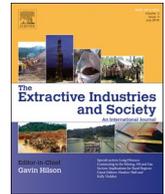




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## Natural gas pipeline compressor stations: VOC emissions and mortality rates

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## ABSTRACT

Increasing reliance on natural gas for energy has resulted in expansion of the natural gas infrastructure, including pipelines and compressor stations to transport gas. Compressor stations emit numerous particulate and gaseous pollutants including volatile organic compounds (VOCs) but studies of human health in association with compressor stations are almost completely absent from the literature. The objective of the study was to test for associations between VOC emissions from compressor stations and adjusted mortality rates. We conducted a county-level ecological study, using VOC emission data from the 2017 National Emissions Inventory, 2017 age-adjusted total mortality per 100,000 population from CDC data, and covariates from the County Health Rankings data. Results of multiple linear regression models showed that total age-adjusted mortality, controlling for covariates (race/ethnicity, education, poverty, urbanicity, smoking and obesity rates), was significantly higher in association with greater non-methane VOC emissions from compressor stations. Twelve individual VOCs were also associated with significantly higher adjusted mortality. Results provide preliminary evidence that compressor stations along natural gas pipelines are sources of pollutant exposures that may contribute to adverse human health outcomes.

## 1. Introduction

Natural gas supplies 35% of US electricity generation, more than any other single fuel source and surpassing coal in recent years (EIA 2019). At the point of power plant combustion, natural gas produces less particulate and gaseous pollutants per unit of energy generation than coal (Gaffney and Marley 2009). However, for any fuel source, consideration of total pollution emissions must include not only combustion but also extraction, processing, transportation and waste disposal. The use of natural gas for electricity generation has been increasing in recent years in the US corresponding to the growth of unconventional natural gas development (i.e., drilling by hydraulic fracturing or ‘fracking’ techniques) (Jacquet et al. 2018). Research evidence indicates that fracking generates air, water and noise pollution that may be harmful to the health of nearby populations (Balise et al., 2016; Ferrar et al. 2013; Garcia-Gonzales et al., 2019; McDermott-Levy and Garcia 2016; Saberi et al., 2014).

Compared to drilling and combustion, less information is available regarding human health effects related to the transportation of natural gas. Much of this transportation occurs via pipelines. There are 315,000 miles of interstate and intrastate natural gas pipeline in the US, and additional miles are being laid or are proposed as the natural gas infrastructure expands around the country (DOE 2015). Along pipelines,

compressor stations are placed as frequently as every 25–100 miles to keep gas pressurized and moving through the pipeline (NaturalGas.org 2013). About 1650 pipeline compressor stations exist in the US (MECF 2020). Compressor stations emit multiple pollutants including volatile and semi-volatile organic compounds, nitrogen oxides, sulfur dioxide and particulate matter (Brown et al., 2015; Russo and Carpenter 2019). Relationships between compressor station emissions and possible human health impacts have been suggested (Koehler et al. 2018; Russo and Carpenter 2017; Russo and Carpenter 2019), but research on measured associations between emissions and human health outcomes is almost completely absent (Nethery et al., 2019).

The current study examines associations between pollutant emissions from natural gas pipeline compressor stations and age-adjusted mortality rates for the US population. The study focuses on emissions of volatile organic compounds (VOCs) because they are known to occur from compressor stations, because they are known to have direct adverse human health impacts (Bolden et al., 2018; Cheng et al. 2019) and because they interact with nitrogen oxides to generate ground ozone, which is an established human health risk (EPA, 2019b). We test the hypothesis that higher compressor station VOC emissions will be associated with higher age-adjusted mortality rates, controlling for covariate risks.

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## 2. Methods

### 2.1. Study design

The study employs a cross-sectional ecological design. We measured amounts of VOC emissions from pipeline natural gas compressor stations from the 2017 National Emissions Inventory (EPA, 2019a). Age-adjusted total mortality rates for 2017 were collected from the Centers for Disease Control and Prevention (CDC) Underlying Cause of Death data (CDC, 2019). We obtained covariate data from the County Health Rankings data (CHR, 2019). Emissions only from natural gas pipeline compressor stations are included (North American Industry Classification System (NAICS) codes 4862, 48621 and 486210); emissions from natural gas extraction activities are not included. There were 3141 counties with covariate data in the County Health Rankings data; counties were deleted if they had missing data on age-adjusted mortality rate from the CDC ( $N = 78$  or 2.5%) for a final sample size of 3063.

### 2.2. Measures

The National Emissions Inventory (NEI) data included compressor station facilities that are “major sources” of VOC emissions. VOCs are classified as Hazardous Air Pollutants (HAPs). Compressor stations are included as major sources in the NEI if they meet the Clean Air Act Title V reporting requirements, meaning that they emit more than 10 tons of any single HAP or 25 tons of combined HAPs (EPA, 2020b). Quality assurance and quality control (QA/QC) procedures regarding data submission quality, and regarding sampling and analysis procedures for VOC emissions from point sources are described by the EPA (EPA, 2019c; EPA, 2020a)

VOC emissions in pounds for each of 32 chemicals released from

more than five facilities included in the study are shown in Table 1. (Methane was included in a supplementary analysis but not included in the primary VOC measures because, although it is a potent greenhouse gas, it has not been shown to have direct population health impacts when released into the outdoor ambient environment.)

We also included a measure of total VOC emissions. In the NEI this is reported as a separate emission category from individual VOC species as part of the Criteria Air Pollutant (CAP) reporting requirement. As described by the EPA (Pope et al., 2002), the total CAP quantity is used to estimate total VOC emissions when the CAP quantity is greater than the sum of the HAP-reported emissions, which was the case for 94% of facilities. Higher estimated VOCs reported as CAP versus amounts of individual VOCs reported as HAPs has been previously observed (Rao et al., 2013). In the remaining 6% of facilities, the HAP sum was greater than the CAP sum, and we used the higher HAP sum in these cases.

The primary outcome measure was county-level total age-adjusted mortality per 100,000 population from the CDC data. As a counterfactual test we also analyzed external causes of morbidity and mortality (ICD-10 codes V01-Y89 including accidents, homicide and suicide.)

For covariates we used the 2019 County Health Rankings data. These data in turn are based on data sources that covered the year 2017 or in some cases earlier years or averages over more than one prior year up to 2017. Specifically, we included as covariates measures on percent population by race/ethnicity (African American, Asian, Native American, Hispanic, and non-Hispanic White) based on 2017 Census estimates; percent with high school only education (based on the difference between the high school graduation percent for 2016–2017 minus the percent of the adult population with some college); urban county (based on 75% or more urban population in the 2010 US Census); percent children in poverty in 2017; adult smoking rate in 2016; and adult obesity rate in 2015.

**Table 1**

VOC types and emissions in pounds (except methane, which is measured in tons).

VOC	N of facilities with emissions >0	Total emissions	Mean emissions per facility (SD)	Minimum	Maximum
1,1,2,2-Tetrachloroethane	652	5041.33	7.73 (17.73)	<.001	216.80
1,1,2- Trichloroethane	342	4068.29	11.90 (20.01)	<.001	198.28
1,3-Butadiene	1267	167389.97	132.12 (240.36)	<.001	3085.03
1,3-Dichloropropene	342	3332.45	9.74 (16.44)	<.001	164.78
1,4-Dichlorobenzene	9	.03	.004 (.005)	<.001	.016
2,2,4-Trimethylpentane	700	75658.32	108.08 (211.29)	<.001	3182.79
2,4-Dinitrophenol	74	37318.05	504.30 (136.68)	.003	540.84
Acetaldehyde	1359	922773.01	679.01 (1696.85)	<.001	29,13.96
Acrolein	1341	773942.09	577.14 (1558.17)	<.001	29289.00
Benzene	1427	296481.22	207.77 (693.20)	<.001	17640.50
Carbon tetrachloride	228	1928.88	8.46 (14.60)	<.001	82.42
Chlorobenzene	418	3802.82	9.10 (16.49)	<.001	167.04
Chloroform	421	6002.03	14.26 (78.88)	<.001	1439.32
Ethyl benzene	1176	59128.27	50.28 (178.25)	<.001	3343.40
Ethyl chloride	542	181.74	0.34 (0.50)	<.001	4.04
Ethyl dibromide	231	2339.60	10.13 (17.79)	<.001	99.67
Ethylene dichloride	392	3321.12	8.47 (15.48)	<.001	158.78
Ethylene glycol	6	30.86	5.14 (10.69)	0.20	26.840
Ethylidene dichloride	389	3124.01	8.03 (14.63)	<.001	147.09
Formaldehyde	1445	6046353.64	4184.33 (11203.3)	0.10	209790.25
Hexane	1060	333714.01	314.82 (2208.15)	.002	61159.90
Hydrogen sulfide	10	17467.81	1746.78 (4092.45)	.02	13305.32
Methane (measured in tons)	465	109229.25	234.90 (455.11)	<.001	4455.50
Methyl chloride	10	400.76	40.07 (38.67)	0.01	116.85
Methylene chloride	1022	14186.98	13.88 (32.73)	<.001	553.04
Propylene dichloride	391	3545.24	9.07 (16.57)	<.001	167.81
Propylene oxide	120	1929.95	16.08 (26.24)	<.001	144.29
Styrene	428	4285.05	10.01 (19.14)	<.001	206.18
Tetrachloroethylene	104	113.14	1.09 (5.19)	<.001	52.41
Toluene	1423	312124.44	219.34 (1089.89)	<.001	34212.38
Vinyl chloride	783	3225.96	4.12 (7.18)	<.001	92.91
Xylene isomers	1377	170078.95	123.51 (625.12)	<.001	19145.60

### 2.3. Analysis

Descriptive summaries of study variables were calculated. Then, we conducted multiple linear regression analyses to examine total age-adjusted mortality rates at the county level in association with pounds of VOC emissions from compressor stations. Regression models controlled for race/ethnicity, education, poverty, urban county, smoking, and obesity rates. We measured VOC emissions in several alternative forms, including total pounds of emissions, the natural log of total emissions to adjust for right skewed emission distributions, and a dummy variable indicating whether the county had zero compressor station emissions or emissions greater than zero. Models included total VOC emissions as well as emissions of specific VOCs. We conducted a descriptive analysis at the state level to display the graphical association between VOC emissions and adjusted mortality rates.

Two counterfactual models were estimated, including one using mortality from external causes as the dependent variable, and one replacing VOC emissions with methane emissions; in both cases, associations with mortality rates were expected to be non-significant. Analyses were conducted using SAS software version 9.4.

### 3. Results

The NEI data included 1633 compressor station facilities, which were located across 45 states and 767 US counties. Among the states with at least one facility, the average facilities per state was 36.3, with the highest number in Oklahoma (279), followed by Texas (180). Emissions from 32 VOCs that occurred from more than five facilities were present in the database. Table 1 shows the amounts of these VOC releases in pounds, except for methane which is shown in tons of emissions. At the county level ( $N = 3063$ ), total emissions (excluding methane) summed to 52305308.91 pounds (mean = 17065.35, standard deviation = 67732.36, minimum = 0, maximum = 1,230,254).

A descriptive summary of mortality rate outcomes and study population covariates is provided in Table 2. The table shows averages in counties with at least one compressor station with emissions versus counties without stations. Counties with emissions on average had higher age-adjusted mortality rates, higher percentage of Hispanic populations, lower percentage of non-Hispanic White populations, higher percent urban population, and higher obesity rates. There were no significant differences on other covariates.

Regression analyses showed significant associations between VOC emissions and total adjusted mortality rates, controlling for covariates (Table 3). Significant associations were present for total emissions, log of total emissions, and for emissions dichotomized into yes or no.

Associations were not significant for any of the emission variables for external causes of mortality as a counterfactual test (results not

**Table 2**  
Descriptive summary of population characteristics ( $N = 3063$ ).

Variable	Overall Mean (SD)	Mean (SD) in counties with one or more emission sites ( $N = 767$ )	Mean (SD) in counties with no emission sites ( $N = 2296$ )	$P < *$
Age-adjusted mortality per 100,000	830.47 (165.52)	843.2 (155.0)	826.2 (168.7)	.01
Percent race/ethnicity				
African American	9.14 (14.38)	9.23 (14.25)	9.11 (14.42)	.83
Hispanic	9.44 (13.71)	11.54 (15.97)	8.74 (12.80)	.0001
Asian	1.52 (2.76)	1.56 (2.57)	1.51 (2.82)	.62
Native American/PI**	2.32 (7.22)	2.45 (6.62)	2.28 (7.41)	.59
White non-Hispanic	76.32 (19.66)	74.01 (20.30)	77.09 (19.79)	.0002
Percent high school only education	30.97 (12.77)	31.48 (12.86)	30.80 (12.74)	.20
Percent children in poverty	21.55 (9.07)	21.61 (8.82)	21.53 (9.15)	.84
Percent adult obesity	32.13 (4.57)	32.62 (4.75)	31.96 (4.49)	.001
Percent adult smokers	17.90 (3.59)	17.90 (3.48)	17.90 (3.63)	.99
Percent high urban population	25.0 (43.3)	30.12 (45.91)	23.26 (42.26)	.001

\*  $P$ -values based on unpaired two-tailed  $t$ -test, with Satterthwaite correction for unequal variances where appropriate.

\*\* Includes Native American, Alaska Native, Native Hawaiian and Pacific Islander.

**Table 3**

Association between 2017 NEI natural gas pipeline compressor station emissions and 2017 age-adjusted mortality per 100,000 controlling for covariates.\*

Variable	Regression Coefficient	Standard error	$P <$
Total VOC emissions per 1000 pounds	0.086	0.032	.008
Log of total VOC emissions	1.34	0.50	.008
VOC emissions (yes/no)	14.04	5.05	.005
Methane emissions per 1000 tons	-0.002	.005	.70

\* Covariates include percent African American, Hispanic, Asian, Native American (non-Hispanic White as referent); percent only high school education; percent children in poverty; urban county (yes/no); adult obesity rate; and adult smoking rate.

shown). Associations were also not significant for methane emissions (Table 3).

Analysis of specific VOCs showed significant associations between emission quantities and total adjusted mortality rates for 12 of the VOCs (e.g., styrene, 2,2,4-trimethylpentane, ethylene dichloride, vinyl chloride, and others.) Others not significant included toluene, xylene isomers, ethyl benzene, etc. (Table 4). Other VOCs with lower emission frequencies were not tested.

Fig. 1 shows the association between total VOC emissions and adjusted mortality rates at the state level. The bivariate correlation between these state-level measures was  $r = 0.32$  ( $p < .02$ ). States where emissions and adjusted mortality were relatively high included Oklahoma, West Virginia, Louisiana, Georgia, New Mexico and Ohio.

### 4. Discussion

Findings were supportive of the study hypothesis. Greater amounts of VOC emissions from natural gas pipeline compressor stations were associated with higher age-adjusted mortality rates controlling for covariates. Associations were significant for total emissions, the natural log of emissions, and when comparing counties with emissions to counties without. As expected, methane emissions were not significantly related to mortality rates, but rather associations were specific to other VOCs of potential exposure concern.

The specific VOC compounds that were associated with higher adjusted mortality rates were somewhat unexpected. In particular, some VOCs of known health concern from previous research were not significantly associated with mortality, including 1,3-butadiene, benzene, ethyl benzene, toluene, xylene isomers and formaldehyde. We suspect that this may be due in part to key individual VOCs reported as HAPs being differentially underestimating true emissions. In 94% of cases, the

**Table 4**  
Regression results for specific VOCs emissions in pounds released into 100 or more counties.

VOC	N of counties with emission	Coefficient	Standard error	P <
1,1,2,2-Tetrachloroethane	435	.771	.232	.001
1,1,2- Trichloroethane	290	.672	.275	.01
1,3-Butadiene	543	.007	.005	.11
1,3-Dichloropropene	290	.853	.334	.01
2,2,4-Trimethylpentane	425	.052	.017	.003
Acetaldehyde	710	.002	.002	.26
Acrolein	703	.003	.002	.12
Benzene	724	.005	.004	.18
Carbon tetrachloride	143	-.568	.451	.21
Chlorobenzene	309	.648	.307	.03
Chloroform	311	-.002	.073	.97
Ethyl benzene	655	.016	.018	.36
Ethyl chloride	310	10.06	4.72	.03
Ethylene dibromide	146	-.378	.375	.31
Ethylene dichloride	282	.918	.337	.01
Ethylidene dichloride	282	.863	.357	.02
Formaldehyde	738	.0003	.0002	.21
Hexane	548	-.001	.001	.35
Methanol	548	.005	.003	.16
Methylene chloride	527	.308	.097	.002
Propylene dichloride	281	.833	.315	.01
Propylene oxide	100	-.363	.356	.31
Styrene	313	.586	.265	.03
Toluene	721	.0003	.003	.90
Vinyl chloride	441	1.29	.403	.001
Xylene isomers	703	-.0002	.005	.75

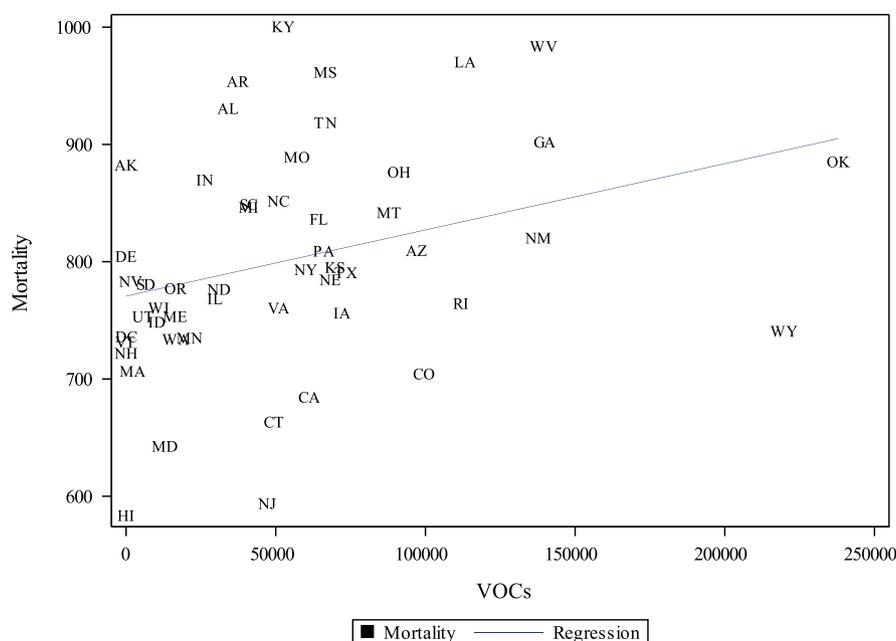
total VOC emissions measured as a Criteria Air Pollutant (CAP) was an amount greater than the total of all VOCs measured as HAPs. As described by Pope et al. (2002), CAPs are reported as part of the Title I, Section 110 Clean Air Act State Implementation Plan requirements, whereas HAPs are separately reported under Title I, Section 112 to assess whether Clean Air Act programs are successful in reducing emissions. Given the uncertainty about complete HAP reporting in the NEI, our results for individual VOC species in association with mortality should be interpreted cautiously, and results should primarily focus on the total VOC emissions variable.

Information about health harms of individual VOCs is available for

some chemicals but not for others. Some VOCs including 1,3-butadiene, acetaldehyde, benzene, ethylbenzene, formaldehyde, propylene dichloride, styrene, and vinyl chloride are classified as known or possible human carcinogens (ATSDR 2007; ATSDR 2009; ATSDR 2015; IARC 2018; NCI 2011; NCI 2018; NIH 2010; WHO 2010). These VOCs and others (e.g., acrolein, methylene chloride) may also compromise cardiovascular, respiratory, neurological, reproductive, chromosomal, or immune function (ATSDR, 2014a; ATSDR, 2014b; ATSDR, 2015; WHO, 2010). But prior information on health effects of some other VOCs associated with mortality in this study is sparse to absent. It seems that almost nothing is known, for example, on human health effects of 2,2,4-trimethylpentane exposure (EPA, 2000). Also largely unknown are effects that may result from chronic, low level ambient exposures or co-exposures to mixtures of VOCs such as those that may be occurring from compressor stations. An important future research endeavor would include formal health risk assessments of chronic exposures to VOC mixtures from compressor stations (Bari and Kindzierski, 2018; Xiong et al., 2020).

In addition to the direct adverse effects of VOC exposure, VOCs also interact with NOx in the presence of sunlight to form ground ozone. Ozone exposures can result in exacerbations of asthma, bronchitis and emphysema, and can lead to symptoms such as chest pain, coughing, throat irritation, inflammation, and reduced lung function. Children and older adults, or people with pre-existing respiratory disease, may be most vulnerable (EPA, 2019b). Observed health outcomes in the current study may be related to ozone exposures or direct VOC exposures.

Limitations of the study include the county level ecological design. The study suggests associations between compressor station emissions and mortality risk, but additional research that examines individual level exposures in relationship to health are needed. The study examined only a cross-sectional association between emissions and mortality rates over one year. There may also be time-lagged relationships such that outcomes may occur years after exposure, and to the extent that this occurs our results may be conservative. Information on the length of time that compressor stations have been in operation would be useful for more in-depth analyses of these associations. We examined overall mortality rates but non-missing county-level data were insufficient to examine finer diagnostic groups. We did not assess effects of other pollution sources (e.g., transportation, other industry) that might be correlated with the presence of compressor stations, although



**Fig. 1.** State level scatterplot of VOC emissions and adjusted mortality rate per 100,000.

we partially accounted for this by measuring the urbanicity of the county. Significant associations found, or not found, for individual VOCs may reflect underestimated VOC reporting under the HAP provisions of the Clean Air Act, but may also reflect the limitations of the cross-sectional ecological design. Significant associations may also reflect co-exposures rather than exposure to a specific VOC, as a station with high emissions of one VOC may be likely to have high emissions of others.

The study has implications for the ongoing expansion of the natural gas infrastructure, as well as for a proposed EPA rule change that would rescind all requirements for monitoring VOC emissions at pipeline compressor stations (EPA, 2019d). At the time of this writing, the EPA has proposed this change but final implementation has not yet taken place. Results of the current study suggest that rescinding this rule may be ill-advised until such time as health effects of emissions from compressor stations may be more thoroughly evaluated.

Previous research has suggested that hydraulic fracturing impacts the health of nearby populations (Bamber et al., 2019; Ferrar et al., 2013; Rasmussen et al., 2016), and the current study begins to expand the examination of health effects from drilling sites to other components of the natural gas production and distribution cycle. As the natural gas infrastructure is likely to keep expanding, it will be especially important to examine health consequences in states or other places where that expansion is concentrated, as well as places where long term exposure effects may be better investigated. States identified in Fig. 1 with high emissions and high adjusted mortality may be places to focus efforts on emissions reductions. Also important will be to examine social and political implications of expanding natural gas infrastructure for the welfare of surrounding communities (Witt et al., 2018).

Finally, in addition to health impacts from non-methane exposures related to these activities, methane emissions during extraction, processing, transportation and use are of great concern from a climate change perspective (Howarth, 2014; Howarth et al., 2011). The results of the current study, along with findings from other research, challenge the conventional wisdom that natural gas is a clean fuel that we may rely on to provide for our energy needs with little adverse effect.

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