for commercial hexane, which contains about 50-53% n-hexane. Toxicity information on the individual hydrocarbons of this fraction suggests nervous system effects. Some of the compounds have central nervous system effects, as well as liver and kidney effects. Exposure to n-hexane and commercial hexane has also been associated with peripheral neuropathy in a few studies. However, a chronic provisional Reference Concentration (RfC) for commercial hexane based on nasal and laryngeal lesions is considered protective for the peripheral neuropathy by the USEPA.

- **The C9–C18 aliphatic fraction** of the total petroleum hydrocarbons was based on a provisional reference concentration and a provisional inhalation unit risk for midrange aliphatic hydrocarbons streams and solvents and have minimal (<1.0%) aromatic content. The major effects include functional disturbances in the central nervous system including memory and learning impairment. The USEPA derived subchronic and chronic provisional RfCs are based on the critical effects of nasal and/or adrenal lesions.

- **The C9–C16 aromatic fraction** of the total petroleum hydrocarbons was based on a provisional inhalation reference concentration for high flash naphtha. The US EPA has not derived a provisional inhalation unit risk value for this fraction because of the lack of availability of adequate data. The common effects of hydrocarbons in this fraction are kidney, liver, and body weight effects. Also, hematological effects have been observed associated with some of these compounds. The EPA derived chronic provisional RfC is based on the critical effect of maternal body weight depression. Finally, 17 COPCs with no inhalation toxicity values were omitted altogether from any quantitative inhalation risk estimation (Table 3).

## 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) were evaluated in this risk assessment.

### 4.1 Cancer Risk Estimation

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

\[
\text{Risk}_x = \text{EPC}_x \times \text{IUR}_x
\]

Where:
- \(\text{Risk}_x\) = the risk of the \(X^{th}\) COPC at a monitor;
- \(\text{EPC}_x\) = the exposure point concentration of the substance (i.e., most stringent of the 95% UCL or maximum air concentration); and
- \(\text{IUR}_x\) = 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 one chance in one million (or one additional person in 1,000,000) is written as $1 \times 10^{-6}$. 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 were calculated as follows:

$$HQ_x = \frac{EPC_x}{RfC_x}$$

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.

Noncancer hazards are estimated separately for shorter term (acute and subchronic) and long-term (chronic) exposures.

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, characterizing 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 23 out of 90 COPCs, the health risk for 23 COPCs with known toxicity values are quantitatively estimated (Section 4.4.1), the health risks of 50 aliphatic and aromatic fractions of the total petroleum hydrocarbons are semi-quantitatively evaluated using the USEPA (2009) recommended approach (Section 4.4.2), and the health risks for the remaining 17 COPCs with no available toxicity values are qualitatively evaluated (Section 4.4.3). Overall the results are discussed for the following quantitative, semi-quantitative, and qualitative evaluation of COPCs:

1) Quantitative and semi-quantitative evaluation of total cancer risks and noncancer hazard for all 73 COPCs including 23 COPCs with known toxicity values and 50 aliphatic and aromatic hydrocarbons with screening-level surrogate toxicity values (Section 4.4.1)

2) Semi-quantitative evaluation of cancer risks and noncancer hazards for 50 aliphatic and aromatic hydrocarbons with surrogate toxicity values (Section 4.4.2)

3) Qualitative evaluation of cancer risks and noncancer hazards for 17 COPCs with no available toxicity values (Section 4.4.3)
4.4.1 Quantitative and Semi-Quantitative Evaluation of Potential for Cancer and Noncancer Health Effects for All COPCs with Available Toxicity Values

Twenty three chemicals from all four monitoring sites (Bell, Rulison or Battlement Mesa, Parachute, and Rifle) are carried through for evaluation in the risk characterization (Table 1). Of these chemicals, six are evaluated for cancer risk and 23 are evaluated for noncancer hazards quantitatively using the available toxicity values. In addition, 50 aliphatic and aromatic hydrocarbons are discussed in order to address the total risk estimates and the major risk contributing chemicals (Table 2).

4.4.1.1. Cancer Risk Estimates

Six COPCs for cancer risk across the four monitoring sites include acetaldehyde, 1, 3-butadiene, benzene, crotonaldehyde, ethylbenzene, and formaldehyde (Table 1). The majority of these chemicals are classified as “known” or “probable” human carcinogens by the federal agencies (Table G1). In addition, EPA’s provisional screening-level surrogate toxicity values are available to evaluate 45 aliphatic hydrocarbons C₅-C₁₈ with suggestive evidence of the carcinogenic potential (Table G1).

The major findings are briefly discussed below by evaluating the estimated risks in the following four ways across the five monitoring sites from 2008 to 2012:

1) Pattern changes from 2008 to 2012 in the estimated cancer risks (% contribution to total risk) for all carcinogenic COPCs evaluated quantitatively and semi-quantitatively including acetaldehyde, 1, 3-butadiene, benzene, crotonaldehyde, ethylbenzene, formaldehyde, and aliphatic hydrocarbons

2) Total cancer risks estimated quantitatively for six COPCs (acetaldehyde, 1, 3-butadiene, benzene, crotonaldehyde, ethylbenzene, formaldehyde) with known toxicity values and cancer risks

3) Total estimated cancer risks for all carcinogenic COPCs without crotonaldehyde (i.e., acetaldehyde, 1, 3-butadiene, benzene, ethylbenzene, formaldehyde and 45 aliphatic hydrocarbons)

4) Total estimated cancer risks for five COPCs without crotonaldehyde and aliphatic hydrocarbons C₅-C₁₈ (i.e., acetaldehyde, 1, 3-butadiene, benzene, ethylbenzene, and formaldehyde)

Overall, this evaluation indicates a trend toward decreasing cumulative cancer risk estimates for all COPCs evaluated quantitatively and semi-quantitatively from 2008 to 2012 (Table 8 and Figures 1a and1b). The estimated cumulative lifetime cancer range from 98 to 443 in one million, with the highest estimate of 443 in one million in 2008 and the lowest estimate of 98 in one million in 2012 (vs. EPA’s acceptable cancer risk range of 1 to 100 excess cancers in one million or 1E-06 to 1E-04) across all five monitoring