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Non-Methane Hydrocarbon Source Apportionment and BTEX Risk Assessment of Winter 2015 in Roosevelt, Utah

Jerimiah Lamb
Utah State University

NON-METHANE HYDROCARBON SOURCE APPORTIONMENT AND BTEX RISK ASSESSMENT OF WINTER 2015 IN ROOSEVELT, UTAH

by

Jerimiah Lamb

A thesis submitted in partial fulfillment
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of

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in

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Approved:

_____	_____
Paul Grossl, Ph.D. Major Professor	Seth Lyman, Ph.D. Committee Member
_____	_____
Roger Coulombe, Ph.D. Committee Member	Randy Martin, Ph.D. Committee Member

_____

Mark R. McLellan, Ph.D.
Vice President for Research and
Dean of the School of Graduate Studies

UTAH STATE UNIVERSITY
Logan, Utah

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ABSTRACT

Non-Methane Hydrocarbon Source Apportionment and BTEX Risk Assessment of Winter 2015 in Roosevelt, Utah

by

Jerimiah Lamb, Master of Science

Utah State University, 2017

Major Professor: Paul Grossl, Ph.D.
Department: Plants, Soils, and Climate

Non-Methane Hydrocarbons (NMHC) monitored in Roosevelt Utah including Benzene, Toluene, Ethylbenzene and Xylene (collectively known as BTEX) are associated with deleterious effects. This study addressed two points: 1) Source identification using the USEPA's Positive Matrix Factorization (PMF) and NOAA's Hybrid Single Particle Lagrangian Integrated Trajectory (HYSPLIT) model and 2) A human health risk assessment based on ambient concentrations of BTEX collected at the Roosevelt site. Model fit indicated that the primary contributor to total NMHCs was local oil and gas operations. Assessment of ambient BTEX concentrations was associated with slightly elevated carcinogenic risk.

(68 pages)

PUBLIC ABSTRACT

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Non-Methane Hydrocarbons (NMHC) monitored in Roosevelt Utah including Benzene, Toluene, Ethylbenzene and Xylene (collectively known as BTEX) are associated with deleterious effects including cancer. This study was designed to assess the origin and effect of the toxicants and addressed two points: 1) Source identification using the USEPA's Positive Matrix Factorization (PMF) and NOAA's Hybrid Single Particle Lagrangian Integrated Trajectory (HYSPLIT) model and 2) A human health risk assessment based on ambient concentrations of BTEX collected at the Roosevelt site. Model fit indicated that the primary contributor to total NMHCs was local oil and gas operations and was supported by previous assessments. Assessment of ambient BTEX concentrations was associated with slightly elevated carcinogenic risk.

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I would like to acknowledge Deborah McKean of the USEPA for her help and guidance in carrying out a toxicological risk assessment according to USEPA standards and the Uintah Impact Mitigation and Special Services District for funding the continued operation of the air monitoring station in Roosevelt, Utah.

I also want to acknowledge my wife, Alyssa. She has been my best friend since the first day I met her. She talks me down from metaphorical cliffs, encourages me to be the best that I can be, and makes me just a bit better every day.

Jerimiah Lamb

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INTRODUCTION

1. Background

Roosevelt City is located in the Uintah Basin of eastern Utah. Occasionally wintertime ground-level ozone concentrations in this region exceed current United States Environmental Protection Agency (USEPA) standards (Oltmans et al., 2014). To better assess ozone formation, a monitoring station located in a residential area of Roosevelt, Utah was established by the Utah Department of Air Quality (DAQ). Operation funding to monitor non-methane hydrocarbon (NMHC) ozone precursors was provided by the Uintah impact mitigation special service district (UIMSSD). While this station's primary purpose was to assess the production of ozone, some of the NMHC ozone precursors monitored were also Hazardous Air Pollutants (HAPs), as listed in the USEPA's 1990 amendment to the clean air act, and have direct implications on the health of the community (US EPA, 2015a).

Analyses in the greater area of the Uintah Basin has shown that NMHCs are locally derived and strongly associated with oil and natural gas (O&NG) operations(Helmig et al., 2014; Stoeckenius, 2015). Closer consideration of the individual sources contributing to the overall oil and gas source has revealed that individual well pads are thought to be the main emission source (Warneke et al., 2014).

While several studies assessed air concentrations and sources of NMHC in regions of oil and gas operations, only a few have assessed concentrations of NMHC in urban areas of the Uintah Basin (Lyman and Tran, 2015), and none have assessed source

characterization in urban areas of the Uintah Basin. Kim et al., (2005) found that in urban areas where oil and gas operations are present that there is some NMHC dependence on urban automotive and other anthropogenic emissions (Kim et al., 2005)

Source apportionment receptor models such as USEPA's Positive Matrix Factorization (PMF) use mathematical approaches to determine individual source contributions to environmental ambient air. They have been used to successfully identify and quantify sources of NMHC in the ambient air based on concentrations at a sample site (Brown et al., 2007; Choi et al., 2011; Fujita, 2001; Song et al., 2007). PMF has advantages above other receptor models in that it does not require emissions inventories of all possible sources. It only requires pollutant concentrations in ambient air, associated concentration uncertainties and the number of factors to provide the factor contributions and factor profiles (Norris and Duvall, 2014; Polissar et al., 1998).

The use of PMF in concert with a risk assessment of ambient NMHC has been employed previously and can assist in prioritization of management of sources based on adverse health effects (Choi et al., 2011). Since some of the NMHCs monitored in Roosevelt, Utah were also identified by USEPA as HAPs; it is important to determine their source and to quantify the risk to this urban population.

In accordance with the clean air act, USEPA lists Benzene, Toluene, Ethylbenzene and Xylene (collectively known as BTEX) as HAPs (US EPA, 2015a). A previous study for the greater region of the Uintah Basin revealed elevated concentrations of NMHC that are 1-2 orders of magnitude greater than average ambient concentrations for large US cities and that benzene was regularly found at concentrations consistent with

adverse health effects (Helmig et al. 2014). Elevated levels of NMHC in the region were shown to be consistent with times of strong temperature inversions occurring during the winter months (Stoeckenius, 2015). Strong temperature inversions decrease atmospheric mixing and allow the buildup of pollutants, including NMHCs in a shallow layer of atmosphere near the ground (Lyman and Tran, 2015).

In this study, area sources of NMHCs were determined using PMF followed by a risk assessment on ambient air concentrations to identify the adverse health effects associated with BTEX in Roosevelt, Utah. The following is a hazard identification summary of the impact that Benzene, Toluene, Ethylbenzene, Xylene, and BTEX mixtures have on human health.

2. Hazard Summary

Of the chemicals monitored, BTEX is a mixture that has implications for adverse human health effects(Badjagbo et al., 2010; Bolden et al., 2015; Choi et al., 2011; Liu et al., 2015). A synopsis of chemical and physical properties of BTEX is given in Appendix Table A2.

2.1 Benzene

Benzene is a liquid at room temperature that ranges from colorless to light yellow with an aromatic odor (Wilbur et al., 2007). Benzene is found in crude oils and as a by-product in oil-refining (US EPA, 2002). Inhalation is the major route of exposure, and it is readily absorbed by humans and other animals (Huff, 2007). In order to have physiological toxic effects, benzene must be bio activated through metabolism (Bernauer

et al., 1999). Exposure can result in cancerous and noncancerous adverse health effects (Wilbur et al., 2007).

Acute effects of benzene include dizziness, nausea, sleepiness, rapid heartbeat, and convulsions (US EPA, 2002). However, chronic toxicity exhibited in blood formation (hematopoiesis) is of greatest concern. Noncancerous hematopoietic toxicity of benzene includes decreased bone marrow functionality; resulting in anemia (reduction in red blood cell count), leukopenia (reduction in white blood cell count) or thrombocytopenia (reduction in platelet count) (Wilbur et al., 2007). Pancytopenia (reduction in all three forms) with necrosis of bone marrow is also associated with benzene exposure and is diagnostic of aplastic anemia (US EPA, 2002)

Benzene has been shown to have a carcinogenic risk in humans and USEPA classifies benzene as a known human carcinogen (Category A) for its weight of evidence characterization (Huff, 2007). A weight of evidence is a valuation of a chemical's ability to produce mutations and to interact with DNA (National Research Council (US), 2011). The carcinogenic risk has been predominantly associated with acute nonlymphocytic leukemia also known as acute myelogenous leukemia (AML), but also with other cancers including multiple myeloma, non-hodgkin's lymphoma and chronic myelogenous leukemia (Huff, 2007; US EPA, 2002). These details of benzene are supported by the current knowledge about its mode of action (Figure 1).

Metabolic Scheme for Benzene

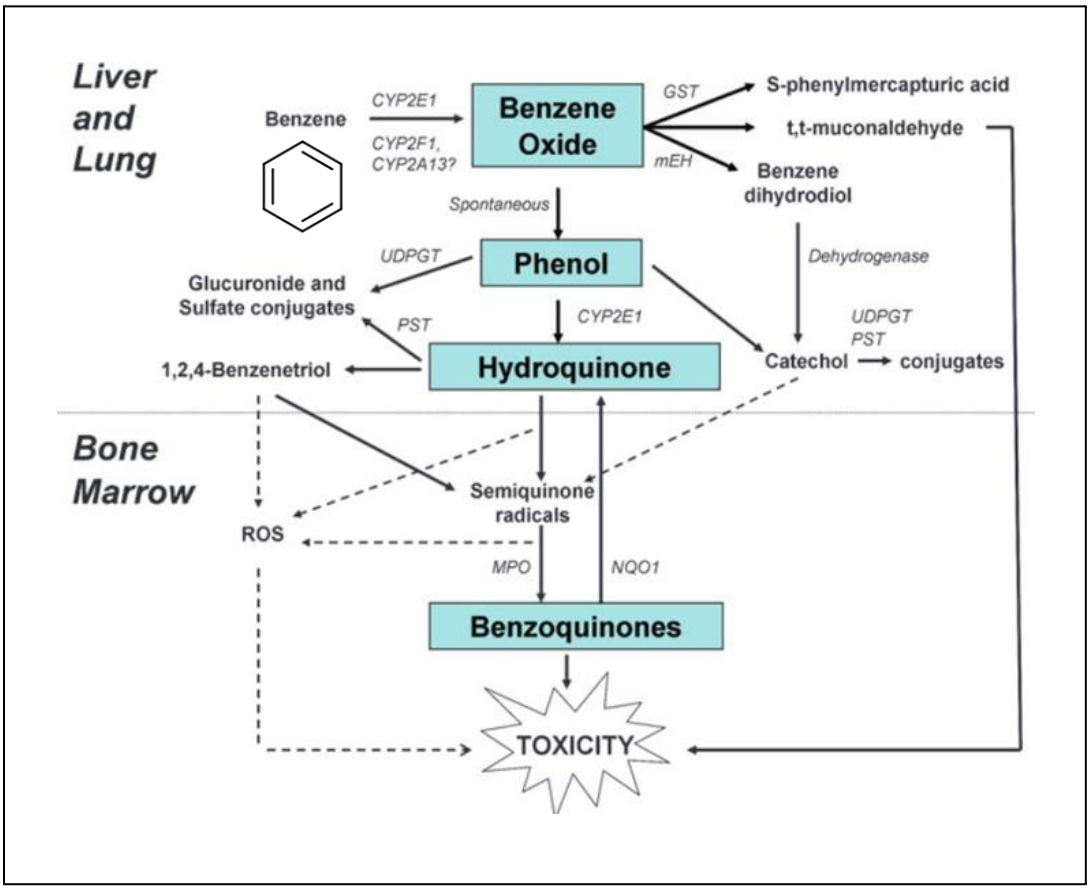

Figure 1. Simplified metabolic scheme for benzene including major pathways. Benzene is metabolized in the lung and the liver to form benzene oxides which can be further metabolized into toxic and non-toxic species or conjugated and excreted. Benzene's site of toxicity is in the bone marrow where it has hematopoietic effects. Reprinted from "Current Understanding of the mechanism of benzene-induced leukemia in humans: implications for risk assessment," by C.M. Mchale et.al, 2012, Carcinogenisis, 33(2), p.248

The mode of action of benzene is as follows: first, benzene is metabolized either in the liver by CYP2E1, or in the lung by CYP2F1 and CYP2A13 resulting in the production of benzene oxides, muconaldehydes or hydroquinones (Bernauer et al., 1999). The muconaldehydes have an open ring structure that is a very reactive electrophile and is immediately toxic (Bleasdale et al., 1996). Other toxic species can be formed by conversion of hydroquinone to 1, 2, 4-Benzenetriol by CYP2E1 in the liver or by converting hydroquinone in the bone marrow to either semiquinone radicals or benzoquinones via myeloperoxidase (Smith, 1996). Detoxification of potentially toxic species is thought to take place by phase 2 enzymes glutathione-s-transferase, UDP-glucuronosyltransferase or phenol sulfotransferase (McHale et al., 2012)

2.2 Toluene

Toluene is a clear liquid with a scent similar to benzene. It is also a component of crude oil and is an additive in gasoline mixtures, paints and solvents, coatings, inks, and adhesives (Williams et al., 2015). Inhalation is the most common route of exposure and toluene also is often abused for its euphoric properties (Von Burg, 1993).

Inhalation uptake has been shown to be approximately 55% for a person at rest (US EPA, 2005). Distribution is throughout the body with highest concentrations being found in the liver, brain, kidneys, and blood (Reese and Kimbrough, 1993). Toluene has been shown to cross the placental barrier entering the fetus and to be found in breast milk (Williams et al., 2015).

Cytochrome p450 enzymes in the liver catalyze the transformation of toluene to benzyl alcohol, to which oxidation then occurs to form benzoic acid (Figure 2) (Hammer,

2002). Benzoic acid is then conjugated with glycine to form hippuric acid which is excreted and is the major metabolite found in urine (Reese and Kimbrough, 1993). Three to five percent of toluene metabolites excreted involve a secondary metabolic pathway of cresol formation that is believed to have a potential genotoxic effect (Hammer, 2002).

Acute effects of toluene at high concentrations are ataxia, tremor, anosmia, sensorineural hearing loss, dementia and epileptic seizures (US EPA, 2005). Chronic exposure may result in low birth weight, immunological effects, and an increased likelihood of asthma symptoms and cardiovascular disease (Bolden, Kwiatkowski, and Colborn 2015). USEPA's weight of evidence (WOE) lists Toluene under inadequate information to assess carcinogenic potential (US EPA, 2005).

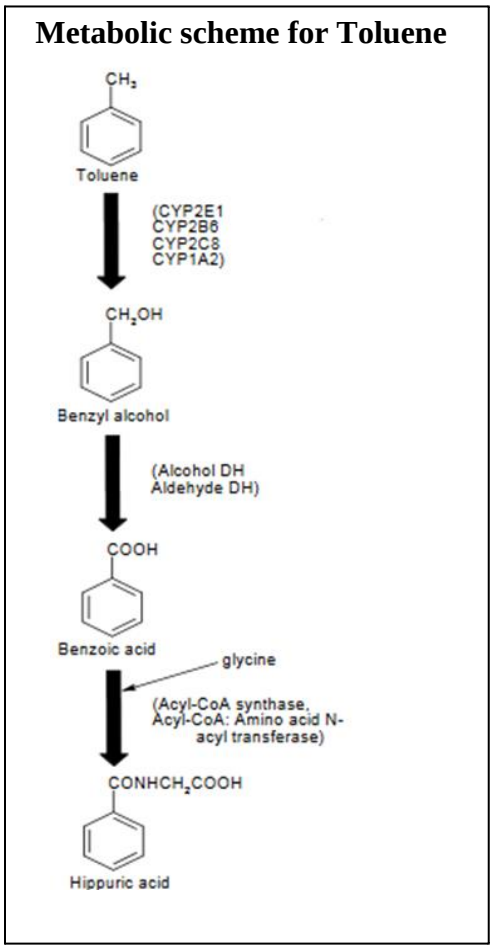

Figure 2. Scheme of major pathway of Toluene metabolism. Metabolism happens in the liver and eventual excretion happens via glycine conjugation. Adapted from "Toxicological Profile for Toluene." R. Williams et.al, 2015, Agency for Toxic Substances and Disease Registry, p. 220

2.3 Ethylbenzene

Ethylbenzene is a colorless liquid at room temperature with an aromatic odor. It is naturally occurring in petroleum and a component in fuels (Cannella, 2000). It is found in hydrocarbon solvents, varnishes, and paint. However, it is in the production of styrene where it is utilized most often. Styrene is an important intermediate in the manufacturing of many polymers (Taylor et al., 2010).

Inhalation from automotive emissions is the primary source of environmental exposure to ethylbenzene and inhalation is the most common route of exposure (Sweeney et al., 2015). Acute exposure to high concentrations of ethylbenzene causes eye and throat irritation, and narcotic effects. Prolonged occupational exposure has been shown to cause an increased incidence of hearing loss (Taylor et al., 2010).

Ethylbenzene is listed by the USEPA's weight of evidence as Class D (not classifiable as to human carcinogenicity) but its status is currently under review. The International Agency for Research on Cancer (IARC), a group associated with the World Health Organization (WHO), lists ethylbenzene as group 2B (possibly carcinogenic to humans). This designation is based on a study performed by the National Toxicology Program (NTP) (National Toxicology Program, 1999).

This study showed multiple possible routes of carcinogenicity. The primary route of metabolism occurs in the liver and the lung resulting in 1-phenylethanol. Distribution to the kidney of 1-phenylethanol in rats has caused an exacerbation of Chronic Progressive Nephropathy (CPN), an age-related disease resulting in kidney tumor development (National Toxicology Program 1999). However, this disease is unique to

rats and it has been suggested that these toxic effects cannot be extrapolated to humans (Sweeney et al., 2015). Other lesser routes of metabolism at high concentrations may result in oxidative stress which can lead to lung tumors (National Toxicology Program 1999). The primary route of excretion is in the urine as 1-phenylethanol.

2.4 Xylenes

Xylenes encompass three isomers of xylene either ortho, meta, or para depending on the arrangement of the methyl groups on the benzene ring (Cannella, 2000). Xylene is also known as xylol or dimethylbenzene. It is colorless and flammable with a sweet aromatic odor. Xylene is extensively produced in the United States and is used as a solvent in printing, rubber and leather production, cleaning agents, and varnishes (Fay et al., 2007). It is also found in fuels, and some isomers of xylene are used to manufacture certain polymers (US EPA, 2003).

Xylene's primary environmental source is petroleum production, and the main route of exposure is inhalation (Fay et al., 2007). Xylene is readily absorbed via inhalation and is distributed more selectively into adipose, liver, and brain tissues (Langman, 1994; US EPA, 2003).

Xylene isomers are listed individually in Appendix Table A2, but each isomer is thought to be toxicologically equivalent (Fay et al., 2007; Langman, 1994). Xylene is primarily metabolized by p450s in the liver to form methylbenzoic acids (Figure 3) with the para isomer being more readily oxidized than the other isomers (Langman, 1994). The major route of conjugation and excretion of methylbenzoic acids is via glycine conjugation and excretion in the urine as hippuric acid. Other routes of conjugation

involve glucuronidation leading to excretion. However, that pathway is much less preferential (Rajan, 2014; US EPA, 2003).

Metabolic Scheme for Xylenes

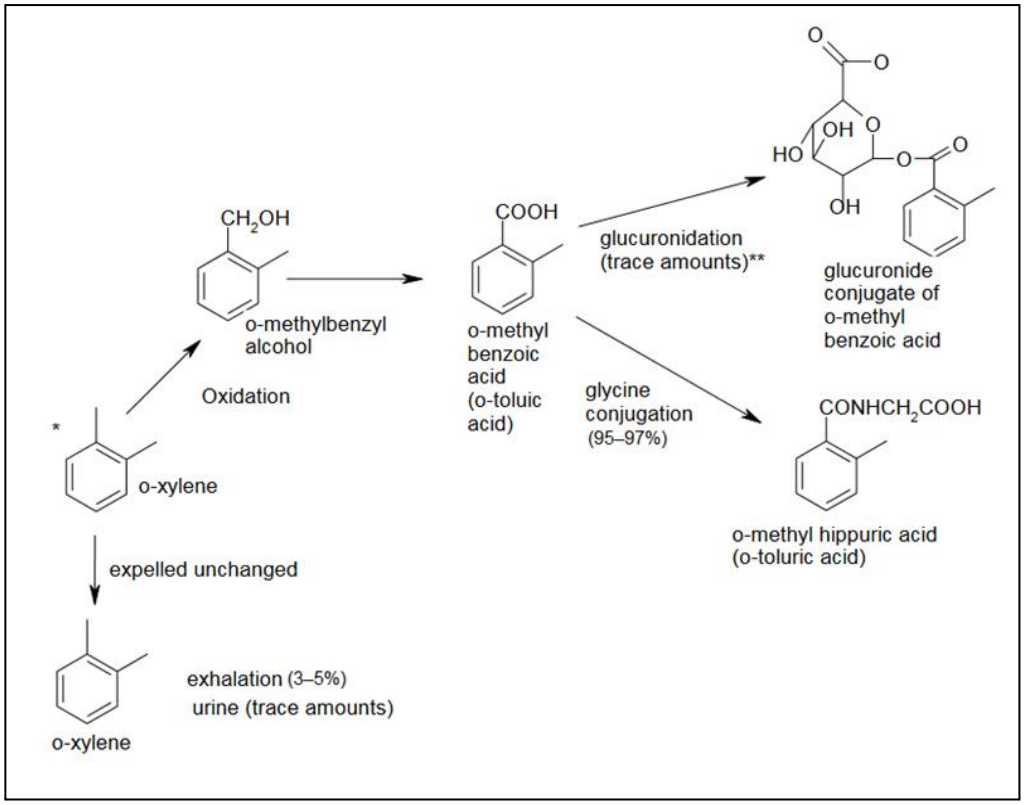

Figure 3. Scheme of major pathway of Xylenes using O-xylene as a model. Metablolism of xylene occurs mostly in the liver to o-methylbenzoic acid. Glycine conjugation is the major metabolite found in urine. Glucuronidation also is a conjugation pathway leading to excretion. ** Significant amounts of glucuronic derivative under high concentrations of administration. Adapted from "Toxicological review of Xylenes." US EPA, 2003, US EPA, p. 10

Acute exposure to xylene can cause irritation of the nose and throat and impair motor ability (US EPA, 2003). Chronic occupational exposure sometimes causes eye irritation, sore throat, inability to concentrate, feelings of weightlessness, and decreased lung function (Bolden et al., 2015; Rajan, 2014).

USEPA's weight of evidence characterization for carcinogenic effects of xylenes states that there is inadequate data for assessment of human carcinogenic effects. Furthermore, the USEPA states that animal data is inconclusive and evaluations of genotoxicity have consistently given negative results (US EPA, 2003).

2.5 BTEX Mixtures

Many mixtures have been studied for BTEX chemicals. Of those, BTEX mixtures demonstrated either no interactions, inhibitory interactions or additive effects. However, there have been relatively few studies on the full mixture of BTEX (Wilbur and Bosch, 2004). Of the articles found, some suggested respiratory impairment and low birth weight when exposed to ambient concentrations of BTEX (Bolden, Kwiatkowski, and Colborn 2015). Physiological modeling suggests that the effects of combined BTEX are additive (Wilbur and Bosch, 2004). However, one study suggested a synergistic action in the genotoxic effects of benzene when accompanied by other BTEX chemicals (Mazzeo et al., 2011).

This study addressed both the determination of the origins of the NMHCs in Roosevelt, Utah and the risk associated with continual exposure to them. Origin determination was performed by a source apportionment using USEPA's multilinear regression model, Positive Matrix Factorization. A complete list of the NMHC used in

the PMF analysis with their associated concentrations is given (Appendix Table A1). Risk associated with BTEX was determined by assessing ambient concentrations using USEPA guidelines for inhalation exposure.

METHODS

1. Data Collection

An NMHC dataset that was collected during the winter of 2015 by USU-Bingham Research Center was used in this study. The dataset had 57 NMHCs that were measured each hour from January through March. The air samples were taken at a remotely operated site located in a residential area of Roosevelt, Utah near a public park. There was low traffic within the immediate vicinity; however, the site was located within one kilometer of two operating oil locations and about a kilometer away from US Highway 40. (Figure 4)

Sampling was performed on an automated 2-column Perkin Elmer Clarus 600 gas chromatograph with a flame ionization detector, preconcentration cryogenic trap and thermal desorber in accordance to USEPA Method TO-12 for measuring ambient NMHCs. A PLOT column was used for lower molecular weight hydrocarbons and then an in line general BP1 column for higher molecular weight molecules. A standard calibration gas with known concentrations of the 57 NMHCs was run in triplicate every three days. The data was then stored as chromatograms which were verified to have properly assigned peaks and were placed into a time series for ease of statistical analysis and to be assessed for source identification.

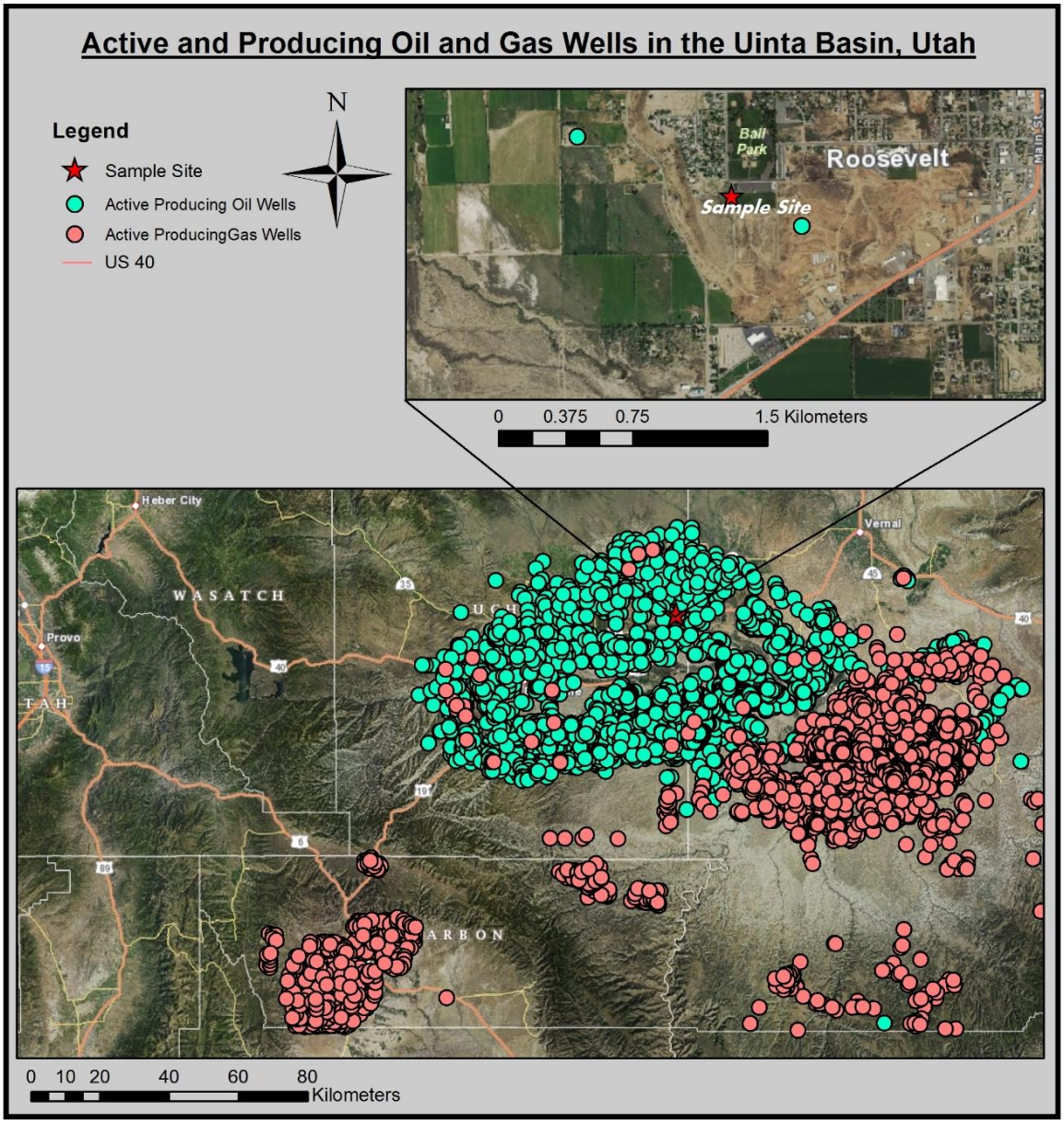

Figure 4. General map of oil and gas operations in the Uintah Basin. Sample site, active oil wells, active gas wells, and major highways are marked.

Additional area samples were obtained from five locations: Fruitland, Wells Draw, Horsepool, Vernal, and Seven Sisters, (Figure 5) in 6 L evacuated Summa canisters during a seven-day period from 1 February to 8 February. These samples were analyzed on a Perkin Elmer automated gas chromatograph by USU-Bingham Research Center information regarding methods for data acquisition is provided by Lyman and Tran (Lyman and Tran, 2015).

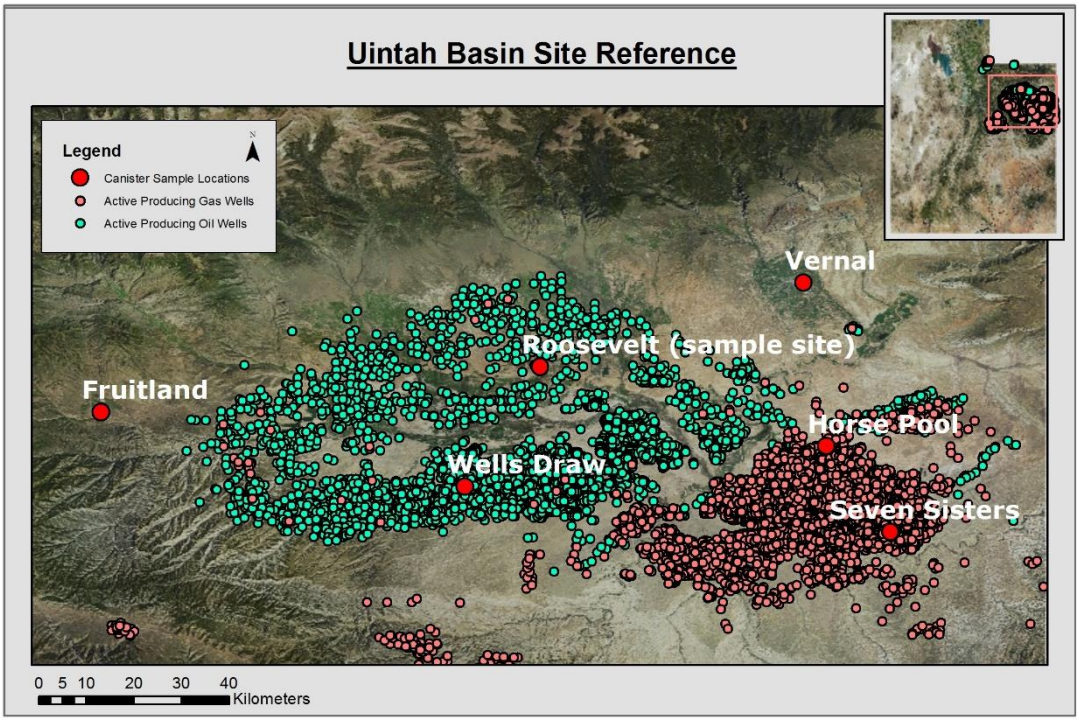

Figure 5. Area air canister sample locations are shown relative to active producing oil wells and active producing gas wells, and are marked in red. Areas used for comparison to factored source profiles are in dense areas of O&NG operations.

2. Source Apportionment

2.1 PMF Data Preparation

Factor analysis of ambient NMHCs was conducted using USEPA Positive Matrix Factorization (PMF) 5.0 (Norris and Duvall, 2014). The PMF method has been thoroughly described by Norris et.al (2014) and widely used for VOC and particulate matter factor analysis.

Simply stated, PMF determines the contribution of sources to ambient samples. The compositions of sources are determined mathematically by viewing ambient sample data as a matrix of time sample verses NMHC species which it then decomposes into two matrices - factor contributions and factor profiles. The user then interprets factor profiles into source types by using known or measured profiles.

The air monitoring data is represented in Eq. 1. Sample $\boldsymbol{x_{ij}}$ is the ***jth*** species concentration measured in the ***ith*** sample. $\boldsymbol{g_{ik}}$ is the contribution in the ***ith*** sample from the ***kth*** source, $\boldsymbol{f_{kj}}$ is the ***jth*** species fraction from the ***kth*** source, $\boldsymbol{e_{ij}}$ is the residual of the ***jth*** species concentrations of the ***ith*** sample and ***p*** is the number of factors. The goal is to identify the number of factors ***p***, the source profiles ***f*** and their respective mass contribution ***g*** along with residuals ***e*** (Norris and Duvall, 2014; Wu et al., 2016).

$$x_{ij} = \sum_{k=1}^{p} g_{ik} f_{kj} + e_{ij} \qquad \text{(eq. 1)}$$

Potentially, there could be an infinite number of solutions produced by PMF due to rotations of a solution. A rotation of a solution is a matrix transformation that results in

a solution that is mathematically equivalent to the original solution but has different matrix values including negative values (Paatero et al., 2002). PMF employs a non-negativity parameter since a negative solution would not be plausible. That parameter alone sometimes makes other solutions impossible. However, PMF also uses other parameters and diagnostic methods to assess and guide the matrix rotation into the most plausible, proper solution (Paatero et al., 2005).

The PMF solution minimizes the loss function Q which is defined by Eq. 2 as follows:

$$Q = \sum_{i=1}^{n} \sum_{j=1}^{m} \left[\frac{x_{ij} - \sum_{k=1}^{p} g_{ik} f_{kj}}{u_{ij}} \right]^2 \qquad \text{(eq. 2)}$$

where $\boldsymbol{\mu_{ij}}$ is the uncertainty associated with the ***jth*** species concentration in the ***ith*** sample.

PMF requires a data matrix without any missing values. Therefore, samples for which no NMHC concentrations were available were excluded from the analyses (Kim et al., 2005). Also, species with large portions of missing data were excluded from analysis as done previously (Zhoa et. al 2004). A total of 826 samples and 42 species were used for analysis (Table A1). NMHC were only collected during the ozone (winter) months. The automated sampling was out of operation for one month from 1/24 – 2/24.

PMF also requires an uncertainty value for each concentration value given. The uncertainty matrix must have the same number of species and samples as the concentration matrix and no empty cells (Norris and Duvall, 2014). Uncertainty

calculations were made based on the uncertainty calculations given by (Polissar et al. 1998) for samples with measured values and values below the detection limit. Determined values were used as given. Uncertainty values for determined values were calculated by using the analytical uncertainty plus ⅓ the detection limit (DL) value. Determined values below the detection limit were also used as given due to an increased model fit and as a potential reduction in modeling error as described by (Paatero et al., 2014). Uncertainty values below the detection limit were calculated by using ⅚ *DL.

Analytical uncertainty calculations were based on calculations of background noise and method detection limits (Berthouex and Brown, 2002; Skoog et al., 2007). Background noise was determined by the standard deviation of the mean of all standard calibration runs, taken every three days. The method detection limit was calculated by choosing a multiple of the background noise so that the probability of any given measurement being a false measurement was less than one percent.

2.2 PMF Analysis

In order to compensate for species with low signal to noise, some species needed to be down-weighted so that their effect on the solution was diminished. The down-weighting was performed using the PMF's signal-to-noise ratio. Species with a signal-to-noise ratio greater than 1.0 were attributed as "Strong." Species with a signal-to-noise less than 0.5 were recognized as "Bad" and species between 0.5 and 1.0 were recognized as weak as directed by Norris et.al (2014). Bad species were excluded from the analysis, weak species were given an added uncertainty, and strong species were processed as

given (Norris et.al 2014). This designation limited the species available for analysis to 16 species.

Analysis with PMF required an input of the number of sources to perform a run. A varying number of sources were tested while performing hundreds of runs to find a value with the most physically reasonable results (Kim et al., 2003). The Q-value as an indicator of a good starting point for interpretation was used as a guide as outlined by Reff et. al (2007).

Other analysis methods were used to lessen rotational ambiguity and to find the best possible solution, including displacement error estimation (DISP), FPEAK and g-space plotting. DISP is a diagnostic method within PMF that explicitly explores rotational ambiguity by adjusting factor profile values and then assessing those adjustments' effect on Q. If the adjustments caused a large change in Q, the solutions were not as valid (Brown et al., 2015; Norris and Duvall, 2014). FPEAK explored possible solution rotations allowing a better scope of the solution, and g-space plotting plotted one factor against another to determine if factors were dependant upon one another, thus, not fully resolved (Norris and Duvall 2014). A FPEAK value of -1.0 in coordination with g-space plotting was used to avoid unrealistic rotations and find the best solution as outlined by (Paatero et al. 2005).

Source identification was performed by using NMHC area sample data obtained from locations in surrounding O&NG dense areas on days of wintertime temperature inversions during 2013, utilizing the USEPA's web –based source information database, SPECIATE, and researching other relevant source profiles found in similar studies

(Logue et al., 2010; US EPA, 2015b; Wu et al., 2016). Area sample data and source profiles were taken from the SPECIATE browser and used as aids to identify source groups for each factor (Ito et. al 2004). Further identification efforts were made by taking PMF factor outputs and correlating them to the area sample data.

Correlation plots were made by normalizing both area sample data and PMF factors so that the average of all the contributions for each factor was one, consistent with previous publications (Choi et al., 2010a; Kim et al., 2005; Norris and Duvall, 2014).

Locations that were used for comparative area sampling included Fruitland, Wells Draw, Seven Sisters, Horse Pool and Vernal, Utah (Figure 5). Except for Fruitland and Vernal, these are areas of dense oil and natural gas operations in the Uintah Basin and were thought to be indicative of source profiles dominated with oil and gas sources.

3. HYSPLIT

National Oceanic and Atmospheric Administration (NOAA) HYSPLIT (Draxler, Roland et al. 2016) was used to simulate the emission, transport, and dispersion of the contributing factors to the sample site in Roosevelt, Utah in order to help compute the time history of air pollutant concentrations. Twenty-four hour air mass back trajectories were calculated to identify the origin and transport of air masses arriving at the sample site for times where individual factors were modeled by PMF to be dominant. Dominant times for individual factors were selected as the top ten highest times of individual factor contribution to the total air composition. HYSPLIT simulations were then performed for each time selected using HYSPLIT's ensemble method with NAM (North American

Mesoscale) 12km tile data to create an estimate of contribution location and uncertainty in HYSPLIT's modeled calculations (Draxler, Roland et al., 2016; Stein et al., 2015).

The ensemble method within HYSPLIT was used to gain an understanding of the variation associated with initial errors in particle trajectories (Stein et al., 2015). Ensemble calculated the dispersion from the same starting location but shifted the meteorological grid in an effort to determine initial transport errors (Stein et al., 2015).

A more in depth study of HYSPLIT would be necessary to gain definitive results. Here, HYSPLIT was used as a means by which PMF could be supported by using assumptions made by PMF outputs and checking those assumptions by performing HYSPLIT simulations in order to provide an indication of potential sources that made up each factor resolved by PMF.

4. Risk Assessment

The guidelines for receptor identification, exposure, and calculation of risk followed the guidelines available in the USEPA's Risk Assessment for Superfund Part F: Supplemental Guidance for Inhalation Risk Assessment (US EPA, 2009). Data collected for ambient air concentrations were consistent with USEPA Method TO-12 to measure ambient VOCs.

4.1 Data collection and analysis

Concentrations of BTEX taken from the ambient air were converted from ppb to $\mu g/m^3$ using standard temperature and pressure of 760 mmHg and 295.16K according to the reference conditions established by 40 CFR50.3. The concentration of the

contaminant was then calculated as the median and 95% upper confidence limit (UCL) of the mean for all risk calculations as performed previously by (McKenzie et al., 2012).

Potential human receptors in Roosevelt, Utah during the time of sampling were identified as a residential receptor, an occupational receptor, and a recreational receptor. A residential receptor was assumed to be exposed to the city's ambient air for 24 hours per day, 365 days per year and for 30 years. An occupational receptor such as an outdoor worker in Roosevelt, Utah, was determined to be exposed to the city's ambient air for 8 hours per day, five days per week for two years, conservatively assuming a long-term (two-year) outdoor occupational project. Finally, a recreational receptor was determined to be exposed to the city's ambient air for up to 24 hours a day for 100 days per year or less. Residential and occupational receptors were assessed under chronic exposure durations that would apply to cancer risk calculations for applicable carcinogenic chemical species and all three receptors (residential, occupational and recreational) were assessed under chronic and acute exposure durations for all other non-cancer risks consistent with USEPA guidelines (US EPA, 2009).

4.2 Exposure Assessment

Methods of risk calculation outlined by the USEPA and defined specifically for inhalation exposure were used in determining non-cancer and cancer risks for receptors. The non-cancer risk was represented by a hazard quotient (HQ) for individual chemicals and by a hazard index (HI) for a summation of multiple chemicals. Cancer risk was represented by a unit-less risk value which determines the risk relative to a set benchmark. Non-cancer and cancer risk values were both assessed as functions of

exposure concentration (EC) and toxicant-specific values. Toxicant specific values for non-cancer risk associated with inhalation exposure were defined as reference concentrations (RfC), and toxicant-specific values for cancer risk associated with inhalation exposure were defined as inhalation unit risk values (IUR) consistent with USEPA guidelines (US EPA, 2009). This section outlines the relevant information and equations necessary for both non-cancer and cancer risk determination.

4.2.1 Non-cancer risk

Non-cancer risk calculations were assessed for each receptor exposed to chronic, subchronic and acute durations of toxicants by first estimating the exposure concentration (EC). The EC was defined as the toxicant concentration time-weighted for the duration of the exposure and is measured in $\mu g/m^3$ (US EPA, 2009)

EC was calculated for receptors exposed to chronic exposure durations by the following equation:

$$EC = \frac{CA * ET * EF * ED}{AT} \qquad \text{(eq. 3)}$$

where CA was the concentration of the contaminant in air, ET was the exposure time in hours, EF was the exposure frequency in Days, ED was the exposure duration in years and AT was the averaging exposure time (ED in years * 365 days/year * 24 hours/day).

For acutely exposed individuals, EC was calculated simply as the given concentration CA, as follows:

$EC = CA$ (eq. 4)

Exposure duration was assessed differently for different receptors because effects from a single or short-term exposure could differ from effects of a long-term exposure depending on how the chemical accumulated in the body, was metabolized, detoxified, and excreted.

It should also be noted that body weight and inhalation rate were found in some calculations of exposure (Choi et al., 2011; Demirel et al., 2014). However, those inputs were not used because USEPA recommends against making such adjustments since the amount of chemical that would reach the target site is not a simple function of inhalation rate and body weight (US EPA, 2009).

After determining EC, a reference concentration (RfC) for each toxicant was obtained via the Integrated Risk Information System (IRIS) established by the USEPA to determine non-cancer calculations for inhalation (US EPA 2016c).

RfCs were defined as a conservative estimate of concentration that will be without appreciable deleterious effects given continuous inhalation exposure for a population. They included adjustments for sensitive subgroups and spanned an order of magnitude for uncertainty (US EPA, 2009).

After obtaining the RfC for each inhaled toxicant, the Hazard Quotient (HQ) was calculated for non-cancer risk. The HQ is a risk identifier that followed the equation:

$HQ = EC/RfC$ (eq. 5)

Where EC was the calculated exposure concentration measured in units of μg/m^3 and RfC was the reference concentration in μg/m^3. HQ was inherently unitless so that any value above one was associated with elevated risk whereas any value below one was associated with a negligible or acceptable risk (US EPA, 2016).

The hazard index (HI) was defined as an aggregate non-cancer risk of exposure to multiple chemicals and was calculated by the sum of HQ values from multiple chemicals that were assessed at the same location. If the HI was greater than one, it would be necessary to derive separate HIs for each target organ of concern (Choi et al., 2011; McKenzie et al., 2012).

4.2.2 Cancer risk

The carcinogenic risk was also initially quantified by calculating the EC as shown in Equation 3. Cancer calculations for inhalation exposure then required an inhalation unit risk (IUR). IUR was defined as the estimated increased lifetime cancer risk to result from continuous exposure to a toxic chemical at a concentration of 1 μg/m^3 in air (US EPA 2015c). It was defined as a slope factor expressed in units of risk per μg/m^3 that was derived from an extrapolation of observed exposures in animal and human occupational studies (US EPA, 2009). To determine cancer risk, IUR was multiplied by EC as shown below:

$$Risk = IUR * EC \quad \text{(eq. 6)}$$

IUR values were obtained for benzene and ethylbenzene through the USEPA IRIS database and via the California EPA (Monserrat, 2016; US EPA, 2016). The calculation of carcinogenic risk was only applied to benzene and ethylbenzene since IUR values indicating carcinogenic risk were not associated with toluene or xylene.

The USEPA expressed the risk as a probability such as 10^{-6}, or 1 in a 1,000,000 chance. Risk values that are above the 10^{-6} are viewed as increased risk, but the acceptable range of risk is 10^{-4} to 10^{-6} (40 C.F.R. § 300.430).

RESULTS AND DISCUSSION

1. PMF results

Results from PMF indicated three factors. The three factors represented were a mixed source of vehicle exhaust and combustion labeled mixed combustion, and two separate oil and natural gas related sources labeled O&NG 1 and O&NG 2. The mixed combustion source was thought to be comprised of gasoline exhaust, diesel exhaust, and residential wood burning due to its composition of acetylene, propene, benzene and other comparatively higher molecular weight molecules (Logue et al., 2010; US EPA, 2015b; Wu et al., 2016). The sources thought to indicate oil and natural gas sources were dominated by short chain alkanes, which are indicative of fugitive natural gas (Figure 6) (Choi, Choi, and Yi 2011). The determination of three factors as an input was chosen because it provided the most physically meaningful solution of various factor considerations (Kim et al. 2005). Analysis with diagnostic tools DISP and FPEAK in PMF showed that there were no potential rotational swaps indicating a stable and reliable solution.

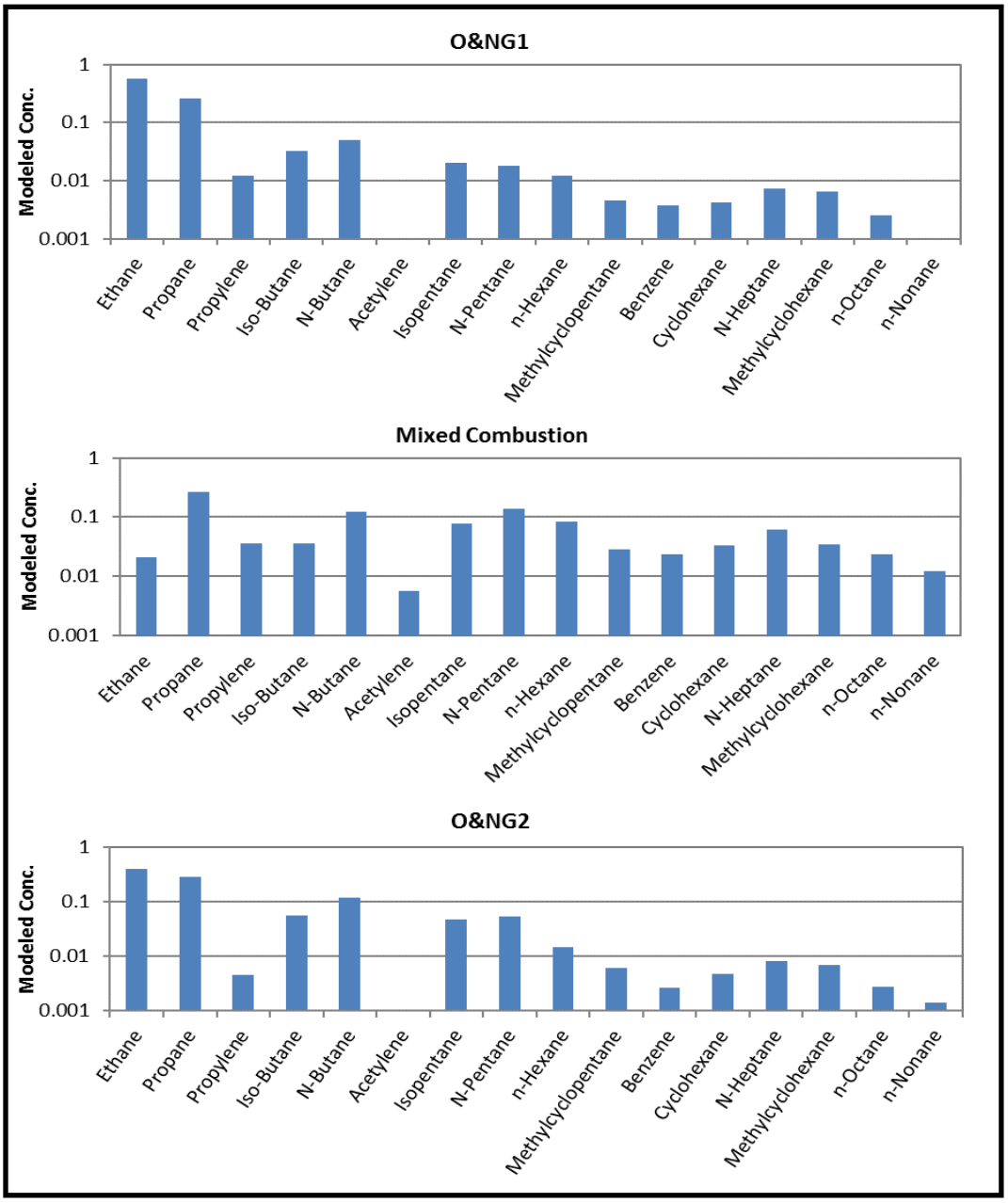

Figure 6. Predicted source profiles resolved by PMF from NMHC samples measured in Roosevelt, Utah. Predicted species concentrations are measured in parts per billion and shown on a log scale. Relative concentrations of species give an indication of which regional sources may be contributing to individual factors. The graph is normalized so that the average of all the contributions for each factor is one.

Upon comparison of PMF factor profiles O&NG1 and O&NG2, it appeared that they had very comparable relative concentrations of NMHCs, were very closely related, and perhaps even from the same source. However, an appeal to the FPEAK and g-space plots determined that they were indeed separate factors. The lack of oblique edges and the distribution of points extending along the axes illustrated the independence of individual factors through g-space plotting (Paatero et al. 2005). This solution also demonstrated robustness as being well constrained when assessed with the displacement error estimation (DISP) further verifying that a proper selection of the number of sources was made (Brown et al. 2015).

Since source identification as stated by Reff et. al 2007, "is the most subjective and least quantifiable step in the analysis of PMF," an attempt to quantitatively identify the factors was made after qualitatively identifying the potential factors (Reff et al., 2007). PMF derived factors were compared to area samples taken by Lyman and Tran during February 2013 to determine if a more direct correlation to local area O&NG could be achieved (Lyman and Tran, 2015). Results indicated a more direct correlation. Comparative charts normalized for comparison for both O&NG1 and O&NG2 show a high degree of correlation ($R^2 > 0.95$, $p<0.001$) to area samples taken in locations of dense oil and gas activity (Figure 7 and Figure 8). The correlation strongly suggested that these two factored sources were properly identified and were directly associated with oil and gas operations.

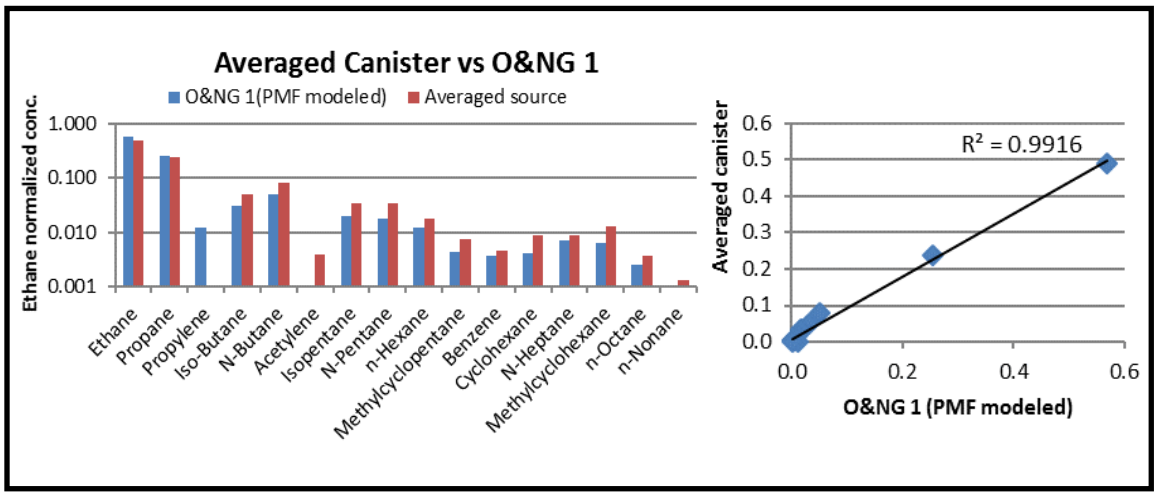

Figure 7. Left: Predicted PMF factor profile of O&NG1 against averaged canister data normalized so that the average of all the contributions for each factor is one. Right: correlation plot of PMF factor O&NG1 against averaged canister data demonstrating association with oil and gas sources.

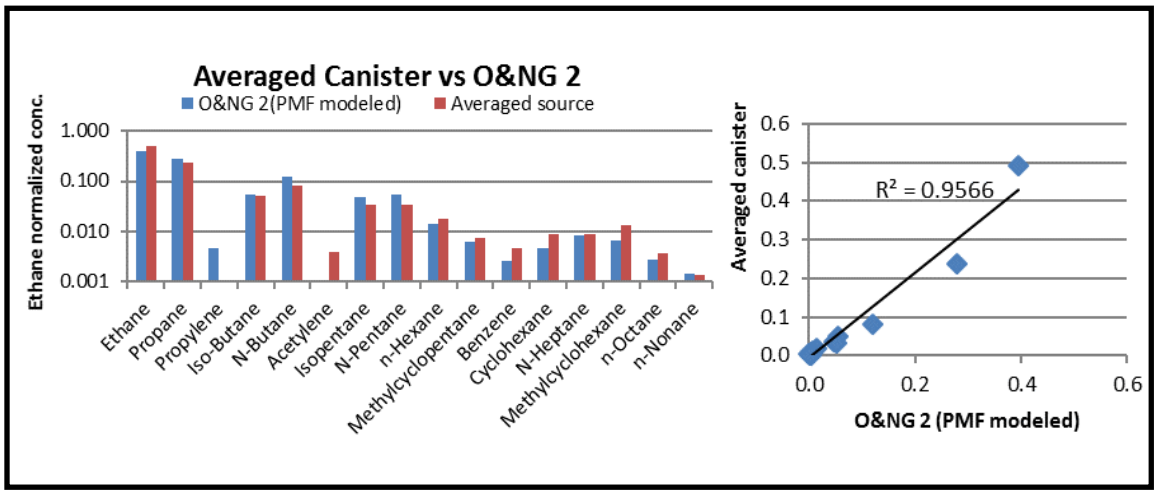

Figure 8. Left: Predicted PMF factor profile of O&NG2 against averaged canister data normalized so that the average of all the contributions for each factor is one. Right: correlation plot of PMF factor O&NG2 against averaged canister data, while not as tightly correlated as O&NG1, demonstrates association with oil and gas sources.

An averaged total concentration of the area sample data was used to show a representation of the oil and gas source profile in the Uintah Basin. This was based on the correlation comparisons of factors to the individual canister sample areas (Table 1) and the high correlation of each area sample to the other (Table 2). The high correlation of O&NG1 and O&NG2 to each site ($R^2 > 0.92$, $p < 0.001$) and high correlation of the area samples to each other ($R^2 > 0.95$, $p < 0.001$) justified averaging the area sample data to demonstrate how each factor compared to a representative O&NG profile for the area.

Individual factor comparisons to regional area profiles (Table 1) indicated that O&NG2 was better correlated to the Vernal area sample than O&NG1 ($R^2 > 0.96$, $p < 0.001$), but that O&NG1 was better correlated to sources across the area ($R^2 > 0.99$, $p < 0.001$). Those comparisons might suggest that factor O&NG2 comes from Vernal, Utah, but drawing definite source conclusions based on this information is not reasonable due to the high correlation of each factor to multiple sampling areas. Furthermore, oil and gas speciation profiles are in ratios on both the east and west sides of the Uintah Basin that are too similar to substantiate the origination of each PMF factor beyond being associated with oil and gas. Even the Vernal area sample is significantly correlated to other area samples despite being located many kilometers from an operating oil and gas well location (Table 2).

Table 1
Correlation of PMF factors to area samples. O&NG factors show a high correlation to area samples taken in regions of dense oil and gas operations. This is indicative that the O&NG factors defined by positive matrix factorization are correct. Mixed combustion shows a very low degree of correlation to area samples, indicating that this factor is not associated directly with oil and gas operations.

Correlation data of factors compared to regional area samples R^2

	Vernal	Wells Draw	Horsepool	Seven Sisters
O&NG1	0.93*	0.99*	0.99*	0.99*
O&NG2	0.96*	0.97*	0.95*	0.95*
Mixed combustion	0.10	0.08	0.06	0.06

* Correlation P-value <0.001

Table 2
Correlation of regional area samples. Area samples taken in regions of dense oil and gas operations are highly correlated to each other. Even the Vernal area sample which is located ~30km from any operating oil or gas location and is highly correlated to the other samples.

Correlations of regional area samples R^2

	Vernal	Wells Draw	Horsepool	Seven Sisters
Vernal	1*	0.9578*	0.9567*	0.9596*
Wells Draw		1*	0.9976*	0.9981*
Horsepool			1*	0.9998*
Seven Sisters				1*

*Correlation P-Value <0.001

Perhaps, the mixed combustion factor may have been generated from an alternative source independent of oil and gas. The mixed combustion source did not significantly correlate with area sampling data ($R^2 < 0.1$). Since oil and gas operations are far and away the dominant industry, local urban/residential or outside sources are most likely associated with the profile of NMHC in the mixed combustion factor.

Yet it should be noted that there is not a clear quantitative way to determine this. Trying to obtain greater resolution for the mixed combustion source with PMF by selecting for more factors resulted in a solution that diminished the high correlation of O&NG sources and factors that were not physically reasonable.

Assessing this profile qualitatively, however as done by many previously, suggested that the higher proportions of midrange carbons may have been indicative of automotive exhaust and that the presence of nonane would have been indicative of diesel exhaust (Wu et al., 2016). This assumption would also be consistent with the proximity of US Hwy 40 (within one kilometer), and the sample location being in an urban area (Logue et al., 2010).

2. Enhancement Ratios

In addition to PMF, it appears that the sources of NMHCs in Roosevelt, Utah are dependent on O&NG based on enhancement ratios. Enhancement ratios are another means to identify source signatures and are equal to the slope of a linear two-sided correlation plot (Figure 9) (Gilman et al., 2013). Enhancement ratios for isopentane versus n-pentane of 2.41, 1.10, 0.809, 0.885 and 0.86 for Pasadena, CA; Boulder, CO;

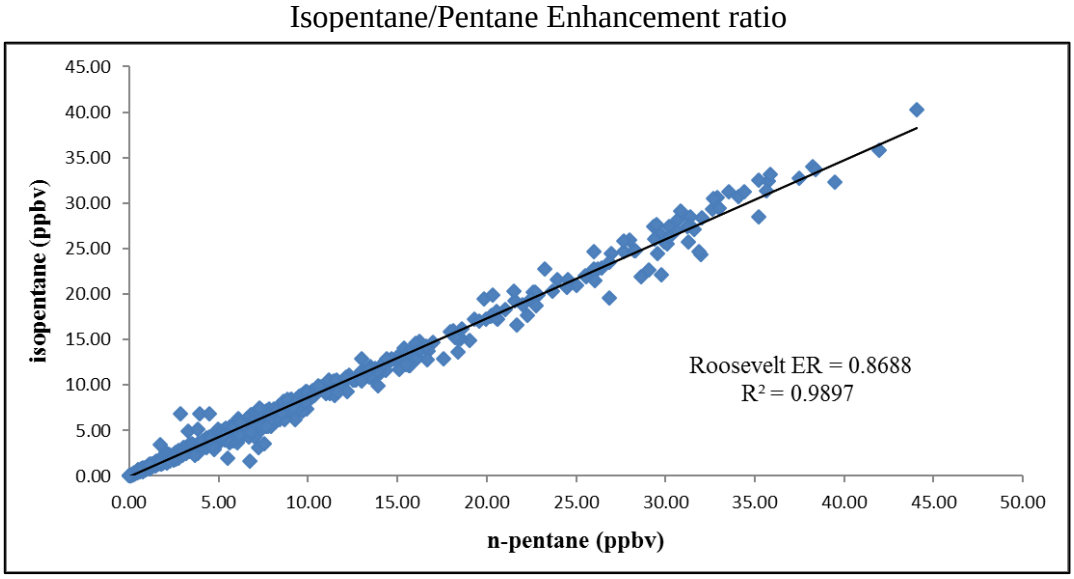

Figure 9. Correlation plot of isopentane verses n-pentane for Roosevelt, Utah. The slope of 0.87 suggests a large influence of raw natural gas based on data from Gilman et. al (2013).

Fort Collins, CO; Boulder Atmospheric Observatory, CO; and Raw natural gas, respectively were previously identified (Gilman et al., 2013). Isopentane/pentane enhancement ratios with higher values were consistent with automotive emissions and gasoline vapors, and lower values were consistent with natural gas dominated air. The

ratio for Roosevelt, Utah was 0.87 (Figure 5). This value corresponds best with raw natural gas, suggesting a strong dependence on O&NG influenced air, supporting PMF determination.

3. HYSPLIT

PMF created outputs that were useful in many ways. One particular output showed the predicted contributions of each factor by time. This was particularly useful when trying to further resolve potential sources using HYSPLIT.

Each factor was illustrated with individual lines showing predicted variations in contributions of each factor at any given time (Figure 10). Days that were represented by a dominant factor were used in HYSPLIT back trajectory simulations in an effort to gauge potential source origins.

Using dominant times modeled by PMF as inputs into HYSPLIT relied heavily on assumptions made by PMF outputs. Based on those assumptions, there was an extra degree of variability introduced; therefore, definitive results were not assessed using HYSPLIT. Rather, HYSPLIT outputs were qualitatively assessed for resulting trends.

Days when O&NG1 was dominantly modeled occurred most frequently (Figure 10). Wind variations showed South, South-West and Northwest origins of air flow with varying air trajectory elevations based on terrain (Figure 11). All of those trajectories crossed areas of active oil production (Figure 4) before reaching the sample location. That trajectory indicated that the oil operations West, Northwest, and South West have been the primary source of air resulting in ambient contributions of O&NG1.

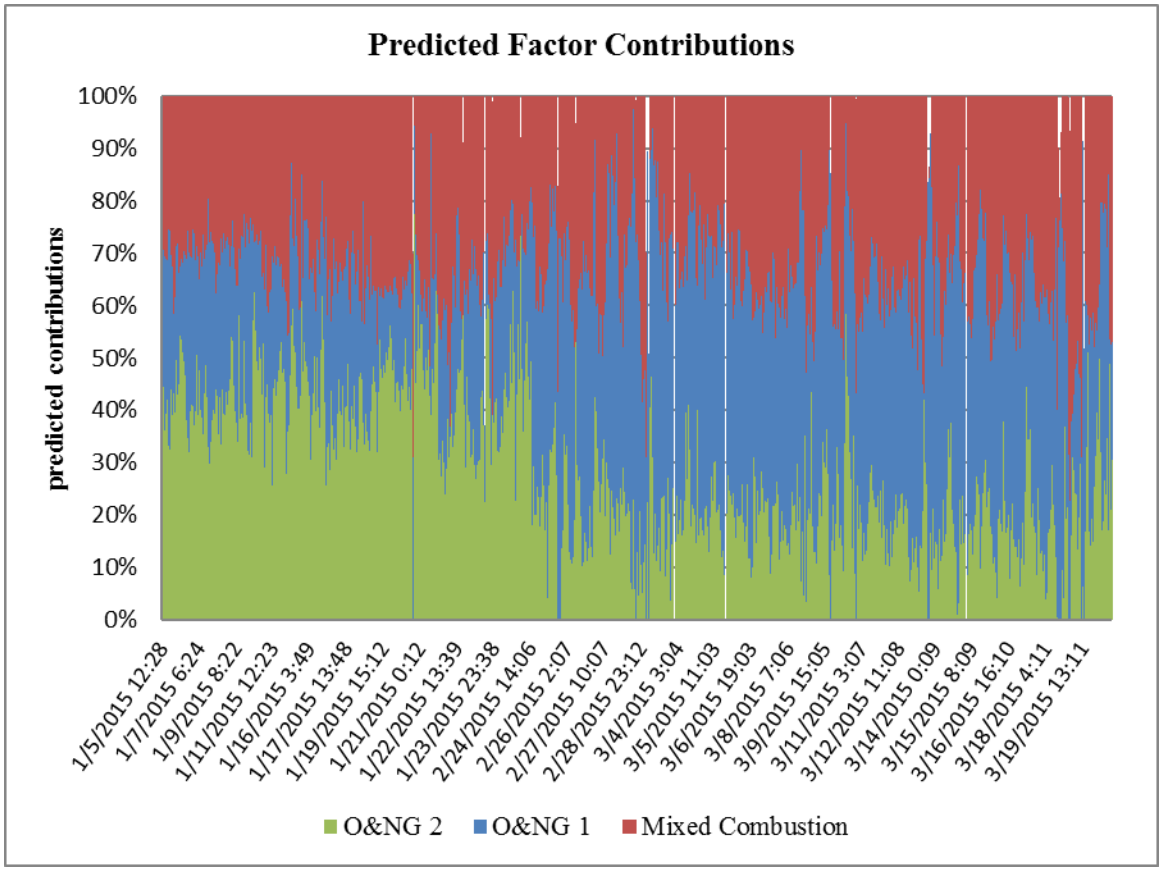

Figure 10. Predicted contributions of each factor by sample. Each sample is normalized so that the average of all contributions of each factor is one. Each factor is represented by a segment of each line at a given time and varies in predicted concentration based on modeled variation by PMF. Instances of one source being dominately represented were used for HSPLIT twenty-four hour back trajectories.

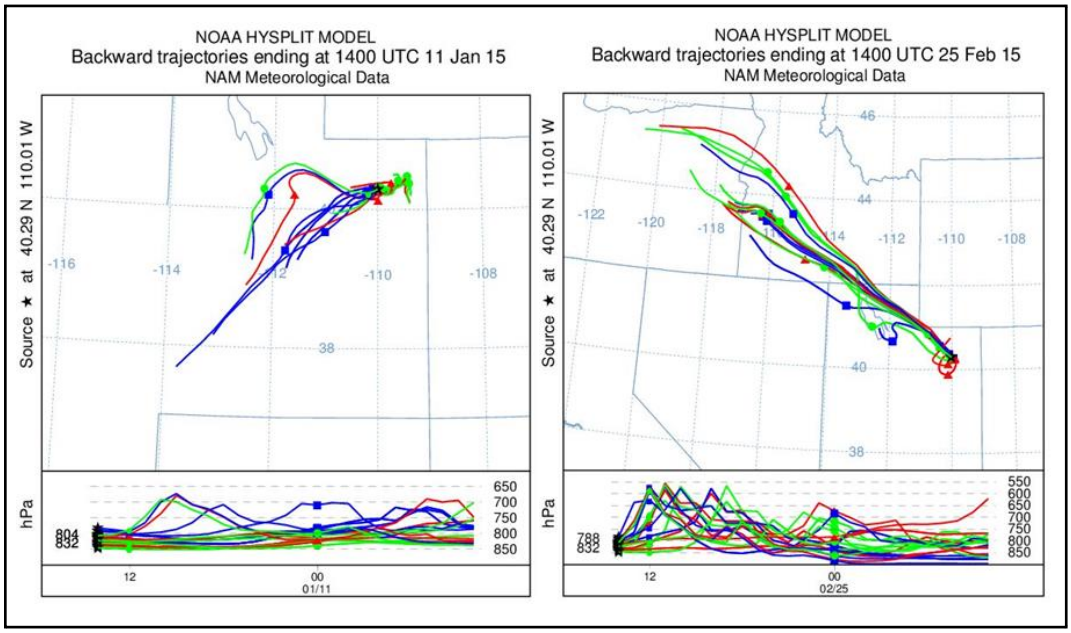

Figure 11. Representative HYSPLIT twenty-four hour back trajectories for O&NG1 PMF modeled dominant days. Various lines demonstrate variability in the simulation. Left: twenty-four hour back trajectory ending at 8:00 MT 11 Jan 15 Right: twenty-four hour back trajectory ending at 8:00 MT 25 Feb 15. Air mass elevation is illustrated in units of hectopascals (hPa).

O&NG1 appeared to be more quintessentially an oil and gas factor without much mixing. The correlations between O&NG1 with area sample data indicated that O&NG1 was extremely well correlated ($R^2 = 0.99$, $P<0.001$) with all area samples that were taken in areas of oil and gas development (Table 1).

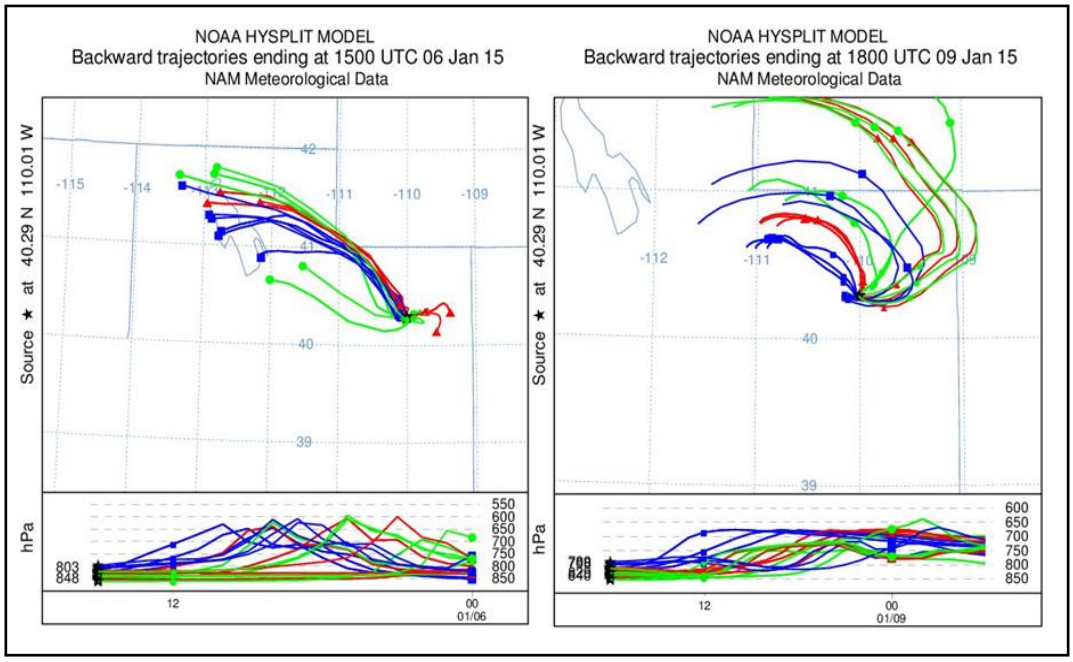

Figure 12. Representative HYSPLIT twenty-four hour back trajectories for O&NG2 PMF modeled dominant days. Various lines demonstrate variability in the simulation. Left: twenty-four hour back trajectory ending at 9:00 MT 06 Jan 15 Right: twenty-four hour back trajectory ending at 10:00 MT 09 Jan 15. Air mass elevation is illustrated in units of hectopascals (hPa).

Days that O&NG2 was dominantly modeled occurred early in January. Twenty-four hour back trajectories for air masses arriving at the sample site location appeared to originate from the North, Northwest, and Northeast with some variability from the East (Figure 12). Both O&NG1 and O&NG2 demonstrated a significant correlation to areas of dense oil and gas operations. Interestingly, O&NG2 demonstrated the best correlation to air sampled in Vernal, Utah ($R^2 = 0.96$, $P<0.001$). One possible explanation for the Eastern variability illustrated in HYSPLIT trajectories is that the O&NG2 source was an air mass that originated in areas of oil and gas development and was influenced by

Vernal, Utah air before ultimately becoming a contributor to the Roosevelt, Utah sample location. However, O&NG2 was nearly equally correlated to other area samples and better correlated to the Wells draw location (Table 1). These confounding variables made it difficult to determine the origin and integrity of these assumptions**.** Whether or not Vernal, Utah air was a contributing factor to the contamination of air at the Roosevelt, Utah sample site is something that requires a further in depth assessment. Yet we surmise that there is a strong argument for O&NG2 being associated with oil and gas operations based on PMF outputs and significant correlation with area samples.

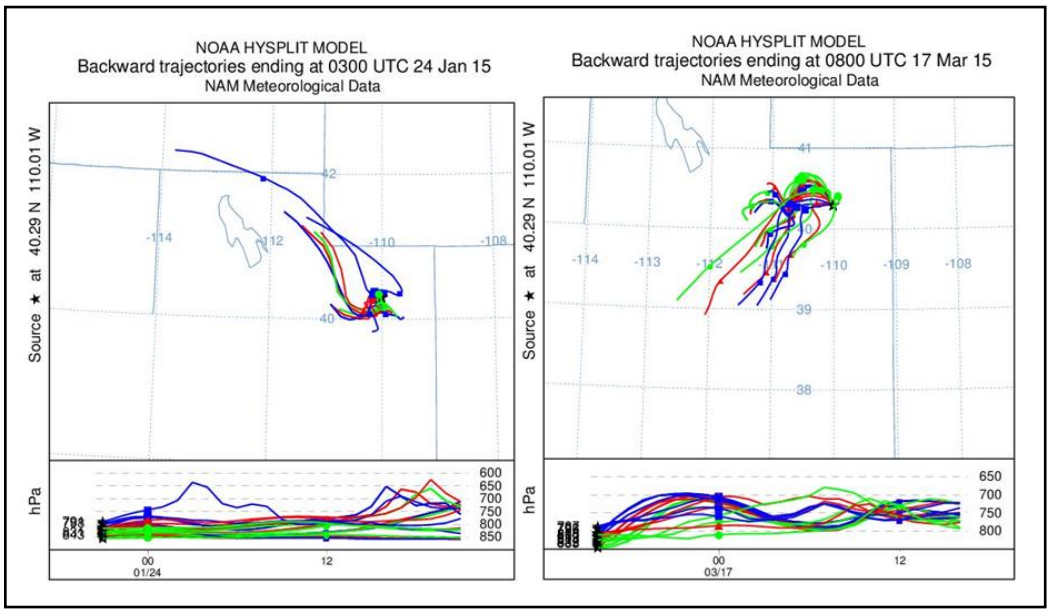

Figure 13. Representative HYSPLIT twenty-four hour back trajectories for Mixed Combustion PMF modeled dominant days. Variation of plotted lines indicates variability in the simulation. Left**:** twenty-four hour back trajectory ending at 21:00 MT 23 Jan 15. Right**:** twenty-four hour back trajectory ending at 2:00 MT 17 Mar 15. Air mass elevation is illustrated in units of hectopascals (hPa).

Finally, the mixed combustion factor appeared to be influenced by a much more locally derived source. Air mass twenty-four hour back trajectories had Northwest and Southwest origination and a pronounced trend of being slow and locally derived (Figure 13). Even though trajectories suggested airflow from oil and gas areas, those contributions could have been attributed to the O&NG components in the sample. The presence of more stagnant air during those time periods suggested that the mixed combustion factor was a local source unassociated with oil and gas operations. Local combustion sources such as gasoline and diesel exhaust and potentially residential wood combustion, given the time of year, were thought to be likely sources contributing to the mixed combustion factor (Logue et al., 2010; US EPA, 2015b; Wu et al., 2016).

4. Risk Assessment

The quantitation and analysis of ambient pollutants directly affected the health of residents who work and live in Roosevelt. Based on ambient air concentrations taken during the early winter months of 2015, a human health risk assessment was performed focusing on the inhalation route of concentrations of Benzene, Toluene, Ethylbenzene and Xylene (BTEX). Receptors accommodated in this assessment were residential, occupational and recreational receptors in Roosevelt, Utah. Residential and occupational receptors were assessed based on both non-carcinogenic and carcinogenic risk and recreational receptors were assessed based only on a non-carcinogenic risk.

4.1 Non-carcinogenic risk

The estimated non-cancer risk values associated with ambient air concentrations for residential, occupational and recreational receptors are summarized in Table 3. Each receptor is assessed with the median and 95% upper confidence limit (UCL) of the mean concentration for each of the BTEX chemicals.

Hazard quotient (HQ) values for all receptors based on the 95% UCL of the mean and the median are below the target HQ value of one, characterizing a negligible non-cancer risk (US EPA, 2009). Any value of HQ or HI below the value of one assumes that the body is capable of metabolizing and excreting the toxicant and its metabolites at that given exposure concentration without injury (US EPA, 2009). Residential and recreational receptors for benzene at the 95% UCL is the largest contributor to non-carcinogenic risk and would require more than an 800% increase to reach an HQ value of one. The aggregate non-cancer risk value, hazard index (HI), is also below one for each receptor characterizing a negligible non-cancer risk associated with both the median and the 95% UCL (US EPA, 2009).

Table 3

Summary of non-cancer risk for residential, occupational and recreational receptors. Each value was calculated for a receptor based on USEPA guidelines found in the Risk Assessment Guidelines for Superfund (RAGS): part F (US EPA, 2009). Values are based on a Hazard Quotient (HQ) value for individual chemicals and a Hazard Index (HI) for summative hazards. Values above unity are indicative of increased risk of negative effects and injury. Concentrations and risk calculations are given for the median and 95% upper confidence level (UCL).

Summary of Non-Cancer Risk (HQ values)

Hydrocarbon	RfC (µg/m³) [a]	Source [b]	Conc. (µg/m³)		Residential Receptor [c]		Occupational receptor [d]		Recreational Receptor [e]	
			median	95%UCL	median	95% UCL	median	95% UCL	median	95% UCL
Benzene	30	IRIS	2.20	3.12	7.34E-02	1.04E-01	1.74E-02	2.47E-02	7.34E-02	1.04E-01
Toluene	5000	IRIS	2.56	3.79	5.12E-04	7.57E-04	1.22E-04	1.80E-04	5.12E-04	7.57E-04
Ethylbenzene	1000	IRIS	0.26	0.36	2.60E-04	3.56E-04	6.18E-05	8.45E-05	2.60E-04	3.56E-04
Xylene	100	IRIS	1.56	2.37	1.56E-02	2.37E-02	3.71E-03	5.62E-03	1.56E-02	2.37E-02
Hazard Index (HI)					8.98E-02	1.29E-01	2.13E-02	3.06E-02	8.98E-02	1.29E-01

[a] Reference concentration (RfC) – A concentration estimate for exposure under continuous inhalation that is likely to be without risk of deleterious effects during a lifetime, including sensitive groups.

[b] IRIS - Integrated Risk Information System

[c] Residential Receptor – Calculated as a person under constant exposure to the chemical. Averaging time is 24 hours/day, 350 days/year, 70 years

[d] Occupational Receptor- Calculated for a person under exposure for 8 hours/day, 5 days/week, 2 years

[e] Recreational Receptor- Calculated for a person under exposure for

It would seem intuitive that a recreational receptor would have the least amount of risk associated with ambient acute exposure, but that was not the case. Reassessing equations 3 and 4, the calculation for a recreational receptor's exposure assumed that the averaging time (AT) was equivalent to the exposure time (ET). An occupational receptor has less acute risk because it has an ET that is less than the AT. Toxicologically, this makes sense due to an occupational receptor’s risk being calculated with a period of non-exposure. The non-exposure period results in a lesser overall dose for metabolism and excretion. On the other hand, a recreational receptor was calculated for the full-length of exposure without assuming any period of non-exposure.

4.2 Carcinogenic risk

The estimated cancer risk values associated with ambient air concentrations for residential, occupational receptors in Roosevelt, Utah were summarized in Table 4.

Each receptor was assessed with the median and the 95% UCL of the mean concentration for benzene and ethylbenzene. The cumulative cancer risk of benzene for the residential and occupational receptor exceeded the 10^{-6} benchmark indicating elevated risk. Cumulative cancer risk for ethylbenzene did not exceed the 10^{-6} benchmark for any associated carcinogenic risk calculations.

Table 4

Summary of cancer risk for residential and occupational receptors. Each value was calculated for a receptor based on USEPA guidelines found in the Risk Assessment Guidelines for Superfund (RAGS): part F (US EPA, 2009). Values are based on a one-in-a-million risk benchmark established by the 40 CFR 300.430. Concentrations and risk calculations are given for the median and 95% upper confidence level (UCL).

Summary of Cancer Risk for Residential and Occupational Receptors

Hydrocarbon	Unit Risk $(\mu g/m^3)^{-1}$	Source	Conc. ($\mu g/m^3$)		Weight of Evidence (WOE) [a]		Residential Receptor		Occupational receptor	
			median	95% UCL	IRIS[b]	IARC[c]	median	95% UCL	median	95% UCL
Benzene	7.80E-06	IRIS	2.20	3.12	A	1	1.72E-05	2.43E-05	4.08E-06	5.77E-06
Ethylbenzene	2.50E-06	CalEPA	0.26	0.36	Not listed	2B	6.51E-07	8.90E-07	1.54E-07	2.11E-07

[a] Weight of Evidence – Evaluation of a chemical's ability to produce mutations and to interact with DNA.
[b] IRIS - Integrated Risk Information System: A- known human carcinogen, Group B- probable human carcinogen, Group C- possible human carcinogen, Group D- Not classifiable as to human carcinogenicity
[c] IARC – International Agency for Research on Cancer: Group 1- carcinogenic to humans, Group 2A- probably carcinogenic to humans, Group 2B- possibly carcinogenic to humans, Group 3- not classifiable as to its carcinogenicity to humans

Closer examination of the ethylbenzene data for the residential receptor indicated a 40.8% increase would be necessary for the median value to reach the one-in-a-million benchmark and that a 12.4% increase in the 95% UCL was necessary to reach the benchmark. Occupational receptor risk values for ethylbenzene are well below the one-in-a-million benchmark in both the median and 95% UCL. Currently, there is no increased risk for cancer associated with ambient air concentrations of ethylbenzene.

Based on the median concentration (2.20 $\mu g/m^3$), a residential receptor has 18.7 times greater risk of developing cancerous effects than the one-in-a-million benchmark, and an occupational receptor has 4.5 times greater risk of developing cancerous effects than the one-in-a-million benchmark. Based on the 95% UCL of the mean, a residential receptor has 24.3 times greater risk of developing cancerous effects than the one-in-a-million benchmark, and an occupational receptor has 5.8 times greater risk associated with benzene exposure than the benchmark.

There appeared to be an even distribution of benzene among the three factors when coupling risk analysis with PMF results for benzene. The mixed combustion factor was the single highest factor contributing 38% of total benzene. However, the sum of the oil and gas factors, O&NG1 and O&NG2 contribute 62% of total benzene (Figure 14).

PMF Factor Contributions to Benzene

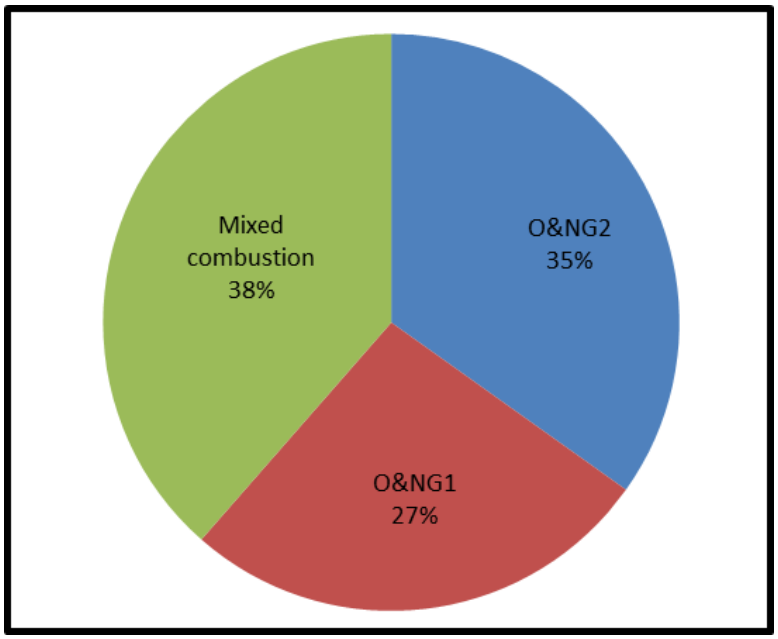

Figure 14. Distribution of total benzene among PMF factors. Mixed combustion contributes the single largest amount of benzene to the total concentration of benzene among factors. However, oil and gas combined contribute the greatest overall.

These concentrations and evaluations of risk were comparable in concentration to studies and assessments for benzene that have occurred in other localities (Fay et al., 2007; Fujita, 2001; Logue et al., 2010; Wallace, 1996). A study that assessed benzene,

toluene and other toxicants in Pittsburgh, PA, and surrounding areas also had risk values in the one-in-one hundred thousand range (Logue et al. 2010). In this case, the main source of benzene for the highest concentration was primarily attributed to metallurgical coke production contributing 66% of the total benzene for that area. The values for benzene listed for that area were comparable to the concentrations seen at the Roosevelt site. Similarly, Busan, South Korea, had risk values greater than the 10^{-6} benchmark for benzene at three sample sites including a background site (Choi et al., 2010b).

The location in Roosevelt, Utah was not a background site where the influence of O&NG was not suspected to be present. As shown in Figure 4, there are two operating oil well locations within one kilometer of the sample site. This may be argued as a confounding factor, however, the sample site was situated in a residential area, and the concentrations taken at that location were immediately applicable to the people living in that area. Therefore, the risk associated with benzene concentrations was arguably indicative of the risk for a residential receiver living in Roosevelt, Utah.

CONCLUSION

A source apportionment performed using positive matrix factorization and HYSPLIT detailed three factors (O&NG1, O&NG2, and Mixed combustion) that contributed NMHC ozone precursors to the ambient air as measured at Roosevelt, Utah. O&NG1 and O&NG2 factors were tightly associated with oil and gas production and mixed combustion was not associated with oil and gas production but was perhaps associated with automotive exhaust, diesel exhaust, and wood burning (Logue et al., 2010; US EPA, 2015b; Wu et al., 2016).

Of the NMHC monitored, the potential non-carcinogenic risk associated with BTEX was determined to be negligible (HQ<1). Cancer risk associated with ethylbenzene was also negligible. However, the cancer risk associated with benzene was greater than the one-in-a-million benchmark set by the USEPA. While the local mixed factor was the single largest to contribute to the total benzene sampled, the combined oil and gas factors in the area played a larger role in the overall concentrations of benzene and benzene associated risk.

These risk calculations for receptors were meant to be a conservative estimate of risk that accounted for sensitive populations and while elevated for benzene, were still within the acceptable range allowed by the USEPA (40 C.F.R. § 300.430). However, given that these concentrations were sampled in a residential area of Roosevelt, Utah, it would be recommended to continue sampling in the area to assess any future potential increases in hazardous air pollutants.

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APPENDICES

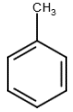
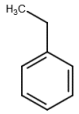
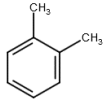
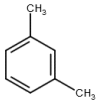
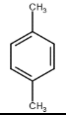
Table A1

NMHC concentrations (ppbv) measured at the sample site in Roosevelt, Utah and used in analysis with Positive Matrix Factorization (PMF). Mean, min, max, median and standard deviation are listed for each.

			Units: g/mol	Units: ppbv				
Number	Name	Chemical classification	Molecular Weight	Mean	S.D.	Min	Max	Median
1	Ethane	Alkane	30.07	72.05	72.45	0.39	396.89	48.90
2	Ethylene	Alkene	28.05	1.05	1.36	0.02	15.32	0.63
3	Propane	Alkane	44.10	45.78	45.26	0.02	243.08	31.77
4	Propylene	Alkane	42.08	1.09	1.43	0.02	7.77	0.38
5	Iso-Butane	Alkane	58.12	7.82	7.93	0.05	44.29	5.16
6	N-Butane	Alkane	58.12	335.47	17.44	0.07	98.38	11.89
7	Acetylene	Alkyne	26.04	0.23	0.17	0.04	1.70	0.18
8	Trans-2-Butene	Alkene	56.11	0.05	0.04	0.01	0.30	0.05
9	1-Butene	Alkene	56.11	1.81	6.83	0.01	84.13	0.10
10	Cis-2-Butene	Alkene	56.11	0.06	0.06	0.01	0.52	0.04
11	Cyclopentane	Alkane	70.10	0.47	0.44	0.01	2.30	0.33
12	Isopentane	Alkane	72.15	6.94	6.96	0.03	40.25	4.83
13	N-Pentane	Alkane	72.15	10.21	12.17	0.02	100.88	5.84
14	Trans-2-Pentene	Alkene	70.14	0.03	0.03	0.01	0.25	0.02
15	Cis-2-Pentene	Alkene	70.14	0.04	0.22	0.01	4.23	0.02
16	2,3-Dimethylbutane	Alkane	86.18	0.10	0.36	0.01	10.16	0.05
17	isoprene	Alkene	68.12	0.33	0.77	0.01	6.40	0.04
18	1-hexene	Alkene	84.16	0.06	0.10	0.01	1.91	0.02
19	n-Hexane	Alkane	86.18	3.24	2.90	0.06	17.09	2.44
20	Methylcyclopentane	Alkane	84.16	1.27	1.18	0.05	6.69	0.93
21	2,4-Dimethylpentane	Alkane	100.21	0.31	0.03	0.26	0.36	0.30
22	Benzene	Aromatic	78.11	0.86	0.62	0.07	5.52	0.69
23	Cyclohexane	Alkane	84.16	1.15	1.07	0.02	6.38	0.87
24	2-Methylhexane	Alkane	100.21	0.51	0.44	0.01	2.60	0.40
25	2,3-Dimethylpentane	Alkane	100.21	0.15	0.13	0.01	0.78	0.12
26	3-Methylhexane	Alkane	100.21	0.52	0.45	0.02	2.49	0.42
27	2,2,4-Trimethylpentane	Alkane	114.23	0.30	0.30	0.01	5.17	0.25
28	N-Heptane	Alkane	100.20	1.99	1.78	0.03	10.18	1.54
29	Methylcyclohexane	Alkane	98.19	1.47	1.39	0.02	7.49	1.12
30	2,3,4-trimethylpentane	Alkane	114.23	0.10	0.05	0.02	0.21	0.10
31	Toluene	Aromatic	92.14	0.88	0.74	0.05	5.12	0.69
32	2-Methylheptane	Alkane	114.23	0.39	0.40	0.01	2.10	0.28
33	3-Methylheptane	Alkane	114.23	0.17	0.14	0.01	0.75	0.13
34	n-Octane	Alkane	114.23	0.67	0.62	0.02	3.47	0.50
35	Ethylbenzene	Aromatic	106.17	0.09	0.06	0.01	0.36	0.07
36	m&p-Xylene	Aromatic	106.17	0.42	0.34	0.01	1.80	0.31
37	Styrene	Aromatic	104.15	0.09	0.05	0.01	0.46	0.09
38	o-Xylene	Aromatic	106.17	0.08	0.07	0.01	0.40	0.06
39	n-Nonane	Alkane	128.20	0.33	0.30	0.01	1.63	0.23
40	m-Ethyltolunene	Aromatic	120.19	0.04	0.02	0.01	0.10	0.04
41	1,3,5-Trimethylbenzene	Aromatic	120.19	0.06	0.04	0.01	0.18	0.05
42	1,2,3-Trimethylbenzene	Aromatic	120.19	0.06	0.03	0.01	0.15	0.05

Table A2

General chemical and physical attributes for BTEX including xylene isomers. Molecular weight, structure, vapor pressure, boiling point, K_{ow}, and LC-50 shown for each BTEX chemical.

Molecule	Molecular Weight	Structure	Vp at 25 C^0	Bp (C^o)	Log K_{ow}[a]	LC-50 (mouse)[b]	References
Benzene	78.11 g/mol		94.5 mmHg	80.1	2.13	33000 mg/m³/4hr	(McCarthy, Hafner, and Montzka 2006), (Reese and Kimbrough 1993), (Fruscella 2000)
Toluene	92.13 g/mol		28 mmHg	110.6	2.73	32800 mg/m³/4hr	(Reese and Kimbrough 1993), (Von Burg 1993), (Williams et al. 2016)
Ethylbenzene	106.7 g/mol		9.53 mmHg	136.19	3.15	35500 mg/m³/2hr	(Cannella 2000), (Taylor et al. 2016), (Welch, Fallon, and Gelbke 2000)
Ortho-xylene	106.7 g/mol		6.61 mmHg	138.37	3.12	20000 mg/m³/6hr	(Cannella 2000), (Welch, Fallon, and Gelbke 2000), (Fay, Risher, and Wilson 2016)
Meta-xylene	106.7 g/mol		8.29 mmHg	139.12	3.2	23000 mg/m³/6hr	(Cannella 2000), (Welch, Fallon, and Gelbke 2000), (Fay, Risher, and Wilson 2016)
Para-xylene	106.7 g/mol		8.84 mmHg	144.41	3.15	17000 mg/m³/6hr	(Cannella 2000), (Welch, Fallon, and Gelbke 2000), (Fay, Risher, and Wilson 2016)

[a] K_{ow} : Octanol-water partition coefficient

[b] LC-50: Concentration that is lethal in 50% of mice. Given in mg/m³ and for the duration exposed