any quantitative inhalation risk estimation (Tables A2 and A2.1).

4 Risk Characterization

Risk characterization is the culmination of the risk assessment process. It integrates the information from the exposure assessment and the toxicity assessment to provide an estimate of the magnitude of potential risks, and the strength of the conclusions based on the uncertainty in the information used to generate these estimates. As mentioned above, both cancer and noncancer health effects (acute and chronic) are evaluated in this risk assessment.

4.1 Cancer Risk Estimation

The lifetime cancer risk for each COPC at each monitoring location is derived by multiplying the 95th percent upper confidence limit on the mean of the monitored ambient air concentrations (Table A4) by the respective IUR value, as shown in the following equation.

$$\text{Risk}_x = \text{EPC}_x * \text{IUR}_x$$

Where:

Risk_x = the risk of the Xth COPC at a monitor;

EPC_x = the exposure point concentration of the substance (i.e., most stringent of the 95% UCL or maximum air concentration); and

IUR_x = the inhalation unit risk of the substance.

Estimates of cancer risk are expressed as a probability, represented in scientific notation as a negative exponent of 10. For example, an additional lifetime risk of contracting cancer of 1 chance in 1,000,000 (or one additional person in 1,000,000) is written as 1×10^{-6} . It can also be written as 1E-06 with an exponential notation.

The EPA classifies chemicals according to their carcinogenicity using an approach based on “weight of evidence”. For example, the chemical is classified as a known human carcinogen (Category A) when there is sufficient evidence from human studies. If the evidence from human studies is judged to be limited or inadequate, but there is sufficient evidence of carcinogenicity in animals, the chemical is classified as a probable human carcinogen (Category B).

The level of cancer risk that is of concern is a matter of individual, community, and regulatory judgment. However, the EPA typically considers risks below 1E-06 to be so small as to be negligible (USEPA 1991). Therefore, the EPA uses a cancer risk of one in a million (1E-06) as a regulatory goal, which means that regulatory programs are generally designed to try to reduce risk to this level. When it is not feasible to meet this regulatory goal, the EPA considers cancer risks lower than 1 in 10,000 (1E-04) to be acceptable.

4.2 NonCancer Hazard Estimation

In contrast to cancer risks, noncancer hazards are not expressed as a probability of an individual suffering an adverse effect. Instead, the noncancer hazard to individuals is expressed in terms of the hazard quotient (HQ). For a given air toxic, exposures below the reference concentration ($HQ < 1$) are not likely to be associated with an appreciable risk of adverse health effects. With exposures increasingly greater than the reference concentration, the potential for adverse effects increases. HQs are calculated as follows:

$$HQ_x = EPC_x / RfC_x$$

Where:

HQ_x = the hazard quotient of the X^{th} COPC at a monitor;

EPC_x = the exposure point concentration of the substance (i.e., most stringent of the 95% UCL or maximum air concentration); and

RfC_x = the reference concentration of the substance.

When used in the assessment of noncancer risks, the hazard quotient is commonly reported to one significant figure (USEPA, 1989). For example, a hazard quotient of 0.13 is rounded to 0.1, and a hazard quotient of 1.6 is rounded to 2.

4.3 Cumulative Risks for Multiple Chemicals

Emissions from oil and gas development activities represent a complex mixture of more than 100 aliphatic and aromatic hydrocarbons in the ambient air. These exposures may occur acutely or chronically, and commonly occur concurrently with exposure to other chemicals and stressors. The toxicity of chemicals in complex mixtures may differ greatly from that of a single compound. Therefore, estimating cancer risks or noncancer hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several compounds. The consequences of the multiple exposures can be quantified, within some limitations, based on EPA's default assumption of additivity.

For cancer risk, the individual chemical risks are added to estimate the total risk for the site. This summation is based upon the principle that the addition of each risk produces a combined total cancer risk estimate.

For non-carcinogenic chemicals, the hazard quotients for each exposure pathway can be summed to develop a hazard index (HI) for that exposure pathway. For screening purposes, it is acceptable to sum all HQ values in order to derive an HI value. If the resulting HI is less than one, no further evaluation is necessary and it can be concluded that no unacceptable risks are present. If the HI is greater than one as a consequence of summing several hazard quotients of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group.

4.4 Results of Risk Estimation

Since the toxicity values are available for 21 out of 86 COPCs, the health risk for 21 COPCs with known toxicity values are quantitatively estimated (Section 4.4.1), and the health risks for the remaining 68 COPCs are qualitatively evaluated (Section 4.4.2).

4.4.1 *Quantitative Evaluation of Potential Cancer and Noncancer Health Effects for COPCs with Known Toxicity Values*

Eighteen chemicals from the Bell, Brock, Parachute, and Rifle monitoring stations are carried through for evaluation in the risk characterization. Of these chemicals, 6 are evaluated for cancer risk and 20 are evaluated for noncancer hazards.

Cancer Risk Estimates

COPCs for cancer risk across the four monitoring sites include acetaldehyde, 1,3-butadiene, benzene, crotonaldehyde, ethylbenzene, and formaldehyde. The majority of these chemicals are classified as “known” or “probable” human carcinogens by the federal agencies (Table A5). The combined theoretical lifetime cancer risk estimates are found to range from 1.1E-04 (110 excess cancers per 1 million individuals) to 1.7E-04 (170 excess cancers per 1 million individuals) across the four monitoring sites. Crotonaldehyde, a possible human carcinogen, is one of the major contributors to the total cancer risk at each monitoring site, with risk estimates ranging from 6.0E-05 to 1.4E-04 (at the Brock monitoring site). Formaldehyde, a probable human carcinogen, is the second major contributor to the total risk at each monitoring site, with risk estimates ranging from 1.5E-05 to 2.8E-05 (at the Rifle monitoring site). Benzene, a known human carcinogen, is the third major contributor to the total cancer risk at each monitoring site, with risk estimates ranging from 7.5E-06 to 2.6E-05 (at the Parachute monitoring site). The cumulative cancer risk estimates for all 6 COPCs across the four monitoring locations are at or slightly above the high-end of EPA’s acceptable cancer risk range of one to one-hundred in a million. This suggests that the estimated cancer risks associated with the 6 COPCs are not likely to result in significant health impacts. Table 1 shows a comparison of cancer risks across all monitoring sites. The cumulative cancer risk estimates are similar across all four monitoring sites. However, the cumulative cancer risk estimates without crotonaldehyd are slightly higher (<2-fold) across both sites in the urban area than those in the rural area.

Noncancer Hazard Estimates: Chronic and Acute

COPCs for noncancer hazards across the four monitoring sites include acetaldehyde, acetone, 1,3-butadiene, benzene, cyclohexane, ethylbenzene, formaldehyde, hexane, isopropylbenzene, methylcyclohexane, nonane, propionaldehyde, propylene, propylbenzene, styrene, toluene, 1, 2, 4-trimethylbenzene, 1,3,5-trimethylbenzene, m-xylene/p-xylene, and o-xylene. The majority of these chemicals are known to affect the respiratory, immune, and/or nervous systems (Table A5). None of the individual chemicals that are assessed at any monitoring site are found to have a HQ exceeding a value of one for chronic or acute exposure durations. Table 1 shows a comparison of chronic HQs and HIs across all monitoring sites. None of the HIs exceed a value of one; however, HIs across both sites in the urban area are nearly equal to one (HI= 0.8 or 0.9).

The major contributing chemicals to this hazard index are acetaldehyde (HQ=0.2), formaldehyde (HQ= 0.2), 1,2,4-trimethylbenzene (HQ=0.2), 1,3,5-trimethylbenzene (0.1), and benzene (0.1). These chemicals are associated with effects on the respiratory, immune, and nervous systems. The cumulative HI of 1 indicates a low increased potential for respiratory, neurologic, and immunologic effects based on continuous exposure at the two urban locations.

Acute HQs for benzene, based on the maximum detected concentration and ATSDR's acute MRL of 30µg/m³, are found to be 0.1 (Brock), 0.5 (Bell), 0.4 (Parachute), and 0.1 (Rifle). Acute HQs are similar across the urban and rural oil & gas development areas. Overall, these results suggest that significant acute or chronic noncancer health effects are not likely to occur.

Table 1. Estimated Potential Lifetime Cancer Risks and Noncancer Hazards (HQ)

Compound	BELL		BROCK		PARACHUTE		RIFLE	
	Cancer Risk	HQ	Cancer Risk	HQ	Cancer Risk	HQ	Cancer Risk	HQ
Acetaldehyde	2.01E-06	0.1	1.96E-06	0.1	2.64E-06	0.1	3.81E-06	0.2
Acetone	NC	0.0001	NC	0.0001	NC	0.0001	NC	0.0001
1,3-Butadiene	1.59E-06	0.03	1.59E-06	0.03	3.33E-06	0.06	4.44E-06	0.07
Benzene	1.19E-05	0.05	7.52E-06	0.03	2.15E-05	0.09	1.45E-05	0.06
Crotonaldehyde*	8.4E-05	NA	1.4E-04	NA	6.0E-05	NA	1.0E-04	NA
Cyclohexane	NC	0.0010	NC	0.0004	NC	0.0008	NC	0.0005
Ethylbenzene	1.44E-06	0.0006	4.78E-07	0.0002	1.82E-06	0.0007	1.32E-06	0.0005
Formaldehyde	1.47E-05	0.1	1.53E-05	0.12	2.43E-05	0.2	2.76E-05	0.2
n-Hexane	NC	0.01	NC	0.007	NC	0.01	NC	0.007
Isopropylbenzene	NC	0.0002	NC	0.0002	NC	0.0002	NC	0.0002
Methylcyclohexane	NC	0.002	NC	0.002	NC	0.004	NC	0.002
n-Nonane	NC	0.004	NC	0.002	NC	0.014	NC	0.005
Propionaldehyde	NC	0.01	NC	0.01	NC	0.02	NC	0.02
Propylene	NC	0.0001	NC	0.0001	NC	0.0003	NC	0.0003
n-propylbenzene	NC	0.0001	NC	0.00007	NC	0.0002	NC	0.0002
Styrene	NC	0.0004	NC	0.0001	NC	0.0002	NC	0.0001
Toluene	NC	0.002	NC	0.0004	NC	0.002	NC	0.001
1,2,4-Trimethylbenzene	NC	0.04	NC	0.03	NC	0.2	NC	0.1
1,3,5-Trimethylbenzene	NC	0.03	NC	0.03	NC	0.1	NC	0.06
m-Xylene/p-Xylene	NC	0.02	NC	0.01	NC	0.05	NC	0.03
o-Xylene	NC	0.006	NC	0.002	NC	0.009	NC	0.007
Cumulative Risk	1.2E-04	0.40	1.7E-04	0.4	1.1E-04	0.9	1.5E-04	0.8

NC = NonCarcinogen; NA = Not Available

* The cancer risk estimates for crotonaldehyde are considered uncertain because these are calculated using EPA's oral cancer toxicity value (i.e., route-to-route extrapolation). See Section 5.2 for details of the uncertainty discussion.

4.4.2 Qualitative Evaluation of Potential Health Risks of COPCs Without Toxicity Values

As already mentioned above, cancer risks and noncancer hazards for 65 COPCs cannot be quantitatively estimated because inhalation toxicity values are not available. Of these 65 COPCs, 59 are comprised of alkanes and alkenes, and 6 are aldehydes. Alkanes and alkenes are the primary components of natural gas, petroleum and/or gasoline vapor.

It appears that the majority of the 59 COPCs are present at very low concentrations because the ambient air concentration attributable to nine COPCs (ethane, propane, n-butane, iso-butane, n-pentane, iso-pentane, n-decane, n-dodecane, and n-undecane) accounts for approximately 85% of the combined exposure point concentrations for all 59 COPCs. The highest EPCs for these nine COPCs at the Parachute site range between 13 and 117 $\mu\text{g}/\text{m}^3$ (Table A2.1).

At low concentrations, the toxicity of alkanes and alkenes is generally considered to be minimal (Sandmeyer, 1981). For example, the occupational exposure limits (NIOSH-RELS) for four COPCs (n-butane, iso-butane, n-propane, and n-pentane) range between 350,000 and 1,900,000 $\mu\text{g}/\text{m}^3$. It should, however, be noted that the occupational exposure limits are not intended to be used as acceptable levels for residential exposures that are evaluated in this assessment.

At high concentrations, health effects that are associated with alkanes and alkenes include acting as anesthetics and subsequently asphyxiants, showing narcotic or other central nervous system depression effects, and dermal and pulmonary irritation. Unlike the alkanes, the alkenes do not exhibit neurotoxic properties (Sandmeyer, 1981). Some aliphatic hydrocarbons (propane, butane and isobutane) may be weak cardiac sensitizers in humans following inhalation exposures to high concentrations (greater than 5% for isobutane and greater than 10% for propane).

Six out of 65 COPCs include aldehydes, which generally act as irritants of the eyes, skin, and respiratory tract. It is important to note that some aldehydes have also been shown to be mutagenic and/or carcinogenic. The variation in toxicity among the individual aldehydes is large. Investigations are needed to further characterize the health effects of the common aldehydes.

Overall, based on the qualitative evaluation of health risks, it appears that exposure to 65 COPCs individually is not likely to result in significant cancer and noncancer effects, but the cumulative health effects of the 65 COPCs cannot be estimated. It should be noted that the current state of the science is unable to assess exposures to complex mixtures of air toxics, especially, synergistic and antagonistic interactions at low levels.

5 Uncertainty Evaluation

The risk estimates presented in this air toxic risk assessment are not fully probabilistic estimates but conditional estimates given a considerable number of assumptions about exposure and toxicity. Therefore, quantitative evaluation of the risks to humans from environmental contamination is frequently limited by uncertainty (lack of knowledge) regarding a number of important exposure and toxicity factors. This lack of knowledge is usually addressed by making estimates based on whatever limited data that are available, or by making assumptions based on professional judgment when no reliable data are available. Because of these assumptions and estimates, the results of risk calculations are themselves uncertain, and it is important for risk managers and the public to keep this in mind when interpreting the results of a risk assessment. A qualitative discussion of the major uncertainties in this risk assessment is provided below.

5.1 Uncertainties in Exposure Assessment

5.1.1 Air Monitoring

The collection and monitoring of ambient air is one of the largest sources of uncertainty. The uncertainty stems from the inability to realistically monitor continuously at all places of interest. Thus, a decision is made to monitor a portion of the time and in specific locations and apply the results to a broader area. The monitoring data at each station reflect one year of chemical concentrations in air. It is uncertain how well this dataset reflects the lifetime exposure assumed in this risk assessment as changes in meteorology and chemical emissions could lead to lower or higher concentrations in air from year-to-year. To reduce this uncertainty would require monitoring over several years, or modeling based on changes in meteorology and chemical emissions.

Monitoring locations may or may not be representative of air concentrations to which an individual is exposed in the breathing zone 24 hours a day for a lifetime. Some of these monitoring locations were placed in areas with mixed industrial use or heavier traffic patterns. Potential health impacts associated with contaminant concentrations at these locations could over estimate the true risk since they may not reflect the actual long-term residential exposure concentration. Additionally, they could underestimate true risk to people living near sources of high concentrations of contaminant emissions.

Finally, a large uncertainty stems from inability to monitor intermittent peak exposure. The nature of oil and gas operations is such that emissions vary strongly with time. To reduce this uncertainty, short-term air monitoring is needed.

Air Quality Data

Overall, the available data for all VOCs is of high analytical quality. For the GCPHD 2008 study, sampling was conducted on a once every 6th day for the speciated non-methane organic compounds (approximately 60 samples per year) and once every 12th day for the carbonyls (approximately 30 samples per year). While this follows general EPA protocols, the quantity of data is less than ideal for a robust statistical analysis on a

one-year basis and can lead to an increased uncertainty. The ideal scenario would be to have sampling conducted daily.

5.1.2 Other Exposure Sources

It is important to note that indoor sources such as paints, home furnishings, cleaning products, building materials, and other indoor sources of air toxics are not evaluated in this assessment. Many chemicals have been shown to accumulate in indoor environments, which could increase exposure. In addition, there are other multiple local outdoor emission sources that can impact air quality in the Garfield County. Among these are mobile and other stationary sources (e.g., traffic along the I-70 corridor, seasonal forest fires, and mining of coal and uranium). The contribution from different outdoor sources is not evaluated in this assessment. However, the findings of the 2008 air quality monitoring report indicated that some of the primary chemicals related to petroleum and natural gas emission sources are higher in Garfield County than in areas outside the County. Specifically,

- Concentrations of light alkanes (ethane, propane, butane, and pentane) were 2 to 5 times higher across the four sites in Garfield County than sites outside of Garfield County (GCPHD, 2009). These alkanes are the primary components of natural gas.
- Concentrations of benzene, toluene, ethylbenzene, and/or m/p-xylenes (BTEX) across the four sites in Garfield County were higher than most averages reported across the United States. Some or all of the BTEX compounds were higher than the nearby, more urban, Grand Junction site. These compounds are the primary components of petroleum.
- Concentrations of styrene and n-hexane, especially, at the Bell site were higher than other Garfield sites, and higher than most regional sites.

It should be noted that the measured concentrations of carbonyls (e.g., aldehydes) at the Garfield County sites were lower than most other sites in the United States, and lower than the nearby urban Grand Junction site.

5.1.3 Exposure Parameters

Another source of uncertainty in estimating exposures is the assumption that all individuals will receive the same amount of chemical. This assumption does not take into account variability in parameters such as breathing rates, absorption rates, body weight, lung surface area, and frequency of exposure. This range of variability is, however, difficult to assess. Therefore, standard EPA default factors representing the upper limit of exposure parameters are used in this assessment.

5.1.4 Air Toxics of Potential Concern

Approximately 90 chemicals were both monitored and analyzed. However, there are additional chemicals (e.g., metals, halogenated hydrocarbons, and polycyclic aromatic hydrocarbons) that may need to be monitored and analyzed to fully understand the potential risks associated with oil and gas activities in the region. In view of this situation, it is possible that this evaluation may underestimate the potential risks posed by oil and gas activities.

5.2 Uncertainties in Toxicity Assessment

One of the most important sources of uncertainty in a risk assessment is in the RfC values used to evaluate noncancer risk and in the inhalation unit risk values used to quantify cancer risk. In many cases, these values are derived from a limited toxicity database, and this can result in substantial uncertainty, both quantitatively and qualitatively. In order to account for these and other uncertainties associated with the evaluation of toxicity data, both RfCs and IURs are derived by the EPA in a way that is intentionally conservative; that is, risk estimates based on these RfCs and IURs are more likely to overestimate risk. Three major sources of uncertainty in this toxicity assessment are briefly discussed below.

First, the largest source of uncertainty in this toxicity assessment is unavailability of toxicity values for 65 out of 86 COPCs. In addition, 4 compounds (2-hexene, 2,5-dimethylbenzaldehyde, 2-ethyl-1-butene, and propyne) that were not detected are not evaluated at half the detection limit because no toxicity values are available. Thus, cancer risks and noncancer hazards are likely to be underestimated for monitored air toxics.

Second, the EPA has calculated a range of IURs for benzene between 2.2×10^{-6} and 7.8×10^{-6} per $\mu\text{g}/\text{m}^3$. The upper-bound value is used in this evaluation in accordance with the EPA Air Toxic guidance, which may slightly overestimate risk (up to 3-fold). The set of risk estimates falling within this interval reflects both the inherent uncertainties in the risk assessment of benzene and the limitations of the epidemiologic studies in determining dose-response and exposure data.

Third, uncertainties in the EPA cancer toxicity value (i.e., IURs) for crotonaldehyde are notable. The EPA Integrated Risk Information System (IRIS) has not calculated an inhalation cancer toxicity value (IUR) for crotonaldehyde. However, crotonaldehyde is evaluated using a cancer toxicity value derived in the EPA Health Effects Assessment Summary Tables from oral exposure studies. Although conversion of oral dose-response information to inhalation exposure is not optimal risk assessment practice, the alternative would be to omit this substance altogether from any quantitative evaluation. Crotonaldehyde is classified as a possible human carcinogen (Category C). This classification was assigned based on the increased incidence of hepatic neoplastic nodules and hepatocellular carcinomas in only one available animal carcinogenicity study that was limited by only one sex of one species. There is insufficient evidence that inhalation is a route that results in crotonaldehyde-induced liver lesions or neoplasia. Given the substantial uncertainty surrounding this toxicity value, the total cancer risk estimates are recalculated without crotonaldehyde for the four monitoring sites. The total cancer risks without crotonaldehyde (and with crotonaldehyde) are noted below.

- Bell 3.2E-05 (1.2E-04 with crotonaldehyde; 65% contribution)
- Brock 2.7E-05 (1.7E-04 with crotonaldehyde; 82% contribution)
- Parachute 5.3E-05 (1.1E-04 with crotonaldehyde; 54% contribution)
- Rifle 5.2E-05 (1.5E-04 with crotonaldehyde; 67% contribution)

It should be noted that the total cancer risks at all monitoring sites remain above the mid-point of EPA's acceptable cancer risk range of 1E-06 to 1E-04 even when crotonaldehyde is excluded.

5.3 Uncertainty in Risk Estimation due to Multiple Contaminants

Interactions among components within petroleum, as well as other air and chemical exposures are not well understood. However, the interactions among the components of petroleum should be considered since petroleum may contain several hundred hydrocarbons. The hydrocarbons present in the petroleum mixture principally include alkanes, alkenes, and aromatic BTEX compounds. Therefore, the number of possible interactions in a complex mixture of petroleum is very large. However, it is not possible to reliably predict the effects of these complex interactions.

Both carcinogenic and noncarcinogenic risks for multiple contaminants are assumed to be additive, in accordance with the EPA guidance for health risk assessment of chemical mixtures. This assumption, however, is associated with several limitations and, therefore, there is potential for under- or over-estimation of risk. For example, the assumption of additivity of risk does not account for synergistic or antagonistic chemical interactions.

6 Conclusions

At the request of the GCPHD, CDPHE conducted a screening-level risk assessment of potential human health impacts from inhalation of air toxics using the 2008 ambient air quality data collected by the GCPHD. It should be noted that this risk assessment report is a part of the ongoing joint APCD and DCEED assistance to the GCPHD. CDPHE conducted this risk assessment in accordance with the EPA air toxic risk assessment methodology. Overall, due to typical uncertainties and limitations in the methods used to assess exposure and toxicity, this investigation is best viewed as a "snapshot" of air quality. Further monitoring and analysis would be required for more than a "snapshot" view.

The available information suggests a potential for public health impacts across the oil and gas development areas in Garfield County because of the following:

- The estimated cumulative lifetime cancer risks for the 6 air toxics with known toxicity values are at or slightly above the high-end of EPA's acceptable cancer risk range of 1 to 100 excess cancers in a million (1E-06 to 1E-04) across all monitoring sites. These total risk estimates are based on all carcinogenic chemicals including crotonaldehyde, which as discussed in Section 5.2 has a highly uncertain cancer toxicity value. It should be noted that the total cancer risks at all monitoring sites remain above the mid-point of EPA's acceptable cancer risk range of 1E-06 to 1E-04 even when crotonaldehyde is excluded. The major contributors to this risk are formaldehyde and benzene. The

estimated risks with and without crotonaldehyde indicate a low to moderate increased risk of developing cancer during a lifetime. Overall, it is important to note that the cancer risks are likely to be underestimated in this assessment because cancer toxicity values are only available for a small number of air toxics.

- Each of the 20 individual air toxics assessed at any monitoring site have a chronic noncancer hazard estimate well below an acceptable value of one. However, when accounting for the cumulative chronic noncancer hazards for all of these 20 air toxics, the chronic noncancer hazard estimate is just below the acceptable level of one across the two monitoring sites. The major contributing chemicals to the cumulative hazard estimate are acetaldehyde, formaldehyde, trimethylbenzenes, and benzene. This indicates a low increased risk of developing noncancer health effects (e.g., respiratory, immunological, and nervous system effects). Overall, it is important to note that the noncancer hazards are likely to be underestimated in this assessment because noncancer toxicity values are not available for 65 air toxics.
- Based on the available 24-hour air monitoring data, the estimated acute noncancer hazards for benzene are well below an acceptable value of one indicating a low increased potential for acute health effects of benzene (e.g., immune system effects).
- The cumulative health impacts of 86 detected ambient air toxics cannot be determined due to the absence of toxicity values for 65 air toxics.

7 Identification of Key Data Gaps

The areas where future data collection and research efforts need to focus are identified below.

- Short-term (acute) air sample of less than 24-hr duration are needed in order to evaluate health risks posed by intermittent peak exposures.
- Source-specific air monitoring data are needed in order to determine the contribution of various local sources.
- Air monitoring data are needed for all contaminants of potential concern (e.g., metals, polycyclic aromatic hydrocarbons, and Tentatively Identified Compounds).
- Direct measurements of air concentrations for toxics in the breathing zone are needed. Personal air monitors provide information on exposure to that individual, rather than to the general area in which an individual might be moving.
- Toxicity values are needed for 65 air toxics.

- Research studies are needed to assess exposures to complex mixtures of air toxics, especially, synergistic and antagonistic interactions at low levels.

8 Recommendations

The findings of this risk assessment support the need for the following:

- Continued long-term air monitoring; increased frequency of sampling; and development of a complete list of contaminants associated with oil and gas development.
- Short-term (acute) air monitoring by collecting 1-hour air samples in order to evaluate health risks posed by intermittent peak exposures.
- Source apportionment including sources other than the oil and gas operations, such as stationary industrial sources and mobile traffic sources.
- Continued management of the risk posed by potential exposures to air toxics as a result of increase in oil and gas development activities (e.g., additional monitoring, sample analysis, and action as appropriate).

9 References

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Appendix A

Data Summary for All Chemicals of Potential Concern (COPCs) and Toxicity Values

Table A1. Chemicals with Toxicity Values at the Bell and Brock Sites in the Rural Oil & Gas Development Area.

Compound	BELL			BROCK		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
Acetaldehyde	1.964	100.0%	0.943	1.591	100.0%	0.889
Acetone	5.392	100.0%	3.113	6.366	100.0%	3.269
Formaldehyde	2.237	100.0%	1.128	2.102	100.0%	1.175
1,3-Butadiene	0.053	5.1%	0.053	0.053	1.7%	0.053
Benzene	13.631	100.0%	1.521	2.401	100.0%	0.964
Crotonaldehyde	0.467	93.5%	0.155	0.519	100.0%	0.253
Cyclohexane	104.985	100.0%	5.010	5.347	100.0%	2.413
Ethylbenzene	4.337	96.6%	0.576	0.482	96.6%	0.191
n-Hexane	22.089	100.0%	7.319	24.262	100.0%	4.606
Isopropylbenzene	0.298	22.0%	0.090	0.094	18.6%	0.084
Methylcyclohexane	21.973	100.0%	6.812	9.810	100.0%	4.855
n-Nonane	2.501	100.0%	0.786	1.463	100.0%	0.487
Propionaldehyde	0.204	96.8%	0.097	0.183	100.0%	0.091
Propylene	0.597	100.0%	0.287	0.757	100.0%	0.295
Propylbenzene	0.710	81.4%	0.101	0.164	76.3%	0.074
Styrene	3.445	5.1%	0.374	0.431	15.3%	0.088
Toluene	79.140	100.0%	9.371	4.883	100.0%	2.226
1,2,4-Trimethylbenzene	3.091	100.0%	0.304	0.661	100.0%	0.211
1,3,5-Trimethylbenzene	0.836	84.7%	0.185	0.412	72.9%	0.159
m-Xylene/p-Xylene	9.879	100.0%	1.608	3.707	100.0%	1.179
o-Xylene	3.610	100.0%	0.577	0.522	100.0%	0.232

TableA1.1. Chemicals with Toxicity Values at the Parachute and Rifle Sites in the Urban Oil & Gas Development Area

Compound	PARACHUTE			RIFLE		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
Acetaldehyde	1.838	100.0%	1.201	2.901	100.0%	1.732
Acetone	5.915	100.0%	3.709	6.746	100.0%	3.988
Formaldehyde	3.257	100.0%	1.865	4.818	100.0%	2.124
1,3-Butadiene	0.033	52.5%	0.111	0.486	81.7%	0.148
Benzene	11.076	100.0%	2.755	4.079	100.0%	1.862
Crotonaldehyde	0.238	100.0%	0.110	0.436	100.0%	0.186
Cyclohexane	13.080	100.0%	4.721	7.401	100.0%	2.811
Ethylbenzene	2.616	100.0%	0.726	1.167	100.0%	0.526
n-Hexane	18.799	100.0%	6.940	15.920	100.0%	5.110
Isopropylbenzene	0.250	40.7%	0.099	0.120	51.7%	0.080
Methylcyclohexane	35.283	100.0%	11.300	14.343	100.0%	5.494
Nonane	13.348	100.0%	2.727	2.285	100.0%	0.916
Propionaldehyde	0.283	93.1%	0.134	0.371	93.5%	0.192
Propylene	1.417	100.0%	0.765	2.782	100.0%	0.973
Propylbenzene	1.092	96.6%	0.213	0.326	95.0%	0.164
Styrene	1.917	15.3%	0.258	0.352	28.3%	0.090
Toluene	118.441	100.0%	11.830	15.020	100.0%	4.890
1,2,4-Trimethylbenzene	7.374	100.0%	1.124	1.595	100.0%	0.690
1,3,5-Trimethylbenzene	5.347	98.3%	0.765	0.803	100.0%	0.361
m-Xylene/p-Xylene	11.833	100.0%	4.543	5.916	100.0%	2.612
o-Xylene	3.175	100.0%	0.911	1.623	100.0%	0.709

Table A2. Chemicals with No Toxicity Values Measured at the Bell and Brock Monitoring Sites.

Compound	BELL			BROCK		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
1,2,3-Trimethylbenzene	0.841	39.0%	0.098	0.135	42.4%	0.070
1-Decene	0.057	0.0%	n/a	0.057	0.0%	n/a
1-Dodecene	0.998	27.1%	0.175	1.503	22.0%	0.320
1-Heptene	2.484	96.6%	0.781	1.113	91.5%	0.497
1-Hexene	0.221	64.4%	0.102	0.222	67.8%	0.098
1-Nonene	0.426	55.9%	0.117	0.252	44.1%	0.100
1-Octene	1.365	20.3%	0.223	0.232	22.0%	0.101
1-Pentene	0.322	96.6%	0.109	0.256	100.0%	0.107
1-Tridecene	0.133	3.4%	0.121	0.120	1.7%	0.120
1-Undecene	0.205	10.2%	0.057	0.349	16.9%	0.070
2,2,3-Trimethylpentane	1.635	49.2%	0.288	0.281	50.8%	0.129
2,2,4-Trimethylpentane	2.155	33.9%	0.394	0.940	52.5%	0.233
2,2-Dimethylbutane	2.338	100.0%	0.776	1.022	100.0%	0.428
2,3,4-Trimethylpentane	1.793	57.6%	0.228	0.280	55.9%	0.086
2,3-Dimethylbutane	4.935	100.0%	1.540	1.845	100.0%	0.787
2,3-Dimethylpentane	1.850	100.0%	0.612	0.820	98.3%	0.383
2,4-Dimethylpentane	1.095	100.0%	0.426	0.509	100.0%	0.263
2-Ethyl-1-butene	0.123	0.0%	n/a	0.123	0.0%	n/a
2-Methyl-1-butene	2.455	45.8%	0.610	2.903	47.5%	0.647
2-Methyl-1-pentene	0.152	3.4%	0.125	0.123	3.4%	0.123
2-Methyl-2-butene	0.417	39.0%	0.136	0.248	50.8%	0.101
2-Methylheptane	2.926	100.0%	0.820	1.267	98.3%	0.528
2-Methylhexane	4.842	100.0%	1.653	2.535	100.0%	1.092
2-Methylpentane	20.561	100.0%	6.728	10.339	100.0%	3.619
3-Methyl-1-butene	0.200	1.7%	0.064	1.073	8.5%	0.113
3-Methylheptane	3.533	100.0%	0.544	0.899	100.0%	0.352
3-Methylhexane	4.403	100.0%	1.548	2.160	98.3%	1.015
3-Methylpentane	10.574	100.0%	3.501	10.104	100.0%	2.210
4-Methyl-1-pentene	4.676	20.3%	0.547	0.418	11.9%	0.140
Acetylene	1.816	100.0%	0.600	1.108	100.0%	0.576
a-Pinene	3.365	79.7%	0.463	1.008	59.3%	0.277
b-Pinene	1.432	3.4%	0.118	1.605	16.9%	0.322
cis-2-Butene	0.153	39.0%	0.063	0.185	54.2%	0.073
cis-2-Hexene	0.700	22.0%	0.146	0.123	11.9%	0.121
cis-2-Pentene	0.145	13.6%	0.061	0.079	22.0%	0.057
Cyclopentane	2.937	100.0%	0.907	1.021	100.0%	0.460
Cyclopentene	0.669	76.3%	0.235	0.825	66.1%	0.218
Ethane	411.389	100.0%	103.400	193.703	100.0%	63.740
Ethylene	1.514	100.0%	0.735	1.744	100.0%	0.768
Isobutane	118.261	100.0%	32.020	32.626	100.0%	12.300

Table A2. Continued (Chemicals with no Toxicity Values)

Compound	BELL			BROCK		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
Isobutene/1-Butene	4.727	79.7%	1.685	5.341	81.4%	2.372
Isopentane	123.349	93.2%	39.230	32.578	91.5%	12.300
Isoprene	3.332	52.5%	0.724	0.964	52.5%	0.306
m-Diethylbenzene	0.530	30.5%	0.118	0.369	27.1%	0.085
Methylcyclopentane	8.892	100.0%	3.266	4.567	100.0%	1.938
m-Ethyltoluene	1.628	98.3%	0.202	8.739	100.0%	1.727
n-Butane	136.684	100.0%	35.460	34.587	100.0%	13.630
n-Decane	69.831	100.0%	6.799	1.158	100.0%	0.442
n-Dodecane	71.407	100.0%	9.256	2.049	98.3%	0.598
n-Heptane	9.543	100.0%	3.231	4.713	100.0%	2.078
n-Nonane	2.501	100.0%	0.786	1.463	100.0%	0.487
n-Octane	5.665	100.0%	1.868	3.305	100.0%	1.233
n-Pentane	61.970	100.0%	17.390	35.057	100.0%	8.222
n-Tridecane	3.828	33.9%	0.492	0.463	32.2%	0.147
n-Undecane	254.561	100.0%	31.790	1.871	100.0%	0.707
o-Ethyltoluene	1.202	71.2%	0.247	0.563	59.3%	0.174
p-Diethylbenzene	0.421	18.6%	0.058	0.714	11.9%	0.104
p-Ethyltoluene	0.907	96.6%	0.202	0.274	88.1%	0.110
Propane	315.646	100.0%	82.470	98.602	100.0%	35.500
Propyne	0.350	1.7%	0.063	0.049	0.0%	n/a
trans-2-Butene	3.345	69.5%	0.367	0.262	69.5%	0.120
trans-2-Hexene	0.123	0.0%	n/a	0.123	0.0%	n/a
trans-2-Pentene	0.318	49.2%	0.081	0.170	52.5%	0.074
2,5-Dimethylbenzaldehyde	0.005	0.0%	n/a	0.005	0.0%	0.005
Benzaldehyde	0.195	96.8%	0.085	0.217	92.6%	0.094
Butyraldehyde	0.218	93.5%	0.092	0.177	92.6%	0.085
Hexaldehyde	0.098	74.2%	0.092	0.172	81.5%	0.071
Isovaleraldehyde	0.113	9.7%	0.026	0.074	3.7%	0.018
Tolualdehydes	0.251	93.5%	0.094	0.256	100.0%	0.130
Valeraldehyde	0.081	48.4%	0.066	0.063	55.6%	0.062

Table A2.1. Chemicals with No Toxicity Values Measured at the Parachute and Rifle Monitoring Sites.

Compound	PARACHUTE			RIFLE		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
1,2,3-Trimethylbenzene	3.485	91.5%	0.503	0.358	90.0%	0.150
1-Decene	0.057	0.0%	n/a	0.057	0.0%	n/a
1-Dodecene	7.114	76.3%	1.609	0.981	36.7%	0.203
1-Heptene	2.467	93.2%	1.068	1.675	96.7%	0.655
1-Hexene	0.200	74.6%	0.099	0.182	85.0%	0.101
1-Nonene	1.899	84.7%	0.248	0.410	68.3%	0.117
1-Octene	1.021	32.2%	0.282	0.524	30.0%	0.123
1-Pentene	0.648	96.6%	0.172	0.981	98.3%	0.253
1-Tridecene	0.282	5.1%	0.127	0.120	3.3%	0.120
1-Undecene	1.228	16.9%	0.216	0.278	15.0%	0.066
2,2,3-Trimethylpentane	1.069	89.8%	0.397	0.467	75.0%	0.252
2,2,4-Trimethylpentane	3.632	39.0%	0.576	0.940	100.0%	0.213
2,2-Dimethylbutane	1.921	100.0%	0.859	1.439	100.0%	0.596
2,3,4-Trimethylpentane	0.392	78.0%	0.138	0.339	90.0%	0.130
2,3-Dimethylbutane	3.713	100.0%	1.512	2.820	100.0%	1.132
2,3-Dimethylpentane	4.104	100.0%	0.899	1.288	100.0%	0.603
2,4-Dimethylpentane	1.499	100.0%	0.549	0.831	100.0%	0.408
2-Ethyl-1-butene	0.123	0.0%	n/a	0.123	0.0%	n/a
2-Methyl-1-butene	2.639	78.0%	0.804	4.394	88.3%	0.709
2-Methyl-1-pentene	0.177	10.2%	0.123	0.181	36.7%	0.111
2-Methyl-2-butene	1.342	79.7%	0.223	1.819	96.7%	0.417
2-Methylheptane	4.911	100.0%	1.654	1.962	100.0%	0.783
2-Methylhexane	12.002	98.3%	2.760	3.425	100.0%	1.591
2-Methylpentane	14.921	100.0%	6.135	11.808	100.0%	5.029
3-Methyl-1-butene	0.209	3.4%	0.067	0.314	8.3%	0.088
3-Methylheptane	3.749	100.0%	1.291	1.314	100.0%	0.584
3-Methylhexane	16.920	100.0%	2.894	3.431	100.0%	1.530
3-Methylpentane	8.753	100.0%	3.576	7.167	100.0%	2.800
4-Methyl-1-pentene	0.254	25.4%	0.129	0.344	25.0%	0.144
Acetylene	2.498	100.0%	1.302	4.968	100.0%	1.865
a-Pinene	6.018	88.1%	0.472	0.830	88.3%	0.292
b-Pinene	2.017	8.5%	0.270	0.168	1.7%	0.061
cis-2-Butene	0.481	91.5%	0.144	1.876	100.0%	0.519
cis-2-Hexene	0.223	18.6%	0.122	0.363	21.7%	0.140
cis-2-Pentene	0.352	66.1%	0.086	0.895	91.7%	0.171
Cyclopentane	2.679	100.0%	0.841	1.721	100.0%	0.652
Cyclopentene	1.109	76.3%	0.301	0.658	90.0%	0.214
Ethane	318.535	100.0%	116.900	204.772	100.0%	74.860
Ethylene	4.210	98.3%	2.039	7.801	98.3%	2.381
Isobutane	274.556	100.0%	43.190	46.948	100.0%	17.350

Table A2.1. Continued (Chemicals with no toxicity values)

Compound	PARACHUTE			RIFLE		
	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$	Max. Concentration $\mu\text{g}/\text{m}^3$	% Samples Detected	EPC $\mu\text{g}/\text{m}^3$
Isobutene/1-Butene	6.483	78.0%	3.691	7.057	85.0%	2.462
Isopentane	125.120	96.6%	34.020	40.369	95.0%	17.810
Isoprene	1.588	81.4%	0.615	1.817	96.7%	0.579
m-Diethylbenzene	2.256	66.1%	0.325	0.708	61.7%	0.144
Methylcyclopentane	10.040	100.0%	3.858	6.081	100.0%	2.492
m-Ethyltoluene	2.458	100.0%	0.589	0.961	100.0%	0.467
n-Butane	54.317	100.0%	21.710	53.366	100.0%	19.790
n-Decane	112.893	100.0%	13.150	1.688	100.0%	0.820
n-Dodecane	82.437	100.0%	16.420	3.576	100.0%	0.834
n-Heptane	19.437	100.0%	5.281	7.025	100.0%	2.644
n-Octane	12.556	100.0%	4.393	4.684	100.0%	1.825
n-Pentane	150.498	100.0%	16.640	34.703	100.0%	11.050
n-Tridecane	5.371	57.6%	0.826	0.748	51.7%	0.167
n-Undecane	225.501	100.0%	36.800	3.877	100.0%	0.991
o-Ethyltoluene	6.336	96.6%	0.501	0.484	98.3%	0.257
p-Diethylbenzene	1.751	39.0%	0.232	0.184	48.3%	0.078
p-Ethyltoluene	3.457	100.0%	0.447	0.545	100.0%	0.257
Propane	155.719	100.0%	59.030	128.663	100.0%	42.280
Propyne	0.049	0.0%	n/a	0.049	0.0%	n/a
trans-2-Butene	1.050	94.9%	0.289	1.922	100.0%	0.602
trans-2-Hexene	0.209	6.8%	0.126	0.212	35.0%	0.116
trans-2-Pentene	0.906	93.2%	0.157	1.790	100.0%	0.354
2,5-Dimethylbenzaldehyde	0.005	0.0%	0.005	0.005	0.0%	n/a
Benzaldehyde	0.247	100.0%	0.131	0.313	100.0%	0.148
Butyraldehyde	0.711	93.1%	0.233	0.360	100.0%	0.179
Hexaldehyde	0.221	86.2%	0.102	0.348	100.0%	0.131
Isovaleraldehyde	0.159	10.3%	0.033	0.134	22.6%	0.076
Tolualdehydes	0.226	96.6%	0.120	0.246	100.0%	0.162
Valeraldehyde	0.113	72.4%	0.060	0.208	80.6%	0.139

Table A3. Selection of COPCs for Acute Risk Evaluation: Comparison of the highest maximum concentration with chronic noncancer toxicity values.

Compound	Maximum Concentration (µg/m ³)	Chronic Toxicity Value (µg/m ³)	Acute COPC Selected
Acetaldehyde	2.9	9	No
Acetone	6.7	30,000	No
1,3-Butadiene	0.49	2	No
Benzene	13.6	10	Yes
Crotonaldehyde	0.52	NA	No
Cyclohexane	105.0	6000	No
Ethylbenzene	4.3	1000	No
Formaldehyde	4.8	9	No
n-Hexane	24.2	700	No
Isopropylbenzene	0.298	400	No
Methylcyclohexane	35.3	3010	No
Nonane	13.348	200	No
Propionaldehyde	0.4	8	No
Propylene	2.8	3000	No
Propylbenzene	1.09	1000	No
Styrene	3.4	1000	No
Toluene	118.4	5000	No
1,2,4-Trimethylbenzene	7.3	7	Yes
1,3,5-Trimethylbenzene	5.3	6	No
m-Xylene/p-Xylene	11.8	100	No
o-Xylene	3.6	100	No

Table A4. Exposure Point Concentrations (EPC) for COPCs with Toxicity Values.

Compound	Bell EPC (µg/m³)	Brock EPC (µg/m³)	Parachute EPC (µg/m³)	Rifle EPC (µg/m³)
Acetaldehyde	0.943	0.889	1.201	1.732
Acetone	3.113	3.269	3.709	3.988
1,3-Butadiene	0.053	0.053	0.111	0.148
Benzene	1.521	0.964	2.755	1.862
Crotonaldehyde	0.155	0.253	0.110	0.186
Cyclohexane	5.010	2.413	4.721	2.811
Ethylbenzene	0.576	0.191	0.726	0.526
Formaldehyde	1.128	1.175	1.865	2.124
n-Hexane	7.319	4.606	6.940	5.110
Isopropylbenzene	0.090	0.084	0.099	0.080
Methylcyclohexane	6.812	4.855	11.300	5.494
Nonane	0.786	0.487	2.727	0.916
Propionaldehyde	0.097	0.091	0.134	0.192
Propylene	0.287	0.295	0.765	0.973
Propylbenzene	0.101	0.074	0.213	0.164
Styrene	0.374	0.088	0.258	0.090
Toluene	9.371	2.226	11.830	4.890
1,2,4- Trimethylbenzene	0.304	0.211	1.124	0.690
1,3,5- Trimethylbenzene	0.185	0.159	0.765	0.361
m-Xylene/p-Xylene	1.608	1.179	4.543	2.612
o-Xylene	0.577	0.232	0.911	0.709

Table A5. Cancer and Noncancer Toxicity Values for COPCs: Inhalation Unit Risk (IUR) and Chronic Reference Level (RfC).

Compound	Cancer		Noncancer	
	IUR ($\mu\text{g per m}^3$) ⁻¹	Cancer classification	Chronic RfC ($\mu\text{g/m}^3$)	Target organ (Critical effect)
Acetaldehyde	0.0000022 I	Probable human carcinogen(B2) <i>Nasal and laryngeal tumors in animals</i>	9.0 I	Respiratory (<i>Degeneration of Olfactory epithelium</i>)
Acetone	NC	NC	30000.0 A	Neurological (<i>delayed visual reaction, general weakness, headache</i>)
1,3-Butadiene	0.00003 I	Known human carcinogen(A) <i>Lymphohematopoietic cancer and leukemia in humans</i>	2.0 I	Reproductive (<i>Ovarian atrophy</i>)
Benzene	0.0000078 I	Known human carcinogen (A) Leukemia in humans	30.0 I	Immunological (<i>Decreased lymphocyte count</i>)
Crotonaldehyde	0.000543 ^a H	Possible human carcinogen (C) <i>Hepatic neoplastic nodules and hepatocellular carcinoma in animals (oral study)</i>	NA	NA
Cyclohexane	NC	NC	6000.0 I	Reproductive/developmental (<i>Reduced pup weight</i>)
Ethylbenzene	0.0000025 C	Probable human carcinogen(B2) Renal tumors in animals	1000.0 I	Developmental (<i>Kit mortality</i>)
Formaldehyde	0.000013 I	Probable human carcinogen(B1) <i>Nasopharyngeal and lung cancer in humans (limited) and nasal cancer in animals</i>	9.8 A	Respiratory (<i>Histopathological changes in nasal tissue in humans</i>)
n-Hexane	NC		700.0 I	Neurological (<i>Peripheral neuropathy</i>)
Isopropylbenzene (cumen)	NC		400.0 I	Renal/Adrenal (<i>Increased kidney and adrenal weight</i>)
Methylcyclohexane	NC		3010.0 H	Renal (<i>Mineralization, papillary hyperplasia in animals</i>)
Nonane	NC		200.0 P	Salivation, lacrimation, and reduced body weight
Propionaldehyde	NC		8.0 I	Respiratory (<i>Atrophy of olfactory epithelium</i>)
Propylene	NC		3000.0 C	Respiratory (<i>Squamous metaplasia, epithelial hyperplasia, and inflammation of the nasal cavity in animals</i>)
Propylbenzene	NC		1000.0 P	Derived using ethylbenzene as a surrogate
Styrene	NC		1000.0 I	Neurological (<i>Increase in reaction time, decreased memory, impaired visual perception</i>)
Toluene	NC		5000.0 I	Neurological and respiratory (<i>Neurological effects; other effects: degeneration of nasal epithelium</i>)
1,2,4-Trimethylbenzene	NC		7.0 I	Neurologic, Respiratory, Immunologic (<i>Vertigo, headaches, drowsiness, anemia, altered blood clotting, chronic asthma-like bronchitis</i>)
1,3,5-Trimethylbenzene	NC		6.0 I	Neurologic, Respiratory, Immunologic (<i>Vertigo, headaches, drowsiness, anemia, altered blood clotting, chronic asthma-like bronchitis</i>)
m-Xylene/p-Xylene	NC		100.0 I	Neurological (<i>Impaired motor coordination</i>)
o-Xylene	NC		100.0 I	Neurological (<i>Impaired motor coordination</i>)

Note: Sources of toxicity values: A= ATSDR –Minimal Risk Level (MRL); C = Cal EPA; H= EPA-Heast; I- EPA IRIS; P= PPRTV

^a Based on route -to-route extrapolation of EPA's oral cancer slope factor

NC = Non Carcinogen; NA= Not Available