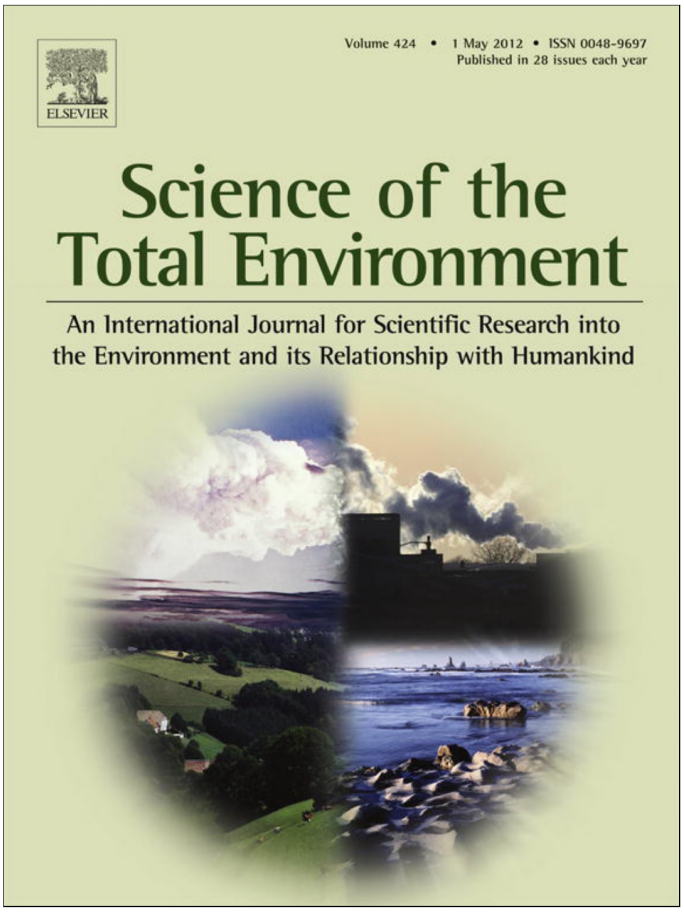

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Human health risk assessment of air emissions from development of unconventional natural gas resources☆,☆☆

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ABSTRACT

Background: Technological advances (e.g. directional drilling, hydraulic fracturing), have led to increases in unconventional natural gas development (NGD), raising questions about health impacts.
Objectives: We estimated health risks for exposures to air emissions from a NGD project in Garfield County, Colorado with the objective of supporting risk prevention recommendations in a health impact assessment (HIA).
Methods: We used EPA guidance to estimate chronic and subchronic non-cancer hazard indices and cancer risks from exposure to hydrocarbons for two populations: (1) residents living >½ mile from wells and (2) residents living ≤½ mile from wells.
Results: Residents living ≤½ mile from wells are at greater risk for health effects from NGD than are residents living >½ mile from wells. Subchronic exposures to air pollutants during well completion activities present the greatest potential for health effects. The subchronic non-cancer hazard index (HI) of 5 for residents ≤½ mile from wells was driven primarily by exposure to trimethylbenzenes, xylenes, and aliphatic hydrocarbons. Chronic HIs were 1 and 0.4. for residents ≤½ mile from wells and >½ mile from wells, respectively. Cumulative cancer risks were 10 in a million and 6 in a million for residents living ≤½ mile and >½ mile from wells, respectively, with benzene as the major contributor to the risk.
Conclusions: Risk assessment can be used in HIAs to direct health risk prevention strategies. Risk management approaches should focus on reducing exposures to emissions during well completions. These preliminary results indicate that health effects resulting from air emissions during unconventional NGD warrant further study. Prospective studies should focus on health effects associated with air pollution.

1. Introduction

The United States (US) holds large reserves of unconventional natural gas resources in coalbeds, shale, and tight sands. Technological advances, such as directional drilling and hydraulic fracturing, have led to a rapid increase in the development of these resources. For example, shale gas production had an average annual growth rate of 48% over the 2006 to 2010 period and is projected to grow almost fourfold from 2009 to 2035 (US EIA, 2011). The number of unconventional natural gas wells in the US rose from 18,485 in 2004 to 25,145 in 2007 and is expected to continue increasing through at least 2020 (Vidas and Hugman, 2008). With this expansion, it is becoming increasingly common for unconventional natural gas development (NGD) to occur near where people live, work, and play. People living near these development sites are raising public health concerns, as rapid NGD exposes more people to various potential stressors (COGCC, 2009a).

The process of unconventional NGD is typically divided into two phases: well development and production (US EPA, 2010a; US DOE, 2009). Well development involves pad preparation, well drilling, and well completion. The well completion process has three primary stages: 1) completion transitions (concrete well plugs are installed in wells to separate fracturing stages and then drilled out to release gas for production); 2) hydraulic fracturing ("fracking": the high pressure injection of water, chemicals, and propants into the drilled well to release the natural gas); and 3) flowback, the return of fracking and geologic fluids, liquid hydrocarbons ("condensate") and natural gas to the surface (US EPA, 2010a; US DOE, 2009). Once development is

Abbreviations: BTEX, benzene, toluene, ethylbenzene, and xylenes; COGCC, Colorardo Oil and Gas Conservation Commission; HAP, hazardous air pollutant; HI, hazard index; HIA, health impact assessment; HQ, hazard quotient; NATA, National Air Toxics Assessment; NGD, natural gas development.

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complete, the "salable" gas is collected, processed, and distributed. While methane is the primary constituent of natural gas, it contains many other chemicals, including alkanes, benzene, and other aromatic hydrocarbons (TERC, 2009).

As shown by ambient air studies in Colorado, Texas, and Wyoming, the NGD process results in direct and fugitive air emissions of a complex mixture of pollutants from the natural gas resource itself as well as diesel engines, tanks containing produced water, and on site materials used in production, such as drilling muds and fracking fluids (CDPHE, 2009; Frazier, 2009; Walther, 2011; Zielinska et al., 2011). The specific contribution of each of these potential NGD sources has yet to be ascertained and pollutants such as petroleum hydrocarbons are likely to be emitted from several of these NGD sources. This complex mixture of chemicals and resultant secondary air pollutants, such as ozone, can be transported to nearby residences and population centers (Walther, 2011; GCPH, 2010).

Multiple studies on inhalation exposure to petroleum hydrocarbons in occupational settings as well as residences near refineries, oil spills and petrol stations indicate an increased risk of eye irritation and headaches, asthma symptoms, acute childhood leukemia, acute myelogenous leukemia, and multiple myeloma (Glass et al., 2003; Kirkeleit et al., 2008; Brosselin et al., 2009; Kim et al., 2009; White et al., 2009). Many of the petroleum hydrocarbons observed in these studies are present in and around NGD sites (TERC, 2009). Some, such as benzene, ethylbenzene, toluene, and xylene (BTEX) have robust exposure and toxicity knowledge bases, while toxicity information for others, such as heptane, octane, and diethylbenzene, is more limited. Assessments in Colorado have concluded that ambient benzene levels demonstrate an increased potential risk of developing cancer as well as chronic and acute non-cancer health effects in areas of Garfield County Colorado where NGD is the only major industry other than agriculture (CDPHE, 2007; Coons and Walker, 2008; CDPHE, 2010). Health effects associated with benzene include acute and chronic nonlymphocytic leukemia, acute myeloid leukemia, chronic lymphocytic leukemia, anemia, and other blood disorders and immunological effects. (ATSDR, 2007a, IRIS, 2011). In addition, maternal exposure to ambient levels of benzene recently has been associated with an increase in birth prevalence of neural tube defects (Lupo et al., 2011). Health effects of xylene exposure include eye, nose, and throat irritation, difficulty in breathing, impaired lung function, and nervous system impairment (ATSDR, 2007b). In addition, inhalation of xylenes, benzene, and alkanes can adversely affect the nervous system (Carpenter et al., 1978; Nilsen et al., 1988; Galvin and Marashi, 1999; ATSDR, 2007a; ATSDR, 2007b).

Previous assessments are limited in that they were not able to distinguish between risks from ambient air pollution and specific NGD stages, such as well completions or risks between residents living near wells and residents living further from wells. We were able to isolate risks to residents living near wells during the flowback stage of well completions by using air quality data collected at the perimeter of the wells while flowback was occurring.

Battlement Mesa (population ~5000) located in rural Garfield County, Colorado is one community experiencing the rapid expansion of NGD in an unconventional tight sand resource. A NGD operator has proposed developing 200 gas wells on 9 well pads located as close as 500 ft from residences. Colorado Oil and Gas Commission (COGCC) rules allow natural gas wells to be placed as close as 150 ft from residences (COGCC, 2009b). Because of community concerns, as described elsewhere, we conducted a health impact assessment (HIA) to assess how the project may impact public health (Witter et al., 2011), working with a range of stakeholders to identify the potential public health risks and benefits.

In this article, we illustrate how a risk assessment was used to support elements of the HIA process and inform risk prevention recommendations by estimating chronic and subchronic non-cancer hazard indices (HIs) and lifetime excess cancer risks due to NGD air emissions.

2. Methods

We used standard United States Environmental Protection Agency (EPA) methodology to estimate non-cancer HIs and excess lifetime cancer risks for exposures to hydrocarbons (US EPA, 1989; US EPA, 2004) using residential exposure scenarios developed for the NGD project. We used air toxics data collected in Garfield County from January 2008 to November 2010 as part of a special study of short term exposures as well as on-going ambient air monitoring program data to estimate subchronic and chronic exposures and health risks (Frazier, 2009; GCPH, 2009; GCPH, 2010; GCPH, 2011; Antero, 2010).

2.1. Sample collection and analysis

All samples were collected and analyzed according to published EPA methods. Analyses were conducted by EPA certified laboratories. The Garfield County Department of Public Health (GCPH) and Olsson Associates, Inc. (Olsson) collected ambient air samples into evacuated SUMMA® passivated stainless-steel canisters over 24-hour intervals. The GCPH collected the samples from a fixed monitoring station and along the perimeters of four well pads and shipped samples to Eastern Research Group for analysis of 78 hydrocarbons using EPA's compendium method TO-12, Method for the Determination of Non-Methane Organic Compounds in Ambient Air Using Cyrogenic Preconcentration and Direct Flame Ionization Detection (US EPA, 1999). Olsson collected samples along the perimeter of one well pad and shipped samples to Atmospheric Analysis and Consulting, Inc. for analysis of 56 hydrocarbons (a subset of the 78 hydrocarbons determined by Eastern Research Group) using method TO-12. Per method TO-12, a fixed volume of sample was cryogenically concentrated and then desorbed onto a gas chromatography column equipped with a flame ionization detector. Chemicals were identified by retention time and reported in a concentration of parts per billion carbon (ppbC). The ppbC values were converted to micrograms per cubic meter ($\mu g/m^3$) at 01.325 kPa and 298.15 K.

Two different sets of samples were collected from rural (population<50,000) areas in western Garfield County over varying time periods. The main economy, aside from the NGD industry, of western Garfield County is agricultural. There is no other major industry.

2.1.1. NGD area samples

The GCPH collected ambient air samples every six days between January 2008 and November 2010 (163 samples) from a fixed monitoring station located in the midst of rural home sites and ranches and NGD, during both well development and production. The site is located on top of a small hill and 4 miles upwind of other potential emission sources, such as a major highway (Interstate-70) and the town of Silt, CO (GCPH, 2009; GCPH, 2010; GCPH, 2011).

2.1.2. Well completion samples

The GCPH collected 16 ambient air samples at each cardinal direction along 4 well pad perimeters (130 to 500 ft from the well pad center) in rural Garfield County during well completion activities. The samples were collected on the perimeter of 4 well pads being developed by 4 different natural gas operators in summer 2008 (Frazier, 2009). The GCPH worked closely with the NGD operators to ensure these air samples were collected during the period while at least one well was on uncontrolled (emissions not controlled) flowback into collection tanks vented directly to the air. The number of wells on each pad and other activities occurring on the pad were not documented. Samples were collected over 24 to 27-hour intervals, and samples included emissions from both uncontrolled flowback and

diesel engines (i.e., from. trucks and generators supporting completion activities). In addition, the GCPH collected a background sample 0.33 to 1 mile from each well pad (Frazier, 2009). The highest hydrocarbon levels corresponded to samples collected directly downwind of the tanks (Frazier, 2009; Antero, 2010). The lowest hydrocarbon levels corresponded either to background samples or samples collected upwind of the flowback tanks (Frazier, 2009; Antero, 2010).

Antero Resources Inc., a natural gas operator, contracted Olsson to collect eight 24-hour integrated ambient air samples at each cardinal direction at 350 and 500 ft from the well pad center during well completion activities conducted on one of their well pads in summer 2010 (Antero, 2010). Of the 12 wells on this pad, 8 were producing salable natural gas; 1 had been drilled but not completed; 2 were being hydraulically fractured during daytime hours, with ensuing uncontrolled flowback during nighttime hours; and 1 was on uncontrolled flowback during nighttime hours.

All five well pads are located in areas with active gas production, approximately 1 mile from Interstate-70.

2.2. Data assessment

We evaluated outliers and compared distributions of chemical concentrations from NGD area and well completion samples using Q–Q plots and the Mann–Whitney *U* test, respectively, in EPA's ProUCL version 4.00.05 software (US EPA, 2010b). The Mann–Whitney *U* test was used because the measurement data were not normally distributed. Distributions were considered as significantly different at an alpha of 0.05. Per EPA guidance, we assigned the exposure concentration as either the 95% upper confidence limit (UCL) of the mean concentration for compounds found in 10 or more samples or the maximum detected concentration for compounds found in more than 1 but fewer than 10 samples. This latter category included three compounds: 1,3-butadiene, 2,2,4-trimethylpentane, and styrene in the well completion samples. EPA's ProUCL software was used to select appropriate methods based on sample distributions and detection frequency for computing 95% UCLs of the mean concentration (US EPA, 2010b).

2.3. Exposure assessment

Risks were estimated for two populations: (1) residents >½ mile from wells; and (2) residents ≤½ mile from wells. We defined residents ≤½ mile from wells as living near wells, based on residents reporting odor complaints attributed to gas wells in the summer of 2010 (COGCC, 2011).

Exposure scenarios were developed for chronic non-cancer HIs and cancer risks. For both populations, we assumed a 30-year project duration based on an estimated 5-year well development period for all well pads, followed by 20 to 30 years of production. We assumed a resident lives, works, and otherwise remains within the town 24 h/day, 350 days/year and that lifetime of a resident is 70 years, based on standard EPA reasonable maximum exposure (RME) defaults (US EPA, 1989).

2.3.1. Residents >½ mile from well pads

As illustrated in Fig. 1, data from the NGD area samples were used to estimate chronic and subchronic risks for residents >½ mile from well development and production throughout the project. The exposure concentrations for this population were the 95% UCL on the mean concentration and median concentration from the 163 NGD samples.

2.3.2. Residents ≤½ mile from well pads

To evaluate subchronic non-cancer HIs from well completion emissions, we estimated that a resident lives ≤½ mile from two well pads resulting a 20-month exposure duration based on 2 weeks per well for completion and 20 wells per pad, assuming some overlap in between activities. The subchronic exposure concentrations for this population were the 95% UCL on the mean concentration and the median concentration from the 24 well completion samples. To evaluate chronic risks to residents ≤½ mile from wells throughout the NGD project, we calculated a time-weighted exposure concentration (C_{S+c}) to account for exposure to emissions from well completions for 20-months followed by 340 months of exposure to emissions from the NGD area using the following formula:

$$C_{S+c} = (C_c \times ED_c/ED) + (C_S \times ED_S/ED)$$

where:

C_c Chronic exposure point concentration ($\mu g/m^3$) based on the 95% UCL of the mean concentration or median concentration from the 163 NGD area samples

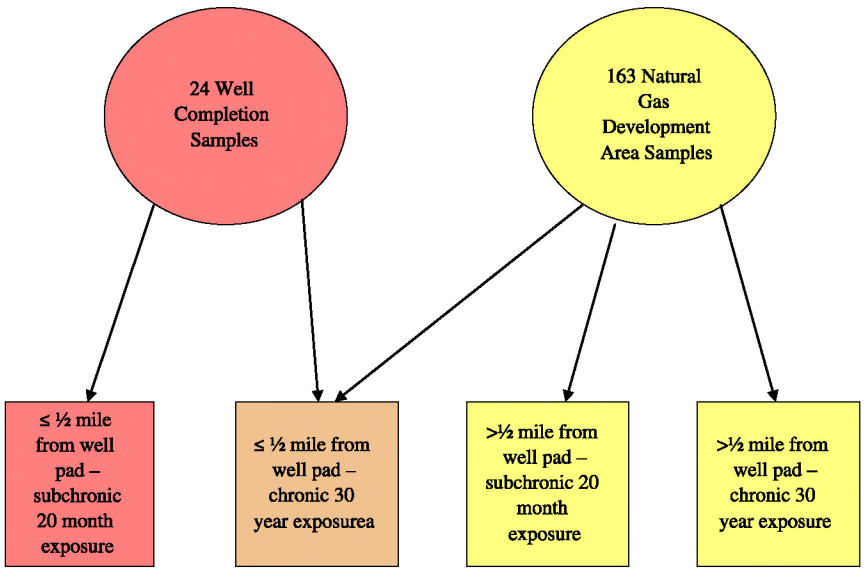

Fig. 1. Relationship between completion samples and natural gas development area samples and residents living ≤½ mile and >½ mile from wells. [a]Time weighted average based on 20-month contribution from well completion samples and 340-month contribution from natural gas development samples.

ED_c Chronic exposure duration
C_S Subchronic exposure point concentration (μg/m^3) based on the 95% UCL of the mean concentration or median concentration from the 24 well completion samples
ED_S Subchronic exposure duration
ED Total exposure duration

2.4. Toxicity assessment and risk characterization

For non-carcinogens, we expressed inhalation toxicity measurements as a reference concentration (RfC in units of μg/m^3 air). We used chronic RfCs to evaluate long-term exposures of 30 years and subchronic RfCs to evaluate subchronic exposures of 20-months. If a subchronic RfC was not available, we used the chronic RfC. We obtained RfCs from (in order of preference) EPA's Integrated Risk Information System (IRIS) (US EPA, 2011), California Environmental Protection Agency (CalEPA) (CalEPA, 2003), EPA's Provisional Peer-Reviewed Toxicity Values (ORNL, 2009), and Health Effects Assessment Summary Tables (US EPA, 1997). We used surrogate RfCs according to EPA guidance for C_5 to C_{18} aliphatic and C_6 to C_{18} aromatic hydrocarbons which did not have a chemical-specific toxicity value (US EPA, 2009a). We derived semi-quantitative hazards, in terms of the hazard quotient (HQ), defined as the ratio between an estimated exposure concentration and RfC. We summed HQs for individual compounds to estimate the total cumulative HI. We then separated HQs specific to neurological, respiratory, hematological, and developmental effects and calculated a cumulative HI for each of these specific effects.

For carcinogens, we expressed inhalation toxicity measurements as inhalation unit risk (IUR) in units of risk per μg/m^3. We used IURs from EPA's IRIS (US EPA, 2011) when available or the CalEPA (CalEPA, 2003). The lifetime cancer risk for each compound was derived by multiplying estimated exposure concentration by the IUR. We summed cancer risks for individual compounds to estimate the cumulative cancer risk. Risks are expressed as excess cancers per 1 million population based on exposure over 30 years.

Toxicity values (i.e., RfCs or IURs) or a surrogate toxicity value were available for 45 out of 78 hydrocarbons measured. We performed a quantitative risk assessment for these hydrocarbons. The remaining 33 hydrocarbons were considered qualitatively in the risk assessment.

3. Results

3.1. Data assessment

Evaluation of potential outliers revealed no sampling, analytical, or other anomalies were associated with the outliers. In addition, removal of potential outliers from the NGD area samples did not change the final HIs and cancer risks. Potential outliers in the well completion samples were associated with samples collected downwind from flowback tanks and are representative of emissions during flowback. Therefore, no data was removed from either data set.

Descriptive statistics for concentrations of the hydrocarbons used in the quantitative risk assessment are presented in Table 1. A list of the hydrocarbons detected in the samples that were considered qualitatively in the risk assessment because toxicity values were not available is presented in Table 2. Descriptive statistics for all hydrocarbons are available in Supplemental Table 1. Two thirds more hydrocarbons were detected at a frequency of 100% in the well completion samples (38 hydrocarbons) than in the NGD area samples (23 hydrocarbons). Generally, the highest alkane and aromatic hydrocarbon median concentrations were observed in the well completion samples, while the highest median concentrations of several alkenes were observed in the NGD area samples. Median concentrations of benzene, ethylbenzene, toluene, and m-xylene/p-xlyene were 2.7, 4.5, 4.3, and 9 times higher in the well completion samples than in the NGD area samples, respectively. Wilcoxon–Mann–Whitney test results indicate that

Table 1
Descriptive statistics for hydrocarbon concentrations with toxicity values in 24-hour integrated samples collected in NGD area and samples collected during well completions.

Hydrocarbon (μg/m^3)	NGD area sample results[a]							Well completion sample results[b]						
	No.	% > MDL	Med	SD	95% UCL[c]	Min	Max	No.	% > MDL	Med	SD	95% UCL[c]	Min	Max
1,2,3-Trimethylbenzene	163	39	0.11	0.095	0.099	0.022	0.85	24	83	0.84	2.3	3.2	0.055	12
1,2,4-Trimethylbenzene	163	96	0.18	0.34	0.31	0.063	3.1	24	100	1.7	17	21	0.44	83
1,3,5-Trimethylbenzene	163	83	0.12	0.13	0.175	0.024	1.2	24	100	1.3	16	19.5	0.33	78
1,3-Butadiene	163	7	0.11	0.020	0.0465	0.025	0.15	16	56	0.11	0.021	NC	0.068	0.17
Benzene	163	100	0.95	1.3	1.7	0.096	14	24	100	2.6	14	20	0.94	69
Cyclohexane	163	100	2.1	8.3	6.2	0.11	105	24	100	5.3	43	58	2.21	200
Ethylbenzene	163	95	0.17	0.73	0.415	0.056	8.1	24	100	0.77	47	54	0.25	230
Isopropylbenzene	163	38	0.15	0.053	0.074	0.020	0.33	24	67	0.33	1.0	1.0	0.0	4.8
Methylcyclohexane	163	100	3.7	4.0	6.3	0.15	24	24	100	14	149	190	3.1	720
m-Xylene/p-Xylene	163	100	0.87	1.2	1.3	0.16	9.9	24	100	7.8	194	240	2.0	880
n-Hexane	163	100	4.0	4.2	6.7	0.13	25	24	100	7.7	57	80	1.7	255
n-Nonane	163	99	0.44	0.49	0.66	0.064	3.1	24	100	3.6	61	76	1.2	300
n-Pentane	163	100	9.1	9.8	14	0.23	62	24	100	11	156	210	3.9	550
n-Propylbenzene	163	66	0.10	0.068	0.10	0.032	0.71	24	88	0.64	2.4	3.3	0.098	12
o-Xylene	163	97	0.22	0.33	0.33	0.064	3.6	24	100	1.2	40	48.5	0.38	190
Propylene	163	100	0.34	0.23	0.40	0.11	2.5	24	100	0.41	0.34	0.60	0.16	1.9
Styrene	163	15	0.15	0.26	0.13	0.017	3.4	24	21	0.13	1.2	NC	0.23	5.9
Toluene	163	100	1.8	6.2	4.8	0.11	79	24	100	7.8	67	92	2.7	320
Aliphatic hydrocarbons C_5–C_8[d]	163	NC	29	NA	44	1.7	220	24	NC	56	NA	780	24	2700
Aliphatic hydrocarbons C_9–C_{18}[e]	163	NC	1.3	NA	14	0.18	400	24	NC	7.9	NA	100	1.4	390
Aromatic hydrocarbons C_9–C_{18}[f]	163	NC	0.57	NA	0.695	0.17	5.6	24	NC	3.7	NA	27	0.71	120

Abbreviations: Max, maximum detected concentration; Med, median; Min, minimum detected concentration; NGD, natural gas development; NC, not calculated; No., number of samples; SD, standard deviation; % > MDL, percent greater than method detection limit; μg/m^3 micrograms per cubic meter; 95% UCL 95% upper confidence limit on the mean.

[a] Samples collected at one site every 6 six days between 2008 and 2010.

[b] Samples collected at four separate sites in summer 2008 and one site in summer 2010.

[c] Calculated using EPA's ProUCL version 4.00.05 software (US EPA, 2010b).

[d] Sum of 2,2,2-trimethylpentane, 2,2,4-trimethylpentane, 2,2-dimethylbutane, 2,3,4-trimethylpentane, 2,3-dimethylbutane, 2,3-dimethylpentane, 2,4-dimethylpentane, 2-methylheptane, 2-methylhexane, 2-methylpentane, 3-methylheptane, 3-methylhexane, 3-methylpentane, cyclopentane, isopentane, methylcyclopentane, n-heptane, n-octane.

[e] Sum of n-decane, n-dodecane, n-tridecane, n-undecane.

[f] Sum of m-diethylbenzene, m-ethyltoluene, o-ethyltoluene, p-diethylbenzene, p-ethyltoluene.

Table 2
Detection frequencies of hydrocarbons without toxicity values detected in NGD area or well completion samples.

Hydrocarbon	NGD area sample[a] detection frequency (%)	Well completion sample[b] detection frequency (%)
1-Dodecene	36	81
1-Heptene	94	100
1-Hexene	63	79
1-Nonene	52	94
1-Octene	29	75
1-Pentene	98	79
1-Tridecene	7	38
1-Undecene	28	81
2-Ethyl-1-butene	1	0
2-Methyl-1-butene	29	44
2-Methyl-1-pentene	1	6
2-Methyl-2-butene	36	69
3-Methyl-1-butene	6	6
4-Methyl-1-pentene	16	69
Acetylene	100	92
a-Pinene	63	100
b-Pinene	10	44
cis-2-Butene	58	75
cis-2-Hexene	13	81
cis-2-Pentene	38	54
Cyclopentene	44	94
Ethane	100	100
Ethylene	100	100
Isobutane	100	100
Isobutene/1-Butene	73	44
Isoprene	71	96
n-Butane	98	100
Propane	100	100
Propyne	1	0
trans-2-Butene	80	75
trans-2-Hexene	1	6
trans-2-Pentene	55	83

Abbreviations: NGD, natural gas development.
[a] Samples collected at one site every 6 six days between 2008 and 2010.
[b] Samples collected at four separate sites in summer 2008 and one site in summer 2010.

concentrations of hydrocarbons from well completion samples were significantly higher than concentrations from NGD area samples ($p<0.05$) with the exception of 1,2,3-trimethylbenzene, n-pentane, 1,3-butadiene, isopropylbenzene, n-propylbenzene, propylene, and styrene (Supplemental Table 2).

3.2. Non-cancer hazard indices

Table 3 presents chronic and subchronic RfCs used in calculating non-cancer HIs, as well critical effects and other effects. Chronic non-cancer HQ and HI estimates based on ambient air concentrations are presented in Table 4. The total chronic HIs based on the 95% UCL of the mean concentration were 0.4 for residents >½ mile from wells and 1 for residents ≤½ mile from wells. Most of the chronic non-cancer hazard is attributed to neurological effects with neurological HIs of 0.3 for residents >½ mile from wells and 0.9 for residents ≤½ mile from wells.

Total subchronic non-cancer HQs and HI estimates are presented in Table 5. The total subchronic HIs based on the 95% UCL of the mean concentration were 0.2 for residents >½ mile from wells and 5 for residents ≤½ mile from wells. The subchronic non-cancer hazard for residents >½ mile from wells is attributed mostly to respiratory effects (HI = 0.2), while the subchronic hazard for residents ≤½ mile from wells is attributed to neurological (HI = 4), respiratory (HI = 2), hematologic (HI = 3), and developmental (HI = 1) effects.

For residents >½ mile from wells, aliphatic hydrocarbons (51%), trimethylbenzenes (22%), and benzene (14%) are primary contributors to the chronic non-cancer HI. For residents ≤½ mile from wells, trimethylbenzenes (45%), aliphatic hydrocarbons (32%), and xylenes (17%) are primary contributors to the chronic non-cancer HI, and trimethylbenzenes (46%), aliphatic hydrocarbons (21%) and xylenes (15%) also are primary contributors to the subchronic HI.

3.3. Cancer risks

Cancer risk estimates calculated based on measured ambient air concentrations are presented in Table 6. The cumulative cancer risks based on the 95% UCL of the mean concentration were 6 in a million for residents >½ from wells and 10 in a million for residents ≤½ mile from wells. Benzene (84%) and 1,3-butadiene (9%) were the primary contributors to cumulative cancer risk for residents >½ mile from wells. Benzene (67%) and ethylbenzene (27%) were the primary contributors to cumulative cancer risk for residents ≤½ mile from wells.

4. Discussion

Our results show that the non-cancer HI from air emissions due to natural gas development is greater for residents living closer to wells. Our greatest HI corresponds to the relatively short-term (i.e., subchronic), but high emission, well completion period. This HI is driven principally by exposure to trimethylbenzenes, aliphatic hydrocarbons, and xylenes, all of which have neurological and/or respiratory effects. We also calculated higher cancer risks for residents living nearer to wells as compared to residents residing further from wells. Benzene is the major contributor to lifetime excess cancer risk for both scenarios. It also is notable that these increased risk metrics are seen in an air shed that has elevated ambient levels of several measured air toxics, such as benzene (CDPHE, 2009; GCPH, 2010).

4.1. Representation of exposures from NGD

It is likely that NGD is the major source of the hydrocarbons observed in the NGD area samples used in this risk assessment. The NGD area monitoring site is located in the midst of multi-acre rural home sites and ranches. Natural gas is the only industry in the area other than agriculture. Furthermore, the site is at least 4 miles upwind from any other major emission source, including Interstate 70 and the town of Silt, Colorado. Interestingly, levels of benzene, m,p-xylene, and 1,3,5-trimethylbenzene measured at this rural monitoring site in 2009 were higher than levels measured at 27 out of 37 EPA air toxics monitoring sites where SNMOCs were measured, including urban sites such as Elizabeth, NJ, Dearborn, MI, and Tulsa, OK (GCPH, 2010; US EPA, 2009b). In addition, the 2007 Garfield County emission inventory attributes the bulk of benzene, xylene, toluene, and ethylbenzene emissions in the county to NGD, with NGD point and non-point sources contributing five times more benzene than any other emission source, including on-road vehicles, wildfires, and wood burning. The emission inventory also indicates that NGD sources (e.g. condensate tanks, drill rigs, venting during completions, fugitive emissions from wells and pipes, and compressor engines) contributed ten times more VOC emissions than any source, other than biogenic sources (e.g. plants, animals, marshes, and the earth) (CDPHE, 2009).

Emissions from flowback operations, which may include emissions from various sources on the pads such as wells and diesel engines, are likely the major source of the hydrocarbons observed in the well completion samples. These samples were collected very near (130 to 500 ft from the center) well pads during uncontrolled flowback into tanks venting directly to the air. As for the NGD area samples, no sources other than those associated with NGD were in the vicinity of the sampling locations.

Subchronic health effects, such as headaches and throat and eye irritation reported by residents during well completion activities

Table 3
Chronic and subchronic reference concentrations, critical effects, and major effects for hydrocarbons in quantitative risk assessment.

Hydrocarbon	Chronic		Subchronic		Critical effect/ target organ	Other effects
	RfC ($\mu g/m^3$)	Source	RfC ($\mu g/m^3$)	Source		
1,2,3-Trimethylbenzene	5.00E+00	PPTRV	5.00E+01	PPTRV	Neurological	Respiratory, hematological
1,3,5-Trimethylbenzene	6.00E+00	PPTRV	1.00E+01	PPTRV	Neurological	Hematological
Isopropylbenzene	4.00E+02	IRIS	9.00E+01	HEAST	Renal	Neurological, respiratory
n-Hexane	7.00E+02	IRIS	2.00E+03	PPTRV	Neurological	–
n-Nonane	2.00E+02	PPTRV	2.00E+03	PPTRV	Neurological	Respiratory
n-Pentane	1.00E+03	PPTRV	1.00E+04	PPTRV	Neurological	–
Styrene	1.00E+03	IRIS	3.00E+03	HEAST	Neurological	–
Toluene	5.00E+03	IRIS	5.00E+03	PPTRV	Neurological	Developmental, respiratory
Xylenes, total	1.00E+02	IRIS	4.00E+02	PPTRV	Neurological	Developmental, respiratory
n-propylbenzene	1.00E+03	PPTRV	1.00E+03	Chronic RfC PPTRV	Developmental	Neurological
1,2,4-Trimethylbenzene	7.00E+00	PPTRV	7.00E+01	PPTRV	Decrease in blood clotting time	Neurological, respiratory
1,3-Butadiene	2.00E+00	IRIS	2.00E+00	Chronic RfC IRIS	Reproductive	Neurological, respiratory
Propylene	3.00E+03	CalEPA	1.00E+03	Chronic RfC CalEPA	Respiratory	–
Benzene	3.00E+01	ATSDR	8.00E+01	PPTRV	Decreased lymphocyte count	Neurological, developmental, reproductive
Ethylbenzene	1.00E+03	ATSDR	9.00E+03	PPTRV	Auditory	Neurological, respiratory, renal
Cyclohexane	6.00E+03	IRIS	1.80E+04	PPTRV	Developmental	Neurological
Methylcyclohexane	3.00E+03	HEAST	3.00E+03	HEAST	Renal	–
Aliphatic hydrocarbons C_5–C_8[a]	6E+02	PPTRV	2.7E+04	PPTRV	Neurological	–
Aliphatic hydrocarbons C_9–C_{18}	1E+02	PPTRV	1E+02	PPTRV	Respiratory	–
Aromatic hydrocarbons C_9–C_{18}[b]	1E+02	PPTRV	1E+03	PPRTV	Decreased maternal body weight	Respiratory

Abbreviations: 95%UCL, 95% upper confidence limit; CalEPA, California Environmental Protection Agency; HEAST, EPA Health Effects Assessment Summary Tables 1997; HQ, hazard quotient; IRIS, Integrated Risk Information System; Max, maximum; PPTRV, EPA Provisional Peer-Reviewed Toxicity Value; RfC, reference concentration; $\mu g/m^3$, micrograms per cubic meter. Data from CalEPA 2011; IRIS (US EPA, 2011); ORNL 2011.

[a] Based on PPTRV for commercial hexane.

[b] Based on PPTRV for high flash naphtha.

occurring in Garfield County, are consistent with known health effects of many of the hydrocarbons evaluated in this analysis (COGCC, 2011; Witter et al., 2011). Inhalation of trimethylbenzenes and xylenes can irritate the respiratory system and mucous membranes with effects ranging from eye, nose, and throat irritation to difficulty in breathing and impaired lung function (ATSDR, 2007a;

Table 4
Chronic hazard quotients and hazard indices for residents living >½ mile from wells and residents living ≤½ mile from wells.

Hydrocarbon	>½ mile		≤½ mile	
	Chronic HQ based on median concentration	Chronic HQ based on 95% UCL of mean concentration	Chronic HQ based on median concentration	Chronic HQ based on 95% UCL of mean concentration
1,2,3-Trimethylbenzene	2.09E−02	1.90E−02	2.87E−02	5.21E−02
1,2,4-Trimethylbenzene	2.51E−02	4.22E−02	3.64E−02	2.01E−01
1,3,5-Trimethylbenzene	1.96E−02	2.80E−02	3.00E−02	1.99E−01
1,3-Butadiene	5.05E−02	2.23E−02	5.05E−02	2.25E−02
Benzene	3.03E−02	5.40E−02	3.32E−02	8.70E−02
Cyclohexane	3.40E−04	9.98E−04	3.67E−04	1.46E−03
Ethylbenzene	1.63E−04	3.98E−04	1.95E−04	3.23E−03
Isopropylbenzene	3.68E−04	1.78E−04	3.90E−04	3.05E−04
Methylcyclohexane	1.18E−03	2.00E−03	1.36E−03	5.32E−03
n-Hexane	5.49E−03	9.23E−03	5.76E−03	1.47E−02
n-Nonane	2.11E−03	3.14E−03	2.95E−03	2.31E−02
n-Pentane	8.71E−03	1.32E−02	8.79E−03	2.39E−02
n-propylbenzene	9.95E−05	9.59E−05	1.28E−04	2.64E−04
Propylene	1.09E−04	1.27E−04	1.10E−04	1.30E−04
Styrene	1.43E−04	1.25E−04	1.42E−04	4.32E−04
Toluene	3.40E−04	9.28E−04	4.06E−04	1.86E−03
Xylenes, total	1.16E−02	1.57E−02	1.54E−02	1.71E−01
Aliphatic hydrocarbons C_5–C_8	4.63E−02	7.02E−02	4.87E−02	1.36E−01
Aliphatic hydrocarbons C_9–C_{18}	1.22E−02	1.35E−01	1.58E−02	1.83E−01
Aromatic hydrocarbons C_9–C_{18}	5.44E−03	6.67E−03	7.12E−03	2.04E−02
Total Hazard Index	2E−01	4E−01	3E−01	1E+00
Neuorological Effects Hazard Index[a]	2E−01	3E−01	3E−01	9E−01
Respiratory Effects Hazard Index[b]	1E−01	2E−02	2E−02	7E−01
Hematogical Effects Hazard Index[c]	1E−01	1E−01	1E−01	5E−01
Developmental Effects Hazard Index[d]	4E−02	7E−02	5E−02	3E−01

Abbreviations: 95%UCL, 95% upper confidence limit; HQ, hazard quotient.

[a] Sum of HQs for hydrocarbons with neurological effects: 1,2,3-Trimethylbenzene, 1,2,4-Trimethylbenzene, 1,3,5-Trimethylbenzene, 1,3-butadiene, benzene, cyclohexane, ethylbenzene, isopropylbenzene, n-hexane, n-nonane, n-pentane, n-propylbenzene, styrene, toluene, xylenes, aliphatic C_5–C_8 hydrocarbons.

[b] Sum of HQs for hydrocarbons with respiratory effects: 1,2,3-Trimethylbenzene, 1,2,4-Trimethylbenzene, 1,3-butadiene, ethylbenzene, isopropylbenzene, n-nonane, propylene, toluene, xylenes, aliphatic C_9–C_{18} hydrocarbons, aromatic C_9–C_{18} hydrocarbons.

[c] Sum of HQs for hydrocarbons with hematological effects: 1,2,3-trimethylbenzene, 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene, benzene.

[d] Sum of HQs for hydrocarbons with developmental effects: benzene, cyclohexane, toluene, and xylenes.

Table 5
Subchronic hazard quotients and hazard indices residents living >½ mile from wells and residents living ≤½ mile from wells.

Hydrocarbon (μg/m^3)	>½ mile		≤½ mile	
	Subchronic HQ based on median concentration	Subchronic HQ based on 95% UCL of mean concentration	Subchronic HQ based on median concentration	Subchronic HQ based on 95% UCL of mean concentration
1,2,3-Trimethylbenzene	2.09E−03	1.90E−03	1.67E−02	6.40E−02
1,2,4-Trimethylbenzene	2.51E−03	4.22E−03	2.38E−02	3.02E−01
1,3,5-Trimethylbenzene	1.18E−02	1.68E−02	1.29E−01	1.95E+00
1,3-Butadiene	5.04E−02	2.23E−02	5.25E−02	8.30E−02
Benzene	1.14E−02	2.02E−02	3.25E−02	2.55E−01
Cyclohexane	1.13E−04	3.33E−04	2.93E−04	3.24E−03
Ethylbenzene	1.81E−05	4.42E−05	8.56E−05	5.96E−03
Isopropylbenzene	1.63E−03	7.92E−04	3.62E−03	1.14E−02
Methylcyclohexane	1.18E−03	2.01E−03	4.67E−03	6.47E−02
n-Hexane	1.92E−03	3.23E−03	3.86E−03	3.98E−02
n-Nonane	2.11E−04	3.14E−04	1.80E−03	3.78E−02
n-Pentane	8.71E−04	1.32E−03	1.05E−03	2.13E−02
n-propylbenzene	9.95E−05	9.57E−05	6.36E−04	3.26E−03
Propylene	1.43E−04	3.80E−04	4.12E−04	6.02E−04
Styrene	5.68E−04	4.16E−05	4.00E−06	1.97E−03
Toluene	4.18E−05	9.28E−04	2.46E−04	1.84E−02
Xylenes, total	2.91E−03	3.93E−03	2.05E−02	7.21E−01
Aliphatic hydrocarbons C_5–C_8	1.07E−03	1.63E−03	2.07E−03	2.89E−02
Aliphatic hydrocarbons C_9–C_{18}	1.3E−02	1.41E−01	7.9E−02	1.03E−00
Aromatic hydrocarbons C_9–C_{18}	6.00E−04	6.95E−04	3.7E−03	2.64E−02
Total Hazard Index	1E−01	2E−01	4E−01	5E+00
Neuorological Effects Hazard Index[a]	9E−02	8E−02	3E−01	4E+00
Respiratory Effects Hazard Index[b]	7E−02	2E−01	2E−01	2E+00
Hematogical Effects Hazard Index[c]	3E−02	4E−02	2E−01	3E+00
Developmental Effects Hazard Index[d]	1E−02	3E−02	5E−02	1E+00

Abbreviations: 95%UCL, 95% upper confidence limit; HQ, hazard quotient.
[a] Sum of HQs for hydrocarbons with neurological effects: 1,2,3-Trimethylbenzene, 1,2,4-Trimethylbenzene, 1,3,5-Trimethylbenzene, 1,3-butadiene, benzene, cyclohexane, ethylbenzene, isopropylbenzene, n-hexane, n-nonane, n-pentane, n-propylbenzene, styrene, toluene, xylenes, aliphatic C_5–C_8 hydrocarbons.
[b] Sum of HQs for hydrocarbons with respiratory effects: 1,2,3-Trimethylbenzene, 1,2,4-Trimethylbenzene, 1,3-butadiene, ethylbenzene, isopropylbenzene, n-nonane, propylene, toluene, xylenes, aliphatic C_9–C_{18} hydrocarbons, aromatic C_9–C_{18} hydrocarbons.
[c] Sum of HQs for hydrocarbons with hematological effects: 1,2,3-trimethylbenzene, 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene, benzene.
[d] Sum of HQs for hydrocarbons with developmental effects: benzene, cyclohexane, toluene, and xylenes.

ATSDR, 2007b; US EPA, 1994). Inhalation of trimethylbenzenes, xylenes, benzene, and alkanes can adversely affect the nervous system with effects ranging from dizziness, headaches, fatigue at lower exposures to numbness in the limbs, incoordination, tremors, temporary limb paralysis, and unconsciousness at higher exposures (Carpenter et al., 1978; Nilsen et al., 1988; US EPA, 1994; Galvin and Marashi, 1999; ATSDR, 2007a; ATSDR, 2007b).

4.2. Risk assessment as a tool for health impact assessment

HIA is a policy tool used internationally that is being increasingly used in the United States to assess multiple complex hazards and exposures in communities. Comparison of risks between residents based on proximity to wells illustrates how the risk assessment process can be used to support the HIA process. An important component of the HIA process is to identify where and when public health is most likely to be impacted and to recommend mitigations to reduce or eliminate the potential impact (Collins and Koplan, 2009). This risk assessment indicates that public health most likely would be impacted by well completion activities, particularly for residents living nearest the wells. Based on this information, suggested risk prevention strategies in the HIA are directed at minimizing exposures for those living closet to the well pads, especially during well completion activities when emissions are the highest. The HIA includes recommendations to (1) control and monitor emissions during completion transitions and flowback; (2) capture and reduce emissions through use of low or no emission flowback tanks; and (3) establish and maintain communications regarding well pad activities with the community (Witter et al., 2011).

4.3. Comparisons to other risk estimates

This risk assessment is one of the first studies in the peer-reviewed literature to provide a scientific perspective to the potential health risks associated with development of unconventional natural

Table 6
Excess cancer risks for residents living >½ mile from wells and residents living ≤½ mile from wells.

Hydrocarbon	WOE		Unit Risk (μg/m^3)	Source	>½ mile		≤½ mile	
	IRIS	IARC			Cancer risk based on median concentration	Cancer risk based on 95% UCL of mean concentration	Cancer risk based on median concentration	Cancer risk based on 95% UCL of mean concentration
1,3-Butadiene	B2	1	3.00E−05	IRIS	1.30E−06	5.73E−07	1.30E−06	6.54E−07
Benzene	A	1	7.80E−06	IRIS	3.03E−06	5.40E−06	3.33E−06	8.74E−06
Ethylbenzene	NC	2B	2.50E−06	CalEPA	1.75E−07	4.26E−07	2.09E−07	3.48E−06
Styrene	NC	2B	5.00E−07	CEP	3.10E−08	2.70E−08	3.00E−08	9.30E−08
Cumulative cancer risk					5E−06	6E−06	5E−06	1E−05

Abbreviations: 95%UCL, 95% upper confidence limit; CalEPA, California Environmental Protection Agency; CEP, (Caldwell et al., 1998); IARC, International Agency for Research on Cancer; IRIS, Integrated Risk Information System; Max, maximum; NC, not calculated; WOE, weight of evidence; μg/m^3, micrograms per cubic meter. Data from CalEPA 2011; IRIS (US EPA, 2011).

gas resources. Our results for chronic non-cancer HIs and cancer risks for residents >than ½ mile from wells are similar to those reported for NGD areas in the relatively few previous risk assessments in the non-peer reviewed literature that have addressed this issue (CDPHE, 2010; Coons and Walker, 2008; CDPHE, 2007; Walther, 2011). Our risk assessment differs from these previous risk assessments in that it is the first to separately examine residential populations nearer versus further from wells and to report health impact of emissions resulting from well completions. It also adds information on exposure to air emissions from development of these resources. These data show that it is important to include air pollution in the national dialogue on unconventional NGD that, to date, has largely focused on water exposures to hydraulic fracturing chemicals.

4.4. Limitations

As with all risk assessments, scientific limitations may lead to an over- or underestimation of the actual risks. Factors that may lead to overestimation of risk include use of: 1) 95% UCL on the mean exposure concentrations; 2) maximum detected values for 1,3-butadiene, 2,2,4-trimethylpentane, and styrene because of a low number of detectable measurements; 3) default RME exposure assumptions, such as an exposure time of 24 h per day and exposure frequency of 350 days per year; and 4) upper bound cancer risk and non-cancer toxicity values for some of our major risk drivers. The benzene IUR, for example, is based on the high end of a range of maximum likelihood values and includes uncertainty factors to account for limitations in the epidemiological studies for the dose–response and exposure data (US EPA, 2011). Similiarly, the xylene chronic RfC is adjusted by a factor of 300 to account for uncertainties in extrapolating from animal studies, variability of sensitivity in humans, and extrapolating from subchronic studies (US EPA, 2011). Our use of chronic RfCs values when subchronic RfCs were not available may also have overestimated 1,3-butadiene, n-propylbenzene, and propylene subchronic HQs. None of these three chemicals, however, were primary contributors to the subchronic HI, so their overall effect on the HI is relatively small.

Several factors may have lead to an underestimation of risk in our study results. We were not able to completely characterize exposures because several criteria or hazardous air pollutants directly associated with the NGD process via emissions from wells or equipment used to develop wells, including formaldehyde, acetaldehyde, crotonaldehyde, naphthalene, particulate matter, and polycyclic aromatic hydrocarbons, were not measured. No toxicity values appropriate for quantitative risk assessment were available for assessing the risk to several alkenes and low molecular weight alkanes (particularly $<C_5$ aliphatic hydrocarbons). While at low concentrations the toxicity of alkanes and alkenes is generally considered to be minimal (Sandmeyer, 1981), the maximum concentrations of several low molecular weight alkanes measured in the well completion samples exceeded the 200–1000 μg/m^3 range of the RfCs for the three alkanes with toxicity values: n-hexane, n-pentane, and n-nonane (US EPA, 2011; ORNL, 2009). We did not consider health effects from acute (i.e., less than 1 h) exposures to peak hydrocarbon emissions because there were no appropriate measurements. Previous risk assessments have estimated an acute HQ of 6 from benzene in grab samples collected when residents noticed odors they attributed to NGD (CDPHE, 2007). We did not include ozone or other potentially relevant exposure pathways such as ingestion of water and inhalation of dust in this risk assessment because of a lack of available data. Elevated concentrations of ozone precursors (specifically, VOCs and nitrogen oxides) have been observed in Garfield County's NGD area and the 8-h average ozone concentration has periodically approached the 75 ppb National Ambient Air Quality Standard (NAAQS) (CDPHE, 2009; GCPH, 2010).

This risk assessment also was limited by the spatial and temporal scope of available monitoring data. For the estimated chronic exposure, we used 3 years of monitoring data to estimate exposures over a 30 year exposure period and a relatively small database of 24 samples collected at varying distances up to 500 ft from a well head (which also were used to estimate shorter-term non-cancer hazard index). Our estimated 20-month subchronic exposure was limited to samples collected in the summer, which may have not have captured temporal variation in well completion emissions. Our ½ mile cut point for defining the two different exposed populations in our exposure scenarios was based on complaint reports from residents living within ½ mile of existing NGD, which were the only data available. The actual distance at which residents may experience greater exposures from air emissions may be less than or greater than a ½ mile, depending on dispersion and local topography and meteorology. This lack of spatially and temporally appropriate data increases the uncertainty associated with the results.

Lastly, this risk assessment was limited in that appropriate data were not available for apportionment to specific sources within NGD (e.g. diesel emissions, the natural gas resource itself, emissions from tanks, etc.). This increases the uncertainty in the potential effectiveness of risk mitigation options.

These limitations and uncertainties in our risk assessment highlight the preliminary nature of our results. However, there is more certainty in the comparison of the risks between the populations and in the comparison of subchronic to chronic exposures because the limitations and uncertainties similarly affected the risk estimates.

4.5. Next steps

Further studies are warranted, in order to reduce the uncertainties in the health effects of exposures to NGD air emissions, to better direct efforts to prevent exposures, and thus address the limitations of this risk assessment. Next steps should include the modeling of short- and longer-term exposures as well as collection of area, residential, and personal exposure data, particularly for peak short-term emissions. Furthermore, studies should examine the toxicity of hydrocarbons, such as alkanes, including health effects of mixtures of HAPs and other air pollutants associated with NGD. Emissions from specific emission sources should be characterized and include development of dispersion profiles of HAPs. This emissions data, when coupled with information on local meteorological conditions and topography, can help provide guidance on minimum distances needed to protect occupant health in nearby homes, schools, and businesses. Studies that incorporate all relevant pathways and exposure scenarios, including occupational exposures, are needed to better understand the impacts of NGD of unconventional resources, such as tight sands and shale, on public health. Prospective medical monitoring and surveillance for potential air pollution-related health effects is needed for populations living in areas near the development of unconventional natural gas resources.

5. Conclusions

Risk assessment can be used as a tool in HIAs to identify where and when public health is most likely to be impacted and to inform risk prevention strategies directed towards efficient reduction of negative health impacts. These preliminary results indicate that health effects resulting from air emissions during development of unconventional natural gas resources are most likely to occur in residents living nearest to the well pads and warrant further study. Risk prevention efforts should be directed towards reducing air emission exposures for persons living and working near wells during well completions.

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