Public Comment on NC DEQ Industrial Hog Operation Air Quality Monitoring Study: Scientific Review

Co-signed by 12 scientists in fields relating to
air quality, public health, and environmental sciences

Hog waste being sprayed into the air in North Carolina. Photo: Donn Young

Major Points:

1. There are **numerous limitations** with the study design that must be considered when drawing conclusions or generalizing results.
2. We **disagree with the report's summary findings:** that air quality monitoring of IHOs in North Carolina should cease.
3. Instead, we believe that evidence, including this report, suggests that **monitoring the air quality impacts of IHOs <u>should both continue and increase</u>** in North Carolina.

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Executive Summary

N.C. Department of Environmental Quality
Attn: Jim Bowyer
217 West Jones Street
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January 30, 2020

Dear Jim Bowyer,

We are 12 undersigned scientists in fields such as air quality monitoring, public health, and environmental sciences. We have each read the NC Department of Environmental Quality (NC DEQ) draft report on study of the air quality impacts of industrial hog operations (IHO) in Duplin County, North Carolina.

In this public comment, we outline **nine issues that limit the findings and quality of this air monitoring study.** These are grouped in three areas: the siting of monitoring locations; concentrations and thresholds; and completeness, quality, and attribution issues.

Given these limitations, **we disagree with the report's conclusion,** that the NC Department of Environmental Quality should cease monitoring the overall and specific impacts to air quality of IHOs, both in these counties and in North Carolina more generally.

Instead, in the light of the limitations, we interpret these initial study results as evidence that **air quality monitoring of the range of impacts of IHOs should both continue and increase in North Carolina.**

We consider the protection of the environmental quality of North Carolina for both its current and future residents an essential activity. This includes measuring and monitoring the impact of IHOs on air quality at ambient levels focused on NAAQS compliance in areas of moderate density, as was done in this report. However, we believe it also includes **measuring, monitoring, and action** to minimize the negative **acute, proximate, and highest-level exposures** that people experience, **acknowledging sub-compliance level impacts to quality of life and public health.** We ask that you continue and expand this study to represent and defend the environments and health of people whose experiences this study cannot represent.

Sincerely,

Sarav Arunachalam, PhD, MS
Calvin Cupini
Radhika Dhingra, PhD, MSPH, MS
Lawrence Engel, PhD, MS
Mike Dolan Fliss, PhD, MPS, MSW
Chris Heaney, PhD, MS
Brian Magi, PhD
Sarah Rhodes, PhD
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Signatories / Co-Authors

1. **Sarav Arunachalam**, PhD, MS. sarav@email.unc.edu. Deputy Director & Research Professor with UNC Institute for the Environment, Acting Director, Center for Environmental Modeling for Policy Development, Adjunct Professor, Environmental Sciences and Engineering, UNC Gillings School of Global Public Health. Dr. Arunachalam has over 25 years of experience studying air quality at local to regional scales, with a focus on developing and applying multi-scale chemistry – transport models to study nonattainment issues and developing methods for source attribution. He previously studied O3 formation in North Carolina and identified emissions controls from coal-fired power plants, that was in part, the basis of the 2002 NC Clean Smokestacks Act. Recently, Dr. Arunachalam has led the development of a series of web-based reduced-form screening tools to study near-source air pollution from traffic-related and other emissions sources for community applications focused on developing mitigation options. Dr. Arunachalam is on the Scientific Advisory Board of the Clean Air Carolina's Citizen Science program, the ICAO Committee on Aviation Environmental Protection's Impacts and Sciences Group, and Chair of the American Meteorological Society's Committee on Meteorological Aspects of Air Pollution. https://ie.unc.edu/people/arunachalam/

2. **Calvin A. Cupini**, Program Manager of Citizen Science and AirKeepers for Clean Air Carolina. Calvin works on localized pollution impacts in environmental justice areas, manages and implements the state-wide AirKeeper network of engaged citizens and low cost sensors, and advises staff in matters of public science and environmental economics. Calvin lead the community collaboration and deployment for the development of the Collocation Guide and Macro Analysis Tool for EPA's Air Sensor Toolbox for Citizen Scientists. He is a technical advisor to Earthwatch, SciStarter, The Pollution Detectives and the Science Museum of Virginia. Calvin is also a session chair for the Air Sensors International Conference.

3. **Radhika Dhingra**, PhD, MSPH, MS.rdhingra@unc.edu. Assistant Professor, Environmental Sciences and Engineering, UNC Gillings Global School of Public Health. Radhika has a doctorate in environmental epidemiology and a masters in environmental engineering. As an environmental epidemiologist she has 8 years of experience in environmental health research that includes the study of community-wide contamination from industrial sources, air pollution and vector-borne disease. Website: https://sph.unc.edu/adv_profile/radhika-dhingra-PhD/

4. **Lawrence Engel,** PhD, MS. larry.engel@unc.edu. Associate Professor, Epidemiology, UNC Gillings Global School of Public Health. Dr. Engel has over 20 years of experience in environmental and occupational health research. His current research in North Carolina focuses on the transport of environmental contaminants from point sources to nearby communities and the health impacts of these contaminants in these communities. Website: https://sph.unc.edu/adv_profile/lawrence-s-engel-phd/.

5. **Mike Dolan Fliss**, PhD, MPS, MSW. Mike.Dolan.Fliss@unc.edu. Research Scientist with NC Injury Prevention Research Center; epidemiologist & public health data scientist with NC Division of Public Health, Injury & Violence Prevention Branch. Mike has a PhD in Epidemiology, a Masters in Public Health Informatics, and a Masters in Social Work. He has been a research volunteer with the NC Environmental Justice Network focusing on CAFOs and other human experiences of pollution in North Carolina since 2015. Mike's previous studies have included supporting the Title VI complaint analysis and documenting the widespread and cumulative exposure to CAFOs in NC. Mike is one of the primary authors of this public comment. Website: http://epimike.web.unc.edu/

6. **Chris Heaney**, PhD MS. cheaney1@jhu.edu. Associate Professor, Department of Environmental Health and Engineering, Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health. Chris's research interests include environmental epidemiology, occupational and environmental health, infectious diseases, water and health, global climate change, and community-based participatory website. Recent related projects include the health impacts of industrial poultry integration and exposure to airborne swine-specific fecal pollution. Website: https://www.jhsph.edu/faculty/directory/profile/2595/christopher-d-heaney

7. **Brian Magi**, PhD. brian.magi@uncc.edu. Associate Professor, UNC Charlotte, Department of Geography and Earth Sciences. Brian has a PhD in Atmospheric Sciences. He is a member of the Mecklenburg County Air Quality Commission, and on the Board of Directors of Clean Air Carolina, and the Scientific Advisory Board of Clean Air Carolina's Citizen Science program. His expertise is related to aerosol/particle physics and chemistry, and he has been studying low-cost air monitoring as a tool for learning about local scale variability in ambient PM2.5. He also teaches courses in Atmospheric Chemistry and Global Environmental Change at UNC Charlotte. Brian is one of the primary authors of this public comment. His research group webpage is http://brianmagi.uncc.edu/

8. **Sarah Rhodes**, PhD. smrhodes@live.unc.edu. Research Affiliate in the Department of Environmental Sciences and Engineering, University of North Carolina at Chapel Hill. For the past six years, Dr. Rhodes has been conducting research in partnership with the Rural Empowerment Association for Community Help and Johns Hopkins University to examine the role of swine CAFOs in the evolution and spread of antibiotic-resistant bacteria among hogs, swine CAFO workers, community residents, and the environment (e.g. air, water, surfaces) in North Carolina. Dr. Rhodes is currently working as a consultant in environmental/occupational epidemiology in Toronto, Ontario.

9. **Ana Maria Rule**, PhD MHS. arule1@jhu.edu. Assistant Professor, Department of Environmental Health and Engineering, Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Education and Research Center for Occupational Safety and Health. Dr. Rule's background is in aerosol research and exposure assessment. Her previous projects include collaboration with FDA researchers in Wisconsin optimizing air sampling techniques to evaluate potential exposures from manure irrigation systems; she has led an effort to investigate emissions from food animal transport vehicles, and is helping characterize bacterial aerosols and antibiotic resistance related to hog and dairy operations.She is currently director of the Environmental Exposure Assessment Lab, where she develops and applies methods for the assessment of airborne exposures to adult and pediatric populations, which include biological aerosols. Website: https://www.jhsph.edu/faculty/directory/profile/1984/ana-mar-a-rule

10. **Sacoby Wilson**, PhD, MS. swilson2@umd.edu. Associate Professor with the Maryland Institute for Applied Environmental Health and Department of Epidemiology and Biostatistics, School of Public Health, University of Maryland-College Park. Dr. Wilson has over 15 years of experience as an environmental health scientist in the areas of exposure science, environmental justice, environmental health disparities, community-engaged research including crowd science and community-based participatory research (CBPR), air pollution studies, built environment, industrial animal production, climate change, and community resiliency. He works primarily in partnership with community-based organizations to study and address environmental justice and health issues and translate research to action. Dr. Wilson is Director of the Community Engagement, Environmental Justice and Health (CEEJH) Initiative. CEEJH is focused on providing technical assistance to communities fighting against environmental injustice and environmental health disparities in the DMV region and across the nation.

He is a member of the USEPA's National Environmental Justice Advisory Council (NEJAC), on the board of the Citizen Science Association, a past Chair of the APHA Environment Section, past board member of Community-Campus Partnerships for Health, and a former Chair of the Alpha Goes Green Initiative, Alpha Phi Alpha Fraternity, Inc. He is also a senior fellow in the Environmental Leadership Program. Dr. Wilson, a two-time EPA STAR fellow, EPA MAI fellow, Udall Scholar, NASA Space Scholar, and Thurgood Marshall Scholar. Website: www.ceejhlab.org.

11. **Courtney Woods**, PhD. cgwoods@email.unc.edu. Assistant Professor, Department of Environmental Sciences and Engineering. Dr. Courtney Woods has over 10 years of experience in toxicology research and community-based participatory research in partnership with rural communities and environmental justice organizations across the southeastern US. Website: https://sph.unc.edu/adv_profile/courtney-g-woods/

12. **William Vizuete**, PhD, MS. vizuete@unc.edu. Associate Professor, Environmental Sciences and Engineering, UNC Gillings Global School of Public Health. In his research Dr. Vizuete seeks novel environmental engineering solutions to solve public health problems associated with air quality. Website: http://vizuete.web.unc.edu.

Concerns about DEQ CAFO Air Monitoring Report

Siting of monitoring locations

Selection of monitoring sites for compliance with regulatory standards may not capture public health exposure and may exclude maximum/peak exposure. How representative of community exposure are these sites?

1. Monitor strategy based on ambient compliance, not public health impact

   The siting strategy DEQ implemented is in line with compliance with National Ambient Air Quality Standards (NAAQS), which is geared towards county/regional air quality assessment. In contrast, the complaints due to odors associated with hog-farm operation are a specific emission source of pollution, and **the experiences of a community or even a household are as important to consider as county/regional air quality**.

   The quality of life and public health impacts of sub-compliance exposures may be as significant to these specific communities and households as meeting regional NAAQS thresholds. **Sub-compliance experiences (and unmonitored, higher intensity experiences) still represent a lowered environmental quality for residents that may continue to prompt complaints.** Additionally, measuring air quality experiences in rural areas may require unique monitoring coverage plans when compared to more dense city environments.

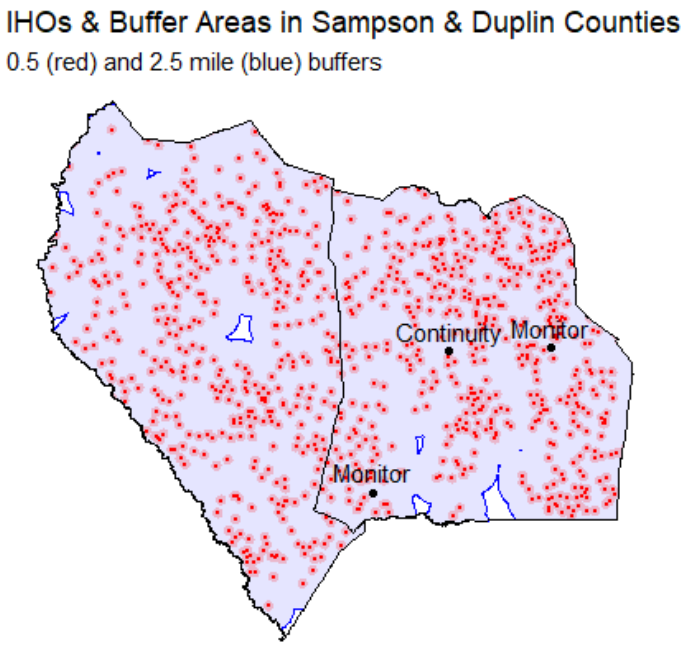
   As presented, the DCAMS Report **does not help understand community or household exposure**, so we would suggest that the DCAMS Report conclusion (that there is no significant air quality issue) is flawed because the analysis and study design themselves were aimed at understanding county/regional air quality assessment. This design strategy is different than one aimed at understanding quality of life and public health impacts. **Studies that combine air quality measurement with the people's lived experiences can help bridge this gap** - residents can (and have) identified that intense odorant chemicals and acute spikes in already high ambient exposures negatively impact activities of daily living, including being awoken from sleep at night by smells. This DCAMS report does not include experiences of individuals, households, or specific communities associated with this air monitoring data, so it is difficult to interpret the lived experience parallels of this data.

2. Study exclusions leave out tens of thousands with most proximate exposure

By following the EPA monitoring placement guidelines for ambient PM2.5, the study excluded the possibility of placement of monitoring locations within 0.5 miles of industrial hog operations (IHOs). However, given the extremely high density of IHOs in Sampson and in Duplin counties specifically (99% of each county is within 2.5 miles of an IHO), this **0.5 mile exclusion excludes over 30% (by area) of these counties (see below).**

These are not unpopulated areas; **nearly 100,000 people in NC (most of them in these two counties) live within 0.5 miles of an IHO.** By excluding these areas from study, the air quality experiences of these people are not represented by DCAMS Report conclusions. These areas, most proximate to IHOs, may also include some of the most intense air quality impacts, as they may be much closer to lagoons and spray fields in ways the monitors were not. Moreover, given that around one million North Carolina residents live within 3 miles of an IHO (from Title VI Complaint), ending this IHO-focused air quality monitoring program leaves the experiences of many North Carolinians largely unmonitored. **Given how many people are exposed to IHOs, and how many people are left out of this study, we recommend more monitoring to better understand how and when complaints arise. Less or no monitoring would undermine the lived experience of NC citizens in Duplin County**

Monitor locations, industrial hog operations (IHOs), & 0.5, 2.5 mile buffer areas



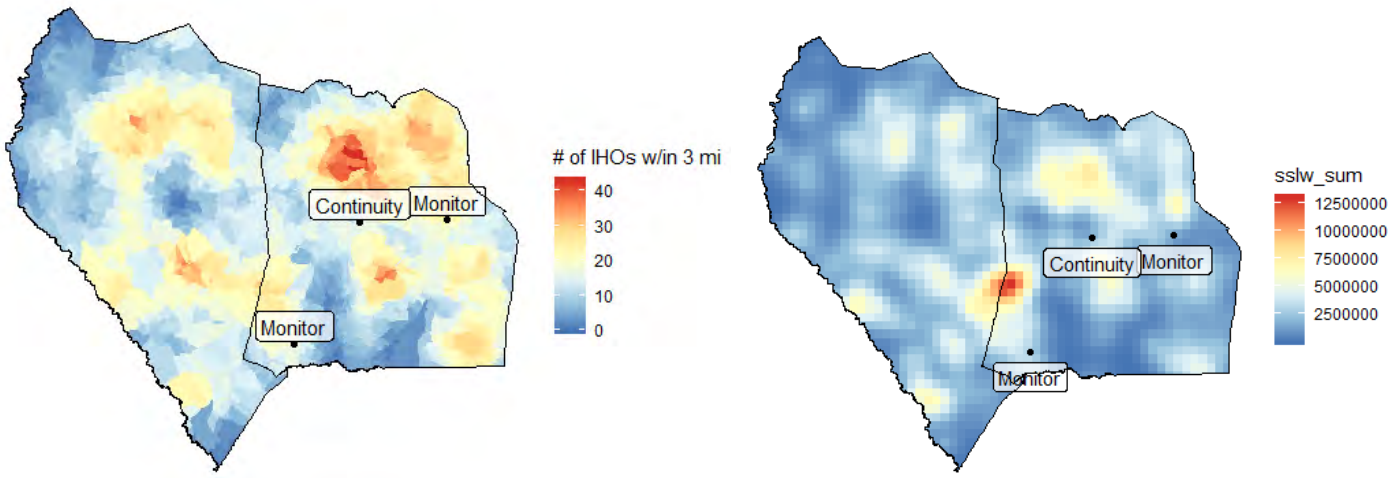
Includes 2,029 Industrial Hog Operations (IHOs) from 205 data of permitted CAFO locations as cleaned for the Title VI analysis. 0.5 mile buffers exclude 20 and 35% of Sampson and Duplin from the study respectively. 99% of each county area is within 2.5 miles of an IHO. Analysis and map by Mike Dolan Fliss, PhD, UNC Chapel Hill.

3. Monitor locations are placed in relatively low-density locations

The DEQ placement strategy, based on ambient PM2.5 measurement, precluded the study from measuring the closest, most proximate exposures to CAFOs. In addition, the monitor locations seem to suggest the **study is measuring the lower end of density of IHOs, whether by number of IHOs or proxy measures for manure density**, like steady state live weight (SSLW). This suggests the study is limited to measurement of less-proximate IHO impacts to ambient air quality in (relatively) lower IHO density areas in Sampson and Duplin Counties.

It is important to remember that the relative lower-density areas in these two counties are still some of the highest density IHO exposures in both the US and the world. Still, this study may not represent the upper range of ambient exposures given the additive impact of multiple IHO exposures in other places of the county. This is different than the highest proximate exposures (excluded from analysis) and acute exposures (likely sprayfield mechanisms) that may also be left out of the study entirely.

Monitor location vs. IHO density by count of IHOs and steady state live weight (SSLW)



Monitor locations are not placed in the highest density areas, whether by number of IHOs (top: sum of IHOs within 3 miles of a block centroid) or modeled steady state live weight (bottom: additive raster - +100% of SSLW at 0 miles, +0% at 4 miles or farther, bisquare in between). Analysis and map by Mike Dolan Fliss, PhD, UNC Chapel Hill.

4. Limited study does not represent range of people's lived experience

This study, including limiting monitoring in one county, is not representative of the range impacts Industrial Hog Operations (IHOs) have on air quality and people's lives in North Carolina. Residents experience the air quality impacts of IHO pollution in different ways.

To break down this range of people's lived experiences, we might consider a simplified table of exposures. (1) **IHO density** (by headcount, steady state live weight, estimated manure load, etc.) may be higher or lower. (2) Relevant **concurrent exposures** with negative impacts to PM2.5 and odorant chemical by co-located facilities (e.g. poultry feed operations locations, landfills) may likewise be high or low, partly determined by the high or low density of those co-located facilities. (3) The **exposure type**, related to the **mechanism**, includes at least (a) ambient lagoons as their source and (b) higher intensity proximate lagoon or (c) acute spray exposures.

Given limited monitoring locations, the comparison of monitor locations and high density CAFO exposures (above), the study design exclusions (0.5 miles), and the lack of sprayfield data or spikes in the monitors, we estimate the DEQ study includes an attempt to model ambient lagoon air quality impacts with lower IHO density and (assumed, little evidence in report) lower concurrent exposures. **Therefore, it is our estimation that this DEQ study could not capture the range of these air quality impacts or quantify the experiences of people in these diversity of settings**. However, it is our understanding that this diversity of human experiences (at least!) matters for communities living proximate to IHOs. Should DEQ continue its monitoring program, it should aim to provide both evidence for and ongoing surveillance of changes to at least these air quality impact combinations.

DEQ study: coverage of IHO-related air quality impact experiences

			IHO Density (Head, SSLW)			
			Lower		Higher	
			Concurrent Exposures			
			Low	High	Low	High
Exposure Type & Mechanism	Ambient	Lagoon	DEQ Study	NA	NA	NA
	Acute / Proximate	Lagoon	NA	NA	NA	NA
		Spray	NA	NA	NA	NA

Table by Mike Dolan Fliss, PhD, UNC Chapel Hill.

Concentrations and thresholds

5. Time-series graphs need improvement to accurately communicate results

We suggest that the statistical summary of the valid data points be presented more clearly and completely to better convey the full scope of the data collected for PM2.5. Figure 5 and Figure 6 in the DEQ DCAMS Report summarize the hourly and daily PM2.5 at the various sites with all data points from all sites overlaid as a series of individual hourly and daily average PM2.5 for the full time span of the longest sampling (June 2018 to October 2019). Certainly the graphs contain all the information, but the presentation of the data could be much clearer. We suggest the following:

I. Separately plot the time series of data from each site using mathematical smoothing to generally guide the eye.
II. On the graph, include the average PM2.5 concentration from each site, the fraction of hours or days when PM2.5 > 12 ug/m3 (Annual NAAQS) and the fraction of hours or days when PM2.5 > 35 ug/m3 (24 hour NAAQS). **This additional information would better quantify instances when PM2.5 concentrations were high** (relative to 24 hour NAAQS) and quantify how high the general PM2.5 concentrations were (relative to the annual NAAQS).
III. Figure 5 and 6 should both denote the annual PM2.5 NAAQS and 24 hour NAAQS thresholds. Both are relevant in the conversation of acute exposure (something more along the lines of the 24 hour NAAQS) and prolonged exposure (annual NAAQS). We also would think both are relevant in and amongst CAFOs in Duplin county that operate with spraying schedules (leading to acute exposure) and operate all year round to generate prolonged exposure.

To prompt this discussion, we plotted the PM2.5 data from Sarecta and Williamsdale available at the DEQ Special Studies website (https://deq.nc.gov/about/divisions/air-quality/air-quality-data/special-studies/duplin-county) that was marked with the QA flag of “Ok” and that was greater than 0 ug/m3 since negative mass concentration is physically meaningless. We further partitioned the filtered PM2.5 data into day (05:00-20:00) and nighttime (21:00-04:00) values and calculated various bulk statistics for Sarecta and Williamsdale.

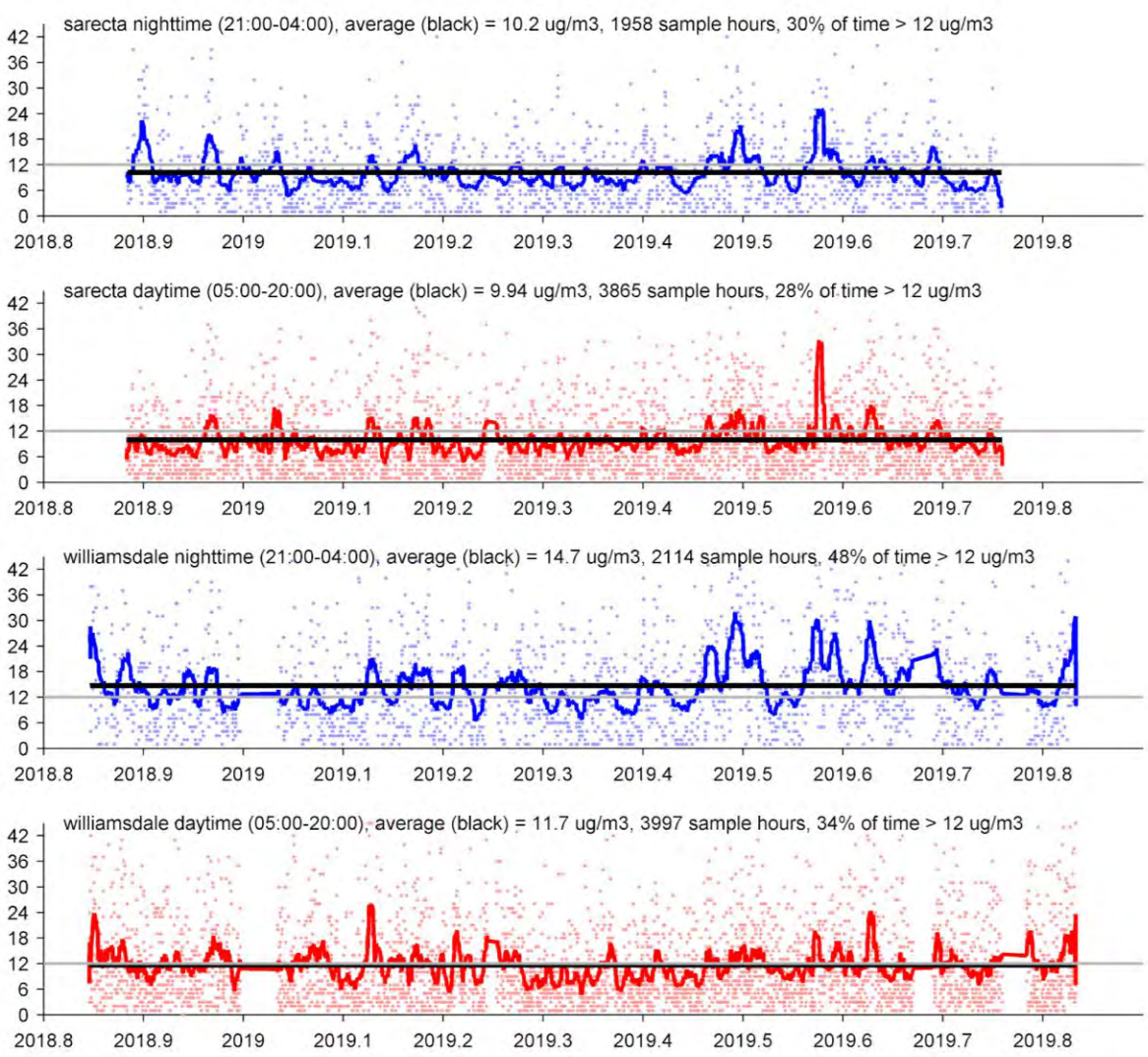
Sarecta day and night time averages were similar at 9.9 and 10.2 ug/m3, with 3865 and 1958 sample hours, respectively. Both day and night had values of PM2.5 > 12 ug/m3 about 30% of the time. Sarecta had 55 hours of PM2.5 > 35 ug/m3, or about 1% of the 5823 sample hours.

Williamsdale day and night time averages were noticeably different at 11.7 and 14.7 ug/m3, with 3997 and 2114 sample hours, respectively. Day and night had values of PM2.5 > 12 ug/m3 about 34% and 48% of the time, also consistent with the differences in the average values. Finally, Williamsdale had 226 hours of PM2.5 > 35 ug/m3, or about 4% of the 6111 sample hours.

If extrapolated over multiple years, **Williamsdale site data leans towards a violation of annual PM2.5 NAAQS, and also has about 4x more hours than Sarecta with PM2.5 > 35 ug/m3**. This, we suggest, should warrant further study using the E-BAMS deployed for additional years. If the findings continue to show that PM2.5 annual NAAQS is violated, then a downstream result could be an additional regulatory monitoring site for PM2.5 being located in Duplin County.

The caveat is that we have not necessarily explored all the dimensions of the data, and our results may change with different QA filtering or by considering the influence of wind speed and direction. However, even with that caveat, **the conclusion that there is no air quality problem (for PM2.5) seems to be the result of an analysis that ignores or minimizes the acute exposures that communities experience** – the lived experience of feeling the effect of the high PM2.5 for 4% of the time. For scale, if the Williamsdale and Sarecta analysis we provide holds up, 4% of a given year is about 15 days in any given year with high (>35 ug/m3) PM2.5 in Williamsdale area, and about 4 days in any given year for Sarecta area.

PM2.5 monitoring results in Sarecta and Williamsdale (daytime and nighttime)



Re-analysis & graph by Brian Magi, Associate Professor of Atmospheric Sciences, UNC Charlotte, Department of Geography and Earth Sciences. The y-axis is PM2.5 in units of ug/m3, the individual dots are the hourly PM2.5, and the thick red and blue lines represent a multi-hour mathematically smoothed average to guide the eye. Data greater than about 42 ug/m3 are not shown on the graphs, but represent a relatively tiny fraction of total sample hours.Specific data being plotted is available from DEQ DCAMS website with our filtering choices described in our text above (QA flag = Ok, and hourly PM2.5 > 0 ug/m3).

6. Annual PM2.5 average exceeds NAAQS

In DCAMS, assessment of PM2.5 was strictly limited to the 24 hour NAAQS value of 35 ug/m3. We would suggest that persistent emissions from year-round CAFO emissions make the DCAMS study area one where the annual PM2.5 NAAQS is also quite relevant. See the description of Point 5, but we argue that it is reasonable to surmise that **the annual PM2.5 NAAQS is in danger of violation near Williamsdale** (if we extrapolate out to three years assuming that the single sample year is "representative").

The US EPA acknowledges these annual PM2.5 measures, quantifying potential chronic exposures, have both a primary standard to protect sensitive populations and a secondary standard to protect the general public. If the current monitoring results (implying already high annual PM2.5 at monitoring locations) underestimates the annual ambient exposures of some groups in the highest density or most proximate areas, **there may be tens of thousands in these counties experiencing PM2.5 concentrations that exceeds the annual NAAQS health-based standard for PM2.5.** This is not grounds for ending monitoring, but the opposite: monitoring should be increasing, particularly to determine **whether some populations not well-captured by the DCAMS study are breathing air with PM2.5 concentration that exceeds annual NAAQS PM2.5 standard**. Those studies should also examine whether with most proximate exposures are receiving acute spray field exposure greater than the 24 hour levels, as well as non-violation-level impacts to quality of life impacts and non-criteria air pollutants not included on this list.

Pollutant		Primary/ Secondary	Averaging Time	Level	Form
Carbon Monoxide (CO)		primary	8 hours	9 ppm	Not to be exceeded more than once per year
			1 hour	35 ppm	
Lead (Pb)		primary and secondary	Rolling 3 month average	0.15 µg/m3 (1)	Not to be exceeded
Nitrogen Dioxide (NO2)		primary	1 hour	100 ppb	98th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		primary and secondary	1 year	53 ppb (2)	Annual Mean
Ozone (O3)		primary and secondary	8 hours	0.070 ppm (3)	Annual fourth-highest daily maximum 8-hour
Particle Pollution (PM)	PM2.5	primary	1 year	12.0 µg/m3	annual mean, averaged over 3 years
		secondary	1 year	15.0 µg/m3	annual mean, averaged over 3 years
		primary and secondary	24 hours	35 µg/m3	98th percentile, averaged over 3 years
	PM10	primary and secondary	24 hours	150 µg/m3	Not to be exceeded more than once per year on
Sulfur Dioxide (SO2)		primary	1 hour	75 ppb (4)	99th percentile of 1-hour daily maximum concentrations, averaged over 3 years
		secondary	3 hours	0.5 ppm	Not to be exceeded more than once per year

Table taken from US EPA website. See https://www.epa.gov/criteria-air-pollutants/naaqs-table for footnotes.

Completeness, Quality, and Attribution Issues

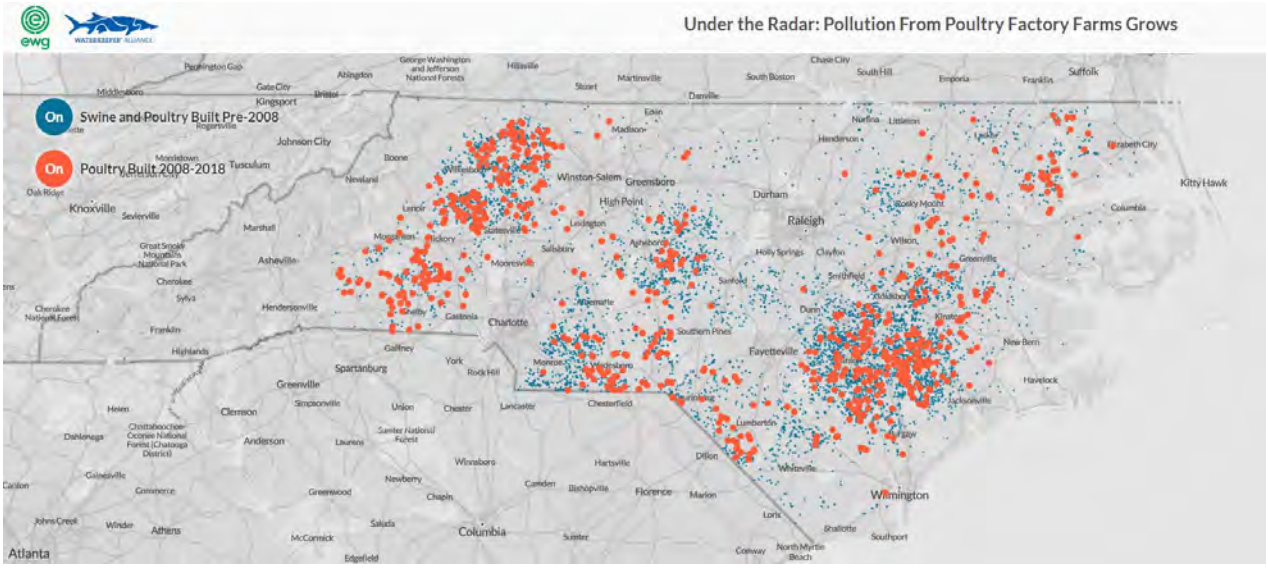
7. Study does not include cumulative impact, e.g. unpermitted poultry CAFOs

The ambient air quality of North Carolina is impacted by more than industrial hog operations (IHOs). If the monitoring locations were not located to represent the cumulative impact of both IHOs and permitted and non-permitted releases, then the monitoring plan may not represent the ambient levels experienced elsewhere.

Poultry concentrated animal feed operations (CAFOs) are of particular concern, since (1) they are currently unpermitted in North Carolina, yet (2) have documented air quality impacts that would be expected to add to the air quality impact of IHOs and (3) according to community data, are often co-located with IHOs, especially in the county under study.

These estimated **nearly 5,000 unpermitted poultry operations in 2019, up from an estimated 4,000 operations ten years ago, add to the air quality impacts of permitted IHOs**. Future air quality monitoring studies should incorporate the expected impact of nearby poultry operations and other sources of air quality pollution. Without this, it's difficult to attribute what impacts air quality their study, designed for IHOs, is actually capturing and how representative the monitoring locations are.

Growth of poultry operations from pre 2008 to 2018



Data collected by the Environmental Working Group. Presented on web page of Waterkeeper Alliance.
https://www.ewg.org/research/under-radar

8. *No sprayfield activity data*

Spray fields are fundamental to understanding acute exposure to reduced air quality due to industrial animal hog operations, but, given the ambient focus, are **not included in the study analysis plan or documented to have been monitored**. Given the exclusion area of 0.5 miles within IHOs, it's possible the monitoring plan may have captured few or no proximate spray events. Without documenting actual spray activity near the monitors, farms may be able to to shield the monitors from high acute peak readings.

DEQ likely knows this analysis challenge exists (since it was notified of the practice of selective spray field applications to reduce monitoring in Title VI documentation); without accommodating for this lack of data by collecting other data on documented and actual spray field practices, it is possible this monitoring effort captured no spray field activity. **The report does not mention spray fields**, offering the only direct attribution of a peak to a transient smoke plume. If no measured contaminant peaks were due to hog waste, then monitoring sites may be mis-located to capture nearby spray events and under-measuring the impact on the air quality.

Hog waste being sprayed into the air. Photo: Donn Young

9. Numerous small questions remain about missingness and attribution

We have outstanding questions that we consider more minor, but worth mentioning.

The report lists that the **monitor in Candor** didn't pass expected **data completeness** checks. Why? Could this have been anticipated and fixed in future studies?

Many 24-hour average periods are excluded due to **missing data**. In some cases during (H2S peaks) there seemed to be a significant amount of missingness. Why is this, and how can it be fixed in future studies?

Though these issues are not as substantial as the previous 8 listed, we suggest the final report add more to contextualize these questions and how to avoid these issues in the future.

A. Appendix: Useful References & Links

DEQ Release on the open comment period
https://deq.nc.gov/news/press-releases/2019/12/16/release-comment-period-open-draft-air-monitoring-study-report

Title VI Complaint
https://earthjustice.org/sites/default/files/files/North-Carolina-EJ-Network-et-al-Complaint-under-Title-VI.pdf

Title VI Analysis & Findings
https://www.ncpolicywatch.com/wp-content/uploads/2014/09/UNC-Report.pdf

EPA Letter of Concern
https://www.epa.gov/sites/production/files/2018-05/documents/letter_of_concern_to_william_g_ross_nc_deq_re_admin_complaint_11r-14-r4_.pdf

Attachment A

Attachment A1

Malodor as a Trigger of Stress and Negative Mood in Neighbors of Industrial Hog Operations

Rachel Avery Horton, PhD, Steve Wing, PhD, Stephen W. Marshall, PhD, and Kimberly A. Brownley, PhD

Objectives. We evaluated malodor and air pollutants near industrial hog operations as environmental stressors and negative mood triggers.

Methods. We collected data from 101 nonsmoking adults in 16 neighborhoods within 1.5 miles of at least 1 industrial hog operation in eastern North Carolina. Participants rated malodor intensity, stress, and mood for 2 weeks while air pollutants were monitored.

Results. Reported malodor was associated with stress and 4 mood states; odds ratios (ORs) for a 1-unit change on the 0-to-8 odor scale ranged from 1.31 (95% confidence interval [CI]=1.16, 1.50) to 1.81 (95% CI=1.63, 2.00). ORs for stress and feeling nervous or anxious were 1.18 (95% CI=1.08, 1.30) and 1.12 (95% CI=1.03, 1.22), respectively, for a 1 ppb change in hydrogen sulfide and 1.06 (95% CI=1.00, 1.11) and 1.10 (95% CI=1.03, 1.17), respectively, for a 1 $\mu g/m^3$ change in semivolatile particulate matter less than 10 μm in aerodynamic diameter (PM_{10}).

Conclusions. Hog odor, hydrogen sulfide, and semivolatile PM_{10} are related to stress and negative mood in disproportionately low-income communities near industrial hog operations in eastern North Carolina. Malodor should be considered in studies of health impacts of environmental injustice. (*Am J Public Health.* 2009;99:S610–S615. doi:10.2105/AJPH.2008.148924)

Odor, noise, heat, and crowding are environmental stressors[1] that may affect physical and mental health. Malodor is reported in neighborhoods near hazardous waste facilities, petroleum refineries, certain industrial facilities, and confined animal feeding operations; people in these areas may report sensations of irritation, respiratory problems and other physical health symptoms, interference with activities of daily living, and concerns about chronic diseases and property values.[1–37] Because polluting facilities are disproportionately located in low-income communities and communities of color,[38,39] malodor is an important aspect of environmental injustice that threatens physical, mental, and social well-being.[40]

Several studies have evaluated relationships among malodor, negative mood, and reduced quality of life in neighbors of industrial hog operations. Schiffman et al.[26] found that a small sample of neighbors of industrial hog operations reported more tension, depression, anger, fatigue, and confusion, and less vigor, compared with an unexposed rural sample. Bullers[4] found higher mean scores on a short form of the Center for Epidemiologic Studies Depression Scale (CES-D) in neighbors of industrial hog operations than in control participants (2.24 vs 1.84). Wing and Wolf[36] assessed effects on quality of life, determined by asking how often neighbors of hog operations could open windows or go outside during nice weather. By that metric, neighbors reported greatly reduced quality of life relative to other demographically comparable rural residents.

The Community Health Effects of Industrial Hog Operations (CHEIHO) study was a collaborative community-based participatory research project conducted in the predominantly low-income African American communities of rural eastern North Carolina where industrial hog operations are disproportionately located.[35] The purpose of this study was to evaluate longitudinal relationships among malodor, airborne emissions, stress, and negative mood. We hypothesized that malodor from industrial hog operations is an environmental stressor that may also negatively affect mood.

METHODS

We have previously described the CHEIHO study, including details of its community-based design and its links to education and organizing for environmental justice.[41] Research on health effects in neighbors of industrial hog operations is community-based at its origin. Community-based organizations brought the issue to the attention of researchers at the School of Public Health at the University of North Carolina and have continued as partners in all research that has been conducted. In the CHEIHO study, members of community-based organizations participated as advisors in the study design and design of study instruments. They were integrally involved in the recruitment and training of study participants. Indeed, community organizers were essential to the recruitment and retention of study participants in predominantly African American communities with historic distrust of researchers and research institutions.[42]

Study Participants

Eligible participants in the CHEIHO study were nonsmoking adults who lived within 1.5 miles of at least 1 industrial hog operation and were willing to collect data twice daily for approximately 2 weeks. Between September 2003 and September 2005, participants collected data on odor, stress, mood, physical health symptoms, blood pressure, immune function, and lung function; outcomes analyzed in this study are described in more detail in the paragraphs that follow.

At a central location in each neighborhood, research staff set up a monitoring trailer to collect data on hydrogen sulfide (H_2S; MDA Scientific Single Point Monitor, Honeywell Analytics Inc North America, Lincolnshire, IL), particulate matter less than 10 μm in aerodynamic diameter (PM_{10}) and semivolatile PM_{10} (Tapered Element Oscillating Microbalance Series 1400a Ambient Particulate Monitor with a Series 8500 Filter Dynamics Measurement

System, Thermo Fisher Scientific, Waltham, MA), and weather (Vantage Pro Weather Station, Davis Instruments, Hayward, CA, and Young Model 05103VM-42 Wind Monitor, R.M. Young Company, Traverse City, MI).

Selection of the particular pollutants to be monitored was based on previous work that has documented emissions of both H_2S (a product of the anaerobic decomposition of hog waste) and particulate matter from feed, dried feces, skin cells, hair, and bioaerosols, at confinement buildings and waste lagoons.[6,43] Furthermore, we found that H_2S and PM_{10} were related to reported malodor in the CHEIHO study; H_2S was associated with reported malodor in models that adjusted for the study's longitudinal design, as was PM_{10} during times when wind speed was greater than 6.75 miles per hour.[44]

The average distance from the monitoring platform to the nearest industrial hog operation in each neighborhood was 0.51 miles; the minimum distance to the nearest industrial hog operation was 0.20 miles and the maximum distance to the nearest industrial hog operation was 1.42 miles. In 2 of the 16 neighborhoods, the platform was located within 2 miles of 1 industrial hog operation; in the other 14 neighborhoods, however, the platform was located within 2 miles of at least 3 industrial hog operations (maximum of 16 industrial hog operations). We therefore calculated, for each neighborhood, the average distance between the platform and the industrial hog operations within 2 miles of the monitoring platform. The average distance across all neighborhoods was 1.10 miles, with a range by neighborhood from 0.56 miles to 1.50 miles. In contrast, the average distance between participant households and the monitoring platform across 15 of the 16 neighborhoods was 0.20 miles, with a range by neighborhood from 0.03 miles to 0.36 miles.

In 1 neighborhood, the average distance between participant households and the platform was 0.95 miles. In this and 3 other neighborhoods where participant homes were more geographically dispersed, we deployed additional H_2S monitors at homes farthest from the platform. All of the data on particulate matter, however, were collected at the platform and assigned to all participants in the neighborhood.

Participants attended a 3-hour training session during which they learned to complete the required data collection activities. They selected a morning time and an evening time at which they would collect data (for example, 6:00 AM and 6:00 PM). In addition, participants completed an assessment of coping style using the John Henryism Active Coping scale[45,46] and an assessment of threshold odor sensitivity using butanol standards.[47]

At the preselected, twice-daily times, participants spent 10 minutes outdoors at home and then returned indoors to rate any odor present during that 10-minute period on a 9-point scale ranging from 0 (no odor) to 8 (very strong odor). Hourly average H_2S, PM_{10}, and semivolatile PM_{10} values were calculated for the hour immediately preceding the odor rating. Following the odor rating, they responded to 5 mood state questions: "How do you feel now: (a) stressed or annoyed?, (b) nervous or anxious?, (c) gloomy, blue, or unhappy?, (d) angry, grouchy, or bad-tempered?, (e) confused or unable to concentrate?" They rated these mood questions on a 9-point scale ranging from 0 (not at all) to 8 (extremely). The "stressed or annoyed?" question was an ad-hoc single-item measure,[48,49] and the remaining 4 questions came from the Profile of Mood States instrument,[26,50] specifically, from the Tension–Anxiety, Depression–Dejection, Anger–Hostility, and Confusion–Bewilderment subscales. (The Fatigue–Inertia and Vigor–Activity subscales were not used.)

Statistical Analyses

We used logistic mixed models to evaluate malodor, H_2S, PM_{10}, and semivolatile PM_{10} as predictors of reported stress and negative mood (NLMIXED procedure in SAS version 9.1.3, Cary, NC). We used 2-level (within person and between person) mixed models to take into account the correlated structure of longitudinal data for individuals. The stress and mood variables were recoded as binary; for stressed or annoyed and nervous or anxious, 0 and 1 on the original scale were coded as 0 and 2 to 8 on the original scale were coded as 1. For the remaining 3 mood variables, 0 on the original scale was also coded as 0 and 1 to 8 on the original scale were coded as 1. These coding decisions were based on the distribution of the data such that approximately 90% of the records for each outcome variable were coded as 0 and approximately 10% were coded as 1. We included all predictor variables as linear terms. We conducted all analyses with records for which the ratings of malodor, stress, and mood, and the airborne emissions data, were not missing.

Random intercepts were included in the mixed logistic models to capture the variation between participants in baseline (average) levels of stress and negative mood. Models included the following time-dependent covariates: time of day (morning vs evening), study day (1 to ≥14), and study week (first vs second). For analyses of odor as a predictor of stress and mood, the models also included whether participants reported a cold, flu, or stomach virus at any time during data collection (yes or no). We hypothesized that illness could affect a participant's ability to smell or perception of odor and negative mood. We did not consider time-independent confounders, such as age or gender, because their relationship with exposure and outcome did not vary over time. A sample logistic mixed model follows.

Level 1 (time, within person):

$$(1)\ \text{Logit}\left(\Pr[\text{Stress}_{ij}=1]\right)=b_{0j}+b_{1j}(\text{odor})+b_{2j}(\text{time of day}),$$

where $\Pr[\text{Stress}_{ij}=1]$ is the probability that stress reported by person j at timepoint i equaled 1, b_{0j} is the person-specific intercept, b_{1j} is the effect of the time-dependent odor rating, and b_{2j} is the effect of time of day (morning vs evening).

Level 2 (between person):

$$(2)\ b_{0j}=\gamma_{00}+\gamma_{01}(person_j)+\mu_{0j};\ \mu_{0j}\sim N(0,\tau_{00}),$$

where b_{0j} is the person-specific intercept, γ_{00} is the mean of the person-specific intercepts (i.e., fixed intercept), $\gamma_{01}(\text{person}_j)$ is the contribution to the overall mean from person j, and μ_{0j} is the residual between-person variation in the intercept.

We also evaluated several potential modifiers. For analyses of H_2S as a predictor of stress and negative mood, we considered modification by wind speed (low [≤0.57 mph], medium [0.58 mph–6.75 mph], and high [>6.75 mph]) because of previous work that suggested modification of the relationship between H_2S and reported malodor by wind speed.[44] Based on previous research,[3,29,30,37]

we considered age, dichotomized at the median (≤53.7 years vs >53.7 years), and coping style, dichotomized at the median, (John Henryism Active Coping scale score <52 vs ≥52)[46,47] as potential modifiers of any association between reported odor and stress. We also considered threshold odor sensitivity (low or moderate [<320 ppm] vs high [≥320 ppm]) as a potential modifier of the relationships between odor, stress, and mood to evaluate whether more-sensitive individuals responded differently than less-sensitive ones.

RESULTS

There were 2895 records from 101 individuals in 16 neighborhoods. Complete data on reported odor, stress, and mood were available for 2666 records. Of the 2666 records with complete odor, stress, and mood data from study participants, 78 records were missing data on H_2S and 741 records were missing data on PM_{10} because of monitoring equipment malfunction.

Demographics

Table 1 presents demographic information about study participants. The median age was 53.7 years; age ranged from 19.2 years to 89.5 years. Approximately two thirds of the participants were female, and approximately 85% were African American. Seventy-five percent of participants reported that they grew up around livestock. Six neighborhoods were within 2 miles of 1 to 4 industrial hog operations, 4 were within 2 miles of 5 to 9 industrial hog operations, and 6 were within 2 miles of 10 or more industrial hog operations. Average H_2S values in the 16 neighborhoods ranged from less than 0.01 ppb to 1.5 ppb, and the highest measured H_2S values ranged from 2 ppb to 90 ppb. Average PM_{10} values ranged from 10.8 μg per cubic meter (μg/m^3) to 28.7 μg/m^3, and average semivolatile PM_{10} values ranged from –3.2 μg/m^3 (negative values occurred because of measurement imprecision at very low concentrations) to 9.2 μg/m^3.[44]

TABLE 1—Participant Characteristics: Community Health Effects of Industrial Hog Operations Study, Eastern North Carolina, 2003–2005

	No. of Records	No. of Participants
Age		
>53.7 y	1377	50
≤53.7 y	1289	51
Gender		
Female	1737	66
Male	929	35
Race		
Black	2167	85
Non-Black[a]	499	16
Grew up around livestock		
Yes	1998	76
No	591	22
Missing	77	3
Total	2666	101

[a]Fifteen White participants and 1 Latino participant.

The distribution of twice-daily odor ratings during the preselected 10-minute exposure times is presented in Table 2. Of the 2666 odor ratings recorded after participants spent 10 minutes outdoors, approximately 50% equaled zero. An additional 30% were low (a rating of 1 or 2) on the 9-point scale. Approximately 20% were 3 or higher, and 1% of the data were in each of the 2 highest categories. Most of the ratings of stress and mood state also equaled zero. For "stressed or annoyed," 81% of reports were zero; 87% were zero for "nervous or anxious," 88% for "gloomy, blue, or unhappy," 93% for "angry, grouchy, or bad-tempered," and 95% for "confused or unable to concentrate" (Table 2).

Mixed Models

Table 3 presents parameter estimates, standard errors, *t* values, odds ratios (ORs), and 95% confidence intervals (CIs) for H_2S, PM_{10}, semivolatile PM_{10}, and reported malodor as predictors of binary stress and negative mood. Variables considered as time-dependent confounders produced little change in the magnitude of the parameter estimates for the independent variables. However, we adjusted all models for time of day (morning vs evening) because time is an important predictor of odor. Reporting stress or annoyance was strongly associated with increasing levels of H_2S; the OR for a 1 ppb change in H_2S was 1.18 (95%CI=1.08, 1.30). Hydrogen sulfide was also strongly associated with feeling nervous or anxious (OR=1.12; 95% CI=1.03, 1.22). Hydrogen sulfide did not appear to be associated with the other 3 mood state variables, and wind speed did not modify any of the relationships between H_2S and stress or mood.

We found that PM_{10} did not appear to be associated with stress or negative mood, with the exception of a marginal association with feeling confused or unable to concentrate (Table 3). Semivolatile PM_{10} was most strongly associated with feeling stressed or annoyed and nervous or anxious. Associated ORs for a 1 μg/m^3 increase in semivolatile PM_{10} were small (1.06 and 1.10, respectively), though ORs associated with a 10 μg/m^3 increase, for example, were 1.73 and 2.59, respectively. Semivolatile PM_{10} appeared to be only marginally associated with feeling gloomy, angry, or confused or unable to concentrate.

Table 3 also presents parameter estimates, standard errors, *t* values, ORs, and 95% CIs for reported malodor as a predictor of binary stress and negative mood. All parameter estimates were large relative to their standard errors. The ratio of the odds of reporting stress for a 1-unit increase in reported odor on a 0-to-8 scale was 1.81 (95% CI=1.63, 2.00). Consequently, a 4-unit change on the odor scale (from odor=0 to odor=4, for example) yielded an OR of 10.6. Odds ratios for feeling nervous, gloomy, angry, and unable to concentrate, associated with a 1-unit change in odor, were 1.60 (95% CI=1.41, 1.81); 1.43 (95% CI=1.25, 1.63); 1.52 (95% CI=1.37, 1.70) and 1.31 (95% CI=1.16, 1.50), respectively.

Coping, but not age, appeared to modify the relationship between reported odor and stress. The parameter estimate for participants who scored below the median on the John Henryism Active Coping scale was 0.45 (standard error [SE]=0.07), whereas the parameter estimate for participants who scored above the median was 0.73 (SE=0.08). Threshold odor sensitivity did not appear to modify the associations between reported malodor and stress or negative mood.

DISCUSSION

We used a longitudinal design to evaluate relationships between malodor from industrial hog operations, H_2S, PM_{10}, semivolatile PM_{10}, and the stress and negative mood reported by neighboring residents. We found that ratings of

TABLE 2—Number and Percentage of Records and Number of Participants in Each Category of the Odor, Stress, and Mood Variable Ratings: Community Health Effects of Industrial Hog Operations Study, Eastern North Carolina, 2003–2005

	Twice-Daily Odor Rating		Stressed or Annoyed		Nervous or Anxious		Gloomy, Blue, or Unhappy		Angry, Grouchy, or Bad-Tempered		Confused or Unable to Concentrate	
Rating	No. of Records (%)	No. of Participants	No. of Records (%)	No. of Participants	No. of Records (%)	No. of Participants	No. of Records (%)	No. of Participants	No. of Records (%)	No. of Participants	No. of Records (%)	No. of Participants
0	1374 (51.5)	88	2162 (81.1)	98	2314 (86.8)	100	2337 (87.7)	98	2479 (93.0)	99	2529 (94.9)	100
1	472 (17.7)	82	290 (10.9)	60	217 (8.1)	40	198 (7.4)	44	109 (4.1)	40	96 (3.6)	24
2	273 (10.2)	72	95 (3.6)	39	80 (3.0)	24	42 (1.6)	20	22 (0.8)	11	20 (0.8)	9
3	196 (7.4)	68	50 (1.9)	20	34 (1.3)	12	45 (1.7)	13	10 (0.4)	7	10 (0.4)	4
4	123 (4.6)	47	14 (0.5)	10	10 (0.4)	3	12 (0.5)	6	6 (0.2)	5	7 (0.3)	2
5	73 (2.7)	39	22 (0.8)	13	8 (0.3)	6	13 (0.5)	6	17 (0.6)	9	3 (0.1)	3
6	108 (4.1)	40	19 (0.7)	10	1 (<0.1)	1	8 (0.3)	4	10 (0.4)	4	1 (<0.1)	1
7	22 (0.8)	12	6 (0.2)	4	1 (<0.1)	1	6 (0.2)	3	5 (0.2)	3	0 (0.0)	0
8	25 (0.9)	12	8 (0.3)	6	1 (<0.1)	1	5 (0.2)	3	8 (0.3)	3	0 (0.0)	0
Total	2666 (100.0)	101	2666 (100.0)	101	2666 (100.0)	101	2666 (100.0)	101	2666 (100.0)	101	2666 (100.0)	101

feeling stressed or annoyed, nervous or anxious, gloomy or unhappy, angry or grouchy, and confused or unable to concentrate increased with ratings of malodor. Of the 5 outcome variables, odor was most strongly related to feeling stressed or annoyed. Active coping appeared to modify the relationship between odor and stress or annoyance, with those with higher John Henryism scores more affected by malodor. Hydrogen sulfide appeared to be associated with feeling stressed or annoyed and nervous or anxious but not with the other 3 mood variables. We found that PM_{10} was not associated with the outcome variables, with the exception of a marginal association with feeling confused or unable to concentrate. Semivolatile PM_{10}, however, appeared to be associated with feeling stressed or annoyed and nervous or anxious and only marginally associated with the remaining 3 mood variables.

Though we are not aware of other work that has sought to link airborne emissions to reported stress and negative mood, there is a consistent literature documenting the effect of malodor on annoyance, both in laboratories[1,37,51–53] and other settings.[3,29,30] Several authors have also considered coping style as a potential effect modifier.[1,3,29,30,37] In field studies of annoyance response to industrial odors, people with higher scores for problem-oriented coping, or action-oriented coping, tended to report more annoyance following odor exposure than did people with lower scores.[3,29,30,37] In a laboratory study, however, Asmus and Bell did not find coping style to be an effect modifier.[1]

We found a stronger relationship between odor and stress in participants with high scores on the John Henryism Active Coping scale. Our findings are consistent with odor studies by Steinheider and Winneke,[29] Winneke et al.,[37] Sucker et al.,[30] and Both et al.[3] The John Henryism Active Coping scale was developed by Sherman James in studies conducted among African Americans in eastern North Carolina[46] and, therefore, may be especially appropriate in the context of the present investigation. It measured "the degree to which [Black Americans] felt they could control their environment through hard work and determination."[46(p259)] James hypothesized a poorer health outcome (higher blood pressure) in men who scored high on the scale but lacked the resources to control their environments.[46] Consistent with our a priori hypothesis, it appears that study participants who perceived that they had more control over their environment found an unpredictable and uncontrollable malodor more stressful than those who perceived they had less control.

Strengths and Limitations

The longitudinal design was a particular strength of this research. There were approximately 28 repeated measures for each participant. In the analyses, each participant served as her or his own control. Perceptions of stress and adverse mood vary between people, and we were able to statistically model the between-person variation in such perceptions. Physical measures of pollution are an additional strength of this research; previous studies have relied entirely on self-reported measures of exposure and outcome. We did, however, measure only several constituents of a chemically complex odor plume that includes, potentially, hundreds of volatile organic compounds.[23]

A further design limitation was the contemporaneous assessment of both exposure and outcome for the analyses of odor as a predictor of stress and negative mood. Because both exposure and outcome were assessed by self-report, it is difficult to determine how the assessment of one affected the assessment of the other. Participants spent 10 minutes outdoors before returning indoors to complete the required data collection activities; they rated the intensity of any malodor present and then rated stress and mood. Rating the odor while stressed or annoyed for reasons unrelated to odor may have induced a higher rating than the participant would have rated in the absence of feeling stressed or annoyed. Though the results of the analyses of odor and stress or mood must be interpreted in light of this design limitation, odor as a marker of exposure is important because it captures information on numerous other pollutants with odorant properties that we were unable to explicitly measure in this study. Furthermore, it permits consideration of the mixture of chemicals emitted from industrial hog

TABLE 3—Logistic Mixed Model Results for Associations Between Hydrogen Sulfide, PM_{10}, Semivolatile PM_{10}, Odor, Stress, and Negative Mood: Community Health Effects of Industrial Hog Operations Study, Eastern North Carolina, 2003–2005

Main Exposure and Outcome Variable	b	SE	*t*	OR (95% CI)
Hydrogen sulfide (ppb)				
Stressed or annoyed	0.17	0.048	3.54	1.18 (1.08, 1.30)
Nervous or anxious	0.11	0.044	2.55	1.12 (1.03, 1.22)
Gloomy, blue, or unhappy	0.012	0.063	0.18	1.01 (0.89, 1.15)
Angry, grouchy, or bad-tempered	0.039	0.047	0.84	1.04 (0.95, 1.14)
Confused or unable to concentrate	–0.074	0.12	–0.63	0.93 (0.73, 1.17)
PM_{10} ($\mu g/m^3$)				
Stressed or annoyed	0.00065	0.0051	0.13	1.00 (0.99, 1.01)
Nervous or anxious	0.0029	0.0052	0.57	1.00 (0.99, 1.01)
Gloomy, blue, or unhappy	0.012	0.010	1.11	1.01 (0.99, 1.03)
Angry, grouchy, or bad-tempered	0.0035	0.0057	0.61	1.00 (0.99, 1.01)
Confused or unable to concentrate	0.010	0.0070	1.43	1.01 (1.00, 1.02)
Semivolatile PM_{10} ($\mu g/m^3$)				
Stressed or annoyed	0.055	0.025	2.15	1.06 (1.00, 1.11)
Nervous or anxious	0.095	0.033	2.91	1.10 (1.03, 1.17)
Gloomy, blue, or unhappy	0.058	0.043	1.35	1.06 (0.97, 1.16)
Angry, grouchy, or bad-tempered	0.027	0.026	1.05	1.03 (0.98, 1.08)
Confused or unable to concentrate	0.043	0.036	1.22	1.04 (0.97, 1.12)
Twice daily odor rating (0–8)				
Stressed or annoyed	0.59	0.051	11.50	1.81 (1.63, 2.00)
Nervous or anxious	0.47	0.064	7.37	1.60 (1.41, 1.81)
Gloomy, blue, or unhappy	0.36	0.067	5.35	1.43 (1.25, 1.63)
Angry, grouchy, or bad-tempered	0.42	0.055	7.70	1.52 (1.37, 1.70)
Confused or unable to concentrate	0.27	0.065	4.20	1.31 (1.16, 1.50)

Note. OR = odds radio; CI = confidence interval; PM_{10} = particulate matter less than 10 μm in aerodynamic diameter. Adjusted for time of day, morning versus evening.

operations as opposed to its individual constituent parts.

Conclusions

In a community-based, longitudinal study of neighbors of industrial hog operations, we observed associations among malodor, several airborne emissions, stress, and negative mood. Specifically, we observed increased reporting of stress and negative mood in response to increasing malodor. Additionally, increases in H_2S and semivolatile PM_{10}, both odorous in nature, were associated with reported stress and 1 or more mood variables. Our findings complement a large literature on malodor as an environmental stressor. Malodor and concomitant airborne emissions do appear to trigger stress and negative mood in nearby residents unwillingly exposed at home.

It is important to contextualize the effect of malodor on the lives of nearby residents. People who cannot afford air conditioning, clothes dryers, membership at a gym, and entertaining in restaurants depend on opening their windows for ventilation, drying their clothes outside, exercising in their yards, and entertaining family and friends in and around their homes. In ethnographic interviews, neighbors of industrial hog operations report that they refrain from gardening, walking, chores, and having cookouts with family and friends because of hog odor, and they report interruption of their sleep because of hog odor inside their homes.[54] This is significant, because physical activity, social support, and sleep are important for health. Industrial hog operations in North Carolina are located disproportionately in low income, African American communities[35] that have limited financial resources to prevent the influx of polluting industries as well as to manage the impacts of uncontrollable environmental malodors on physical and mental health. Recognizing that health is a state of well-being, and not merely the absence of disease,[40] public health and environmental professionals should consider the impacts of environmental malodor and its potential role in magnifying health disparities. ■

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Contributors

R. Avery Horton had primary responsibility for the study, completed the analyses, and wrote the first draft. S. Wing actively provided consultation throughout all phases of the research. S. W. Marshall provided statistical expertise in the design and analysis of data. K. A. Brownley consulted in the design phase and in the interpretation and contextualization of the results. All authors contributed to the writing of the article.

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Human Participant Protection

This study was approved annually by the institutional review board of the University of North Carolina at Chapel Hill. All study participants provided informed consent.

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Attachment A2

Air Pollution from Industrial Swine Operations and Blood Pressure of Neighboring Residents

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Background: Industrial swine operations emit odorant chemicals including ammonia, hydrogen sulfide (H_2S), and volatile organic compounds. Malodor and pollutant concentrations have been associated with self-reported stress and altered mood in prior studies.

Objectives: We conducted a repeated-measures study of air pollution, stress, and blood pressure in neighbors of swine operations.

Methods: For approximately 2 weeks, 101 nonsmoking adult volunteers living near industrial swine operations in 16 neighborhoods in eastern North Carolina sat outdoors for 10 min twice daily at preselected times. Afterward, they reported levels of hog odor on a 9-point scale and measured their blood pressure twice using an automated oscillometric device. During the same 2- to 3-week period, we measured ambient levels of H_2S and PM_{10} at a central location in each neighborhood. Associations between systolic and diastolic blood pressure (SBP and DBP, respectively) and pollutant measures were estimated using fixed-effects (conditional) linear regression with adjustment for time of day.

Results: PM_{10} showed little association with blood pressure. DBP [β (SE)] increased 0.23 (0.08) mmHg per unit of reported hog odor during the 10 min outdoors and 0.12 (0.08) mmHg per 1-ppb increase of H_2S concentration in the same hour. SBP increased 0.10 (0.12) mmHg per odor unit and 0.29 (0.12) mmHg per 1-ppb increase of H_2S in the same hour. Reported stress was strongly associated with BP; adjustment for stress reduced the odor–DBP association, but the H_2S–SBP association changed little.

Conclusions: Like noise and other repetitive environmental stressors, malodors may be associated with acute blood pressure increases that could contribute to development of chronic hypertension.

Key words: agriculture, air pollution, community-based participatory research, environmental justice, epidemiology, health disparities, odors, psychosocial stress. *Environ Health Perspect* 121:92–96 (2013). http://dx.doi.org/10.1289/ehp.1205109 [Online 28 October 2012]

The rapid global expansion of confined animal feeding operations (CAFOs) has created environmental health concerns at local, regional, and global scales, including infectious and respiratory diseases, reduced quality of life, impacts on the built environment, and environmental injustice (Pew Commission on Industrial Food Animal Production 2008). CAFO airborne emissions, including ammonia, hydrogen sulfide (H_2S), volatile organic compounds, and endotoxins, originate from confinement buildings, waste storage areas, and land application of animal waste (National Research Council 2003).

North Carolina experienced a rapid transformation of swine production during the 1980s and 1990s. The number of producers declined, the size of operations grew, the swine population increased from approximately 2.5 million to 10 million, and production shifted to the eastern coastal plain region of the state (Furuseth 1997). In North Carolina, swine CAFOs are concentrated in low-income communities of color (mostly African American), where older housing and lack of central air conditioning could increase human exposure to air pollutants (Wing et al. 2000). Studies conducted in Germany and the United States reported that neighbors describe odors from swine CAFOs as annoying and offensive (Schiffman 1998; Tajik et al. 2008; Thu 2002, 2003; Thu and Durrneberger 1998; Radon et al. 2007). In a previous study of communities neighboring North Carolina CAFOs (Schinasi et al. 2011), we found that self-reported hog odor and H_2S are associated with acute irritation of the eyes, nose, and throat, and also that particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}) is associated with eye irritation. In addition to physical symptoms and negative mood (Bullers 2005; Horton et al. 2009; Schiffman et al. 1995), CAFO neighbors have reported that because of frequent and unpredictable episodes of malodor, they were unable to engage in valued traditions of rural life, including gardening, family gatherings, cookouts, visiting neighbors, and drying laundry (Tajik et al. 2008; Thu 2002, 2003; Thu and Durrneberger 1998).

Several studies have found relationships between malodor from swine CAFOs and chronic (Schiffman et al. 1995) or acute (Horton et al. 2009) stress in neighbors. Other studies have reported that environmental stressors are associated with increased blood pressure (BP) (Attarchi et al. 2012; Belojevic and Evans 2012; Djindjic et al. 2012) and that odorant compounds perceived as pleasant attenuated exercise-related increases in BP (Nagai et al. 2000). African Americans and low-income people experience an excess prevalence of chronic hypertension (Carson et al. 2011; Keenan and Rosendorf 2011; Liao et al. 2011), as well as hypertension-related morbidity (Liao et al. 2011) and mortality (Fiscella and Holt 2008). Identification of environmental factors that contribute to BP elevations could inform efforts to prevent upward shifts of BP in populations.

In this study we evaluated whether measures of swine CAFO air pollution were associated with acute changes in BP among neighbors during follow-up of approximately 2 weeks. We did not compare BPs of CAFO neighbors and other people; rather, we compared each participant's BP during times of more and less exposure to swine CAFO air pollution. In this design each participant served as her or his own control. Characteristics that were essentially constant during the short follow-up (e.g., age, socioeconomic status,

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medical history, body mass, occupation, personality) could not cause bias in estimates of the exposure–outcome relationship. Chronic effects of exposure, however, could not be evaluated.

Methods

Setting and data collection. The study was conducted in partnership with the Concerned Citizens of Tillery (CCT), a community-based organization in Halifax County that promotes the health, environmental, and political interests of predominantly African-American communities in eastern North Carolina (Wing et al. 1996). CCT has partnered with universities to provide medical care through the Tillery People's Clinic and to conduct research on health and environmental justice (Tajik and Minkler 2006). For this study, the CCT staff organized community meetings in areas with a high density of swine CAFOs and provided information about our ongoing study to attendees, who were invited to contact CCT or University of North Carolina–Chapel Hill researchers if they were interested in participating in the study (Wing et al. 2008a). We sequentially enrolled between 4 and 10 volunteers in each of 16 rural communities from 2003 to 2005, and participants began data collection within 24–36 hr. Enrollment did not take place between mid-December and mid-February because of holidays and cold weather. Numbers of nearby swine CAFOs, participants, and other community-specific characteristics have been reported previously (Wing et al. 2008b).

To be eligible, participants had to be ≥ 18 years of age and nonsmokers, and live within 1.5 miles of at least one swine CAFO (Wing et al. 2008a), defined as a facility housing > 250 animals and using a liquid waste management system (Wing et al. 2000). At an initial training session, participants chose morning and evening times when they would sit outside each day for approximately 2 weeks (in three neighborhoods participants chose to continue up to 1 more week). They provided information about regular use of medications, and each participant's odor sensitivity was tested using a standard set of butanol dilutions to evaluate the lowest concentration that could be distinguished from zero (e.g., Croy et al. 2009). Participants completed the John Henryism Active Coping (JHAC) scale, which measures the predisposition to respond behaviorally to psychosocial environmental stressors (James et al. 1987); higher values indicate a greater predisposition to cope actively. Participants were classified by reported use (yes/no) of antihypertensive medications (e.g., drugs classified as beta blockers, calcium channel blockers, angiotensin-converting-enzyme inhibitors, diuretics). They learned how to use a structured diary to record levels of swine odor, stress, and symptoms, and they practiced measuring their BP with an automated oscillometric device. Time spent outdoors and times of diary completion were tracked using a digital clock provided and set by researchers. Informed consent was obtained at the training session using a procedure approved by the University of North Carolina Institutional Review Board, which reviewed the study annually. We obtained a Certificate of Confidentiality from the National Institutes of Health (Wing et al. 2008a) because of prior attempts by the pork industry to obtain confidential records (Wing 2002).

Each morning and evening, participants sat outside for 10 min and completed the first of four pages of a data-collection diary. They then returned indoors to complete the remaining pages and measure their BP (Wing et al. 2008a). They rated the strength of swine odor during the 10-min period outdoors on a nine-level Likert-type scale [0 (none) to 8 (very strong)], and evaluated perceived stress ("How do you feel now … stressed or annoyed?) on a nine-level scale [0 (none) to 8 (extremely)]. Participants measured their BP twice in a seated position. They were instructed to wait 1 min between readings, raising their right arm above their head for the first 30 sec and then resting for the remaining time before taking their BP again. They printed the results and taped the printout with the systolic (SBP) and diastolic (DBP) values and current time into the diary. We treated the average of the two readings as dependent variables.

While participants collected data, we monitored air pollution at a central location in each neighborhood. The mean and median distance from air monitors to participant homes was 0.2 miles and 0.1 miles, respectively (Wing et al. 2008a). Swine CAFOs release many odorant chemicals including ammonia, H_2S, and hundreds of volatile organic compounds (Schiffman et al. 2001). Odorant chemicals may occur as gases or particles. We quantified H_2S, which is produced by the anaerobic decomposition of fecal waste, as a marker of this complex mixture that is related to hog odor intensity (Wing et al. 2008b; Schiffman et al. 2005). H_2S is a specific marker of swine CAFO pollution in the study areas because other H_2S-emitting industries such as waste water treatment plants, petrochemical plants, and paper mills, were not present. Average ambient H_2S concentrations measured every 15 min with an MDA Scientific Single Point Monitor (Zellweger Analytics Inc., Lincolnshire, IL) were used to calculate hourly averages; 15-min values below the detection limit of 2 ppb were treated as zero. We considered average concentrations during the 1 hr before BP measurements as predictors of SBP and DBP.

We measured hourly levels of PM_{10} using a Series 1400a tapered element oscillating microbalance Ambient Particulate Monitor (Rupprecht and Patashnick Co. Inc., East Greenbush, NY). A Series 8500 FDMS Filter Dynamics Measurement System (Rupprecht and Patashnick Co. Inc.) was used to quantify semivolatile PM_{10}. Semivolatile particles consist of compounds that are present in both vapor and condensed phases. Airborne PM is ubiquitous; although CAFOs are one source, particles are not a specific marker of CAFO pollutants. We reported previously that semivolatile PM_{10} showed little association with hog odor in the study neighborhoods and that PM_{10} was related to hog odor only when wind speeds were high (Wing et al. 2008b).

Statistical analysis. In this repeated-measures design, each participant served as her or his own control. The sample size is a function of the number of participants and the number of observations (records) per person. We used linear fixed-effects regression to model repeated measures for individuals (Allison 2005). This approach estimates the average within-person associations between exposure measures and BP by conditioning on person, and eliminates bias from any measured or unmeasured confounding factors that do not change during follow-up. Relationships between SBP and DBP and air pollution appeared linear across categories of exposure (data not shown), so they were modeled as continuous variables. BP varies diurnally, as do hog odor and H_2S (Wing et al. 2008b); therefore, time of day (AM vs. PM) was included as a covariate in all models. In separate analyses, we also adjusted for self-reported stress, a potential mediator of associations between pollutants and BP. Sex and odor detection threshold (dichotomized at the median) were considered potential modifiers related to odor perception, whereas JHAC score (dichotomized at the median) and use of antihypertensive medication (yes/no) were considered potential modifiers of BP reactivity to environmental stressors. We also considered modification by age (dichotomized at the median) because it could influence either odor perception or BP reactivity.

Observations (records) with missing values for a variable were dropped from models including that variable. Model coefficients represent the average within-person change in BP for each unit increase in pollution. In nonrandomized studies, confidence limits and *p*-values do not quantify the confidence or probability that a point estimate would occur within a specified interval due to chance; therefore, we report standard errors of the regression coefficients as a measure of precision and *t*-values as indicators of the improvement in the fit of the model associated with the exposure variable. Degrees of freedom for *t*-tests, n-1, are large and can be considered equivalent for comparing *t* values.

Results

Descriptive characteristics of the 101 participants are given in Table 1. Half of the participants were > 53 years of age, and two-thirds were women. Among the 97 participants whose odor detection threshold was determined, 55 had a butanol odor detection threshold of ≤ 40 ppm. Forty-two participants reported taking one or more BP medications. Among the 96 participants who completed the JHAC, 46 had a score > 52. Most participants (85) identified themselves as black.

Table 2 presents distributions of reported hog odor intensity during the 10 min outdoors, average pollutant concentrations in the hour before BP measurement, SBP, and DBP. Odor ratings were missing in 6% of the records, and no odor was reported in 48% of the records. Very strong odor (a rating of 6, 7, or 8) was reported 6% of the time. Hourly H_2S measurements were missing in approximately 9% of the records, and most (88%) were below the limit of detection (2 ppb). PM measures were missing in 32.2% of the records, primarily because of equipment malfunction during periods of high temperature and humidity (Wing et al. 2008b). For 12.4% of records, semivolatile particle concentrations were < 0; this occurs when concentrations are low because microbalance estimates are derived by subtraction of sequential mass values that are measured with error (Wing et al. 2008b). BP was missing in 1.4% of the records. SBP readings were < 120 mmHg in approximately 30% of the records and > 140 mmHg in approximately 25% of the records. DBP was < 80 mmHg in 61% of the records and ≥ 90 mmHg in 11% of the records. No participants were missing data for all their records.

Associations between air pollutants and BP adjusted for time of day (AM or PM) are presented in Table 3. Each unit increase in reported hog odor on the 0–8 intensity scale was associated with average estimated increases [β (SE)] of 0.10 (0.12) and 0.23 (0.08) mmHg for SBP and DBP, respectively. A 1-ppb increase in H_2S was associated with increases of 0.29 (0.12) mmHg for SBP and 0.12 (0.08) mmHg for DBP. PM_{10} was not associated with BP. Semivolatile PM_{10} was not associated with SBP but had a small negative association with DBP [–0.06 (0.03)].

Table 4 provides beta coefficients for hog odor and H_2S according to potential modifying variables. Coefficients for PM_{10} and semivolatile PM_{10} are not shown because their main effect estimates were small, they are not specific markers of swine CAFO air pollution, and data are missing for almost one-third of the records. Hog odor coefficients for SBP were all positive, but none had *t*-values > 1.17. Coefficients for DBP were positive and all had *t*-values near or above 2 except for participants ≤ 53.7 years of age, for whom the β (SE) is 0.08 (0.12). Coefficients for both SBP and DBP were larger for older participants than younger participants [0.14 (0.15) and 0.33 (0.10) vs. 0.04 (0.18) and 0.08 (0.12), respectively] and for men than women [0.20 (0.23) and 0.36 (0.15) vs. 0.07 (0.13) and 0.19 (0.09), respectively]. Associations between hog odor and SBP were larger for participants with JHAC scores ≤ 52 compared with those for persons with JHAC scores > 52 [0.18 (0.17) compared with 0.01 (0.16)] and for participants who reported no use of antihypertensive drugs compared with those with regular use [0.19 (0.16) compared with 0.01 (0.17)]. For H_2S, coefficients for both SBP and DBP were larger for men than women [0.56 (0.30) and 0.48 (0.19) compared with 0.24 (0.13) and 0.05 (0.08), respectively]; participants with butanol odor sensitivity thresholds > 40 ppm than for those with thresholds ≤ 40 ppm [0.33 (0.14) and 0.13 (0.09) compared with 0.17 (0.22) and 0.07 (0.14), respectively]; and participants with JHAC scores of ≤ 52 than those with scores > 52 [0.36 (0.14) and 0.17 (0.09) compared with 0.02 (0.24) and –0.07 (0.15), respectively]. The SBP coefficient was larger for participants who did not report taking BP medications compared with those who did [0.38 (0.14) compared with 0.07 (0.22)].

SBP and DBP were strongly associated with reported stress, increasing on average 0.82 (0.21; t = 3.98) and 0.57 (0.13 mmHg; t = 4.28), respectively, for every unit increase on the 0–8 scale. We included stress in models reported above (in addition to time of day) to evaluate whether associations of BP with hog odor and H_2S change after adjustment for this potential mediator. With adjustment for reported stress, coefficients for the association between hog odor and DBP declined from 0.23 (0.08) to 0.15 (0.08), whereas the coefficient for SBP decreased from 0.10 (0.12) to –0.04 (0.12). With adjustment for reported stress, there was little change in the coefficient for the association between H_2S and DBP [0.15 (0.08) vs. 0.12 (0.08) before adjustment] or SBP [0.26 (0.12) vs. 0.29 (0.12) before adjustment].

Table 1. Characteristics of participants [*n* (%) of nonmissing observations], Community Health Effects of Industrial Hog Operations study.

Variable	Participants (*N* = 101)	Records (*N* = 2,949)
Age (years)		
≤ 53.7	51 (50.5)	1,410 (47.9)
> 53.7	50 (49.5)	1,539 (52.2)
Gender		
Women	66 (65.3)	1,945 (66.0)
Men	35 (34.7)	1,004 (34.0)
Odor threshold		
Missing[a]	4 (4.0)	91 (3.1)
Butanol ≤ 40ppm	55 (56.7)	1,559 (54.5)
Butanol > 40ppm	42 (43.3)	1,299 (45.5)
BP medication used		
No	59 (58.4)	1,680 (57.0)
Yes	42 (41.6)	1,269 (43.0)
JHAC score[b]		
Missing[a]	5 (5.0)	117 (4.0)
≤ 52	50 (52.1)	1,480 (52.3)
> 52	46 (47.9)	1,352 (47.7)

[a]Percent of all observations. [b]Higher JHAC score indicates higher active coping with psychosocial stressors.

Discussion

In this community-based participatory repeated-measures study we found that, on average, BP of participants living near swine CAFOs increased in association with increases in markers of transient plumes of odorant air pollution. Because each participant served as her or his own control, factors that did not change during the 2-week study—including body mass, race, socioeconomic position, medical and dietary history, and prior BP—could not

Table 2. Distributions of odor, H_2S, and BP from the total of nonmissing records (*N* = 2,949), Community Health Effects of Industrial Hog Operations study.

Variable (scale)	*n* (%)
Odor (0–8)	
Missing[a]	177 (6.0)
None	1,419 (48.1)
1–2	779 (26.4)
3–5	407 (13.8)
6–8	167 (5.7)
Stress (0–8)	
Missing[a]	58 (2.0)
None	2,331 (80.6)
1–2	436 (15.1)
3–5	91 (3.2)
6–8	33 (1.2)
H_2S (ppb)	
Missing[a]	255 (8.6)
0	2,412 89.5
0–2	170 (6.3)
2–4.99	77 (2.9)
5–47.5	35 (1.3)
PM_{10} (μg/m^3)	
Missing[a]	948 (32.1)
< 10	415 (20.7)
10–19.9	783 (39.1)
20–29.9	528 (26.4)
30–502.0	275 (13.7)
Semivolatile PM_{10} (μg/m^3)	
Missing[a]	948 (32.2)
< 0	366 (18.3)
0–2.99	638 (31.9)
3–7.99	767 (38.3)
> 8	230 (11.5)
SBP (mmHg)	
Missing[a]	41 (1.4)
< 120	897 (30.8)
120–139	1,257 (43.2)
140–159	510 (17.5)
> 160	244 (8.4)
DBP (mmHg)	
Missing[a]	41 (1.4)
< 80	1,804 (62.0)
80–89	781 (26.9)
90–99	221 (7.6)
> 100	102 (3.5)

[a]Percent of all records.

confound these associations. Estimated DBP was almost 2 mmHg higher during periods of very strong odor (a rating of 8) compared to none, and estimated SBP was almost 3 mmHg higher when H_2S concentrations were 10 ppb compared with times when H_2S was zero (below the limit of detection). This magnitude of effect could have public health importance because of the frequency and duration of odor episodes near CAFOs. The 101 people who participated in this study for approximately 2 weeks reported 1,655 episodes of outdoor hog odor, 38% of which lasted > 1 hr, and 17% of which had a mean odor ≥ 5 (on the scale of 0–8); participants also reported 500 episodes of indoor odor (Wing et al. 2008b). If the associations were causal and if malodors from other sources such as sewage, landfills, and chemical refineries produce similar effects, then control of environmental malodor might help prevent repeated acute elevations of BP that could contribute to development of chronic hypertension.

With approximately 29 measures per person, the sample size for this study was primarily suited to examining within-person covariation in exposures and outcomes. Although estimates within subgroups defined by non–time-varying factors are imprecise, some interactions are of interest. Associations between H_2S and SPB were similar for both older and younger participants, whereas the odor–DBP association was observed primarily among older participants. Beta coefficients for both odor and H_2S were larger for men than women. The magnitude of the association between BP and hog odor was not related to the butanol odor sensitivity threshold. Because the effectiveness of peoples' active coping is reduced by lack of resources, persons with high JHAC scores and low socioeconomic position are expected to be more physiologically reactive to psychosocial stressors than people with high JHAC scores and high socioeconomic position, or people with low JHAC scores (James et al. 1987). Contrary to our expectation, even though all participants in this study lived in low-income areas, associations between hog air pollution markers and BP were not stronger among participants with high JHAC scores. Associations for SBP were generally weaker among participants who were taking BP medications, which may reduce responses to environmental stimuli.

Table 3. Linear fixed effects beta coefficients (SEs) and *t*-values for associations of one-unit increases in pollutants with SBP and DBP, adjusted for time-of-day (AM or PM), Community Health Effects of Industrial Hog Operations study.

	SBP		DBP	
Pollutant	β (SE)	*t*-Value	β (SE)	*t*-Value
Odor (0–8)	0.10 (0.12)	0.86	0.23 (0.08)	3.02
H_2S (ppb)	0.29 (0.12)	2.45	0.12 (0.08)	1.52
PM_{10} (μg/m^3)	–0.01 (0.01)	–0.78	–0.00 (0.01)	–0.41
Semivolatile PM_{10} (μg/m^3)	–0.02 (0.05)	–0.45	–0.06 (0.03)	–1.66

Table 4. Linear fixed effects beta coefficients (SEs) and *t*-values for potential modifiers of associations of BP with one-unit increases in hog odor and H_2S, adjusted for time-of-day (AM or PM), Community Health Effects of Industrial Hog Operations study.

	SBP		DBP	
Modifier	β (SE)	*t*-Value	β (SE)	*t*-Value
Hog odor (0–8)				
Age ≤ 53.7 years	0.04 (0.18)	0.23	0.08 (0.12)	0.68
Age > 53.7 years	0.14 (0.15)	0.93	0.33 (0.10)	3.34
Women	0.07 (0.13)	0.50	0.19 (0.09)	2.11
Men	0.20 (0.23)	0.85	0.36 (0.15)	2.37
Butanol threshold ≤ 40 ppm	0.10 (0.15)	0.67	0.21 (0.10)	2.17
Butanol threshold > 40 ppm	0.10 (0.19)	0.54	0.24 (0.12)	2.03
JHAC score ≤ 52	0.18 (0.17)	1.07	0.22 (0.11)	2.05
JHAC score > 52	0.01 (0.16)	0.06	0.20 (0.11)	1.92
No BP meds	0.19 (0.16)	1.17	0.25 (0.11)	2.31
Any BP meds	0.01 (0.17)	0.04	0.21 (0.11)	1.96
H_2S (ppb)				
Age ≤ 53.7 years	0.30 (0.15)	1.97	0.13 (0.10)	1.32
Age > 53.7 years	0.28 (0.19)	1.45	0.10 (0.12)	0.78
Women	0.24 (0.13)	1.85	0.05 (0.08)	0.58
Men	0.56 (0.30)	1.90	0.48 (0.19)	2.51
Butanol threshold ≤ 40 ppm	0.17 (0.22)	0.78	0.07 (0.14)	0.48
Butanol threshold > 40 ppm	0.33 (0.14)	2.40	0.13 (0.09)	1.49
JHAC score ≤ 52	0.36 (0.14)	2.67	0.17 (0.09)	1.90
JHAC score > 52	0.02 (0.24)	0.08	–0.07 (0.15)	–0.45
No BP medication	0.38 (0.14)	2.70	0.10 (0.09)	1.12
Any BP medication	0.07 (0.22)	0.34	0.15 (0.14)	1.07

Although the repeated-measures design and fixed-effects analysis precludes confounding from time-independent factors that differ between people, time-related factors associated with both air pollution and BP could have either attenuated or exaggerated associations. Time of day (AM vs. PM) was included in all models; therefore, potential time-related factors would need to be associated with pollution and BP within times of day in order to act as confounders. Time-related confounding could occur if a cause of acute BP change that is not a consequence of CAFO air pollution covaried with the CAFO air pollutants in participants' neighborhoods.

Measurement errors could also impact estimates of association between odorant pollutants and BP. In a clinical or experimental setting, BP is typically measured by a trained technician in a standardized manner. In contrast, in the present study, each participant measured her or his own BP twice each day at home, which could reduce the precision of the effect estimates. Use of a portable printer with a time stamp to record BP values in the diaries prevented transcription errors that could have introduced systematic errors related to odor intensity. The temporal sequence of sitting outside prior to BP measurement was reversed in < 2% of records (Schinasi et al. 2009).

Although participants recognized hog odor and could rate it on the 0–8 scale from "none" to "very strong," we did not evaluate the reproducibility of their ratings, which could be affected by physical and social context. For example, participants might rate an odor as more intense on a day that they expected company if they were ashamed of their expected guests' reactions to the presence of fecal odor at their home. More precise measures of odor can be made in units of dilution to threshold using an olfactometer (Lambert et al. 2000); however, it was not feasible to use such a device in this participatory study. We evaluated participants' odor sensitivity threshold using a butanol standard and expected that associations between hog odor and BP might be attenuated among participants with poorer odor sensitivity; however, associations with hog odor differed little by odor sensitivity. In an experiment including 44 volunteers, van Thriel et al. (2008) reported that butanol odor threshold was not related to ratings of environmental odorants.

H_2S was the chemical marker of odorant swine CAFO air pollution that we could quantify over short time period; these measures cannot be affected by response bias. Because there are no other major industrial sources of H_2S in the study communities, it is a specific marker of swine CAFO emissions; however, this marker is not sensitive, in part, because of the detection threshold of the instrument (~ 2 ppb/15 min). Hog

odor, which has a distinctive character due to a complex mixture of volatile organic compounds (Schiffman et al. 2001; Karageorgos et al. 2010), was often reported when H_2S levels were below the detection limit. Another source of measurement error comes from the placement of the H_2S monitor at a central location in rural neighborhoods, which was as far as approximately 1 mile from some participants' residences (median, 0.1 mile). Narrow plumes of odorant compounds from swine CAFOs could be present at participants' homes but not at the monitor, or vice versa. We expect this type of exposure misclassification would attenuate any real associations between H_2S and BP.

Relationships between odorant air pollutants and BP could be produced by psychophysiological or pharmacological mechanisms (Shusterman 1992). Our findings that odor and H_2S, but not PM, were associated with BP increases are consistent with a psychophysiological mechanism. The lack of an association with PM could also be related to the lower levels or different composition of PM in rural communities compared with urban areas typically studied. Furthermore, many observations were missing for PM. We evaluated BP in this study because environmental exposure to swine odor in this population has been associated with self-reported stress (Horton et al. 2009), and acute stress is associated with transient BP elevation (Sparrenberger et al. 2009). Odorant pollution could also produce other changes in a person's environment that cause acute changes in BP, for example, irritability of a household member.

The pharmacological actions of swine CAFO air emissions on BP are unknown and difficult to predict because emissions include many chemical compounds and fine particles (Schiffman et al. 2001). Although we measured H_2S as an indicator of the odorant component of this mixture, growing evidence suggests that H_2S, an endogenous gasotransmitter, acts as a vasodilator (Wagner 2009). To the extent that exogenous H_2S plays a similar role, its presence in odorant plumes could therefore attenuate associations between swine odor and BP.

The setting for our study, the coastal plain of eastern North Carolina, has one of the highest densities of swine production in the world (Pew Commission on Industrial Food Animal Production 2008). Historically, it is part of both the Black Belt (home to a majority of rural African Americans) and the stroke belt (an area of high mortality from cerebrovascular and cardiovascular diseases) (Casper et al. 1995). Swine CAFOs in the state are highly disproportionately located in low-income communities of color (Wing et al. 2000). If swine CAFO air pollution contributes to high BP in this region, the associated cardiovascular morbidity and mortality would be among the consequences of environmental injustice.

Malodors are produced by other types of CAFOs, waste disposal sites, refineries, chemical plants, waste water treatment plants, and land application of sewage sludge. These facilities and activities expose communities that lack political power to environmental malodors while benefiting consumers and producers in nonimpacted areas. Therefore, the generalizability of findings reported here is relevant to public health protection. Communities with low levels of political influence are less able to prevent siting of such facilities than are communities with political power, and they are less able to demand the best technologies for reducing resulting pollutants. Repeated acute physical environmental stressors, such as malodor and noise, may be aspects of the built environment that contribute to racial and economic disparities in high BP and its sequelae.

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Attachment A3

Public Health Concerns for Neighbors of Large–Scale Swine Production Operations

K. M. Thu

Abstract

This article provides a review and critical synthesis of research related to public health concerns for neighbors exposed to emissions from large–scale swine production operations. The rapid industrialization of pork production in the 1990s produced a generation of confined animal feeding operations (CAFOs) of a size previously unseen in the U.S. Recent research and results from federally sponsored scientific symposia consistently indicate that neighbors of large–scale swine CAFOs can experience health problems at significantly higher rates than controlled comparison populations. Symptoms experienced by swine CAFO neighbors are generally oriented toward irritation of the respiratory tract and are consistent with the types of symptoms among interior confinement workers that have been well documented in the occupational health literature. However, additional exposure assessment research is required to elucidate the relationship of reported symptoms among swine CAFO neighbors and CAFO emissions.

Keywords. *Swine, CAFOs, Public health, Neighbors.*

The report "Agriculture at Risk: A Report to the Nation" (Merchant et al., 1989) played a central role in elevating agricultural health issues to national prominence. The cast of agricultural health issues laid forth in the report set the stage for shaping priority research issues. However, the rapid pace of agricultural industrialization resulted in tremendous change in the agricultural sector between the time of the report in 1989 and the present. Nowhere is this more evident than in the livestock sector, particularly the swine industry. The rapid structural shift in the swine sector towards concentration and consolidation created environmental and public health problems largely unforeseen in the 1989 report, or even in the Surgeon General's Report on Agricultural Safety and Health issued three years later (Myers et al., 1992). Specifically, the swift growth of large–scale confined animal feeding operations (CAFOs) in the 1990s has resulted in the emergence of whole new agricultural health issues previously unmentioned.

In this article, I focus on emergent agricultural health concerns related to CAFOs, namely public health concerns for neighbors exposed to CAFO emissions. Concerns have been directed at a broad spectrum of CAFOs, including dairy, cattle, and poultry. However, the swine industry has received the most attention from both a public health and public policy standpoint. In addition, considerable research on occupational

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health challenges among swine confinement workers was in fact present at the time of the "Agriculture at Risk" report, and numerous subsequent conferences provide a backdrop for consideration of CAFO neighbor health issues. Consequently, this article will focus directly, though not exclusively, on reviewing research and concomitant agricultural and public health issues related to CAFO emissions and neighbor health concerns in the swine sector.

Background

The industrialization of agriculture dates back to at least the previous century and is characterized, in part, by the substitution of fossil fuels and capital intensive production technology for human labor (Thu and Durrenberger, 1998). The substitution of fossil fuel–based technology in place of farmers has resulted in the consolidation of production into fewer hands and the attendant growth of large–scale production operations. The swine industry is a classic example of this industrialization process. There is very little difference between the total U.S. inventory of hogs in the year 2000 (59.3 million) compared with the total inventory of hogs produced over 80 years earlier in 1915 (60.6 million) (USDA NASS). However, while overall production volume has changed little, the structure of the industry has shifted radically. As revealed in figure 1, the number of hog producers in the U.S. declined precipitously from the 1960s to the present. Notable in this regard is the rapid rate of decline and concurrent emergence of relatively large production operations. For example, in a mere six–year period from 1993 to 1999, there was a 250% increase in the total U.S. hog inventory concentrated in operations with 5,000 or more hogs each (USDA NASS).

The structural shift in the hog industry paralleled technological changes. Most notable among these is the transformation from pasture–based and open–lot hog production to totally confined production beginning in the 1970s. Movement to totally confined production provided a seeming advantage to the bulk of hog farmers in the Midwest by providing an antidote to harsh climatic conditions that impeded growth rates and time to market. Moreover, enclosed production units provided an opportunity for stricter control of feed rationing and reproduction. However, as is so often the case, technological changes produced unforeseen costs.

Worker Health Problems and Swine CAFOs

One of the principles underlying confined animal production is to control the climatic elements that hinder swine growth, particularly in temperate regions. To accomplish this, an enclosed environment is created to keep outdoor conditions, most notably the frigid cold, from coming indoors. This also means that elements inherent to hog production will, to varying extents, be confined to the inside. Soon after confinement production emerged in the 1970s, studies began to reveal that confining pigs also meant confining airborne elements injurious to both workers and pigs. In a 25–year period beginning in the 1970s, nearly 30 published studies consistently revealed a variety of health problems among swine confinement workers, the most notable of which are a series of respiratory problems (see reviews in Thorne et al., 1996; Cole et al., 2000).

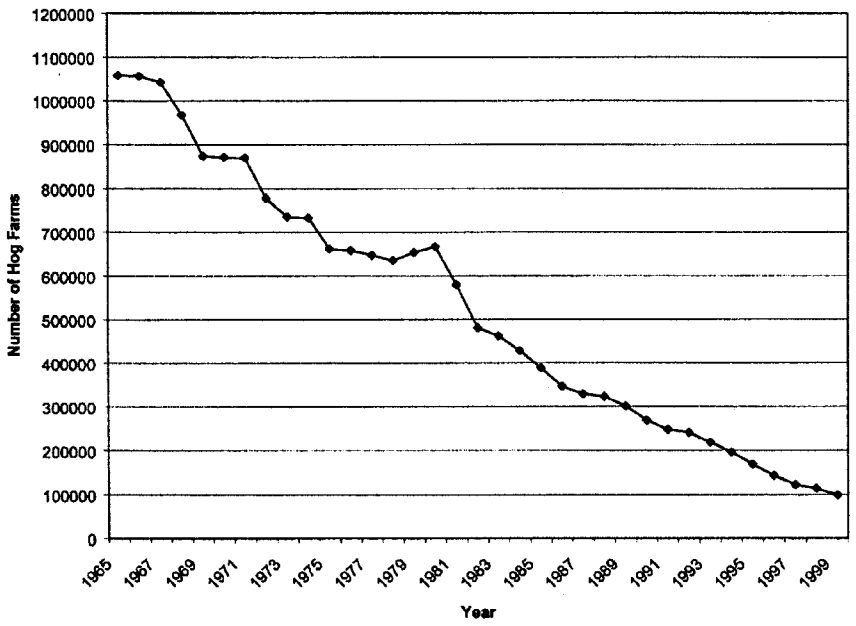

Figure 1. Total U.S. hog farmers from 1965 to 1999 (USDA NASS).

Respiratory problems include a series of overlapping conditions such as chronic bronchitis, occupational asthma, and organic dust toxic syndrome that have been documented to occur in up to 30% of swine confinement workers. Symptomatic signals of chronic problems include nagging cough with production of phlegm persisting for more than three months in a year, chest tightness and/or wheezing associated with work in a confinement, persistent fatigue in conjunction with headaches and difficulty breathing, symptoms of a cold that won't go away (stuffy nose, sore throat, and "popping" ears), and decreases in pulmonary function (Thorne et al., 1996, p. 163–164). Six or more years of exposure heightens the potential for chronic problems, while a minimal two–hour exposure inside a confinement may result in shorter–term acute symptoms such as itchy, watery eyes and chest tightness. Moreover, many of these symptoms and overarching conditions can co–occur, making them difficult to discern and assess. It is reasonably clear that persons with medical pre–conditions, such as asthma, are more susceptible to these constellations of problems. It has also been suggested that some of these conditions may result in irreversible lung damage (Thorne et al., 1996, p. 164).

More detailed research identified the concentration levels of interior ambient airborne elements, including their interactive dynamics, which put exposed worker populations at risk. For example, recommended gas (7 ppm ammonia), dust (2.5 mg/m^3 total dust; 0.23 mg/m^3 respirable dust), and endotoxin (100 EU/m^3) levels have been developed for interior swine confinement operations based on dose–response research (Donham et al., 1995; Reynolds et al., 1996). Researchers have also noted that when these elements combine (e.g., ammonia attached to small dust particles), they may have an added negative health consequence.

At the time of the "Agriculture at Risk" report, occupational health problems among swine confinement workers were clearly recognized. The occupational health research over the past ten years has resulted in further clarification of exposures and the connections to health symptomologies and defined medical conditions. However, virtually unknown in 1989 was the existence of health problems among neighbors exposed to swine CAFOs. Since the mid–1990s, research examining the health of neighbors of large–scale swine production has opened a new chapter concerning the health costs of confined swine production.

An Emergent Public Health Issue: Swine CAFO Neighbors

There is no indication in the earliest occupational health literature that swine CAFOs posed any real or potential health problems to neighbors. This is understandable since the relatively modest size of early–generation swine CAFOs simply did not seem likely to pose an air–quality challenge beyond nuisance odor. Indeed, an entire cottage research industry developed among agricultural engineers to understand and control emissions from livestock facilities as an odor problem largely devoid of any heath reference (Hobbs, 1995; Miner, 1975; O'Neill and Phillips, 1992). Nonetheless, a close examination of early odor literature provides clues that a lingering health issue could be present.

For example, early agricultural engineering reports (Overcash, 1984) indicated the possibility that livestock odors could elicit deleterious physiological responses, including nausea, vomiting, headaches, coughing, and irritation of eyes, nose, and throat. In addition, health science literature showed odor exacerbating pre–existing conditions such as asthma (Chang and Williams, 1986). Within the tradition of livestock odor research, Warner et al. (1990) examined emissions from a swine facility in Michigan in order to assess the intensity and distance that odor traveled in creating a nuisance, or quality of life, problem. Moreover, embedded in this work was a health survey that recorded health symptoms among neighbors as an early indication that something beyond an unpleasant nuisance was involved. These early studies reveal that odor has a tangible physical property that elicits a physiological response. Moreover, odor is only one dimension of the panoply of swine CAFO emissions, which include over 160 identified compounds (see Ritter, 1989; and overview odor discussion in Melvin et al., 1996). Consequently, this early research may have mistakenly assumed that odor was the primary constituent of CAFO emissions exposure responsible for reported health symptoms.

In 1995, Dr. Susan Schiffman and colleagues at Duke University published the first research specifically focused on the negative health effects of exposure to emissions among neighbors of swine CAFOs. The results were based on a matched control study examining the psychological effect of odors from commercial swine operations in North Carolina (Schiffman et al., 1995). Researchers administered a standardized mood states (POMS) and total mood disturbances (TMD) scale to 44 neighbors of commercial swine operations and 44 controls not living near such operations who were matched based on age, gender, education, and race. Results showed that the neighbors subjected to odors scored significantly higher on both scales, exhibiting higher rates of tension, depression, anger, and fatigue than did the control group. Elsewhere, Schiffman describes a variety of mechanisms that explain how odor can have a deleterious human health effect, including a physiological pathway between the olfactory lobe and the immune system, which directly implicate odor as a health risk (Schiffman et al., 1998).

As a follow–up to the Schiffman study, Thu et al. (1997) published the results of a control study based on physical and psychological health data from 18 neighbors living within a two–mile radius of a 4,000–sow swine confinement production facility. They compared the results with data from 18 demographically comparable rural residents who did not live near concentrations of livestock in Iowa. The results indicated that the neighbors of the swine operation reported significantly higher rates of four clusters of symptoms previously documented to represent toxic or inflammatory effects of the respiratory tract among confinement workers. One cluster reported by swine CAFO neighbors includes symptoms such as coughing, sputum production, breath shortness, chest tightness, and wheezing. A second cluster includes symptoms of nausea, weakness, and feelings of dizziness. A third cluster consists of headaches and plugged ears, while a fourth cluster encompassed symptoms of a runny nose, scratchy throat, and burning eyes. Most notable is the fact that for the first time the configuration of respiratory symptoms among neighbors was documented to be consistent with the scientifically well–established pattern of respiratory health problems among swine confinement workers discussed previously.

In April of 1998, a scientific workshop on odors and health problems was held at Duke University and spearheaded by Dr. Susan Schiffman. The workshop, in which I participated, was sponsored by Duke University, the Environmental Protection Agency, and the National Institute on Deafness and Other Communication Disorders and brought together nearly 50 experts from a broad array of scientific fields to assess the current state of scientific knowledge concerning the health consequences of odor exposures, particularly odors generated by large livestock operations. The results, published two years later (see Schiffman et al., 2000), revealed three general paradigms whereby ambient odors can indeed produce health problems: (1) physiological irritation from emission levels causing toxicological effects in which odor is simply a marker, (2) symptoms produced by malodors but without concomitant toxicological irritation, and (3) odors as part of a mixture of co–pollutants. In paradigms one and three, the odor serves as a signal that other ambient emission elements are creating the health symptoms. The over–riding conclusion of the workshop was that odors and their interconnected airborne emission partners can cause health problems:

> "Our current state of knowledge clearly suggests that it is possible for odorous emissions from animal operations, wastewater treatment, and recycling of biosolids to have an impact on physical health. The most frequently reported symptoms attributed to odors include eye, nose, and throat irritation, headache, nausea, hoarseness, cough, nasal congestion, palpitations, shortness of breath, stress, drowsiness, and alterations of mood" (Schiffman et al., 2000, p. 57).

In June of 1998, the same year as the Duke conference, the National Center for Environmental Health, Centers for Disease Control and Prevention, responded to growing nationwide concerns expressed through state health agencies about the environmental and public health consequences of CAFOs by sponsoring their own scientific workshop, "Public Health Issues Related to Concentrated Animal Feeding Operations," in which I also participated. The scientific workshop brought together over 35 experts and divided them into two groups to focus on water and air contamination. The CDC workshop produced a report consistent with the Duke results, indicating that exposure to odor–related emissions from large–scale livestock facilities can result in health problems for neighbors. The group of scientists focusing

on CAFO–related air contamination agreed that "adequate evidence currently exists to indicate airborne emissions from large–scale swine facilities constitute a public health problem" (CDC, 1998, p. 30).

In 1999, a landmark study funded by the North Carolina Department of Health and Human Services, Division of Public Health, was released and later published in *Environmental Health Perspectives* (Wing and Wolf, 2000). This was the largest study to date examining the health and quality of life of residents living in proximity to large swine operations. The researchers examined three different rural communities, which included residents living near a 6,000–swine CAFO, residents living near two intensive cattle operations, and a control area where residents lived at least two miles from any livestock operation that used a liquid manure system. After controlling for age, gender, smoking, and work, the findings among the 155 participants were consistent with Thu et al. (1997) and Schiffman et al. (1995), as well as with the two 1998 scientific workshops, in revealing elevated health problems among residents living in proximity to the swine CAFO. There were significantly elevated rates of headaches, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes among neighbors of the swine operation.

The results of the Wing and Wolf (2000) study are notable for two reasons. First, the results indicate that reporting bias was not a problem. It is clear that participants in this study, as well as in the comparable study by Thu et al. (1997), could be subject to reporting bias because residents may simply want to provide negative responses to the pork industry. However, if that is the case, then it is extremely difficult to explain the highly selective nature of reported symptoms and the fact that participants chose not to report elevated rates of all symptoms when given the opportunity. That did not occur in either the Thu et al. (1997) study or the Wing and Wolf (2000) research. Indeed, in the Wing and Wolf research, many of the reported symptoms were similar in all three of their research groups in North Carolina. Second, the configuration of reported symptoms is consistent with well–documented symptoms among swine confinement workers. It appears highly unlikely that two groups of swine CAFO neighbors would stumble upon a consistent set of symptoms that match symptoms among swine confinement workers.

Further evidence for health problems among swine CAFO neighbors comes from the Bureau of Epidemiology within the Utah Department of Health (Keller and Ball, 2000). In 1993, one of the largest hog operations in the country began construction near the town of Milford (population 1,305) in Beaver County, southern Utah. In the following two years, the facility was populated with pigs whose sow base reached 44,000 (with a reported target of 120,000 total sows). At the request of the Southwest Utah District Health Department, Utah's Bureau of Epidemiology responded to concerns about increases in diarrheal and respiratory disease among nearby Milford residents. A retrospective study of hospital discharge records from nearby Milford was compared with two comparison populations in the region, in addition to statewide averages for Utah as a whole. The investigation examined whether hospital discharge records from Milford indicated higher rates of respiratory and diarrheal illness during the years 1992 to 1998 (when the swine CAFO was constructed and became operational) compared to other communities in the region and the state as a whole. The findings demonstrated an increase in both diarrheal and respiratory illness cases in Milford during the period from 1992 to 1998 when the swine CAFO became operational. For example, Milford experienced a quadrupling of hospital diarrheal case rates, from 88 per 10,000 in 1992, to 409 per 10,000 in 1997. During the same period, Milford reflected a tripling of hospital cases involving respiratory illness, from 159 cases per 10,000 in 1992, to 517 cases per 10,000 in 1997. Moreover, the

incidence rates were significantly higher than those found in comparison populations and in the state as a whole, leading the authors to conclude: "The results of this investigation found evidence that suggests elevated incidence of diarrheal and respiratory illness in Milford as compared to Parowan, Panuitch, and the State of Utah during the time period of 1992 through 1998" (Keller and Ball, 2000, p. 7). The investigators do not ascribe causation to the neighboring swine CAFO, but the implication is clear.

The evidence is mounting that a public health problem appears to be present for not only swine confinement workers but for neighbors of swine CAFOs as well (Thu, 1998). These studies, as well as federal and state reports, independently and consistently show that neighbors, including farmer neighbors, of large-scale swine production are experiencing abnormally high rates of health problems compared with other populations. Moreover, the health symptoms experienced by neighbors are consistent with, and similar to, the types of health symptoms that have been well documented among interior confinement workers. However, in contrast to occupational health research among CAFO workers, comparable exposure studies investigating potential toxicants and their relationship to neighbor health problems are notably lacking.

There are clues from the occupational health literature, the odor research, and investigations in other industries that suggest the direction for future human exposure research. Among the airborne elements to examine are hydrogen sulfide, volatile organic compounds (VOCs), dusts, endotoxin, and perhaps ammonia, in addition to the irritant dimension of odors (see Reynolds et al., 1997). It should be noted that many of these ingredients have an interactive and synergistic effect, such as gas molecules attaching to dust particles.

While the likely culprit is some combination of emission constituents, exposure to hydrogen sulfide merits particularly attention. Hydrogen sulfide is a known toxic gas produced by the anaerobic digestion of protein in swine CAFOs. Chronic or acute occupational exposure to hydrogen sulfide concentrations near or above 500 ppm is known to result in Acute Respiratory Distress Syndrome (ARDS) or pulmonary edema among swine confinement workers, which can be fatal. However, less attention has been devoted to examining the consequences of longer-term exposure to low levels of hydrogen sulfide. In the oil refinery industry, exposure to low levels of hydrogen sulfide produces symptoms such as shortness of breath, headache, eye irritation, small-airway obstruction, and diarrhea (Kilburn and Warshaw, 1995). Moreover, longer-term exposure to low levels of hydrogen sulfide can result in permanent neurological dysfunction with accompanying physiological damage (Kilburn, 1993, 1997). This threat has resulted in six states establishing hydrogen sulfide or reduced sulfur standards. Hydrogen sulfide monitoring of swine CAFOs in Minnesota by the Minnesota Pollution Control Agency (MPCA) confirms the relevance of research on chronic exposure to low levels of hydrogen sulfide (Sullivan, 1999). The MPCA data reveal that swine CAFOs can emit hydrogen sulfide onto neighboring property at levels that exceed World Health Organization recommended standards (Roth, 1993). Moreover, recent work prepared in conjunction with Minnesota policy planning provides a comprehensive review of livestock emission constituents and their potential human health consequences (Earth Tech, Inc., 2001).

Discussion

The decade following the release of "Agriculture at Risk: A Report to the Nation" (Merchant et al., 1989) witnessed a dramatic change in the structure of the livestock industry, particularly the swine sector. The rapid industrialization of pork production produced a generation of confined production facilities at a scale never before experienced in the U.S. The enormous size of these operations has given rise to the necessity of moving beyond a traditional odor–based research paradigm and toward a more encompassing public health research paradigm. Nearly three decades of research on the interior environment of swine CAFOs and the well–documented exposure consequences for confinement workers provide important direction for research on external emissions to better understand the health consequences for CAFO neighbors. Recent research and results from federally sponsored scientific symposia consistently indicate that neighbors of swine CAFOs can experience health problems at significantly higher rates than controlled comparison populations. Moreover, such problems can be created by several different CAFO emission constituents acting alone or synergistically. It should be noted that odor is but one component of emissions and can both create problems by itself and act as a marker for the presence of irritant toxins. Symptoms experienced by swine CAFO neighbors are generally oriented toward irritation of the respiratory tract and are consistent with the types of symptoms that have been well documented among swine confinement workers. However, additional exposure assessment research is required to elucidate the relationship of reported symptoms among swine CAFO neighbors and emissions from CAFOs.

Research is currently underway and progress is being made toward identifying emission elements potentially responsible for health problems. However, much research continues to be technically focused, with lesser attention on the people involved, particularly from the neighbors' standpoint. Many of these issues involve rural quality of life expectations and considerations that can and should be investigated in concert with technical research. Indeed, common law nuisance and state air pollution statutes may focus more directly on the experiences, expectations, and quality of life norms of neighbors than technical measurements of various emission constituents. Hence, we need not only additional epidemiological and exposure assessment research, we also need additional anthropological and social science research to more fully understand quality of life norms and expectations among rural residents.

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Attachment A4

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The Effect of Environmental Odors Emanating From Commercial Swine Operations on the Mood of Nearby Residents

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ABSTRACT: The effect of environmental odors emanating from large-scale hog operations on the mood of nearby residents was determined using the POMS (Profile of Mood States). The scores for six POMS factors and the TMD (total mood disturbance score) for 44 experimental subjects were compared to those of 44 control subjects who were matched according to gender, race, age, and years of education. The results indicated a significant difference between control and experimental subjects for all six POMS factors and the TMD. Persons living near the intensive swine operations who experienced the odors reported significantly more tension, more depression, more anger, less vigor, more fatigue, and more confusion than control subjects as measured by the POMS. Persons exposed to the odors also had more total mood disturbance than controls as determined by their ratings on the POMS. Both innate physiological responses and learned responses may play a role in the impairment of mood found here.

KEY WORDS: Odors, Mood, Pollution, Swine, Psychological effects, Brain-immune connections.

INTRODUCTION

Odors have always been associated with livestock and poultry production [24,55,72,78,79,86,88]. However, odors have recently become a major challenge for the livestock industry due to the present trend toward intensive livestock operations in which large numbers of animals are confined on small areas of land [8,19,51,69,120,122–124,127]. Environmental odors can have a considerable impact upon a population's general well-being, affecting both physiological and psychological status [93,103,128]. Miner [70] concluded that unpleasant odors can affect well-being by "eliciting unpleasant sensations, triggering possible harmful reflexes, modifying olfactory function and other physiological reactions." He also reported that annoyance and depression can result from exposure to unpleasant odors along with nausea, vomiting, headache, shallow breathing, coughing, sleep disturbances, and loss of appetite. Odorous compounds associated with livestock production that are at low concentrations but above odor thresholds are still likely to generate complaints [18,52].

Neutra et al. [77] studied people living near hazardous waste sites, and found that those complaining of odors had a higher number of symptoms than those who did not complain, regardless of proximity to the site. Shusterman [103] reviewed several studies [e.g., 4,37,47,95–97] in which there was a direct relationship between nontoxicological odors and symptomatology. In a variety of settings (municipal, agricultural, and industrial) where airborne toxicants were negligible and odors had been complained about, there was a strong relationship between reported symptoms and odor exposure.

The sources of the odors from swine operations include ventilation air released from swine buildings, waste storage and handling systems including lagoons, and land application of manure to fertilize fields [15]. The odors are produced by a mixture of fresh and decomposing feces, urine, and spilled feed. The more objectionable odors appear to result from anaerobic microbial decomposition of the feces [90]. A broad range of compounds has been identified in livestock manure including volatile organic acids, alcohols, aldehydes, amines, fixed gases, carbonyls, esters, sulfides, disulfides, mercaptans, and nitrogen heterocycles [30,70,71,73,104]. It is likely that the mixture of compounds rather than a single component contributes to the mood changes measured here.

A variety of techniques for reducing odor have been evaluated, but overall the results have been disappointing [123]. Aerobic treatment has been found to be the most effective method to date for deodorizing pig slurry [2,9,11,54,105–107,127]. Odorous compounds can be carried in a plume, and the concentration of these compounds in the plume may not be significantly reduced at distances of 750–1500 feet or more downwind from a source [36]. Dispersion models have been developed to predict the peak and mean concentrations of odors and environmental air pollutants at various distances from the source [20,36,46,80], and complaint patterns at a variety of distances from an odor source have been studied [21].

The purpose of the present study was to use a well-standardized scale to quantify objectively the moods of people living near large-scale hog operations who are exposed to odors. The Profile

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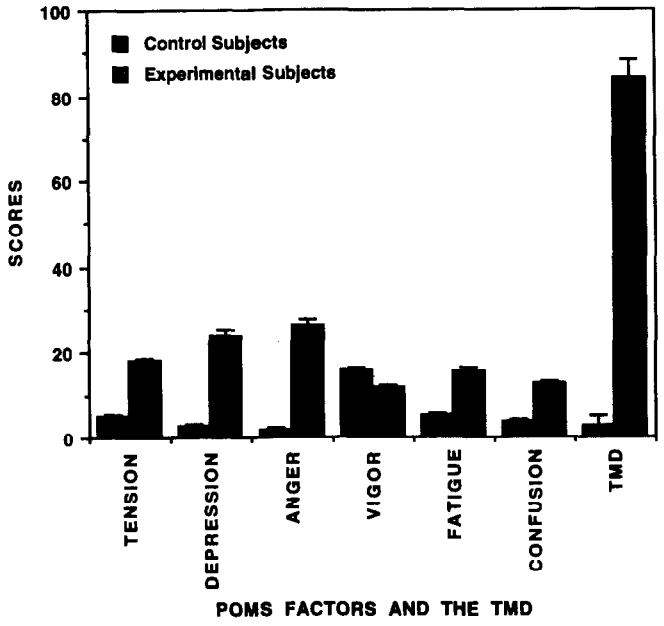

FIG. 1. Mean POMS scores of each factor and the total mood disturbance score (TMD) for experimental and control subjects.

of Mood States questionnaire [65,66] was used to assess mood in persons living near swine operations and in control subjects. This scale has been used extensively in many situations including previous studies that evaluated the effect of pleasant odors on mood [98,99]. The study of mood in persons exposed to odors is important because negative mood has been found to play a role in immunity [16,81,111,125] and can potentially affect subsequent disease.

METHOD

Subjects

Forty-four experimental (persons living near hog operations) and 44 control subjects participated in the study; all of the subjects were residents of North Carolina. The subjects in the two groups (control and experimental) were matched according to gender, race, age, and years of education. Twenty-six subjects in each group were female, and 18 subjects were male. The mean age of the experimental group was 52.0 ± 13.4 years, and the mean age of the control group was 51.7 ± 8.3 years. The experimental group had an average of 12.8 ± 3.3 years of education, and the control group had an average of 13.0 ± 3.1 years of education. The majority of subjects in both groups were employed as skilled laborers. The groups were also matched for the number of chronic illnesses that they had experienced; 14 subjects in each group suffered from allergies. The experimental group lived an average of 5.3 ± 6.5 years near hog operations, with a maximum of 27 years and a minimum of 8 months.

Materials

Subjects in both groups signed a consent form and filled out a general information questionnaire that asked demographic, medical, and dietary information. Mood ratings were obtained from all subjects by filling out Profile of Mood States questionnaires (POMS). The POMS was chosen to measure the impact of the hog odors on mood because it has been shown to be sensitive to transient mood shifts [65,66]. There are 65 adjectives/feelings on the POMS, most of which may be grouped into one of six factors: tension/anxiety, depression/dejection, anger/hostility, vigor/activity, fatigue/inertia, and confusion/bewilderment. Each feeling is rated on a scale from 0 (not at all) to 4 (extremely). The feelings for each factor were added together, according to the POMS manual, to get a total score for that factor. The totals for each factor were then added together, with the vigor/activity factor weighted negatively, to derive a total mood disturbance score (TMD).

Procedure

At the beginning of the study, all subjects filled out the consent form as well as the general information questionnaire. Experimental subjects were asked to complete one POMS questionnaire per day on 4 days when the hog odor could be smelled. The 4 days did not have to be consecutive, and subjects had as long as needed to complete all four POMS questionnaires. Control subjects were asked to complete one POMS per day for 2 days. All subjects were asked to complete the POMS based upon how they recently had been feeling, including at that particular time.

RESULTS

Figure 1 shows the means and standard errors for the experimental group vs. the control group for all POMS factors and the TMD. An analysis of variance was performed to determine if there were any main effects or interactions between group (control or experimental) and gender for each POMS factor and the TMD. Subjects were nested within group and gender. Table 1 gives the results of the analysis. There was a significant difference (at $p < 0.0001$ level) between the control group and the experimental group for all of the POMS factors as well as the TMD. The experimental group had significantly worse scores than the control group for every factor and the TMD. There was a significant main effect of gender for the anger factor, $p < 0.01$, and a significant gender × group interaction for the confusion factor, $p < 0.005$. Males had significantly higher (worse) anger scores than the females. For the confusion factor, scores for experimental males were significantly higher than those for experimental females and control males and females; scores for ex-

TABLE 1

RESULTS OF THE ANALYSIS OF VARIANCE

Effect	Tension	Depression	Anger	Vigor	Fatigue	Confusion	Total Mood Disturbance Score
Group	*	*	*	*	*	*	*
Gender			*				
Group × gender						*	
Subject (group, gender)	*	*	*	*	*	*	*

* Significant at $\alpha = 0.05$ level.

perimental females were significantly higher than those of control males and females. Only scores for control males and control females were not significantly different from each other.

DISCUSSION

The main finding of this study is that persons living near the swine operations who experienced the odors had significantly more tension, more depression, more anger, less vigor, more fatigue, and more confusion than control subjects as measured by the Profile of Mood States (POMS). In addition, persons exposed to the odors also had more total mood disturbance than controls as determined by their ratings on the POMS. These findings are consistent with previous studies in which odors of varying hedonic properties have been found to affect mood [7,32,93,98,99,103,128]. In other settings, odors have also been reported to affect cognitive performance [57,62] and physiological responses including heart rate and electroencephalographic patterns [56,58–61,64].

Possible Causes of Altered Mood

A variety of factors may play a role in the altered mood of residents who are exposed to odors from nearby swine operations. These factors include: a) the unpleasantness of the sensory quality of the odor; b) the intermittent nature of the stimulus; c) learned aversions to the odor; d) potential neural stimulation of immune responses via direct neural connections between odor centers in the brain and lymphoid tissue; e) direct physical effects from molecules in the plume including nasal and respiratory irritation; f) possible chemosensory disorders; and g) unpleasant thoughts associated with the odor.

At moderate to high odor intensities, most persons rate the quality of the odor from the swine operations as unpleasant. The odor is not only perceived while breathing outdoor air but can also be perceived within the homes of nearby residents due to air circulation through open windows and air conditioning systems. The odorant molecules can be absorbed by clothing, curtains, and building materials which act as a sink; the molecules are then released slowly over a period of time from textiles and other materials after the plume has passed the house increasing the temporal exposure to the odor. The intermittent nature of the odors may also be a factor in the mood of persons living near swine operations. Studies of noise have shown that intermittent stimuli produce more arousal and are more likely to affect performance negatively than constant noise [22]. This is due in part to feelings of lack of control over the timing of unwanted transient stimuli. Differences in responses to irregular noise and predictable noise are not only found in humans but in animals as well [27].

Learning (via conditioning) may also play a role in the psychological and physical effects from odors. Conditioned aversions to odors are well-documented in the scientific literature [31,38,44,67,75,119]. Aversive conditioning can occur if environmental odors are associated with an irritant or other toxic chemicals such as pesticides [103]. In addition, conditioned alterations in immune responses using chemosensory (smell and taste) stimuli provide strong evidence for functional relationships between chemosensory centers in the brain and the immune system [1]. Both conditioned immunosuppression and immunoenhancement have been reported using chemosensory stimuli as the conditioned stimulus [1,31,42,43,109,110].

There is a potential for unpleasant odors to influence physical health without involvement of learning or conditioning due to the direct anatomical connections between the olfactory system and the immune system. Brain structures broadly involved in smell [12,35,39,49,82–85,101,112,114–116] can modulate immune responses, especially via the integrated circuitry of the limbic cortex, limbic forebrain, hypothalamus, and brain stem [13,25,26,48,50,76,92,118]. These studies provide an anatomical basis for the possibility that sensory stimulation of the limbic forebrain, hypothalamus, and other odor projection areas of the brain can directly alter immune status. The links between the brain and the immune system are bidirectional [108] so that immune responses can also affect odor centers in the brain [10,94].

Components in the odorous plume may also have direct physical effects on the body. Some of the odorant molecules implicated in malodor from hog farms can cause nasal and respiratory irritation [15,23,29,70,103]. Nasal irritation has been shown to elevate adrenalin [3] which may contribute to feelings of anger and tension. The volatile organic compounds (VOCs) responsible for odors may also be absorbed directly by the body (into the bloodstream and fat stores) via gas exchange in the lungs. Many VOCs that are inhaled into the lungs are known to reach blood and adipose tissue [4,6,53,63,126]. Persons who have absorbed odorants through the lungs can sometimes smell the odor for hours after exposure due to slow release of the odorants from the bloodstream into expired air activating the olfactory receptors. Volatile organic compounds are well known to be eliminated in breath after exposure [89,121], and methods for measuring VOCs in breath have been described [87,89,117]. It is also theoretically possible for some compounds in the plume to be transmitted to the brain via olfactory neurons because a range of agents have been found to reach the brain through the nasal route [28,33,45,74,91,102]. Endotoxin, a component of bacteria, found in the swine house air environment [29], may also be present in the plume. Persons with olfactory dysfunction caused by factors unrelated to swine odor such as concurrent medical conditions, drugs they are taking, or pesticide exposure [100], may find the odor even more objectionable due to their abnormal smell functioning.

Finally, odors may alter mood because they are associated with unpleasant thoughts. Some persons consider the smell from hog farms a taboo odor, which they should not have to endure. For other persons, the odors generate environmental concerns, fear of loss of use and value of property, or a conviction that odors interfere with their enjoyment of life and property. Livestock odors may also be considered inappropriate in certain environments. Odor complaints have been reported to be most frequent among new, large, or recently expanded facilities that are located near existing residences or shopping areas [70,113]. Part of the motivation for odor complaints may be the increased awareness of other environmental agents, such as tobacco smoke, which is malodorous and is considered dangerous to one's health.

Lack of Legislation to Monitor Odor Levels

Odors are not regulated by the Clean Air Act because they are generally regarded as nontoxic [15]. In addition, nonfederal legislation for controlling odors from swine operations is imprecise or lacking in many states. For example, North Carolina Administrative Code Title 15A-02D.0522(c) specifies that "a person shall not cause, allow, or permit any plant to be operated without employing suitable measures for the control of odorous emissions including wet scrubbers, incinerators, or such other devices as approved by the Commission." This regulation is subjective because it gives no provision for either emission standards or ambient air standards. Under this regulation, it appears that as long as a plant has suitable control devices, it is lawful for them to emit offensive odors. In addition, it is unclear what type of operation is to be considered a plant. In contrast, Connecticut's laws on odor emissions set specific standards, as shown in Table

TABLE 2

ACCEPTANCE LIMITS FOR ODORS (FROM 17)

Chemical	ppm by Volume
Acetaldehyde	0.21
Acetic acid	1.0
Acetone	100.0
Acrolein	0.21*
Acrylonitrile	21.4*
Allyl chloride	0.47
Amine, dimethyl	0.047
Amine, monomethyl	0.021
Amine, trimethyl	0.00021
Ammonia	46.8*
Aniline	1.0
Benzene	4.68
Benzyl chloride	0.047
Benzyl sulfide	0.0021
Bromine	0.047
Butyric acid	0.001
Carbon disulfide	0.21
Carbon tetrachloride (chlorination of CS_2)	21.4*
Carbon tetrachloride (chlorination of CH_4)	100.0*
Chloral	0.047
Chlorine	0.314
Dimethylacetamide	46.8*
Dimethylformamide	100.0*
Dimethyl sulfide	0.001
Diphenyl ether	0.1
Diphenyl sulfide	0.0047
Ethanol (synthetic)	10.0
Ethyl acrylate	0.00047
Ethyl mercaptan	0.001
Formaldehyde	1.0
Hydrochloric acid gas	10.0*
Hydrogen sulfide gas	0.00047
Methanol	100.0
Methyl chloride	(above 10 ppm)
Methylene chloride	214.0*
Methyl ethyl ketone	10.0
Methyl isobutyl ketone	0.47
Methyl mercaptan	0.0021
Methyl methacrylate	0.21
Monochlorobenzene	0.21
Monomethylamine	0.021
Nitrobenzene	0.0047
Paracresol	0.001
Paraxylene	0.47
Perchloroethylene	4.68
Phenol	0.047
Phosgene	1.0*
Phosphine	0.021
Pyridine	0.021
Styrene (inhibited)	0.1
Styrene (uninhibited)	0.047
Sulfur dichloride	0.001
Sulfur dioxide	0.47
Toluene (from coke)	4.68
Toluene (from petroleum)	2.14
Toluene diisocyanate	2.14*
Trichloroethylene	21.4

* Exceeds the Threshold Limit Value adopted by the American conference of Industrial Hygienists for 1971.

2 [17]. Similarly, in the Netherlands, regulations are based on accurate records of manure production and bookkeeping, and violations are considered a criminal offense [14].

Regulations need to be established in all 50 states because animal wastes contain high levels of volatile organic compounds that can produce strong odors. The annual production of animal manure in the US in 1987 was estimated at 1.5 billion tons per year, which is enough to apply one ton per acre on each of the 1.9 billion acres of the continental US [14].

Persons exposed to high levels of odor from agricultural sources generally use nuisance laws to protect their rights. However, there are many caveats in nuisance laws that consider a) which party was there first; b) the character of the neighborhood; c) the reasonableness of the use of the land; and d) the nature and degree of the interference [40]. In addition, most states have right-to-farm statutes that supersede nuisance laws in some circumstances [40]. Strong support against nuisance suits involving agriculture is not specific to the United States but is found in the laws of many countries [5]. Suits against agricultural activities based on odor nuisance are harder to prove than those based on water pollution [68]. In addition, nuisance claims fall under state laws, while suits on water pollution are most frequently filed in federal courts.

Conclusion

Odors from swine operations have a significant negative impact on mood of nearby residents. Methods must be found to lower the concentrations of compounds responsible for the odors so that swine operations do not affect the emotional lives of residents in the local vicinities. This may involve legislation that sets standards for odor. In addition, technological solutions must be found to reduce the concentrations of the offending compounds.

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Attachment A5

Intensive Livestock Operations, Health, and Quality of Life among Eastern North Carolina Residents

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People who live near industrial swine operations have reported decreased health and quality of life. To investigate these issues, we surveyed residents of three rural communities, one in the vicinity of an approximately 6,000-head hog operation, one in the vicinity of two intensive cattle operations, and a third rural agricultural area without livestock operations that use liquid waste management systems. Trained interviewers obtained information about health symptoms and reduced quality of life during the previous 6 months. We completed 155 interviews, with a refusal rate of 14%. Community differences in the mean number of episodes were compared with adjustment for age, sex, smoking, and employment status. The average number of episodes of many symptoms was similar in the three communities; however, certain respiratory and gastrointestinal problems and mucous membrane irritation were elevated among residents in the vicinity of the hog operation. Residents in the vicinity of the hog operation reported increased occurrences of headaches, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes as compared to residents of the community with no intensive livestock operations. Quality of life, as indicated by the number of times residents could not open their windows or go outside even in nice weather, was similar in the control and the community in the vicinity of the cattle operation but greatly reduced among residents near the hog operation. Respiratory and mucous membrane effects were consistent with the results of studies of occupational exposures among swine confinement-house workers and previous findings for neighbors of intensive swine operations. Long-term physical and mental health impacts could not be investigated in this study. *Key words*: African Americans, agricultural health, air pollution, epidemiology, respiratory conditions, rural health. *Environ Health Perspect* 108:233–238 (2000). [Online 8 February 2000]
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Industrial hog production has grown rapidly in North Carolina since the early 1980s. Once characterized by relatively small independently owned farms scattered across the state, hog production in North Carolina is now concentrated in the coastal plain region, under the domain of large corporate growers, and dominated by large-scale intensive operations (*1,2*). Persons who live near large hog operations have reported reduced quality of life as well as health problems related to airborne emissions from animal confinement houses, open waste lagoons, and spray fields (*3–8*). Airborne emissions include hydrogen sulfide, ammonia, dusts, endotoxins, and complex mixtures of volatile organic compounds. Health effects from environmental exposures could occur through inflammatory, immunologic, irritant, neurochemical, and psychophysiologic mechanisms (*5*).

In contrast to the many studies of occupational exposures of swine confinement-house workers (*9–25*), only a few field studies have investigated the health effects of lower level environmental exposures. In a study of residents near hog facilities in North Carolina, Schiffman et al. (*26*) reported that persons exposed to odors from intensive hog operations experienced "more tension, more depression, more anger, more fatigue, and more confusion" than a group of unexposed persons. A study in Iowa (*7*) compared physical and mental health symptoms among people residing within a 2-mile radius of a 4,000-head swine operation and a control group in an area with no intensive livestock operation. Those who lived in the vicinity of the intensive hog operation reported higher frequencies of 14 of 18 physical health symptoms, especially respiratory symptoms. The Iowa study did not find an excess of mental health symptoms but, in contrast to the North Carolina study (*26*), it was not designed to evaluate symptoms at the time that odors were present.

The present study addressed a number of issues raised by previous research. Unlike studies of volunteers, the sample was drawn systematically from defined populations. To increase the levels of participation and prevent exclusions based on literacy or the ability to participate in a longer study, we did not ask participants to keep a diary or respond to questions at the times that airborne emissions from livestock operations were noticeable. Instead, we asked questions about the number of times that participants experienced the symptoms of interest during the previous 6 months. Because mood disturbance and mental health effects may be acute responses to the presence of odors, we focused on physical health and quality of life rather than on short-term mood changes. We achieved high levels of participation in the study by establishing cooperative relationships with local community based organizations in planning and conducting the research.

This study compared health symptoms in residents of three North Carolina communities, one in the vicinity of an intensive hog operation, one in the vicinity of two intensive cattle operations, and a third in a rural agricultural area where no livestock operations used liquid waste management systems. Although the primary motivation for the study came from an interest in airborne emissions from swine operations, the inclusion of people residing near cattle operations afforded an opportunity to examine possible health effects from a different kind of livestock, and also offered a second comparison community that may share other features common to communities with intensive livestock production.

Materials and Methods

Selection of communities. The North Carolina Division of Water Quality (Raleigh, NC) maintains a database on intensive livestock operations that use liquid waste management systems (*27*). Information on livestock operations included in the database as of January 1998 was merged with 1990 U.S. Census block group data (U.S. Census Bureau, Suitland, MD). Data for block groups, which average approximately 500 households, included information on population size, race, and poverty levels. Maps of the eastern part of North Carolina

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were prepared showing the locations of livestock operations, towns, roads, and churches. Community consultants experienced with the hog industry and the health concerns of community members met with university researchers to review the maps and choose potential study sites. Our goal was to choose three areas with similar economic and demographic characteristics where residents would be willing to participate in an interview and where existing community based organizations would be interested in working with researchers. We sought livestock areas with 80–100 households within a 2-mile radius of the livestock facility so that we would be able to obtain approximately 50 participants in each area.

The hog and cattle study areas were defined by a < 2-mile radius around the operations and each study area was contained within a single census block group. The hog operation was a feeder-to-finish facility with a head capacity of approximately 6,000, a steady-state live weight of approximately 800,000 pounds, and one lagoon. The cattle community contained two neighboring dairy operations with a combined head capacity of approximately 300, live weight of approximately 200,000 pounds, and two lagoons. The area with no intensive livestock operations extended across two block groups. Parts of two block groups were included to ensure that eligible households were at least 2 miles away from any livestock operation using a liquid waste management system. The median annual family income of the census block groups from which the study areas were chosen ranged from approximately $17,000–23,000 and the populations were between 65 and 90% African American.

All habitable dwellings in the study areas were enumerated. The location of each dwelling was noted on an enlarged area map and was assigned a unique study number. Information on street or road location and the type of dwelling was entered into a computerized database.

Questionnaire. A structured questionnaire was developed based on previous research findings and on discussions with community members who had experienced exposures from intensive livestock operations. In addition to symptoms identified by previous studies or community residents as possibly related to airborne emissions from livestock operations, we included symptoms that we did not believe would be related to airborne emissions to evaluate the possibility that residents of exposed communities might report excesses of all types of symptoms because of negative feelings about intensive livestock operations. The questionnaire was designed to obtain information about the frequency of occurrence of each symptom over the 6 months preceding the interview. Possible responses were never; rarely (once or twice over the past 6 months); sometimes (1–3 times per month); often (1 per week); and very often (twice a week or more over the past 6 months). After all of the structured questions had been asked, respondents were asked about aspects of the environment that may have affected their own health or the health of others in the household. Interviewers took notes to summarize the types of responses. At the end of the interview, participants were asked their age, occupation, household size, source of drinking water, and whether they or others in the household smoked tobacco. The interviewers recorded race, sex, and whether anyone other than the participant and interviewer were present during the interview.

Household interviews. Adults 18 years of age or older with no serious speech or mental impairment who lived in the current residence for 6 months or longer were eligible to respond to the questionnaire. The households of dairy operators who lived beside the cattle facility were excluded to avoid the complication of occupational exposures; the household of the swine facility operator was not within the 2-mile enumeration area of the facility. Interviews were conducted on Fridays and Saturdays in January and February 1999 by university-based staff. Interviewers were accompanied by a community consultant, a local resident recruited from the membership of the community based organization. The community consultant introduced the interviewer to the prospective respondent, explained the purpose and importance of the survey, and encouraged each person to participate. Interviewers were trained to administer the survey instrument systematically and uniformly to all respondents. The participant interview was conducted in a location of the participant's choosing. The questionnaire required less than 15 min to complete. The community consultant was not present for the interview unless the participant specifically asked the consultant to remain.

One adult from each household was invited to participate in the survey. Preference was given to the first person to answer the door if

Table 1. Characteristics of study households, listed by type of livestock operation.

Characteristic	Livestock operation			
	None	Cattle	Hogs	Total
Inhabited houses	104	116	92	312
Households ineligible[a]	5	2	3	10
Not home	29	44	19	92
Rescheduled or not contacted	5	14	10	29
Completed interviews	50	50	55	155
Refused	15	6	5	26
Refusal rate[b]	23.1%	10.7%	8.3%	14.4%

[a]Not living in the house for 6 months; difficulty understanding survey questions. [b]Refusal rate = completed interviews/completed interviews + refusals.

Table 2. Characteristics of respondents.

Characteristic	Livestock operation, no. (%)			
	None	Cattle	Hogs	Total
Age				
19–44 years	19 (38)	13 (26)	23 (42)	55 (36)
45–64 years	19 (38)	19 (38)	20 (36)	58 (37)
65–90 years	12 (24)	18 (36)	12 (22)	42 (27)
Race/ethnicity				
African American	45 (90)	49 (98)	48 (87)	142 (92)
White	5 (10)	1 (2)	6 (11)	12 (8)
Latino	0 (0)	0 (0)	1 (2)	1 (1)
Sex				
Female	31 (62)	33 (66)	36 (65)	100 (65)
Male	19 (38)	17 (34)	19 (35)	55 (35)
Smoking				
Yes	14 (28)	13 (26)	7 (13)	34 (22)
No	36 (72)	37 (74)	48 (87)	121 (78)
Employed outside of the home				
Yes	26 (52)	15 (30)	34 (62)	75 (48)
No	24 (48)	34 (68)	21 (38)	79 (51)
Not completed	0 (0)	1 (2)	0 (0)	1 (1)
Number in household				
1	12 (24)	8 (16)	3 (5)	23 (15)
2	21 (42)	21 (42)	20 (37)	62 (40)
3–4	12 (24)	15 (30)	15 (27)	42 (27)
5–12	5 (10)	6 (12)	17 (31)	28 (18)
Total respondents (*n*)	50 (100)	50 (100)	55 (100)	155 (100)

the person was over 18 years old and lived in the household. Those who declined to participate because the time was inconvenient were offered alternative times and the visit was rescheduled. If no one was at home, the information was recorded on the tracking form. These households were visited a second time. Households were visited sequentially using the enumeration map in approximate order of distance from the intensive livestock operation until a minimum sample size of 50 was reached. Informed consent was requested verbally by the trained interviewer.

Statistical methods. Differences in symptoms among the three communities were evaluated by comparing the average number of episodes experienced over the last 6 months for each symptom. The number of episodes over the 6 months preceding the interview was scored according to the instructions given to respondents for responding to the frequency of symptoms. A response of "never" corresponded to 0 episodes. A response of "occasionally" corresponded to two episodes. "Sometimes" corresponded to 12 episodes (2/month), "often" corresponded to 26 episodes (1/week), and "very often" corresponded to 52 episodes (2/week). Adjusted mean differences in the numbers of episodes were calculated using linear regression to control for sex, age (19–44, 45–64, or 65–90 years), respondent's smoking status (yes or no), and employment outside the home (yes or no). These variables were considered potential confounders because they may be associated with exposure to airborne emissions and experience or reporting of symptoms. Because the five response categories for the number of episodes were highly skewed, regression models were also run with the dependent variable coded as the square root of the number of episodes and as 0–4.

The ratio of the β-coefficient (adjusted mean difference in number of episodes) to its SE yields a t-value. Larger absolute values of t indicate that the livestock variable is more important for statistically predicting numbers of symptom episodes. Significance tests are not presented because exposures were not randomized in this observational study; however, t-values > 1.66 would produce a significant one-tailed test of the hypothesis that average numbers of symptoms are greater in the livestock than in the control community at $p < 0.05$. Values > 1.98 would produce a significant two-tailed test at $p < 0.05$.

Table 3. Number and percent of respondents reporting 12 or more episodes, and mean number of episodes.

	Livestock operation					
	None		Cattle		Hogs	
Symptom	No. (%)[a]	Mean[b]	No. (%)[a]	Mean[b]	No. (%)[a]	Mean[b]
Total respondents	50 (100.0)	–	50 (100.0)	–	55 (100.0)	–
Upper respiratory/sinus						
Headache	16 (32.0)	7.8	18 (36.0)	9.4	34 (61.8)	15.5
Stuffy nose/sinuses	14 (28.0)	7.2	17 (34.0)	8.8	24 (44.4)	10.2
Runny nose	8 (16.0)	3.9	10 (20.0)	5.4	16 (29.1)	8.5
Burning nose/sinuses	11 (22.0)	4.1	9 (18.0)	3.4	14 (25.5)	6.7
Sore throat	2 (4.0)	0.9	6 (12.0)	2.5	9 (16.4)	4.7
Plugged/popping ears	10 (20.0)	5.5	11 (22.0)	5.2	11 (20.0)	4.6
Scratchy throat	6 (12.0)	2.2	10 (20.4)	3.8	10 (18.2)	4.4
Lower respiratory						
Mucus/phlegm	14 (28.0)	5.9	14 (28.6)	7.2	16 (29.1)	8.5
Excessive coughing	5 (10.0)	1.8	6 (12.0)	3.7	12 (21.8)	6.3
Shortness of breath	12 (24.0)	7.0	13 (26.0)	6.1	11 (20.0)	5.5
Tightness in chest	6 (12.0)	3.0	9 (18.0)	4.9	11 (20.0)	3.9
Wheezing	8 (16.0)	4.4	7 (14.0)	3.7	9 (16.4)	3.6
Strange breathing sounds	10 (20.0)	5.2	5 (10.2)	3.0	6 (10.9)	2.3
Gastrointestinal						
Heartburn	10 (20.4)	5.2	10 (20.0)	8.1	17 (30.9)	7.1
Nausea/vomiting	7 (14.0)	3.0	7 (14.0)	4.8	15 (27.3)	5.9
No appetite	8 (16.0)	2.8	8 (16.3)	4.1	12 (21.8)	5.5
Diarrhea	2 (4.0)	1.7	4 (8.2)	1.3	10 (18.2)	4.3
Skin/eye irritation						
Burning eyes	8 (16.0)	3.8	5 (10.0)	3.4	19 (35.2)	9.4
Tearing eyes	16 (32.0)	9.5	14 (28.0)	8.7	20 (36.4)	9.3
Dry/scaly skin	10 (20.0)	4.4	11 (22.0)	7.1	12 (21.8)	7.1
Skin rash or irritation	4 (8.0)	1.6	4 (8.0)	2.0	8 (14.6)	4.0
Skin redness	1 (2.0)	1.2	0 (0.0)	0.1	4 (7.3)	1.3
Miscellaneous						
Joint/muscle pain	24 (48.0)	16.1	26 (52.0)	17.2	28 (50.9)	16.7
Unexplainably tired	19 (38.0)	12.8	19 (38.0)	10.5	23 (41.8)	13.7
Blurred vision	15 (30.0)	8.8	9 (18.0)	5.4	16 (29.6)	9.7
Dizzy/faint	11 (22.0)	5.5	10 (20.0)	5.3	12 (21.8)	4.1
Hearing problems	7 (14.0)	7.4	5 (10.0)	2.0	6 (10.9)	2.7
Chest pain	10 (20.0)	3.4	6 (12.0)	1.6	6 (10.9)	2.7
Fever/chills	5 (10.0)	2.3	2 (4.0)	1.2	5 (9.3)	1.9
Fainted	0 (0.0)	0.04	0 (0.0)	0.04	1 (1.9)	1.0
Quality of life						
Can't open windows	7 (14.3)	3.2	4 (8.2)	1.8	31 (57.4)	18.5
Can't go outside	5 (10.0)	2.1	3 (6.0)	1.2	30 (55.6)	15.4

[a]Number and percentage of respondents answering sometimes (1–3 times/month), often (1/week), and very often (≥ 2 times/week over the past 6 months). [b]Average number of episodes per person over 6 months.

Results

Table 1 shows the numbers of households enumerated and surveyed. Enumerated households were within 2 miles of an intensive livestock operation in the cattle and hog communities. In the control area, enumerated households were > 2 miles from an intensive livestock operation in the control area. Approximately 100 households were enumerated in each area. Fifty interviews were completed in the cattle and control communities, and 55 interviews were completed in the hog community. The refusal rate was 23.1% in the control community, 10.7% in the cattle community, and 8.3% in the hog community.

Characteristics of the respondents are shown in Table 2. The cattle community had the largest proportion of respondents older than 65 years of age. All three communities were predominantly African American. Approximately two-thirds of the participants were female. The proportion of respondents who reported smoking tobacco was lower in the hog community than in the other two communities, whereas the proportion employed outside of the home was higher. None of the study participants reported that they worked in the livestock industry. Household size was largest in the hog community.

Responses to the symptom questions in the three communities are shown in Table 3. The symptoms were categorized in six groups: upper respiratory and sinus, lower respiratory, gastrointestinal, skin and eye irritation, miscellaneous, and quality of life. For each community we tallied the number of persons who answered "sometimes," "often," or "very often" corresponding to ≥ 12 episodes during the 6-month period. Table 3 also shows the percentage of "sometimes" or more often and the average number of episodes for the 6 months.

Most of the percentages in Table 3 are < 50; the majority of participants responded "never" or "occasionally" to most of the symptom questions. Among the upper respiratory and sinus conditions, the percentage of respondents reporting ≥ 12 episodes was the largest in the hog community except for

plugged ears and scratchy throats. Percentages were generally intermediate in the cattle community. The percentage of respondents reporting ≥ 12 episodes was generally smaller for lower respiratory, gastrointestinal, and skin or eye irritation symptoms. Percentages were the highest in the hog community for all four gastrointestinal symptoms. In all three communities, more than one-third of the participants reported experiencing joint or muscle pain and unexplained tiredness ≥ 12 times. By far the biggest differences between the communities were seen in the quality-of-life questions. Over half of the respondents in the hog community, as compared to less than one-fifth in the other two communities, reported not being able to open windows or go outside, even in nice weather, ≥ 12 times over the last 6 months.

Table 4 presents the results of the linear regression showing differences between the average number of episodes in each livestock community as compared to the community with no intensive livestock. Table 4 shows the difference in the mean number of episodes adjusted for sex, age, smoking, and work outside the home; the SE of the β-coefficient; and the *t*-value, which is the ratio of the β-coefficient to its SE (see "Statistical Methods"). The adjusted mean differences for the cattle community were generally small, with lower mean scores (negative β-coefficients and *t*-values) for many symptoms in the cattle as compared to the control community. Only episodes of excessive coughing and heartburn occurred on average > 2 times more in the cattle than in the control community (β > 2), and the *t*-values for these differences were only approximately 1.0. All of the symptoms in the miscellaneous category appeared less frequently in the cattle than in the control community. Hearing problems showed the largest difference in adjusted mean episodes, although this is based on a small number of people in the higher categories (Table 3).

In contrast, there were many mean differences of more than two episodes for the hog as compared to the control community. The average number of episodes was the most consistently elevated for upper respiratory and sinus conditions, gastrointestinal conditions, and skin or eye irritation. *t*-Values for headache, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes showed that residence in the hog community was an important predictor of these physical health symptoms. In contrast, none of the miscellaneous symptoms showed important excesses in the hog community.

Responses to the quality-of-life questions were very different in the control and cattle communities as compared to the hog community. The adjusted number of episodes during which participants could not open windows or go outside even in nice weather differed little for the cattle and control communities, whereas excesses of approximately 13–15 episodes were seen in the hog as compared to the control communities. *t*-Values for these β-coefficients were large.

To evaluate the sensitivity of the regression results to the coding of the dependent variable, the models shown in Table 4 were rerun using values of the square root of the number of episodes and as 0, 1, 2, 3, and 4. *t*-Values for differences between the hog community and the control community were larger in these models. The *t*-value for nausea/vomiting was 1.61 with the original metric, 2.68 using the square root of the number of episodes, and 2.88 with a coding of 0–4. To consider whether elevated gastrointestinal symptoms in the hog community might be related to well contamination, the models shown in Table 4 were rerun for the four gastrointestinal symptoms including a variable for well versus municipal water supply. The coefficients for well water were small and had little influence on the estimates of differences between livestock and control communities.

Responses to open-ended questions about how the environment around the home affected the life or health of the respondent or members of her household are shown in Tables 5 and 6. Responses that were given by two or more persons in the study are shown. Most participants from the control and cattle communities had little to report in response to these open-ended questions, although eight participants in the cattle community mentioned livestock odor. In contrast, livestock odor was noted as a problem for many residents of the hog community and for members of the residents' households.

Discussion

To our knowledge this is the first population-based study of physical health symptoms and

Table 4. Linear regression results: average number of episodes in two livestock communities as compared to a community with no intensive livestock.

	Livestock operation					
	Cattle			Hogs		
Symptom	β[a]	SE[b]	*t*-Value	β[a]	SE[b]	*t*-Value
Upper respiratory/sinus						
Headache	1.57	3.02	0.52	7.62	2.94	2.60
Stuffy nose/sinuses	1.33	2.86	0.47	2.97	2.79	1.06
Runny nose	1.26	2.44	0.52	5.18	2.37	2.18
Burning nose/sinuses	-0.42	2.19	-0.19	1.99	2.13	0.93
Sore throat	1.71	1.52	1.12	3.64	1.48	2.45
Plugged/popping ears	-1.07	2.28	-0.47	-0.79	2.22	-0.35
Scratchy throat	1.63	1.49	1.09	2.09	1.45	1.44
Lower respiratory						
Mucus/phlegm	0.56	2.65	0.21	3.91	2.57	1.52
Excessive coughing	2.15	2.06	1.04	4.74	2.01	2.36
Shortness of breath	-1.62	2.66	-0.61	-0.74	2.59	-0.29
Tightness in chest	1.45	2.08	0.70	1.37	2.02	0.68
Wheezing	-0.63	2.05	-0.31	-0.50	1.99	-0.25
Strange breathing sounds	-2.31	2.16	-1.07	-2.57	2.09	-1.23
Gastrointestinal						
Heartburn	2.35	2.86	0.82	1.94	2.78	0.70
Nausea/vomiting	1.15	2.20	0.52	3.46	2.15	1.61
No appetite	0.92	2.02	0.46	3.03	1.96	1.55
Diarrhea	-0.92	1.44	-0.64	2.96	1.39	2.13
Skin/eye irritation						
Burning eyes	-1.39	2.47	-0.56	5.58	2.42	2.31
Tearing eyes	-1.70	3.24	-0.52	0.64	3.16	0.20
Dry/scaly skin	1.85	2.81	0.66	2.67	2.74	0.98
Skin rash or irritation	0.54	1.72	0.31	2.28	1.67	1.36
Skin redness	-1.25	1.01	-1.23	0.12	0.99	0.12
Miscellaneous						
Joint/muscle pain	-0.22	4.03	-0.06	1.22	3.93	0.31
Unexplainably tired	-3.43	3.78	-0.91	0.76	3.68	0.21
Blurred vision	-4.67	3.14	-1.49	1.25	3.07	0.41
Dizzy/faint	-1.22	2.17	-0.56	-1.32	2.11	-0.63
Hearing problems	-6.44	2.50	-2.57	-3.58	2.44	-1.47
Chest pain	-2.30	1.32	-1.74	-0.35	1.29	-0.27
Fever/chills	-1.32	1.04	-1.27	-0.39	1.02	-0.38
Fainted	-0.18	0.86	-0.20	1.02	0.84	1.21
Quality of life						
Can't open windows	-1.33	2.88	-0.46	14.74	2.80	5.26
Can't go outside	-0.79	2.38	-0.33	12.73	2.32	5.47

[a]Difference in the average number of episodes between communities with and without livestock operations, adjusted for sex, age, smoking, and work outside of the home. [b]Of the β-coefficient.

quality of life among community residents in North Carolina that focused on the possible health effects of airborne emissions from intensive livestock operations. The study sample was drawn from areas of the state with a majority of African American residents who have low median income. This was not unexpected because intensive hog operations in North Carolina are located disproportionately in poor and nonwhite areas (*27*). Despite the legacy of distrust of biomedical research in the African American community (*28*), refusal rates were low because of the participation of community based organizations in introducing researchers to participants. The preponderance of women in the study reflects, in part, who was at home and who answered the door when approached by the community consultant and interviewer.

A number of symptoms previously reported as elevated among persons occupationally exposed in swine confinement houses were elevated among the residents of the hog community as compared to the community with no livestock operations. In particular, headache, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes were reported more frequently in the hog community. Members of the cattle community did not report similar elevations, nor did they report reduced quality of life. The quality of life measures (not opening of windows and not going outside even in nice weather) showed a large excess in the hog community.

As in all studies, measurement problems and differences between the communities other than the exposure of interest could have influenced the results. Recall bias is an issue in any survey. We were particularly concerned that residents living in proximity to a hog operation might report a greater number of symptoms because of negative feelings about the effect of the operation on their lives and their community. Therefore, we were careful to present the study as a rural health survey, not as a livestock and health study, and we did not include any questions in the survey that referred to hogs, livestock, or odors. During debriefings after the field work, interviewers reported that some respondents did not understand that questions about the environment referred to problems including odor. Such misunderstandings would have led to an underestimate of the impact of livestock operations on health and quality of life.

It is possible that residents of the hog community could have reported more symptoms because of their feelings about the negative impact of the hog operation on their community. However, if this had occurred, we would have expected excess reports for most symptoms. In fact, the eight symptoms in the miscellaneous category, none of which were expected to be related to exposure to airborne emissions, occurred with about the same frequency in the hog and control communities (Table 4). This suggests that there was not a tendency for over-reporting among residents of the hog community. Negative feelings might also have been evident in the open-ended questions, when respondents had the opportunity to report concerns beyond the environmental health and quality-of-life issues addressed in the structured questionnaire. As shown in Table 6, two persons in the hog community expressed concerns about property values.

Other circumstances of the survey may have led to an underestimate of the impact of swine operations on health of area residents. Perhaps most important, we studied an area with only one intensive hog operation. We would have expected to see larger effects in areas of the state with larger and more numerous operations and consequently heavier airborne emissions. Differences between the livestock and control communities may also have been reduced because of exposures to agricultural chemicals and dusts from row cropping in the control community.

Levels of emissions and weather conditions at the time interviewers were in the field may also have influenced the findings. With one exception, interviewers did not notice an odor from the hog operation while conducting the interviews. If interviews had been conducted when odors were strong, respondents may have reported a greater frequency of health symptoms.

The lack of environmental exposure monitoring data is also a concern in this study. We assumed that if persons resided within 2 miles of the hog operations, they were exposed to the emissions. We were not able to distinguish higher or lower exposure levels within the community. Exposure differences could occur because of differences in distance, direction, elevation, physical barriers, the amount of time spent at home, the amount of time spent outdoors, and the availability of air conditioning and filters in the home. Quantitative evaluation of exposure differences between individuals would increase the ability of an epidemiologic study to identify health effects of airborne emissions.

Similarly, clinical or biologic measures of outcome would strengthen information about relationships between environmental exposures to emissions from livestock operations and health. Future studies could be designed to obtain information on respiratory and immune function and standardized clinical evaluation of physical and mental health conditions. Such studies could evaluate possible mechanisms linking environmental exposures and health.

This study was not able to evaluate specific populations that may be more susceptible to health impacts of environmental exposures. These groups include children, asthmatics, and older persons with compromised pulmonary or cardiovascular function. Future studies should evaluate whether these subgroups are more sensitive to airborne emissions from intensive livestock operations. We were also unable to evaluate the acute impact of odors on mental health or the long-term impacts of reduced quality of life on mental, physical, or community health.

This study supports previous research suggesting that community members experience health problems due to airborne emissions from intensive swine operations (*7*). In North Carolina there are approximately 2,500 intensive hog operations, and they are located disproportionately in areas that are poor and nonwhite (*27*). The public health

Table 5. Problems that affect respondents' own life or health.[a]

Problem	Livestock operation		
	None	Cattle	Hogs
Livestock odor	0	8	25
Livestock odor (limits adult recreation)	0	0	14
Livestock odor (respiratory symptoms)	0	0	6
Livestock odor (can't open windows)	0	0	4
Livestock effluent (contaminated well)	0	0	4
Livestock odor (try not to breathe)	0	0	3
Livestock odor (nausea)	0	0	3
Livestock operation (flies and insects)	0	0	3
Crop sprayers (dust or noise)	1	0	2

[a]Respondents were asked, "Has the environment around your house affected your life and health?"

Table 6. Problems that affect family members' life or health.[a]

Problem	Livestock operation		
	None	Cattle	Hogs
Livestock odor	0	0	18
Livestock odor (limits child recreation)	0	0	10
Livestock odor (limits adult recreation)	0	1	4
Livestock odor (try not to breathe)	0	0	4
Livestock odor (respiratory symptoms)	0	0	4
Respiratory ailments	3	0	3
Complaints of skin symptoms	1	0	2
Livestock effluent (contaminated well)	0	0	2
Livestock odor (decreases property value)	0	0	2

[a]Respondents were asked, "Has the environment around your house affected the life or health of other members of your household?"

and environmental injustice implications of this geographical pattern extend beyond the physiologic impact of airborne emissions to issues of well-water contamination (*29*) and the negative impact of noxious odors (*8*) on community economic development (*30,31*). Populations in these areas may be at greater risk of health impacts due to high disease rates (*32,33*), low income (*27*), and poor housing conditions. Future research could provide a better understanding of the health effects of intensive livestock operations by combining individual exposure assessment, physiologic measures, clinical evaluation of physical and mental health, and follow-up of exposed communities.

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Attachment A6

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Examination of atmospheric ammonia levels near hog CAFOs, homes, and schools in Eastern North Carolina

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Abstract

Hog concentrated animal feeding operations (CAFOs) release ammonia (NH_3) in Eastern North Carolina (NC) to the atmosphere which is potentially hazardous for nearby human populations at community locations particularly homes and schools. We present NH_3 weekly average concentrations that were collected using passive diffusion tubes from October 2003 to May 2004 (20 sites) and from July 2004 to October 2004 (23 sites) near community locations in close proximity to hog CAFOs. The data for each phase of sampling was stratified by distance from the nearest hog CAFO. The mean Phase I levels were 16, 8, 7 and 5 ppb for distances <0.5, 0.5–1, 1–2, and 2 km or more, respectively. The mean levels for Phase II were 29, 16, and 11 ppb for distances <0.5, 0.5–1, and 1 km or more, respectively. The results of the distance stratification are the best results of this study and provide the strongest evidence that distance to one or more CAFOs is the key variable in controlling weekly NH_3 atmospheric concentration at the community level in Eastern NC. Statistical analyses confirmed that source terms such as distance to a hog CAFO and live weight per operation, as well as temperature, wind speed and wind direction were important predictors of atmospheric NH_3 at community locations. The results indicate potential zones of exposure for human populations who live or go to school near hog CAFOs.

Keywords: Ammonia; Hog; Passive diffusion; CAFOs; Emission; Exposure

1. Introduction

The exposure of rural populations to industrial hog farm pollution including ammonia (NH_3), hydrogen sulfide (H_2S), VOCs, bioaerosols, endotoxins, and particulate matter (National Academy of Sciences (NAS), 2002; Iowa Study Group (ISG), 2002) may lead to adverse health outcomes (e.g., respiratory ailments, immune suppression, stress, mental health problems) (Mirabelli et al., 2006; Merchant et al., 2005; Bullers, 2005; Avery et al., 2004; Hodne, 2001; Wing and Wolf, 2000; Cole et al., 2000; Thu et al., 1997; Schiffman et al., 1995, 2000). In addition, the burden of hog confined agricultural feeding operations (CAFOs) on rural communities can negatively impact quality of life

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including: (1) decreasing the ability of residents to enjoy their property (Thu, 2002), (2) reducing property values (Palmquist et al., 1997), (3) eroding social capital (Wright et al., 2001; Thu, 1996), (4) preventing healthy economic development (Durrenberger and Thu, 1996), and (5) hampering community sustainability.

In recent years, scientists have performed considerable research on industrial hog operations to quantify the emissions of these pollutants from confinement houses, lagoons, sprayfields and reemission from soils (see Iowa Study Group (ISG), 2002 for literature review), while, other researchers have produced studies to assess occupational exposures to hog CAFO pollution and related health effects (Iowa Study Group (ISG), 2002). This work has a limited utility in understanding chronic exposure of rural populations to intermittent levels of NH_3 and other CAFO pollutants and the development of appropriate exposure limits not based on occupational data and a paucity of community-based epidemiology studies.

Researchers have performed a paucity of work to examine the spatiotemporal variation of atmospheric NH_3 near populations at the community level (Reynolds et al., 1997; Secrest, 1999; Walker et al., 2004; Robarge et al., 2002; Phillips et al., 2004). Our lack of knowledge on atmospheric NH_3 at the community level is an important problem, because, without it, we are unable to develop accurate exposure profiles for residents who live near CAFOs and ascertain the burden of CAFOs and negative health responses on exposed populations.

This paper is a part of a larger project that examines spatiotemporal variation of atmospheric ammonia levels in Eastern North Carolina near hog CAFOs, homes and schools (Wilson, 2005). Previous work has demonstrated the importance of source and meteorological parameters in predicting NH_3 levels on the CAFO property or at a limited number of ambient sites. The purpose of this paper is to answer the question: What are the variables that explain NH_3 variation in a region of variable hog CAFO density near homes and schools at the community level? This paper seeks to answer this question by exploring the relationship between distance to emission source and measured NH_3 levels and examining the association between concentration and meteorological parameters across community locations.

2. Methods

2.1. Passive monitoring of atmospheric ammonia in Eastern NC

Passive diffusion tubes were utilized to measure atmospheric ammonia levels near industrial hog CAFOs, homes and schools in Eastern NC. Passive sampling occurred in two phases from 2003 to 2004 across a region of variable CAFO density including Duplin, Greene, and Lenoir (high emission density counties) and Nash, Edgecombe, and Wilson (low emission density counties). Phase I occurred from October 2003 to May 2004 (20 sites) to measure atmospheric NH_3 levels across different seasons and low and high emission density counties. Phase-II sampling occurred from July to October 2004 (23 sites) to measure NH_3 during the warmest months of the year in Duplin, Greene, and Lenoir counties. Passive samplers were placed near homes and schools less than 2 km from the nearest hog CAFO (exposed sites) and near homes and schools greater than 2 km from the nearest hog CAFO (less-exposed sites). Passive samplers were exposed for 1 week at a time, so that the NH_3 data presented in this work correspond to weekly average of atmospheric NH_3 concentration. More information about the passive method, sampling framework, and results can be found in Wilson (2005).

2.2. Calculation of distance

Latitude/longitude coordinates of industrial hog operations were supplied by the North Carolina Department of Environment and Natural Resources (North Carolina Department of Environment and Natural Resources (NCDENR), 2002). Additional information on the location of hog CAFOs was derived from the work presented in Wing et al. (2000). The ArcGIS 8.3 (ESRI, Redlands, CA, USA) find distance tool was employed to find the distance from each passive monitoring station to the closest hog CAFO and these distances were checked in the field. These distances were recorded and placed in an Excel spreadsheet.

2.3. Stratification of distance

NH_3 data were stratified based on distance from the emission source. Four distance categories were established for Phase I: (1) <0.5 km from a CAFO,

(2) 0.5–1 km from a CAFO, (3) 1–2 km from a CAFO and (4) >2 km from a CAFO. Only three distance categories were used <0.5 km from a CAFO, 0.5–1 km from a CAFO, and >1 km from a CAFO for Phase II because sampling occurred at only one site >2 km from a CAFO (site #23). The Swine Farm Siting Act requires that swine houses or lagoons holding animal waste shall be located at least 1500 ft from any occupied residence; at least 2500 ft from any school, hospital, or church; and at least 500 ft from any property boundary (North Carolina General Statutes (NCGS), 1995). Distance categories 1 and 2 were comparable to setback distances for livestock operations at 1500 ft (~0.5 km) from a residence and 2500 ft (~0.8 km) from a school. The use of stratification will allow us to ascertain the average NH_3 levels in the mandated buffers between livestock operations and homes and schools at the community level.

2.4. Extraction of weather data from the North Carolina Climate Office database

Meteorological parameters were obtained for nearby weather stations in the Eastern NC study area during Phase I and Phase II sampling. The data were extracted from the North Carolina Climate database administered by the State Climate Office of North Carolina (SCONC) housed at North Carolina State University (State Climate Office of North Carolina (SCONC), 2003; ⟨www.nc-climate.ncsu.edu⟩. This office maintains the database that collects all of the available meteorological data for the state of NC. The weather stations collect hourly temperature, wind speed and direction, humidity, and precipitation, and barometric pressure data. Fig. 1 shows the location of the weather stations used in this study in relation to the Phase I and II monitoring sites and industrial hog operations.

2.5. Prediction of weather parameter at each passive monitoring station using ArcGIS

Due to the unavailability of weather data at each passive monitoring station, the SCONC data was used. In the ArcGIS 8.3 platform, the Geostatistical Analyst tool (Johnston et al., 2001) was employed to predict unknown weather parameter levels at different locations in the study. Twenty-five stations provided available temperature, wind speed, wind direction, pressure, humidity, temperature and other data. From this data, ordinary kriging was

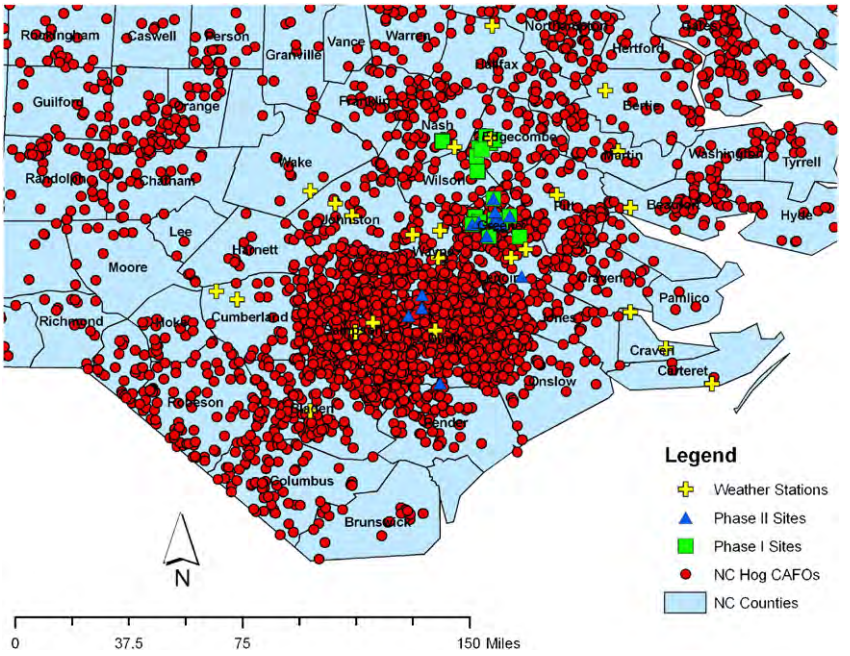

Fig. 1. Location of weather stations in relation to Phase I and II NH_3 sites in Eastern NC. This map shows 25 weather stations in Eastern NC which were used to create weather surfaces.

used to predict and develop a continuous surface map for each weather variable for each sampling period.

A map of the NH_3 sampling stations was overlaid on the continuous surface map for each weather parameter created for each deployment period. A value for each weather parameter was then obtained with the ArcGIS locate tool. For example, the continuous temperature surface showed the kriged predictions of temperature over the entire study area. Using the ArcGIS locate tool, a temperature value was obtained for each of the passive NH_3 stations for Phase-I and Phase-II sampling. These temperature values were recorded and placed in an Excel spreadsheet. This process was repeated for each meteorological parameter for each deployment period.

2.6. Source-related variables obtained using ArcGIS 8.3

Information was obtained from NC DENR on the number of animals and steady state live weight (SSLW) per hog CAFO. ArcGIS 8.3 was used to obtain source-related variables including the distance of each monitoring site to the closest hog CAFO, animals at the nearest CAFO, and SSLW at the nearest CAFO. A spatial calculator was employed to calculate the total and mean number of CAFOs, animals, live weight, and emissions in a 1 km radius around each NH_3-sampling site. These steps were repeated for radial buffers of 2 and 5 km.

2.7. Regression of weather parameters with source variables vs. NH_3 concentration

SPSS 10.0 (SPSS Inc., Chicago, IL) was used to ascertain which weather and source variables were statistically significant predictors of atmospheric ammonia during Phase I and II sampling at community locations. Several regression options were available in SPSS including Forward, Backward and Stepwise. The stepwise procedure was utilized as the primary regression procedure because it allows the user to retain the most statistically significant terms in the model. Mean NH_3 was regressed against meteorological variables (temperature, wind speed, pressure, humidity, average wind direction, maximum wind direction, and precipitation) and source-related variables mentioned previously to obtain the best predictive model for each set of data.

3. Results and discussion

3.1. Location of hog CAFOs in relation to NH_3 passive monitoring sites

Table 1 shows the distance zone of each passive monitoring site employed during Phase I and II sampling in relation to the nearest industrial hog operation regardless of direction.

During Phase-I sampling, the distance from the passive monitoring sites to the nearest hog CAFOs ranged from 0.25 to 4.77 km. Four sites (#6, #8, #10, and #13) were located at distances greater than 2 km from the nearest CAFO in either Edgecombe or Wilson. These sites were designated as "less-exposed" sites. Table 1 also shows the distance zone of Phase II monitoring sites to the nearest industrial hog operation. All of these sites were primarily "exposed sites" located in high emission density counties as defined by Walker et al. (2004). Phase-II sites were located 0.20–5.94 km from the nearest industrial hog operation.

Table 1
Distance zone of each passive monitoring station to nearest hog CAFO

Phase I station ID	County	Zone[a]	Phase II station ID	County	Zone[b]
1	Nash	1	1	Duplin	3
2	Greene	3	2	Duplin	3
3	Greene	2	3	Duplin	3
4	Greene	3	4	Duplin	2
5	Edgecombe	1	5	Duplin	2
6	Edgecombe	4	6	Duplin	2
7	Edgecombe	3	7	Greene	3
8	Wilson	4	8	Greene	2
9	Greene	2	9	Greene	2
10	Wilson	4	10	Greene	1
11	Greene	3	11	Greene	1
12	Nash	2	12	Greene	2
13	Edgecombe	4	13	Greene	3
14	Greene	1	14	Greene	2
15	Edgecombe	1	15	Greene	3
16	Edgecombe	2	16	Greene	3
17	Greene	3	17	Duplin	1
18	Greene	2	18	Duplin	1
19	Greene	2	19	Duplin	1
20	Greene	3	20	Duplin	1
			21	Duplin	3
			22	Duplin	3
			23	Lenoir	3

[a]For Phase I, Zone 1: 0–0.5 km, Zone 2: 0.5–1 km, Zone 3: 1–2 km, and Zone 4: 2 km or more from nearest hog CAFO.

[b]For Phase II, Zone 1: 0–0.5 km, Zone 2: 0.5–1 km, and Zone 3: 1 km or more from nearest hog CAFO.

3.2. Stratification of distance vs. NH_3 concentration

Walker et al. (2004) reported a mean NH_3 level of $0.58\,\mu g\,m^{-3}$ (0.84 ppb) at an agricultural site in Martin County, NC (emission density = 320 kg NH_3-N $km^{-1}\,yr^{-1}$). This value is higher than the value reported by Pryor et al. (2001) at an agricultural site in an area of low local emissions (Walker et al., 2004). The mean value at the Martin County site is within the range of values reported for non-agricultural sites and US urban areas (Walker et al., 2004). Walker et al. (2004) also reported a mean value of $2.46\,\mu g\,m^{-3}$ (3.50 ppb) at the Kinston site (emission density = 2290 kg NH_3-N $km^{-1}\,yr^{-1}$). We report levels in the following section that are higher than the ambient levels mentioned above.

Phase I and II data are considered separately in Figs. 2 and 3. The concentrations are stratified by distance from the nearest hog CAFO. We made the assumption that the closest emission source would be the most dominant emission source near each passive monitoring site. Overall concentrations are lower for Phase-I sampling and concentrations drop off more steeply from <0.5 to 0.5–1.0 km, but the same declining trend is apparent in both phases. Fig. 2 shows that during Phase-I sampling the mean concentration drops by more than half between sites at 0–0.5 and 0.5–1 km. The mean NH_3 concentration at distances greater than 2 km from a CAFO (5.3 ppb) remained higher than ambient levels

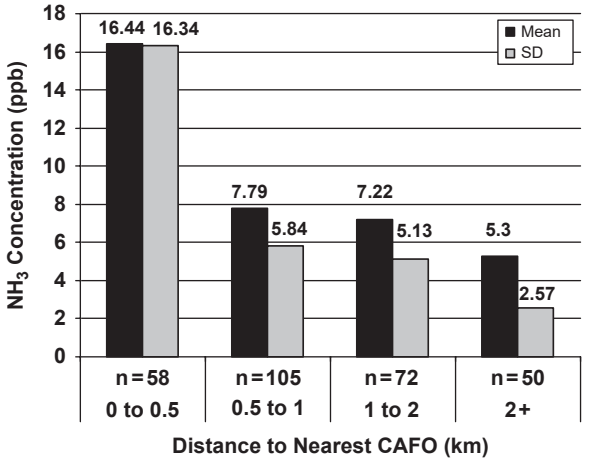

Fig. 2. NH_3 concentrations stratified by distance to nearest hog CAFO (Phase I). The figure shows the mean and standard deviation (SD) of pooled NH_3 averages for four distance zones. Zone 1: 0–0.5 km, Zone 2: 0.5–1 km, Zone 3: 1–2 km, and Zone 4: 2 km or more from nearest hog CAFO.

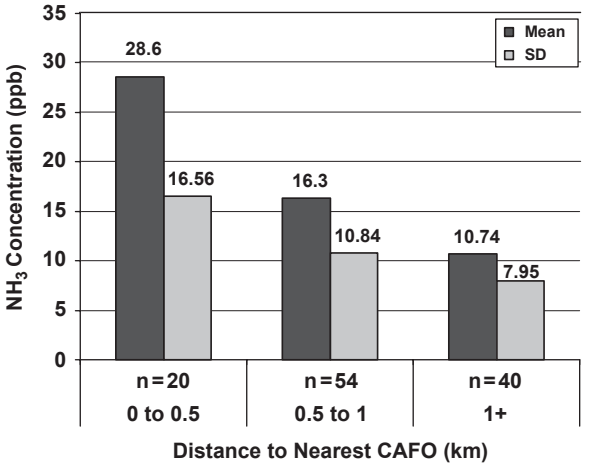

Fig. 3. NH_3 concentrations stratified by distance to nearest hog CAFO (Phase II). The figure shows the mean and standard deviation (SD) of pooled NH_3 averages for three distance zones. Zone 1: 0–0.5 km, Zone 2: 0.5–1 km, and Zone 3: 1 km or more from nearest hog CAFO.

thought to range from less than 1 to 3 ppb for an agricultural region (Pryor et al., 2001; Walker et al., 2004; Warneck, 1988).

Fig. 3 displays the stratified Phase II data. We see that the mean concentration of 29 ppb for sites located in the exposure zone 0.5 km or less from the nearest hog CAFO is two times greater than the mean concentration at sites between 0.5 and 1 km. Additionally, the mean concentration obtained for pooled sites that were 1 km or greater from the nearest CAFO was 10.74 ppb. This figure illustrates the decline in mean NH_3 levels as distance increases between community locations and emission source.

Tables 2 and 3 present data for Phase I and II sampling sites that demonstrate the negative trend that exists between distance to emission source and NH_3 concentration. From these tables, we see that the mean NH_3 levels are generally higher than ambient levels reported in other work (Pryor et al., 2001; Walker et al., 2004). Some of the Phase-I sites located less than 2 km from the nearest hog CAFO had lower than expected mean NH_3 levels. This could be due to their placement in low emission density counties.

We placed all Phase II passive samplers in high emission density counties. This factor in conjunction with their close proximity to not only the nearest CAFO but a cluster of operations within less than 1 km may explain why the mean NH_3 concentrations obtained for each site at distances near or less than 1 km (4–49 ppb) during Phase-II

Table 2
Arithmetic average (mean) of the weekly NH_3 (ppb) concentrations obtained for each site during Phase I

Site	Distance (km)	Mean	Site	Distance (km)	Mean
1	0.71	7.38	11	1.01	7.92
2	1.12	6.28	12	0.9	6.65
3	0.56	10.36	13	3.63	4.23
4	1.93	5.51	14	0.2	19.59
5	0.25	21.09	15	0.46	8.64
6	2.1	5.00	16	0.694	4.80
7	1.18	4.91	17	1.91	11.25
8	4.77	5.20	18	0.644	8.42
9	0.958	9.57	19	0.527	7.34
10	4.92	6.77	20	1.29	7.45

Distance represents distance from each NH_3 monitoring site to nearest hog CAFO.

Table 3
Arithmetic average (mean) of the weekly NH_3 (ppb) concentrations obtained for each site during Phase II

Site	Distance (km)	Mean	Site	Distance (km)	Mean
1	1.01	10.54	13	1.26	4.6
2	1.02	7.91	14	0.53	8.81
3	1.01	9.13	15	1.8	11.56
4	0.79	20.11	16	1.01	4.25
5	0.81	19.48	17	0.44	36.63
6	0.84	21.51	18	0.45	32.12
7	1.12	10.59	19	0.48	48.95
8	0.69	8.56	20	0.48	32.12
9	0.64	18.17	21	1.83	11.57
10	0.2	20.37	22	1.8	12.64
11	0.21	23.2	23	5.94	3.34
12	0.56	9.57			

Distance represents distance from each NH_3 monitoring site to nearest hog CAFO.

sampling were higher than the mean concentrations obtained during Phase-I sampling for the same distance range (7–21 ppb). In addition, Phase-II sampling occurred mainly during the summer when the concentrations at or near animal operations are typically higher than the concentrations in the fall, winter and spring months (Walker et al., 2004; Robarge et al., 2002; Pryor et al., 2001; Lefer et al., 1999; Langford et al., 1992; Asman, 1998).

The mean and maximum concentration (not shown) for each of these sites can be used to infer that human populations in the surrounding area may be exposed to appreciable amounts of ammonia released from nearby hog CAFOs, which creates a relative index of exposure. The relative index of exposure is further influenced by the location of community locations (i.e., homes and schools) proximal to clusters of hog CAFOs at distances less than 2 km. The results from Phase-II sampling demonstrate the strength of the relationship between distance to the nearest hog operation and atmospheric ammonia concentration. The mean atmospheric NH_3 concentrations were generally higher than the concentrations obtained for sites during Phase I-sampling because of the placement of the monitoring stations in high emission density counties such as Greene and Duplin.

Sites 10–11 (Greene) (Table 2) and 17–20 (Duplin) (Table 3) were located at distances less than 0.5 km from the nearest operation. Sites 10 and 11 both located approximately 200 m from the nearest hog CAFO had mean concentrations of 20 and 23 ppb, respectively, and a combined concentration range between 12 and 47 ppb. These sites had elevated concentration levels that were appreciably above ambient background levels. The location of multiple hog operations in close proximity to sites 17–20 resulted in the highest levels of atmospheric ammonia observed during data collection. Sites 17–20 were placed at a residential location with multiple hog operations less than 0.5 km from the collocated passive samplers. The mean concentration for the pooled data of these sites was 28 ppb and the concentration range fell between 4 and 81 ppb.

Conversely, Phase-I sites located at distances greater than 2 km from an industrial hog operation (sites #6, #8, #10, and #13) (Table 2) were designated as "less-exposed" sites. Due to their distance from an emission source, these sites were located in low emission density counties with lower numbers of hogs raised in a confined environment than in Duplin and Greene counties. The NH_3 data for these "less-exposed" sites indicate on average there are lower NH_3 levels at community sites located at greater distances from industrial hog operations. Moreover, human populations that reside at distances greater than 2 km from an industrial hog operation may have a lower risk or frequency of exposure to atmospheric ammonia when they reside in communities, attend school or travel to receptor locations in counties with a low density of operations (i.e. sparse hog population). Even though ammonia emissions released to the atmosphere undergo dry or wet deposition, as well as transformation processes, these processes do not completely eliminate atmospheric concentration of

ammonia down to ambient levels (1–3 ppb) for sites that are more than 2 km from the CAFO source in counties with low emission density as expected.

Figs. 2 and 3 reveal that the distance-to-the-closest-farm curve does not decline quickly either for Phase-I or Phase-II data. This result leads us to believe that in areas similar to Eastern NC, the presence of a high density of CAFOs in a region will have a cumulative effect and result in higher background levels of atmospheric NH_3 than agricultural regions with few operations. Therefore, we believe the density of hog CAFOs is equally or a more important determinant of atmospheric NH_3 levels than distance to the nearest CAFO. This supposition is further validated by the fact that we found a high background level even at sites that we located far from hog CAFOs. The implication of these findings (see Section 4) is that we expect a regional increase of atmospheric NH_3 across high emission density counties where no resident is far from any hog operation, so that the density of hog operations is a critical component of any intervention to minimize community exposure to NH_3.

However, in states like Iowa, Oklahoma and Minnesota, where ammonia emissions are reduced by the use of deep pit building systems and alternative waste management techniques instead of the uncovered lagoons used at most NC operations (Iowa Study Group (ISG), 2002), this supposition may not hold true. Therefore, the results in Eastern NC may have limited generalizability to areas in hog production states that use alternative waste management systems.

3.3. Regression of weather variables and source data vs. NH_3 concentration

Stepwise regression was used to examine the relationship between meteorological, source-related terms, and measured ammonia concentration for Phases I and II. The deployment averages collected for 40 weeks were pooled for each variable and analyses were run in SPSS 10.0. Tables 4a, 4b, 5a and 5b show the stepwise regression models and coefficients for Phases I and II.

The Phase-I model only explained a small portion (21%) of the variation of the NH_3 concentration. This model was an improvement over the regression of NH_3 using only Phase I meteorological variables which only explained 3% of the variation (not shown). The source-related variables provide information on the major source of ammonia near the passive monitoring stations which was assumed in this work to be industrial hog operations. The inclusion of the source variables greatly improved the regression models. For example, the R^2 increased from Phase I to Phase II (21–43%) because during Phase II most of the sites were located less than 1.5 km from the nearest CAFO and sampling occurred only in high emission density counties during the summer when NH_3 levels should be the highest. Many of the Phase-I sites were located greater than 1.5 km from the nearest hog operation in low emission density counties. Less ammonia was available for sampling and lower mean NH_3 levels were obtained.

Live weight acts as an important predictor of NH_3 (Walker et al., 2004, 2000a, b; Dragosits et al., 2002) and has similar Beta coefficients for Phase I and II data. However, the distance to closest farm variable is only present in the Phase-I model and not

Table 4a
Stepwise regression coefficients for Phase I

Model	R^2	R^2 change
1	0.062	0.062
2	0.099	0.037
3	0.187	0.088
4	0.211	0.024

Model 1: Constant + DISCLOSF.
Model 2: Constant + DISCLOSF + LIVEWT.
Model 3: Constant + DISCLOSF + LIVEWT + SUMAN2KM.
Model 4: Constant + DISCLOSF + LIVEWT + SUMAN2KM + AVGTEMP.
R^2: Amount of NH_3 variation accounted for by the model.

Table 4b
Final regression model coefficients for Phase I

Variable	Beta coefficient
Constant (B_0)	11.6
Distance from each NH_3 monitoring station to the nearest industrial hog farm (DISCLOSF)	−6.20
Steady state live weight of nearest hog CAFO to NH_3 monitoring station (LIVEWT)	1.40×10^{-5}
Sum of the number of hogs on all of the industrial operations located within a 2 km radius of each NH_3 monitoring station (SUMAN2KM)	-7.20×10^{-4}
Average temperature (AVGTEMP)	0.30

Table 5a
Stepwise regression for Phase II

Model	R^2	R^2 change
1	0.300	0.308
2	0.381	0.087
3	0.411	0.036

Model 1: Constant + LIVEWT.
Model 2: Constant + LIVEWT + WDMAXWS.
Model 3: Constant + LIVEWT + WDMAXWS + AWS.
R^2: Amount of NH_3 variation accounted for by the model.

Table 5b
Final regression model coefficients for Phase II

Variable	Beta coefficient
Constant (B_0)	15.3
Steady state live weight of nearest hog CAFO to NH_3 monitoring station (LIVEWT)	1.20×10^{-5}
Wind direction of maximum wind speed (WDMAXWS)	6.70×10^{-2}
Average wind speed (AWS)	−7.00

the Phase-II model. This may be explained by a spatial gradient during Phase-I sampling (several sites located 2 or more km from the nearest CAFO) while only one site was located 2 or more km from the nearest CAFO during Phase-II sampling resulting in no spatial gradient. Monitoring sites located in close proximity to hog CAFOs during Phase II indirectly controlled for distance and helped to exclude it from the final model.

Temperature, which was previously demonstrated to control atmospheric NH_3 levels (Robarge et al., 2002; Walker et al., 2004, 2000a, b; Aneja et al., 2000; Asman, 1998; Lefer et al., 1999) was included in the Phase-I model and not the Phase-II model because passive sampling occurred during fall, winter and spring months. Phase-II sampling only occurred during the summer months, giving no appreciable temperature gradient and the variable was dropped from the model. If Phase II-sampling occurred from summer 2004 to spring 2005, we believe the temperature variable would have had a greater chance of inclusion in the final model. In addition, Phase II regression model shows that wind speed is an important variable. The model indicates as wind speed increases, there is more mixing and dispersion and less ammonia at the community level. This relationship between wind speed and pollutant level has been established previously (Tate, 2002; Brown, 2000; Thijsse et al., 1998; Robarge et al., 2002; Rabaud et al., 2001).

In the Phase I regression model, SUMAN2KM (sum of animals within 2 km) has a negative value for its regression coefficient. Since we controlled for live weight (which has a positive regression coefficient), this means that for a given live weight more animals produce less NH_3, or put in other words, that smaller animals produce less NH_3 per unit weight. This does not necessarily follow the usual wisdom, and therefore this observation will have to be studied in more detail. It could be that the number of animals captures some confounding effect not accounted for in our model (i.e. this relationship could be more representative of variation at the farm level when animals are present on the operation vs. when they are not present on the operation or type of operation (i.e., farrowing, feeding, finishing). Unfortunately, the inclusion of both live weight and number of animals may limit the generalizability of this model to other hog production regions.

The regression analyses indicate that there is a positive relationship between wind direction and NH_3. An explanation is NH_3 sampling occurred in an area with multiple sources. While the nearest source does have an impact, we cannot ignore the overall elevation of ammonia in the sampling area due to the contribution of multiple sources. Robarge et al. (2002) used loess regression to develop model for a site surrounded by several CAFOs and found NH_3 would be highest when wind direction was near 278° (blowing from the northwest). These results emphasize the importance of wind direction in relation to source strength (one or more sources) and highlight the potential use of NH_3 as a tracer for CAFO pollution based on relative wind direction.

The regression analyses reiterate the importance of hog CAFOs as the primary source of NH_3 emissions released to the local environment in Eastern NC. Walker et al. (2000a, b) support the role of animal population in determining NH_3 variation and therefore supports the inclusion of LIVEWT in the regression model. The distance, direction, and population of an industrial hog operation contribute to potential exposure including frequency and duration for humans at community locations contiguous to the operation or a hog-densified area.

4. Conclusions

The purpose of this paper was to answer the question: What are the variables that explain NH_3 variation at the community level? The distance stratification results (Figs. 2 and 3) demonstrate how concentration changes over space as a function of distance. These results are the best results of this study and provide the strongest evidence that answers the above question: distance to one or more CAFOs is the key variable in controlling atmospheric NH_3 at the community level in Eastern NC. The results also show that the passive diffusion method was successful at measuring NH_3 at the community level.

The results suggest that maximally exposed populations in Eastern NC (average exposure levels $\geqslant 10$ ppb) will live in environments characterized by one or more hog CAFOs (<1–5 km away) and above-average levels of hogs measured by either CAFO density, hog population density, etc. at the county level. These conclusions are supported by the regression results. Distance to the nearest CAFO and SSLW were shown to be strong predictors of NH_3 concentration. Meteorological parameters such as temperature, wind speed and direction were found to be good predictors of ammonia as well. The low reported R^2s for Phase I (21%) and Phase II (43%) could be an effect of geographic or temporal differences in the weather variables, measurement variability, or an increase in the regional background levels of atmospheric NH_3. High baseline levels limit the effect that individual variables such as wind speed and direction, temperature, humidity, etc. have on NH_3 levels at the regional scale. However, these findings are consistent with other studies that have examined or discussed the influence of source terms (Walker et al., 2004, 2000a, b; Fowler et al., 1998; Robarge et al., 2002) and weather variables (Robarge et al., 2002; Aneja et al., 2000).

The results also support the theory that zones of exposure exist for human populations who live near industrial hog operations in Eastern NC. These zones are a function of distance from the emission source where frequency of exposure over time increases the closer a community location is to the source (as shown in Figs. 2 and 3). Human populations residing at locations with a density of operations within close proximity (2 km) could be exposed to very high levels of ammonia, up to 40 times higher than normal ambient levels (1–3 ppb). As started earlier, we believe that the density of hog CAFOs is a critical emission factor belying the effectiveness of nuisance and public health policies focusing on individual facility setback distances from homes and schools. Furthermore, this finding may indicate the potential ineffectiveness of setback distances even where hog facilities are low density, but clustered.

These findings are crucial for the development of an exposure zone theory to model hog CAFO pollution in low and high emission density counties. The challenge of developing such a model is to identify variables controlling important fate and transport mechanisms for which data is currently available or attainable through more research. Our findings indicate that currently available data exists for (a) distance to the location of CAFOs, (b) live weight, (c) wind speed and wind direction, and (d) temperature. We recommend that future modeling approaches combine both mechanistic modeling of key fate and transport mechanisms (e.g. additive plumes arising from each CAFO, etc.) to account for the general characteristics of the spatiotemporal distribution of NH_3 in regions with high emission density, as well as geostatistical processing of NH_3 monitoring data to account for other random space/time processes for which data is lacking. The aim of such future work will be to model the effect of the *density* of CAFOs and their emissions, which this work points to as being a critical concern for chronic community exposure to hog CAFO air pollution. *It is not just how far CAFOs are from a residence, but also what is the density of CAFOs in that community*.

We expect this paper and additional work on the spatiotemporal variation of human exposure to atmospheric ammonia to help fill the gap between the work to monitor NH_3 and related odors at or near the CAFO property and limited number of environmental epidemiology studies performed in exposed communities. The linkages between the two are obvious for populations negatively affected by chronic exposure to intermittent levels of NH_3 and other hog CAFO pollutants. It is important that rigorous scientific inquiry examines this chronic exposure and the efficaciousness of buffers between CAFOs and residences and schools to improve quality of life and protect public health. Data from these studies will provide a clearer picture of the burden that hog CAFOs have on nearby communities and support legislation needed to redress the health and quality of life

burdens that hog CAFOs have on exposed human populations.

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Attachment A7

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Use of passive samplers to measure atmospheric ammonia levels in a high-density industrial hog farm area of eastern North Carolina

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Abstract

Hog concentrated animal feeding operations (CAFOs) in North Carolina release ammonia (NH_3), hydrogen sulfide, VOCs, and particulate matter to the atmosphere. These operations are located mainly in the NC coastal plain and can create potential health hazards for nearby human populations. Limited work has been performed to measure NH_3 at the community level to assess potential human exposure. In an effort to address this issue, a study was designed to measure NH_3 levels near hog CAFOs and community locations (i.e. homes and schools) in Eastern NC.

NH_3 was collected using passive diffusion tubes in triplicate exposed primarily in weekly intervals. Sampling occurred from October 2003 to May 2004 (20 sites) and from July 2004 to October 2004 (23 sites) at varying distances from hog CAFOs in close proximity to homes and schools. Average weekly NH3 levels were measured as mass (μg NH_3-N) and converted to concentration (ppb). Mean level of 13.8 ppb near homes and schools (<2 km) was 4–12 times greater than ambient background levels (1–3 ppb), reaching as high as 80 ppb. Exposed sites (<2 km from a hog CAFO) had a mean level of 12.8 ppb which was over 2 times higher than the mean level of 5.5 ppb at less exposed sites (>2 km from a hog CAFO).

The study establishes that passive sampling can be effectively used to measure average atmospheric ammonia levels at community locations near hog CAFOs in Eastern NC. The collected data indicate the relative exposure for human populations who live near a hog CAFO. The closer a populace is to the hog CAFO, the more intense the exposure. These results require more validation in the field by comparison to a reference method.

Keywords: Atmospheric ammonia; Passive sampling; Industrial swine operations; Community level; Exposure

1. Introduction

Industrial swine facilities release a variety of pollutants that may pose health concerns to human populations living near the operations. Air pollutants are released during the decomposition of the swine waste and have three major sources on the

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hog farms: swine confinement structures, anaerobic lagoons, and sprayfields. These pollutants include ammonia (NH_3), hydrogen sulfide, SO_2, VOCs, bioaerosols, particulate matter, methane, carbon monoxide and carbon dioxide (Schnoor et al., 2002; Cole et al., 2000).

There has been a substantial increase in the number of large intensive swine operations in the state of North Carolina over the past 10 years (Kase, 2003). Before 1990, the number of hogs in the state was 2.5 million (North Carolina Department of Agriculture (NCDA), 1998). The state produces approximately 10 million hogs which are located on 2500 concentrated animal feeding operations (CAFOs) primarily in Eastern NC (United States Department of Agriculture (USDA), 2002) (Fig. 1). North Carolina is currently second in US hog production behind Iowa with a production increase of approximately 282 percent from 1987 to 1997 (Scorecard, 2003). Approximately 90 percent of the NC hog population is located in the coastal plain region (Walker, 1998; Walker et al., 2000a, b) with most of the hog production focused on a six-county area (Duplin, Sampson, Greene, Lenoir, Bladen, and Wayne) (Walker et al., 2000a, b). This nexus of hog production in the NC coastal plain area has an average hog population density of 528 hogs km^{-2} and the remaining coastal plain has a hog population density of 65 hogs km^{-2}. Ammonia emissions from the six-county area contribute 36 percent of the total NH_3 emissions with over 75 percent coming from the swine operations located in the region (Chauhan, 1999).

Passive samplers were selected for use in this research project instead of active samplers to measure atmospheric NH_3 levels near hog CAFOs in Eastern NC. Passive samplers are less expensive, more inconspicuous, easily secured, can be deployed for extended periods and do not require access to electricity (Carmichael et al., 2003; Roadman et al., 2003; Rabaud et al., 2001). Passive samplers can be deployed in large numbers, over wide areas for monitoring and modeling dispersion and the fate of gases released from various sources (Roadman et al., 2003). The efficacy of passive samplers in measuring atmospheric ammonia shown in previous research (Walker, 2005, 2006; Walker et al., 2004a; Carmichael et al., 2003; Thoni et al., 2003; Tate, 2002) helped in the selection of passive sampling for use in this work.

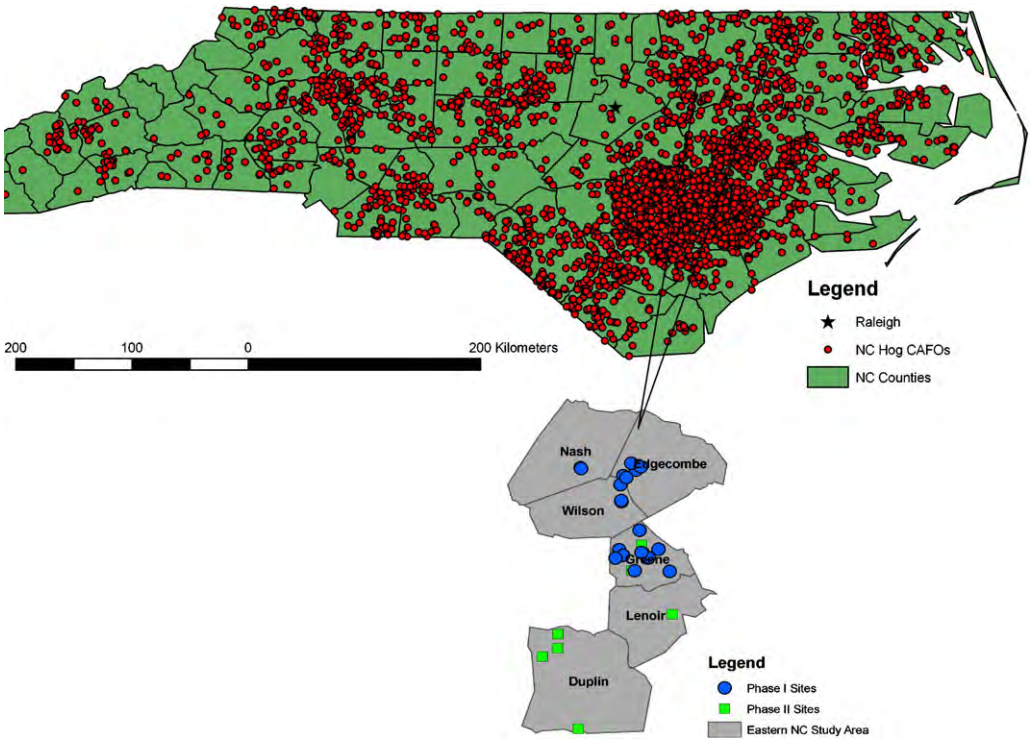

Fig. 1. Location of hog CAFOs in North Carolina in relation to Eastern NC study area. This map visually shows that majority of hog CAFOs (~2500) are located in NC coastal plain.

Researchers have demonstrated the ability of passive samplers to measure NH_3 levels near poultry operations (Fowler et al., 1998; Roadman et al., 2003). However, passive samplers have not been used extensively to measure the ambient NH_3 levels near intensive agricultural operations (e.g. hog CAFOs) at the community level. Communities that neighbor hog CAFOs and residences in close vicinity (2 miles) to the operations have expressed concerns about exposure to air emissions and contaminants in ground water (Wing and Wolf, 2000; Cole et al., 2000). Although the ambient levels of air pollutants may be lower than occupational exposures, there is a need to quantify the ambient concentration in the residential environment. Previous epidemiological studies have shown these pollutants may adversely impact the air quality and respiratory health of neighboring communities and present risk to susceptible populations (Schiffman, 1998; Wing and Wolf, 2000; Thu et al., 1997).

Limited work has been performed to characterize atmospheric NH_3 levels at fenceline locations or in neighborhoods near CAFOs (Secrest, 1999; Reynolds et al., 1997). A few studies (Robarge et al., 2002; Walker et al., 2004b) assessed ambient off-site levels of atmospheric NH_3 in Eastern NC. Most of the research associated with measuring atmospheric NH_3 levels has been performed on or near the hog operation (Aneja et al., 2000, 2001; APWMC, 2003; Walker, 2006). The research presented in this paper expands upon previous work performed at the operation utilizing the method described by Fowler et al. (1998) and Walker (2006).

A major objective of the study was to answer the question: is it possible to develop and implement a monitoring strategy using passive samplers that would detect atmospheric ammonia at the community level? The aim of utilizing passive diffusion tubes was to assess the efficiency of a robust, inexpensive, power-free and unobtrusive analytical measurement technique that could eventually be deployed by community members to provide data which can be used to characterize potential exposure to average NH_3 near homes and schools at the community level.

2. Methods

2.1. Site selection

Swine production in North Carolina is mainly concentrated in the eastern part of the state in six counties (Duplin, Sampson, Greene, Wayne, Bladen, and Lenoir) (Walker et al., 2000b; Robarge et al., 2002). Walker et al. (2004b) defined counties in this region as high emission density counties and potential sampling sites in this region and low emission density counties (Walker et al., 2004b) were selected based on other studies (Edwards and Ladd, 2000; Wing et al., 2000). Sampling sites were deemed "exposed sites" if they were within 2 km of the nearest hog CAFO and other sites were deemed "less exposed sites" if they were located >2 km from the nearest hog CAFO. There was a tradeoff between selecting sites in close proximity to a desired source (<2 km from a hog CAFO) or population receptor in terms of visibility, accessibility, safety and security of the sites.

2.2. Phase I and II sampling

Sampling took place in two phases in 2003 and 2004. Passive monitoring sites were selected in both high and low emission density counties as defined by Walker et al. (2000a, b). High emission density counties (3000 kg NH_3-N km^{-2} or more) were Greene, Duplin and Lenoir and low emission density counties (<1000 kg NH_3-N km^{-2}) were Nash, Edgecombe, and Wilson (Walker et al., 2004b). The passive samplers were typically exposed in weekly intervals to obtain integrated NH_3 concentrations. We followed previous research (Walker, 2006; Walker et al., 2004a; Tang et al., 2001) and sampled on a weekly basis to accumulate enough NH_3 mass to calculate a time-integrated concentration. This weekly exposure is particularly important in environments with variable amounts of NH_3 where concentrations are typically at the ppb range, instead of the ppm range present on industrial hog operations.

Phase I sampling occurred from October 2003 to May 2004 in Nash, Edgecombe, Wilson and Greene counties. Phase II sampling occurred from July 2004 to October 2004 in Greene, Lenoir, and Duplin counties only. This sampling was conducted to capture elevated ammonia levels found during the summer. Previous research (Walker, 2005, 2006; Walker et al., 2004a; Robarge et al., 2002) indicated that there are higher concentrations of atmospheric ammonia on CAFOs and locations near CAFOs during the summer than during the rest of the year. This is due in part to the temperature dependency of ammonia emissions and the exchange of ammonia between different surfaces as defined by Henry's Law (Robarge et al., 2002).

Phase II sampling was employed to further characterize the atmospheric ammonia levels found in the vicinity of hog CAFOs, in particular, the levels of human exposure found at locations within 1 km versus locations that were >2 km from hog CAFOs. Passive monitors were placed near residential and school locations. Passive samplers were also placed at four residential sites (three at each site) to collect data on microvariation of atmospheric NH_3 at residential locations and to help estimate at locations in very close proximity to hog CAFOs (less than 1 km) to represent the protective buffer area between CAFOs and community locations.

Less exposed sites were also selected. These sites were located >2 km from an existing hog CAFO or any other large intensive livestock operation. These sites were located in areas with similar neighborhood characteristics as the exposed monitoring sites, and were located in the same counties.

2.3. Description of samplers

Passive diffusion tubes were utilized and assembled with parts supplied by Gradko International (Winchester, UK). The usual method is to have two stainless steel grids act as support for an acidic absorbent (typically 30 μl of 5% sulfuric acid). The grids are placed into a colored polyethylene cap which is fitted onto a PTFE diffusion tube body 35 mm long (Sutton et al., 2001; Walker, 2006; Walker et al., 2004a). The internal diameter of the tube was 11.1 mm (Walker, 2006; Walker et al., 2004a). A turbulence barrier consisting of a porous PTFE filter supplied by Gradko International was held in place by a polyethylene ring (Sutton et al., 2001) at the top of the diffusion tube. Assembled tubes were placed in polystyrene vials (Sutton et al., 2001) and then placed in Ball glass jars (Robarge, 2003) which were stored in the laboratory refrigerator at 4 °C until exposure in the field.

2.4. Coating, extraction and analysis of passive samplers

Preparation and analysis of passive tubes was performed by the North Carolina State University Analytical Service Laboratory (Raleigh, NC; http://www.soil.ncsu.edu/services/ASL). The laboratory utilized the method described in Fowler et al. (1998) (1% v/v solution of sulfuric acid) to prepare the passive samplers. The use of 30 μl of 1% v/v solution of sulfuric acid was sufficient to capture ammonia at the distances from the sources identified in this project. Typically, 2.5 ml of deionized water is used for extraction of the exposed samplers. This volume was reduced during the winter months in an attempt to increase the sensitivity of the samplers to detect NH_3 above blank values. Given the relatively low amounts of NH_3-N mass collected by the samplers, maintaining low blanks is critical in order to prevent analytical variance from exerting an influence on the results for the exposed samplers.

Analysis for the ammonium concentration in the distilled water extracts of the samplers was performed using a Dionex Model 600 Ion Chromatograph (Dionex Corp, Sunnyvale, CA) equipped with a Dionex CG12A 4 mm × 50 mm guard column and a CS12A 4 mm × 250 mm analytical column. The Dionex CSRS (cation self-regenerating) suppressor was set at 50 mV. The detector was a Dionex CD25A conductivity detector. The eluent was methanesulfonic acid (MSA).

2.5. Ammonia calculations and method validation

To calculate the ammonia collected by each diffusion tube, the following equations were used (Sutton et al., 2001; Wilson, 2005):

$$NH_{3a} = (Q/V), \quad (1)$$

where NH_{3a} is the concentration of NH_3 in the air in units of μg $NH_3\,m^{-3}$, Q is the mass of NH_3-N collected by the sampler, and V is the effective volume of air sampled,

$$NH_{3b} = NH_{3a} \times 1.44, \quad (2)$$

NH_{3b} is the concentration of NH_3 in air in units of ppb NH_3 and 1.44 is the conversion factor used to convert from $\mu g\,m^{-3}$ to ppb.

$$V = DAt/L, \quad (3)$$

where D (diffusion coefficient) equals $0.07524\,m^2\,h^{-1}$ at 25 °C. A (tube cross-sectional area) equals $9.5 \times 10^{-5}\,m^2$, t is the sampling duration (in h) and L (diffusion tube length) equals 0.035 m. V equaled $0.137\,m^3$ per month assuming a temperature of 25 °C.

Our confidence in the passive sampler to measure ammonia in Eastern NC was bolstered by studies which validated the use of passive samplers against reference methods (Walker, 2006; Sutton et al., 2001; Tang et al., 2001). Walker (2006) compared the passive sampler to a TEI Model 17C chemiluminescent NH_3 analyzer in the laboratory and during field deployment. Laboratory tests were

conducted to develop a calibration curve for the passive sampler and to assess potential saturation effects at high concentrations. The passives were exposed to a range of NH_3 concentrations in an exposure chamber to generate a calibration curve. Good agreement was found between the passives and NH_3 analyzer. The passive sampler underestimated concentrations by $<5\%$ in laboratory and field comparisons and a correction of 1.05 was applied to exposed samplers (Walker, 2006).

Saturation tests revealed that the sampler worked well at high NH_3 levels (Walker, 2006). While, Sutton et al. (2001) found at low levels (<3 ppb), there is more uncertainty associated with values, the method is less accurate, and the values are possibly less reliable. NH_3 levels above background were observed at locations >0.5 km from the emissions source (Walker, 2006; Walker et al., 2004a) which also supported our use of the passive method at community locations. We also feel that the issue of measurement accuracy at low levels is minimized by use of measurement error modeling (see Sections 2.6 and 2.7).

2.6. Lab blanks and adjusted NH_3 measurement

Laboratory blanks (LBs) were prepared with each set of field samplers. After coating in the laboratory, LBs ($n = 6$) were sealed in glass jars and stored at 4 °C. The purpose of the LBs was to assess the background NH_3 levels during handling of the samplers in the laboratory. Having six LBs provided a sample large enough from which to calculate a representative mean value. Therefore, a good estimate of the corresponding LB is the average $\overline{NH_{3blank\,i}}$ of the six LBs prepared for the sampler deployment when measurement i was taken.

For each sampler deployment, we exposed three samplers at the same monitoring location. Hence, for each i, we have triplicate raw NH_3 concentration measured values, $NH_{3raw\ i,j}$, $j = 1,2,3$. By subtracting the average LB $\overline{NH_{3blank\,i}}$ using equations from Sutton et al. (2001), we obtain the triplicate NH_3 adjusted measurements $NH_{3adjusted\ i,j} = NH_{3raw\ i,j} - \overline{NH_{3blank\,i}}$, $j = 1,2,3$.

Having triplicate NH_3 adjusted measurements for each i allowed us to obtain both the average NH_3 adjusted measurement value,

$$N_{mi} = \frac{1}{3}\sum_{j=1}^{3} N_{3\text{adjusted}\,i,j}, \tag{4}$$

as well as its measurement error variance:

$$\sigma_{Ni}^2 = \frac{1}{3}\sum_{j=1}^{3}(N_{3\text{adjusted}\,i,j} - N_{\text{m}i})^2. \tag{5}$$

The measurement error variance σ_{Ni}^2 provides an assessment of all the uncertainty associated with i, including uncertainty from sampling error and analytical measurement error for the exposed samplers as well as the LBs. The measurement error variances are used to develop the linear measurement error model (MEM) presented next.

2.7. Measurement error model and detection limit

We use a MEM for the true NH_3 concentration N given its measured value N_{m}. The variance of N, denoted as σ_N^2, provides an assessment of the measurement uncertainty associated with N_{m}. We used the following equation to obtain an adequate MEM for σ_N (Serre et al., 2006; Lee, 2005):

$$\sigma_N = \sigma 0 + kN_{\text{m}}. \tag{6}$$

The standard deviation σ_0 characterizes the smallest measurement uncertainty corresponding to very small measured values, and is directly related to the NH_3 detection limit (DL). The coefficient k is a positive slope quantifying the increase in measurement error as N_{m} increases. Insight is gained by defining the ratio σ_N/N_{m} of the measurement error standard deviation over the measured value, which we will refer to as the *relative error*. It follows directly from Eq. (3) that the relative error $\sigma_N/N_{\text{m}} = \sigma_0\ /N_{\text{m}} + k$ is asymptotically equal to k for large N_{m}, so that k is the asymptotic relative error. On the other hand, as we try to measure smaller NH_3 concentrations, the relative error increases rapidly in σ_0/N_{m}. In fact, at some point the relative error is too large to be acceptable. The concentration below which the relative error is unacceptable is called the DL. The following DL was used in this study (Robarge et al., 2002):

$$DL = 2\sigma_0. \tag{7}$$

2.8. Sampler deployment

The prepared passive samplers were obtained from the North Carolina State Analytical Services Laboratory and deployed at the study locations on the same day. The recovered exposed samplers were returned to the NC State laboratory also on the same day as deployment. This same day process was

used to ensure that there was minimal opportunity for the unexposed and exposed passive samplers to be exposed to potential ammonia contamination when the passive samplers were beyond the confines of the Analytical Services Laboratory.

To protect the passive tube samplers from adverse weather conditions such as high winds and precipitation, a shelter manufactured by Gradko International (Winchester, UK) was employed when the tube samplers were exposed to ambient air. The shelters were placed at heights between 1.5 and 3 m above the ground as in previous research (Tate, 2002; Rabaud et al., 2001; Walker, 2005). To expose the samplers to the air, caps were removed and the samplers oriented face-down in the shelter so that the flow of wind moved across the open face of the tube (Rabaud et al., 2001). Sheltering was also important because there can be a difference in the performance of sheltered and unsheltered tubes (Bush et al., 2001).

3. Results

3.1. Site selection and description of sampling sites

A network of passive sampling sites was established to monitor atmospheric ammonia levels in the Eastern NC study area. Fig. 2 displays the locations of 20 passive sampling sites during Phase I sampling of this research project. Samplers were deployed for 1 week time periods at sites in: Nash ($n = 2$), Edgecombe ($n = 6$), Wilson ($n = 2$), and Greene ($n = 10$) counties.

Additional sampling from July to October 2004 (Phase II) was focused in several of the counties (Fig. 3) that are part of the high emission density zone (Walker et al., 2000a, b) where most of the North Carolina hog inventory is located. These counties included Greene ($n = 9$), Duplin ($n = 13$), and Lenoir ($n = 1$), which equaled a total of 23 passive monitoring stations.

3.2. Measurement error and detection limit

Each adjusted measured NH_3 value N_{mi} obtained using Eq. (1) and the corresponding measurement error standard deviation σ_{Ni} obtained using Eq. (2) are shown in Fig. 4. In addition, the MEM obtained by fitting Eq. (3) on the experimental N_{mi} and σ_{Ni} data is shown with a plain lines in Fig. 4. The parameters of the MEM obtained by fitting to the experimental data are $\sigma_0 = 1.91$ ppb and $k = 0.104$. The corresponding DL $= 2\ \sigma_0 = 3.82$ ppb. In Fig. 4a, we show measurement error standard deviation σ_N as a function of the measured value N_m. This plot is

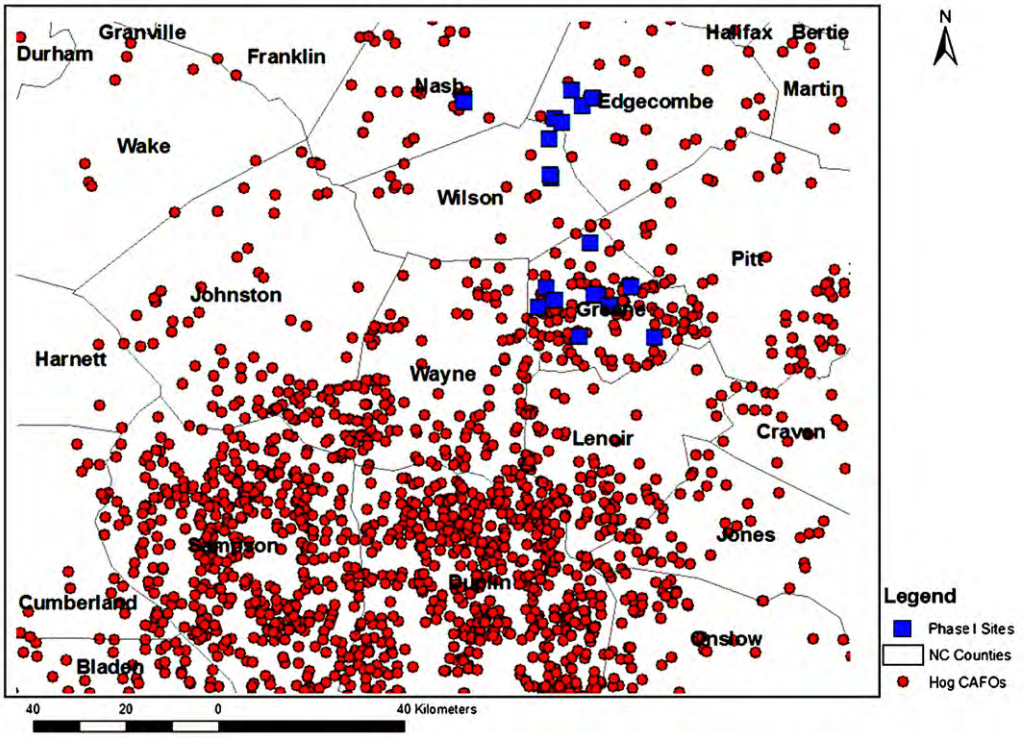

Fig. 2. Location of NH_3 monitoring sites and hog CAFOs during Phase I sampling.

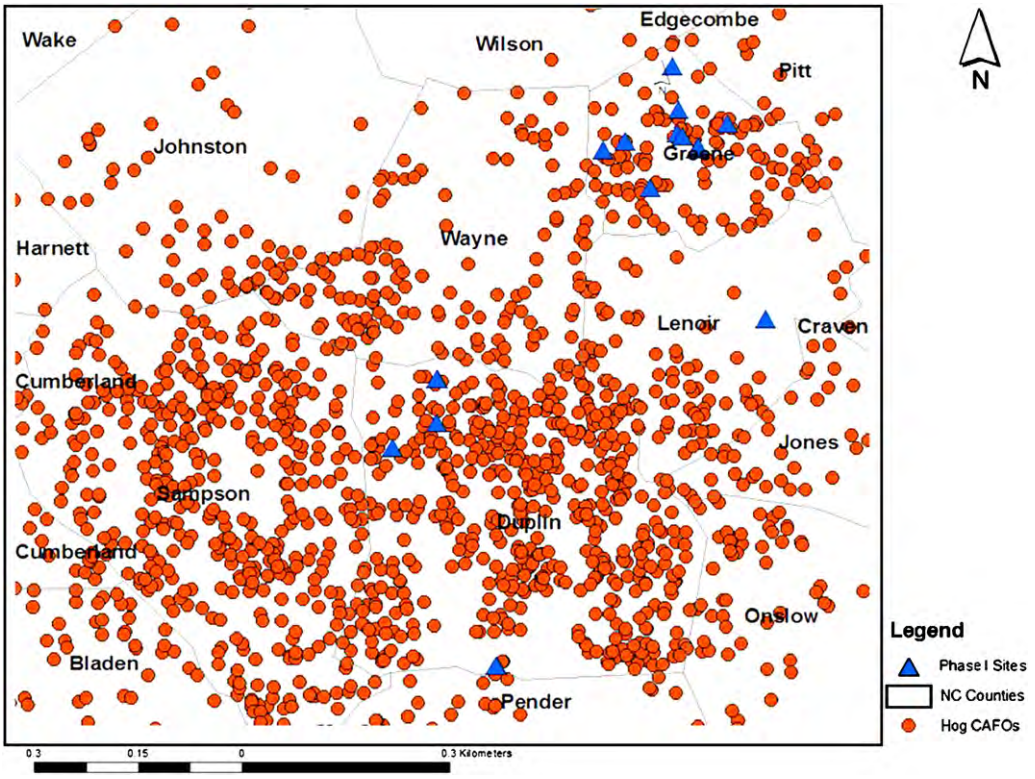

Fig. 3. Location of NH_3 monitoring sites and hog CAFOs during Phase II sampling.

useful to see the linear increase of σ_N with respect to the measured value N_m. In Fig. 4b, we show relative error σ_N/N_m as a function of the measured value N_m. This plot is useful to see the asymptotic behavior of the relative error for large N_m (i.e. σ_N/N_m tend to k when N_m is large). Conversely, when N_m becomes small, the relative error increases rapidly. Fig. 4b shows measurements greater than the DL (3.82 ppb) have small (acceptable) errors, while measurements below DL have large (unacceptable) errors.

A cumulative frequency plot (Fig. 5) shows that 35% of the expected NH_3 values were below the DL and 65% of the values were above the *DL*. Thus, the passive samplers were able to detect NH_3 at the community level in about 65% of the measurements detected. This finding is the best evidence that we completed our goal of validating the use of passive samplers to measure average NH_3 levels at the community level.

3.3. Statistical results

3.3.1. Phase I sampling

The blank-adjusted concentration of NH_3 (ppb) was obtained for each sampling site during Phase I sampling (October 2003 to May 2004). Table 1 displays descriptive statistics for Phase I sampling sites.

The mean concentration ranged from 4 ppb ($\pm$3 ppb) to 21 ppb ($\pm$18 ppb). The lowest value of 4 ppb was obtained for site 13. Similar levels near 5 ppb were obtained for site 16 and sites 6–8 (less exposed sites). Average levels near 20 ppb were obtained at sites 5 and 14. These sites were located in close proximity to one or more hog CAFOs (<1 km). The relationship between sampled concentration and distance from source will be explored in future publications.

3.3.2. Phase II sampling

Sampling occurred from July to October 2004 at 23 sites during Phase II sampling. The concentrations during Phase II sampling were higher than the concentrations obtained during Phase I sampling due to several factors: (1) there were higher atmospheric ammonia concentrations during the summer, (2) all of the sites were located in the high emission density zone, and (3) many of the sites were located at distances less than 1 km from a hog

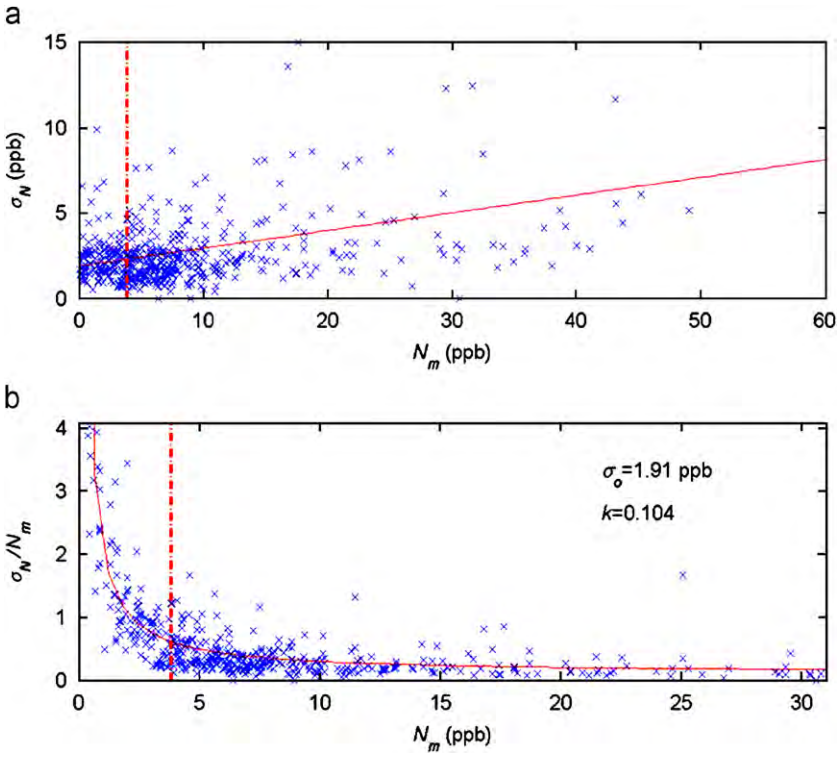

Fig. 4. Plot of (a) measurement error standard deviation σ_N versus measured value N_m, and (b) relative error σ_N/N_m versus N_m. Experimental values (Eqs. (1) and (2)) are shown with crosses, while MEM (Eq. (3)) is shown with a plain line.

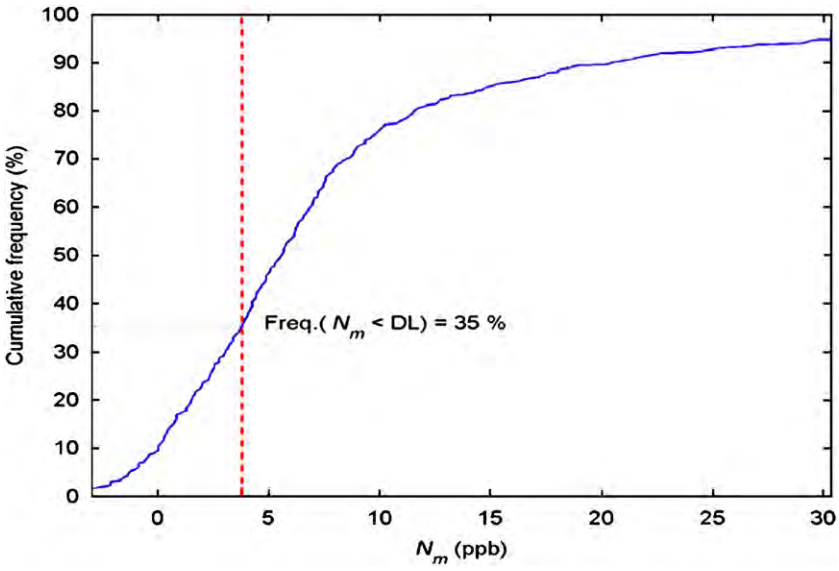

Fig. 5. Cumulative frequency plot of ammonia measured values N_m. Vertical dot-dashed line corresponds to detection limit (DL) = 3.82 ppb.

CAFO. Table 2 provides descriptive statistics for Phase II sampling sites.

The mean NH_3 levels ranged from 3 to 49 ppb. The highest mean level of 49 ppb was obtained at site 19, one of the sites at a residential location near a large number of CAFOs. Sites 17, 18, and 20 were collocated with site 19 and had average levels that ranged from 32 to 37 ppb. Elevated levels near

Table 1
Descriptive statistics for each passive sampling station during Phase I[a]

ID	Start date	Stop date	NH_3 concentration[b]	
			Mean	SD
1	16/10/03	11/5/04	7.38	8.08
2	16/10/03	11/5/04	6.28	3.00
3	16/10/03	11/5/04	10.36	8.45
4	16/10/03	16/3/04	5.51	1.92
5	16/10/03	11/5/04	21.09	17.5
6	16/10/03	11/5/04	5.00	1.76
7	16/10/03	11/5/04	4.91	1.52
8	16/10/03	11/5/04	5.20	2.32
9	16/10/03	11/5/04	9.57	4.84
10	4/11/03	11/5/04	6.77	2.96
11	2/12/04	11/5/04	7.92	3.13
12	2/12/04	11/5/04	6.65	2.40
13	2/12/04	23/3/04	4.23	2.89
14	2/12/04	11/5/04	19.59	17.8
15	9/12/04	11/5/04	8.64	8.81
16	23/12/04	11/5/04	4.80	1.63
17	23/12/04	11/5/04	11.25	9.82
18	30/12/04	11/5/04	8.42	3.53
19	16/3/04	11/5/04	7.34	1.78
20	23/3/04	11/5/04	7.45	3.24

SD: standard deviation. NA: not available.
[a]Phase I sampling occurred from October 2003 to May 2004.
[b]Airborne NH_3 concentration (in ppb) calculated from measured mass of NH_3-N collected.

Table 2
Descriptive statistics for each passive sampling station during Phase II[a]

ID	Start date	Stop date	NH_3 concentration[b]	
			Mean	SD
1	8/7/04	30/9/04	10.54	6.80
2	8/7/04	14/10/04	7.91	5.33
3	8/7/04	14/10/04	9.13	8.26
4	8/7/04	14/10/04	20.11	13.83
5	8/7/04	14/10/04	19.48	11.24
6	8/7/04	14/10/04	21.51	12.79
7	8/7/04	14/10/04	10.59	4.28
8	8/7/04	14/10/04	8.56	3.57
9	8/7/04	14/10/04	18.17	8.40
10	8/7/04	23/8/04	20.37	9.66
11	22/7/04	14/10/04	23.20	10.93
12	22/7/04	5/8/2004	9.57	3.93
13	5/8/04	2/9/2004	4.60	1.67
14	5/8/04	14/10/04	8.81	4.93
15	5/8/04	9/9/04	11.56	10.66
16	5/8/04	23/9/04	4.25	NA
17	2/9/04	14/10/04	36.63	3.82
18	2/9/04	14/10/04	32.12	3.98
19	2/9/04	14/10/04	48.95	28.76
20	9/9/04	14/10/04	32.12	2.18
21	30/9/04	14/10/04	11.57	NA
22	30/9/04	14/10/04	12.64	NA
23	23/8/04	14/10/04	3.34	NA

SD: standard deviation. NA: not available.
[a]Phase II sampling occurred from July 2004 to October 2004.
[b]Airborne NH_3 concentration (in ppb) calculated from measured mass of NH_3-N collected.

20 ppb were also obtained at sites 4–6 and 9–11. Sites 4–6 were collocated sites located at a residential location in close proximity to several CAFOs. Sites 9–11 were sites where monitoring occurred during Phase I and were located in close proximity to multiple CAFOs. Statistics for Phases I and II are displayed in Fig. 6.

3.3.3. Comparison of results to other North Carolina research

The NH_3 levels obtained in this study compare well with the NH_3 levels obtained in other studies on the levels of atmospheric NH_3 at or near hog CAFOs in eastern NC. The mean seasonal values obtained for this study were 10.9 ppb (spring), 16.7 ppb (summer), 10 ppb (fall) and 8.1 ppb (winter). The spring, summer and fall values are comparable to the values obtained by Robarge et al. (2002), Phillips et al. (2004), and McCulloch et al. (1998). When NH_3 values from this study are compared to the seasonal values from other studies, we see that the data corresponds well to the measurements found in published literature. The data presented in this research are consistent with what is expected for eastern NC. The comparison between the data from this work and previous studies also validates that passive sampling was able to measure elevated atmospheric NH_3 at the community level.

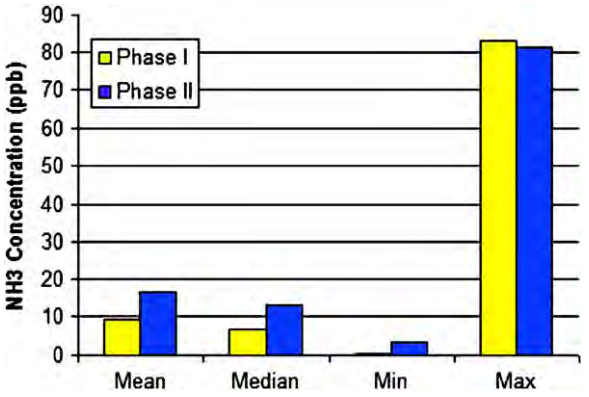

Fig. 6. Descriptive statistics for pooled Phases I and II sampling.

4. Discussion

In an effort to obtain NH_3 data at the community level needed for human exposure assessment, we devised and executed a study to address this gap. The first obstacle to overcome was the selection of an appropriate sampling technology. A review was undertaken of the available real-time sampling technologies that met the following criteria: (1) low detection limit (ppb), (2) accuracy at low levels, (3) high precision, (4) and quick response time. This review included examining technologies such as the annular denuder system (ADS) used by Robarge et al. (2002) and NH_3 chemiluminescent analyzers and field testing some equipment.

Unfortunately, most of these systems were too expensive. For example, the TEI Model 17C NH_3 chemiluminescent analyzer costs $17,000, the Pranalytica Nitrolux costs $36,000, and denuder system equipment and analysis costs at one location for a year exceed $30,000. While, passive diffusion tubes cost $7 each and require minimal analysis costs. Active systems also required complex maintenance, temperature controlled environments, electricity, and various calibrations. We selected passive diffusion tubes because they were: (1) inexpensive, (2) able to detect gases at relatively low levels, (3) easy to handle and transport, (4) small and inconspicuous, (5) readily secured, (6) non-electric, and (7) able to provide good spatial coverage. The passive tube method based on the Fowler design (Fowler et al., 1998) was chosen because of the minimal purchase, processing and analysis costs. In addition, the work would be performed by a local NC State University laboratory very proficient using the method and had proper QA/QC procedures.

Passive monitors were positioned at community locations near swine CAFOs within a 1, 1–2, and 2–5 km buffer. Sites less than 2 km from the nearest CAFO were selected as exposure sites based on previous work that: (1) measured and modeled odors emanating from CAFOs from <100 to 5000 m from the nearest source (Chen et al., 1998; Carney and Dodd, 1989; Chastain and Wolak, 1999), (2) reported frequency of respiratory symptoms and mood disorders in a community within 3 km of a hog CAFO in comparison to a community >3 km from the nearest hog CAFO (Wing and Wolf, 2000); and (3) measured atmospheric ammonia levels as a function of distance from CAFOs (Fowler et al., 1998). The conservative distance of 2 km was selected to delineate between exposed sites (<2 km from the nearest hog CAFO) and less exposed sites (>2 km from the nearest CAFO). It was assumed that sites within 2 km of the nearest CAFO would experience a higher frequency of elevated ammonia than less exposed sites. Defining exposed sites as those within 2 km of a hog CAFO presumed that CAFOs would be the dominant ammonia source for population receptors at these locations.

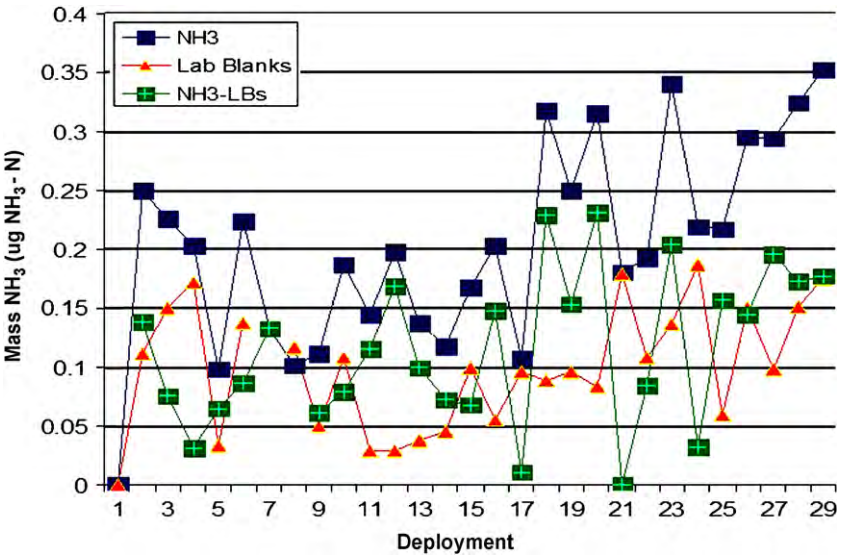

Fig. 7. NH_3 versus Lab Blank Data for Phase I. Line with solid squares represents average mass NH_3-N (signal) collected across all sites during each Phase I deployment. Line with triangles represents average mass of NH_3-N in the laboratory blanks (noise) for each Phase I deployment. Line with squares containing + signs represents difference between signal and noise.

Fig. 7 shows that after adjustment, mean NH_3 levels (signal) > LBs (noise) were above zero for 27 out of 28 deployments. It provides evidence that supports the ability of the passive diffusion method to measure atmospheric ammonia levels at community locations near hog CAFOs. Figs. 4a and 4b present MEM evidence (a relatively low DL of 3.82 ppb), combined with the fact that as much as 65% of the adjusted NH_3 values were above DL, signify that the passive sampling method was effective at measuring ammonia at homes and schools in Eastern NC near hog CAFOs. The research was able to positively answer the question: is it possible to develop and implement a monitoring strategy to measure atmospheric ammonia at the community level?

NH_3 was measured above ambient levels during Phases I and II. The mean weekly levels ranged from 4 to 21 ppb during Phase I and 3 to 49 ppb during Phase II (see Tables 1 and 2). The highest mean NH_3 levels (>10 ppb) were obtained at sites 3, 5, 14 and 17 (Phase I) and sites 1, 4–7, 9–11, 15, 17–22 (Phase II) which were all located less than 2 km from the nearest hog CAFO. Mean level of 13.8 ppb near homes and schools (less than 2 km from a hog CAFO) was 4–12 times greater than ambient background levels (1–3 ppb), reaching as high as 80 ppb. Exposed sites had a mean level of 12.8 ppb, which was two times higher than the mean level of 5.5 ppb at less exposed sites. Mean NH_3 level of 15.4 ppb at sites in high emission density counties was 3 times higher than the mean level of 5.3 ppb at sites in low emission density counties.

The values obtained during Phase II sampling were approximately twice as high as the mean concentrations obtained during Phase I sampling. This observation can be explained by the seasonal variation of atmospheric ammonia. Phase I sampling occurred during the fall, winter and spring while Phase II sampling occurred primarily during the warmest period of the year (July to mid-October 2004). Similar results were found in previous research performed to measure ambient NH_3 levels in Eastern NC in an area with a high density of animal production facilities (Robarge et al., 2002; Walker, 2005; Walker et al., 2004b).

The Phase II results indicate that the exposed sites had a high mean concentration. It can be inferred that there may be possible exposure risks for human populations that reside near the exposed sampling sites or at community locations with similar atmospheric NH_3 concentrations such as residential locations in Duplin and Sampson, high emission density counties in Eastern NC. These two counties have more than 60 hogs per person which means populations will be potentially exposed to higher levels of atmospheric NH_3 due to the higher hog density in the local environment.

The strength of the data stems from its ability to be employed as a measure of potential frequency of exposure to atmospheric NH_3 levels at community locations near industrial hog CAFOs. It is important to employ the data as a relative measure of exposure instead of an absolute measure of exposure. Additional work needs to be performed using a reference method to validate passive data and illustrate the temporal relationship between exposure and possible adverse health effects.

5. Conclusions

This study shows that the passive diffusion method can be used to measure NH_3 levels beyond the property line of the hog operation and above known ambient levels near homes and schools in Eastern NC. We see from Tables 1 and 2 and Fig. 7 that the method was able to obtain a NH_3 signal after adjustment using the LBs. With longer deployment times, even lower detection limits could have been obtained. This could have provided more evidence to support the supposition: passive samplers were able to measure NH_3 representative of average concentrations present at the community level near exposed homes and schools. The conclusion that the method worked is further supported by the relationship found between distance and concentration. NH_3 was measured near community locations above 1–3 ppb at distances < 1–5 km from the nearest hog operation in high and low emission density counties.

Our results reveal there is human exposure to NH_3 beyond the property line of hog CAFOs. Appreciable integrated NH_3 concentrations were found at exposed sites above ambient levels for an agricultural region in Eastern NC (Walker et al., 2004b). These results imply there are potential zones of exposure for human populations who reside in neighborhoods within 2 km and beyond from industrial hog operations. As the distance between the human population increases from the industrial hog operation, the levels of ammonia decrease due to dispersion, mixing and transformation. In future papers and research, we will explore the "exposure zone theory" and use calibration

curve data (Walker, 2006) as "soft data" in our spatiotemporal assessment of atmospheric NH_3 (Serre et al., 2006).

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Attachment A8

Air Pollution and Odor in Communities Near Industrial Swine Operations

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Background: Odors can affect health and quality of life. Industrialized animal agriculture creates odorant compounds that are components of a mixture of agents that could trigger symptoms reported by neighbors of livestock operations.

Objective: We quantified swine odor episodes reported by neighbors and the relationships of these episodes with environmental measurements.

Methods: Between September 2003 and September 2005, 101 nonsmoking volunteers living within 1.5 mi of industrial swine operations in 16 neighborhoods in eastern North Carolina completed twice-daily odor diaries for approximately 2 weeks. Meteorological conditions, hydrogen sulfide, and particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}) were monitored in each neighborhood. We used mixed models to partition odor variance within and between people and between neighborhoods, and to quantify relationships between environmental factors and odor.

Results: Participants reported 1,655 episodes of swine odor. In nine neighborhoods, odor was reported on more than half of study-days. Odor ratings were related to temperature, PM_{10}, and semivolatile PM_{10} in standard but not mixed models. In mixed models, odor increased 0.15 ± 0.05 units (mean ± SE) for a 1-ppb increase in H_2S, and 0.45 ± 0.14 units for a 10-μg/m^3 increase in PM_{10} at wind speeds > 6.75 miles per hour. The odds of reporting a change in daily activities due to odor increased 62% for each unit increase in average odor during the prior 12 hr (*t*-value = 7.17).

Conclusions: This study indicates that malodor from swine operations is commonly present in these communities and that the odors reported by neighbors are related to objective environmental measurements and interruption of activities of daily life.

Key words: agriculture, air pollution, community-based participatory research, environmental justice, epidemiology, quality of life, rural health. *Environ Health Perspect* 116:1362–1368 (2008). doi:10.1289/ehp.11250 available via *http://dx.doi.org/* [Online 5 June 2008]

There is a long history of medical interest in the health impacts of environmental malodor, from Hippocrates to William Farr, England's first Registrar General. In recent decades, scientific consideration of the health consequences of malodors has increased in the context of residential exposures to malodors from municipal solid waste landfills; wastewater treatment; land application of treated sewage sludge; industrialized animal operations; and the production, storage, and transport of industrial chemicals (Schiffman et al. 2000). Environmental malodors may prompt reports of annoyance, worry, and physical symptoms (Shusterman 2001). The extent to which malodor is an aesthetic issue versus a threat to health is a subject of scientific investigation and litigation that has important implications for environmental regulation, public health, and environmental justice (Thu 1998).

Odorant compounds can affect human health via several mechanisms (Schiffman et al. 2000; Shusterman 1992). First, at concentrations high enough to stimulate the trigeminal nerve, odorant chemicals may produce irritation of the eyes, nose, and throat, or other toxicologic effects. In this case, the toxicologic properties of the odorous molecules, rather than odor, produce symptoms. Second, via innate aversion, conditioning, or stress responses, odorant compounds can induce symptoms such as nausea, vomiting, headaches, stress, negative mood, and a stinging sensation at concentrations higher than the olfactory nerve threshold but below the trigeminal nerve threshold (Schiffman 1998; Schiffman et al. 2000; Shusterman 1992, 2001; Shusterman et al. 1991). Third, symptoms occurring in response to odorant mixtures may be due to a nonodorant component such as endotoxin, which can induce inflammation and airflow obstruction (Kline et al. 1999).

Odors may be quantified in natural settings or by laboratory analysis of ambient air samples using trained odor panels, scentometers, olfactometers, or electronic noses (Schiffman et al. 2001, 2005); however, transient and unpredictable odors are difficult to quantify. Although spontaneous reports of malodor may be quantified (e.g., Aitken and Okun 1992; Drew et al. 2007), this approach mixes variation in odor with variation in people's propensities to report odors and the limited availability of public agencies or researchers to track reports.

Research on malodors from concentrated animal feeding operations (CAFOs) and the consequences of these malodors for the health and quality of life of nearby neighbors has increased with expansion of industrial animal agriculture. Recent studies report that CAFO neighbors experience elevated levels of gastrointestinal and respiratory tract symptoms (Thu et al. 1997; Wing and Wolf 2000), wheezing and asthma (Merchant et al. 2005; Mirabelli et al. 2006; Radon et al. 2007), and decreased secretion of salivary IgA during episodes of high odor (Avery et al. 2004). Research on malodor is of interest in the context of broader impacts of industrial livestock production on energy use, diet, air and water pollution, and occupational health and safety (Donham et al. 2007; Thu 2002).

The purpose of this study was to quantify the reports of hog odors made by neighbors of swine CAFOs. To address a common limitation of research into connections between odor and health based on self-report without objective measures, we measured hydrogen

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This paper has not been subjected to the U.S. EPA's required peer and policy review, and therefore does not necessarily reflect the views of the agency and no official endorsement should be inferred. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

In exchange for a report and testimony, a law firm representing plaintiffs in a civil suit about impacts of an industrial swine operation on its neighbors contributed $2,000 to the University of North Carolina in support of S.W.'s research. K.T. has served as a consultant and provided testimony in civil suits regarding impacts of industrial swine operations on neighbors, and has received funding from the Iowa Pork Producers Association and the National Pork Producers Council. S.S.S. has received funding from the National Pork Board, the North Carolina Pork Council, and the Smithfield Agreement between Smithfield Foods and the State of North Carolina. The remaining authors declare they have no competing financial interests.

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sulfide, a product of anaerobic decomposition of hog waste, and particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}), which can transport odorant chemicals (Bottcher 2001); at the same time participants rated the strength of hog odor. Swine CAFOs are located disproportionately in low-income communities of color (Wilson et al. 2002; Wing et al. 2000), where fear of reprisals and community discord may discourage residents from reporting malodors and health concerns to health or environmental officials (Wing 2002), thus limiting the possibility of obtaining data about odor from public records. The Community Health Effects of Industrial Hog Operations study used community-based participatory research methods to increase the completeness and quality of data collection while promoting community organizing for environmental justice (Wing et al. 2008).

Materials and Methods

Setting and data collection. From September 2003 through September 2005 we collected data in eastern North Carolina, an area with one of the world's highest densities of swine production. Volunteers were recruited through community-based organizations. Nonsmoking adults ≥ 18 years of age who lived within 1.5 mi of at least one swine CAFO and had a freezer in their home (for storage of saliva samples) were eligible to be enrolled. Participants in each neighborhood attended a structured training session at which they practiced data-collection activities. Odor sensitivity threshold was evaluated by asking participants to choose which of two vials had an odor; one vial contained distilled water and the other contained butanol. Participants were presented up to 12 pairs of vials in series. The concentration of butanol increased 2-fold with each successive pair, beginning with 10 ppm. We defined odor sensitivity as the lowest concentration of a series of five correct choices.

Twice daily for 2 weeks (three neighborhoods chose to continue up to 7 additional days) participants sat outside their homes for 10 min at times agreed upon during the training session, usually morning and evening. They used a structured diary to report the strength of hog odor and information about health and quality of life. During their 10 min outside, participants were asked to recall the strength of hog odor inside at home, outside at home, and away from home for each hour of the day since their last diary entry. In this study we examined the ratings of hourly outdoor odor as well as hourly indoor odor reported in this portion of the diary. Participants also rated the current strength of hog odor at the end of the 10-min period. We analyzed these twice-daily odor ratings, which were made in the same locations at preselected times of day, in relation to odor sensitivity and environmental variables. Odor was rated on a 9-point scale from 0 (none) to 8 (very strong). Participants also indicated whether they had changed activities or decided not to do something because of hog odor.

We placed a small farm trailer with air monitoring equipment in each neighborhood. Locations were chosen to be as inconspicuous as possible but free from trees or structures that could affect air flow. We used a tapered element oscillating microbalance ambient particulate monitor Series 1400a with a Series 8500 filter dynamics measurement system (Rupprecht and Patashnick Co, Inc., East Greenbush, NY) to record hourly values of PM_{10} and semivolatile PM_{10}. Semivolatile particles are composed of compounds that simultaneously have meaningful concentrations in both vapor and condensed phases. PM_{10} values were updated every 6 min. An MDA Scientific single point monitor (Zellweger Analytics, Inc., North America, Lincolnshire, IL) provided concentrations of H_2S (parts per billion) averaged over 15-min intervals. Temperature, humidity, wind speed, and wind direction were recorded every 10 min with a Vantage Pro Weather Station (Davis Instruments, Hayward, CA), and every 30 min with a Young Model 05103VM-42 Wind Monitor (R.M. Young Company, Traverse City, MI). The Davis wind speed data were more complete, but the instrument was less sensitive, with values about 2 mi/hr (mph) lower than the Young monitor. To fill in missing data from each machine, values from the two machines were collectively categorized as low (≤ 0.57 mph), medium (0.58–6.75 mph), or high (> 6.75 mph). In four communities, data were missing for both weather instruments for some periods. In these cases, which comprise about three percent of total records, data were obtained from the nearest airport weather station, which was about 4.5 mi away for three communities and 18.5 mi away in one.

In each neighborhood a local "community monitor" was shown how to check the operation status of the monitoring equipment and was asked to call research staff on a toll-free line to report any outage or error message. In 12 neighborhoods a study participant served in this capacity.

We calculated the number of swine CAFOs within 2 mi of the monitoring platform using latitude and longitude coordinates derived from online satellite imagery and operating permits issued by the North Carolina Division of Water Quality (Raleigh, NC). Although we used 1.5 mi as the criterion for study elgibility, we counted operations within 2 mi because *a*) odor reports are made from that far away; *b*) that distance has been used in previous research (Thu et al. 1997; Wing and Wolf 2000); and *c*) excess wheezing symptoms have been reported as far as 3 mi from swine CAFOs (Mirabelli et al. 2006). Coordinates for the monitoring trailer and each participant's home were determined using a handheld global positioning system device.

Following input and approval from the Community Research Advisory Board of the Concerned Citizens of Tillery (Tillery, NC) the study protocol and survey instruments were approved by the University of North Carolina's Institutional Review Board for research involving human subjects, which follows national and international standards. All participants gave informed consent. We obtained a Certificate of Confidentiality from the National Institutes of Health because of legal measures taken by the North Carolina Pork Council to obtain identifiable participant information from a prior study (Wing 2002).

Statistical analysis. We evaluated relationships between environmental measurements and twice-daily odor by stratification, standard linear regression, and linear mixed models. We chose the measure of twice-daily odor for these analyses because these odor ratings were provided in real time and at preselected periods, and therefore should be less susceptible to recall bias than ratings of hourly odor since the previous diary entry. The sample sizes for these analyses varied based on the numbers of missing values for environmental measurements. Although hog-odor ratings were highly right-skewed, the number of observations was adequate to produce normal sampling distributions for the regression coefficients (Lumley et al. 2002); therefore, untransformed odor was considered as a continuous dependent variable in our linear regression models. Hourly average H_2S, temperature, humidity, and wind speed for hours centered at the time of sitting outside were considered as predictors of odor. We considered H_2S levels for hours when all measurements were below the detection limit of 2 ppb to be zero.

Mixed models with twice-daily odor as the dependent variable and environmental measures as independent variables were fit using the SAS MIXED procedure (SAS Institute Inc., Cary, NC) to account for variance within people, between people, and between neighborhoods. We compared Akaike information criterion (AIC) statistics for fixed-slope and random-slope models and chose models with lower AIC statistics for presentation. We fit models with intercepts when the only predictor of odor is coded as an indicator variable, providing a test of the difference between the omitted category and the other category or categories. For models with the interaction of a variable coded as continuous and one coded as an indicator, we fit models with no intercept to provide an estimate of the effect of the continuous variable, its SE, and a test of difference from zero, at each level of the indicator variable.

We used mixed logistic regression for analyses of activity limitation as the dependent variable. Average hourly outdoor odor since the previous diary entry was the independent variable. Models were fitted using the SAS GLIMMIX procedure. Random intercepts and fixed effects of average odor ratings of 1 to < 2, 2 to < 3, 3 to < 5, and ≥ 5 compared with no odor were estimated as predictors of activity limitation due to odor, coded as a 0/1 variable. A model was also fit with average hourly odor as a continuous variable.

SEs of regression coefficients are presented as measures of precision in order to reduce the probabilistic interpretations implied by the use of confidence intervals. For the same reason, we assessed contributions of predictors to the fit of models by *t*-tests instead of *p*-values because this is not a randomized study (Greenland 1990).

Results

Neighborhood and participant characteristics. A total of 102 volunteers from 16 neighborhoods enrolled in the study. One person who had difficulty with the study protocol was excluded from analyses. Analyses here include 84 people who collected data for 2 weeks, 15 (from three neighborhoods) who chose to continue an additional 4–7 days, and 2 who stopped before 2 weeks. Sixty-six women and 35 men participated. Age ranged from 19 to 89 years, with a mean age of 53. Eighty-four participants identified themselves as black, 15 as white, one as black/Native American, and one as Latino.

Characteristics of study neighborhoods, labeled A–P, are given in Table 1. Two neighborhoods had one swine CAFO within 2 mi of the monitoring trailer, and six neighborhoods had ≥ 10 within 2 mi. Approximately two-thirds of participants lived in neighborhoods within 2 mi of ≥ 5 swine CAFOs. In nine neighborhoods, participants reported outdoor swine odor on more than half the study days. Mean temperature on study days ranged from 47°F in neighborhood A to 82°F in neighborhood K; no neighborhoods participated during January. Mean H_2S was 0.004 ppb in neighborhood E, where 99.8% of readings were below the detection limit (2 ppb). Neighborhoods O and C had the highest mean values, 1.02 and 1.48 ppb, respectively, and the highest values recorded in neighborhood O were at the upper limit of detection, 90 ppb. Average PM_{10} varied from 10.8 μg/m^3 in neighborhood A to 28.7 μg/m^3 in neighborhoods C and E, whereas semivolatile PM_{10} was highest (9.2 μg/m^3) in neighborhood O and lowest in H (–3.2 μg/m^3), indicating the high degree of measurement error when using the microbalance to characterize semivolatile particle levels over short time periods.

Frequency, magnitude, and duration of odor episodes. We calculated the average daily odor that participants reported following the twice-daily preselected 10-min periods of sitting outdoors, as well as the average hourly outdoor odor reported each day. Study participants collected data on 1,495 days, although twice-daily odor was missing for 39 of these days. Results for the 1,456 days with twice-daily odor information are reported here (Table 2). The average twice-daily odor was zero for 563 days (38.7%), and > 5 on 51 days (3.5%). Average hourly outdoor odor was zero for 591 days (40.6%) and > 5 on 33 days (2.3%). Average twice-daily odor was zero on fewer days than average hourly odor. This is possible because participants could report nonzero odor during twice-daily times sitting outdoors when there was no odor at other times during the hour.

Reported hourly outdoor odor was highest in the mornings and evenings and lowest in the middle of the day and night (Figure 1). Morning odor was highest around 0300 hours (mean = 1.7) when 12.2% of ratings were ≥ 5. Mean hourly odor was 2.1 at 2000 hours, when 19.2% of odor ratings were five or greater.

Based on hourly outdoor odor ratings, participants reported 1,655 odor episodes (Table 3). The duration of an episode is the number of consecutive hours that swine odor was reported to be above zero. The majority of episodes (62.1%) lasted 1 hr, whereas 9 episodes (0.5%) lasted ≥ 9 hr. Average odor was < 2 for about 39% and > 5 for about 16% of odor episodes lasting 1 or 2 hr. Average strength was ≥ 5 for > 21% of odor episodes of ≥ 3 hr.

Hog odor was reported inside homes on 185 of 1,456 person-days of follow-up (12.5%). Five hundred episodes of indoor hourly odor were reported, of which 233 (46.6%) lasted 1 hr, 179 (35.8%) lasted 2–3 hr, and 88 (17.6%) lasted ≥ 4 hr. Three of the 1-hr indoor odor episodes, rated 3, 6 and 8, were reported in the middle of time periods when consistent sleep was indicated.

Butanol odor sensitivity threshold was estimated for 98 participants, of whom 39 had a threshold of 10 or 20 ppm (Table 4). Most odor ratings were provided by people with butanol detection thresholds between 10 and 160 ppm. Average reported odor declined with sensitivity from 20 to 160 ppm. Among the 12 participants with odor thresholds of ≥ 320 there was not a clear relationship between odor sensitivity and average odor.

Environmental correlates of odor. Analyses of environmental correlates were based on the

Table 2. Daily averages of twice-daily and hourly outdoor odor ratings (scale of 0–8).

Mean odor rating	Twice-daily odor [no.(%)]	Hourly outdoor odor [no. (%)]
0	563 (38.7)	591 (40.6)
> 0 to < 2	541 (37.2)	581 (39.9)
> 2 to < 5	301 (20.7)	251 (17.2)
≥ 5	51 (3.5)	33 (2.3)
Total	1,456 (100.0)	1,456 (100.0)

Table 1. Characteristics of neighborhoods and CAFOs within 2 mi of the monitoring platform.

Site	Swine CAFOs (no.)	Participants (no.)	Mean 10-min odor	Days with any odor outdoors (%)	Days with any odor indoors (%)	Mean temp (F)	Mean H_2S (ppb)	H_2S values < 2 ppb (%)	Highest H_2S (ppb)[a]	Mean PM_{10} (μg/m^3)	Mean semivolatile PM_{10} (μg/m^3)
A	1	7	0.4	26	2	47	0.01	99.7	4	10.8	1.1
B	1	6	0.7	48	10	50	0.09	97.0	9	13.6	1.8
C	3	5	1.4	70	14	60	1.48	77.1	28	28.7	2.7
D	3	6	0.8	68	9	59	0.41	90.7	20	13.7	1.4
E	4	7	0.5	20	15	77	> 0.00	99.8	2	28.7	5.9
F	4	4	2.7	95	46	77	0.15	94.2	10	28.4	3.9
G	5	4	0.6	41	2	51	0.07	96.7	3	17.5	5.0
H	9	6	1.0	45	9	63	0.02	98.9	3	16.8	–3.2
I	9	9	2.9	88	23	80	0.40	90.9	20	27.0	7.5
J	9	4	1.9	63	15	79	0.40	91.2	52	21.7	3.5
K	10	8	1.3	73	12	82	0.28	93.3	21	22.8	8.6
L	12	7	0.8	43	3	71	0.05	97.6	4	23.0	4.6
M	12	10	2.1	73	11	75	0.05	98.6	27	17.1	1.6
N	15	5	0.9	49	13	59	0.01	99.5	4	27.3	4.6
O	15	5	1.8	68	26	77	1.02	91.1	90	18.7	9.2
P	16	8	1.2	66	10	59	0.08	97.3	9	19.1	6.5

temp, temperature.
[a]Based on 15-min average values.

twice-daily odor ratings reported at preselected times of day when participants sat outdoors for 10 min. Table 5 provides results of bivariate simple linear regression models for each environmental variable as a predictor of 10-min odor ratings. Odor ratings increased 0.26 ± 0.02 (mean ± SE) for every 10°F increase in temperature; the *t*-test value is large (11.65). Odor ratings increased 0.17 ± 0.02 for every 1-ppb increase in H_2S, 0.04 ± 0.02 for a 10-μg/m^3 increment in PM_{10}, 0.03 ± 0.01 per 1 μg/m^3 of semivolatile PM_{10}, and 0.06 ± 0.02 for a 10% increase in relative humidity. Average odor at moderate wind speeds was 1.02. Compared with moderate wind speeds, odor was higher by 0.43 ± 0.08 at low wind speeds and higher by 0.72 ± 0.15 at high wind speeds.

Temperature and semivolatile PM_{10} showed little association with 10-min odor ratings as main effects in mixed models (data not shown). Table 6 presents effect parameters from mixed models with other environmental variables. The relationship between H_2S and odor was best fit with a random-intercept, random-slope model, in which odor increased 0.15 ± 0.05 (mean ± SE) for every 1-ppb increase in H_2S (*t*-value for H_2S = 3.10).

Because there is a strong main effect for H_2S, we considered odor sensitivity as a modifier of its association with odor. H_2S was positively related to odor among participants with detection thresholds of ≤ 160 ppm (0.17 ± 0.06/1 ppb, mean ± SE), but not among participants with thresholds of ≥ 320 ppm (0.02 ± 0.14/1 ppb).

The relationship between wind speed and odor was adequately fit with a random-intercept, fixed-slope model. Parameters for low and high wind speeds were estimated in mixed models with medium wind speed as the referent (Table 6). Average odor was lowest at medium wind speed (1.23 ± 0.20, mean ± SE). Compared with the odor at medium wind speed, odor was higher by 0.18 ± 0.07 units at low wind speeds and by 0.38 ± 0.13 units at high wind speeds.

Relationships between odor, H_2S, and PM_{10} depended on wind speed (Table 6). A mixed model with fixed effects for wind speed and random effects for H_2S showed that H_2S and odor were not associated at medium wind speed (–0.09 ± 0.10/1 ppb, mean ± SE). At low wind speeds, odor increased 0.28 ± 0.11/1 ppb (*t* = 2.49), and at high wind speed there was an increase of 0.77 ± 0.44/1 ppb (*t* = 1.75). In contrast, PM_{10} was associated with odor at high wind speeds (0.45 ± 0.14/10 μg/m^3; *t* = 3.14), but not at low or medium wind speeds.

Activity limitation. On 118 occasions 34 participants reported that they cancelled or changed an activity because of hog odor. Typical changes included closing windows, avoiding sitting in the yard and socializing with friends, cancelling plans to barbecue, not putting clothes out to dry, declining exercise via outdoor walks, not putting up Christmas lights, not being able to garden or mow the lawn, not washing the car, or not being able to sit on the porch. One participant reported on two occasions that odor made it difficult to sleep. Whereas in other records this participant reported 6–8 hr of sleep during the previous night, on these two occasions he or she indicated having slept either 0 or 4 hr. The common theme in these disruptions was the adverse impact of odor on people's social and

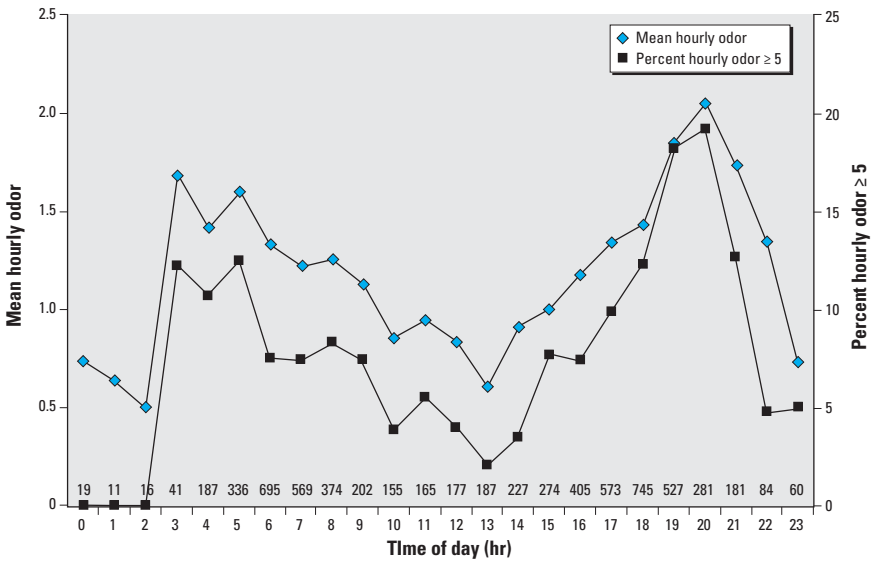

Figure 1. Time of day and odor. Numbers above the *x*-axis indicate the number of hourly ratings for that time point.

Table 3. Duration and strength of reported outdoor odor episodes.

	Duration of hourly outdoor odor episode (hr)					
Mean odor	1 [no.(%)]	2 [no.(%)]	3 [no.(%)]	4–8 [no.(%)]	≥ 9 [no.(%)]	Total
1 to < 2	398 (38.8)	126 (38.5)	30 (18.9)	29 (21.8)	3 (33.3)	586 (35.4)
2 to < 5	462 (45.0)	152 (46.5)	89 (56.0)	76 (57.1)	4 (44.4)	783 (47.3)
≥ 5	167 (16.3)	49 (15.0)	40 (25.2)	28 (21.1)	2 (22.2)	286 (17.3)
Total	1,027 (100.0)	327 (100.0)	159 (100.0)	133 (100.0)	9 (100.0)	1,655 (100.0)

Table 4. Butanol odor sensitivity threshold and mean twice-daily odor.

Butanol (ppm)	No. of participants	No. of twice-daily odor ratings	Mean odor
10	18	503	1.51
20	21	575	1.64
40	15	405	1.32
80	14	396	1.08
160	17	479	0.85
320	4	97	1.39
640	5	125	1.25
1,280	1	20	1.55
2,560	1	27	4.89
5,120	1	28	2.07
20,480	1	28	1.00

Table 5. Simple linear regression coefficients for environmental predictors of odor.

	No. of records	Coefficient	SE	*t*-Value
Temperature (× 10)	2,772	0.26	0.02	11.42
H_2S (ppb)	2,701	0.17	0.02	8.73
PM_{10} (10 μg/m^3)	2,005	0.03	0.02	1.89
Semivolatile PM_{10} (μg/m^3)	2,005	0.03	0.01	2.90
Humidity (10%)	2,772	0.05	0.02	2.91
Low wind	1,617	0.43	0.08	5.73
Medium wind (intercept)	972	1.02	0.06	16.96
High wind	183	0.73	0.15	4.87

personal space. There was an association between activity change and average outdoor odor intensity during the 12 hr prior to a diary record, with odor grouped into several levels (Table 7). Participants noted changes in activity due to odor from 1.4% of occasions when average odor was < 1.0 up to 16.2% when average odor was ≥ 5.0. Estimates from logistic mixed models with random intercepts and a fixed slope for odor show a similar relationship; all model coefficients are substantially larger than their SEs, and *t*-values are large. A separate model was estimated for odor as a continuous variable; the log odds ratio of activity change for a one-unit increase in odor is 0.48 ± 0.07, a 62% increase in the odds of activity change per odor unit ($t = 7.17$).

Discussion

In the present study 101 participants from 16 neighborhoods in eastern North Carolina reported on the strength of hog odor inside and outside their homes for approximately 2 weeks while temperature, humidity, wind speed, H_2S, and PM_{10} were monitored nearby. One to 16 swine CAFOs were located within 2 mi of the monitoring platform in each neighborhood. Odor was reported outside on more than half the study days in 9 neighborhoods. Odor ratings made during 10-min periods of sitting outside twice a day were associated with weather conditions, H_2S, and PM_{10}. One-third of participants reported ceasing or changing their activities due to malodor, and the intensity of odors reported between diary entries was strongly associated with these reports. This study indicates that malodor from swine operations is commonly present in these communities and that the odors reported by neighbors are related to objective environmental measurements.

Neighborhoods were included in the study if at least several members were interested in participating in a 2-week study that required a 3-hr training session and a twice-daily routine of reporting and measurement. Neither the neighborhoods nor participants are a representative or systematic sample of the region. We relied on local knowledge to select neighborhoods where hog odor had been reported to community organizers and where individuals might be interested in participating. However, there are > 2,000 swine CAFOs in the region, and we had no way to identify those CAFOs with higher releases of odorant chemicals. Although it is unlikely that neighborhoods with the highest exposures were included in this study, neighborhoods with no odor problems, if they exist, would not have been included either. Pollution levels and odor strength in this study may also have been affected by actions taken by operators of swine CAFOs near the study sites; participants in several neighborhoods reported cessation or relocation of hog waste sprayers, as well as reduced odor, during their period of study participation.

Other analyses indicated that the completeness and consistency of data in this study were high (Schinasi 2007). Participants reported twice-daily odor ratings in 94% of 2,949 total journal entries and at least one such rating on 97% of 1,495 study days. On the 1,456 study days with at least one twice-daily odor rating, the mean and median percentages of hours of the day for which hourly odor ratings were provided were 96% and 100%, respectively. On 95% of study days, participants reported information on whether hog odor had altered their daily activities.

We evaluated the hypothetical possibility that, due to their access to the H_2S monitor, odor ratings of 12 study participants who were asked to check for malfunctions with the environmental monitoring equipment could have been influenced by the value on the display screen; in this case the relationship between H_2S and odor might be overestimated. We refit the random-intercept, random-slope model for H_2S and odor excluding these 12 participants; the β coefficient and its SE rounded to the same values reported in Table 6.

Although the structured reporting of odor by neighbors of swine CAFOs is a strength of our study, the frequency, duration, and intensity of reported hog odor episodes must be interpreted in the context of participants' daily activity patterns. Participants reported being indoors at home 30.0%, outdoors at home 17.1%, away from home 25.5%, and sleeping 27.4% of hours in the study. The large proportion of time spent indoors and away from home limits information on outdoor odor episodes. The duration of outdoor odor episodes is also truncated by going indoors or away from home to avoid odor; this may contribute to the shorter duration of reported outdoor hourly odor episodes (62.1% lasted 1 hr) compared with indoor hourly odor (46.6% lasted 1 hr).

With the exception of PM_{10} in higher wind conditions, temperature, PM_{10}, and semivolatile PM_{10} were correlated with hog odor ratings only if the within-person, between-person, and between-neighborhood structure of the data was ignored. This might reflect the lack of seasonal variation of these variables within neighborhoods sampled for only about 2 weeks, which is a limitation of the study design. H_2S, in contrast, was strongly related to odor in mixed models. Unlike the weather variables, H_2S levels varied markedly within neighborhoods. In a recent chamber experiment, naïve volunteers exposed to swine CAFO air with a 24 ppb concentration of H_2S reported an average odor of 5.29 on a 0–8 scale (Schiffman et al. 2005). The predicted odor at 24 ppb in the present study, based on the linear regression function from Table 4 [odor = 1.25 + 0.17 × H_2S (ppb)] produces a similar value of 5.33.

In theory, a stronger relationship between odor ratings and the concentration of odorant compounds should have been observed among people with a better sense of smell. We considered butanol detection threshold as a modifier of the H_2S effect because, unlike PM_{10}, it was strongly associated with odor even without taking into account the modifying effect of wind speed. The observation that this association was restricted to people with detection thresholds < 320 ppm suggests that this simple threshold test distinguishes a subgroup of participants (87.8%) who are more responsive to H_2S.

The microbalance produced many negative values for semivolatile PM_{10}, indicating large measurement error relative to the semivolatile

Table 6. Mixed-model coefficients for environmental predictors of odor.

	Effect	SE	*t*-Value
Wind speed[a,b]			
Low	0.18	0.07	2.62
Medium (intercept)	1.23	0.20	6.03
High	0.38	0.13	2.91
Relative humidity ≥ 50%	0.29	0.11	2.59
H_2S (ppb)[c]	0.15	0.05	3.10
H_2S × wind speed[d]			
Low	0.28	0.11	2.49
Medium	–0.09	0.10	–0.83
High	0.77	0.44	1.75
PM_{10} (10 μg/m^3) × wind speed[e]			
Low	–0.01	0.05	–0.23
Medium	0.00	0.02	0.25
High	0.45	0.14	3.14

[a]Random-intercept, fixed-slope model. [b]Low, ≤ 0.57 mph; 0.57 < medium ≤ 6.75; high, > 6.75. [c]Random intercepts, random slopes. [d]Random intercept, random slope for H_2S, random intecept, fixed slope for wind. [e]Random intercept, fixed slope for wind and PM_{10}.

Table 7. Reports of change in activities due to odor in relation to average odor during the previous 12 hr.

12-hr average	No. of changes in activity reports	Percentage of times with change in activity	Rate ratio	Log_e odds ratio[a]	SE	*t*-Value
Odor < 1	22	1.4	1.0	Referent	—	—
1 ≤ odor < 2	23	5.1	3.6	1.32	0.38	3.46
2 ≤ odor < 3	19	7.1	5.0	1.56	0.40	3.93
3 ≤ odor < 5	30	11.0	7.7	2.12	0.39	5.46
Odor ≥ 5	24	16.2	11.3	2.78	0.43	6.39

[a]From mixed model with random intercepts and fixed slope for odor terms.

particle signal. This reduced the power of the study to detect associations between reported odor and semivolatile compounds in particle phase, including ammonia, an important odorant chemical emitted by swine CAFOs (Lim et al. 2003; Reynolds et al. 1997; Wilson and Serre 2007). We did not have the capacity to directly measure ammonia or other odorant compounds for this study.

The presence of air pollution from swine CAFOs in neighboring communities depends on wind direction and speed. We did not evaluate wind direction because there were at least several CAFOs in different directions near most neighborhoods in the study. Wind speed was related to odor and was also a modifier of relationships between air pollution levels and the strength of odors reported by neighbors. Although odor was highest at high wind speeds, mean H_2S levels were lowest at high wind speeds (0.05 ppb) compared with medium (0.09 ppb) and low (0.45 ppb) wind speeds. H_2S was strongly related to odor at low wind speeds (0.28 ± 0.11/1 ppb). Although the point estimate of the odor–H_2S relationship at high wind speeds was very large (0.77), its SE was also large (0.44), reflecting the limited range of H_2S values and smaller sample size at higher wind speeds.

In contrast, PM_{10} was related to odor in mixed models only during periods of higher wind speed. This observation is consistent with the greater capacity of stronger winds to transport PM, and provides evidence that organic dusts from swine CAFOs may be inhaled by CAFO neighbors during higher wind conditions. Although PM_{10} is associated with a variety of health outcomes, most studies have been conducted among populations where the composition of PM is largely affected by combustion by-products and urban dusts. Although PM from animal dander, dried feces, feed, pharmaceuticals, and endotoxin is known to affect occupational health of workers in swine confinement buildings (Donham 1990, 1993; Donham et al. 1995, 2000), its effect at lower levels and among nonworker populations is poorly understood.

Among the 98 participants who answered questions about residential history, 76 grew up on farms where they had experience with animal odors, and 82 had lived in their homes for > 5 years. Thus, adaptation and loss of sensitivity to malodors from swine operations could have occurred. On the other hand, the study protocol prompted participants to pay attention to swine odors, thus, physiologic adaptation or reduced attention to odor as a means of coping may have been offset by the odor-reporting protocol. In considering the effects of odor, it is important to note that adaptation occurs most readily when there is little variation in the concentration of odorant chemicals, whereas swine odors are transient. Like other environmental agents that act as stressors, unpredictable acute odor episodes may cause more of a stress response in susceptible persons than nonepisodic stressors.

The health significance of malodorous compounds is due, in part, to diseases related to pollutants such as PM that would occur even among persons with no sense of smell. However, malodor also should be considered in the context of scientific interest in end points that are not specific diseases. For example, biological markers of exposure to or effects of toxicants, genetic markers of susceptibility, and physiologic states associated with increased risk of disease are widely recognized as relevant to understanding and improving environmental health, even though they are not specific diseases. Similarly, environmental malodor is an important subject for inquiry, not only because it may be involved in causation of specific diseases but because of its potential to affect health, considered as not merely the absence of disease, but as a state of physical, mental, and social well-being (World Health Organization 2002). Environmental malodors may be markers of agents that can produce inflammatory, immunologic, infectious, or toxicologic responses; additionally, they may affect physical, mental, and social well-being due to their psychological and cultural meaning (Schiffman et al. 2000). Odors that are viewed as unpleasant, embarrassing, or sickening may interfere with mood, beneficial uses of property, and social activities that are central to quality of life.

We found that average odor over a 12-hr period relates strongly to changes in activities because of hog odor. Both reports of activity limitations and the three reported episodes of indoor odor that occurred during the middle of time periods of sleep suggest that odor interrupted participants' sleep in the middle of the night. Other studies have shown that the odor of feces and urine from liquid waste management systems can negatively impact neighbors' quality of life. Among a subsample of participants in the present study, odor was found to be related to levels of stress reported in daily diaries (Horton 2007). However, numerical relationships between hog odor and disrupted activity are insufficient to capture the full impacts of quality of life disruptions. Ethnographic interviews conducted with a subsample of study participants demonstrate that malodor, when present, limited many daily physical and social activities that have been shown to reduce stress and promote health (Tajik et al. 2008). Even when odor is not present, anticipation of the potential impact of irregular and unpredictable odor events may create stress and anxiety about daily routines and about social events that could cause embarrassment if odor occurs when relatives, friends, or out-of-town guests are present (Tajik et al. 2008).

Previous studies indicate that North Carolina swine CAFOs are located disproportionately in low-income communities of color (Edwards and Ladd 2000; Ladd and Edwards 2000; Wing et al. 2000). These communities may be more adversely affected by CAFOs because of their limited resources, higher disease rates, poor food supplies, poor housing, and unprotected sources of groundwater for drinking. Lower levels of formal schooling and less access to legal and political resources make it more difficult for such communities to bring about more protective environmental policies and enforcement. The present study adds to a growing body of literature suggesting that malodor from swine CAFOs, and the physical and chemical agents with which it is associated, have the potential to negatively impact public health, especially in communities that are already vulnerable (Donham et al. 2007).

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Attachment A9

Does Animal Feeding Operation Pollution Hurt Public Health? A National Longitudinal Study of Health Externalities Identified by Geographic Shifts in Livestock Production

Stacy Sneeringer

The Environmental Protection Agency is currently gathering data to regulate livestock facilities under the Clean Air Act, legislation that purports to protect public health. To set rational policy, estimates of health externalities associated with livestock farming are necessary. This study uses geographic shifts in the industry to measure the impact of pollution on infant health. The article finds that a doubling of production leads to a 7.4% increase in infant mortality. This finding is robust with respect to the inclusion of multiple fixed and time-varying controls. The mortality increases are driven by elevated levels of respiratory diseases, providing suggestive evidence of an air pollution mechanism.

Key words: externalities, livestock, pollution, public health.

Pollution and accompanying public health effects from livestock operations have been a growing concern for the last several decades. Most recently, concern has centered on air pollution arising from livestock facilities, leading the Environmental Protection Agency (EPA) to attempt to regulate them under the Clean Air Act (CAA). However, no consistent air pollution emissions data exist for these facilities that could be used to write feasible regulations. Recognizing this lack, in 2005 the EPA entered into an agreement by which operations could voluntarily monitor their own air quality in exchange for exemption from regulation during and prior to monitoring (EPA 2005a). One goal of the CAA is to protect the public health, hence regulatory measures should take into account whether livestock facilities effect health. As the EPA creates regulations, consideration of the effects of pollution externalities on health is necessary for rational policy.

The empirical strategy employed in the article exploits spatial variation in livestock operations to identify the effects of the industry on measures of infant health. This provides a method of discerning effects in the absence of pollution data. Using county-level national data spanning two decades, the article documents a positive relationship between the concentration of industrial production in livestock farming and infant mortality rates, controlling for many observed and time-constant unobserved variables that also impact infant mortality. I find that a 100,000 animal unit increase in a county corresponds to 123 more deaths of infants under one year per 100,000 births, and 100 more deaths of infants under twenty-eight days per 100,000 births.[1] A doubling of production induces a 7.4% increase in infant mortality. In practical terms, counties with increases in animal units between 1982 and 1997 experienced on average a 35% gain. This corresponds to a 2.8% increase in infant mortality in these counties, or an additional 3,500 infant deaths between 1982 and 1997. Using the EPA's

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[1] An animal unit is a method of normalizing across animal types and represents 1,000 pounds of average live weight. This metric is used by the USDA (USDA 2001) and the EPA in environmental permitting because it creates equal standards for all animals, regardless of size and manure production. One animal unit is equal to 250 layer chickens, 1.14 fattened cattle, or 2.67 breeding hogs.

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$6.2 million value of a statistical life, this amounts to $21.7 billion in externality costs associated with increases in livestock production.[2] Complementary evidence on causes of death and other health measures suggest that the causal effect is rooted in increasing levels of air-based pollution in the proximity of livestock farming operations.

In the past fifty years, technological change and growing economies of scale were the impetus for a sharp decline in the number of U.S. livestock farms. At the same time, the livestock population in inventory has remained nearly constant (see figures A1 and A2 in the appendix [Sneeringer 2008]). The consequent rise in the average size of livestock farms has led to the industry's status as one of the top polluters of rivers and streams and agricultural run-off (EPA 2000, 2005b). It has also evoked concern about the impact of livestock operations on surrounding populations. In 2003, the American Public Health Association called for a moratorium on new livestock operations until more could be understood about their effects on the environment and infant health (American Public Health Association 2003). Several states have temporarily halted construction of large livestock facilities, with some calls for a nationwide and permanent ban, but there is almost no research on public health effects to inform policy decisions.

As the primary source of variation in livestock farming, the fixed-effects estimates use differences over time within counties, controlling for characteristics of states in each time period. As noted by the EPA in the 2005 agreement, data on the pollutants most directly associated with livestock farms do not exist on a national basis, or for the time period of interest. This study therefore employs careful reduced form analysis subject to a battery of specification tests and complementary analysis. The strategy of examining changes in infant health, particularly causes of death associated with pollution, allows me to provide empirical evidence on the pollution mechanism and is therefore one of the main contributions of the research.

There are two immediate concerns of the empirical strategy. The first is that farms specifically choose to locate in areas with low and declining infant health. The literature strongly suggests that livestock farming's location shifts are due to technological advances, presence of environmental amenities such as temperature and land availability, geographic proximity to markets, and state legislation (Steelman, Page, and Burton 2004; Stirm and St. Pierre 2003) rather than public health features. The second concern is that livestock farms systematically move with another industry that affects infant health. While lower land and labor costs may be factored into location decisions and may be correlated with decreased infant health, I control for a rich variety of time-varying observables, including per-capita income, labor characteristics, medical care availability and use, climate, other industries, and housing and land-use covariates in subsequent analyses. While it is unlikely that livestock farming legislation is systematically correlated with infant health, I add state-time fixed effects to control for legislation occurring within states in each time period. If anything, community members who witness poor health outcomes when a new livestock facility moves into the area may pressure the legislature for the farm to move out of the area, hence we might see correlation between lower infant health and reduced numbers of livestock. Finally, the inclusion of fifteen variables for the presence of other industries and supplementary analyses relating these other industries to livestock farming and infant health suggests that this livestock production is not systematically correlated with another industry that affects infant health.

Research on the causes of death, effects by well-water usage, and other health outcomes suggest that increased infant mortality associated with livestock farming is primarily driven by air rather than water pollution. Examination of the causes of death provides a falsification test that the infant mortality effect is an artifact of unobserved heterogeneity. If we believed that the negative health effects correlated with increased animal production were the result of unhealthier mothers moving to the area or giving birth, we would expect a rise in mortality from all causes of death. Instead, only certain causes are affected. Respiratory and perinatal causes are associated with livestock farming, suggesting an air pollution mechanism. Moreover, greater effects of livestock production occur in areas with low well-water usage, suggesting less scope for a water pollutant mechanism. Finally, an effect on Apgar score mirrors findings in other research of an effect of air pollution.

These findings have implications for current policy discussions on the regulation of livestock farming. Past federal attempts to minimize negative public health impacts of

[2] In 2002 dollars.

livestock farming have been via Clean Water Act regulations. Recent discussions center on air quality impacts. This study helps to explore whether regulatory concerns should turn more towards air pollution. The article finds that the negative health effects of livestock farming are plausibly arising via air pollution, suggesting that more attention needs to be paid to air quality regulations of livestock farms if negative public health effects are to be avoided.

Livestock Production, Pollution, and Public Health

A confluence of factors has led to the concentration and location shifts of livestock production. Vertical integration coupled with technological advances has generally divorced meat production from heavy dependency on fixed land resources. Disconnected from crop production, livestock producers have been able to locate based more strongly on market forces, thereby moving to where profit could be maximized and costs minimized (Abdalla, Lanyon, and Hallberg 1995; Roe, Irwin, and Sharp 2002). Location decisions differ by product, but chief among the factors cited are availability and quality of water supplies, availability of land for waste management, and geographic proximity to markets and product finishers (Abdalla, Lanyon, and Hallberg 1995; Stirm and St. Pierre 2003; MacDonald et al. 1999). Environmental policies are cited as a decision factor only when compliance is difficult (Stirm and St. Pierre 2003; Sullivan, Vasavada, and Smith 2000). Factors affecting ease of compliance are type of ownership, landscape, and legislation complexity. Notably, labor costs are rarely mentioned as a reason for location choice.

A concentration in the byproducts of animal production accompanies the intensification of livestock farming. A large number of livestock concentrated in a small area leads to a vast amount of excrement in that same area. This concentration leads to concerns over the effects of animal wastes on the surrounding environment and public health.

Anxiety over pollution from livestock farms focuses on contamination of groundwater and air. The main groundwater pollutants are coliform bacteria and nitrogen as nitrate.[3] Coliform bacteria has been linked to gastroenteritis outbreaks (see, for example, Health Canada 2000). Ingestion of nitrate can lead to human health problems, particularly among pregnant women and infants. Medical events associated with nitrate include infant methemoglobinemia, problems originating in the perinatal period, and spontaneous abortion. Infant methemoglobinemia is a relatively rare but easily undiagnosed disorder related to reduced oxygen in the blood stream.[4]

Certain gases associated with livestock farming have been found to be toxic to humans, and to contribute to overall air pollution levels.[5] The main gases in question are hydrogen sulfide and ammonia, which have been linked to respiratory infections in infants and respiratory distress syndrome (Donham 2000 CDC 1986). Exposure in utero has been linked to perinatal disorders (Hannah, Hayden, and Roth 1989) and spontaneous abortion (Merchant et al. 2002).

Aside from hydrogen sulfide and ammonia, livestock farming has also been associated with air-borne particulate matter. Because ambient particulate matter is more commonly measured, there are a host of studies examining its effects on health. The epidemiologic research in relation to infant health generally examines correlations of pollutant exposure during pregnancy with birth outcome.[6] Results are mixed but compelling in implicating particulate air pollution in infant death and perinatal effects. More recent methodological innovations in this type of study use quasi-experiments to exogenously assign pollutant levels to similar groups. Using this type of identification strategy that controls for unobservable characteristics that may bias results, Chay and Greenstone (2003a and 2003b) find effects of particulate matter on neonatal infant mortality and Apgar score.[7]

[3] For research linking concentrated animal production to groundwater contamination, see Becker et al. 2003; Mugel 2002; CDC 1998; Gould 1995.

[4] See Fan and Steinberg 1996 for a review; CDC 1996; Wolfe and Patz 2002. Nitrates have also been linked to adult cases of bladder, stomach, ovarian, and liver cancers and adult central nervous system tumors (Wolfe and Patz 2002).

[5] On types and level of gases emitted by livestock facilities, see Hoff et al. 2002. These include hydrogen sulfide, ammonia, VOCs, and particulates.

[6] Glinianaia SV et al. (2004) provide an overview of these studies, which show a general relationship between particulate matter exposure and perinatal problems as well as death. In relation to studies on health outcomes other than death, pollutants are generally examined in conjunction; only studies that examine particulates in conjunction with sulfur dioxide show effects on birthweight (Bobak 2000; Maisonet et al. 2001; Ritz et al. 2000).

[7] The five-minute Apgar score is a composite of five tests given to an infant five minutes after its birth. The five tests are each scored on a scale of 0 to 2, with 2 being the best outcome. "Low birthweight" is defined as 2,499 grams or under. "Premature" refers to a birth that occurred after less than 37 weeks of gestation.

The connection between livestock production and pollution has been well documented, but the direct connection between livestock and public health is understudied. While several small-scale epidemiologic studies have examined incidence and found livestock farming to contribute the cause, larger-scale or studies with control groups have been rare. Only two studies attempt to quantify a relationship between public health outcomes and livestock farming. Thu et al. (1997) perform a case-control study examining the physical and mental health of eighteen residents living near a large-scale swine operation, and compare these to those of demographically comparable controls. The researchers find that neighbors of swine farms are significantly more likely than controls to experience toxic or inflammatory effects on their upper respiratory tracts. Wing and Wolf (2000) perform a similar study with 155 respondents; they also find elevated levels of respiratory effects, as well as gastrointestinal outcomes.

Empirical Strategy

The identification strategy employs the geographic shifting of livestock operations over the past two decades. As livestock production has become more concentrated in larger farms, production has become more concentrated in certain areas, a trend documented by several authors (for example, see Kellogg et al. 2000). Figure 1 shows the percentage of change from 1982 to 1997 in animal units by county. While most livestock farming still occurs in the Cornbelt states in the middle of the country, this map reveals the growth disparity by region.

This variation within states allows for identification of the effects of the industry while controlling for fixed unobservable characteristics of counties. This uses a least-squares regression model of the form:

$$H_{ikt} = \delta L_{ikt} + \mathbf{X}'_{ikt}\boldsymbol{\beta} + \gamma_{ik} + \gamma_t + \gamma_{kt} + e_{ikt} \quad (1)$$

The subscripts i, k, and t refer to county, state, and period, respectively. H_{ikt} refers to the health outcome variable, L_{ikt} refers to the number of animal units, and X_{ikt} is a vector of observable regressors that vary by county and period. γ_{ik} is a constant term that absorbs any unobserved characteristics of county i in state k that do not vary over time. γ_t is a constant term that captures unobserved events that affect all counties in period t. γ_{kt} is a dummy variable that absorbs effects occurring to all counties in state k in time period t.

The focus on reproductive and infant mortality provides a better consideration of public health effects than adult outcomes, which may reflect exposure that has occurred earlier in life or in prior areas of residence. Infants have had no opportunity to be exposed to livestock in previous time periods, and low migration rates for pregnant women and infants suggest that exposure occurs in the same area as the documented outcome.

Measuring the effect of livestock production on infant health would ideally account for the proximity of each individual mother and infant to the production facility. The data used here are on the level of the county, thus exact distances between pollution sources and those exposed is unavailable. In subsequent analyses, I attempt to control for exposure by dividing

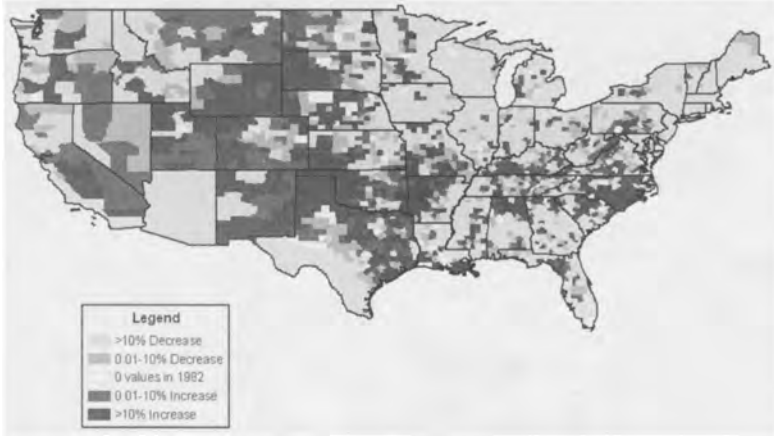

Figure 1. Percent change in animal units by county, 1982 to 1997

the sample between geographically small and large counties, and by regressing health outcomes on animal units per square mile.

The demographic covariates include mother's age, mother's age squared, race, marital status, and place of origin. Birth characteristics include sex, number of prenatal visits, whether the birth occurred in a hospital, and month of birth. County-level controls include per capita income, farm employment, physicians and hospital beds per capita, education levels, and percent Hispanic. The fixed effects account for unchanging characteristics such as predisposition to illness and preferences for health care, including attitudes toward prenatal care.

In the event that livestock operation movement is systematically correlated with another industry's movement, and this other industry affects health, then the coefficient on livestock may be capturing the effects of the other industry. While the literature makes no suggestion of a link between livestock farming and any other specific industry, I control for the number of establishments in fifteen other industries. Results of regressing livestock on these fifteen other industries show no significant negative (positive) correlations with industries that also have significant negative (positive) correlations with infant mortality (see table A2.1 in the appendix [Sneeringer 2008]).

Densely populated places may have more human waste per square mile of land and more air pollution. Hence, a population density variable is included in the analysis. Housing characteristics may play a role in pollutant exposure, so I also include well water and septic tank usage variables. A final characteristic of the built environment potentially correlated with livestock farming and infant health is land cost. The average number of building permits in a time period proxies for this factor.

A number of geologic and weather variables may affect the concentrations of pollution from animal feedlots in the ambient groundwater and air, as well as be correlated with health. I control for precipitation and temperature, and the percentage of counties in cultivated cropland, forest, rural transportation land, and other rural land.

The only federal regulation of livestock farms in effect between 1980 and 1999 was the Clean Water Act (CWA). The CWA requires facilities above a certain size that confine livestock to obtain National Pollution Discharge Elimination System permits. In order to obtain a permit, the facility must fulfill certain engineering requirements concerning the structure of the lagoon, and must observe certain limits on application of manure. I include a variable in the analysis for whether or not a county has permits.

Data

Information on livestock numbers[8] comes from a proprietary data set created by Robert Kellogg at the National Resource Conservation Service. Dr. Kellogg created this data set using the 1982, 1987, 1992, and 1997 Censuses of Agriculture. Public-use data by county from the Census of Agriculture is censored when it is possible to discern specific farms within county. As livestock operations have become increasingly concentrated, the observations by county have been increasingly censored. The data from Dr. Kellogg does not suffer from this impediment. Type of animal is not available in this restricted-use data.

Restricted-use birth and death records from the National Center for Health Statistics (NCHS) provide the health and mortality data for 1980 to 1999 (U.S. Department of Health and Human Services, 1980–1999). The NCHS's unrestricted public-use natality and mortality files after 1988 are censored when the birth or death occurred in a county with less than 100,000 inhabitants. This amounts to a serious problem, as approximately a quarter of all births are censored in each year. Because I expect the largest effects from livestock farming will occur in rural and therefore less populated counties, censoring such counties would yield a biased picture. Hence, I use restricted-use data in which all counties are enumerated. I use these data to construct a number of outcome measures including infant mortality at different ages, cause of death, and birth outcomes such as gestation and Apgar score.

For the many time- and county-varying controls, I garner data from a variety of sources, including the National Resource Conservation Service, U.S. Census, Bureau (U.S. Department of Commerce, 2001), Bureau of Economic Activity, National Climactic Data Center, and the EPA's online Envirofacts Data Warehouse. The appendix (Sneeringer 2008) provides more detail and describes data sources.

All observations are on a county basis. To create consistent units of time across the data

[8] "Livestock" in this study include beef, dairy, swine, and poultry. Sheep, goats, horses, and other animal types are excluded.

sets and to prevent sample size reduction (particularly for the mortality rates by cause of death), I group the data into the following four five-year time periods, each with a Census of Agriculture at its middle: 1980–84, 1985–89, 1990–94, and 1995–99. The appendix (Sneeringer 2008) shows results with other time periods as the unit of analysis (table A2.2).

Although the combined data are rich and cover a large number of factors that might be correlated with livestock and affect infant health, there are potentially important factors that I am unable to measure. While the natality records provide rich information on mothers and medical use, certain variables are still unavailable for births in all time periods. If a certain group of people with high levels of infant mortality is employed in livestock farming, and if I do not have information on the characteristic leading to poor health, then I run the risk of attributing the outcome to livestock farming instead of the characteristic. In subsequent analyses below, I also examine this hypothesis using causes of death as a falsification test.

One possibly omitted variable is access to abortion. Suppose that people in areas with more livestock have less access to abortion services and that fetuses that are aborted are higher risk. Women in high-livestock areas may be more likely to carry high-risk fetuses to full gestation, and the relationship between livestock farming and infant mortality may be due to higher-risk babies not being aborted. In a county fixed effects model, this requires that abortion services systematically change with the presence of livestock. Plausibly, decreased livestock farming and increased abortion services may be correlated with urbanization. However, the population density variable should capture this effect, and exclusion of the most densely populated counties from analysis does not change the results. Finally, the supposition that aborted fetuses would be more likely to die if born may not be tenable.

Table 1 provides descriptive statistics for a subset of the included variables by period.[9] The table shows that approximately 97% of all births occurring in the contiguous United States are covered by the sample.[10] The average number of animal units per county remains fairly constant over the four periods, reflecting Appendix figure A2. The next set of rows in table 1 provides means of a selected set of demographic variables.[11]

Results

Table 2 shows fixed-effect regression results on the infant mortality rate (IMR) or the neonatal IMR.[12] In order to account for possible serial correlation, disturbance terms are clustered on the level of the county. Models 1 through 3 show results with different confounders in order to examine robustness. Only controlling for fixed effects, an increase in 100,000 animal units in a county corresponds to an increase in the IMR of 1.64 and an increase in the neonatal IMR of 1.24.[13] With the addition of the time-varying covariates and state-time effects, these correlations drop to 1.12 and 1.03, respectively. The addition of the fifteen industry controls changes the coefficients only slightly, leaving a sizable and statistically significant correlation between livestock and infant death.[14] About 80% of the deaths associated with livestock farming occur within the first twenty-eight days of life. With all controls, a 100,000 animal unit increase corresponds to 123 more infant deaths per 100,000 births.[15]

A log-log regression (Appendix table A2.3) estimates the adjusted elasticity between infant mortality and provides a robustness check with respect to functional form. In Model III,

[9] The total number of counties or combined counties for which I have livestock data in each period is 2,424. The reduction to around 2,300 county units is caused mostly by no births occurring in a county unit, or in a few situations a lack of data on covariates.

[10] Those that are not covered by the sample occur almost entirely in the "consolidated cities" of Virginia, for which I do not have livestock data.

[11] A possible source of selection bias in my sample arises because I use a population of live births, and do not include the results of changes in fetal death. If presence of livestock causes damage to a fetus before birth, then this may increase the likelihood of miscarriage or stillbirth. If these fetal deaths would have been more likely to die once born, then excluding fetal deaths from the sample means that estimates of infant mortality associated with livestock are understated.

[12] The IMR is defined as the number of deaths under one year of age per 1,000 births. The neonatal mortality rate is the number of deaths under twenty-eight days per 1,000 births. These are both period (rather than cohort) measures.

[13] This corresponds to 164 additional deaths under one year of age per 100,000 births, and 124 additional deaths under twenty-eight days per 100,000 births.

[14] The cross-sectional estimates range from 0.024 to 0.497 as an effect of animal units on infant mortality. In all periods except the third the effects are highly significant. This underestimation in the cross sections is to be expected if there are fixed characteristics of an area that are negatively correlated with livestock and infant health.

[15] Cigarette smoking and alcohol use during pregnancy are only systematically recorded on the natality records beginning in 1989. Hence, I cannot use these two covariates in regressions using the full 1980–99 sample. Regressions of animal units on cigarette smoking and alcohol use during Period 3 and 4 show no significant correlation.

Table 1. Sample Statistics, by Period

	All Periods	Period 1 1980–84	Period 2 1985–89	Period 3 1990–94	Period 4 1995–99
Number of counties or combined counties	9,223	2,300	2,303	2,303	2,317
Total births in sample	74,422,510	17,480,722	18,545,151	19,525,687	18,870,950
Total births in U.S.[a]	76,470,652	17,930,501	19,071,312	20,075,545	19,393,294
Percentage of births covered by data	97	97	97	97	97
Fatalities per 1,000 live births					
At 1 month	6.0	7.6	6.4	5.4	4.8
	(3.3)	(3.9)	(6.4)	(1.7)	(1.5)
At 1 year	9.3	11.5	10.0	8.5	7.2
	(4.4)	(5.1)	(5.8)	(2.3)	(2.1)
Mean county-level variables					
Number of animal units	39,674	40,522	38,690	39,046	40,432
	(48,835)	(45,659)	(46,516)	(48,422)	(54,250)
Percentage of houses with well-water	0.308	0.319	0.311	0.304	0.297
	(0.196)	(0.181)	(0.187)	(0.199)	(.214)
Percentage of houses with septic tank	0.063	0.168	0.040	0.025	0.018
	(0.080)	(0.086)	(0.041)	(0.029)	(0.027)
Mean demographic and socioeconomic characteristics					
Percentage of mothers married	0.734	0.805	0.758	0.701	0.678
	(0.102)	(0.083)	(0.088)	(0.096)	(0.089)
Percentage white	0.804	0.817	0.806	0.796	0.800
	(0.150)	(0.149)	(0.149)	(0.152)	(0.148)
Age of mother	26.3	25.3	26.1	26.6	27.0
	(1.3)	(0.91)	(1.07)	(1.25)	(1.44)
Number of prenatal visits	10.5	9.4	9.8	11.2	11.6
	(2.4)	(3.2)	(2.9)	(2.3)	(2.1)
Percentage of Hispanic in county	0.033	0.027	0.029	0.034	0.042
	(0.081)	(0.075)	(0.079)	(0.083)	(0.088)
Percentage of county with less than high school education	0.308	0.364	0.321	0.285	0.262
	(0.102)	(0.106)	(0.097)	(0.090)	(0.083)
Percentage of county with just high school education	0.357	0.372	0.359	0.350	0.348
	(0.059)	(0.062)	(0.057)	(0.057)	(0.056)
Percentage of county with some college	0.216	0.160	0.206	0.240	0.257
	(0.068)	(0.052)	(0.058)	(0.061)	(0.059)
Percentage of county with college degree or above	0.119	0 .104	0.115	0.125	0.134
	(0.052)	(0.043)	(0.049)	(0.053)	(.056)
Mean infant health variables					
Birthweight (grams)	3,339	3,346	3,348	3,338	3,323
	(63)	(63)	(63)	(63)	(61)
Percentage of low birthweight (<2500 grams)	0.070	0.067	0.068	0.071	0.074
	(0.015)	(0.014)	(0.014)	(0.015)	(0.014)
Mean Apgar score	8.9	9.0	8.9	8.9	8.9
	(0.21)	(0.28)	(0.22)	(0.17)	(0.15)
Percentage of premature (<37 weeks of gestation)	0.101	0.086	0.097	0.107	0.112
	(0.022)	(0.019)	(0.020)	(0.020)	(0.018)

[a] Excluding Hawaii, Alaska, and D.C.

a 100% increase in the number of animal units corresponds to a 7.4% increase in the infant mortality rate.[16]

[16] Results for different time period ranges (in the appendix) show even stronger results.

How Does Proximity Affect Outcomes?

Two analyses help elucidate the effects of proximity of the mother or infant to the livestock operation. Controlling for distance between the polluting facility and the infant or mother is impossible with the data I have. However,

Table 2. Fixed Effect Regressions of Infant Mortality Rates on Number of Animal Units

	Dependent Variable					
	Model I IMR	Model III IMR	Model III IMR	Model I Neonatal IMR	Model III Neonatal IMR	Model III Neonatal IMR
Number of animal units (100,000)	1.641 (0.382)*	1.116 (0.497)*	1.232 (0.485)*	1.242 (0.307)*	1.034 (0.400)*	1.005 (0.387)*
Average prenatal visits		0.157 (0.099)	0.088 (0.106)		0.079 (0.077)	0.045 (0.084)
Log of per capita income		−6.402 (2.741)*	−6.096 (2.517)*		−4.461 (2.060)*	−4.359 (1.927)*
Physicians per capita (1,000)		−3.877 (1.124)*	−3.747 (1.187)*		−3.003 (0.823)*	−2.926 (0.869)*
Farm employment (/1,000)		0.630 (0.241)*	0.465 (0.229)*		0.474 (0.212)*	0.347 (0.213)
Population density (/1,000)		−1.610 (1.003)	−3.099 (1.141)*		−1.060 (0.729)	−2.108 (0.869)*
Percentage of homes with septic tank		−1.659 (1.920)	−1.100 (1.902)		−1.849 (1.679)	−1.624 (1.683)
Percentage of homes with well water		−7.354 (2.196)*	−7.509 (2.148)*		−5.734 (1.721)*	−5.820 (1.701)*
Demographic controls?[a]	N	Y	Y	N	Y	Y
Controls for month of birth?	N	Y	Y	N	Y	Y
Other medical use controls?[b]	N	Y	Y	N	Y	Y
Land use covariates?[c]	N	Y	Y	N	Y	Y
Precipitation and temperature covariates?	N	Y	Y	N	Y	Y
CWA permits and building permits covariates?	N	Y	Y	N	Y	Y
Other industry levels?[d]	N	N	Y	N	N	Y
Period fixed effects?	Y	Y	Y	Y	Y	Y
County fixed effects?	Y	Y	Y	Y	Y	Y
State × Time controls?	N	Y	Y	N	Y	Y
R^2	0.675	0.751	0.759	0.659	0.738	0.746
N	9,208	9,134	9,012	9,223	9,149	9,027

Notes: Observations weighted by number of births. Robust standard errors shown in parentheses. Standard errors clustered by county. Asterisk (*) refers to significance at the 5% level.

[a]Refers to controls for county education levels, percentage of county that is Hispanic, percentage of mothers that are foreign-born, mother's race, mother's marital status, mother's age, and mother's age squared.

[b]Refers to hospital beds per capita and percentage of births occurring in hospitals.

[c]Refers to percentages of county in cultivated cropland, forest, rural transportation land, and other rural land.

[d]Refers to the number of establishments for fifteen different industries; for listing see appendix.

the size of a county should provide an indication of proximity, as infants subject to health consequences of livestock are more likely to live closer to the animal feeding operations in geographically smaller counties than in larger ones. Results of dividing the sample by geographic size (Appendix table A2.4) are not precisely estimated but suggest that small counties have an effect of animal units that is nearly four times the size of the effect in the overall sample. Large counties have a smaller effect. This suggests that the closer a person is to a livestock operation, the more probable the negative health effect.

A second method of controlling for proximity is regression of health outcomes on animal units per square mile. Results (Appendix table A2.5) show that in the most restricted regression, a 100-animal unit increase in animal units per square mile significantly correlates with 2.0 more infant deaths per 1,000 births. Thus regardless of performing the analyses with number of animal units or the number of animal units per square mile, the result in terms of number of excess deaths is the same.

Does Water or Air Pollution Provide the Mechanism?

Given a lack of pollution data that would correspond in scale and time period to the results presented above, I examine the relationship between pollution and health outcomes using

Table 3. Fixed Effect Regressions of Infant Mortality Rate on Number of Animal Units, by Level of Well Water Usage of County

Dependent Variable: Infant Mortality Rate			
	Model		
	(I)	(II)	(III)
Number of animal units*Indicator for low well usage	1.994 (0.502)*	1.311 (0.655)*	1.572 (0.632)*
Number of animal units*Indicator for medium well usage	1.045 (0.371)*	0.896 (0.434)*	0.984 (0.450)*
Number of animal units*Indicator for high well usage	0.749 (0.463)	0.903 (0.495)*	0.857 (0.501)*
Demographic and socioeconomic controls?[a]	N	Y	Y
Housing controls?[b]	N	Y	Y
Land use, precipitation, temperature, and permit covariates?[c]	N	Y	Y
Other industry levels?[d]	N	N	Y
County fixed effects?	Y	Y	Y
Period fixed effects?	Y	Y	Y
State × Period controls?	N	Y	Y
R^2	0.675	0.751	0.759
Observations	9,208	9,134	9,012

Notes: Observations weighted by number of births. Robust standard errors shown in parentheses. Standard errors clustered by county. Asterisk (*) refers to significance at the 5% level.

[a] Refers to average number of prenatal visits, physicians per capita, hospital beds per capita, percentage of births occurring in hospitals, log of per capita income, farm employment, population density, controls for county education levels, percentage of county that is Hispanic, percentage of mothers that are foreign-born, mother's race, mother's marital status, mother's age, mother's age squared, and month of birth.

[b] Refers to percentage of county with well water and percentage with septic tank.

[c] Refers to mean county temperature, mean county precipitation, existence of Clean Water Act permit in county, number of average annual building permits, cultivated cropland, forest, rural transportation land, and other rural land.

[d] Refers to the number of establishments for fifteen different industries; for listing see appendix.

the data on livestock operations and health. First, I examine whether health effects vary by well-water usage. Second, I examine livestock's effect on causes of death associated with pollution. Third, I examine other health outcomes that have been related to pollution.

Water from individual wells is more likely to be contaminated by livestock farming byproducts than is water in a "city" system because individual wells are generally not subject to regulations, unlike city-wide systems. If livestock production was associated with water pollution, and this was the source of increased infant mortality, then counties with higher well usage would see greater negative effects from livestock. To test this premise, I interact animal units with low, medium, and high well usage.[17] Table 3 shows that high well-water usage counties are less likely to have an effect from livestock on infant mortality than low well-water usage counties, suggesting that water pollution does not provide the mechanism.

A second method of investigating whether the increased infant deaths associated with livestock farming could be related to pollution is to examine the causes of death associated with livestock farming. If causes of death that have been shown to be related to pollution are also related to livestock farming, this provides evidence for a link between livestock farming and worse public health outcomes. Table 4 provides mean infant mortality rates by cause of death and a summary of results of regressing the infant mortality rate from selected causes of death[18] on the number of animal units. These regressions follow the same specification as those described in Models I through III above.

Certain causes of death are extremely unlikely (as evidenced by the means in the first column of table 4), and thus it is difficult to discern statistical significance of livestock's effects on these. We would expect the more

[17] These levels are set according to the 33rd and 67th percentiles of the well usage distribution. One-third of counties have 18% or fewer houses with well water, another third has between 18% and 37% with well water, and the final third of all counties has more than 37% of homes with well water.

[18] The IMR from a specific cause of death is defined as the number of deaths attributed to the cause per 1,000 births.

Table 4. Causes of Infant Death Associated With Livestock

IMR from Cause of Death (Dependent Variable)	Mean Number of Deaths per 1,000 Births by County	Coefficient on Animal Units (100,000)		
		Model I	Model II	Model III
Chromosomal anomalies	0.226	0.013	−0.006	0.011
	(0.181)	(0.014)	(0.020)	(0.021)
Accidents and homicides	0.305	0.030	0.012	0.024
	(0.249)	(0.028)	(0.024)	(0.030)
Gastritis, duodenitis, and noninfective enteritis and colitis	0.028	0.009	0.005	0.003
	(0.061)	(0.006)	(0.006)	(0.007)
Certain intestinal infections	0.037	0.008	0.006	−0.001
	(0.064)	(0.009)	(0.008)	(0.008)
Diseases of blood and blood-forming organs	0.022	0.007	0.002	0.004
	(0.047)	(0.006)	(0.007)	(0.008)
Acute upper respiratory infections	0.010	0.005	0.019	0.029
	(0.057)	(0.012)	(0.014)	(0.018)
Bronchitis and bronchiolitis	0.027	−0.005	−0.012	−0.004
	(0.064)	(0.009)	(0.010)	(0.010)
Respiratory distress syndrome	0.702	0.290	0.222	0.150
	(0.716)	(0.080)*	(0.104)*	(0.098)
Infections specific to the perinatal period	0.220	0.051	0.060	0.052
	(0.213)	(0.021)*	(0.023)*	(0.024)*
Certain conditions originating in the perinatal period	3.367	0.529	0.488	0.514
	(1.930)	(0.143)*	(0.177)*	(0.195)*
Symptoms, signs, and ill-defined conditions	1.408	0.179	−0.009	0.001
	(0.725)	(0.080)*	(0.080)	(0.082)
Congenital anomalies (excluding chromosomal anomalies)	1.756	0.411	0.288	0.323
	(1.093)	(0.122)*	(0.175)*	(0.159)*
Demographic and socioeconomic controls?[a]		N	Y	Y
Housing controls?[b]		N	Y	Y
Land use, precipitation, temperature, and permit covariates?[c]		N	Y	Y
Other industry levels?[d]		N	N	Y
County fixed effects?		Y	Y	Y
Period fixed effects?		Y	Y	Y
State × Period controls?		N	Y	Y

Notes: For means, standard deviations shown in parentheses. Results of 39 regressions shown. An example of interpretation is in Model I, a 100,000 animal unit increase in a county corresponds to 0.013 more infant deaths under one year per 1,000 births from chromosomal anomalies. For regression results, robust standard errors clustered by county are shown in parentheses.

[a]Refers to average number of prenatal visits, physicians per capita, hospital beds per capita, percentage of births occurring in hospitals, log of per capita income, farm employment, population density, controls for county education levels, percentage of county that is Hispanic, percentage of mothers that are foreign-born, mother's race, mother's marital status, mother's age, mother's age squared, and month of birth.

[b]Refers to percentage of county with well water and percentage with septic tank.

[c]Refers to mean county temperature, mean county precipitation, existence of Clean Water Act permit in county, number of average annual building permits, cultivated cropland, forest, rural transportation land, and other rural land.

[d]Refers to the number of establishments for fifteen different industries; for listing see appendix.

common causes of death to have more variance across counties, allowing us greater possibility for identification of effects. The first two rows of this table provide the results for two relatively common causes of death that we would not expect to be correlated with livestock farming. These include chromosomal anomalies and accidents and homicides.[19] While the mean mortality rates from causes of death are relatively high, we see that livestock are not significantly correlated with either of these. The third through fifth rows examine possible causes of death associated with water pollution. Consumption of fecal coliform, linked to livestock farms, gives rise to bacterial and intestinal infections like gastroenteritis. I find no significant relationship between gastritis or intestinal infections and number of animal units, although the rates of these causes of death are low enough to inhibit identification of effects.

[19] While children above the age of one who live on farms are also twice as likely as their urban counterparts to die from accidents (Webster and Mariger 1999), this difference does not apply to infants.

The fifth row shows the relationship between diseases of the blood and blood forming organs and livestock, which is the cause of death associated with methemoglobinemia (associated with nitrate-contaminated water). While positive, the relationship is not significant, although again the mean rate is extremely low. The next three rows show causes of death that we might believe are associated with air pollution arising from livestock farming. The only one of the three that has a relatively higher mean rate is also strongly and positively correlated with livestock. The final four rows of the table show causes of death that could be related to air or groundwater pollution, or some other element for which I have not accounted. These are also much more common causes of infant mortality. These rows show that livestock are positively and significantly correlated with infections and conditions arising in the perinatal period. This table suggests that the causes of death most associated with livestock farming are predominantly respiratory distress syndrome or conditions arising in the perinatal period. This suggests that livestock farming may also be associated with prebirth damage to the fetus. The relatively weak correlation with congenital anomalies supports the conclusion of fetal interference.

Examination of different causes of death also provides a falsification test as to whether livestock production is the cause of elevated infant mortality. While I control for a variety of demographic, socioeconomic, and fixed characteristics in prior analyses, some omitted variable correlated with livestock production may be driving the positive correlation, rather than animal operations themselves. If this were the case, then we might expect to see a rise in mortality from all causes of death as livestock operations grow, particularly the more common causes of death. Instead, only certain causes are affected, providing evidence that the effects are the result of livestock production, not a changing health distribution due to underlying population composition changes.

As a final examination of the pollution-livestock-health link, I investigate other measures of reproductive health, including birthweight, prematurity, and five-minute Apgar score, to elucidate whether negative effects of feedlots are occurring before or after birth. Table 5 shows that the only statistically significant coefficient is on Apgar score; a

Table 5. Fixed Effect Regressions of Other Infant Health Measures on Number of Animal Units

	Dependent Variable			
	Birthweight (grams)	% Births That Were Low Birthweight (*100)	Five-Minute Apgar Score	% Births That Were Premature (*100)
Number of animal units (100,000)	1.628	−0.023	−0.060	0.016
	(2.739)	(0.067)	(0.031)*	(0.106)
Demographic and socioeconomic controls?[a]	Y	Y	Y	Y
Housing controls?[b]	Y	Y	Y	Y
Land use, precipitation, temperature, and permit covariates?[c]	Y	Y	Y	Y
Other industry levels?[d]				
County fixed effects?	Y	Y	Y	Y
Period fixed effects?	Y	Y	Y	Y
State × Period controls?	Y	Y	Y	Y
R^2	0.970	0.955	0.766	0.950
Observations	9,027	9,027	8,969	9,027

Notes: Model III in previous analyses. Observations weighted by number of births in the county. Robust standard errors shown in parentheses. Standard errors clustered by county. Asterisk (*) refers to significance at the 10% level.

[a] Refers to average number of prenatal visits, physicians per capita, hospital beds per capita, percentage of births occurring in hospitals, log of per capita income, farm employment, population density, controls for county education levels, percentage of county that is Hispanic, percentage of mothers that are foreign-born, mother's race, mother's marital status, mother's age, mother's age squared, and month of birth.

[b] Refers to percentage of county with well water and percentage with septic tank.

[c] Refers to mean county temperature, mean county precipitation, existence of Clean Water Act permit in county, number of average annual building permits, cultivated cropland, forest, rural transportation land, and other rural land.

[d] Refers to the number of establishments for fifteen different industries; for listing see Sneeringer 2008.

100,000 animal unit increase correlates to a 0.06 decrease in the average Apgar score at a 10% level of significance. These results are robust with respect to model specification.[20]

In summary, livestock farming is strongly correlated with infant mortality. This finding is robust with respect to inclusion of covariates and specification changes. Most of this effect occurs within the first twenty-eight days of life, with causes of death relating to respiratory distress syndrome and perinatal conditions. The other significant effect on other markers of infant health is on Apgar score. This correlation with infant mortality and Apgar score but not birthweight is not surprising given evidence from Almond, Chay, and Lee (2002) showing Apgar score to be a better predictor of infant death than birthweight. These findings suggest systematic negative public health effects associated with livestock farming.

The effects relating to neonatal mortality, perinatal causes of death, and Apgar score suggest that the animal units are also associated with prebirth damage to the fetus, although other outcomes associated with prebirth damage are not correlated with livestock farming.

Viewing the results together suggest a pollution mechanism arising from air rather than water. The effects on specific causes of death but other (particularly more common) ones provides evidence against a theory of worse health mothers moving in tandem with livestock and towards a pollution mechanism. The effect on Apgar score and the implicated causes of death coupled with the larger effects in areas of low well-water usage point toward air-borne pollution.

Discussion of Results and Policy Implications

This article demonstrates a strong positive relationship between livestock farming and infant mortality. Animal production appears to damage the fetus as evidenced by higher rates of neonatal infant mortality, causes of death related to problems in the perinatal period, and lowered Apgar score. A 100,000 animal unit increase corresponds to 123 more deaths per 100,000 births. The 2.3% increase in infant mortality in counties with increased livestock presence can be compared to the 8.5% decrease in infant mortality brought on by Medicaid expansions found by Currie and Gruber (1996). In terms of percentage of GDP, offsetting the 2.3% increase associated with livestock would require 0.11% of GDP.[21]

The results of this article suggest that the mechanism by which this effect operates may be increased air pollution. Careful monitoring of groundwater and air pollutants near livestock farms will be necessary to form an accurate picture of their effect on public health. Unfortunately, existing air and water quality data are poorly suited for this purpose; an important use of resources would be to improve data quality.

The results of this analysis show that the new legislative efforts to regulate large-scale livestock farms under the CAA are appropriate. While the EPA is correct in stating that the current data on air pollution from livestock operations is inadequate for generating regulations, exempting large animal feeding operations from CAA regulations may be inadvisable if the EPA wishes to uphold the CAA's aim of protecting public health.

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[20] In a study of twin births Almond et al. (2002) find that smoking during pregnancy is associated with lower Apgar score. As noted above, cigarette smoking during pregnancy is only systematically recorded on the natality records beginning in 1989. Hence, I cannot control for smoking during pregnancy in the full 1980–99 sample. To check whether this might be contaminating the correlation between animal units and Apgar score, I regress Apgar score on number of animal units using Periods 3 and 4, including percent of mothers who smoke in a county as a covariate. Inclusion of these variables does not change the estimated coefficient on number of animal units.

[21] This number is calculated in the following manner: Currie and Gruber (1996) found an 8.5% decline in infant mortality based on the Medicaid expansions between 1970 and 1990, when Medicaid spending grew from 0.3% to 0.7% of GDP (CBO 2002). Thus a 0.4% increase in GDP led to an 8.5% decline in infant mortality. To offset a 2.3% increase in infant mortality would therefore require 27% of 0.4%, or 0.11% of GDP.

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Attachment A10

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ORIGINAL ARTICLE

Hydrogen sulfide concentrations at three middle schools near industrial livestock facilities

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Safe school environments are essential for healthy development, yet some schools are near large-scale livestock facilities that emit air pollution. Hydrogen sulfide (H_2S) from decomposing manure is an indicator of livestock-related air pollution. We measured outdoor concentrations of H_2S at three public middle schools near livestock facilities in North Carolina. We used circular graphs to relate H_2S detection and wind direction to geospatial distributions of nearby livestock barns. We also used logistic and linear regression to model H_2S in relation to upwind, distance-weighted livestock barn area. Circular graphs suggested an association between upwind livestock barns and H_2S detection. The log-odds of H_2S detection per 1000 m^2 increased with upwind weighted swine barn area (School A: β-coefficient (β) = 0.43, SE = 0.06; School B: β = 0.64, SE = 0.24) and upwind weighted poultry barn area (School A: β = 0.05, SE = 0.01), with stronger associations during periods of atmospheric stability than atmospheric instability (School A stable: β = 0.69, SE = 0.11; School A unstable: β = 0.32, SE = 0.09). H_2S concentration also increased linearly with upwind swine barn area, with greater increases during stable atmospheric conditions (stable: β = 0.16 parts per billion (p.p.b.), SE = 0.01; unstable: β = 0.05 p.p.b., SE = 0.01). Off-site migration of pollutants from industrial livestock operations can decrease air quality at nearby schools.

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Keywords: air pollution; animal feeding operations; children's health; hydrogen sulfide; livestock

INTRODUCTION

A healthy school environment is important for both proper physiologic development and maximal educational performance. Children spend many hours each week at schools where pollutants can impact their health.[1,2] Schools in low-income communities of color are especially vulnerable to pollutants and there is a lack of federal policies in the United States to protect schools.[1,3] Good air quality is a key determinant of healthy school environments.

One source of air pollution in rural North Carolina (NC) is industrial livestock production facilities. Over the past several decades, the majority of livestock agriculture in NC and across the United States has shifted from small farms with relatively few livestock and complementary crop production to industrialized facilities with large barns housing thousands of animals in confinement. In NC, 99.8% of swine are produced at facilities selling at least 2000 swine annually, and 99.6% of broilers (meat chickens) are produced at facilities selling at least 30,000 broilers or more annually.[4] Such facilities are concentrated in Eastern NC, an area that includes the 10 most swine-dense counties in the United States.[5]

Although numerous studies indicate that children who grow up on farms have a lower incidence of allergy and asthma-related symptoms,[6,7] other studies have found detrimental effects from exposures related to industrial livestock production. Excess asthma-related outcomes have been associated with home exposures to swine facilities, especially among children growing up on swine farms that add antibiotics to feed for growth promotion.[8] In addition, children whose residences are frequently downwind of industrial swine facilities have increased prevalence of physician-diagnosed asthma and self-reported wheeze medication usage compared with children with lower relative swine facility exposures.[9] Several cross-sectional studies have reported excess asthma or asthma symptoms among children who attend schools near industrial swine facilities.[10,11] In NC, there are higher proportions of low-income children and children of color at schools near industrial swine facilities than at more distant schools.[12]

Livestock facilities produce a mixture of harmful pollutants, including hydrogen sulfide (H_2S), respirable particulate matter, and ammonia. H_2S is an odorous gas generated by anaerobic decomposition of manure[13,14] and can serve as a marker for this mixture. Ammonia, another odorant compound, is released from urinary urea and fecal urease enzymes in animal waste.[14] In addition, hundreds of volatile organic compounds (VOCs) contribute to characteristic malodors associated with concentrated animal feeding operations (CAFOs).[15] Particulate matter $< 10\,\mu m$ in aerodynamic diameter (PM_{10}) is generated directly from animal feed, fecal matter, dander, endotoxins, bacteria, and molds, as well as indirectly from gaseous emissions. Ammonia can react with acidic atmospheric species and form fine particulates.[13,14,16,17] The ventilation of barns, the storage of animal waste, and the spraying or spreading of waste on fields release these particles and gases into the air.

To further investigate school exposures, a team of researchers from the University of North Carolina at Chapel Hill (UNC) and

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members of the Rural Empowerment Association for Community Help (REACH) collaboratively developed the Rural Air Pollutants and Children's Health (RAPCH) study. We aimed to measure industrial livestock-related air quality at schools in rural eastern NC and potential associations with acute student health outcomes, while also providing educational benefits to students and their communities.[18] Here, we summarize the measured pollutant concentrations at school sites and quantify their relationships with the geospatial distributions of nearby industrial animal confinements and concurrent meteorological conditions.

MATERIALS AND METHODS

School Recruitment

In 2008, REACH staff contacted local school administrators and recruited four schools to participate in a pilot study to determine whether we could measure livestock-related pollutants onsite. In 2009, two schools from the pilot study plus a third school recruited by REACH were invited to participate in a longer exposure assessment study. After administrators approved participation, representatives from both REACH and UNC met with school staff to review study protocols and discuss logistics such as data collection schedules and placement of air quality monitors. The UNC institutional review board (IRB) annually reviewed and approved study activities.

Air Pollutant and Meteorological Data

We monitored H_2S and PM_{10} during three time periods in 2009: 21 February to 20 March, 28 March to 8 May, and 6 September to 25 November. Each sampling period lasted at least 4 weeks and consisted of measurements at a single school. We placed a set of active air monitors outside the school buildings at a site recommended by school staff. Instruments were stored in large cases with intake tubing for protection from tampering and weather.

We used MDA Scientific Single Point Monitors (SPMs) (Honeywell Analytics, Lincolnshire, IL, USA) to measure 15-min concentrations of H_2S in parts per billion (p.p.b.) at all three schools. The SPM had a limit of quantification (LOQ) of 1.0 p.p.b. determined via laboratory tests by UNC researchers (M Boundy, personal communication). At School A, we also deployed a more sensitive Thermo Hydrogen Sulfide—Sulfur Dioxide Analyzer (Thermo Fisher Scientific, Waltham, MA, USA) to measure H_2S alongside the SPM. The Thermo had recently become available for use and had an LOQ of 0.5 p.p.b. for 5-min concentrations, similar to the minimum reported odor threshold of 0.5 p.p.b.[19] For both H_2S monitors, we retained all detected values that were recorded, including those below the LOQ. We replaced non-detected values for the SPM with LOQ/$\sqrt{2}$.[20–22] For example, in 1 h with 15-min concentration readings of non-detect, 0.58, 1.85, and 3.52 p.p.b. from the SPM, we would have reported an hourly average H_2S concentration of $((1/\sqrt{2})+0.58+1.85+3.52)/4 = 1.66$ p.p.b. We also performed sensitivity analyses for SPM results in which all non-detected values were (1) replaced with 0, (2) replaced with LOQ/2, and (3) replaced with the LOQ.

We also used DustTrak Aerosol Monitors (TSI, Shoreview, MN, USA) to measure PM_{10} concentrations as low as 1 $\mu g/m^3$ every 5 min. Observed measurements were divided by 2.41 to account for the difference in light scatter between Arizona road dust, to which the DustTrak is calibrated, and particulates in rural eastern NC (J Tarman, unpublished results). REACH staff members checked instrument function almost daily and notified UNC staff of malfunctions for prompt resolution. Every week we downloaded data and performed quality control procedures, including using a high and low standard to verify the performance of the optical system. H_2S instruments were calibrated using varied H_2S permeation tubes with a range of concentrations (1.59, 3.28, 4.29, 6.19, 11.13, 54.2, and 75 p.p.b.) before deployment.

We obtained meteorological data for weather stations nearest to the schools from the online NC Climate Retrieval and Observations Network of the Southeast (CRONOS) database operated by the NC State Climate Office and NC State University. We used hourly data from two NC Environment and Climate Observing Network (ECONet) weather stations, assigning the closest weather station to each school (13.7–17.4 km away). We downloaded solar radiation, wind speed, and wind direction data. We used solar radiation and wind speed to generate hourly Pasquill–Gifford atmospheric stability categories ranging from A to F using the Solar radiation/delta-T (SRDT) method.[23] During the day—defined as time when solar radiation was >0 W/m^2—classification of atmospheric stability was based on solar radiation and wind speed (in m/s). During the night—defined as time when solar radiation was equal to 0 W/m^2—atmospheric stability classification was based solely on wind speed because data on vertical temperature gradient were not available. Using these measurements, we then collapsed categories to generate a binary atmospheric stability variable for every hour with categories A–C as "unstable" and categories D–F as "stable." We then created hourly records by merging pollutant concentrations, meteorological variables, and atmospheric stability class by date and hour.

Geospatial Data on Livestock Barn and Waste Lagoon Locations

There are no publicly available data with geospatial information for all livestock operations in NC. Therefore, we used high-resolution digital aerial imagery data (henceforth, orthoimagery)[24] to generate an original database containing information on the location and size of livestock barns and swine waste lagoons near the participating schools. We obtained complete orthoimagery for eastern NC through the NC OneMap Geospatial Portal,[25] a publicly available online source for geographic information systems (GIS) data. We downloaded tiled raster data (GeoTIFF format, 6-inch ground resolution) published online in December 2010 by the Geospatial and Technology Management Office in the NC Department of Crime Control and Public Safety;[26] no accurate orthoimagery for our study area was available before 2010. All data were referenced to the NC State Plane Coordinate System, NAD83, North American Datum, readjusted to the National Spatial Reference System of 2007. To integrate the NC OneMap orthoimagery with data for geocoded schools and weather stations, we created a file geodatabase in ArcGIS software, version 10.1, Service Pack 1 for Desktop.[27] We made a mosaic data set from the tiled raster data, compressed it using the LZ77 lossless method,[27] and used the closest equivalent spatial reference available in ArcGIS to overlay all data.

After establishing the geodatabase, we defined three distinct study areas by demarcating a circle with radius of 5 km around each school's geocoded central point; this 5-km radius corresponds to the documented distance H_2S can travel from large swine facilities[28] as well as the proximity of industrial livestock facilities in previous studies that documented excess asthma-related symptoms.[9,10] Within each school's surrounding 79-km^2 circle, we visually inspected orthoimagery in ArcGIS (at scale 1:15,000) to identify swine and poultry barns and swine waste lagoons.

We identified livestock barns based on their unique shapes and sizes, and by the presence of adjacent feed towers; we were careful not to include greenhouses, hoop houses, or storage sheds. We assumed any livestock barns without a nearby waste lagoon were poultry barns that typically use a dry waste system.[29] To convert the imagery to data on the size and location of each livestock barn, we manually added a polygon feature to the geodatabase (at scales ranging 1:200–1:800) over the boundary of each barn.

We identified swine waste lagoons based on their color, shape, size, and proximity to swine barns. Although farm ponds are common at swine facilities, their varied shapes and locations are easily distinguished from the standard border types for swine waste lagoons near barns. We used state permit data from NC Department of Environment and Natural Resources Division of Water Quality to ensure that we did not misclassify any poultry layer operations—which also produce liquid waste—as swine facilities. Just as we did for livestock barns, at scales ranging 1:200–1:2500, we encoded a polygon feature over the boundary of each swine waste lagoon. Using the polygons to represent barns and lagoons, we calculated the area of each barn and lagoon, as well as the location of the feature's centroid relative to its corresponding school.

Data Analysis

Lacking accurate data on the number and weight of animals at each facility during the study period, we used barn area, which typically reflect a standard density of animals, as a proxy for the intensity of pollution-generating activities. We used hourly measurements of meteorological conditions and time-fixed geospatial data on livestock barn locations to compute hourly values for inverse distance-weighted livestock barn area located upwind of each school. Our computations were based on a previously published method for generating an exposure index.[9]

Within the circle around each school defined by a 5-km radius, we measured the area of each barn, A, in m^2 and the distance, d, between the barn centroid and the school centroid in m. We computed the weighted area for each barn by multiplying its area by the inverse of distance-squared ($1/d^2$), assuming non-linear distance decay.[28] For each hour of the day, using compass directions in degrees (°) to characterize the average

wind direction and barn locations, we defined upwind barns as those having a centroid within 15° of the average wind direction. Thus, for each school we computed hourly values for the sum of distance-weighted upwind barn area, $E(h)$, separately for swine and poultry barns as follows,

$$E(h) = \sum_{i=1}^{n} \frac{A_i}{d_i^2} I[w(h) - 15 \leq c_i < w(h) + 15]$$

where n is the number of barns around the school, A is the area of barn i, and d is the distance between the centroid of barn i and the school centroid. A barn centroid, c_i, has to fall within 15° of the hourly wind direction at hour h, $w(h)$, to contribute to the sum of distance-weighted upwind barn area. Therefore, $I[w(h)\text{-}15 \leqslant c_i < w(h)+15]$ returns a value of 1 if $w(h)$ is within 15° of c_i, else 0. To conceptualize this degree-based criterion using distance, the required proximity (in km of arc length) of a barn to the hourly wind direction is given by $15/360 \times 2\pi d$; for example, a barn $d = 3.6$ km from a school must be $\leqslant 0.94$ km from the hourly wind direction to contribute a detected value to that school's $E(h)$ measure in that hour.

For the circular graphs, we plotted the locations of swine and poultry barns within 5 km of each school, as described above, using ArcGIS software.[27] For clarity of display, we clustered barns together when located within 76.2 m (250 ft) of one another and represented the summed area of clustered barns with proportionately sized symbols. We categorized hourly average wind direction into 72 overlapping 30° categories, with the center of each subsequent category increasing 5°, for example, centered on 0° and ranging 345–15°, centered on 5° and ranging 350–20°, and so on. We then generated figures showing the wind rose for each school's data collection period. These show the relative frequency of average hourly wind directions and atmospheric stability for each direction, using 12 categories for wind direction for clarity of display. We generated a third series of figures showing the proportion of hourly records with $H_2S \geqslant$ LOQ, using 72 categories for wind direction. The wind rose and circular plots were generated using R software (R Foundation for Statistical Computing, Vienna, Austria) with the following packages: plotrix[30] and openair.[31]

We used logistic and linear regression models to compare upwind weighted barn area (independent variable) with measured H_2S concentrations (dependent variable). Dependent variables in logistic regression models were detection *versus* non-detection of H_2S detection *versus* non-detection (i.e., SPM: $\geqslant$ 1.0 p.p.b. *versus* $<$ 1.0 p.p.b.; Thermo: $\geqslant$ 0.5 p.p.b. *versus* $<$ 0.5 p.p.b.) in relation to upwind weighted barn area (per 1000 m^2 increase in barn area); these models were fit separately for swine and poultry barns.

For School A, where the Thermo instrument provided more sensitive H_2S detection, we also used linear regression to model changes in H_2S concentration (p.p.b.) in relation to upwind weighted barn area (per 1000 m^2 increase in barn area). For analysis at School A, we also ran separate models under each classification of atmospheric stability (i.e., stable and unstable), fit separately for swine and poultry barns. We used SAS software Version 9.3 (SAS Institute, Cary, NC, USA) to run regression models.

Because our study involved neither random sampling nor random allocation, results may be due to the factors under investigation, unmeasured factors, or measurement error, but not chance. Therefore, we do not report *P*-values or confidence intervals.[32,33] For logistic models, we report β-coefficients and SE; 95% confidence intervals can be calculated by adding and subtracting $1.96 \times$ SE from β. We also report χ^2 test statistics with 1 degree of freedom (d.f.). Higher χ^2 values indicate greater improvement in model fit upon addition of the variable for upwind weighted barn area; in a randomized study with 1 d.f., χ^2 values >3.84 would indicate a two-tailed $P < 0.05$. For linear regression models we report β, SE, and *t*-values (i.e., β/SE) as indicators of improved model fit. With large sample sizes, as in this study using hourly measurements, *t*-values >1.96 would approximate a two-tailed $P < 0.05$ in a randomized study.

Code availability. SAS code for computing distance-weighted upwind barn area and conducting regression analyses can be obtained by contacting the corresponding author.

Table 1. School exposure characteristics and student enrollment.

Characteristic	*School A*	*School B*	*School C*
Total number within 5 km			
Swine barns	95	68	68
Swine lagoons	29	31	29
Poultry barns	70	31	43
Total area within 5 km (1000 m^2)			
Swine barns	60.6	60.3	58.6
Swine lagoons	150.4	163.1	140.8
Poultry barns	130.5	70.3	67.5
Distance from schools (km)			
Swine barns			
Minimum	0.9	1.0	2.3
25th percentile	2.3	3.0	3.3
Median	2.7	3.9	4.1
75th percentile	4.3	4.6	4.4
Maximum	5.0	5.0	4.8
Swine lagoons			
Minimum	0.9	0.9	2.2
25th percentile	2.3	2.4	3.4
Median	3.0	3.2	4.3
75th percentile	4.4	4.6	4.6
Maximum	5.0	4.9	4.9
Poultry barns			
Minimum	0.5	1.0	1.9
25th percentile	2.8	3.4	3.9
Median	3.5	4.6	4.2
75th percentile	4.2	4.7	4.4
Maximum	4.9	4.8	4.9
Days of data collection	69	35	28
Hours of data collection	1588	798	648
Student enrollment for 2009–2010	428	493	223

RESULTS

School A had both the greatest number of livestock barns nearby and the closest barns (Table 1). Despite differences in total numbers, the cumulative swine barn area was similar across schools because individual swine barns around School A were smaller than around other schools (median: School A = 636 m^2; School B = 822 m^2; School C = 774 m^2). The ratio of total swine lagoon area to barn area was consistent across schools (range: 2.4–2.7). The larger total area of the poultry barns reflects their

Table 2. Frequency of hydrogen sulfide (H_2S) detection and mean hourly concentrations (p.p.b.).

School (instrument)	*Number of hourly records (N = 3034)*	*Number (%) of hours with $H_2S \geqslant LOQ$*[a]	*Mean (SE) concentration for hours with $H_2S \geqslant LOQ$ (p.p.b.)*[b]	*Number (%) of hours with detected H_2S*	*Mean (SE) concentration for hours with $H_2S > 0$ (p.p.b.)*[b]
School A (Thermo)	1325	764 (57.7)	0.79 (0.02)	1325 (100.0)	0.53 (0.02)
School A (SPM)	1546	94 (6.1)	1.35 (0.11)	533 (34.5)	0.81 (0.02)
School B (SPM)	797	5 (0.6)	1.12 (0.28)	67 (8.4)	0.71 (0.02)
School C (SPM)	647	25 (3.9)	1.26 (0.11)	204 (31.5)	0.78 (0.02)

[a]LOQ for Thermo was 0.5 p.p.b.; LOQ for SPM was 1.0 p.p.b. [b]Mean of observed detected measurements and, for the SPM, replacement of zeros with LOQ/$\sqrt{2}$.

larger size per barn in comparison with standard swine barns (median: School A = 2032 m^2; School B = 2528 m^2; School C = 1383 m^2). The longest data collection period was at School A.

For most records, 15-min H_2S concentrations measured with the SPM were below the LOQ. Therefore, we present the number and percentage of hours with detected H_2S, followed by the mean concentrations only among these hours (Table 2). However, using the more sensitive Thermo at School A, 58% of hours had measured concentrations above the LOQ, and 100% of hours had mean concentrations >0 p.p.b. Schools' hourly mean concentrations were more similar when detected values below the LOQ were retained and non-detected values were replaced by LOQ/$\sqrt{2}$. SPM results were similar regardless of the method used to replace values below the LOQ.

Mean hourly PM_{10} concentrations ranged from 9.47 $\mu g/m^3$ (SE = 0.18) at School B to 12.74 $\mu g/m^3$ (SE = 0.24) at School C, and based on the 95th percentile the greatest hourly concentration was 29.15 $\mu g/m^3$ at School A. Hourly PM_{10} values were low, on average, and we observed little association with upwind livestock barn area in circular plots and regression models (data not shown).

Figure 1 shows data for School A regarding the geographic distribution of surrounding swine and poultry barns (Figure 1a), the wind rose demonstrating atmospheric stability measurements during data collection (Figure 1b), and the proportion of measurements with detected H_2S by direction based on the SPM (Figure 1c) and Thermo (Figure 1d) instruments. Swine and poultry barns were frequently collocated around School A, with most barns located to the southwest, south, and southeast of the school (Figure 1a). During the study period, the wind infrequently came from the directions with the greatest concentration of livestock barns (Figure 1b). Although there were relatively fewer livestock barns within 5 km northeast of the school, the wind most

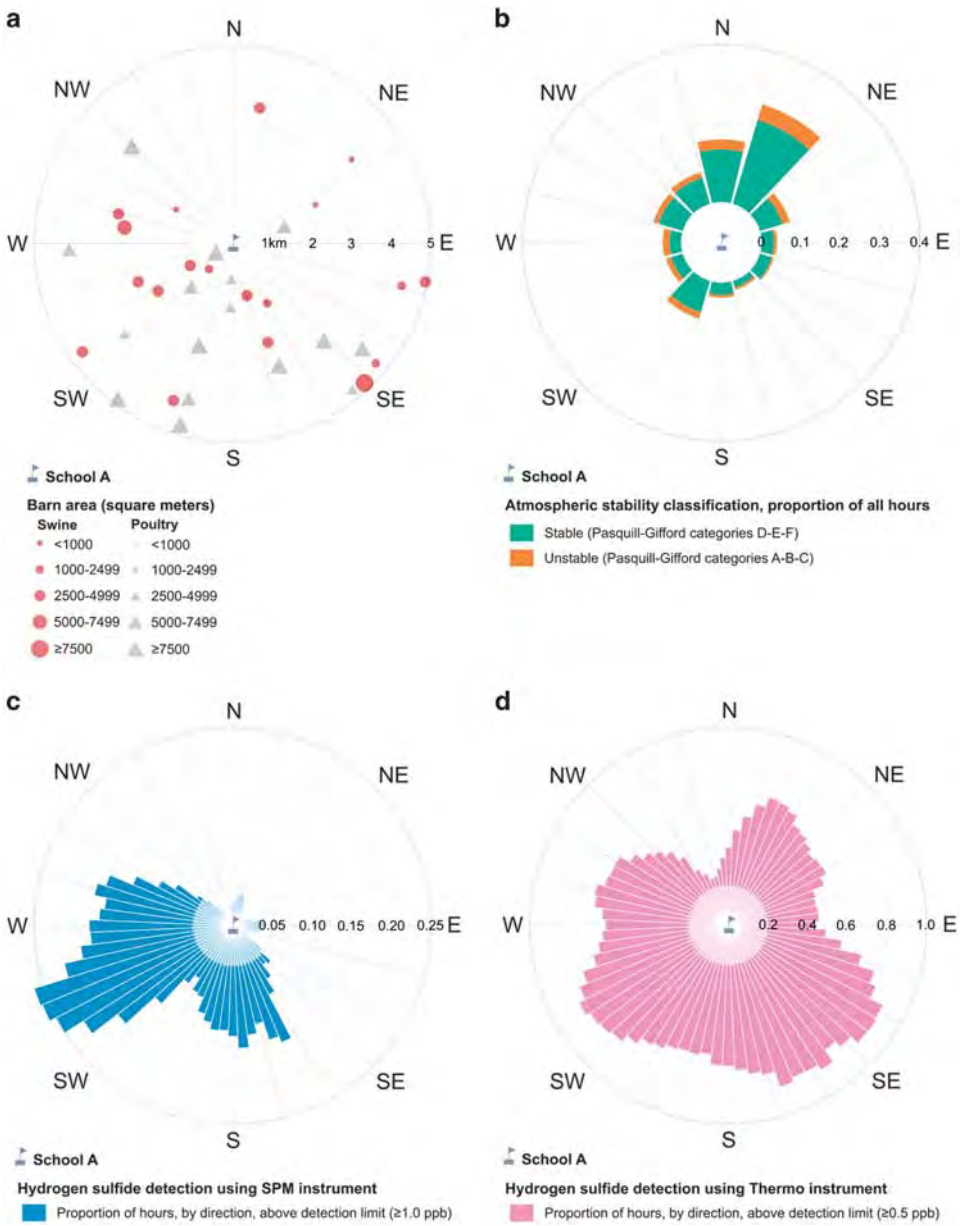

Figure 1. School A. (a) Livestock barns within 5 km, (b) wind rose and atmospheric stability by wind direction during data collection, (c) hydrogen sulfide detection ($\geqslant$1.0 p.p.b., SPM instrument) by wind direction, and (d) hydrogen sulfide detection ($\geqslant$0.5 p.p.b., Thermo instrument) by wind direction.

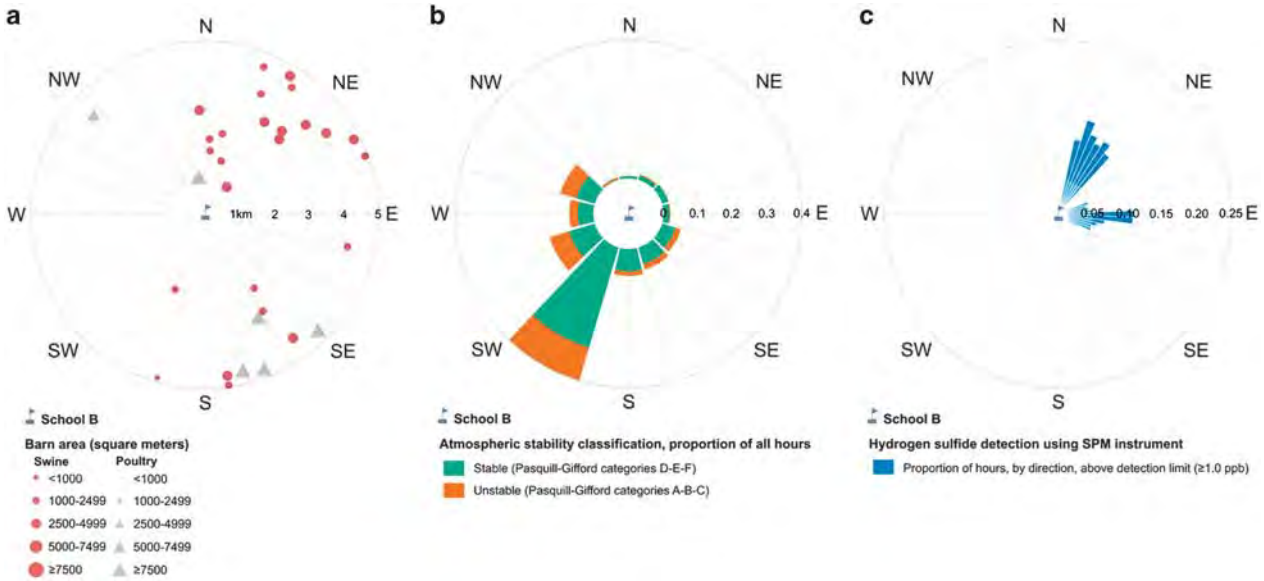

Figure 2. School B. (a) Livestock barns within 5 km, (b) wind rose and atmospheric stability by wind direction during data collection, and (c) hydrogen sulfide detection (⩾1.0 p.p.b., SPM instrument) by wind direction.

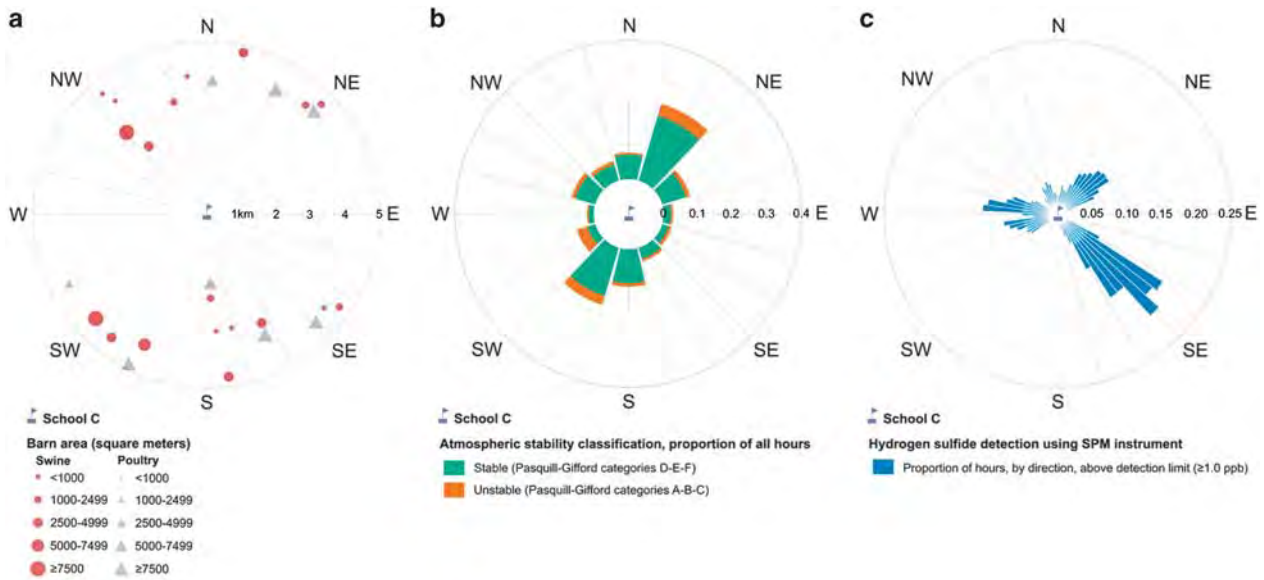

Figure 3. School C. (a) Livestock barns within 5 km, (b) wind rose and atmospheric stability by wind direction during data collection, and (c) hydrogen sulfide detection (⩾1.0 p.p.b., SPM instrument) by wind direction.

frequently came from that direction, sometimes with wind speeds >16 km/h (10 mph) (Figure 1b) that could facilitate the transport of pollutants and increase the occurrence of detected concentrations.[34] We observed a high proportion of records with detected H_2S ⩾ LOQ when the wind was from the southwest, south, and southeast (often in excess of 0.70, based on Thermo measurements). The proportion of hours with detected H_2S ⩾ LOQ was lower for the SPM instrument (Figure 1c) than the Thermo (Figure 1d). Using the Thermo instrument, we also observed a high proportion of records with detected H_2S ⩾ LOQ when the wind was coming from the northeast (Figure 1d).

For School B, most nearby swine barns were located in the northeast quadrant, several poultry barns were in the southeast, and few livestock barns were in close proximity to the school (Figure 2a). During data collection the wind mostly originated from the southwest (Figure 2b), an area with few livestock barns within 5 km (Figure 2a). Using the SPM, the proportion of measurements with detected H_2S ⩾ LOQ was very low (Figure 2c), measured during the infrequent hours when wind originated from the northeastern and eastern directions.

For School C, there were livestock barns toward both north and south, but none were less than 2 km from the school (Figure 3a). Wind direction was more evenly distributed for School C than for the other schools (Figure 3b). We observed lower proportions of SPM measurements with detected H_2S ⩾ 1.0 p.p.b. by wind direction at School C compared with SPM measurements at

Table 3. Change in log odds of hydrogen sulfide (H_2S) detection per 1000 m^2 increase in upwind weighted livestock barn area at each school.

School (instrument)	*Swine*			*Poultry*		
	β	*SE*	X^2 *(1 d.f.)*	*β*	*SE*	X^2 *(1 d.f.)*
School A (Thermo)	0.43	0.06	46.00	0.05	0.01	24.84
School A (SPM)	0.43	0.05	66.82	0.07	0.01	61.10
School B (SPM)	0.64	0.24	6.96	−1.94	3.33	0.34
School C (SPM)	−0.94	0.69	1.88	−0.27	0.36	0.57

School A, and we did not see a consistent pattern between detected H_2S and upwind barn locations (Figure 3c).

In Table 3, we present the results of separate logistic models for swine and poultry barns that quantify relationships between H_2S detection ($\geqslant$ LOQ) and upwind weighted barn area. At School A, which had the longest data collection and the most sensitive H_2S instrument, the log-odds of H_2S detection increased by 0.43 (SE = 0.06) with each additional 1000 m^2 of upwind weighted swine barn area, and 0.05 (SE = 0.01) for each additional 1000 m^2 of upwind weighted poultry barn area. The large χ^2 values (1 d.f.) for these coefficients are indicative of the substantial contribution of upwind weighted livestock barn area to prediction of H_2S detection. The log-odds for upwind weighted swine barn area increased by 0.64 (SE = 0.24) at School B; upwind weighted poultry barn area was negatively associated with H_2S detection, but this relation was imprecise. Relationships for School C were negative but imprecise for both upwind swine and poultry area. We repeated these analyses including observed concentrations below the LOQ and replacing SPM 15-min non-detects with LOQ/$\sqrt{2}$; *β*-coefficients and SEs were within 0.01.

We also assessed relationships between upwind weighted barn area and H_2S concentrations across strata of atmospheric stability at School A, the only school with sufficient data for this analysis (Table 4). For swine barns, we observed a greater increase in log-odds of H_2S detection during stable conditions ($\beta = 0.69$, SE = 0.11) compared with unstable conditions ($\beta = 0.32$, SE = 0.09). Using linear regression to model H_2S concentration at School A, we found that an increase of 1000 m^2 of upwind weighted swine barn area was associated with an additional 0.16 p.p.b. of additional H_2S (SE = 0.01) during stable conditions; during unstable conditions, we observed an attenuated ($\beta = 0.05$ p.p.b., SE = 0.01) positive association between upwind weighted swine barn area and H_2S p.p.b. We observed similar patterns with upwind poultry barns, although the *β*-coefficients for increases in log-odds of H_2S detection and H_2S concentration were considerably smaller.

Table 4. Change in log odds of hydrogen sulfide (H_2S) detection (using logistic regression) and change in H_2S concentration (using linear regression) per 1000 m^2 increase in upwind weighted livestock barn area at School A, stratified by atmospheric stability classification.

	Swine			*Poultry*		
Detection ($\geqslant$0.5 p.p.b.)	*β*	*SE*	X^2 *(1 d.f.)*	*β*	*SE*	X^2 *(1 d.f.)*
All stability categories	0.43	0.06	46.00	0.05	0.01	24.84
Stable	0.69	0.11	42.86	0.07	0.02	20.68
Unstable	0.32	0.09	12.84	0.05	0.01	11.96
Concentration (p.p.b.)	*β*	*SE*	*t-value*	*β*	*SE*	*t-value*
All stability categories	0.12	0.012	10.45	0.02	0.002	9.39
Stable	0.16	0.014	11.11	0.03	0.003	10.07
Unstable	0.05	0.011	4.28	0.01	0.002	4.30

All hydrogen sulfide data from the Thermo instrument.

DISCUSSION

Ambient air pollution at schools is a particular concern for children because of their developmental susceptibility to pollutants and high breathing rates compared with adults.[35] Furthermore, during the academic year, children spend a substantial proportion of their waking hours at school. The concentration of livestock production since the mid-1900s has brought industrial air pollution problems, formerly the province of urban areas, to rural farming communities including public schools attended by a majority of children.

H_2S is one of many chemical compounds released by industrial livestock facilities. It is also emitted by other industrial facilities including petrochemical refineries, paper mills, wastewater treatment plants, and landfills;[36] however, it is not a ubiquitous regional pollutant. There are no other point sources of hydrogen sulfide within the 5-km radii around each school; the closest potential source is a wastewater treatment plant 10.1 km from one of the study schools. Therefore, we consider detection of this compound as an indicator of the presence of livestock-related air pollution at school. Furthermore, using hourly concentrations and wind direction we demonstrated strong spatial and temporal associations between the size and distance of upwind livestock facilities and H_2S concentrations. Our analysis did not account for temporal autocorrelation between H_2S measurements. At the study school with the largest numbers of measurements and the more sensitive monitor, we found that associations between H_2S and upwind weighted barn area were stronger during stable compared with unstable atmospheric conditions. These data provide convincing evidence that upwind livestock facilities were the source of H_2S at the study schools.

H_2S is only one component of a complex mixture of gases and particles emitted by livestock facilities, including over 300 VOCs and ammonia.[13,15,16] H_2S concentrations were low outside of study schools, but indicated the presence of livestock-related plumes. H_2S concentrations have been used as a specific marker of the complex mixture of swine-related air pollution in studies of adult neighbors that have found H_2S to be related to reported hog odor,[34] mucous membrane irritation and respiratory symptoms,[37,38] stress and anxiety,[39] and increased blood pressure.[40] Because of its relatively low concentration and the co-occurrence of a large number of other pollutants, these effects on health and quality of life cannot be attributed to H_2S *per se*, but rather to the complex mixture of which H_2S is a part.

Although these studies have found harmful effects from air pollution exposures related to industrial livestock production, other studies have found livestock-related air pollution exposure to be protective, even in adults who do not live on farms.[41] One study found a lower prevalence of chronic obstructive pulmonary disease among adults living near farms, although adults already diagnosed with COPD were more likely to experience exacerbations.[42] Conflicting results may occur because of differences in livestock operation size, livestock production practices, microbial exposures, or susceptibility of nearby populations.

Although PM_{10} is another component of this mixture, we did not find it to be related to upwind weighted livestock barn area. There are several possible explanations for this finding. PM_{10} is a ubiquitous air pollutant that is not specific to livestock production. In addition to common stationary and mobile sources, PM_{10} arises from common agricultural activities including tilling, planting, fertilizing, harvesting, and burning. We did not have measures of these sources for the present study. Furthermore, gases can persist longer and travel farther than coarser particles.[13] The average hourly PM_{10} concentrations we observed (9.47–12.74 $\mu g/m^3$ across schools) are within range of expected values in this rural area. The closest US Environmental Protection Agency ambient air quality

monitor reports 22 and 28 $\mu g/m^3$ as the annual second maximum 24 h average for 2009 and 2010 respectively.[43]

In this study we lacked measurements of many key variables that contribute to H_2S concentrations at schools. These include operating schedules at individual livestock facilities (use of ventilation fans, opening side walls, flushing liquid manure, or removing dry manure), three-dimensional wind speed and direction between the livestock facilities and schools, presence of barriers (like forests), and extent of oxidation of H_2S into sulfur dioxide (SO_2) and sulfate compounds.[36] Such information would potentially improve the modeling of H_2S concentrations at schools downwind of livestock facilities. Furthermore, we were not able to monitor H_2S for extended time periods (multiple seasons at each school), and the prominent wind directions during the study did not correspond to the directions of higher livestock density. This is not a reflection of the annual wind directions in the areas; rather, it is a consequence of the time periods of monitoring.

In conclusion, we detected H_2S, a signature livestock-related air pollutant, at school sites and found it to be associated with upwind livestock barn area. In the rural study area, which lacks other point sources of H_2S, this evidence shows that air pollution from industrial livestock operations reaches nearby public schools. H_2S serves as a marker for a complex mixture of emissions from livestock operations that may be harmful to the respiratory health and educational experience of children who live in communities in eastern NC. In addition, children attending these schools are disproportionately low-income students of color, and therefore these exposures constitute an environmental injustice. Future research should explore the impacts that school exposures to livestock-related air pollutants have on health and educational outcomes.

CONFLICT OF INTEREST

SW and JJ provided a report in a civil rights complaint regarding re-permitting of industrial hog operations in NC. SW provided a declaration for plaintiffs in federal suits regarding community exposures to industrial hog operation air pollution. Neither received nor expects any financial compensation for these activities. The other authors declare no conflict of interest.

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Attachment A11

ARTICLE

Asthma Symptoms Among Adolescents Who Attend Public Schools That Are Located Near Confined Swine Feeding Operations

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ABSTRACT

OBJECTIVES. Little is known about the health effects of living in close proximity to industrial swine operations. We assessed the relationship between estimated exposure to airborne effluent from confined swine feeding operations and asthma symptoms among adolescents who were aged 12 to 14 years.

METHODS. During the 1999–2000 school year, 58 169 adolescents in North Carolina answered questions about their respiratory symptoms, allergies, medications, socioeconomic status, and household environments. To estimate the extent to which these students may have been exposed during the school day to air pollution from confined swine feeding operations, we used publicly available data about schools ($n = 265$) and swine operations ($n = 2343$) to generate estimates of exposure for each public school. Prevalence ratios and 95% confidence intervals for wheezing within the past year were estimated using random-intercepts binary regression models, adjusting for potential confounders, including age, race, socioeconomic status, smoking, school exposures, and household exposures.

RESULTS. The prevalence of wheezing during the past year was slightly higher at schools that were estimated to be exposed to airborne effluent from confined swine feeding operations. For students who reported allergies, the prevalence of wheezing within the past year was 5% higher at schools that were located within 3 miles of an operation relative to those beyond 3 miles and 24% higher at schools in which livestock odor was noticeable indoors twice per month or more relative to those with no odor.

CONCLUSIONS. Estimated exposure to airborne pollution from confined swine feeding operations is associated with adolescents' wheezing symptoms.

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Key Words
asthma, environmental health, epidemiology, school age children, school health

Abbreviations
CAFO— confined animal feeding operation
PR—prevalence ratio
NCSAS—North Carolina School Asthma Survey
SSLW—steady-state live weight
CI— confidence interval

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During the past 2 decades, the process of raising swine and other livestock has grown into a major industry in the United States. Production has shifted from smaller, family-owned farms to larger, industrialized confined animal feeding operations (CAFOs). Animals in North Carolina's industrialized operations are raised in confinement buildings, housing hundreds to thousands of hogs per operation. Residues of food additives, bedding, dried waste, and animal dander are vented from confinement buildings, and animal waste from the confinement houses is flushed into on-site cesspools, where it begins to decompose and aerosolize anaerobically before being sprayed onto nearby land. There are concerns about the health impacts of exposure to particulate matter, antibiotic residues, volatile organic compounds, and bioaerosols that are present in air that is downwind from confinement buildings, waste lagoons, and spray fields.[1–4]

In occupational settings, adverse respiratory symptoms and changes in bronchial responsiveness and lung function have been observed among confinement building workers.[5–12] Studies that have compared swine CAFO neighbors with other rural residents showed that neighbors reported more frequent respiratory symptoms and mucosal membrane irritation.[13] This literature about health impacts of residential exposures that arise from CAFOs focuses on adults[2,13–15] and may describe inadequately the potential respiratory health effects among children, who may experience notably different physical, educational, and social impacts from such exposures. We designed this research to assess the relationship between self-reported wheezing symptoms among adolescents who were aged 12 to 14 years and estimated exposure to airborne effluent from swine CAFOs.

METHODS

This study combined data about adolescents' respiratory health symptoms, data from a survey of school environments, and location data about swine CAFOs and public schools in North Carolina. Random-intercepts binary regression models were used to estimate prevalence ratios (PRs) that assessed the association between airborne swine pollutants and the prevalence of wheezing symptoms.

North Carolina School Asthma Survey Data

During the 1999–2000 school year, the North Carolina Department of Health and Human Services conducted a statewide respiratory health surveillance project to assess the prevalence of respiratory symptoms among middle school–aged children.[16] Approximately 67% (128 568 of 192 248) of all eligible students participated in the survey, which included core wheezing questions from the International Study of Asthma and Allergies in Childhood questionnaire, a standardized and validated instrument that combines a traditional written questionnaire with a series of video scenes that show children with asthma symptoms.[17–20] To complete the video-based survey questions, students viewed a sequence of video vignettes that showed adolescents experiencing asthma-related symptoms; each scene was followed by time to complete a written survey question, allowing each student to indicate whether he or she had experienced symptoms like those illustrated in the scene.[19,20] We analyzed the prevalence of any wheezing symptoms within the past year ("current wheezing"), as determined by responses to questions about wheezing at rest, waking at night as a result of wheezing, exercise-induced wheezing, and severe wheezing attacks. The definition of current wheezing used here is consistent with that applied in previous analyses of the North Carolina School Asthma Survey (NCSAS) data.[16,21–23]

To evaluate whether the estimated exposure had an impact other asthma-related outcomes, we assessed "severe wheezing" using responses to survey questions about waking at night as a result of wheezing and having a severe wheezing attack during the past year; considered the severe wheezing symptoms to be frequent when they occurred at least once per month ("frequent severe wheezing"); and evaluated physician-diagnosed asthma, medical care, and behavioral consequences of asthma-related symptoms.

Each adolescent also answered questions about age, race, Hispanic ethnicity, allergies, socioeconomic status, cigarette smoking history, and home environment. We included age as a continuous variable (centered at 13) and categorized all other variables: race (black/white); Hispanic ethnicity (yes/no); allergies to cat, dog, dust, grass, or pollen (yes/no); ever smoked cigarettes (yes/no); number of other smokers in household (0, 1, 2, or $\geq$3); and use of a gas stove at home ($<$1 time per month vs $\geq$1 times per month). Socioeconomic status was assessed using responses to a question about payment for lunch at school, with lower economic status designated by receiving free or reduced-price lunch at school compared with paying full price for lunch or bringing lunch to school.

School Environment Data

During the 2003–2004 school year, we mailed 4 copies of a survey to principals of 337 public schools and asked each to distribute the surveys to current school employees. More than 800 anonymous survey respondents, employed in 265 (79%) of the targeted schools, answered questions about their observations of the environmental conditions in and around the school buildings. The survey responses indicated whether there was visible evidence of the presence of cockroaches, rodents, or mold and noticeable odors from indoor (eg, mold) and outdoor (eg, nearby industries) sources of airborne pollutants. Responses were used to create school-level indicator variables for the presence of indoor respiratory

irritants and sources of outdoor air pollution from agriculture and industries that are located near the school. Because of concerns about response bias resulting from social and political conflict surrounding industrial swine production in North Carolina, we asked survey respondents to answer a question about livestock odor generically rather than about odor specifically arising from swine operations. When we received >1 survey from a single school, schools were categorized as positive for a given survey question when any respondent reported the given condition.

Swine CAFO Exposure Estimates

Estimates of exposure to airborne pollution from 2343 swine CAFOs were generated using data from permits that were issued by the North Carolina Division of Water Quality to all CAFOs that house at least 250 animals and use a liquid waste management system. Records contained mandatory information about each CAFO facility, including geographic coordinates and the number, type, and weight of animals (called steady-state live weight [SSLW]) at each operation.[3,24] CAFO operators who filed applications for liquid waste management permits with the state agency provided latitude and longitude coordinates of their operations; the coordinates were verified and corrected, when necessary, when state inspectors visited the operations, although the extent to which the information was corrected by agency inspectors was not recorded in the data (S. Lewis, personal communication, 2002).

Separate exposure estimates were developed on the basis of distances between schools and swine CAFOs and of survey responses about noticeable odors from livestock farms. Distances and geographic directions between schools and CAFOs were calculated using the formulas given by Goldberg et al[25] and Sinnott,[26] respectively. We used calculations of proximity to create 3 metrics of potential exposure for each school: (1) distance to the nearest operation; (2) SSLW within 3 miles; and (3) a weighted SSLW based on the distance between the school and nearby swine CAFOs, the SSLW of each operation, and the proportion of wind measurements in the direction from the operation to the school. We obtained measurements of wind speed and direction recorded at 16 automated weather stations located throughout the state from the State Climate Office of North Carolina (Raleigh, NC). Hourly averages from January 1999 through December 1999 and from the weather station located nearest each school–CAFO pair were used to compute the proportion of time when the wind was blowing from the operation to the school. Weighted SSLW values for each CAFO within 3 miles of a school were the product of the squared inverse of the distance between the school–CAFO pair, the operation's SSLW value, and the proportion of time that regional wind measurements indicated that wind was blowing from the operation toward the school. For each school, weighted SSLW values were summed and the schools were assigned categories of low, medium, and high exposure on the basis of tertiles of the distribution of values among schools with 1 or more swine CAFOs located within 3 miles. A 3-mile radius was selected on the basis of previous research about the impacts of swine CAFOs on health and quality of life among neighbors who live within a 2-mile radius[2,13]; for this research, we expanded the potential zone of exposure to 3 miles because odors from swine CAFOs sometimes are reported at distances of >2 miles.

Study Population

Students in 499 public schools participated in NCSAS, and each student provided data about his or her respiratory health. Schools in 14 counties that did not contain a swine CAFO or border a county with at least 1 swine CAFO (n = 45), schools within the city limits of the 6 cities with populations >100 000 (n = 61), schools within 5 miles of the state border (n = 18), schools with <25 students surveyed (n = 34), schools that had closed or relocated since 2000 (n = 11), and schools that did not respond to the survey about in-school environmental conditions (n = 72) were excluded from our study. The remaining 265 public schools were included in our study. From these 265 schools, a total of 73 305 boys and girls who were aged 12 to 14 years responded to NCSAS. Of those, 58 169 (79%) who reported black or white race and provided complete data for all asthma survey variables of interest constituted our final study population.

Statistical Analyses

Multivariate analyses were conducted separately for individuals with and without self-reported allergies to cat, dog, dust, grass, and/or pollen. To assess the relationship between the prevalence of wheezing symptoms and the estimates of in-school exposure, we used random-intercepts binary regression. This method accounted for the hierarchical clustering of student-level data within schools. Specifically, we used a variation of the generalized linear mixed model $E(Y|x) = \exp(\alpha + \Sigma\beta x)$ similar to those described by Singer[27] and McLeod,[28] in which the student's outcome is modeled by a combination of student-level (level 1) and school-level (level 2) models. The student-level model was defined as

$$\log_e(P_{ij}) = \beta_{0j} + \beta_1 x_{1j} + \beta_2 x_{2j} + \ldots + \beta_n x_{nj} \text{ (level 1)},$$

where P_{ij} is the probability of outcome y = 1 for individual i in school j, $p_{ij} \sim$ binomial; β_{0j} is school-specific intercept (intercept for school j); and β is the effect of individual-level predictor x_{ij}. Level 1 models included student-level variables for age, gender, race, Hispanic ethnicity, economic status, allergy status, ciga-

rette smoking experience, number of other smokers in the household, and use of a gas kitchen stove at home. The school-level (level 2) model was defined as

$$\beta_{0j} = \beta_0 + \mu_1 z_1 + \mu_2 z_2 + \ldots + \mu_n z_m + \mu_{0j} \text{ (level 2)},$$

where β_0 is the mean of school-level means for outcome y (ie, fixed intercept); μ is the effect of school-level predictor z_j; z_j is the school-level predictor for school j; $\mu_{0j} \sim N(0,\tau_{00})$; and τ_{00} is between-school variance. The level 2 models included main exposure variable(s) and indicator variables for rural school locale, survey-reported presence of indoor respiratory irritants (cockroaches, rodents, mold visible, mold odor, or flooding of school buildings within the past 5 years), and survey-reported industry other than a swine CAFO located near the school. The level 2 model, substituted into the level 1 model, results in a final 2-level random-intercepts model,

$$\log_e(P_{ij}) = \beta_{0j} + \beta_1 x_{1j} + \beta_2 x_{2j} + \ldots + \beta_n x_{nj} + \mu_1 z_1 + \mu_2 z_2 + \ldots + \mu_n z_m + \mu_{0j},$$

where μ_{0j} is the random intercept term. Associations were estimated as PRs (exp[μ]) using SAS statistical software version 8.2 (SAS Institute Inc, Cary, NC).

RESULTS

More than 26% (15 250 of 58 169) of students who participated in NCSAS during the 1999–2000 school year reported wheezing during the past year (ie, current wheezing). Table 1 shows adjusted PRs for individual- and school-level characteristics. Of the individual-level characteristics, the highest PR was observed for self-reported allergy status (PR: 2.20; 95% confidence interval [CI]: 2.14–2.27). Variations in the prevalence of current wheezing by school-level characteristics and indicators of school-specific environmental health conditions were less pronounced.

Of the 265 schools, 66 (25%), including 10 518 (18%) surveyed students, were located within 3 miles of at least 1 (range: 1–27) swine CAFO. More than 50% of the schools were within 7 miles of the nearest operation (median: 6.7 miles; range: 0.22–42.0 miles). The average SSLW capacity of operations that were located within 3 miles of a school was slightly lower than that of operations that were located beyond 3 miles (556 283 lb vs 605 139 lb), and, overall, the SSLW capacity of swine CAFOs increased with increasing distance from the nearest surveyed school (β [SE] per mile = 15 948 [4791]). On the basis of the environmental health surveys and according to survey respondents, livestock odor was noticeable outside buildings in 86 (33%) schools and inside the buildings in 39 (15%) schools.

Table 2 presents adjusted PRs for wheezing using each exposure measure separately for students with and without allergies. PRs were 1.05 (95% CI: 1.00–1.10) and 1.02 (95% CI: 0.94–1.11) for adolescents who did and did not have allergies, respectively, and attended schools that were located within 3 miles of the nearest swine CAFO. PRs were approximately unity for schools that were closer than 2 miles, compared with schools with no nearby swine CAFOs, and were 1.12 (95% CI: 1.04–1.19) and 1.08 (95% CI: 0.95–1.21), respectively, for students who did and did not have self-reported allergies and attended schools that were located between 2 and 3 miles from the nearest operation. Associations with SSLW and the weighted SSLW exposure categories also tended to be highest for the low exposure groups and closer to unity for higher exposure groups compared with schools with no nearby swine CAFOs. Basing potential exposure estimates on survey-reported livestock odor resulted in 20 fewer schools' and 3315 fewer adolescents' being considered unexposed. The prevalence of current wheezing was 24% and 21% higher among allergic and nonallergic students, respectively, at schools in which livestock odor was noted inside the school building 2 or more times per month relative to the prevalence at schools without any survey reports of livestock odor.

Table 3 presents adjusted associations between school proximity within 3 miles of a swine CAFO and alternative asthma outcomes as well as functional consequences of asthma-related symptoms. Results indicate that larger proportions of adolescents who attended school near at least 1 swine CAFO experienced respiratory symptoms, physician diagnosis, asthma-related medical treatment, activity limitations, and missing school because of their symptoms. In the population of all students, the largest PRs were observed for physician-diagnosed asthma (PR: 1.07; 95% CI: 1.01–1.14), medication use (PR: 1.07; 95% CI: 1.00–1.15), and visit to a physician or an emergency department or hospitalization (PR: 1.06; 95% CI: 1.00–1.12). Most associations were slightly higher in adolescents with self-reported allergies; however, the PR for physician-diagnosed asthma was higher among students without (PR: 1.14; 95% CI: 1.01–1.26) compared with those with (PR: 1.06; 95% CI: 0.99–1.12) self-reported allergies. Adjusted associations between these outcomes and the presence of livestock odor in and around the schools indicate only slightly elevated proportions of wheezing symptoms, physician diagnosis, use of asthma-related medical care, activity limitations, and missed school among students in schools where employees reported noticeable livestock odor (Table 4). When school-level exposures were assigned on the basis of reported livestock odor (Table 4), the PRs for severe wheezing (PR: 1.05; 95% CI: 1.00–1.10) and frequent severe wheezing (PR: 1.06; 95% CI: 0.98–1.14) were higher than when exposure was assigned on the basis of distance to the nearest swine CAFO (severe wheeze, ≤3 miles: 1.02 [95% CI: 0.97–

TABLE 1 **Characteristics of North Carolina School Asthma Survey Participants and Public Schools in North Carolina**

	N	Students Who Reported Current Wheezing, *n* (%)	PR (95% CI)[a]
Total	58 169	15 250 (26.2)	—
Age, y[b]			
12	17 905	4873 (27.2)	1.06 (1.04–1.08)
13	28 130	7268 (25.8)	1.00[c]
14	12 134	3109 (25.6)	0.95 (0.93–0.96)
Race			
White	43 590	10 919 (25.1)	1.00
Black	14 579	4331 (29.7)	1.04 (1.01–1.08)
Gender			
Male	28 342	6798 (24.0)	1.00
Female	29 827	8452 (28.3)	1.07 (1.04–1.10)
SES indicator			
Lunch not subsidized	41 719	10 088 (24.2)	1.00
Lunch subsidized	16 450	5162 (31.4)	1.16 (1.12–1.20)
Hispanic ethnicity			
No	54 827	14 236 (26.0)	1.00
Yes	3342	1014 (30.3)	1.11 (1.06–1.16)
Allergies			
No	31 480	5149 (16.4)	1.00
Yes	26 689	10 101 (37.9)	2.20 (2.14–2.27)
Ever smoked			
No	40 632	9154 (22.5)	1.00
Yes	17 537	6096 (34.8)	1.35 (1.31–1.39)
No. of other smokers in household[b]			
0	27 662	6138 (22.2)	1.00
1	16 079	4447 (27.7)	1.09 (1.07–1.10)
2	10 209	3178 (31.1)	1.18 (1.15–1.21)
≥3	4219	1487 (35.3)	1.29 (1.24–1.34)
Frequency of gas kitchen stove use			
Less than once per more	45 546	11 384 (25.0)	1.00
Once per month or more	12 623	3866 (30.6)	1.14 (1.11–1.17)
Rural school locale			
No	30 154	8074 (26.8)	1.00
Yes	28 015	7076 (25.6)	0.96 (0.92–1.00)
In-school asthma triggers[d]			
No	4619	1147 (24.8)	1.00
Yes	53 550	14 103 (26.3)	1.03 (0.95–1.11)
Location near non-livestock industry[e]			
No	52 184	13 603 (26.1)	1.00
Yes	5985	1647 (27.5)	1.06 (0.99–1.13)

PR indicates prevalence ratio; SES, socioeconomic status.

[a] Adjusted for all individual-level and school-level covariates in the table.

[b] Included in the model as a continuous variable.

[c] Referent category.

[d] Environmental Health Survey responses about cockroaches, rodents, mold, and/or flooding in school buildings (no: 24 schools; yes: 241 schools).

[e] Environmental Health Survey responses about non-livestock industries located near the school (No: 236 schools; Yes: 29 schools).

1.07]; frequent severe wheeze, ≤3 miles: 1.01 [95% CI: 0.92–1.09]; Table 3).

DISCUSSION

We observed elevated prevalences of current wheezing among 12- to 14-year-old students who attended public schools near swine CAFOs, especially among students with self-reported allergies. Such associations are plausible, given that swine CAFOs are sources of bioaerosols, endotoxins, and other airborne asthma triggers. The availability of standardized symptom data and the independence of symptom and exposure data strengthen confidence in the validity of our findings. Overall, estimates of excess current wheezing symptoms among students who attended schools nearby swine CAFOs are as high as 24% among students who attended schools where livestock odor was reported outside as well as inside 2 or more times per month. Excess prevalence of current wheezing tended to be greater among students who reported allergies. Although the majority of the

TABLE 2 **Associations Between the Prevalence of Wheezing and Exposure to Confined Swine Feeding Operations by Adolescents' Self-Reported Allergic Status, North Carolina**

	Total No. of Schools	Self-Reported Allergies (n = 26 689)			No Self-Reported Allergies (n = 31 480)			All (N = 58 169)		
		Total No. of Students	Wheeze, n (%)[a]	PR (95% CI)[b]	Total No. of Students	Wheeze, n (%)	PR (95% CI)[b]	Total No. of Students	Wheeze, (%)	PR (95% CI)[c]
Current wheeze			10 101 (37.9)			5149 (16.4)			15 250 (26.2)	
Miles to nearest swine CAFO										
>3	199	21 898	8145 (37.2)	1.00	25 753	4138 (16.1)	1.00	47 651	12 283 (25.8)	1.00
≤3	66	4791	1956 (40.8)	1.05 (1.00–1.10)	5727	1011 (17.7)	1.02 (0.94–1.11)	10 518	2967 (28.2)	1.04 (0.99–1.09)
2 to ≤3	22	1865	822 (44.1)	1.12 (1.04–1.19)	2107	396 (18.8)	1.08 (0.95–1.21)	3972	1218 (30.7)	1.10 (1.02–1.18)
≤2	44	2926	1134 (38.8)	1.01 (0.95–1.07)	3620	615 (17.0)	0.99 (0.89–1.09)	6546	1749 (26.7)	1.01 (0.95–1.07)
Hog pounds (in millions) within 3 miles of school										
None	199	21 898	8145 (37.2)	1.00	25 753	4138 (16.1)	1.00	47 651	12 283 (25.8)	1.00
0.1 to <2.0	42	3342	1388 (41.5)	1.07 (1.01–1.12)	4017	713 (17.8)	1.03 (0.93–1.12)	7359	2101 (28.6)	1.05 (1.00–1.11)
2.0 to <5.0	12	733	294 (40.1)	1.04 (0.93–1.14)	858	150 (17.5)	0.99 (0.81–1.16)	1591	444 (27.9)	1.01 (0.91–1.12)
≥5.0	12	716	274 (38.3)	1.00 (0.89–1.11)	852	148 (17.4)	1.04 (0.85–1.23)	1568	422 (26.9)	1.02 (0.91–1.13)
Exposure category										
None	199	21 898	8145 (37.2)	1.00	25 753	4138 (16.1)	1.00	47 651	12 283 (25.8)	1.00
Low	21	1655	711 (43.0)	1.10 (1.03–1.18)	1922	359 (18.7)	1.09 (0.95–1.23)	3577	1070 (29.9)	1.09 (1.01–1.18)
Medium	22	1741	771 (40.8)	1.04 (0.97–1.12)	2139	378 (17.7)	1.01 (0.89–1.13)	3880	1089 (28.1)	1.03 (0.96–1.11)
High	23	1395	534 (38.3)	1.01 (0.93–1.08)	1666	274 (16.5)	0.97 (0.84–1.10)	3061	808 (26.4)	1.00 (0.92–1.08)
Livestock odor										
None	179	19 055	7188 (37.7)	1.00	22 438	3694 (16.5)	1.00	41 493	10 882 (26.2)	1.00
Outside school only	47	4625	1766 (38.2)	1.04 (0.98–1.09)	5593	843 (15.1)	0.94 (0.85–1.02)	10 218	2609 (25.5)	1.00 (0.95–1.06)
Outside + inside <2 times/mo	36	2745	1022 (37.2)	0.99 (0.93–1.06)	3137	550 (17.5)	1.04 (0.93–1.15)	5882	1572 (26.7)	1.01 (0.94–1.07)
Outside + inside ≥2 times/mo	3	264	125 (47.4)	1.24 (1.03–1.44)	312	62 (19.9)	1.21 (0.85–1.57)	576	187 (32.5)	1.23 (1.01–1.44)

[a] Any wheeze in the past 12 months (current wheeze).
[b] Adjusted for individual-level characteristics (gender, age, race, Hispanic ethnicity, economic status, smoking status, exposure to second-hand smoke at home, and use of a gas stove more than once per month) and school-level characteristics (rural locale, indoor air quality, and reports of other non-livestock industries nearby).
[c] Adjusted for variables listed above plus self-reported allergy to cats, dogs, dust, grass, and/or pollen.

TABLE 3 **Associations Between the Prevalence of Asthma-Related Symptoms and School Location Within 3 Miles of a Confined Swine Feeding Operation by Adolescents' Self-Reported Allergic Status, North Carolina**

	PR (95% CI) for ≤3 vs >3 Miles From Nearest Swine CAFO		
	Self-Reported Allergies (n = 26 689)	No Self-Reported Allergies (n = 31 480)	All (N = 58 169)
Wheezing symptoms			
Current wheeze	1.05 (1.00–1.10)	1.02 (0.94–1.11)	1.04 (0.99–1.09)
Current wheeze without physician diagnosis	1.08 (1.01–1.15)	0.99 (0.90–1.08)	1.04 (0.98–1.11)
Severe wheeze[b]	1.01 (0.96–1.07)	1.05 (0.96–1.14)	1.02 (0.97–1.07)
Frequent severe wheeze[a]	1.02 (0.92–1.11)	0.97 (0.80–1.14)	1.01 (0.92–1.09)
Physician-diagnosed asthma	1.06 (0.99–1.12)	1.14 (1.01–1.26)	1.07 (1.01–1.14)
Medical care			
Asthma-related physician visit, emergency visit, and/or hospitalization in past year	1.06 (1.00–1.13)	1.03 (0.92–1.13)	1.06 (1.00–1.12)
Asthma medication use in past year	1.09 (1.00–1.18)	1.03 (0.88–1.18)	1.07 (1.00–1.15)
Functional consequences of symptoms			
Activity limitations in past year as a result of asthma symptoms	1.09 (1.01–1.16)	—[b]	—
Missed school in past year as a result of asthma symptoms	1.06 (0.98–1.14)	—	—

[a] Among individuals with current wheeze.
[b] Nonconvergent model.

estimates are small in relative terms, the increases are important in absolute terms because of the high prevalence of asthma-related symptoms in this age group; the impact that symptoms have on adolescents' ability to attend school and participate in social, recreational, and physical activities; and the costs and burdens of symp-

TABLE 4 **Associations Between the Prevalence of Asthma-Related Symptoms and the Presence of Livestock Odor at the School by Adolescents' Self-Reported Allergic Status, North Carolina**

	PR (95% CI) for Livestock Odor Reported Outside or Inside School Building Versus No Reported Odor		
	Self-Reported Allergies (n = 26 689)	No Self-Reported Allergies (n = 31 480)	All (N = 58 169)
Wheezing symptoms			
Current wheeze	1.03 (0.98–1.07)	0.99 (0.91–1.06)	1.01 (0.97–1.06)
Current wheeze without physician diagnosis	1.04 (0.97–1.10)	0.99 (0.90–1.07)	1.01 (0.96–1.07)
Severe wheeze[a]	1.06 (1.01–1.12)	1.00 (0.91–1.08)	1.05 (1.00–1.10)
Frequent severe wheeze[a]	1.04 (0.95–1.14)	1.10 (0.92–1.28)	1.06 (0.98–1.14)
Physician-diagnosed asthma	1.00 (0.94–1.06)	1.04 (0.93–1.15)	1.01 (0.95–1.06)
Medical care			
Asthma-related physician visit, emergency visit, and/or hospitalization in past year	0.99 (0.94–1.05)	1.01 (0.91–1.10)	1.00 (0.95–1.05)
Asthma medication use in past year	1.03 (0.96–1.11)	1.02 (0.89–1.15)	1.03 (0.96–1.10)
Functional consequences of symptoms			
Activity limitations in past year as a result of asthma symptoms	1.02 (0.96–1.08)	—[b]	—
Missed school in past year as a result of asthma symptoms	1.02 (0.94–1.09)	—	—

[a] Among individuals with current wheeze.
[b] Nonconvergent model.

tom-related medical care. In these data, the effect estimates for swine CAFO exposures are of similar magnitude to the effects that have been estimated for established risk factors for wheeze, such as age, race, gender, economic status, Hispanic ethnicity, exposure to secondhand cigarette smoke, and use of a gas stove at home.

We estimated potential exposure on the basis of distance and a mailed survey. Although distance is a crude measure of exposure, our findings suggest a consistent trend toward higher symptom prevalence, especially among adolescents with allergies, at schools that were between 2 and 3 miles of a swine CAFO. The finding that schools that were located within 2 miles had a lower prevalence of current wheezing may reflect the lack of a direct relationship between exposure to etiologically active agents and distance. Use of distance and SSLW as exposure measures does not take account of waste management and sanitation practices of swine CAFOs, ages and conditions of the facilities' equipment, localized weather patterns, topography surrounding the school, school building structure, and ventilation practices, all of which may affect the quantity and the duration of the exposures. In addition, swine CAFO practices such as waste and sanitation procedures may be influenced by population density, land availability, and other features of the communities in which the operations are located, although we do not know the extent to which this occurs. Indeed, results of analyses that used exposure metrics of increasing complexity failed to show a monotonic dose-response relationship between the exposure and current wheezing, further suggesting that if the exposure is associated with an increase in respiratory symptoms, then relevant exposure may not correlate directly with the factors that we used for our distance-based exposure categories.

The higher prevalence of current wheezing among students who attended schools that were located 2 to 3 miles from the nearest swine CAFO compared with the prevalence among students who attended schools within 2 miles also may be attributable to exposures that were experienced at home, in the communities where students lived, and in other locations that could not be assessed in our study. In many of the rural areas in North Carolina, students may live many miles from the public schools that they attend. As the distance between the school and the CAFO becomes small, few homes can be equally close or closer to a CAFO; as the distance increases, more of the students' homes can be located closer to a CAFO than the distance between the CAFO and the school, and school-based exposure estimates will underestimate students' total swine CAFO exposures. In addition, reports of odor from swine CAFOs tend to be more common in early morning and evening hours rather than in the daytime, when students are in school. Although this phenomenon may not affect exposures in geographic areas where both schools and homes are far from CAFOs, identifying exposure as the distance between a school and a CAFO may be more problematic in regions where schools are located very near or within several miles of CAFOs if exposure varies throughout the day. Previous research that was conducted in a rural population of school-aged children who may have experienced swine farm exposures at home indicated a higher prevalence of asthma-related symptoms among children who lived on farms where swine were raised

than among children who lived on farms where swine were not raised and among children who did not live on farms,[29] although the extent to which exposures that resulted from residence on a swine farm were attributable to performing chores or occupation-like tasks, rather than simply living close to swine, are unknown. Although information about adolescents' household farming exposures are unavailable in our study population, the majority of swine in North Carolina are raised in nonresidential, factory farm settings; therefore, the proportion of children who perform chores or live on swine farms is expected to be low.

Results of analyses of the distance-based measures of each exposure suggest lower prevalence of wheezing among students who attended schools that were located nearest to CAFOs and located in areas with the highest density of swine compared with those in the highest exposure categories. To assess potential misclassification of exposure, we excluded from all analyses schools with reported livestock odor from the unexposed distance-based categories, schools that were located beyond 3 miles of swine CAFO from the exposed survey-based categories, and schools for which survey respondents specifically identified livestock odor as arising from poultry and found no notable differences in the direction, magnitude, or precision of the PRs generated. An alternative explanation for the lower prevalence of wheezing among students in schools that were located nearby swine CAFOs may be the hygiene hypothesis, which postulates that early-life exposures and childhood infections may confer protection against hay fever, atopy, and asthma.[30,31] Specifically, rural living and early-life exposures to allergens, irritants, and other bioaerosols on farms may be associated with lower rates of atopy and asthma.[29,32–38] In our study, the prevalence of wheezing was slightly lower (−1.2%) in rural compared with nonrural schools. Although we could not assess early-life exposures, higher exposures to animal dander and bacterial endotoxin during early developmental stages among individuals who attend schools closest to swine CAFOs and therefore often live in rural areas could provide some resistance to exposures later in childhood and lead to lower prevalence of wheezing during adolescence compared with students who attend schools farther away.

Twenty-one percent (n = 72) of schools were excluded from our final analysis because of nonparticipation in our mailed survey about in-school environmental conditions. When we compared the populations of schools that participated and those that did not, we found differences in mean distance to the nearest swine CAFO (participating schools: 8.7 miles; nonparticipating schools: 8.0 miles), percentage of nonwhite enrollment (participating schools: 36%; nonparticipating schools: 42%), and percentage of enrolled students who received subsidized school lunches (participating schools: 48%; nonparticipating schools: 51%). Systematic differences between participating and nonparticipating schools in levels of exposure and prevalences of asthma-related symptoms could have influenced our findings.

We received up to 7 completed surveys per school, and for each survey question, we assigned an exposure to a school when any respondent indicated the presence of the exposure. This method of classifying schools' environmental conditions and, in particular, the presence of livestock odor at the school was sensitive to the number of surveys completed and returned from each school and did not take into account the variation in survey responses from a single school. Our intention was to survey employees in several occupations who would be familiar with different aspects of the school building and students' behaviors: teacher, administrator, maintenance or custodial staff, and school nurse or health care personnel. Previous literature about the economic, political, and social impacts of a strong swine industry presence in communities in Iowa and North Carolina suggested that residents who live near swine CAFOs may be reluctant to voice their concerns for fear of social ostracism or conflict in their communities.[39–42] Although our school survey was anonymous and designed to minimize risks for deductive disclosure of respondents' identities, we recognize the possibility that respondents may have underreported livestock odor out of concern for expressing their opinions, and we cannot know fully the extent to which our survey reports were influenced by the social and political context in the communities in which the schools were located.

Lack of data on medical risk factors, environmental asthma triggers, and classification of allergic status on the basis of survey reports rather than of a clinical assessment of atopy are limitations of this study. Because students self-identified asthma-related symptoms, our current wheezing variable may include other respiratory symptoms that the respondents experience and mistake for the symptoms that were illustrated in the video scenes. Cross-sectional asthma-related symptom data and survey-based exposure data prohibit specific assessment of temporal relationships between the symptoms and exposures evaluated here. Our findings are vulnerable to systematic error if students with asthma-related symptoms changed their environments or behaviors because of symptoms that were caused by exposure to airborne pollution that arose from swine CAFOs; such a systematic error would lead to underestimation of associations between swine CAFOs and asthma symptoms.

CONCLUSIONS

This research was designed to estimate exposures to a source of air pollution that is of great concern to swine CAFO neighbors and to investigate relationships between school exposures and respiratory health of middle school–aged children. Our findings identify a plausible

association between exposure to airborne pollution from swine CAFOs and wheezing symptoms among adolescents. Environmental pollution measurement and standardized clinical information about asthma symptoms and atopic status could help to determine better the magnitude and the temporality of the relationships between swine CAFO emissions and respiratory symptoms. Our findings should be used by public health personnel who are interested in understanding possible adverse respiratory health consequences of an important rural environmental exposure.

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Asthma Symptoms Among Adolescents Who Attend Public Schools That Are Located Near Confined Swine Feeding Operations

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Attachment A12

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IMPACT OF ODOR FROM INDUSTRIAL HOG OPERATIONS ON DAILY LIVING ACTIVITIES*

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ABSTRACT

Intensive industrial animal production systems worldwide require confinement of large numbers of animals in small spaces and concentration of enormous quantities of waste. Industrial hog operations, in particular, have raised public concerns about their adverse impact on public health and sustainable development. Using a community-based participatory research approach and qualitative interviews, we explored people's perception of the impact of odor from these industries on daily living activities as they relate to the beneficial use of property and enjoyment of life. Our research indicates that hog odor limits several leisure time activities and social interactions which could have adverse public health consequences. The results of this study can assist the communities and other stakeholders in public policy development that addresses these concerns.

Worldwide, livestock production systems are changing from small farms to intensive industrial production systems [1]. In the case of pigs, significant

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concentrations have developed in areas where access to corn and soybean by-products are available for animal feed, as in the Netherlands, or in regions where government policies and vertical integration have resulted in rapid growth, for example, in North Carolina [2]. In the U.S, which ranks third in pig meat production (9.3 million metric tons annual production) after the People's Republic of China (48.3 million metric tons) and the European Union with 25 member states (21.6 million metric tons), approximately 79% of the 62 million hogs produced annually comes from medium or large corporate facilities [3]. North Carolina, the location of this study, rose from the country's fifteenth- to its second-largest pork-producing state in less than two decades [3], with a rapid shift from small family farms to large-scale corporate production.

In contrast to traditional small farms, industrial production systems involve large concentrations of animals in confined spaces and the generation of enormous quantities of solid, liquid, and gaseous wastes in small geographic areas. In North Carolina, they are disproportionately concentrated in communities of color and/or low-income communities, resulting in environmental injustices and disproportionate exposure to environmental and public health risks [4-7]. Adverse impacts of pollution from hog waste have been examined on ground and surface waters [8-11], economic and employment development [12, 13], devaluation of land and properties [14, 15], respiratory and other health problems, and quality of life [4, 16-19].

However, the effect that concentrated hog waste and odor have on people's daily activities related to beneficial use of property and quiet enjoyment of life requires in-depth exploration. Enjoyment of life and beneficial use of property are two of the components considered in defining a nuisance. A nuisance is generally defined as a use of property or an activity that unreasonably limits or diminishes a person's health, safety, or enjoyment of life; or that interferes with the other person's quiet enjoyment or beneficial use of his or her own property [20]. More specific to farm nuisance in North Carolina, nuisance is defined in N.C. General Statute § 7A-38.3—*Pre-litigation mediation of farm nuisance disputes,* as an action that is injurious to health, indecent, offensive to the senses, or an obstruction to the free use of property.

This article explores community members' perception of odor from industrial hog operations and its impacts on daily living activities related to the beneficial use of property and quiet enjoyment of life. Our goal is to provide public health policy recommendations associated with the built environment rather than recommendations for legal remedies. Our analysis uses data from the qualitative part of a larger community-based participatory research (CBPR) project that explored the impact of industrial hog operations (IHOs) on health and quality of life [21]. Community-based participatory research is a partnership approach to research that equitably involves community members and other stakeholders in the research process and builds on the partners' strengths [22]. Rooted in

part in the revolutionary approaches to research that emerged from works with oppressed communities in Africa, South America, and Asia in the 1970s [23-25], in some respects, CBPR is considered an orientation to research rather than a particular research method [26]. Under this conception, CBPR addresses health from a broad ecological perspective and engages the community in the research process in a manner that is participatory and builds community capacity [22, 27, 28] without creating colonial relationships between research institutions and lay communities. It integrates local and academic or professional knowledge and expertise and focuses on community-driven issues and actions to improve health as part of the research process.

METHODS

Two series of in-depth interviews were conducted with 75 participants in eastern North Carolina in 2002 and again in 2004 and 2005. Participants were adults (18 years or older) who lived near industrial hog operations and were willing to be interviewed. The first series of interviews was conducted with a convenience sample of 26 participants. The results from these interviews were used to guide the development of a study instrument for a second series of semi-structured open-ended interviews. In this series, 34 interviews were conducted with 49 additional participants from 16 neighborhoods and communities. Some interviews were with couples in the same household. Forty-two of the 49 interviewees also participated in a quantitative longitudinal study exploring the impact of hog odor on health prior to their interview. All participants lived within 1.5 miles of one or more hog operations and were non-smokers. The interviews were conducted in the homes of participants and lasted between 30 minutes and two hours. Informed consent and written permission to audio record the interviews were obtained prior to the start of the interviews. A pair of interviewers, consisting of one academic and one community organizer, conducted each interview.

In addition to information on basic demographic characteristics (age, gender, and race), data were collected about context, experience, beliefs and attitudes, coping mechanisms, capabilities, and individual and/or collective actions as they related to hog odor. Table 1 provides a summary of the general categories and rationale for the questions that were explored during the interviews.

Interviews were audiotaped and transcribed, and an evolving list of codes was developed for all interviews by two members of the research team using grounded theory approach [29-31]. During the initial steps of analysis, interview texts were thoroughly read and passages were first open coded and then categorized according to relevant research questions and a code book was created based on axial coding. Open coding involves forming initial categories of information and assigning codes by segmenting the text; in axial coding, data are assembled based on a coding paradigm [31]. Initial validation of the codes

Table 1. Categories and Questions Explored in In-Depth Interviews

Category	Purpose	Questions
Context	To explore social and physical environment	• Do you consider yourself living in a community? If yes, what makes it a community to you? • What do you like about your community? Why? • What do you dislike about your community? Why?
Experiences and their meanings	To explore emotions, feelings, beliefs, and meta-beliefs	• What was it like for you growing up? • What are/were some activities you enjoy doing? • What do/did those activities mean to you? • What are/were some activities you enjoy doing but you no longer do? Why?
Coping mechanism	To explore attitudes and responses to hog odor	• What do you do when the odor comes? [Probe: 1) what are specific lifestyle changes that are related to hog odor? 2) What are specific actions (social and political) taken that are related to hog odor?]
Recognizing capabilities	To create ongoing reflection in participants about looking into realities and 1) *recognizing* something they might want to have but do not currently have e.g., a voice in decision-making process); 2) *understanding* the implications of not having this capability when faced with other similar problems; 3) creating a new ability to fill this void.	• What do you think can be done about the odor? • What role do you think you and/or other community members could play in addressing the problem? (Why or why not?) • What (resources) would you (or others) need to be able to do this?

was undertaken by reviewing and discussing the formulated descriptions and categories of themes with key informed community partners. The second validation step was undertaken by returning to 12 of the 49 participants in the original 34 interviews. To ensure these participants had an equal chance of being selected for the second validation step, we randomly selected 10 of 34 interviews using Random Generator for Microsoft Excel software and conducted the validation sessions with their respective participants. Two participants were lost to follow up and the final validation sessions occurred with eight of the 10 randomly selected interviews. During these validation sessions, the participants were presented with the codes and the transcription of their statements that corresponded with those codes and asked to verify if the codes accurately depicted what they had said during their in-depth interviews.

The questions explored in this article are: From the perspective of the participants, what is beneficial use of property and how does hog odor interfere with those uses? From the perspective of the participants, what is enjoyment of life and how does hog odor interfere with that? What is the extent of the interference in terms of time and place?

RESULTS

Sixteen neighborhoods and communities where the participants lived are located in three counties in eastern North Carolina and have high concentrations of industrial hog operations. Demographic characteristics of interview participants are summarized in Table 2.

Table 2. Demographic Summary of the Participants

Category	No. (%)
Number of participants	49
Number of interviews	34
Number of Black participants	43 (87.8%)
Number of White participants	6 (12.2%)
Number of female participants	32 (65.3%)
Number of male participants	17 (34.7%)
Hours of interview	34 hours, 11 min
Average age of participants	57 years
Age range (in years)	32-84

Data obtained from the U.S. Census Bureau [32] indicated that African Americans constituted from greater than 45% to nearly 90% of the people living in these communities at block group levels. At the time of the interviews, all participants in our study owned their homes and, with the exception of two participants who had lived in the community for fewer than five years, they had been born and raised or had lived in their respective communities most of their lives. More than half of the participants lived on a land that had been in their family for more than one generation. Most participants had lived on or near a farm and were familiar with odor from non-industrial hog farms. Qualitative description of the participants about the proximity of their homes to one or more hog operations showed that the hog facilities and/or their spray fields (spray fields are open fields where liquified hog waste is sprayed using large sprinkle systems) were quite visible and sometimes the sprayfields extended to the participant's "door step" and "driveway" or they were located "down the road." All participants lived within a 1.5-mile radius of a hog facility, which was one of the criteria for participation in the larger exposure study.

Examples of participants' significant statements about activities of daily living that related to and were coded as "beneficial use of property" and "quiet enjoyment of life" included working outside, growing vegetables, sitting outside, eating outside, gardening, playing, barbequing, use of wellwater, sleeping, opening doors and windows, hanging out with neighbors, having family and guests over, and drying laundry among others. They are presented in Tables 3 and 4, respectively, along with the examples of statements about the impact of hog odor on those activities.

These were recurring themes in almost all interviews with differences in participants' qualitative description of how the hog odor schedule, duration, and intensity affected these activities in terms of time of day, frequency, and duration. The impact of hog odor on these activities occurred among all participants with some participants not engaging in those activities "any more" or "as often as they used to," or scheduling those activities around hog odor's "schedule" and/or "intensity" to those who would interrupt what they were doing and "go inside when the odor comes." Participants used words and phrases such as "bad," "terrible," "it just stinks," or "you can't stand it" to qualify the odor and explained when the odor comes, they will "go inside," "lose appetite," or "stop doing what [they] are doing and go inside." Activities that participants did not engage in "any more" were mostly social activities such as family reunions and having guests over to avoid embarrassment and shame due to the possible untimely arrival of hog odor. Two participants reported that the hog odor did not interfere with their beneficial use of property as illustrated in the following direct quote: "Health problems have not affected me . . . a lot of people are experiencing serious respiratory problems though . . . with myself, I continue to do things outside . . . I know there are communities much worse than the one that I live in." In terms of adverse environmental and economic impacts, hog odor was

Table 3. Examples of Participants' Significant Statements about Beneficial Use of Property

Beneficial use of property (BUP)	Hog odor interference with BUP
1. Sit outside	1. Can't sit outside
2. Eat outside	2. Can't have guests over
3. Cook out	3. Can't have cookouts
4. Barbeque	4. Can't have family reunions
5. Dry laundry	5. Can't play outside
6. Have guests over	6. Can't garden
7. Open windows/doors to air the house	7. Can't hang out with neighbors
8. Fresh clean air	8. Can't keep working outside with odor
9. Open window at night [for cool air]	9. Devaluation of property
10. Sleep	10. Unpredictability of odor
11. Garden	11. Can't use well water
12. Play outside [children]	12. Buy bottled water
13. Family reunion on property	13. Had to get and use air conditioner
14. Hang out with neighbors	14. Had to buy a dryer
15. Grow own vegetables	15. Have a hard time sleeping or wake up at night
16. Use well water for drinking	

Examples of participants' statements related to beneficial use of property

"The beautiful landscape that we have here and the animals roaming about. And you could just walk out, you could just sit outside and enjoy in the summertime."

"He [child in the family] likes to go outside. He likes to play basketball."

Examples of participants' statements related to hog odor interference

"You wouldn't invite a person over for a cookout on your deck if you expected hog odor to come in."

"I only had one cookout. One cookout [repeated for emphasis by the participant]. That has changed because I don't invite people over because I don't want them to come in and smell that odor."

"Couldn't invite people over for a cookout, family reunion."

"I had my uncle, my grand daddy, I had my grandma before she died. A lot of my family come and can't stay here. They say, 'god, I can't stand this. How can you live here?'"

"My son has asthma and allergies . . . he just stays inside."

"I had a rose garden . . . do you see those weeds there . . . I haven't done it for the past few years. . . ."

"Sometimes it's so unbearable you couldn't even hardly stand it, not even in the house."

"On a bad day it is not that you can't go outside . . . but the odor determines how long you gonna stay . . ."

"When the smell [hog odor] get in, you can't get rid of it."

"They went up so much that we went in to talk to him. And I had stuff here in writing saying that the property has gone down 20-30 percent because you are near a hog farm."

"It [the odor] could come any time, day or night . . . mostly at night was worse."

"The water turns everything yellow. If I wash my clothes for a good six weeks in that water, I will have to buy new clothes . . . I will have to buy new clothes every six weeks."

"I don't drink the ground water no more because of the hog farms . . . now we have to buy water to drink."

"It [hog odor] woke me up. And I had to get up. I couldn't sleep. I put the covers up over my face and it didn't do any good."

Table 4. Examples of Participants' Significant Statements about Quiet Enjoyment of Life

Quiet Enjoyment of Life (QEL)	Hog odor interference with QEL
1. Enjoy being outside	1. Staying outside
2. Enjoy working outside	2. Socializing
3. Enjoy visiting with neighbors	

Examples of participants' statements related to Quiet Enjoyment of Life:

"We enjoyed the outdoors. That was the main spot. Sitting in the house was not our thing."

"We would get together as a community and do a lot of things."

"I used to like to go outside for walks and breathe fresh air."

"I love to sit on my porch when possible . . . and I will sit out there as long as I can."

"I enjoy gardening . . . woodworking."

"A lot of times when I get home from work . . . like to go outside to visit with neighbors . . . play ball."

"You take away outside for anyone living in the country, to a degree they will cease living."

"We grew up loving the outdoors and now it is a part of us. So when we get together, we still do things outside"

Examples of participants' statements related to hog odor interference

"I had to pay money to buy this place. Why can't I go sit outside and enjoy?"

"I've been in this spot all my life. When me, my mom and my dad first lived here before the hogs . . . we could stay outside late at night until 10-11 o'clock and you couldn't smell anything but ever since the hogs came in we couldn't go out there anymore . . . everybody in the neighborhood used to go to each other's houses and sit outside and enjoy it but now it's just so bad."

". . . we used to stay outside or play late at night or we would cook out at night cuz everyone could sit out there around the trees and have a fire . . . like a family we would but we can't do that anymore because of the smell."

"I used to enjoy walking back and forth down the road . . . but I just won't go outside anymore . . . the flies are so bad . . . the smell is so bad."

"It's really hard to let the kids go out and play even . . . you know if you want to go out and wash the car, sit at the picnic table and even something so simple as sitting on the porch, we just don't enjoy doing those kinds of things because we just never know when he is going to spray. Or if we do go try to enjoy those types of things and we see him pass by . . . nine times out of ten he will go turn those sprayers on . . . and that is absolutely what he does."

mentioned as the reason to buy a dryer (to dry laundry), to install or use air conditioning, and to pay to discontinue the use of well water and use bottled water or get connected to city water.

DISCUSSION

Integrating the results from the analysis of "beneficial use of property" and "quiet enjoyment of life," our research shows that hog odor limits activities of daily living that participants either "enjoyed" doing the most or expected to be able to perform inside and outside their homes. It restricts, for instance, activities like cookouts, barbequing, family reunions, socializing with neighbors, gardening, working outside, playing, drying laundry outside, opening doors and windows for fresh air and to conserve energy, use of well water, and growing vegetables. When we examine these restrictions in terms of types of activities and in the context of our area of study, which includes low-income rural communities with a high percentage of African Americans, the cumulative adverse impact goes beyond mere violation of property rights and has critical public health ramifications.

The types of activities that are restricted by hog odor are social interactions, physical activities, energy- and cost-saving activities, relaxing outside or indoors, and sleeping. Social activities have been shown to positively affect health, improve overall well-being, reduce stress, and strengthen social networks [33-35].

Furthermore, activities like gardening, working, growing vegetables, and playing outside, naturally integrate physical activity into the day-to-day living of rural residents and have enormous health benefits. Research has already shown that residents in rural communities perceive the environmental barriers as a reason for physical inactivity [36, 37]. Therefore, any moderate to severe restriction in these activities could further force the rural residents into an inactive and sedentary lifestyle. In fact, a study published in 2005 by Martin et al. [38] based on a nationwide survey about physical activity in the U.S. revealed that physical **in**activity (PIA) levels were higher in rural areas than in urban areas and that, regionally, the urban-rural differences were most striking in the South. It is estimated that physical inactivity and diet contribute to approximately one-third of all cancers [39]. These statistics are even more alarming for low-income populations and people of color who are at particularly high risk for several chronic diseases [40-41], have limited access to health care [43, 44], and limited means to seek regular treatment for their illnesses [45-47].

Current regulations and enforcement mechanisms for industrial swine operations do not adequately protect public health. Improvements could be brought about by considering their cumulative impacts on the physical, mental,

and social well-being of residents of neighboring communities. Policy recommendations published by Donham et al. in 2007 [48] could help to address these impacts. They include, among other recommendations, the issuance of permits based on the carrying capacity of the local environment and decision-making at local levels. While reiterating their policy points, we additionally recommend that:

- Requirement to include direct and indirect impacts on quality of life and activities of daily living in environmental and public health impact statements for all existing and new concentrated animal feeding operations should be required.
- For existing operations, specific timelines could be mandated for evaluating these issues and implementing necessary protections.
- Strong support for sustainable farming practices and independent farmers.

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Attachment A13

U.C.L.A. Law Review

The Clean Air Act's Blind Spot: Microclimates and Hotspot Pollution

Ann E. Carlson

ABSTRACT

The Clean Air Act (CAA) has delivered enormous pollution reductions in its almost fifty-year history, produced huge health benefits, and saved thousands of lives. Its ambient focus has led to the almost complete elimination of lead and dramatic drops in the other pollutants it regulates. Nevertheless, the CAA has a major blind spot: small "microclimates" that contain levels of deadly pollutants that can far exceed federal standards. These hotspots contain pollutants that exacerbate asthma, increase respiratory and cardiac deaths, may cause developmental problems in children, and increase cancer risks. The most prevalent of these pollution hotspots occur in predictable patterns around heavily trafficked roads and industrial facilities. Low-income communities and communities of color are much more likely to live in polluted microclimates and suffer health effects as a result.

This Article argues that the ambient focus of the CAA, which requires the monitoring and regulation of large air districts to produce background levels of pollution that meet stringent federal standards, actually masks pollution hotspots. Residents who live in air districts that receive the imprimatur of "attainment" under the CAA may nevertheless experience air quality that is considerably worse than federal air standards. Paradoxically, residents of air districts that are out of attainment, even with extreme designations, may be breathing background air that is cleaner than residents in attainment zones who are exposed to hotspot pollution. Our regulatory system helps shape our understanding of health and safety risks, in other words, in ways that are inconsistent with scientific reality. Unfortunately, using the traditional CAA mechanisms, primarily the National Ambient Air Quality Standards (NAAQS), to regulate conventional pollutants is an awkward fit for hotspot pollution. States are the primary regulators of these pollutants, yet they have little authority over the biggest causes of hotspot pollution: cars, heavy-duty trucks, and other vehicles.

Nevertheless, the Article offers suggestions for how states and the Environmental Protection Agency (EPA) may be able to address microclimate pollution using existing statutory authority. Indeed the most effective strategy for dramatically reducing hotspot pollution, electrifying the transportation fleet, also happens to be one of the most effective ways to reduce greenhouse gas emissions. Recognizing the immediate health benefits that greening the fleet would deliver could make action on climate change mitigation more palatable.

AUTHOR

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INTRODUCTION

What we see and what we can measure are often what we know and understand. In government, they are also frequently what we regulate. Consider the following. Despite huge strides in cleaning up the nation's air, children who commute to school on conventional diesel-powered school buses can face pollution levels—of black carbon, hydrocarbons, and nitrous oxides—many times higher than allowable background federal pollution standards.[1] Much of the pollution enters the inside of the buses from the tailpipe exhaust of the very vehicles in which students ride.[2] Similarly, residents of neighborhoods downwind from highways around the country—especially those with significant truck traffic—can experience highly unhealthful air quality several times during the day.[3] The time of day is not necessarily intuitive: Early morning air patterns can create air pollution plumes before many people awaken and can enter homes through open windows and vents.[4] Even the configuration of highway ramps can alter exposure amounts.[5] Residents who live near airports, as well as pedestrians, can also face elevated pollution levels. Living downwind from a small regional airport with only private plane traffic can lead to pollution levels many times higher than nearby neighborhoods outside the planes' routes.[6] Different neighborhoods within the same air basins can experience highly differential risk from exposure to air pollutants, though, again perhaps

1. *See* JOHN WARGO ET AL., ENV'T. & HUMAN HEALTH, CHILDREN'S EXPOSURE TO DIESEL EXHAUST ON SCHOOL BUSES 41 (2002), http://www.ehhi.org/reports/diesel/diesel.pdf (showing exposure levels on buses exceeded 100 ug/m3, significantly above the federal standard of 12.0 ug/m3); Lisa D. Sabin et al., *Characterizing the Range of Children's Air Pollutant Exposure During School Bus Commutes*, 15 J. EXPOSURE ANALYSIS & ENVTL. EPIDEMIOLOGY 377, 384–86 (2004). For a table of federal air pollution standards, see U.S. Environmental Protection Agency, NAAQS Table, https://www.epa.gov/criteria-air-pollutants/naaqs-table.
2. Sabin, *supra* note 1, at 382.
3. *See* Wonsik Choi et al., *Prevalence of Wide Area Impacts Downwind of Freeways Under Pre-Sunrise Stable Atmospheric Conditions*, 62 ATMOSPHERIC ENV'T 318, 326 (2012).
4. *See id.*
5. NEALSON WATKINS & RICHARD BALDAUF, EPA, NEAR-ROAD NO_2 MONITORING TECHNICAL ASSISTANCE DOCUMENT 38 (2012), http://www.epa.gov/ttnamti1/files/nearroad/NearRoadTAD.pdf.
6. Wonsik Choi et al., *Neighborhood-Scale Air Quality Impacts of Emissions From Motor Vehicles and Aircraft*, 80 ATMOSPHERIC ENV'T 310, 316 (2013).

counterintuitively, the risk is often largely due to vehicles rather than large industrial facilities.[7]

Stationary sources, however, can also cause problems for people within close distance of their emissions. Environmental regulators have long relied on engineering estimates—known as emissions factors—to measure the majority of stationary source emissions. Yet evidence about big, industrial sources, like oil refineries, shows that emissions can be orders of magnitude higher than official measures show.[8] And epidemiological evidence demonstrates that residents living downwind of coal-fired power plants face increased risk of cardiovascular mortality and disease.[9]

All of these individual assaults from pollution, from both mobile and stationary sources, can create pollution levels that exceed national air pollution standards or cause health problems. Yet, because of the way pollutants are monitored and measured under the Clean Air Act (CAA), the air district in which the bus, the neighborhood near the freeway or airport, the highway off-ramp, or the oil refinery are located may be considered compliant with national standards.

The scenarios described above share a common characteristic: The areas sampled are relatively small "microclimates" that due to geography, including the built environment and land use choices, create air pollution hotspots. Until relatively recent advances in monitoring and modeling capabilities, the existence of these air pollution hotspots was not well understood. Moreover,

7. *See* Shih Ying Chang et al., *A Modeling Framework for Characterizing Near-Road Air Pollutant Concentration at Community Scales*, 538 SCI. TOTAL ENV'T 905, 917 (2015) (showing that roughly half of near-road exposure comes from heavy duty engines); EPA, INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN—HEALTH CRITERIA, FINAL REPORT (2016) [hereinafter INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN] (showing that 60 percent of NO2 emissions come from mobile sources and that highest health risks are from near-road exposure); S. COAST AIR QUALITY MGMT. DIST., FINAL REPORT: MULTIPLE AIR TOXICS EXPOSURE STUDY IN THE SOUTH COAST AIR BASIN (MATES-III) (2008), http://www.aqmd.gov/home/air-quality/air-quality-studies/health-studies/mates-iii/mates-iii-final-report (showing that the vast majority of toxic emissions in the Los Angeles basin come from mobile sources).

8. *See* Daniel Hoyt & Loren H. Raun, *Measured and Estimated Benzene and Volatile Organic Carbon (VOC) Emissions at a Major U.S. Refinery/Chemical Plant: Comparison and Prioritization*, 65 J. AIR & WASTE MGMT. ASS'N 1020, 1026 (2015); *see also* Alex Cuclis, Hous. Advanced Research Ctr., *Why Emissions Factors Don't Work at Refineries and What to Do About It, Paper for EPA* (Aug. 13–16, 2012), https://www3.epa.gov/ttnchie1/conference/ei20/session7/acuclis.pdf.

9. *See* MORTON LIPPMANN ET AL., NATIONAL PARTICLE COMPONENT TOXICITY (NPACT) INITIATIVE: INTEGRATED EPIDEMIOLOGIC AND TOXICOLOGIC STUDIES OF THE HEALTH EFFECTS OF PARTICULATE MATTER COMPONENTS, RESEARCH REPORT 177, HEALTH EFFECTS INST. 173 (2013), https://www.healtheffects.org/system/files/RR177-Lippmann.pdf.

the health effects of exposure to many of the pollutants that contaminate microclimates, while generally known for many years, have come into much sharper focus as our capacity to understand exposure mechanisms has improved.

I argue in this Article that what we now know and can measure about hotspot air pollution has not caught up with how we regulate. Indeed, our current system of air pollution regulation—while enormously successful—may actually mask pollution hotspots, leaving those who are exposed to them unaware of their health risks and in some cases misleading regulators about their existence. The legal literature, too, has largely failed to recognize the implications of our new measurement capabilities. This Article attempts to fill that gap.

My central contention is that microclimate hotspots raise significant challenges to the Clean Air Act's central regulatory mechanism, the National Ambient Air Quality Standards (NAAQS), for at least two pollutants: fine particulate matter and nitrogen dioxide (NO2). Indeed, I argue that the current system of ambient air regulation contained in the NAAQS can mask bad air quality by providing an imprimatur of clean air for areas of the country that attain the standards even when microclimates within them are unhealthful. Paradoxically, though, using the NAAQS system to tighten the standards to address microclimate pollution creates its own problems. Allowing microclimate pollution to drive setting the NAAQS can distort the regulatory process and divert attention away from tightening diesel and other mobile source emissions regulations, the dominant culprits in creating pollution hotspots, and onto stationary sources. But the current regulatory system also allows stationary sources that emit unhealthful levels of pollutants to continue to do so, as long as overall ambient air pollution limits are met. As a result, tackling microclimate pollution may require new approaches that, while maintaining the NAAQS approach, begin to address its shortfalls.

To date, microclimate pollution has received very little direct regulatory attention under the Clean Air Act. The CAA instead revolves largely—though not exclusively—around the NAAQS system of regulating six common pollutants sufficiently stringently to create healthy background, or ambient, air quality across a large geographic swath.[10] The NAAQS are set at

10. I focus in this Article on microclimate pollution caused by the National Ambient Air Quality Standards (NAAQS) pollutants rather than on hazardous air pollutants (HAP). I do so because the regulatory system to regulate NAAQS differs substantially from the system to regulate HAPs, and thus, I argue, raises unique problems. For an interesting article analyzing data about hazardous air pollutants and arguing that, for the most

levels designed to protect public health, welfare, and the environment.[11] In order to ensure that the NAAQS are met, states and the federal government together regulate emissions from both stationary and mobile sources, with states taking principal responsibility for stationary sources[12] and the federal government and California—through a unique statutory role—leading in regulating mobile sources.[13]

Assuming the NAAQS are sufficiently stringent, attainment with them implies that background outdoor air in attainment areas is healthful.[14] Similarly, a NAAQS nonattainment designation signals that an area has unhealthful air quality. Yet the concept of "ambient" as implemented under the CAA is applied to precisely the opposite of microclimates. Instead, attainment is measured for large geographic areas—air districts designated by the Environmental Protection Agency (EPA) after consultation with individual states[15]—even though those areas may have very different air quality within their jurisdictional borders.[16] Furthermore, the quality of the

part, stationary sources do not cause serious risks of exposure at unhealthful levels, see David E. Adelman, *The Collective Origins of Toxic Air Pollution: Implications for Greenhouse Gas Trading and Toxic Hotspots*, 88 IND. L.J. 273, 277 (2013) ("Industrial sources of air toxins are geographically concentrated, but even where their emissions are the highest they rarely dominate."). For a discussion about why our monitoring system may underestimate pollutants from certain stationary sources, *see supra* note 8 and accompanying text. For the statutory text establishing the NAAQS, see 42 U.S.C. §§ 7408, 7409 (2012).

11. The Clean Air Act (CAA) treats hazardous air pollutants—designated in 42 U.S.C. § 4712 of the CAA—differently by regulating individual sources through a technology-based approach.
12. *See* 42 U.S.C. § 7410 (2012) (setting forth the State Implementation Plan process to implement national standards).
13. *See* 42 U.S.C. §§ 7521, 7543 (2012). California is given special authority to issue its own mobile sources emissions standards provided the standards are "at least as protective of public health and welfare" as the federal standards. 42 U.S.C. § 7543(b)(2). The state must receive a waiver from EPA in order to implement separate mobile source standards. For an explanation of this process and its implications for federalism, *see* Ann E. Carlson, *Iterative Federalism and Climate Change*, 103 NW. U. L. REV. 1097, 1109–28 (2009).
14. For a list of the NAAQS and an explanation of their operation, see EPA, NAAQS Table, https://www.epa.gov/criteria-air-pollutants/naaqs-table (last visited Mar. 22, 2018). The statutory provisions requiring the establishment of NAAQS are included in 42 U.S.C. § 7409 (2012).
15. 42 U.S.C. §§ 7407(b), 7407(c) (2012).
16. To be fair, the setting of the NAAQS has in recent iterations begun to take account of the risks of exposure to pollutants as a result of proximity to roadways with high vehicle intensity. In setting the most recent NAAQS for nitrogen dioxide (NO2), for example, the standard was revised to include not only an annual background standard but also to include a one-hour standard, recognizing problems of peak exposure during rush hour. *See* discussion *infra* notes 114–116.

air is designated based on a relatively small number of monitoring stations, combined with complex modeling, designed to measure that quality.[17]

The NAAQS have been extraordinarily successful in improving the quality of our ambient air, and I do not mean to diminish that accomplishment. I do, however, want to highlight how our system of NAAQS designations and our methods for measuring and modeling background air pollution raise at least two separate problems in what they signal about the relative healthful or unhealthfulness of air quality.

First, the geographic size of air districts can lead to nonattainment areas that are overinclusive in terms of their attainment designation. By relying on readings of monitoring stations in the worst part of a district, a designation of nonattainment may actually overstate how bad the air is in cleaner parts of the district.[18] The South Coast Air Quality Management District (SCAQMD) covering Southern California's notoriously dirty air is a good example. SCAQMD covers 10,743 square miles and has a population of 18 million people. Measurements from the eastern part of the district routinely show levels of pollutants significantly higher than in the western part, including in the City of Los Angeles. In 2016, for example, West Los Angeles experienced no violations of the 2008 eight-hour ozone standard, downtown Los Angeles experienced one violation, and San Bernardino experienced seventy-six

17. The requirements for district monitoring of air pollutants are contained in 40 C.F.R. § 58 (2017).

18. The NAAQS are set based on a particular measurement of a quantity of air averaged over a particular time period. In the case of carbon monoxide, for example, the primary standard (which protects human health) is divided into both a measurement averaged over eight hours (nine parts per million) and over one hour (thirty-five parts per million). Air districts that exceed the standard more than once a year are considered out of attainment. *See* EPA, NAAQS TABLES [hereinafter NAAQS TABLES], https://www.epa.gov/criteria-air-pollutants/naaqs-table [https://perma.cc/646L-AL3M] (last visited Mar. 7, 2018). For ozone, until 2015, the standard was .075 parts per million, averaged over eight hours; it has now been lowered to .070 parts per million. *See* EPA, TABLE OF HISTORICAL OZONE NATIONAL AMBIENT AIR QUALITY STANDARDS (NAAQS) [hereinafter TABLE OF HISTORICAL OZONE], https://www.epa.gov/ozone-pollution/table-historical-ozone-national-ambient-air-quality-standards-naaqs [https://perma.cc/U9E9-2F8W]. The Trump Administration has indicated that it will delay the implementation of the new .070 standard by one year. *See* Timothy Cama, *EPA Delays Obama Air Pollution Rule by One Year*, HILL, (June 6, 2017), http://origin-nyi.thehill.com/policy/energy-environment/336663-epa-delays-obama-air-pollution-rule-deadline-by-one-year?amp=1 [https://perma.cc/9JRK-H2HM]. To determine whether a designated area is out of attainment, EPA takes the annual fourth-highest daily maximum eight-hour concentration and averages it over three years. *See* NAAQS TABLES, *supra* note 18. The further out of attainment an area is, the more serious the regulatory consequences.

violations.[19] Yet the entire district is designated an extreme nonattainment zone for ozone.[20] The district boundaries make regulatory sense because of the geographic configuration of the district and air patterns that typically move pollutants from the west to the east. Nevertheless, common perceptions about how bad air quality is in the City of Los Angeles can be both incorrect and misleading.

Ironically, tightening NAAQS to address microclimate pollution or mandating that monitoring stations measure microclimates might make this overinclusiveness problem worse. Take, for example, near-roadway pollution, a phenomenon well-recognized in the scientific literature. Residents living within 300 to 500 meters of major roadways—and millions of people fall within that category—experience elevated levels of air pollutants, particularly during rush hour.[21] If air districts were to rely on monitors placed to measure this elevated pollution, many could experience readings that would tip them into nonattainment areas under the NAAQS for at least two pollutants: nitrogen dioxide and fine particulate matter. Or, if EPA tightened a NAAQS in recognition of the deleterious effects of near roadway pollution—as it has done for nitrogen dioxide by adding a new short-term exposure standard[22]—more air districts would likely be out of attainment.[23] The nonattainment designation would, in turn, trigger a series of regulatory requirements for these districts aimed at attempting to bring them back into attainment.[24] Yet the most obvious option to increase the likelihood of attainment—tightening various mobile source standards—is outside the regulatory purview of forty-nine states and all local air districts and is instead

19. Cal. Air Res. Bd., Select 8 Summary, https://www.arb.ca.gov/adam/select8/sc8display.php [https://perma.cc/PR5C-3AR9] (selecting for national 2008 eight-hour ozone standard (0.075 ppm) for West Los Angeles/VA Hospital, Los Angeles N. Main and San Bernardino 4th Street).
20. *See* EPA, CURRENT NONATTAINMENT COUNTIES FOR ALL CRITERIA POLLUTANTS, https://www3.epa.gov/airquality/greenbook/ancl.html [https://perma.cc/49K8-7YC8] (last updated Feb. 28, 2018).
21. HEALTH EFFECTS INST., TRAFFIC-RELATED AIR POLLUTION: A CRITICAL REVIEW OF THE LITERATURE ON EMISSIONS, EXPOSURE, AND HEALTH EFFECTS vii–ix (2010).
22. *See* EPA, AIR QUALITY GUIDE FOR NITROGEN DIOXIDE 2, https://www3.epa.gov/airnow/no2.pdf.
23. Environmental Protection Agency (EPA) has yet to designate nonattainment areas for the short-term exposure standard. *See* EPA, NITROGEN DIOXIDE DESIGNATIONS: REGULATORY ACTIONS, https://www.epa.gov/nitrogen-dioxide-designations/nitrogen-dioxide-designations-regulatory-actions [https://perma.cc/CR4C-XKKK] ("EPA has designated all areas of the country as unclassifiable/attainment.").
24. To be sure, the new one-hour standard for nitrogen dioxide allows measurements averaged over a three-year period rather than finding a violation for a single event. *See* EPA, NAAQS TABLES, *supra* note 18.

exclusively federal with the exception of California.[25] Moreover, most other measures that might significantly control transportation emissions—particularly those measures that interfere in any meaningful way with driving—are not mandatory under the CAA. States have not been willing to impose meaningful transportation or land use controls, and the language of the CAA comes close to actively discouraging transportation control measures. The result is that a nonattainment designation likely causes states to target stationary sources, which are not the principal cause of microclimate pollution, for more stringent pollution control.

But leaving microclimates out of the monitoring system hardly makes them go away. The way in which the NAAQS system operates creates a problem of underinclusiveness that is far more troubling than the overinclusiveness problem. Using a relatively small number of monitors to measure ambient air quality over a large geographic area, by definition, simply ignores many microclimates. Again, to use the Los Angeles basin as an example, the air district uses thirty-eight monitoring stations around its almost 11,000-mile basin to measure air quality, a number higher than required by federal regulation.[26] The stations cannot, obviously, measure the quality of the air across the potentially thousands of microclimates that exist within its borders. And, indeed, EPA regulations discourage using microclimate monitoring to establish ambient limits for a number of NAAQS pollutants, including fine particulate matter. The agency has opposed environmental group efforts to require near-road monitors to measure PM 2.5.[27]

Yet microclimates around the country have air quality significantly more unhealthful than the background measurements of ambient air suggest.

25. Even air districts within California are preempted from regulating emissions from mobile sources. The U.S. Supreme Court struck down a South Coast Air Quality Management District (SCAQMD) program that mandated that operators of certain vehicle fleets purchase alternative fuel vehicles on the grounds that the rules were preempted by Section 209(a) of the CAA. *See* Engine Mfrs. Ass'n v. S. Coast Air Quality Mgmt. Dist., 541 U.S. 246, 252–59 (2004).

26. *See* RENE M. BERMUDEZ ET AL., S. COAST AIR QUALITY MGMT. DIST., ANNUAL AIR QUALITY MONITORING NETWORK PLAN 2 (2016), http://www.aqmd.gov/docs/default-source/clean-air-plans/air-quality-monitoring-network-plan/annual-air-quality-monitoring-network-plan.pdf?sfvrsn=10. In addition to the thirty-eight multipollutant monitors, SCAQMD also has five monitoring sites specifically for stationary sources of lead. *Id.*

27. The monitoring regulations are contained in 40 C.F.R. § 52 app. D (2016). A coalition of environmental groups recently sued EPA over its failure to mandate near-road monitors in Southern California to establish ambient limits. *See* Brief for Petitioner at 1–3, Physicians for Soc. Responsibility v. EPA, No. 12-70016 (9th Cir. filed May 18, 2012) (on file with author). EPA opposed the placement of near-road monitors to measure PM 2.5. *See infra* note 118 and accompanying text.

This is true not only in areas of the country that are out of attainment for certain NAAQS pollutants but also in areas of the country considered to comply with Clean Air Act requirements. And it is true in some predictable, measurable ways. To put this more concretely, children who ride buses in central Los Angeles and in rural Connecticut both face in-cabin air pollution significantly dirtier than the outside ambient air.[28] People who live in neighborhoods downwind from freeways in Memphis, Las Vegas, Denver, and Detroit can breathe air that is on average significantly dirtier than ambient air in the worst nonattainment area in the country, Southern California.[29] And residents of communities near oil refineries and chemical plants in Houston, Texas and Torrance, California may be breathing pollutants at levels that exceed federal standards and yet are represented to be compliant.[30] Not surprisingly, these harms disproportionately affect low-income communities of color. National data show that Latinos, African Americans, and Asian/Pacific Islanders are much more likely to live near freeways than whites. Nearly half of near-freeway residents are poor or near-poor.[31] Residential communities surrounding refineries share similar demographics.[32]

The phenomenon I describe in this Article also raises a broader theoretical point, one not confined to air pollution. How we monitor and measure pollution—in water, on the ground, in the air—shapes not only how we regulate pollution but also cultural perceptions about health and safety in ways that do not necessarily comport with the best available scientific information. Our "ways of seeing" these pollution problems are shaped by scientific, technical, and legal practices that grant them a scientific authority that, in turn, shapes—and, in the case of microclimate pollution, limits—our regulatory response and public understanding of the health and safety risks we actually face.[33] By using seemingly authoritative emissions factors rather

28. *See* JOHN WARGO ET AL., ENVIRONMENT AND HUMAN HEALTH INC., CHILDREN'S EXPOSURE TO DIESEL EXHAUST ON SCHOOL BUSES 9–20 (2002), http://www.ehhi.org/reports/diesel/diesel.pdf.
29. *See Traffic-Related Health Impacts*, *supra* note 21, at 10 (summarizing results of studies showing that near-road pollution is elevated and causes health risks).
30. *See* discussion *infra* notes 98–100.
31. Tegan K. Boehmer et al., *Residential Proximity to Major Highways—United States*, 2010, CDC, (2013), https://www.cdc.gov/mmwr/preview/mmwrhtml/su6203a8.htm [https://perma.cc/C3KN-DQ6E] (last visited Mar. 7, 2018).
32. *See* EPA, FACT SHEET: FINAL PETROLEUM REFINERY SECTOR RISK AND TECHNOLOGY REVIEW AND NEW SOURCE PERFORMANCE STANDARDS 3, https://www.epa.gov/sites/production/files/2016-06/documents/2010-0682_factsheet_overview.pdf.
33. *See* William Boyd, *Ways of Seeing in Environmental Law: How Deforestation Became an Object of Climate Governance*, 37 ECOLOGY L.Q. 843, 847–50 (2010). For an account of

than actual measurements of refinery emissions—factors that may systematically understate the risk to residents in adjacent neighborhoods—regulators may simply have been unable to "see" or understand the complaints of residents who can smell the chemicals and regularly experience respiratory problems. By stamping an air district as "in attainment" with all six federal air standards, residents of the district may feel confident that the air they breathe is healthful, even when living close to a heavily trafficked freeway. And by labeling an air district an "extreme non-attainment zone," residents (and observers) may believe they are being poisoned, even when the air quality for many of them is healthier than the air in parts of attainment zones. In addition to identifying the problem of systematic hotspot air pollution and the role the Clean Air Act plays in its continuation, then, part of my aim in this project is to unmask the role that monitoring, measuring, and labeling air pollution play in our collective understanding of the quality of air we breathe.

I turn in Part I to describing the statutory scheme that establishes the NAAQS and its enormous public health accomplishments since its 1970 passage. I then describe our increasing understanding of the health problems that remain as the result of exposure to three of the NAAQS pollutants that remain ubiquitous: ozone, NO2, and fine particulate matter. I also describe the ways in which near-source exposure to the latter two pollutants are of particular concern, and the sources, both mobile and stationary, that cause this exposure. In Part II, I describe the monitoring system EPA requires of states, including the scale of the area to be monitored for the pollutants that cause near-source health risks. Part II also includes a discussion of three ways in which EPA is attempting to address hotspot pollution under its existing authority. In Part III, I turn to a more systematic discussion of the significant shortcomings of the NAAQS for addressing hotspot pollution despite EPA's efforts to address it. In Part IV, I describe ways in which EPA programs that are not necessarily designed to address near-source exposure nevertheless can produce significant reductions in the pollutants that cause it. In fact the single most effective way to reduce near road exposure is also one of the central means to reduce greenhouse gas emissions—cleaning up the transportation fleet with a push toward vehicle electrification. As a result, the push for climate change mitigation in the transportation sector has immediate and important health

how changes in our ability to detect toxins in the environment led to a major shift in our "ways of seeing" chemical hazards in the water, on the land, in our food, and in our workplaces, see William Boyd, *Genealogies of Risk: Searching for Safety, 1930s–1970s*, 39 ECOLOGY L.Q. 895, 944–77 (2012).

co-benefits that could make such regulation more politically palatable. Since that is a multi-decadal strategy, however, I also evaluate additional ways to curtail hotspots in the shorter term, including through transportation measures and land use controls. I conclude by suggesting that Clean Air Act reform may be necessary to address hotspot pollution meaningfully in the shorter and medium term.

I. SETTING NAAQS, EVALUATING THE PUBLIC HEALTH EFFECTS OF POLLUTION EXPOSURE

A. The NAAQS Statutory Framework

The central—though not only—means for cleaning up the nation's air has been through the National Ambient Air Quality Standards.[34] Although familiar to many, a recitation of the ways in which the NAAQS system operates helps set the stage for understanding how the system can both produce dramatically cleaner air and mask the deleterious health effects of certain pollutants that concentrate in small geographic areas.

The Environmental Protection Agency sets NAAQS for pollutants that "endanger public health and welfare" and that are emitted by numerous types of sources.[35] To date, six pollutants have been designated as NAAQS pollutants: lead, carbon monoxide, nitrogen dioxide, ozone, sulfur dioxide, and particulate matter, which is further broken down into particulate matter, known as PM 10 (for particles that are between 10 and 2.5 microns in size), and fine particulate matter, known as PM 2.5 (for particles smaller than 2.5 microns).[36]

The process for designating, and then updating, a pollutant regulated under the NAAQS provisions is set forth in Sections 108 and 109 of the CAA. In addition to the requirements that a pollutant "endanger public health and welfare" and come from ubiquitous sources, Section 108 requires the administrator to issue criteria documents that assess the latest scientific evidence about the health and welfare effects of exposure to the pollutant.[37] Section 109 requires that the EPA Administrator appoint an independent scientific review committee to assist in the promulgation of NAAQS and that

34. 42 U.S.C. §§ 7408, 7409 (2012).
35. *Id.* § 7408(a)(1)(A)–(B).
36. *See Criteria Air Pollutants*, EPA, https://www.epa.gov/criteria-air-pollutants [https://perma.cc/BG4P-VZJU] (last visited April 14, 2018).
37. 42 U.S.C. § 7408(a)(2).

the NAAQS be reevaluated and revised every five years with the assistance of the committee.[38] Based on the evaluation of the scientific data about the health and welfare effects of pollution exposure, NAAQS "shall be ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."[39]

Ambient air standards apply to geographic units known as air quality control regions (AQCRs). EPA first designated a number of AQCRs in the 1960s, as the federal government began to expand its role in regulating air pollution; Section 107 of the CAA recognizes the pre–1970 AQCRs and provides authority to EPA to designate air regions for purposes of compliance with the NAAQS.[40] As an early EPA document about the designations explains, the regions were to be based on "jurisdictional boundaries, urban-industrial concentrations, and other factors, including atmospheric areas, necessary to provide adequate implementation of air quality standards."[41] States have the primary responsibility for "assuring air quality within the entire geographic area comprising such State" while EPA is responsible for determining whether air districts comply with the NAAQS.[42] The EPA Administrator designates districts as either attainment, nonattainment, or unclassifiable depending on whether they meet the standard, do not meet the standard, or lack the requisite information to determine attainment status.[43]

B. Air Pollution Accomplishments

Since the first NAAQS were promulgated shortly after the passage of the modern CAA in 1970, the progress the United States has made in cleaning up the air is remarkable. Despite the fact that systematic evidence of hotspot pollution exists, these accomplishments deserve highlighting. Over the last forty-five years, emissions of the six pollutants regulated under the NAAQS program have dropped by close to 70 percent.[44] EPA estimates that the 1990 amendments to the Clean Air Act will have saved more than 230,000 lives by

38. *Id.* § 7409(d)(1)–(2).
39. *Id.* § 7409(b)(1).
40. *Id.* § 7407.
41. EPA, FEDERAL AIR QUALITY CONTROL REGIONS 1 (1972), https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P10054HI.TXT.
42. § 7407(a)–(b), (d)(1)(B) (2012).
43. § 7407(c)–(d) (2012).
44. *See* EPA, OVERVIEW OF THE CLEAN AIR ACT AND AIR POLLUTION, http://www.epa.gov/air/caa/progress.html#breathe [https://perma.cc/KSY3-4ELZ].

2020, reduced 2.4 million cases of asthma exacerbation, and prevented 17 million sick days.[45] For some pollutants, including lead and sulfur dioxide, only a handful of counties across the country are out of attainment with the standard.[46] Lead has been almost completely eliminated as an air pollutant, with a 98 percent reduction in airborne lead between 1980 and 2014.[47] And for carbon monoxide, the entire country has attained the NAAQS.[48] These accomplishments are all the more impressive given U.S. economic[49] and population growth[50] during that time period.

C. Remaining Health Effects From Pollution Exposure

Significant evidence has mounted over the years, however, that three NAAQS pollutants—PM 2.5, ozone, and nitrogen dioxide (NO2)—continue to cause persistent and ubiquitous health problems and increases in morbidity.[51] Two of those pollutants, fine particulate matter and NO2, can be

45. *See* EPA, BENEFITS AND COSTS OF THE CLEAN AIR ACT, 1990–2020, THE SECOND PROSPECTIVE STUDY, https://www.epa.gov/clean-air-act-overview/benefits-and-costs-clean-air-act-1990-2020-second-prospective-study [https://perma.cc/54LE-MGKS].

46. *See* EPA, LEAD (2008) DESIGNATED AREA/STATE INFORMATION [hereinafter LEAD 2008 STANDARD NONATTAINMENT AREAS], https://www3.epa.gov/airquality/greenbook/mbtc.html (last updated Feb. 28, 2018); EPA, SULFUR DIOXIDE (2010) DESIGNATED AREA/STATE INFORMATION [hereinafter SULFUR DIOXIDE 2010 STANDARD NONATTAINMENT], https://www3.epa.gov/airquality/greenbook/tbtc.html (last updated Feb. 28, 2018). As the 2008 Sulfur Dioxide Integrated Science Assessment explained, "[n]o monitored exceedance of the SO_2 annual ambient air quality standard in the lower 48 States of the U.S. has been recorded between 2000 and 2005, according to the EPA Acid Rain Program (ARP) 2005 Progress Report." EPA, INTEGRATED SCIENCE ASSESSMENT FOR SULFUR OXIDES—HEALTH CRITERIA 2–23 (2008) [hereinafter INTEGRATED SCIENCE ASSESSMENT FOR SULFUR OXIDES].

47. EPA, BASIC INFORMATION ABOUT LEAD AIR POLLUTION, https://www.epa.gov/lead-air-pollution/basic-information-about-lead-air-pollution [https://perma.cc/TL2R-ZRFX] (last visited Mar. 8, 2018).

48. *See* EPA, CARBON MONOXIDE (1971), DESIGNATED AREA/STATE INFORMATION, https://www3.epa.gov/airquality/greenbook/cbtc.html (last updated Feb. 28, 2018).

49. The U.S. economy has grown by 80 percent since 1970 as measured by adjusted GDP. *See* DATA 360, GDP-REAL (ADJUSTED) UNITED STATES, http://www.data360.org/dataset.aspx?Data_Set_Id=354 [https://perma.cc/9FTR-UDZV].

50. The U.S. population grew from 205 million in 1970 to 316 million in 2013. *See* U.S. POPULATION BY YEAR, http://www.multpl.com/united-states-population/table [https://perma.cc/C3XL-RPAY].

51. This is not to suggest that the other three NAAQS pollutants cause no health problems, but rather that their airborne levels have dropped dramatically enough that exposure levels are causing less concern than for the remaining three. *See, e.g.*, INTEGRATED SCIENCE ASSESSMENT FOR SULFUR OXIDES, *supra* note 46, at 2–53 ("Because SO2 concentrations have declined markedly over the past few decades, relatively few recent personal exposure studies have focused on SO2.").

elevated near the sources from which they are emitted and can cause significant health problems for those breathing in the pollutants. Ozone, by contrast, can be elevated in areas away from the primary sources, and thus the system of NAAQS regulation is better suited for its control. Nevertheless, nitrogen dioxide (along with other NOx and volatile organic compounds) contributes to the formation of ground-level ozone so that better control of NO2 will lead to lower ozone levels.[52] And two NAAQS—nitrogen dioxide and sulfur dioxide—can transform in the atmosphere into PM 2.5.[53] The ongoing control of all four of these NAAQS, then, is key to reducing the ongoing negative health effects of air pollution.

Since the passage of the contemporary Clean Air Act in 1970, our scientific understanding of air pollution has significantly improved in two important ways relevant to my analysis. First, we have much better data about the specific health effects of particular pollutants at particular exposure levels. Second, we can measure individual exposure to pollutants in much smaller spaces more precisely. Our regulatory structure to control air pollution, however, has not caught up to these improvements in scientific knowledge.

A number of long-term studies have found a strong relationship between exposure to particular pollutants and increased risk of mortality, and all show that the risk of mortality increases with higher levels of exposure to the particular pollutant.[54] Perhaps the most striking new finding about the

52. EPA, OZONE POLLUTION [hereinafter OZONE POLLUTION], https://www.epa.gov/ozone-pollution [https://perma.cc/JT8J-GWXN] (last visited Mar. 8, 2018); EPA, NITROGEN DIOXIDE BASICS [hereinafter NITROGEN DIOXIDE BASICS], https://www.epa.gov/no2-pollution/basic-information-about-no2 [https://perma.cc/NLN5-K27X] (last visited Mar. 8, 2017). NO2 is one of seven compounds that make up NOx and the only one of the seven that is separately regulated from ozone. NO2 is the most prevalent of the seven NOx compounds. *See* EPA, TECHNICAL BULLETIN, NITROGEN OXIDES (NOX): WHY AND HOW THEY ARE CONTROLLED 1 (1999).

53. EPA, INTEGRATED SCIENCE ASSESSMENT FOR PARTICULATE MATTER 1–4 (2009) [hereinafter ASSESSMENT FOR PARTICULATE MATTER]. *See* INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN, *supra* note 7, at lxxiii.

54. *See* Johanna Lepeule et al., *Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study From 1974 to 2009*, 120 ENVTL. HEALTH PERSP. 965, 967 (2012) (showing that exposure to fine particulate matter increases the risk of mortality from cardiac disease and lung cancer and that "[e]ach 10-$\mu g/m^3$ increase in PM 2.5 was associated with a 14% increased risk of all-cause death . . . a 26% increase in cardio-vascular death . . . and a 37% increase in lung-cancer death"); Michelle C. Turner et al., *Long Term Ozone Exposure and Mortality in a Large Prospective Study*, 193 AM. J. RESPIRATORY & CRITICAL CARE MED. 1134, 1139 (2016) ("We observed significant positive associations between long-term O_3 and all-cause, circulatory, and respiratory mortality with 2% , 3%, and 12% increases in risk per 10 ppb, respectively, in this large-scale study . . . ."); Annunziata Faustini et al., *Nitrogen*

health effects of air pollution is that populations exposed to two of the most ubiquitous pollutants—ozone and PM 2.5—face increased risk of death even at rates lower than the current stringent federal standards. In a remarkably comprehensive study, researchers looked at 61 million Medicare enrollees from across the country, including in less populated (and less frequently studied) parts of the country.[55] They broke the study group down by 1 x 1 km geographic areas. For every increase in average annual exposure of 10 micrograms of PM 2.5 per cubic meter, the mortality rate among Medicare enrollees increased 7.3 percent. Increases of 10 parts per billion of ozone caused less significant, but still real, increases in mortality rates. Surprisingly, for people exposed to levels below the fine particulate matter federal standard, the increase in mortality rate was even larger: For every increase of 10 micrograms per cubic meter of PM 2.5, mortality increased by 13.6 percent.[56]

We also know that exposure to specific air pollutants causes particular health effects in addition to increased risk of death. In the Environmental Protection Agency's most recent PM 2.5 Integrated Scientific Assessment—which comprehensively reviews and assesses the evidence of the health effects of pollutant exposure—the agency concluded that short-term exposure to PM 2.5 causes cardiovascular effects, likely causes respiratory ailments, and causes premature mortality. Long-term exposure has the same effects and even more, with evidence suggesting that exposure is related to reproductive harm, developmental problems in children, and cancer.[57]

Dioxide and Mortality: Review and Meta-Analysis of Long-Term Studies, 44 EUR. RESPIRATORY J. 744, 751 (2014) (showing that exposure to nitrogen dioxide increases overall cardiovascular and respiratory mortality, explaining how the results hold independent of multiple pollutant exposure, including fine particulates, and concluding that "NO_2 effects ranging from 3% to 36% per 10 μgm^{-3} have been reported for cardiovascular mortality in European cohorts as well as an effect of 12% for respiratory mortality").

55. *See generally* Qian Di et al., *Air Pollution and Mortality in the Medicare Population*, 26 NEW ENG. J. MED. 2513 (2017).

56. *Id.* at 2513.

57. *See* ASSESSMENT FOR PARTICULATE MATTER, *supra* note 53, at 2–9. EPA conducts a comprehensive Integrated Science Assessment every time it reviews whether to strengthen a NAAQS. For a description of the process, see EPA, LEARN ABOUT THE ISAS, https://www.epa.gov/isa/learn-about-isas (last visited Mar. 8, 2018). The team conducts an extensive review of existing evidence about the health effects of the pollutant being reviewed and then characterizes the strength of the evidence about particular effects. For some health effects, for example, EPA concludes that there is evidence "sufficient to conclude there is a causal relationship" between exposure to the pollutant and the particular health effect. For others, EPA might find that the evidence is "suggestive of a causal relationship" but not sufficient to infer one. *See* ASSESSMENT FOR PARTICULATE MATTER, *supra* note 53, at 1–21. *See also* Lepeule, *supra* note 54, at 965 (showing that

The improvement in our understanding of the health effects of exposure to fine particulate matter since the 1970 passage of the CAA is quite remarkable. The earliest federal standard regulated only "Total Suspended Particles" (TSP) without regard to particle size. As of 1982, when EPA prepared air quality criteria documents for TSP, the agency concluded that "essentially no epidemiological studies" provided sufficient data to connect "respiratory disease or other types of mortality to chronic (annual average) exposures to PM . . ."[58] The same document did acknowledge that health effects "might most reasonably and directly [be] attributed to fine- and small coarse-mode particles" but also found very little evidence of health effects for exposure even at levels that far exceed what we now know to be highly unhealthful.[59] It was not until 1997 that EPA established a separate NAAQS for fine particulate matter. The new fine particulate standard was set because data from six cities indicated that "[f]ine particles (PM 2.5) showed a consistent and statistically significant relationship to acute mortality" with risks increasing as levels of fine particulates increased.[60] By 2012, the standard had been tightened twice, with the most recent standard set based on the evidence described above of substantial health risks at exposure levels far below what was previously understood.[61]

Our knowledge of the health effects of ozone exposure has increased dramatically as well. We now know that short-term ozone exposure causes respiratory effects and likely causes cardiovascular effects and increases in morbidity, according to EPA's 2013 Integrated Scientific Assessment.[62] Evidence suggests that short-term ozone exposure causes central nervous system effects as well. Additionally, researchers have found associations between short-term exposure and increased hospital visits and increased allergic and asthma-related responses.[63] Epidemiological evidence also exists

exposure to fine particulate matter increases the risk of mortality from cardiac disease and lung cancer).

58. *See* EPA, AIR QUALITY CRITERIA FOR PARTICULATE MATTER AND SULFUR OXIDES 1-97 (Dec. 1982).
59. *Id.* at 1-92, 1-99, 1-103.
60. *See* EPA, AIR QUALITY CRITERIA FOR PARTICULATE MATTER 1-13 (1996).
61. *See* EPA, PARTICULATE MATTER (PM STANDARDS)—TABLE OF HISTORICAL PM NAAQS, https://www3.epa.gov/ttn/naaqs/standards/pm/s_pm_history.html [https://perma.cc/J8CY-VF34] (last visited Mar. 7, 2018).
62. *See* EPA, INTEGRATED SCIENCE ASSESSMENT FOR OZONE AND RELATED PHOTOCHEMICAL OXIDANTS, at 1-5, 2-20 to 2-24 (Feb. 2013) [hereinafter ASSESSMENT FOR OZONE AND RELATED PHOTOCHEMICAL OXIDANTS], https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=247492; *see also* Turner et al., supra note 54, at 1134, 1139., 193 AM. J. RESPIRATORY & CRITICAL CARE MED. 1134, 1139 (2016).
63. *Id.*

showing that short-term exposure is associated with pulmonary inflammation and lung host defenses. Long-term exposure to ozone is connected to similar health outcomes, though the evidence for causation of many of the health outcomes is somewhat weaker for longer term exposure as opposed to shorter term.[64] Health effects from ozone exposure appear to be worse for children, older adults, outdoor workers, and individuals with asthma.[65] Some evidence—still suggestive—exists showing that individuals who are obese and those of lower socioeconomic status may also suffer worse health outcomes.[66] Women, Latinos, and African Americans may as well, though more study is needed.[67]

The sum total of the scientific knowledge of the health effects of ozone, in 1971, by contrast, was described in the Federal Register notice announcing the final adoption of the first standards: "The revised national primary standard . . . is based on evidence of increased frequency of asthma attacks in some asthmatic subjects on days when estimated hourly average concentrations of photochemical oxidant reached . . . 0.10 ppm."[68]

Exposure to NO2 also produces negative health outcomes. In EPA's most recent Integrated Scientific Assessment of Oxides of Nitrogen, the agency concluded that short-term exposure to NO2 likely causes respiratory ailments and may cause cardiovascular disease. Long-term exposure likely causes respiratory disease and may cause increases in cancer, low birth weight, and cardiovascular effects.[69] Vulnerable populations include children, the elderly, and people with asthma.[70]

64. *See id.* at 1-5.
65. *See id.* at 1-8, 8-11 to -15, 8-18 to -23.
66. *See id.* at 8-26 to -27, 8-30 to -31.
67. *See id.* at 8-24 to -26, 8-28 to -29.
68. National Primary and Secondary Ambient Air Quality Standards, 36 Fed. Reg. 8186 (Apr. 30, 1971) [hereinafter Ambient Air Quality Standards]; *see also* Faustini et al., *supra* note 54, at 751 (exposure to nitrogen dioxide increases overall cardiovascular and respiratory mortality and explaining how the results hold independent of multiple pollutant exposure, including fine particulates). In its most recent Integrated Science Assessment of Oxides of Nitrogen, EPA concluded that evidence of exposure to NO2 is suggestive of, but insufficient to establish definitively, a relationship between short- and long-term exposure and mortality. *See* INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN, *supra* note 7, at lxxxii.
69. For a chart describing EPA's conclusions about the relationship between NO2 exposure and health effects, see INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN, *supra* note 7, at lxxxii.
70. *See* EPA, REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY STANDARDS FOR NITROGEN DIOXIDE: RISK AND EXPOSURE ASSESSMENT AND PLANNING DOCUMENT 1–8 to 8–9 (May 2015) [hereinafter REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY

Again in contrast to the most recent EPA assessment, in 1971 EPA eliminated a short-term standard for NO2 (while retaining an annual standard) on the grounds that "[n]o adverse effects on public health or welfare have been associated with short-term exposure to nitrogen dioxide at levels which have been observed to occur in the ambient air."[71] In 2013, in a complete turnaround, EPA added to its annual standard a new short-term exposure standard based on the scientific information demonstrating respiratory and other health effects from near-source exposure.[72]

II. EVIDENCE OF NEAR-SOURCE EXPOSURE

A. Mobile Sources

Not only do we have a much better understanding of the negative health effects of exposure to ozone, PM 2.5, and NO2, we also know significantly more about where and how human exposure occurs. For some pollutants—ozone being the most important—exposure is not tied to living or working near the sources of ozone pollutants. Ozone is a secondary pollutant, one that occurs as the result of the emission of "ozone precursors"—volatile organic compounds (VOCs) and nitrogen oxides (NOx)—that chemically react with sunlight to cause ground level-ozone.[73] Once ozone is formed, those who experience the highest levels of ozone exposure are downwind from the sources, sometimes at significant distances. They are typically suburban and rural residents who live downwind of urban, industrialized areas.[74]

For particulate matter and NO2 emissions, however, near-source exposure matters a great deal. And significant evidence exists that one source in particular, traffic on major roads and highways, is the largest culprit of near-source pollution.

In a 2010 comprehensive review of studies of exposure to traffic-related air pollution, a high-level panel tasked with evaluating the literature concluded that those living within 300 to 500 meters of a major roadway face

STANDARDS FOR NITROGEN DIOXIDE] (reviewing Integrated Science Assessment evidence of health effects of short-term nitrogen dioxide exposure).

71. *See* Ambient Air Quality Standards, *supra* note 68, at 8186.

72. *See* REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY STANDARDS FOR NITROGEN DIOXIDE, *supra* note 70, at 1-3.

73. *See* ASSESSMENT FOR OZONE AND RELATED PHOTOCHEMICAL OXIDANTS, *supra* note 62, at 1-2.

74. *See id.*

the highest health risks from traffic emissions.[75] There is significant evidence, the panel concluded, that near-road exposure exacerbates asthma and suggestive evidence that living near a major roadway causes asthma, increases respiratory symptoms that are non-asthma related, impairs lung function, increases cardiovascular mortality, and increases overall mortality from all causes.[76]

The most recent Integrated Science Assessment for NO2 actually labels NO2 a "traffic-related pollutant," noting that "recent information shows that motor vehicle emissions are the largest single source of NO_2 in the air and that NO_2 concentrations tend to be variable within communities, decreasing with increasing distance from roads."[77] NO2 concentrations also vary depending, unsurprisingly, on traffic conditions, with rush hour traffic, areas of traffic delay, and so forth exhibiting higher emissions than areas with freer flowing traffic.[78]

Near-road PM 2.5 exposure also causes real and significant health effects. Traffic combustion generates significant particulate matter, especially fine and ultrafine particles, and "particulates generated from combustion processes, especially diesel exhaust particulates (DEP), are more potent in posing adverse health effects than those from non-combustion processes."[79] A recent study of two roadways in the United States showed that heavy-duty diesel contributes more than half of fine particulate matter and NOx (including NO2) and thatconcentrations of these pollutants drop dramatically 200 meters away from the roads. Light-duty vehicles are a significant source of other pollutants, including benzene.[80]

More than 11 million Americans live within 150 meters of a major highway.[81] Though this is just under 4 percent of the U.S. population, in urban areas the percentage rises dramatically. In Los Angeles, for example,

75. *See* HEALTH EFFECTS INST., *supra* note 21, at 5.
76. *See* HEALTH EFFECTS INST., *supra* note 21, at 10.
77. INTEGRATED SCIENCE ASSESSMENT FOR OXIDES OF NITROGEN, *supra* note 7, at lxxxvii. *See also* Evelyn S. Kimbrough et al., *Seasonal and Diurnal Analysis of NO2 Concentrations From a Long-Duration Study Conducted in Las Vegas, Nevada*, 63 J. AIR & WASTE MGMT. ASS'N 934 (2013) (showing higher concentrations closer to highways).
78. *See, e.g.*, Luther Smith et al., *Near-Road Measurements for Nitrogen Dioxide and its Association with Traffic Exposure Zones*, 6 ATMOSPHERIC POLLUTION RES. 1082 (2015); Shaibal Mukerjee et al., *Comparison of Modeled Traffic Exposure Zones Using On-Road Air Pollution Measurements*, 6 ATMOSPHERIC POLLUTION RES. 82 (2015) (showing highest exposure levels for heavily trafficked areas as opposed to traffic signals and bus routes).
79. Xianglu Han & Luke P. Naeher, *A Review of Traffic-Related Air Pollution Exposure Assessment Studies in the Developing World*, 32 ENV'T INT'L 106, 108 (2006).
80. *See generally* Shih Ying Chang et al., *supra* note 5.
81. Boehmer et al., *supra* note 31, at 46–50.

more than a third of the population lives within 300 meters of a freeway or major road.[82] Of the 11 million Americans living within 150 meters of a major highway, about a quarter are under the age of 18 and just under half are nonwhite.[83] Latinos, African Americans, and Asian/Pacific Islanders are much more likely to live next to a highway than whites.[84] Close to half of near-highway residents are poor or near-poor.[85]

Exposure to higher levels of NO2 and particulate matter is not limited to residents who live near roads. Children riding on diesel school buses face some of the most alarming exposure rates to carcinogenic particulate matter. In a study of Connecticut school buses, researchers found some buses with fine particulate measurements five to fifteen times higher than ambient levels outside.[86] California researchers found similarly alarming levels inside Los Angeles-area buses.[87] Occupants of vehicles in heavily trafficked areas also experience elevated levels of pollutants. Levels of PM can be elevated by as much as 40 percent for PM 10 and by 16 percent to 17 percent for fine particulate matter at trafficked intersections.[88] Use of the ventilation system in a car, however, can reduce exposure significantly.[89]

Mobile sources also play a major role in making ports one of the most significant contributors to air pollution and one of the most polluted microclimates. EPA estimates that about 39 million people live in close proximity to a port and acknowledges that diesel emissions from drayage, longer-trip truck traffic, ocean-going vessels, and so forth produce serious health risks for residents living in close proximity.[90] In Los Angeles, emissions from the Los Angeles and Long Beach ports constitute the single largest air pollution source in the region.[91]

82. *See* HEALTH EFFECTS INST., *supra* note 21, at 17.
83. BOEHMER ET AL., *supra* note 31, at 48 tbl.
84. *Id.*
85. *Id.*
86. WARGO, *supra* note 1, at 5.
87. Lisa D. Sabin et al., *Characterizing the Range of Children's Air Pollutant Exposure During School Bus Commutes*, 15 J. EXPOSURE ANALYSIS & ENVTL. EPIDEMIOLOGY 377 (2005).
88. *See* Prashant Kumar & Anju Goel, *Concentration Dynamics of Coarse and Fine Particulate Matter at and Around Signalised Traffic Intersections*, 18 ENVTL. SCI. PROCESSES & IMPACTS 1220, 1234 (2016).
89. *See* N. Hudda & S.A. Fruin, *Models for Predicting the Ratio of Particulate Pollutant Concentrations Inside Vehicles to Roadways*, 47 ENVTL. SCI. & TECH. 11,048, 11,054 (2013).
90. EPA, NATIONAL PORT STRATEGY ASSESSMENT: REDUCING AIR POLLUTION AND GREENHOUSE GASES AT U.S. PORTS 1, https://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=P100PGK9.pdf.
91. *See* S. COAST AIR QUALITY MGMT. DISTRICT, CLEAN PORT, http://www.aqmd.gov/home/about/initiatives/clean-port [https://perma.cc/S3X9-U999] (last visited Dec. 25, 2017).

B. Stationary Sources

There is a surprising dearth of data about near-site exposure to emissions of NAAQS pollutants from stationary sources like power plants, refineries, and chemical plants. Somewhat more data exists for toxic emissions from large stationary sources, but those tend to analyze exposure at the census tract-level, where researchers acknowledge that the size of the census tract is not small enough to provide precise estimates of exposure for those closest to the source.[92]

One major concern about emissions from refineries and chemical plants—which emit some of the most toxic pollutants along with conventional NAAQS pollutants—is that there may be systematic underreporting errors in emissions measurements based on measuring techniques the plants use with approval from EPA. In a recent evaluation of emissions in the Houston Shipping Channel, home to one of the largest concentrations of chemical and petrochemical refineries in the world, researchers used testing devices that measured emissions of VOCs and benzene at levels far higher than the estimates produced and reported by the facilities themselves.[93] VOCs emissions were 41 percent higher than emissions inventories reported, and benzene emissions were 94 percent higher.[94] Preliminary results from real-time monitoring of refineries and other sources in the Los Angeles area show similar outcomes, with VOCs emissions three to twelve times higher than emissions inventories report.[95]

92. *See* Adelman, *supra* note 10, at 300 (noting that urban census tracts are on average about two square miles, while rural tracts are much larger). In an extensive analysis of data about toxic releases from stationary sources, culled from EPA data, Adelman concludes that less than 10 percent of toxic air emissions come from large industrial sources, with the exception of Texas, given its high concentration of refineries and chemical facilities. Even in the census tracts packed with large industrial sources, toxic emissions from these facilities comprise just over a quarter of total air toxics emissions and only about 10 percent of the excess cancer risk. *Id.* at 277.

93. Daniel Hoyt & Loren H. Raun, *Measured and Estimated Benzene and Volatile Organic Carbon (VOC) Emissions at a Major U.S. Refinery/Chemical Plant: Comparison and Prioritization*, 65 J AIR & WASTE MGMT. ASS'N 1020, 1021 (2015); see also Cuclis, supra note 8., (Aug. 15, 2012), https://www3.epa.gov/ttnchie1/conference/ei20/session7/acuclis.pdf.

94. Hoyt, *supra* note 93, at 1029.

95. Johan Mellqvist et al., *Quantification of Gas Emissions From Refineries, Gas Stations, Oil Wells and Agriculture Using Optical Solar Occultation Flux and Tracer Correlation Methods*, Presentation for American Geophysical Union Annual Conference Session (Dec. 12, 2016), https://agu.confex.com/agu/fm16/meetingapp.cgi/Paper/180782, *reported in* Emily Guerin, *Refineries in LA Emit Up to 12 Times More Toxic Chemicals*

III. WHY THE NAAQS ARE NOT WELL-DESIGNED TO TACKLE NEAR-SOURCE POLLUTION

A. The Concept of Ambient Air Pollution

The NAAQS system is almost by definition designed not to address near-source pollution. The NAAQS measurements are based on averages, culled from monitoring and models, of the ambient air quality across large geographic areas. They do not, nor are they meant to, measure air quality in small, geographically confined areas.

EPA requires states to monitor background levels of pollution, determine whether those levels are consistent with the NAAQS that has been set, and then issue plans to either stay in attainment with the pollution standard or come into compliance with it.[96] The idea is not to limit any particular individual source to a specific amount of pollution but instead to regulate all sources of a particular NAAQS pollutant within an air basin to a level sufficient to produce attainment with the NAAQS.[97] As a result, EPA requires monitors to be placed in a location that will measure pollutants at an appropriate scale to measure ambient pollution.[98] The number of required monitors provides a good illustration of the ambient nature of the measurements: For metropolitan areas with a population of greater than 10 million, four monitors are required to measure ozone, while three monitors

Than Reported, KPCC (Dec. 29, 2016), http://www.scpr.org/news/2016/12/29/67663/la-area-refineries-emit-up-to-12-times-more-toxic.

96. The monitoring regulations are contained in 40 C.F.R. § 58 (2017). Appendix D provides details about the numbers of monitors required by population for individual pollutants, the types of monitors required, and the ways in which monitoring should proceed. Section 107(a) requires that each state submit "an implementation plan for such State which will specify the manner in which national primary and secondary ambient air quality standards will be achieved and maintained within each air quality control region in such State." 42 U.S.C. § 7407(a) (2012).

97. 42 U.S.C. § 7410(a) requires states to demonstrate how they will either maintain or come into compliance with a NAAQS without requiring any individual source to meet a specified emissions limit. The Prevention of Significant Deterioration provisions require new and modified sources to obtain a permit designed to demonstrate that any emissions from their facilities will not throw the air district in which they are operating out of attainment, rather than to control a set amount of emissions. *See* EPA, PREVENTION OF SIGNIFICANT DETERIORATION BASIC INFORMATION, https://www.epa.gov/nsr/prevention-significant-deterioration-basic-information (last visited Mar. 7, 2018).

98. *See* 40 C.F.R. § 58 app. D (setting forth a number of monitors and placement requirements for various pollutants).

are required to measure PM 2.5 in cities with a population of more than a million.[99]

Although the NAAQS system is designed to measure ambient air in large geographical areas, EPA is not insensitive to the issues posed by microclimate pollution and, as the science has become stronger, has taken several steps to try to address it. But the statutory scheme under which the agency regulates was not designed to simultaneously target ambient air pollution caused by a large number of sources and microclimate pollution that causes near-source harm. Moreover, different NAAQS pollutants pose different measurement and monitoring challenges depending on their primary sources.

B. EPA Measures to Address Near-Source Pollution

Despite the limitations of the NAAQS, EPA has attempted to use three separate regulatory mechanisms in order to address near-source pollution. It has redefined the concept of "ambient" by using different background scales for different pollutants, including a "micro" scale. It has established a new NO2 standard directly addressed to near-source pollution. And it has modified some emissions factors for refineries based on evidence that the factors were underestimating certain pollutant emissions. Beginning in 2018, refineries will be required to monitor certain pollutants at their fence-lines.

1. Definition of Scale for Monitoring Purposes

The first mechanism EPA uses to attempt to address microclimate pollution is to require monitoring based on different scales depending on the NAAQS pollutant being addressed.[100] The monitoring regulations set forth

99. *See* 40 C.F.R. § 58 app. D, tbls. D-1 & D-5.

100. I assume in this paper that air districts place their monitors in a manner that is not only consistent with EPA regulations but that is also designed to measure accurately the ambient pollution levels necessary to establish compliance with the NAAQS. My assumption may be too sanguine. In a forthcoming paper, Corbett Grainger and coauthors evaluate whether air pollution regulators in air districts that are in attainment but only marginally place new monitors designed to measure NO2 and Ozone strategically to avoid kicking their districts into nonattainment. Using remote-sensing and other data to measure actual background levels of the pollutants, Grainger compares the data with collected monitoring data. The authors conclude that these air districts appear to be systematically understating background pollution levels. *See generally* Corbett Grainger, Andrew Schreiber & Wonjun Chaing, *Do Regulators Strategically Avoid Pollution Hot Spots When Siting Monitors? Evidence from Remote Sensing of Monitors* (Oct. 2017) (draft on file with author).

different spatial scales that are designed to be used with different pollutants. There are six scales ranging from smallest (microscale) to largest (national and global scales).[101] The regulations explain that "[p]roper siting of a monitor requires specification of the monitoring objective, the types of sites necessary to meet the objective, and then the desired spatial scale of representativeness."[102] States should use neighborhood, urban, and regional spatial scales to measure ozone, for example, given that it is a secondary pollutant that does not cause near-exposure problems.[103] In recognition of the health consequences of NO2, by contrast, EPA now requires the placement of one microscale monitoring station near roads in metropolitan areas with a population of greater than 1 million and two for populations greater than 2.5 million.[104] NO2 monitoring is not only done at the microscale level, however, but also requires monitoring at the middle and neighborhood scales.[105]

The choice of the appropriate scale for monitoring PM 2.5 is more complex and also demonstrates one of the reasons why the NAAQS system presents an awkward fit to address microclimate pollution.[106] While it is true that near-road exposure to PM 2.5 causes many health problems, PM 2.5 exposure is also a regional pollutant and shows less spatial variability than a pollutant like NO2.[107] The composition of PM 2.5 can vary dramatically

101. 40 C.F.R. § 58 app. D at § 1.2.
102. *Id.* § 1.2(c).
103. *Id.* § 4.1(c).
104. *Id.* § 4.3.2(a). Microscale monitoring is also required for carbon monoxide and sulfur dioxide to accompany the NO2 microscale monitoring. *See* 40 C.F.R. § 58 app. D at §§ 4.4.4(a), 4.2.3(a).
105. *Id.* § 4.3.4(a).
106. Interestingly, lead is the one NAAQS pollutant that may be an exception to my argument that the CAA is an awkward fit for microclimate pollution. Since leaded gasoline was phased out, almost all airborne lead has been eliminated. Some stationary sources that emit lead remain, and they are subject to microclimate monitoring. Very few air districts are out of attainment with the 2008 NAAQS for lead. Those out of attainment have, for the most part, distinct stationary sources of lead that create the nonattainment problem and that, if regulated stringently enough, will bring the district into attainment. *See, e.g.*, Cal. Air Res. Bd., *Proposed State Implementation Plan Revision for the Federal Lead Standard 1–2, 5* (May 11, 2012), https://arb.ca.gov/planning/sip/planarea/scabpbstaffrepfinal.pdf (describing the two battery recycling facilities that occasionally cause Los Angeles County to violate the lead NAAQS and describing measures to eliminate violations). The facilities are also required to engage in enhanced source monitoring. *Id.* at 6. The NAAQS system works well for lead because there are so few sources of lead, such that monitoring and regulation can target them.
107. *See, e.g.*, Kathie L. Dionisio et al., *Development and Evaluation of Alternative Approaches for Exposure Assessment of Multiple Air Pollutants in Atlanta, Georgia*, 23 J. EXPOSURE

depending on its sources, with particulate matter from traffic sources comprised of different pollutants than particulate matter from stationary sources.[108] As a result, research has demonstrated that PM 2.5 composition varies significantly across the country: Boston, for example, has significantly higher elemental carbon and NO2 in its PM 2.5 than Pittsburgh, while Pittsburgh has significantly higher concentrations of carbon monoxide and sulfur dioxide.[109] Cities and regions of the country also exhibit different risk levels for mortality from PM 2.5 exposure; yet a recent analysis that attempted to determine the causes of this difference, as well as the sources of PM 2.5 pollution, concluded that "[w]hile it is clear that each city is impacted by different air pollution source mixtures, it is unclear which sources contribute to the differences in risk estimates between the cities."[110]

As a result of the regional nature of PM 2.5 and its different sources, the choice of what scale to monitor is less obvious than that for NO2, which is more uniformly a traffic-related pollutant. EPA therefore recommends monitoring at the neighborhood scale for PM 2.5, though large cities are required to include with their microscale NO2 monitor a PM 2.5 monitor.[111] EPA also makes clear that microscale or middle-scale monitoring may be appropriate if it is "considered to represent area-wide air quality."[112]

Indeed, EPA has opposed efforts to require air districts to measure and monitor near-source/microclimate pollution for purposes of compliance with the NAAQS. Environmental groups in Southern California recently challenged the South Coast Air Quality Management District's monitoring plan that EPA had approved for PM 2.5. The plaintiffs argued that the plan should be invalidated for failing to include a near-road monitor. EPA explained its opposition to the plaintiffs' argument:

SCI. & ENVTL. EPIDEMIOLOGY 581 (2013) (showing less spatial variability across Atlanta for PM 2.5 than for NOx and other traffic-related pollutants).

108. *See* PM 2.5 ISA (2009) at 2–7.

109. *See generally* Lisa K. Baxter et al., *Examining the Effects of Air Pollution Composition on Within Region Differences in PM(2.5) Mortality Risk Estimates*, 23 J. EXPOSURE SCI. & ENVTL. EPIDEMIOLOGY, 457 (2013).

110. *Id.* at 463.

111. 40 C.F.R. pt. 58, app. D, 4.6(b), 4.7.1(b)(2) (2017).

112. 40 C.F.R. pt. 58, app. D, § 4.71(b) (2017). The monitoring of SO2 raises similar issues. As the 2008 Integrated Science Assessment for SO2 explains:

> If a monitoring site is to be used to determine air quality over a much larger area, such as a neighborhood or city, a monitoring agency should avoid placing a monitor probe, path or inlet near local, minor sources. The plume from the local minor sources should not be allowed to inappropriately influence the air quality data collected.

INTEGRATED SCIENCE ASSESSMENT FOR SULFUR OXIDES, *supra* note 46, at 2-7 to 2-8.

> As a practical matter, the area of maximum concentration will most often be a very small area in the immediate vicinity of a dominating local source. However, if the monitor is intended to "represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers" as Sections 4.7.1(b) and (c)(3) require, then the location of maximum concentration will usually be elsewhere. If every monitor simply had to be sited at the location where the single highest reading in the Air District was expected, then the concept of Spatial Scale—and the information it conveys as to the risk to people throughout the urban area—would be lost.[113]

In other words, the highest maximum concentrations may well be close to the heaviest traffic route but measuring those concentrations would not provide the requisite representativeness of ambient air. Because EPA and the environmental groups settled the case, it is unclear whether EPA's position is correct as a legal matter; however, it is nevertheless helpful for understanding EPA's attempt to balance highly localized impacts with background air quality.

EPA's concerns about scale of monitoring may also reflect serious concerns about what monitoring at the microscale would do to the attainment status of many states and air districts. Moreover, such monitoring as a means for measuring NAAQS compliance would raise serious concerns about the regulatory consequences of a nonattainment designation that could result from near-road measurements. I explore those consequences in Part IV, *infra*. I first turn, however, to the second regulatory requirement EPA has utilized in recognition of near-road pollution, a new standard for NO2.

2. NO2 One-Hour Standard

In 2010, EPA set a new one-hour standard for NO2 directly in response to evidence that NO2 levels are elevated near heavily trafficked roads and highways. As the preamble to the new standard explains:

> [E]stimates . . . suggest that on/near roadway NO2 concentrations could be approximately 80% higher on average across locations than concentrations away from roadways . . . . Because monitors in the current network are not sited to measure

113. Brief for Respondent at 31–32, Physicians for Soc. Responsibility v. EPA, No. 12-70016 (9th Cir. filed Oct. 2, 2012) (on file with author).

> peak roadway-associated NO2 concentrations, individuals who spend time on and/or near major roadways could experience NO2 concentrations that are considerably higher than indicated by monitors in the current area-wide NO2 monitoring network.[114]

The short-term standard is combined with new monitoring requirements for near-road pollution and is meant to recognize the problems of short-term exposure to elevated levels of NO2 pollution and to get nonattainment states to address roadway pollution.[115] We do not at this point know whether the new short-term standard will lead to nonattainment status for many air regions; because states are only beginning to implement the new standard and the supporting monitoring network, EPA has yet to designate attainment/nonattainment standards for it.[116] If the new standard leads to a significant number of nonattainment designations, states will face a real conundrum about how to respond. This conundrum, raised not just by the new NO2 standard but by any attempt to regulate microclimate pollution caused by traffic, is one of several conundrums the NAAQS provisions of the CAA create for the regulation of microclimate pollution more generally. I address these issues in Part IV, *infra*. In the next Section, however, I explore a third way in which EPA is attempting to address near-source pollution.

3. Improvements in Stationary Source Monitoring

In response to concerns that refineries have been using emissions factors that consistently underestimate actual emissions, EPA has begun to refine emissions calculations for flaring and some other activities from refineries.[117] And beginning in 2018, for the first time refineries will be required to monitor fence-line emissions.[118] This is a major advancement in understanding with precision the actual emissions coming from refineries.

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114. 75 Fed. Reg. 6471, 6479 (Feb. 9, 2010) (to be codified at 40 C.F.R. pts. 50 & 58).
115. *See supra* notes 104–105 and accompanying text (describing near-road monitoring requirements for the NO2 standard).
116. *See, e.g.*, Letter from Jared Blumenfeld, EPA Region 9 Administrator, to Edmund G. Brown, California Governor (June 29, 2011) (describing EPA decision to designate all areas of the country "unclassifiable" while new monitoring network is implemented).
117. *See* EPA, NEW AND REVISED EMISSION FACTORS FOR FLARES AND NEW EMISSIONS FACTORS FOR CERTAIN REFINERY PROCESS UNITS AND DETERMINATION FOR NO CHANGES TO VOC EMISSION FACTORS FOR TANKS AND WASTEWATER TREATMENT SYSTEMS [hereinafter NEW EMISSIONS FACTORS], https://www3.epa.gov/ttn/chief/consentdecree/index_consent_decree.html [https://perma.cc/NX7H-ZST6].
118. *See* EPA, FACT SHEET: FINAL PETROLEUM REFINERY SECTOR RISK AND TECHNOLOGY REVIEW AND NEW SOURCE PERFORMANCE STANDARDS, https://www.epa.gov/sites/production/

Since EPA began requiring the monitoring of emissions, regulators have relied extensively on emissions factors. These factors are developed by EPA as an inexpensive means to represent actual emissions, but they are based on averages of available data rather than actual monitoring.[119] EPA and states rely heavily on emissions factors in constructing emissions inventories, making permitting, compliance, and enforcement decisions, and developing emissions reductions strategies.[120] And yet a troubling 2006 U.S. EPA Office of Inspector General (OIG) Report evaluating EPA's use of emissions factors concluded that 62 percent of EPA's emissions factors were of below average or poor quality.[121] To be sure, a number of large industrial facilities do actual monitoring, typically for equipment or facilities, for which a permit is required.[122] Nevertheless, the OIG report estimates that EPA relies on emissions factors for 80 percent of its emissions determinations.[123]

EPA has sometimes taken enforcement actions against facilities whose actual emissions significantly exceed reported emissions based on emissions factors. The OIG report recounts enforcement efforts after the issuance of a critical 1996 OIG report against some refineries, wood products manufacturers, and ethanol producers.[124] More recently, the agency—in response to litigation filed by Texas- and Louisiana-based environmental justice groups concerned that emissions data for refineries was inaccurate—evaluated and revised upward VOCs emissions factors for flaring at refineries and several other refinery operations.[125] And finally, after many years of

files/2016-06/documents/2010-0682_factsheet_overview.pdf [https://perma.cc/B8N9-ELQX]. As with many EPA reforms, this change is the result of a citizen suit. For the final rule itself, see Petroleum Refinery Sector Risk and Technology Review and New Source Performance Standards, 80 Fed. Red. 75,178, 75,178–354 (Dec. 1, 2015) (to be codified at 40 C.F.R. §§ 60–63), *available at* https://www.gpo.gov/fdsys/pkg/FR-2015-12-01/pdf/2015-26486.pdf.

119. *See* EPA, AIR EMISSIONS FACTORS AND QUANTIFICATION, BASIC INFORMATION OF AIR EMISSIONS FACTORS AND QUANTIFICATION, https://www.epa.gov/air-emissions-factors-and-quantification/basic-information-air-emissions-factors-and-quantification [https://perma.cc/4YGW-LPX2].

120. *See* EPA, OFFICE OF INSPECTOR GENERAL, EPA CAN IMPROVE EMISSIONS FACTOR DEVELOPMENT AND MANAGEMENT 1, 4–5 (Rep. No. 2006-00017, 2006).

121. *See id.* at 9.

122. *See, e.g.*, 40 C.F.R. §§ 64.1–64.2 (2017) (specifying circumstances under which compliance assurance monitoring is required).

123. EPA, OFFICE OF INSPECTOR GENERAL, *supra* note 120, at 4.

124. *See id.* at 11–13.

125. EPA entered into a consent decree to evaluate emissions factors in *Air Alliance Houston, Community In-Power and Development Association, Inc., Louisiana Bucket Brigade and Texas Environmental Justice Advocacy Services* ("Plaintiffs"). Air Alliance Houston v. Gina McCarthy, No. 1:13-cv-00621-KBJ (D.D.C. filed May 07, 2014). *See* NEW EMISSIONS FACTORS, *supra* note 117.

environmental justice calls for more effective monitoring of refineries, EPA issued a rule in 2016 that will require monitoring at the fence-lines of refineries to determine actual exposure levels beginning in 2018.

The use of emissions factors to estimate emissions from stationary sources makes sense for measuring ambient pollution at a relatively large scale, even if the emissions factors are not completely accurate. If near-source pollution exposure is not a significant health issue, then, as long as ambient concentrations are measured effectively and are within NAAQS limits, the absolute contribution of any individual source is not as important as the cumulative contributions of all the sources in an air basin. But as our scientific understanding about near-source exposure to pollutants like PM 2.5 and NOx has improved, the importance of the accuracy of individual source emissions has increased. Yet our measuring and monitoring of stationary source emissions appear not to have kept up, with the exception of the new refinery monitoring requirements. Nor is the ambient focus of the CAA particularly well-suited to address individual stationary source emissions in order to address near-source exposure.

I turn to the structural problems NAAQS regulations raise for hotspot pollution next.

IV. UNSUITABILITY OF NAAQS REGULATION FOR TARGETING HOTSPOT POLLUTION

A. State v. Federal Authority Over Sources and the Problem of Near-Road Exposure

The division of CAA authority between states and the federal government for the regulation of different types of sources poses perhaps the biggest structural barrier to the regulation of near-road pollution. The familiar cooperative federalism structure of the CAA is worth describing here in order to explain this barrier.

Once a state is designated as either in or out of attainment with a particular ambient air standard, it must prepare a state implementation plan (SIP) that demonstrates either how the area will maintain its air quality in order to remain in attainment with the NAAQS or how the area will come into attainment.[126] States must submit their SIPs to EPA for approval. Every five years, the CAA requires EPA to review the criteria on which a NAAQS is

126. 42 U.S.C. § 7410(a)(1) (2012).

based and update the standards consistent with the new information.[127] If a standard is tightened, a new series of attainment/nonattainment designations and SIP updating must occur.[128]

The SIP process is designed to allow states discretion to develop their own regulatory regimes that will either maintain or achieve compliance with the NAAQS. Nevertheless, state discretion is limited in important ways. Most significantly, states have very little power to directly regulate emissions from mobile sources and substantially more control over the regulation of stationary sources, though even that discretion is cabined in important ways.[129] Relatively speaking, however, if a state is out of attainment with the NAAQS, it has more power to regulate and tighten up on stationary sources than it does mobile sources.

The establishment of the one-hour NO2 NAAQS brings this distinction between stationary source and mobile source regulatory authority into stark relief: If a state is found to be in nonattainment with the one-hour standard, its options for directly regulating mobile sources are limited. The same would be true if states were required to monitor PM 2.5 at near-road sites, if the monitors produced readings that kicked a state or one of its air districts into nonattainment or a more serious designation.

To be sure, not all options are foreclosed to states. One way to think about how to reduce near-road emissions is to think broadly about ways to reduce human exposure. States could reduce the emissions that are coming out of mobile sources; they could try to channel the emissions away from humans; or they could keep the humans away from the emissions. Below I explore the role of the CAA in reducing mobile source emissions.

B. Directly Reducing Emissions From Mobile Sources

1. Tailpipe Emissions From New Mobile Sources

Mobile source emissions are the cause of most near-road emissions, comprise 60 percent of NO2 emissions, and make up a large share of PM 2.5. Diesel emissions from heavy trucks and other diesel engines are of particular concern, with some studies showing that half of near-road particulate matter

127. 42 U.S.C. § 7409(d)(1) (2012).
128. 42 U.S.C. § 7410(a)(2)(H)(i) (2012).
129. Requirements for technology-based standards for ozone attainment zones are one example of this, as the stringency of the requirement ratchets up the further an air district is out of attainment with the NAAQS.

comes from heavy-duty diesel engines.[130] In the long term, the most direct way of cleaning up these emissions is to prevent them from coming out of tailpipes in the first place.

To be sure, federal and California regulations have been remarkably successful in reducing tailpipe emissions for new passenger automobiles, achieving reductions by as much as 99 percent compared with cars manufactured in the 1960s.[131] EPA has also issued a series of regulations over the years to clean up heavy-duty diesel engines and to require the use of low-sulfur diesel fuel. The most significant of these regulations required new standards for model year engines 2004 and later and standards mandating engine modifications to allow for the use of low-sulfur diesel fuel.[132] EPA has also recently paired with the National Highway Transportation and Safety Administration to tighten fuel economy standards for passenger, medium, and heavy duty vehicles, which I explore in more detail in Part V, *infra*.

Nevertheless, even with stringent emissions standards, as I have already detailed, mobile sources continue to emit huge percentages of many NAAQs pollutants (as much as 60 percent of NO2 pollution and more than a quarter of PM 2.5) and continue to cause near-road exposure problems. As a result, a state facing nonattainment for the new NO2 short-term exposure standard,

130. Shih Ying Chang et al., *supra* note 5, at 916.

131. *See* EPA, *History of Reducing Air Pollution From Transportation in the United States*, https://www.epa.gov/air-pollution-transportation/accomplishments-and-success-air-pollution-transportation (last visited March 26, 2018).

132. *See* EPA, REGULATIONS FOR EMISSIONS FROM VEHICLES AND ENGINES, FINAL RULE FOR CONTROL OF EMISSIONS OF AIR POLLUTION FROM HIGHWAY HEAVY-DUTY ENGINES [hereinafter REGULATIONS FOR EMISSIONS FROM VEHICLES AND ENGINES],https://www.epa.gov/regulations-emissions-vehicles-and-engines/final-rule-control-emissions-air-pollution-highway-heavy [https://perma.cc/Q8DG-CZDZ]; 40 C.F.R. pts. 9, 86 (2016); EPA, REGULATIONS FOR EMISSIONS FROM VEHICLES AND ENGINES, FINAL RULE FOR CONTROL OF EMISSIONS OF AIR POLLUTION FROM 2004 AND LATER MODEL YEAR HEAVY-DUTY HIGHWAY ENGINES AND VEHICLES [hereinafter LATER MODEL YEAR HEAVY-DUTY HIGHWAY ENGINES AND VEHICLES], https://www.epa.gov/regulations-emissions-vehicles-and-engines/final-rule-control-emissions-air-pollution-2004-and-later [https://perma.cc/8K76-3AVT]; EPA, REVISION OF LIGHT-DUTY ON-BOARD DIAGNOSTICS REQUIREMENTS [hereinafter LIGHT-DUTY ON-BOARD DIAGNOSTICS REQUIREMENTS], https://www.epa.gov/regulations-emissions-vehicles-and-engines/final-rule-control-emissions-air-pollution-2004-and-later [https://perma.cc/8K76-3AVT]; EPA, REGULATORY ANNOUNCEMENT, FINAL EMISSION STANDARDS FOR 2004 AND LATER MODEL YEAR HIGHWAY HEAVY-DUTY VEHICLES AND ENGINES (July 2000) [hereinafter FINAL EMISSION STANDARDS FOR 2004 AND LATER MODEL], https://nepis.epa.gov/Exe/ZyPDF.cgi/P1001YS2.PDF?Dockey=P1001YS2.PDF [https://perma.cc/9DP4-2DG4]; EPA, HEAVY-DUTY ENGINE AND VEHICLE STANDARDS AND HIGHWAY DIESEL FUEL SULFUR CONTROL REQUIREMENTS (Dec. 2000) [hereinafter HEAVY-DUTY ENGINE AND VEHICLE STANDARDS AND HIGHWAY DIESEL], https://nepis.epa.gov/Exe/ZyPDF.cgi/P1001CXZ.PDF?Dockey=P1001CXZ.PDF [https://perma.cc/2R9P-85GG].

or a state forced to use near-road monitoring to demonstrate PM 2.5 attainment status for the twenty-four-hour standard, would most likely want to rely on more stringent tailpipe emissions in its SIP to demonstrate how it would come into attainment.

The problem with a strategy that relies on regulating tailpipe emissions, however, is that states have very little authority to do so. Section 209 of the CAA preempts all states from regulating emissions from mobile sources, with the exception of California, which can set standards so long as they are at least as protective of public health and welfare as the federal standards.[133] States may choose to follow either the federal standards or the California standards, but may not impose their own.[134] States are also prohibited from regulating emissions from nonroad vehicles, again with the exception of California, with a similar option to choose to follow the federal standards or the California standards.[135] As a result, a state or air district seeking to reduce near-road emissions can either follow the federal standards or California standards but cannot regulate any class of motor vehicles more stringently than those two standards in an attempt to come into attainment. A state's SIP for nonattainment for near-road sources, then, could not easily rely on the most direct mechanism for addressing the problem. At best, a state that does not already follow the California standards could choose to do so.

Even following California standards does little, however, to address heavy-duty vehicle emissions. Because trucks often enter a state from other states in order to transport cargo, in-state heavy-duty regulation is likely to be insufficient to solve the problem. Even California, which has its own authority to regulate heavy-duty emissions and an economy far larger than most states, faces this problem. Two of California's air districts, along with a number of air districts from across the country, recently petitioned EPA to regulate NOx emissions from heavy-duty engines more stringently in order to assist them in coming into attainment with the new ozone and PM standards.[136] As EPA noted in its response, between 40 and 67 percent of NOx emissions across the country come from mobile sources. In Southern California, this number rises to 88 percent, with the largest source category

133. 42 U.S.C. § 7543 (a)–(b) (2012).
134. *Id.* § 7507.
135. *Id.* § 7543(e).
136. *See* EPA, MEMORANDUM IN RESPONSE TO PETITION FOR RULEMAKING TO ADOPT ULTRA-LOW NO(X) STANDARDS FOR ON-HIGHWAY HEAVY-DUTY TRUCKS AND ENGINES 5 (2016) [hereinafter MEMORANDUM IN RESPONSE TO PETITION], https://www.epa.gov/sites/production/files/2016-12/documents/nox-memorandum-nox-petition-response-2016-12-20.pdf [https://perma.cc/AEV9-9JC8].

being diesel trucks.[137] Without federal regulatory action, many districts will not be able to comply with new standards for ozone and PM.[138] This problem is even more acute for near-road exposure, which is entirely caused by mobile sources, with heavy-duty trucks playing a key role.

2. Regulation of Fuels

Unlike tailpipe emissions, states do have authority to regulate fuels with permission from EPA, subject to some significant limitations, and could attempt to do so to reduce near-road exposure. State authority here, however, is limited in important ways. Under the 1990 Amendments to the CAA, states gained authority to require the use of new fuels under certain conditions with EPA approval. EPA could approve a state fuel requirement, but only if it found that the regulation of fuel is necessary to achieve the NAAQS and that "no other measures . . . would bring about timely attainment . . . ," unless the state could show that other measures "are technically possible to implement, but are unreasonable or impracticable."[139] This authority led EPA to approve seven different fuels used in twelve different states around the country.[140] The proliferation of these so-called "boutique" fuels led to opposition from industry groups and Congress responded in the Energy Policy Act of 2005. The Act restricted state authority by allowing the use of only those seven fuels already approved by EPA as of the time of the amendments.[141] Thus, states can require less polluting fuels, but can only use those already on the boutique fuels list and only with EPA approval.

California, once again, has its own authority to regulate fuels, but unlike the mobile source emissions authority, other states cannot adopt California fuels absent a showing to EPA that adoption of California fuel is necessary for NAAQS attainment.[142] Moreover, for diesel fuel, some of the same interstate

137. *See id.* at 5.
138. Although EPA indicated in its responsive memorandum to the air district petition that it would initiate rule-making proceedings, *see id.* at 20, given the change in EPA leadership such a regulatory move now seems unlikely.
139. 42 U.S.C. § 7545(c)(4)(C).
140. *See* EPA, GASOLINE STANDARDS, STATE FUELS, https://www.epa.gov/gasoline-standards/state-fuels (last visited Mar. 7, 2018) (listing approved fuels).
141. Energy Policy Act of 2005, Pub. L. No. 109-58, § 1541(b), 119 Stat. 1107 (2005).
142. *See* Motor Vehicle Mfrs. Ass'n v. N.Y. State Dep't of Envt'l Conservation, 810 F. Supp. 1331, 1343 (N.D.N.Y. 1993) *modified*, 831 F. Supp. 57 (N.D.N.Y. 1993) *aff'd in part, rev'd in part*, 17 F.3d 521 (2d Cir. 1994) (describing approval requirements).

issues that arise with the regulation of heavy-duty truck emissions also arise with fuels, with cross-border trucks creating significant emissions.

C. Existing v. New Mobile Sources

Just as the CAA requires much more stringent regulation of new stationary sources as opposed to existing ones, it has also long focused much more attention on new vehicles than existing ones. Yet older vehicles of all categories are significantly dirtier than new ones and are the major cause of near-road pollution. A recent and sophisticated study of passenger vehicle emissions showed that 90 percent of NOx emissions came from just 25 percent of automobiles and the top 5 percent of emitters contributed 40 percent of carbon monoxide and black carbon.[143] California estimates that 70 percent of air toxics come from diesel engines.[144]

The CAA does require some states to address pollution from older passenger vehicles in relatively limited ways. In recognition of the contribution of existing mobile sources to ambient air pollution, the 1990 CAA amendments required ozone and carbon monoxide nonattainment areas to establish inspection and maintenance programs for existing vehicles.[145] The programs have until very recently been rife with fraud and largely ineffectual, and they also raise complex distributional questions about how to pay for repairs and upgrades to older cars.[146] And they have been aimed at nonattainment areas rather than near-road exposure.

EPA has done even less to address the largest existing mobile source problem: emissions from heavy-duty vehicles. In 1993, EPA required large cities (greater than 750,000 in population) to retrofit older urban buses, if they were

143. *See* J. M. Wang et al., *Plume-Based Analysis of Vehicle Fleet Air Pollutant Emissions and the Contributions From High Emitters*, 8 ATMOSPHERIC MEASUREMENT TECH. 3263, [3271] (2015) (studying vehicular emissions in Ontario, Canada). Canadian emissions regulations follow U.S. 2007 Tier 2 standards. *See id.* at [3271].

144. Cal. Air Res. Bd., *Overview: Diesel Exhaust and Health*, https://www.arb.ca.gov/research/diesel/diesel-health.htm (last visited March 26, 2018).

145. *See, e.g.*, 42 U.S.C. § 7511a(a)(2)(B) (2012) (requiring inspection and maintenance programs for marginal ozone non-attainment zones).

146. *See* Amihai Glazer et al., *Clean on Paper, Dirty on the Road: Troubles With California's Smog Check*, 29 J. TRANSPORT ECON. & POL'Y 85, 85–86 (1995) (showing large discrepancies between inspection failure rates in Inspection & Maintenance facilities compared with failure rates in roadside audits, lower emissions reductions than predicted, fraud in testing facilities).

being replaced or rebuilt.[147] EPA also has a number of voluntary incentive programs to retrofit heavy-duty diesel engines and, since 2005 with the adoption of the Diesel Emission Reduction Act, has provided some funding for states to retrofit old engines in school buses and other heavy duty vehicles.[148] The Trump Administration has, however, proposed reducing this funding by 83 percent in the 2017–2018 budget.[149] The result is that long-lived heavy-duty engines remain on the road for decades.[150] Moreover, EPA recently proposed rolling back emissions standards for "glider kits," (which is essentially a new chassis with an old engine) even though evidence shows that they emit vastly more particulate matter and NOx than new engines.[151]

D. Issues With Stationary Sources and Their Relationship to Mobile Sources

The regulation of stationary sources under the CAA raises its own issues in how states can address near-road exposure. The first, the application of New Source Review, has already arisen with respect to the NO2 short-term standard. The second, the propensity to regulate stationary sources in response to a problem caused largely by mobile sources, could occur with efforts either to implement the NO2 standard or in response to monitoring of PM 2.5 at roadside. This propensity is even more problematic because of the distinction the CAA makes between new and existing sources. Finally, the likelihood that stationary source-targeting will occur is compounded by the Clean Air Act's requirement that SIPs contain enforceable emissions limitations requirements.

147. *See* 40 C.F.R. § 85.1401 (2016); *United States: Urban Bus Retrofit Rebuild (UBRR) Program*, DIESELNET, https://www.dieselnet.com/standards/us/ubrr.php (last visited Mar. 7, 2018) (describing program).

148. *See* EPA, CLEAN DIESEL AND DERA FUNDING, https://www.epa.gov/cleandiesel [https://perma.cc/3F4E-VNK8] (last visited Mar. 7, 2018) (describing programs and funding levels).

149. *See* Lauren Tyler, *Trump's Proposed Budget Cuts Funding Available for Clean Transportation*, NGT NEWS (May 23, 2017), https://ngtnews.com/trumps-proposed-budget-cuts-funding-available-for-clean-transportation [https://perma.cc/TTN4-KC39].

150. *See* CAL. ENVTL. PROT. AGENCY AIR RES. BD., DRAFT, SUPPORTING INFORMATION FOR TECHNOLOGY ASSESSMENTS: TRUCK AND BUS SECTOR DESCRIPTION, at VI-2 (2016) (describing shelf-life and durability of heavy duty engines).

151. EPA, PROPOSED RULE FOR REPEAL OF EMISSIONS REQUIREMENTS FOR GLIDER VEHICLES, GLIDER ENGINES AND GLIDER KITS; *see also*, *Heavy-Duty Truck "Glider Kit" Rule*, ENVTL. L. HARV., http://environment.law.harvard.edu/2018/02/heavy-duty-truck-glider-kit-rule/ [https://perma.cc/2PN9-BUWD](last visited March 28, 2018).

Before detailing problems with stationary source regulation and microclimate pollution, it is worth noting that EPA has made significant strides in reducing pollutants from these sources, including from existing, rather than stationary, sources. Three programs are particularly noteworthy. The first, the Acid Rain Trading Program, targeted the electric power sector through the country's first large-scale cap-and-trade program. It cut sulfur dioxide from power plants by 50 percent compared to 1980 levels and led to a significant decline in NOx pollution.[152] The second, a series of cap-and-trade programs EPA established to tackle cross-state ozone pollution, have significantly reduced NOx and sulfur dioxide as well.[153] Importantly, these reductions have also led to significant reductions in particulate matter.[154] And finally, a recently adopted rule aimed at toxic pollutants emitted from the power sector, known as the MATS rule,[155] will result in major reductions not only in toxic pollutants like mercury but in particulate matter as well. In fact, the vast majority of the health benefits that will result from the MATS rule will come from reductions in fine particulates, including NO2 and sulfur dioxide particles.[156]

Despite these successful regulatory efforts, problems remain with stationary sources and near-source exposure. The first issue has already arisen with the regulation of NO2 and the new near-road standard. The CAA requires any new or modified stationary source to obtain a permit before beginning operation, whether in an attainment or nonattainment area, if the source will emit a specified number of tons per year of a regulated pollutant.[157] Although

152. *See* U.S. EPA, CLEAN AIR MARKETS, ACID RAIN PROGRAM, https://www.epa.gov/airmarkets/acid-rain-program [https://perma.cc/VVK9-654V] (describing pollution reductions).

153. *See* U.S. EPA, CLEAN AIR MARKETS, INTERSTATE AIR POLLUTION TRANSPORT, https://www.epa.gov/airmarkets/interstate-air-pollution-transport [https://perma.cc/J5WR-L5E7] (describing and linking to programs).

154. *See id.*

155. 40 C.F.R. pt. 63 (2017). The MATS rule was successfully challenged on the grounds that EPA issued it without considering the costs of the rule in *Michigan v. EPA*, 135 S. Ct. 2699 (2015). EPA subsequently reconsidered the rule in light of the Court's decision and found that cost considerations did not change its determination that the rule is appropriate. 81 Fed. Reg. 24,420 (April 25, 2016). The rule is now being put into effect.

156. *See* U.S. EPA, REGULATORY IMPACT ANALYSIS FOR THE FINAL MERCURY AND AIR TOXICS STANDARDS, at ES-1 (Dec. 2011), https://www3.epa.gov/ttnecas1/regdata/RIAs/matsriafinal.pdf (outlining massive co-benefits from reduction of PM 2.5).

157. For an explanation of these New Source Review requirements, see U.S. EPA, NEW SOURCE REVIEW (NSR) PERMITTING, LEARN ABOUT NEW SOURCE REVIEW, https://www.epa.gov/nsr/learn-about-new-source-review [https://perma.cc/ET5D-9HDT]. The PSD (attainment) provisions are contained in 42 U.S.C. §§ 7475(a). The provisions for new sources in non-attainment areas are contained in 42 U.S.C. § 7503 (2012).

the NO2 one-hour standard was explicitly motivated by the health problems associated with exposure to near-road pollution, EPA issued a guidance memorandum shortly after the standard was adopted, making clear (appropriately given the statutory requirements) that the stationary source permitting provisions were triggered by the new standard.[158]

Shortly after the issuance of the initial guidance memorandum, it became clear that a relatively large number of new and modified stationary sources were having trouble getting permitted. These sources apparently found, based on modeling, that they would be in violation of the standard.[159] The sources included emergency electric generating units, pump stations, power plants, paper mills, and refineries.[160] In order to reduce the number of stationary sources having difficulty getting permits given the modeling results, EPA has issued a series of guidance memoranda to "facilitate the permitting of new and modified PSD [Prevention of Significant Deterioration] major stationary sources."[161] Presumably, even with this guidance, seemingly designed to demonstrate that the sources will not need permits, some sources will continue to violate the standard and will need to install the "best available control technology" (BACT) as a result.[162]

There are at least two problems with the fact that new and modified stationary sources may be required to get permits and install BACT technology as a result of the new one-hour NO2 standard. First, the new standard was motivated by near-road pollution, not by stationary source emissions. Thus, requiring permits of new and modified stationary sources will do nothing to solve the problem, nor will it help bring air districts that are out of compliance because of near-road exposure into attainment with the one-hour standard. Moreover, to the degree that stationary sources are causing near-source exposure problems, exposure levels are likely to be far worse at existing stationary sources, like refineries and power plants. Yet existing sources are

158. *See* U.S. EPA, MEMORANDUM, APPLICABILITY OF THE FEDERAL PREVENTION OF SIGNIFICANT DETERIORATION PERMIT REQUIREMENTS TO NEW AND REVISED NATIONAL AMBIENT AIR QUALITY STANDARDS 1 (Apr. 1, 2010), https://www.epa.gov/sites/production/files/2015-07/documents/psdnaaqs.pdf.

159. U.S. EPA, MEMORANDUM, GUIDANCE CONCERNING THE IMPLEMENTATION OF THE 1-HOUR NO(2) NAAQS FOR THE PREVENTION OF SIGNIFICANT DETERIORATION PROGRAM 1 (June 29, 2010), https://www3.epa.gov/ttn/naaqs/aqmguide/collection/nsr/appwno2.pdf.

160. *See id.*

161. U.S. EPA, ADDITIONAL CLARIFICATION REGARDING APPLICATION OF APPENDIX W MODELING GUIDANCE FOR THE 1-HOUR NO(2) NATIONAL AMBIENT AIR QUALITY STANDARD (Mar. 1, 2011), https://www.epa.gov/sites/production/files/2015-07/documents/appwno2_2.pdf.

162. 42 U.S.C. § 7475(a)(4) (2012).

largely exempt from NAAQS regulation under the CAA, unless they are modified in a manner that triggers New Source Review[163] or unless they are in nonattainment areas.[164] The existing/new source division and problem are hardly unique to the one-hour NO2 standard. However, it is worth pointing out that if the aim of the new standard is to reduce near-term exposure to pollutants at dangerous levels, targeting new stationary sources, as the CAA does, is not likely to solve the problem.[165]

The second problem with the structure of the CAA for regulating near-source pollution is that, if states are found to be out of attainment or if near-road monitors are used to measure PM 2.5 and kick a district out of attainment or further out of attainment, then the CAA pushes states to clamp down further on stationary as opposed to mobile sources. This structural incentive exists, again, because states have much less power over mobile source emissions than they do over stationary sources, even when the pollution problem is caused by mobile source emissions. States must impose "reasonably available control measures" on existing sources in the area if out of attainment with a NAAQS.[166]

Finally, the SIP provisions of the CAA contain a strong directive to states to include "enforceable emission limitations" and other direct control measures to demonstrate maintenance or attainment with an air standard.[167] As a result, states are likely to look to stationary sources, on which they can impose enforceable emissions limits, to reduce NO2 or PM 2.5 emissions.

1. Stationary Sources, Emissions Factors, and Near-Source Exposure

As I described above, EPA and states rely extensively on emissions factors for stationary sources to estimate emissions. For ambient concentrations of pollutants that do not create near-source problems (SO2 is an example),[168] emissions factors work reasonably well as long as background monitoring is accurate and effective. For a pollutant like PM 2.5, however, with known near-

163. See 42 § 7479(1) (2012) for a definition of new source.
164. *See* 42 U.S.C. § 7502(c)(1) (2012) (requiring "reasonably available control measures," including "from existing sources").
165. *See, e.g.*, Dialogue, *Grandfathering Coal: Power Plant Regulation Under the Clean Air Act*, 46 ENVTL. L. REP. 10541, 10542 (2016) (discussion of RICHARD REVESZ & JACK LIENKE, STRUGGLING FOR AIR: POWER PLANS AND THE "WAR ON COAL," with Revesz calling the grandfathering provisions of old stationary sources the "tragic flaw" of the Clean Air Act).
166. 42 U.S.C. § 7502(c)(1) (2012).
167. *Id.* § 7410(a)(2)(A).
168. *See* INTEGRATED SCIENCE ASSESSMENT FOR SULFUR OXIDES (2017), at 1-8 to 1-9 (describing exposure).

source consequences, emissions factors may create a significant problem by masking exposure to emissions from stationary sources and consequent health effects for those who live near them.

The PM 2.5 standard was adopted in 1997, but it was not until 2008 that states submitted SIPs that either demonstrated how they would maintain attainment status or come into attainment.[169] The attainment designations themselves, along with the SIPs, were based largely on emissions factors for stationary and mobile sources.[170] Indeed, emissions inventories for PM 2.5 continue to rely extensively on emissions factors to estimate emissions from fuel combustion, refineries, and so forth.[171]

The 2006 OIG report about emissions factors cautioned EPA about the importance of developing emissions factors based on good data.[172] It is difficult to determine, however, whether EPA heeded this advice. A 2004 report commissioned by the U.S. Department of Energy cautioned that:

> There are few existing data regarding emissions and characteristics of fine aerosols from oil, gas and power generation industry combustion sources, and the information that is available is generally outdated and/or incomplete. Traditional stationary source air emission sampling methods tend to underestimate or overestimate the contribution of the source to ambient aerosols because they do not properly account for primary aerosol formation, which occurs after the gases leave the stack.[173]

If emissions factors are systematically overestimating PM 2.5 from these sources, there should not be a problem with near-source exposure. Not only are the sources producing fewer emissions than emissions factors estimate, but regulated parties are also likely to challenge the application of emissions estimates that subject them to more stringent regulation than they would experience with accurate estimates.

But if emissions factors systematically underestimate emissions from stationary sources, like refineries and power plants, our current system of regulation is unlikely to detect these problems. This is likely to be particularly

169. *See* U.S. EPA, EPA CAN IMPROVE EMISSIONS FACTORS DEVELOPMENT AND MANAGEMENT, *supra* note 120, at 14 (describing role of emissions factors in PM 2.5 process).
170. *See id.* at 4–6.
171. *See* U.S. EPA, EPA'S REP. ON THE ENVIRONMENT, PARTICULATE MATTER EMISSIONS, https://cfpub.epa.gov/roe/indicator.cfm?i=19 [https://perma.cc/ZT92-MJWX] (last visited Dec. 28, 2017).
172. *See* OFFICE OF INSPECTOR GENERAL, EMISSIONS FACTORS REPORT, *supra* note 120, at 15.
173. GLENN C. ENGLAND, DEVELOPMENT OF FINE PARTICULATE EMISSION FACTORS AND SPECIATION PROFILES FOR OIL-AND GAS-FIRED COMBUSTION SYSTEMS ix (2004).

true in attainment areas where existing stationary sources are subject to virtually no regulation. These sources are not required to monitor their emissions if they are not otherwise subject to regulation. Moreover, monitoring requirements for PM 2.5 do not require microscale monitoring and thus are unlikely to detect near-source emissions from a stationary source.[174]

Even in nonattainment areas, the use of emissions factors may underestimate near-source emissions from stationary sources and our regulatory apparatus to regulate NAAQS is not well-suited to respond. The monitoring to determine nonattainment, again, is not likely to detect these emissions since the monitoring is at a broader scale. Moreover, existing stationary sources enjoy much more favorable treatment than new sources and thus would not be required to install control technology as effective as that required of new sources.[175]

V. CAN THE CURRENT CAA WORK TO ADDRESS NEAR-SOURCE POLLUTION?

A. In the Long Run, Greenhouse Gas Emissions Regulation May Ultimately Solve the Problem

1. Mobile Sources

Ironically, the problem of microclimate pollution may ultimately be resolved because of a pollution problem at the opposite end of the planetary scale, climate change. Solutions aimed at mitigating climate change will, however, take multiple decades, and, in the meantime, communities that live near highly polluted sources will continue to breathe unhealthful air.

Although greenhouse gas (GHG) emissions, in adding to heat-trapping gases that envelop the planet, create the exact opposite problem of pollution hotspots in their global scale, a large percentage of GHG emissions come from the same sources that create near-source pollution.[176] The transportation and electricity sectors are collectively responsible for more than half of GHG emissions in the United States. When industrial sources are included, the total

174. See discussion of monitoring requirements at notes 106–112, *supra.*
175. See discussion of existing new sources at notes 163–164, *supra.*
176. *See* U.S. EPA, INVENTORY OF U.S. GREENHOUSE GAS EMISSIONS AND SINKS, https://www.epa.gov/ghgemissions/inventory-us-greenhouse-gas-emissions-and-sinks [https://perma.cc/29F3-BES5] (last visited Aug. 14, 2017).

rises to nearly 80 percent.[177] Many observers suggest that the most effective long-run strategy to reduce emissions from the transportation sector is to electrify the vehicle fleet.[178] The electricity used to charge vehicles would come largely from renewable sources, combined with battery storage and other non-carbon mechanisms, eventually making fossil fuel power plants and oil refineries unnecessary or much less prevalent.[179] The major hotspot sources would, in other words, be clean. And the Clean Air Act in its current form could be used to achieve this transition.

How the CAA came to cover greenhouse gases is a well-known story, beginning with the U.S. Supreme Court's holding in *Massachusetts v. EPA* that greenhouse gases are pollutants under the Act and that EPA must determine whether they endanger public health and welfare.[180] Based on that authority, EPA under President Obama made the endangerment finding[181] and then issued two rounds of regulatory requirements focused on vehicles, including passenger, medium, and heavy duty.[182] The details of the standards are less important than the fact of them: The National Highway Transportation and Safety Authority worked with EPA for the first time to issue combined fuel economy standards and GHG regulations to bolster fuel economy from all categories of vehicles.[183] The standards were designed to be the first step on the road to much deeper decarbonization by mid-century.[184]

177. *See id.*

178. *See, e.g.*, MIT & SLOAN AUTO. LAB. ENG'G SYS. DIV., ON THE ROAD TOWARD 2050: POTENTIAL FOR SUBSTANTIAL REDUCTIONS IN LIGHT-DUTY VEHICLE ENERGY USE AND GREENHOUSE GAS EMISSIONS 18 (John Heywood & Don MacKenzie eds., 2015).

179. *See* SUSTAINABLE DEV. SOL. NETWORK, PATHWAYS TO DEEP DECARBONIZATION IN THE UNITED STATES, at vii (U.S. 2050, vol. 1, Technical Report Executive Summary (2015)).

180. 549 U.S. 497 (2007). For an account of how the CAA has been used to address GHG regulations, see generally Ann Carlson, *An Ode to the Clean Air Act*, 30 J. LAND USE & ENVTL. L. 119 (2014).

181. *See* U.S. EPA, ENDANGERMENT AND CAUSE OR CONTRIBUTE FINDINGS FOR GREENHOUSE GASES UNDER THE SECTION 202(A) OF THE CLEAN AIR ACT, https://www.epa.gov/ghgemissions/endangerment-and-cause-or-contribute-findings-greenhouse-gases-under-section-202a-clean [https://perma.cc/X85V-4Y48] (last visited Aug. 18, 2017).

182. For an explanation of all of the regulatory programs, see JAMES E. MCCARTHY & BRENT D. YACOBUCCI, CONG. RESEARCH SERV., R40506, CARS, TRUCKS AND CLIMATE CHANGE: GREENHOUSE GASES FROM MOBILE SOURCES 3–17 (2016). *See also* U.S. EPA, REGULATIONS FOR GREENHOUSE GAS EMISSIONS FROM PASSENGER CARS AND TRUCKS, https://www.epa.gov/regulations-emissions-vehicles-and-engines/regulations-greenhouse-gas-emissions-passenger-cars-and [https://perma.cc/SS5T-J39L] (last visited Aug. 18, 2017).

183. *See* MCCARTHY & YACOBUCCI, *supra* note 182, at 3.

184. *See* U.S. Cover Note, INDC and Accompanying Information (Mar. 31, 2015), http://www4.unfccc.int/ndcregistry/PublishedDocuments/United%20States%20of%20America%20First/U.S.A.%20First%20NDC%20Submission.pdf (describing U.S. climate

At least some of the new regulatory requirements are currently under review by the Trump Administration, and passenger vehicle standards for model years 2021–2025 and standards for glider kits (which combine new trailers with refurbished engines) are likely to be loosened or eliminated.[185] As of this writing, EPA has just issued a revision of a required midterm review stating that the 2022–2025 model year standards are not economically or technologically feasible.[186] The expectation is that the administration will roll back those standards in some way.[187] But because of the special CAA authority California has to regulate mobile sources, the state has already recommitted to maintaining the current standards, has been granted a waiver by EPA to do so, and is investing massive resources into electrifying its vehicle fleet.[188] The state also has a regulatory mandate that 22 percent of vehicles be zero-emission by 2025.[189] Twelve states follow California's vehicle standards, covering more than a third of the country's vehicles.[190] Whether EPA will attempt to revoke the California waiver is unclear as of press time.[191]

commitment as part of a long-term strategy to cut U.S. emissions by 80 percent by 2050).

185. *See* U.S. EPA, EPA ANNOUNCES INTENT TO REVISIT PROVISIONS OF PHASE 2 HEAVY-DUTY RULES (Aug. 17, 2017) [hereinafter EPA ANNOUNCES INTENT TO REVISIT PROVISIONS], https://www.epa.gov/newsreleases/epa-announces-intent-revisit-provisions-phase-2-heavy-duty-rules [https://perma.cc/2AAF-JHB3]; U.S. EPA, EPA TO REEXAMINE EMISSIONS STANDARDS FOR CARS AND LIGHT DUTY TRUCKS—MODEL YEARS 2022-2025 (Mar. 15, 2017) [hereinafter EPA to Reexamine Emissions Standards], https://www.epa.gov/newsreleases/epa-reexamine-emission-standards-cars-and-light-duty-trucks-model-years-2022-2025 [https://perma.cc/4SEB-5LGH]; U.S. EPA, EPA, DOT OPEN COMMENT PERIOD ON RECONSIDERATION OF GHG STANDARDS FOR CARS AND TRUCKS (Aug. 10, 2017) [hereinafter EPA, DOT OPEN COMMENT PERIOD], https://www.epa.gov/newsreleases/epa-dot-open-comment-period-reconsideration-ghg-standards-cars-and-light-trucks [https://perma.cc/Q9T8-5VNC] (adding Model Year 2021 standards for reconsideration).

186. EPA Mid-Term Evaluation of Greenhouse Gas Emissions Standards for Model Year 2022–2025 Light-Duty Vehicles, 83 Fed. Reg. 16,077 (April 13, 2018).

187. *See* Hiroko Tabuchi, *Calling Car Pollution Standards 'Too High,' E.P.A. Sets Up Fight With California,* N.Y. Times (April 2, 2018), https://www.nytimes.com/2018/04/02/climate/trump-auto-emissions-rules.html.

188. *See* Press Release, CARB Finds Vehicle Standards are Achievable and Cost-Effective, Cal. Air Res. Bd. (Mar. 24, 2017), https://www.arb.ca.gov/newsrel/newsrelease.php?id=908 [https://perma.cc/P6JR-EMPU] (describing state re-affirmation of vehicle standards); GOVERNOR'S INTERAGENCY WORKING GRP. ON ZERO-EMISSION VEHICLES, 2016 ZEV ACTION PLAN: AN UPDATED ROADMAP TOWARD 1.5 MILLION ZERO-EMISSION VEHICLES ON CALIFORNIA ROADWAYS BY 2025 (2016) (outlining state programs).

189. CAL. CODE REGS. Tit 13, § 1962.2(b)(1)(a) (2017).

190. *See* CARB Finds Vehicle Standards Are Achievable and Cost-Effective, *supra* note 188.

191. *See* Tabuchi, *supra* note 187.

The current regulatory environment at the federal level is, to say the least, dispiriting with respect to the regulation of greenhouse gases. Moreover, technological challenges remain in electrifying or moving to zero-emissions engines, particularly with respect to the electrification of heavy-duty engines for long-distance travel at reasonable cost.[192] Before long-distance trucks are fully electrified, we may be more likely to see hybrid trucks and improved diesel efficiency,[193] particularly if EPA and/or California use their authority to require additional improvements. Nevertheless, over the long haul, EPA and California collectively have the tools to address the biggest culprit in near-source exposure. The effort will take decades, and the problem of existing engine pollution will remain long after regulations are adopted for new engines. But the problem of climate change may ultimately provide us with the solution to near-road pollution. In fact, the significant and immediate health benefits that would result from dramatically eliminating or reducing entirely the biggest hotspot problem, near-road pollution, could be a more effective and persuasive way, rather than relying on arguments about climate change, to convince the public and its representatives that we should transition away from a fossil-fuel dominated transportation sector.

2. The Power Sector's Transition to Renewable Fuels Will Help the Stationary Source Problem

The Clean Air Act can also assist with the transition of the electricity sector from fossil fuels to renewable energy sources, and market forces in the sector, as well as state policies, are also helping.[194] As with GHG regulations for mobile

192. *See, e.g.*, Peter Fairley, *Can Tractor-Trailers Go Electric Along With Cars?* MIT TECH. REV. (July 29, 2015), https://www.technologyreview.com/s/539741/can-tractor-trailers-go-electric-along-with-cars [https://perma.cc/VX2F-2SAE].

193. *See id.*

194. Twenty-nine states currently have Renewable Portfolio Standards that require their utilities to procure a certain percentage of energy from renewable sources. *See* Jocelyn Durkay, *State Renewable Portfolio Standards and Goals*, NAT'L CONF. ST. LEGISLATURES (Aug. 1, 2017), http://www.ncsl.org/research/energy/renewable-portfolio-standards.aspx [https://perma.cc/AU69-4ZMT]. 2016 saw record renewable energy installations around the world, fueled in large measure by dropping prices. *See* Damian Carrington, *'Spectacular' Drop in Renewable Energy Costs Leads to Record Global Boost*, GUARDIAN, (June 6, 2017, 6:30 PM), https://www.theguardian.com/environment/2017/jun/06/spectacular-drop-in-renewable-energy-costs-leads-to-record-global-boost [https://perma.cc/8GKQ-V74D].

sources, however, the Trump Administration is rolling back CAA regulations for the power sector.[195]

Once EPA made its finding in 2009 that greenhouse gases endanger public health and welfare, the finding set off a cascade of regulatory activity focused on different sources of greenhouse gases.[196] The Obama Administration's Clean Power Plan (CPP) included regulations for new and existing electric generating units.[197] The CPP would, if implemented, cut GHG from the power sector by more than 30 percent below 2005 levels.[198] Importantly, the cuts were based in part on assumptions that the power sector could increase its reliance on renewable, as opposed to fossil, fuels.[199] The regulations would, in other words, begin to transition away from many of the stationary sources that can cause near-source pollution. Though its implementation is highly unlikely given that the Trump Administration has begun proceedings to withdraw the rule, the point is that the CAA in its current form contains mechanisms to reduce greenhouse gas emissions from stationary sources—mechanisms that could, in turn, reduce particulate and NO2 pollution that cause human health problems.[200] Indeed, EPA estimated that the CPP would prevent between 1400 and 3200 premature deaths and 1700 heart attacks, largely from reductions in PM 2.5 and ozone.[201]

195. Review of the Clean Power Plan, 82 Fed. Reg. 16,329 (proposed Apr. 4, 2017) (to be codified at 40 C.F.R. pt. 60) (notice of review of Clean Power Plan (CPP)).

196. For an explanation of this regulatory cascade, see Carlson, *supra* note 180.

197. For a snapshot of EPA's website containing extensive materials about the CPP, including an in-depth description and regulatory materials, see U.S. EPA, OVERVIEW OF THE CLEAN POWER PLAN, CUTTING CARBON POLLUTION FROM POWER PLANTS, https://19january2017snapshot.epa.gov/cleanpowerplan/fact-sheet-overview-clean-power-plan_.html [https://perma.cc/L3HP-ECCW]. The EPA materials about the CPP are no longer available from EPA itself. *Id.* Instead, EPA Administrator Scott Pruitt has launched a review of the CPP and has made clear that he intends to withdraw the rule. *See* Review of the Clean Power Plan, 82 Fed. Reg. at 16,329.

198. *See* U.S. EPA, OVERVIEW OF THE CLEAN POWER PLAN, CUTTING CARBON POLLUTION FROM POWER PLANTS, *supra* note 197.

199. *Id.*

200. The legal status of the CPP was, and remains, uncertain. Numerous regulated parties and a coalition of states led by now EPA Administrator Scott Pruitt, who was the Attorney General of Oklahoma, challenged the plan as outside the scope of EPA's statutory authority. This challenge is pending in the U.S. Court of Appeal for the D.C. Circuit, and the case is currently under abeyance.

201. *See* EPA, REGULATORY IMPACT ANALYSIS FOR THE CLEAN POWER PLAN FINAL RULE, 4–31 (2015). A number of policies and sharp drops in the price of solar and wind technology have led to large increases in wind and solar installations over the past decade. Researchers estimate that this shift from conventional to renewable energy sources has resulted in a reduction in premature deaths of between 3000 and 12,700, and an increase in cumulative economic benefits of between $35 billion and more than $200 billion.

B. In the Nearer Term, CAA Contains Some Potential Regulatory Avenues for Attacking Near-Source Pollution

1. Transportation Control Measures

One potential method for addressing near-source roadway pollution is through transportation control measures, such as the management and flow of traffic, the provision of alternative and cleaner forms of transportation, and the dedication of carpool lanes. While these approaches to reducing near-road exposure are indirect, as compared to direct emissions controls on mobile sources, they can in the short run help reduce emissions levels on heavily trafficked roads.[202]

Though the CAA, is in many ways, prescriptive in requiring states to implement provisions that can be quite directive, the Act's treatment of transportation control measures is striking for—the most part—failing to require any significant adoption of such measures.[203] States are not required to include transportation control measures in SIPs except in serious and extreme ozone nonattainment and serious nonattainment carbon monoxide zones (of which there are none) and for transportation conformity.[204] Even in these nonattainment areas, EPA has taken a remarkably narrow approach to what is required.

The Clean Air Act requires serious and severe ozone nonattainment areas "to offset any growth in emissions from growth in vehicle miles traveled . . . and to attain reduction in motor vehicle emissions as necessary . . . ."[205] In 2004, California submitted a SIP revision for the South Coast Air Quality Management plan to attain the ozone standard, a revision that EPA approved

Dev Millstein et al., *The Climate and Air-Quality Benefits of Wind and Solar Power in the United States*, 2 NATURE ENERGY 1, 1 (2017).

202. *See Multi-Pollutant Emissions Benefits of Transportation Strategies-FHWA*, U.S. DEP'T TRANSP. FED. HIGHWAY ADMIN. (Nov. 14, 2006), https://www.fhwa.dot.gov/environment/air_quality/conformity/research/mpe_benefits [https://perma.cc/AQ5R-RPXU].

203. *See Transportation Control Measures*, U.S. DEP'T TRANSP. FED. HIGHWAY SAFETY ADMIN., http://www.fhwa.dot.gov/environment/air_quality/conformity/research/transportation_control_measures [https://perma.cc/DS5Z-ZVDJ] (describing transportation control measures).

204. In nonattainment zones for several of the criteria pollutants, states must ensure that new transportation projects do not interfere with federal air quality goals. For an explanation of this process, see Fed. Highway Admin., *Air Quality, Transportation Conformity*, https://www.fhwa.dot.gov/environment/air_quality/conformity (last visited March 28, 2018).

205. 42 U.S.C. § 7511a(d)(1)(A) (2012).

in 2009.[206] The plan contained no transportation control measures, even though SCAQMD is an extreme nonattainment zone for ozone.[207] California took the position that no transportation control measures were necessary, because, even though vehicle miles traveled (VMTs) would increase, overall emissions would decline as a result of other regulatory efforts. EPA agreed.[208] Plaintiff environmental groups challenged EPA's approval, and the court held that EPA's interpretation of the statutory language requiring transportation control measures was erroneous. Instead, the court found that the text requires transportation control measures if VMTs have grown over a previous baseline year to offset any emissions growth associated with that growth, not merely if overall emissions increase.[209]

Despite EPA's narrow interpretation, transportation control measures could be used if air districts are designated as out of attainment of the new NO2 one-hour standard. Similarly, were EPA to require near-road monitoring for PM 2.5 or establish a new PM 2.5 standard based on near-road pollution, transportation control measures could play a role in reducing exposure. Given that the standard is directed at near-road pollution and that air districts cannot directly regulate mobile source emissions, transportation control measures are, in fact, likely to be a primary means for controlling exposure. Whether air districts and EPA will have the political will to impose such measures is a different matter.[210]

2. Indirect Source Rules To Control New or Modified Sources

The CAA contains another provision that can be used to control mobile source emissions from new or modified sources that are stationary, but the provision is entirely permissive. Section 110(a)(5) authorizes—but does not require—states to adopt indirect source review to include in State Implementation Plans for new or modified "indirect sources" that will attract emissions from mobile sources.[211] Indirect sources are defined to include "a facility, building, structure, installation, real property, road, or highway

206. Ass'n of Irritated Residents v. EPA, 686 F.3d 668, 672–73 (D.C. Cir. 2011).
207. *Id.* at 673.
208. *Id.*
209. *Id.* at 680–81.
210. *See, e.g.*, U.S. GOV'T ACCOUNTING OFFICE, GAO/RCED-93-169, URBAN TRANSPORTATION: REDUCING VEHICLE EMISSIONS WITH TRANSPORTATION CONTROL MEASURES 3 (1993) (describing some TCMs as "politically painful").
211. 42 U.S.C. § 7410(a)(5)(A) (2012).

which attracts, or may attract, mobile sources of pollution."[212] States using this provision can regulate emissions from the indirect source in order to maintain or come into compliance with a NAAQS even when the cause of those emissions is mobile sources (construction equipment, for example).[213] States do need, however, to ensure that they do not impose direct emissions controls on mobile sources that increase indirect source emissions or they risk running afoul of the mobile source preemption provisions.[214]

The indirect source provision appears to be underutilized but has been used by some air districts, including California's Central Valley, to impose limitations on mobile source emissions on construction sites. Like the transportation control measures described above, the indirect source rule applies only to new or modified sources, not existing ones, so cannot alone solve the problem of pollution hot spots. Nevertheless it is another underutilized mechanism that can address hot spots that arise from new development and construction.

3. Creative Interpretations of Existing CAA Provisions to Tackle Near-Road Pollution

EPA might also use its existing regulatory authority creatively to attempt to require states to address near-source and, particularly, near-road pollution.[215] To begin with, the NAAQS provisions themselves require EPA to establish ambient standards in a way that is sufficient to "protect the public health" with "an adequate margin of safety."[216] This language should provide the agency with sufficient authority to establish mechanisms to regulate near-source pollution and arguably could provide a basis for a citizen suit against the agency for failing to do so. One obvious possibility is to create a near-source standard for PM 2.5 similar to the new NO2 standard. The agency could also explore more unusual regulatory measures, such as establishing air districts for a particular pollutant (PM 2.5 is the obvious one) that encompass only the areas near trafficked roadways or determining that highways are stationary sources.

212. Id. § 7410(a)(5)(C).
213. Id. § 7410(a)(5)(D).
214. *See* Nat'l Ass'n of Home Builders v. San Joaquin Valley Unified Air Pollution Control Dist., 627 F.3d 730, 734–35 (9th Cir. 2010) (discussing tension between CAA mobile source preemption provision and indirect source provision and upholding San Joaquin Air District's rule).
215. These suggestions are preliminary in nature and offered merely to suggest that the expansive nature of and language in the CAA might be deployed to address hotspot pollution.
216. 42 U.S.C. § 7409(b)(1) (2012).

These are preliminary suggestions and raise many complexities, but they highlight the fact that the CAA is a remarkably flexible and expansive statute that can evolve as new pollution problems come to light.[217]

4. State Solutions to Near-Source Pollution

Finally, EPA might look to a number of solutions that some states, particularly California, have adopted in an attempt to address near-source pollution. None of these fully fix the problem in the way that a fully electrified vehicle fleet would, but they can nevertheless reduce exposure levels and consequent health effects of breathing in elevated levels of pollutants and serve as transition measures, as the vehicle fleet turns over and moves toward zero emissions. And of course, other than preemption provisions in the Clean Air Act for new engine emissions standards, nothing prohibits states from attempting to address the near-source problems voluntarily.

Most importantly, California now requires the retrofitting of existing heavy-duty diesel engines and the eventual replacement of old diesel trucks with new technology.[218] Some of this regulatory activity stems from California's special authority to regulate emissions under the Clean Air Act[219] but the requirement that older heavy-duty trucks install engine filters could be done by other states.[220] Other states can also choose to follow new California emissions standards.[221] Though California does not control emissions from all trucks that enter the state, it is still regulating a substantial portion of truck traffic that contributes to near-road emissions.

California's two largest ports, Los Angeles and Long Beach, have also adopted extensive regulatory programs to address port emissions, primarily from mobile sources with diesel engines, including heavy trucks, drayage, and even ocean vessels.[222] The mayors of the two cities that house the ports just

217. For an evaluation of many of the ways in which the CAA has proved adaptable, flexible, and durable, see THE FUTURE OF LONG-TERM ENERGY POLICY: LESSONS FROM THE CLEAN AIR ACT (Dallas Burtraw & Ann E. Carlson eds., forthcoming 2018).

218. *Truck and Bus Regulation: On-Road Heavy-Duty Diesel Vehicles (In-Use) Regulation*, CAL. AIR RES. BD., https://www.arb.ca.gov/msprog/onrdiesel/onrdiesel.htm [https://perma.cc/4FE3-AMS2] (last reviewed Dec. 14, 2017).

219. *See* 42 U.S.C. § 7543(b) (2012).

220. The preemption of state emissions standards for motor vehicles applies to new vehicles. *See id.* § 7543(a).

221. *Id.* § 7507.

222. *See San Pedro Bay Ports Clean Air Action Plan (CAAP)*, PORT L.A., https://www.portoflosangeles.org/environment/caap.asp [https://perma.cc/UYG3-7U5X].

committed to zero emissions from cargo-handling equipment by 2030 and from drayage trucks by 2035.[223]

California is also embarking on a new monitoring program to obtain better data about near-source exposure in disadvantaged communities and to install control measures on sources that are contributing to elevated near-source levels.[224]

Finally, California and some of its localities have restricted some land uses near freeways, including elementary schools,[225] and have required air filters in housing located near major highways.[226] Nevertheless, there are no barriers to the construction of new housing near freeways and a recent study showed that Los Angeles County has 169 child care centers—which, unlike schools, can be built near freeways—located within 500 feet of a freeway.[227]

EPA could look to some of these state solutions, particularly those involving the retrofitting of old heavy-duty engines, as measures available to other states with near-road pollution problems. To do so, however, EPA would need to take other steps outlined above to identify and require the regulation of near-source pollution in order to shoehorn the problem of near-road exposure into the existing statutory structure.

None of these regulatory measures is a magic bullet to solve the microclimate pollution problem. One lesson from the many years of Clean Air Act implementation, however, is that the process of cleaning the air has involved relying on numerous statutory provisions, adopting many iterations of regulations, and engaging in sustained effort to identify and regulate new pollution problems.[228] A legislative solution to the hotspot pollution problem

223. *See* Joint Declaration, Eric Garcetti & Robert Garcia, *Creating a Zero Emissions Goods Movement Future: A Joint Declaration of the Mayors of the Cities of Los Angeles and Long Beach*, https://www.documentcloud.org/documents/3864912-Joint-declaration-of-L-A-Long-Beach-mayors-on.html.

224. *See* Cal. Air Res. Bd., Community Air Protection Program, AB 617 (last viewed March 28, 2018).

225. CAL. PUB. RES. CODE § 21151.8(a)(1)(D) (2009).

226. *See* Tony Barboza, *L.A. Requires Air Filters to Protect Residents Near Freeways. Are They Doing Their Job?*, L.A. TIMES (July 9, 2017, 5:00 AM), http://www.latimes.com/local/lanow/la-me-ln-freeway-pollution-filters-20170709-story.html [https://perma.cc/NLX3-JR6R] (suggesting that the requirement is not well-enforced and the systems often not well-maintained).

227. *See* Deepa Fernandes with Aaron Mendelson, *Polluted Preschools: 169 LA Childcare Centers Are Too Close to Freeways*, 89.3 KPCC, (Mar. 29, 2016), http://www.scpr.org/ news/2016/03/29/58878/pollution-near-preschools-is-impacting-nearly-10-0 [https://perma.cc/5HQY-W87W].

228. For an in-depth analysis of the multiple iterations of passenger vehicle regulations in California and at the federal level, see generally Ann E. Carlson, *Iterative Federalism and*

might well be preferable, but, in the current legislative climate, reform is highly unlikely.

CONCLUSION

The very successful ambient structure of the Clean Air Act may, ironically, also help to mask a ubiquitous and harmful form of pollution: hotspots of emissions in microclimates. Indeed, by requiring air districts around the country to measure, monitor, and regulate pollutants on a large, background scale, the CAA may actually lead residents to misunderstand the health risks they face from the air they breathe. In some instances, background ambient air may actually be cleaner than the labels some air districts receive when out of attainment—this is the case for large parts of Los Angeles that have cleaner ambient air than the air measured by monitors in the dirtiest parts of the basin. But much more troubling, the imprimatur of clean air in many districts may also lead residents to believe the air they breathe is of high quality even when living or playing or working in a microclimate with predictably unhealthful air. These measurements, monitors, and labels may even lull regulators into believing their jobs are done when many of their residents—often the lowest income and disproportionately of color—face unhealthful conditions during many parts of the day. The microclimates that remain largely (though not entirely) unregulated are, in my view, the blindspot of the CAA. This Article is an effort to make us see what the ambient focus of the Act has kept hidden.

Climate Change, 103 NW. U. L. REV. 1097 (2009); *see also* THE FUTURE OF LONG-TERM ENERGY POLICY: LESSONS FROM THE CLEAN AIR ACT, *supra* note 217.

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Attachment A14

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ASSOCIATION BETWEEN PARTICULATE- AND GAS-PHASE COMPONENTS OF URBAN AIR POLLUTION AND DAILY MORTALITY IN EIGHT CANADIAN CITIES

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Although some consensus has emerged among the scientific and regulatory communities that the urban ambient atmospheric mix of combustion related pollutants is a determinant of population health, the relative toxicity of the chemical and physical components of this complex mixture remains unclear. Daily mortality rates and concurrent data on size-fractionated particulate mass and gaseous pollutants were obtained in eight of Canada's

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largest cities from 1986 to 1996 inclusive in order to examine the relative toxicity of the components of the mixture of ambient air pollutants to which Canadians are exposed. Positive and statistically significant associations were observed between daily variations in both gas- and particulate-phase pollution and daily fluctuations in mortality rates. The association between air pollution and mortality could not be explained by temporal variation in either mortality rates or weather factors. Fine particulate mass (less than 2.5 μm in average aerometric diameter) was a stronger predictor of mortality than coarse mass (between 2.5 and 10 μm). Size-fractionated particulate mass explained 28% of the total health effect of the mixture, with the remaining effects accounted for by the gases. Forty-seven elemental concentrations were obtained for the fine and coarse fraction using nondestructive x-ray fluorescence techniques. Sulfate concentrations were obtained by ion chromatography. Sulfate ion, iron, nickel, and zinc from the fine fraction were most strongly associated with mortality. The total effect of these four components was greater than that for fine mass alone, suggesting that the characteristics of the complex chemical mixture in the fine fraction may be a better predictor of mortality than mass alone. However, the variation in the effects of the constituents of the fine fraction between cities was greater than the variation in the mass effect, implying that there are additional toxic components of fine particulate matter not examined in this study whose concentrations and effects vary between locations. One of these components, carbon, represents half the mass of fine particulate matter. We recommend that measurements of elemental and organic carbon be undertaken in Canadian urban environments to examine their potential effects on human health.

Historically, extreme air pollution events, such as those experienced in London in the 1950s and 1960s, clearly demonstrated the potential of ambient air pollution to exacerbate cardiorespiratory disease, as reflected in premature mortality and increased hospital admissions. In the intervening years, considerable effort has been made to reduce atmospheric pollution from the combustion of fossil fuels. Several countries, including Canada and the United States, have established stringent new guidelines and standards for air pollutants such as sulfur dioxide, nitrogen dioxide, carbon monoxide, ozone, and particulate matter. At present, the Canadian National Ambient Air Quality Objectives for these pollutants are rarely violated.

Over the last decade, a series of studies has been published linking daily variations in either deaths or admissions to hospital for cardiorespiratory diseases and daily fluctuations in a number of ambient air pollutants (U.S. EPA, 1996). Although the majority of studies have focused on U.S. and European locations with much higher air pollution levels than normally experienced in Canada, some of the most convincing evidence linking air pollution to health has been obtained from data collected in Canada.

Following Bates and Sitzo's (1983, 1987) initial work, summertime concentrations of both ozone and particulate matter have been linked to respiratory hospitalizations in southern Ontario (Burnett et al., 1994), Toronto, Ontario (Burnett et al., 1997a; Thurston et al., 1994), and in 16 of Canada's largest cities (Burnett et al., 1997b). Summertime ozone levels have also been associated with visits to the emergency department in the Saint John Regional Hospital for patients presenting with asthma (Stieb et al., 1996) and to emergency-department visits for respiratory diseases in Montreal (Delfino et al., 1996). Elevated ambient levels of carbon monoxide have been linked to hospitaliza-

tions for respiratory (Burnett et al., 1997b) and cardiac (Burnett et al., 1997c) diseases in several Canadian cities. Goldberg and collaborators (2000), in a study to identify susceptible populations, found that increased levels of daily particles in Montreal were associated with daily increases in nonaccidental mortality among persons who had cancer, acute lower respiratory diseases, any form of cardiovascular disease, chronic coronary artery diseases, or congestive heart failure. Particulate sulfates were related to respiratory hospitalizations in southern Ontario (Burnett et al., 1994, 1995), and the coefficient of haze was linked to increases in respiratory hospitalizations in 16 cities spanning the breadth of the country (Burnett et al., 1997b). Particulate matter and carbon monoxide were also associated with daily mortality in Toronto, Canada, over the 15-year period 1980 to 1994 (Burnett et al., 1998a).

Gas-phase ambient air pollution was linked to increases in daily mortality rates in 11 Canadian cities (Burnett et al., 1998b). However, particulate mass measurements were not considered in the analysis. Daily variations in particulate mass have been linked to daily variations in nonaccidental mortality rates in a number of locations worldwide (U.S. EPA, 1996). The purpose of this investigation is, for the first time, to examine the association between constituents (mass, elements, and ions) of size-fractionated particulate matter and daily mortality rates, controlling for weather factors, temporal trends, and gaseous copollutants in Canadian urban environments.

METHODS

Environmental Data

Fine ($PM_{2.5}$), coarse ($PM_{10\text{-}2.5}$), and thoracic (PM_{10}) measurements were obtained from dichotomous samplers with Teflon filters operating on a 6-day schedule in 8 Canadian cities (Montreal, Ottawa, Toronto, Windsor, Winnipeg, Calgary, Edmonton, and Vancouver) from 1986 to 1996 inclusive. Locations of the cities are displayed in Figure 1. This database had been described in detail elsewhere (Brook et al., 1997). Forty-seven elemental concentrations were obtained for the fine and coarse fraction using nondestructive x-ray fluorescence techniques. Sulfate concentrations were obtained by ion chromatography.

For each sample, blanks were analyzed in order to identify a limit of chemical detection. If a reading was below the limit of detection for that sample but was positive, we included that data point in the analysis. If a zero reading was recorded, then a value equal to one-half the sample-specific detection limit was used in the analysis.

Each city had one dichotomous sampler operating at any given point in time, except for Montreal and Vancouver, which had two samplers. For these latter two cities, we averaged the available daily data between the two stations to form a single time series for analysis.

Daily average concentrations of nitrogen dioxide (NO_2), carbon monoxide (CO), sulfur dioxide (SO_2), and the coefficient of haze (COH), as well as the daily 1-h maximum concentration of ozone (O_3), were obtained for each

FIGURE 1. Location of study communities.

monitoring station located in a specified geographic area for each city. Data from a monitoring station were included in the study if at least two-thirds of the days of observation had measured concentrations. We selected this inclusion criterion in order to minimize any potential confounding effects on the association between air pollution concentrations and mortality due to differential temporal trends in pollution levels between monitoring stations within a city. Details on the number and location of monitoring stations in addition to the availability of data are given in Table 1. The areas were defined in terms of census subdivision boundaries (Table 2). We made one exception to this decision rule in Winnipeg, which maintained two stations monitoring SO_2 from 1986 to 1991. In order to have some SO_2 data for Winnipeg, we included the time series for this city in our analysis. Edmonton did not maintain

TABLE 1. Monitoring Station by Census Subdivision (CSD), Pollutants Monitored, and Indication of Missing Data (M) or Years With Low Numbers (L) of Daily Monitored Observations (< 100/yr)

City	Station number	CSD	Pollutants monitored	Comments
Montreal	50102	66025	O_3, NO_2, CO, SO_2	CO (L '88), NO_2 (L '89)
	50103	66025	O_3, NO_2, CO, SO_2	NO_2 (M '89–'91, L '92)
	50104	66025	O_3, NO_2, CO, SO_2, PM	CO (L '86), NO_2 (L '87), SO_2 (L '96), PM (M '91)
	50109	66070	O_3, NO_2, CO, SO_2, COH, PM	O_3, NO_2, CO, SO_2, COH (L '86), SO_2 (L '91), PM (M '90, '91, '96)
	50110	66020	O_3, NO_2, CO, SO_2, COH	SO_2 (L '89, M '96), NO_2 (L '91)
	50115	66025	O_3, NO_2, CO, SO_2, COH	
	50116	66025	O_3, NO_2, CO, SO_2, COH	
	50120	66140	O_3, NO_2, CO, SO_2, COH	No data '86, '87, CO (L '89, M '96), NO_2 (L '91, '92), SO_2 (L '89, M '96)
Ottawa-Hull	50203	81020	NO_2, CO, SO_2	No data '95, '96
	60101	06014	O_3, NO_2, CO, SO_2	No data '91, SO_2 (M '95, '96)
	60104	06014	O_3, NO_2, CO, SO_2, COH, PM	
Toronto	60403	20019	O_3, NO_2, CO, SO_2, COH, PM	PM (M '86–'94)
	60410	20001	O_3, NO_2, CO, SO_2, COH	
	60413	20019	O_3, NO_2, CO, SO_2,	
	60415	21005	O_3, NO_2, CO, SO_2	NO_2, CO, SO_2 (M '95, '96)
	60417/ 60424	20004	O_3, NO_2, CO, SO_2, COH, PM	L '90, PM (M '90, '91)–station moved in '90 from 60417 to 60424
	60418	20004	O_3, NO_2	No data '95, '96
	60419	20004	O_3, NO_2	No data '94, '96
	60421	20008	O_3, NO_2	No data '86, '87
	60423	20008	O_3, NO_2	No data '86, '87
Windsor	60204	37039	O_3, NO_2, CO, SO_2, COH, PM	PM (M '86, '95, '96)
	60211	37039	SO_2, COH, PM	PM (M '86–'89)
	60212	37039	SO_2	
Winnipeg	70118	11040	O_3, NO_2, CO, SO_2, COH	SO_2 (M '92–'96)
	70119	11040	O_3, NO_2, CO, SO_2, COH, PM	SO_2 (M '92–'96)
Edmonton	90122	11061	O_3, NO_2, CO, COH	
	90130	11061	O_3, NO_2, CO, COH, PM	
Calgary	90218	06016	SO_2, COH	
	90222	06016	O_3, NO_2, CO, COH	
	90227	06016	O_3, NO_2, CO, COH, PM	PM (M '86)
	90204	06016	PM	PM (M '88–'96)
Vancouver	100110	15025	O_3, NO_2, CO, SO_2, COH	
	100111	15043	O_3, NO_2, CO, SO_2, COH, PM	
	100112	15022	O_3, NO_2, CO, SO_2, COH	
	100118	15022	O_3, NO_2, CO, SO_2, PM	All pollutants (L '86)
	100120	15025	O_3, NO_2	
	100121	15056	O_3, NO_2	NO_2 (L '87)
	100122	15038	O_3, NO_2	
	100124	15043	O_3, NO_2	
	100125	15011	O_3, NO_2	NO_2 (M '86)
	100126	15025	O_3, NO_2	
	100127	15004	O_3, NO_2	
	100128	15015	O_3, NO_2	
	100129	15039	O_3	No data '86

TABLE 2. Selected Characteristics of the Eight Canadian Cities Including Census Subdivision Code, 1991 Population, Percentage of Census Metropolitan Area (CMA) Selected, and Daily Average Nonaccidental Mortality Rate by City

City	Census subdivision	1991 population	% CMA selected	Deaths/day
Montreal	All of Census Division 66	1,775,871	55	38.8
Ottawa-Hull	81015, 81020, 81025, 06006, 06009, 06011, 06012, 06014	728,789	78	11.7
Toronto	20001, 20004, 20006, 20008, 20014, 20019, 21005	2,739,159	70	46.5
Windsor	37039	191,435	73	4.5
Winnipeg	11040	615,215	93	12.6
Calgary	06016	710,795	94	8.5
Edmonton	11061	616,741	73	8.7
Vancouver	15004, 15011, 15015, 15018, 15022, 15025, 15029, 15034, 15038, 15039, 15043, 15051, 15803	1,318,693	82	22.1

any SO_2 stations within the selected census subdivision boundaries. Daily average or daily 1-h maximum values were averaged among available data from all stations in each city to form a single time series for analysis.

Data on daily average temperature, daily average relative humidity, and maximum change in barometric pressure within a day (a measure of frontal activity) were obtained from airports within or near each city.

Mortality Data

We abstracted the number of persons who died from a nonaccidental underlying cause (International Classification of Disease 9th Revision ICD9: <800) on each day from 1 January 1986 to 31 December 1996 and who lived and died in any one of the specified census subdivisions. Deaths were then aggregated at the city level.

Statistical Methods

Adjusting for Temporal Variation The objective of our analysis was to relate daily variations in air pollution levels to daily variations in mortality rates on a temporal basis. There are, however, a number of other factors influencing temporal variation in both deaths and air pollution. More people die in winter than summer. This is likely, in part, due to increased infection in those months in which people spend a greater amount of time indoors (Leech et al., 1996). The average daily number of deaths per city varies from 19.1 to 19.4 from Monday to Saturday, with a lower rate observed on Sunday (18.8 deaths/day). Air pollution levels also vary by season and day of the week. Finally, weather factors, such as temperature and humidity, are predictors of both deaths and air pollution.

We did not wish to attribute variation in mortality to variation in air pollution if the variation in mortality was due, in whole or in part, to common,

but possibly unrelated, temporal cycles and weather factors. We therefore adjusted the time series of the daily number of deaths for temporal trends using a LOESS nonparametric smoothed representation of day of study (Cleveland & Devlin, 1988) and for day-of-the-week effects for each city separately. Bartlett's test for "white noise" and examination of the autocorrelation functions were used to assess serial correlation in the data (Priestly, 1981). The serial correlation structure in the residuals after fitting models with LOESS smoothers was examined. We considered LOESS smooth functions of day of study of length 30, 90, and 150 days.

Determining the Weather Model We also adjusted the mortality series for the influences of weather. The weather variables were adjusted for temporal trends using the LOESS smoother of day of study and dummy variables for day-of-the-week effects. The temporally adjusted effects of weather on mortality were modeled using spline-smoothed functions of daily average temperature, daily average relative humidity, and maximum change in barometric pressure within a day, recorded on the date of death and one day prior to death. In order to identify the smallest number of weather variables required to predict deaths, we used a forward inclusion stepwise regression procedure to select a minimally sufficient set of weather variables needed to statistically predict daily variations in mortality rates. Akaike's Information Criterion (AIC) is a linear function of the residual deviance and the model degrees of freedom and was used to identify important predictors of mortality. We found that daily average temperature and maximum change in barometric pressure recorded on the day of death were sufficient to predict mortality among the variables considered.

The Regression Model We assumed a common form of the relationship between air pollution or weather and mortality among cities and a common effect of air pollution and weather. The city-specific temporally adjusted time series were pooled with parameter estimates obtained from a single model based on data from all cities combined. This approach was adopted due to the limited number of particulate observations in each city. Temporally adjusting all time series prior to relating them was done in order to remove unwanted cycles in the data at a yearly and seasonal time scale that might confound the association among the variables at the daily time scale that we are interesting in detecting.

The air pollution, weather, and mortality time series were adjusted for temporal trends using a LOESS smoother of day of study and dummy variables for day of week. The filtered time series were then related using the model

$$E(y_{tj}) = f_{tj}^{(y)} \exp\{\beta^{T}(\mathbf{x}_{tj} - f_{tj}^{(x)}) + \gamma^{T} s(\omega_{tj} - f_{tj}^{(w)})\}$$

where $E(y_{tj})$ is the expected number of deaths on day t in the jth city; exp is the exponential function; β^{T} is the transpose of the vector of unknown parameters relating the vector of temporally filtered air pollution values on day t of the jth city to temporally filtered deaths; $\mathbf{x}_{tj}$ is the vector of air pollution

values; γ^T is the transpose of the vector of unknown parameters relating the spline smoothed function, $s(\bullet)$, of the temporally filtered weather variables to temporally filtered deaths; and w_{tj} is a vector of weather factors. Here, $f_{tj}^{(y)}$ ($f_{tj}^{(x)}$; $f_{tj}^{(w)}$), are the predicted values of daily deaths (air pollution; weather) as obtained from the generalized additive model with temporal trends and day-of-the-week effects. We assumed that the residual variance was proportional to the expected response, thus accommodating over- (or under-)dispersion relative to Poisson variation.

We used generalize additive models, as implemented in S-PLUS (Statistical Sciences, Inc., 1993), to model the data, thereby obtaining relative excess daily mortality rates expressed for a unit change in air pollution, β, as well as their corresponding standard errors corrected for non-Poisson variation. We report the percentage increase in daily numbers of deaths corresponding to the mean air pollution concentration as well as the ratio of the parameter estimate to its standard error (T value).

Using Principal Component Analysis for Multipollution Regression Models Unstable parameter estimates may be obtained in multipollutant models due to the positive correlation between pollutants in the urban atmosphere. Consequently, we estimate air pollution effects within the urban air pollution mixture using principal components (PC) regression analysis methods with the varimax rotation (SAS Institute, Inc., 1989). We first obtained orthogonal linear combinations (factors) of the prefiltered air pollution variables. With the varimax rotation option, each factor explains a similar amount of the variation in pollutant concentrations. All factors were regressed against temporally prefiltered daily number of deaths simultaneously, adjusting for prefiltered weather variables using the generalized additive models described earlier. Parameter estimates associated with these factors are nearly uncorrelated.

We used the following procedure to translate the results from the principal component regression analysis to estimates of the percent increase in mortality associated with an increase in each pollutant, as evaluated at their mean concentrations. First, the factor loadings (coefficients of the linear combinations of variables for each factor) were normalized such that the loadings for each factor summed to unity. Second, the regression coefficients for each factor were normalized so that they also summed to unity. Then the matrix of normalized factor regression coefficients was multiplied by the vector of normalized regression coefficients of the factors to obtain the percentage contribution for each pollutant to the total effect on mortality. The total effect of the mixture on mortality was determined by multiplying the regression coefficients, obtained from a model including all pollutants, times the corresponding mean value, and summing among pollutants. The effect of each pollutant on mortality was then determined by multiplying the proportion of the effect of the mixture attributable to each pollutant by the total effect of the mixture. Standard errors of these estimates were derived using the standard errors of the factor regression coefficients.

RESULTS

Summary Information

City-specific daily mortality rates are given in Table 2, along with the 1991 population and the percentage of the Census Metropolitan Area (CMA) selected for study in each city. For most cities, 70–80% of the population in the CMA was selected. The selection criterion was based on proximity of the population to a monitoring station. Only 55% of the population in the Montreal CMA (the island of Montreal) was selected, due to limited monitoring data outside the island.

The percentage of missing values for the gaseous pollutants, their distribution, and the number of monitoring stations used in the analysis are given in Table 3 by city, based on the combined air pollution data among stations within a city. In general, there were only a few missing days among the total of 4018 possible days of observation per city. Exceptions were for measurements of SO_2 in Winnipeg (45.6% missing) and Edmonton (100% missing). There were far fewer particulate measurements due to the 6-day sampling schedule.

Windsor had the highest average concentrations for ozone, nitrogen dioxide, sulfur dioxide, and fine particulate mass, with Winnipeg having generally the lowest concentrations for these pollutants.

Temporal Filtering

To select the length of a temporal filtered, we stipulated that the residual time series should be consistent with a white noise process. The 90-day span for day of study did indeed produce such a residual in all cities except Montreal and Windsor. Examination of the time series in Montreal revealed 4 days with very high numbers of deaths (12 July 1987 with 80 deaths; 13 July 1987 with 94 deaths; 17 June 1994 with 95 deaths; and 18 June 1994 with 82 deaths). Removal of these 4 days was sufficient to produce a residual time series consistent with white noise ($p > .05$, Bartlett's test). When the 30-day span was used, we found negative serial correlations for time lags up to 7 days, ranging from −0.05 to −0.10, and the resultant time series was significantly different ($p < .001$) from the expected white noise series. For the 150-day span, the white noise assumption was rejected for Montreal and Toronto ($p < .05$), but not for the other cities. There was some evidence to reject the hypothesis of white noise in Windsor for the 90-day span ($p = .01$). Here, a negative serial correlation of −0.05 at a lag of 4 days was observed. We therefore selected a span of 90 days for the main analysis, as there was little evidence to reject the hypothesis of white noise in 7 out of 8 cities.

Correlation Among Pollutants

Ozone was weakly correlated with the other air pollutants (Table 4), and the other pollutants were moderately correlated with each other. The highest correlation was observed for NO_2 and CO ($r = .65$). The gases were more

TABLE 3. Summary Statistics for Gas- and Particulate-Phase Pollution by City

		City								
Gas/PM (units)	Statistic	Montreal	Ottawa	Toronto	Windsor	Winnipeg	Edmonton	Calgary	Vancouver	All cities
O_3 (ppb)	% Miss	0.0	0.4	0.0	2.2	1.5	0.1	0.0	0.0	0.5
	Mean	28	28	36	36	31	32	34	27	31
	CV	50	44	51	60	39	42	34	43	48
	95th Perc	54	49	72	78	53	55	53	46	59
	Max	116	93	163	159	99	89	89	114	163
	# Mon	8	2	9	1	2	2	2	12	38
NO_2 (ppb)	% Miss	0.4	0.5	0.0	4.6	1.1	0.1	0.0	0.0	0.8
	Mean	22	20	25	26	15	25	26	20	22
	CV	38	42	32	38	46	38	34	30	40
	95th Perc	37	35	38	43	29	43	41	31	38
	Max	80	69	72	134	56	72	77	51	134
	# Mon	8	3	9	1	2	2	2	12	39
CO (ppm)	% Miss	0.3	0.0	0.0	2.6	0.8	0.1	0.0	0.0	0.5
	Mean	0.6	0.8	1.0	0.9	0.6	1.2	1.0	1.1	0.9
	CV	65	52	37	51	42	60	62	51	59
	95th Perc	1.4	1.6	1.6	1.6	1.1	2.7	2.1	2.2	1.8
	Max	4.6	3.3	4.2	4.0	3.0	6.3	7.2	4.5	7.2
	# Mon	8	3	5	1	2	2	2	4	27

SO_2 (ppb)	% Miss	0.3	0.8	0.0	0.1	45.6	100	2.4	0.0	18.6
	Mean	5.6	3.8	5.0	7.3	1.2	NA	3.7	5.1	4.7
	CV	80	88	81	58	107	NA	66	58	81
	95th Perc	15	10	13	15	4	NA	8	11	12
	Max	35	34	30	27	27	NA	23	24	35
	# Mon	8	3	5	3	2	0	1	4	26
$PM_{2.5}$ (μg/m^3)	No. obs.	863	526	900	851	554	571	599	683	5547
	Mean	15.0	11.6	15.4	17.7	9.5	9.9	10.3	12.7	13.3
	CV	69	74	67	64	71	72	69	57	71
	95th Perc	34	29	36	40	21	24	25	29	32
	Max	72	54	71	86	71	56	52	43	86
$PM_{10\text{-}2.5}$ (μg/m^3)	No. obs.	854	434	889	851	541	508	598	565	5240
	Mean	12.4	9.2	10.9	13.4	16.8	14.0	15.2	8.9	12.6
	CV	66	70	60	65	76	73	68	58	72
	95th Perc	29	21	23	29	38	35	36	18	30
	Max	82	52	68	99	78	73	84	35	99
PM_{10} (μg/m^3)	No. obs.	853	433	889	850	538	508	598	565	5234
	Mean	27.3	20.4	26.4	31.0	26.2	23.4	25.5	21.5	25.9
	CV	59	61	56	54	62	57	58	52	59
	95th Perc	60	42	53	63	56	50	55	44	54
	Max	121	76	102	110	112	86	114	63	121

Note. % Miss, percentage of missing observations of a possible 4018; CV, coefficient of variation × 100; 95th Perc, 95th percentile; Max, maximum concentration; # Mon, number of monitoring sites; No. obs., number of observations.

TABLE 4. Correlations (× 100) of Temporally Filtered Gas- and Particulate-Phase Pollution

Pollutant	O_3	NO_2	CO	SO_2	$PM_{2.5}$	$PM_{10\text{-}2.5}$	PM_{10}
O_3	1	12	−5	8	29	23	32
NO_2		1	65	49	53	34	53
CO			1	42	44	29	45
SO_2				1	40	25	40
$PM_{2.5}$					1	37	84
$PM_{10\text{-}2.5}$						1	81
PM_{10}							1

strongly associated with the fine fraction than coarse particulate mass (Table 4). Correlations among pollutants were relatively stable among cities. For example, the correlation between concentrations of CO and NO_2 ranged from .61 to .73, while the correlation between $PM_{2.5}$ and SO_4 ranged from .55 to .80. The city-specific correlations between O_3 and the other gases were also low ($r < .20$). The correlation between $PM_{2.5}$ and $PM_{10\text{-}2.5}$ was also highly stable among cities ($.34 < r < .43$).

Modifying Effects of Gaseous Pollutants on Particulate–Mortality Association

We next considered the association between particulate pollution and mortality after controlling for the gaseous pollutants. For this analysis, only days on which there were measurements of PM_{10} were considered. Here, the gas and particulate air pollution data were filtered with a 90-day LOESS smoother for day of study and adjusted for day-of-the-week effects.

The percent increases in mortality associated with increases in air pollutants evaluated at their mean concentrations are given in Table 5 for time lags of 0 and 1 days. Time lags of 2 to 5 days were also examined. The strongest association with mortality for all pollutants considered were for lags of 0 or 1 days. The effects for $PM_{2.5}$, $PM_{10\text{-}2.5}$, and PM_{10} were adjusted for each of the four gases separately. At lag 0, the effect of O_3 was insensitive to adjustment for the mass measurements. However, the effects at lag 0 of NO_2, SO_2, and CO were explained by the mass measurements. In turn, much of the fine mass effect could be explained by O_3. For lag 1, the ozone effect was reduced by half following adjustment for fine mass. In addition, we found effects reduced by half for fine mass after adjusting for NO_2. The effects of CO and SO_2 were also reduced by half after adjustment for fine mass. Adjusting for the coarse fraction had less of a modifying effect on the gases than fine mass. Adjusting for PM_{10} had a modifying effect on the gases similar to adjusting for fine mass.

The simultaneous effect of $PM_{2.5}$, $PM_{10\text{-}2.5}$, and the four gases on mortality was examined using principal component analysis (see Table 8, Model I). The effects of each of the pollutants were reduced as compared to the effects of the pollutants examined separately. The combined effect of $PM_{2.5}$ and $PM_{10\text{-}2.5}$ (1.6%) was less than the combined effect of the four gases (4.1%),

TABLE 5. Percent Increase in Temporally Filtered Daily Nonaccidental Deaths Associated With Increases in Particulate- and Gas-Phase Pollutants Evaluated at Their Study Mean Adjusted for Particulate Phase Pollutants, Temperature, and Change in Barometric Pressure for Those Days in Which There Were PM_{10} Measurements Available

		Lag 0		Lag 1	
Gas	PM	Gas	PM	Gas	PM
None	$PM_{2.5}$	NA	1.2 (2.3)	NA	1.6 (3.1)
	$PM_{10\text{-}2.5}$	NA	0.6 (1.0)	NA	0.9 (1.4)
	PM_{10}	NA	1.4 (2.1)	NA	1.9 (2.8)
O_3	None	4.0 (4.2)	NA	1.6 (1.8)	NA
	$PM_{2.5}$	3.6 (3.6)	0.6 (1.2)	0.8 (0.9)	1.5 (2.6)
	$PM_{10\text{-}2.5}$	3.9 (4.0)	0.1 (0.2)	1.4 (1.5)	0.7 (1.1)
	PM_{10}	3.7 (3.7)	0.6 (0.9)	0.9 (0.9)	1.7 (2.4)
NO_2	None	1.3 (1.2)	NA	4.0 (3.6)	NA
	$PM_{2.5}$	−0.2 (−0.2)	1.3 (1.9)	3.1 (2.4)	0.7 (1.1)
	$PM_{10\text{-}2.5}$	1.0 (0.9)	0.4 (0.7)	3.9 (3.3)	0.1 (0.2)
	PM_{10}	−0.0 (−0.0)	1.4 (1.7)	3.3 (2.5)	0.7 (0.9)
CO	None	0.4 (0.4)	NA	2.0 (2.3)	NA
	$PM_{2.5}$	−0.7 (−0.7)	1.4 (2.4)	1.1 (1.1)	1.5 (2.2)
	$PM_{10\text{-}2.5}$	0.1 (0.2)	0.6 (1.0)	1.8 (2.1)	0.5 (0.8)
	PM_{10}	−0.5 (−0.6)	1.6 (2.1)	1.2 (1.3)	1.5 (2.0)
SO_2	None	0.3 (0.5)	NA	1.1 (2.0)	NA
	$PM_{2.5}$	−0.3 (−0.5)	1.2 (2.3)	0.6 (0.9)	1.3 (2.1)
	$PM_{10\text{-}2.5}$	0.2 (0.3)	1.3 (2.2)	1.0 (1.7)	0.7 (1.0)
	PM_{10}	−0.2 (−0.4)	0.6 (0.9)	0.6 (1.0)	1.5 (2.0)

Note. Concentrations based on day of death (lag 0) and day prior to death (lag 1) (ratio of log-relative risk to standard error given in parentheses). NA, not applicable.

indicating particulate mass does not fully characterize the effect of the mixture of urban atmospheric pollution on mortality in Canadian cities.

Effects of Elemental and Ion Concentrations From $PM_{2.5}$ on Mortality

The preceding analyses show a positive and statistically significant association between fine mass and mortality, although the association for the coarse fraction was weaker. In order to identify components of the fine fraction that may be related to mortality, we examined the association between elemental and ion concentrations from the fine fraction and daily nonaccidental deaths. Summary statistics for the elemental and ion data from the fine fraction are given in Table 6. Out of a possible 5547 days in which fine mass was collected, concentrations were available on 4255 days for most elements, except for Ce, Pr, Nd, W, Hg, Mg, and Na, as monitoring for these compounds only commenced in May of 1992.

The correlations between filtered mortality and filtered elemental concentrations are given in Table 7. Sulfur (S) showed the highest correlation with fine mass ($r = .74$), with Pb, Si, Fe, K, Zn, Mn, P, and Se modestly correlated with $PM_{2.5}$ ($r = .3–.5$). Several elements were weakly correlated ($r = .2–.3$)

TABLE 6. Summary Statistics for Elemental Data (μg/m^3) from $PM_{2.5}$

Element	Average detection limit (DL)	Percent above DL	Mean	Percentiles			Number of observations
				50th	75th	95th	
Pb	0.0008	97	0.0405	0.0115	0.0447	0.1710	4437
Al	0.0031	91	0.2019	0.0571	0.1570	0.8264	4254
Si	0.0017	93	0.1251	0.0900	0.1560	0.3336	4255
S	0.0007	97	1.0053	0.6840	1.2200	2.9420	4255
Fe	0.0021	99	0.0812	0.0565	0.0971	0.2332	4255
K	0.0023	99	0.0786	0.0603	0.0963	0.1871	4255
Ca	0.0028	98	0.0773	0.0524	0.0902	0.2170	4255
Zn	0.0009	99	0.0258	0.0134	0.0277	0.0903	4255
Br	0.0004	91	0.0110	0.0029	0.0102	0.0464	4442
Mn	0.0020	96	0.0134	0.0103	0.0165	0.0339	4255
P	0.0009	79	0.0266	0.0142	0.0330	0.0945	4255
Cu	0.0018	63	0.0077	0.0046	0.0106	0.0234	4255
As	0.0005	56	0.0009	0.0006	0.0012	0.0027	4255
Ni	0.0010	47	0.0016	0.0006	0.0020	0.0048	4255
Sr	0.0003	52	0.0005	0.0003	0.0006	0.0014	4255
Cl	0.0023	54	0.0492	0.0079	0.0335	0.1982	4255
Se	0.0005	49	0.0010	0.0004	0.0011	0.0036	4255
Ti	0.0047	44	0.0062	0.0029	0.0077	0.0160	4255
Ga	0.0011	36	0.0023	0.0006	0.0022	0.0096	4255
V	0.0037	31	0.0037	0.0020	0.0040	0.0106	4255
Sc	0.0037	29	0.0056	0.0026	0.0050	0.0232	4255
Cr	0.0029	22	0.0022	0.0016	0.0017	0.0060	4255
Co	0.0014	12	0.0009	0.0007	0.0008	0.0021	4255
Ge	0.0007	16	0.0005	0.0004	0.0004	0.0013	4255
Rb	0.0003	12	0.0002	0.0002	0.0002	0.0005	4255
Y	0.0003	7	0.0002	0.0002	0.0002	0.0004	4255
Zr	0.0004	29	0.0003	0.0002	0.0004	0.0010	4255
Nb	0.0004	8	0.0002	0.0003	0.0003	0.0007	4255
Mo	0.0005	32	0.0005	0.0003	0.0005	0.0018	4255
Pd	0.0017	2	0.0009	0.0012	0.0012	0.0014	4255
Ag	0.0018	9	0.0013	0.0013	0.0013	0.0028	3648
Cd	0.0038	22	0.0017	0.0017	0.0017	0.0044	4255
In	0.0023	21	0.0021	0.0018	0.0023	0.0060	4255
Sn	0.0029	47	0.0051	0.0022	0.0068	0.0135	4255
Sb	0.0030	42	0.0048	0.0024	0.0069	0.0164	4255
Te	0.0031	26	0.0036	0.0024	0.0045	0.0136	4255
I	0.0033	9	0.0020	0.0025	0.0025	0.0042	4255
Cs	0.0056	11	0.0032	0.0045	0.0045	0.0055	4255
Ba	0.0074	27	0.0054	0.0060	0.0060	0.0133	4255
La	0.0094	19	0.0059	0.0078	0.0078	0.0129	4255
Ce	0.0023	29	0.0024	0.0014	0.0028	0.0080	2038
Pr	0.0028	31	0.0028	0.0018	0.0033	0.0082	2038
Nd	0.0033	41	0.0039	0.0028	0.0050	0.0109	2038
W	0.0030	49	0.0039	0.0023	0.0058	0.0103	2038
Hg	0.0008	28	0.0006	0.0005	0.0006	0.0012	2038
Mg	0.0040	80	0.0233	0.0141	0.0279	0.0756	2038
Na	0.0463	76	0.2428	0.1510	0.3046	0.7801	2038

TABLE 7. Percent Increase in Filtered Daily Nonaccidental Deaths Associated With an Increase in Filtered Elemental Concentration ($\mu g/m^3$) Equivalent to the Study Mean Based on $PM_{2.5}$

Element	Correlation (× 100) with $PM_{2.5}$	Percent increase	
		Lag 0	Lag 1
Pb	33	−0.0 (−0.1)	0.3 (0.8)
Al	16	0.1 (0.6)	−0.1 (−0.7)
Si	34	0.3 (0.7)	0.4 (1.0)
S	74	0.8 (1.9)	1.4 (3.2)
Fe	48	0.3 (0.5)	1.2 (2.3)
K	41	0.4 (0.9)	0.6 (1.3)
Ca	21	0.8 (2.2)	0.5 (1.2)
Zn	37	0.6 (1.8)	0.8 (2.4)
Br	29	−0.1 (−0.4)	0.1 (0.3)
Mn	47	0.1 (0.3)	0.5 (1.3)
P	46	0.2 (0.9)	0.5 (1.7)
Cu	6	0.4 (1.7)	−0.2 (−0.8)
As	21	0.3 (1.4)	0.1 (0.3)
Ni	26	0.5 (1.5)	0.7 (1.8)
Sr	12	0.1 (1.1)	−0.2 (−1.8)
Cl	16	0.1 (0.8)	−0.1 (−0.7)
Se	35	0.0 (0.1)	0.2 (0.8)
Ti	28	−0.3 (−1.1)	0.2 (0.9)
Ga	−7	0.2 (0.4)	−0.9 (−1.7)
V	26	−0.1 (−0.4)	0.1 (0.2)
Sc	0	0.6 (1.7)	0.1 (0.4)
Cr	13	0.7 (1.4)	0.3 (0.5)
Co	25	1.7 (2.0)	1.4 (1.6)
Ge	7	−0.4 (−0.5)	1.0 (1.2)
Rb	21	0.6 (1.1)	0.2 (0.3)
Y	4	−0.4 (−0.4)	−0.0 (−0.0)
Zr	11	0.9 (2.5)	−0.2 (−0.6)
Nb	−3	−0.0 (−0.0)	−0.8 (−0.7)
Mo	8	−0.6 (−1.1)	0.5 (0.8)
Pd	3	−0.5 (−0.3)	−4.3 (−2.4)
Ag	1	−0.0 (−0.7)	−0.0 (−0.6)
Cd	15	−0.2 (−0.8)	0.2 (0.7)
In	8	0.7 (1.6)	0.2 (0.4)
Sn	5	0.1 (0.5)	0.0 (0.1)
Sb	15	−0.1 (−0.1)	0.1 (0.2)
Te	10	0.4 (1.2)	−0.3 (−0.6)
I	8	−0.2 (−0.2)	0.1 (0.1)
Cs	4	−0.9 (−0.9)	0.2 (0.2)
Ba	11	−0.7 (−1.0)	−0.4 (−0.5)
La	0	0.1 (0.1)	1.4 (1.8)
Ce	−2	−0.4 (−0.6)	0.5 (0.7)
Pr	−7	−0.6 (−0.7)	0.1 (0.2)
Nd	−7	−0.4 (−0.4)	0.2 (0.2)
W	5	−1.8 (−1.3)	0.1 (0.1)
Hg	1	1.7 (1.8)	−1.4 (−1.2)
Mg	21	2.1 (3.0)	−0.7 (−0.9)
Na	17	0.9 (1.6)	0.1 (0.2)

Note. Concentrations recorded on the day of death (lag 0) or the day prior to death (lag 1) (*T* values given in parentheses).

with fine mass (Ca, As, Ni, Ti, V, Co, Rb, Mg). The remaining elements (Al, Cu, Sr, Cl, Ga, Sc, Cr, Ge, Y, Zr, Nb, Mo, Pd, Ag, Cd, In, Sn, Sb, Te, I, Cs, Ba, La, Ce, Pr, Nd, W, Hg, Na) displayed little association with $PM_{2.5}$.

Sulfur, Ca, Zn, Cu, Sc, Co, Zr, and Mg displayed some evidence of a positive association with mortality for concentrations measured on the day of death (T ratio > 1.645, corresponding to a one-sided p value of .05). For those concentrations recorded on the day prior to death, S, Fe, Zn, P, Ni, and La displayed some evidence of an effect on mortality. Note that only S and Zn displayed a positive and statistically significant association for both lagging times.

In further analyses, we examined concentrations of the sulfate ion (SO_4) instead of the element sulfur due to chemical analytical considerations (Brook et al., 1997). There were 4438 days in which sulfate concentrations were obtained from the fine fraction, with a lag 0-day effect of 0.9% ($T = 1.9$) and a lag 1-day effect of 1.2% ($T = 3.5$) evaluated at the study mean of 2.6 $\mu g/m^3$. Sulfates were highly correlated with fine mass ($r = .73$).

We further considered the four components of the fine fraction that were most statistically significantly positively associated with mortality from lag 1 day data: sulfates, zinc, nickel, and iron. We observed some variation in the effect of $PM_{2.5}$ on mortality among cities, ranging from a 1.1% increase in daily mortality to a 6.9% increase. The range in the $PM_{10\text{-}2.5}$ effect was −1.0% to 3.8%, while the range in effect for PM_{10} was from 0.4% to 7.0%. The variability in the effect estimates among cities for the elements and sulfate based on $PM_{2.5}$ was much greater than the corresponding effect for mass. The range in the percent increase in mortality for sulfate was from −4.3 to 3.9, for iron was −5.0 to 3.1, for nickel was −10.7 to 2.2, and for zinc was −3.3 to 3.7. The range in increase in mortality based on the sum of all four compounds was −8.4% to 7.7%, with 2 of 8 cities displaying a negative association with mortality.

Sulfate was weakly correlated with Fe ($r = .28$), Ni ($r = .15$), and Zn ($r = .26$). Ni was also weakly correlated with the other factors ($r < .26$). Fe and Zn were clearly associated ($r = .59$) however. Sulfates, nickel, and zinc were also weakly correlated with the four gases ($r < .32$). However, the correlation between iron and three of the gases (CO, NO_2, and SO_2) was slightly higher ($.35 < r < .39$).

An index of elemental carbon (Groblicki et al., 1981), the coefficient of haze (COH), was monitored daily in each city. The effect for COH at lag 0 days, evaluated at the study mean of 2.6 hundred linear feet, was 0.2% ($T = .3$), while the effect at lag 1 day was 0.7% ($T = 1.1$) based on the days in which PM_{10} were available. COH was positively correlated with fine mass ($r = .57$), iron ($r = .43$), sulfates ($r = .35$), zinc ($r = .33$), and nickel ($r = .22$). However, COH was highly associated with both CO and NO_2 ($r = .71$), modestly correlated with SO_2 ($r = .49$), and weakly related to O_3 ($r = .05$). Note that COH displayed the second largest correlation with $PM_{2.5}$ next to SO_4.

We further considered the combined effects of the four most strongly statistically associated constituents of particulate pollution to mortality based on lagged 1-day measurement data (SO_4, Fe, Ni, and Zn). Four linear combinations or components of these four variables were determined using principal component analysis techniques with varimax rotation, and these four component variables were regressed against filtered mortality counts simultaneously. The component loadings were positive for all pollutants and components examined. The effects of these components on mortality were adjusted separately for the four gases and COH. The effects of sulfur dioxide and COH in this analysis were negative. As the effects of O_3, CO, and NO_2 were positive, we considered a further principal component analysis with seven variables (SO_4, Ni, Fe, Zn, O_3, CO, and NO_2). The factor loading for each component and pollutant were positive, reflecting the correlation structure in the original data. [Ozone concentrations recorded on the day of death were used in this analysis since they displayed a much stronger association with mortality than concentrations recorded on the day prior to death (see Table 5).] These seven components were then related to filtered mortality with the resulting estimates of daily mortality given in Table 8 (Model II). Each of these seven pollutants was positively related to nonaccidental mortality, with the largest effect observed for ozone (2.0%) and the least effect for CO (0.7%). The effect of each individual pollutant on mortality is also given in Table 8. The estimates for the

TABLE 8. Percentage Increase in Daily Filtered Nonaccidental Deaths Associated With Increases in Daily Filtered Air Pollution Concentrations in Single and Multiple Pollutant Model Specifications, Controlling for Weather Variables

Pollutant[a] (units)	Mean concentration	Single-pollutant model	Multiple-pollutant models	
			Model I	Model II
PM_{10} (μg/m^3)	25.9	1.9 (2.8)[b]	NA[d]	NA
$PM_{2.5}$ (μg/m^3)	13.3	1.6 (3.1)	1.0 (2.9)	NA
$PM_{10\text{-}2.5}$ (μg/m^3)	12.6	0.9 (1.4)	0.6 (1.6)	NA
O_3 (ppb)	31	3.4 (2.6)	1.6 (3.4)	2.0 (3.2)
NO_2 (ppb)	22	3.9 (3.0)	1.1 (3.2)	1.2 (2.7)
SO_2 (ppb)	4.7	1.1 (1.6)	0.7 (2.1)	NA[c]
CO (ppm)	0.9	2.1 (2.1)	0.7 (1.9)	0.7 (1.7)
SO_4 (μg/m^3)	2.6	1.2 (3.5)	NA	1.3 (3.5)
Zn (μg/m^3)	0.0258	0.8 (2.4)	NA	0.8 (2.1)
Ni (μg/m^3)	0.0016	0.7 (1.8)	NA	0.8 (1.9)
Fe (μg/m^3)	0.0812	1.2 (2.3)	NA	0.8 (1.8)

Note. Effect estimates in multiple pollutant models obtained using principal component analysis.

[a]Pollutants recorded the day prior to death except for ozone which was recorded on day of death.

[b]*T* statistic.

[c]Negative effect, removed from model.

[d]NA, not included in model.

gases were based on days in which elemental data were available. The effects for all four gases were reduced after adjusting for constituents of particulate matter. The effects for SO_4, Zn, and Ni were not sensitive to adjustment for the gases. However, the effect of Fe on mortality was slightly reduced (1.2% to 0.8%).

Effects of Elemental and Ion Concentrations from $PM_{10\text{-}2.5}$ and PM_{10} on Mortality

Of the 47 components of $PM_{10\text{-}2.5}$ examined for lag 0-day data, 3 displayed positive and statistically significant ($T > 1.645$) associations (indium, sulfate, and ammonium) and 3 displayed negative and statistically significant associations (gallium, tungsten, and lead). For lag 1-day data, 4 compounds displayed positive and statistically significant associations with mortality (scandium, manganese, nickel, and zinc), while a negative, statistically significant association was observed for 6 elements (germanium, iodine, cesium, cerium, praseodymium, and tungsten). These results should be interpreted with caution since we found more components negatively associated with mortality than that displayed positive effects.

Only nickel and zinc displayed a positive and statistically significant association with mortality at the same lagging time for both $PM_{2.5}$ and $PM_{10\text{-}2.5}$ data. We added the concentrations for nickel and zinc obtained from $PM_{2.5}$ and $PM_{10\text{-}2.5}$ to reflect concentrations based on PM_{10} data. A 2.9-$\mu g/m^3$ (study mean concentration) increase in nickel recorded on the day prior to death was associated with a 1.1% increase in nonaccidental mortality (T ratio of 2.5). A 40.8-ng/m^3 (study mean concentration) increase in zinc was associated with a 1.2% increase in mortality (T ratio of 3.1).

We note that the sulfate concentrations recorded on the day of death were associated with mortality (0.7%, $T = 2.2$), but not for concentrations recorded on the day prior to death (−0.1%, $T = 0.2$). Sulfate concentrations were much lower for the $PM_{10\text{-}2.5}$ data (0.3 $\mu g/m^3$) than for $PM_{2.5}$ data (2.6 $\mu g/m^3$). The lag 0-day increase in mortality for sulfates was 0.9% ($T = 2.3$), while the lag 1-day increase was 1.2% ($T = 3.1$) based on a change of 2.9 $\mu g/m^3$. These values were identical to those obtained from the $PM_{2.5}$ data, suggesting that all the information in the association between sulfate concentrations and mortality was in the fine particulate fraction and not the coarse fraction.

DISCUSSION AND CONCLUSIONS

Positive and statistically significant associations were observed in this study between daily variations in both gas and particulate phase pollution and daily fluctuations in mortality rates in eight Canadian cities from 1986 to 1996. The air pollution association with mortality could not be explained by temporal variation in either mortality rates or weather factors.

Health Effects of Components of the Urban Mixture

Fine particulate mass was a stronger predictor of mortality than coarse mass, a result also reported by Schwartz et al. (1996) in six U.S. cities. The results of this study confirmed the association between nonaccidental mortality and concentrations of gas-phase pollution that was observed previously in a study of 11 Canadian cities from 1981 to 1991 (Burnett et al., 1998b). Bates (1998) suggested that the association with the gaseous pollutants, in particular NO_2, and mortality was due to particulate matter. The current study addresses a limitation in our earlier work by including size-fractionated particulate mass. Size-fractionated particulate mass accounted for 28% of the total effect of the urban pollution mix on mortality, as characterized by the four gases and fine and coarse mass. However, if additional constituents of particulate matter are examined, such as elemental concentrations and sulfates, the risk attributable to particulate phase pollution increased to 49%. This suggests that mass may be a relatively crude indicator of the toxicity of particulate phase pollution in urban environments.

We also note that the risk of death associated with the gaseous pollutants was more sensitive to adjustment for other pollutants in the urban mix than the particle-phase pollutants. This suggests that either the gaseous pollutants are indices for other air pollutants and/or theyare subject to greater exposure error than the particulate measures.

Statistical Model

We have chosen to related temporally filtered weather and air pollution values to temporally filtered mortality counts, with filtering conducted on a city-specific basis. In this manner we have removed the potentially confounding temporal cycles in all time series prior to linking them together. Residuals from these temporally filtered series are then compared across cities in a single analysis.

An alternate statistical approach is to simultaneously regress unfiltered air pollution and weather data on daily mortality counts adjusting for temporal trends in the mortality time series only, using a smooth function of day of study. This analysis would be conducted for each city separately and a summary estimate of the air pollution effect given by a weighted average of the city-specific effects, with weights given by the inverse of the estimation error. We conducted this "coadjustment" analysis for $PM_{2.5}$, $PM_{10\text{-}2.5}$, and PM_{10} and compared the results to our "preadjustment" approach.

The percent increase in daily mortality associated with an increase in $PM_{2.5}$ evaluated at the study mean concentration of 13.3 $\mu g/m^3$ was 1.9% under the coadjustment method. This is slightly higher than that obtained by our model in which both mortality and air pollution are temporally filtered prior to linking them together (1.6%). Similar results were observed for $PM_{10\text{-}2.5}$ (1.2% for coadjusted and 0.9% for preadjusted) and PM_{10} (2.1% for coadjusted and

1.9% for preadjusted). These results suggest that there is additional information in the time series data relating air pollution and mortality that is not captured in the high-frequency or day-to-day cycles in the respective series. The preadjusted method removes all non-high-frequency information in the data. The coadjusted approach can leave some residual confounding in the cycles between mortality and air pollution. Particulate levels are highest in the winter, as are mortality counts. In the coadjusted approach, the cycles in these two time series compete to predict mortality. Thus some of the midfrequency or seasonal cycle in mortality may be captured by a corresponding cycle in air pollution, thus inflating the air pollution effect. If the cycles are in conflict, such as with ozone or temperature and mortality, the effect estimates will be biased downward.

The preadjustment approach controls for such confounding. Estimates of the air pollution effect based on the preadjustment approach reflect the true association between short-term (day-to-day) variation in air pollution and mortality, while estimates based on the coadjustment method reflect the sum of short-term and residual medium-term concentrations. This residual median-term concentrations effect on mortality is subject to potential confounding due to other temporally varying risk factors to a greater extent than the short-term concentration signal. We thus recommend the use of the preadjustment method, which, to a greater extent, controls for potentially confounding temporal cycles than the coadjustment technique.

Variation in Effect Among Cities

The variation in the percent increase in mortality associated with concentrations of fine particulate mass was less than the variation in effect of any of the four constituents examined, and of the sum of the effects of the constituents. This suggests that there were unmeasured components of fine particulate matter that affect health. Elemental and organic carbon accounts for approximately half the mass of fine particulate matter in Canadian cities (Brook et al., 1997). Concentrations of these compounds were also more stable between cities than the elemental data (Brook et al., 1997). COH is highly correlated with elemental carbon. We found a positive but statistically weak association between COH and mortality, with a 0.7% increase in daily mortality associated with a 0.26 change in COH per thousand linear feet. Adjustment for the constituents of the atmospheric mix eliminated the COH association with mortality based on days in which elemental data were available.

COH was measured daily in each city. The percent increase in daily deaths associated with COH was 1.1% ($T = 5.1$) for lag 0-day concentrations and 1.2% ($T = 5.7$) for lag 1-day concentrations, with a 1.7% ($T = 6.6$) effect observed for 2-day average values. The COH effect persisted after adjustment for the four gases (results not shown). We thus urge caution in interpreting the

negative results for COH in this study and recommend that daily observations of elemental and organic carbon be taken in Canadian urban environments to address one of the major components of fine particulate matter and its association with human health.

Sources of Particulate Pollution

The four components of the fine fraction that were most statistically significantly positively associated with mortality based on lag 1-day data were sulfate, zinc, nickel, and iron. Fine-fraction Ca, Cu, Sc, Co, Zr, P, La, and Mg were also found to have some association with mortality. The chemical constituents on fine particles are known to be associated with certain sources (Rahn & Lowenthal, 1985), and some elements are more source specific than others are. Sulfur or sulfate is widely known to be from SO_2 emissions, and the main Canadian sources are coal and oil combustion at power plants, smelters, and the oil and gas extraction and refining industry. Many of these sources are located away from cities, but their SO_2 emissions contribute to sulfate particles on a regional scale, which then impacts upon cities. Within cities the main SO_2 sources, which are generally small relative to the sources just listed, are power plants, refineries, industry (e.g., steel manufacturing, pulp and paper), and transportation. However, not all of these sources, with the exception of transportation, are found in each of the cities in included in this study.

The sources for the trace elements are not as widely known. Elemental analysis of fine particles from a wide range of sources, some of which has been carried out to support chemical mass balance modeling (Watson et al., 1994), indicates that zinc emissions are associated with steel production, road dust, possibly from tire wear, and incinerators. In addition, but to a lesser degree, zinc is found in the emissions from oil-fired power plants and even motor vehicle emissions. Iron is also significantly enriched in the emissions associated with steel production and is prevalent in road dust and soil. Iron can also be emitted from power plants. One of the main sources of nickel is oil-fired power plants. To a lesser degree nickel is emitted from oil refining, steel production, and certainly from nickel smelters. Traces of nickel are also emitted from motor vehicles, and it can be found in road dust and soil. The other elements listed earlier also have some predominant sources. For example, magnesium and zirconium are found in coal-fired power plant emissions. Copper is found in fine-particle samples collected near traffic (i.e., roadside sampling) as well as from copper smelters, steel production, incineration, and even vegetation burning. Magnesium is also associated with vegetation burning and even cooking (e.g., meat), while calcium has a wide variety of sources, although soil is one of the main ones. Calcium can be also found in emissions from coal-fired power plants and steel production. There is clearly some overlap in the sources contributing to the ambient trace elements, which makes it difficult to point toward specific sources that may be more responsible for

the fine particles associated with mortality. However, some possible source distinctions can be hypothesized. Zn may be indicative of a road dust and possibly a tire wear source and secondarily of incineration. Fe and Zn are linked to steel production and Ni linked to oil combustion.

Toxicological Evidence of Elemental Effects

Our analyses reveal a stronger association between mortality and a subset of the particle-associated metals, namely, iron, nickel, and zinc, all of which are known pneumotoxicants (Nemeri, 1990), than to mass alone. Iron, a prototypical redox cycling metal of toxicological relevance, mediates the toxicity and carcinogenicity of several types of particles and compounds (Fubini, 1997; Weinberg, 1999; Ghio et al., 1999). Sulfate plays a role in the conversion of iron into a bioavailable, catalytically active form, a phenomenon directly relevant to particulate matter toxicity (Ghio et al., 1999).

The lung toxicity of ambient particulate matter (Adamson et al., 1999; Vincent et al., 1997a) has also been shown to be related to the presence of soluble zinc (Adamson et al., 2000). High doses of zinc chloride aerosols induce a progressive clinical course resembling adult respiratory distress syndrome (Evans, 1945; Hjortso et al., 1988). Zinc, iron, and nickel, at the internal tissue doses produced from inhalation of low ambient concentrations of particles, would not act directly through mechanisms of acute injury. However, even small amounts of these metals are biologically active, and it is plausible that these metals could aggravate an already existing pathology.

The role that fine particulate mass plays in the induction of stress genes is related to chemistry of the particles, and in particular to bioavailable transition metals (Vincent et al., 1997b). Recent studies indicate that variation in the chemical composition of ambient particles–for example, a decrease in the bioavailability of transition metals–results in lower potency of particles in cell cultures, and also correlates with lower rates of morbidity in human populations (Frampton et al., 1999). Iron from inhaled particles could amplify inflammatory processes through Fenton-type reactions in the lungs.

Several nickel compounds are toxic and carcinogenic to the lungs (Oller et al., 1997). The genetic and epigenetic mechanisms of action of nickel at low doses, which may not be acutely toxic to the respiratory tract, could interfere with gene expression in target cells, resulting in adverse impacts on cell regulation pathways critical to lung defense (Timblin et al., 1998). Zinc plays a central role in gene regulation and in the activation of metalloproteases and endopeptidases. Deposition of free, ionized zinc (e.g., as zinc sulfate) in the alveoli could create local cellular loads of the metal, interfering with cell regulation pathways (e.g., Ellerbroek & Stack, 1999; Klug, 1999; Palacek et al., 1999; Samet et al., 1998). Our observations begin to provide some insight into potential chemical determinants of the potency of ambient particles.

CONCLUSIONS

We conclude that daily variations in urban pollution mixtures in Canadian cities are statistically associated with daily variations in mortality rates. There are many components of the atmospheric urban mixture that contribute to its toxicity, including pollutants in both the particulate and gas phase. When evaluating the potential population health benefits of air pollution mitigation strategies, we urge consideration of the health benefits of the predicted reduction in all pollutants attributed to the proposed strategy and not just a single pollutant.

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Attachment A15

Associations between Air Pollution and Mortality in Phoenix, 1995–1997

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We evaluated the association between mortality outcomes in elderly individuals and particulate matter (PM) of varying aerodynamic diameters (in micrometers) [PM_{10}, $PM_{2.5}$, and PM_{CF} (PM_{10} minus $PM_{2.5}$)], and selected particulate and gaseous phase pollutants in Phoenix, Arizona, using 3 years of daily data (1995–1997). Although source apportionment and epidemiologic methods have been previously combined to investigate the effects of air pollution on mortality, this is the first study to use detailed PM composition data in a time-series analysis of mortality. Phoenix is in the arid Southwest and has approximately 1 million residents (9.7% of the residents are > 65 years of age). PM data were obtained from the U.S. Environmental Protection Agency (EPA) National Exposure Research Laboratory Platform in central Phoenix. We obtained gaseous pollutant data, specifically carbon monoxide, nitrogen dioxide, ozone, and sulfur dioxide data, from the EPA Aerometric Information Retrieval System Database. We used Poisson regression analysis to evaluate the associations between air pollution and nonaccidental mortality and cardiovascular mortality. Total mortality was significantly associated with CO and NO_2 ($p < 0.05$) and weakly associated with SO_2, PM_{10}, and PM_{CF} ($p < 0.10$). Cardiovascular mortality was significantly associated with CO, NO_2, SO_2, $PM_{2.5}$, PM_{10}, PM_{CF} ($p < 0.05$), and elemental carbon. Factor analysis revealed that both combustion-related pollutants and secondary aerosols (sulfates) were associated with cardiovascular mortality. *Key words*: cardiovascular, composition, factor analysis, particulate matter, $PM_{2.5}$, PM_{10}, sources. *Environ Health Perspect* 108:347–353 (2000). [Online 25 February 2000] *http://ehpnet1.niehs.nih.gov/docs/2000/108p347-353mar/abstract.html*

The associations between air pollution, especially particulate matter (PM), and adverse human health effects have been well documented (*1–10*). PM is associated with decreased respiratory function, aggravation of existing respiratory and cardiovascular conditions, altered defense mechanisms, and even premature death. The most susceptible populations include those with preexisting respiratory or cardiovascular conditions, asthmatics, children, and the elderly (*8,11*).

To date, few epidemiology studies have used PM measures other than size-segregated mass as the exposure metric. Schwartz et al. (*12*) looked at episodes of high coarse particle concentration in Spokane, Washington, and found that windblown dust episodes were not associated with increased mortality. In the Harvard Six Cities Study, Schwartz et al. (*3*) found a significant association between nonaccidental mortality and particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and sulfur. They did not find a significant association with particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}) or the coarse fraction of PM [PM_{CF} (PM_{10} minus $PM_{2.5}$)]. In contrast, Ostro et al. (*13*) found that PM_{10} dominated by coarse particles was associated with an increase in mortality in the Coachella Valley in California. The differences in the results from these two studies may be due to the particulate composition as well as the difference in the amount of PM_{CF}. In the eastern United States, $PM_{2.5}$ is dominated by sulfates (34%), whereas in the western and central United States it is dominated by organic carbon (OC) from motor vehicles and vegetative burning (39%) (*14*). The average $PM_{2.5}/PM_{10}$ ratio for the Six Cities Study (*3*) was 0.6 (based on the 50th percentiles) as compared to a ratio of 0.3 for Phoenix, Arizona (*15*).

The goal of the present study was to evaluate the associations between daily air pollution and total nonaccidental and cardiovascular mortality in Phoenix. Phoenix is an arid southwestern city with a population of approximately 1 million residents (*16*). It is an interesting location because of its large proportion of elderly people (9.7% of the population is > 65 years of age). The elderly are more susceptible to air pollution than the general public (*2*). The primary sources of PM in Phoenix are motor vehicles, paved road dust, and vegetative burning (*15*).

This study focused on the effects of air pollution on cardiovascular mortality for several reasons. First, the association between air pollutants and cardiovascular mortality has been consistent in previous studies (*1,2,9,17*). Second, a study in Baltimore, Maryland, found that heart rate variability was associated with $PM_{2.5}$ in elderly subjects with cardiovascular conditions (*18*). Finally, in this study cardiovascular mortality had the largest sample size, accounting for 45% of the total nonaccidental deaths in the study region (based on zip codes). This may be reflective of the increased size of Phoenix's elderly population, which is more prone to cardiovascular disease.

A unique aspect of this study is that our pollution data include daily information not only on traditional gaseous pollutants, but also on PM in various size fractions and the chemical composition of $PM_{2.5}$. From 1995 to 1997, the U.S. Environmental Protection Agency (EPA) National Exposure Research Laboratory (NERL) operated a comprehensive monitoring platform in Phoenix. They collected daily $PM_{2.5}$ samples and subsequently analyzed them for various chemical components of PM. This provided an opportunity to examine more specific metrics for PM than simply mass, as well as an opportunity to identify selected chemical components of PM that are associated with mortality.

In addition to PM, this study also evaluated the association between total nonaccidental and cardiovascular mortality and other measured air pollutants: carbon monoxide, nitrogen dioxide, sulfur dioxide, and ozone. These EPA criteria pollutants are also associated with mortality (*7,17,19,20*).

Methods

Study area and data. Mortality data for all of Maricopa County from 1995 to 1997 were obtained from the Arizona Center for Health Statistics in Phoenix. Death

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certifi-cate data included residence zip code and the primary cause of death as identified by the *International Classification of Diseases, Ninth Revision* (*ICD-9*, World Health Organization, Geneva). Only the deaths of residents in the zip codes located near the air pollution platform were included in this study. This zip-code region was recommended by the Arizona Department of Environmental Quality (Phoenix, AZ). We evaluated total nonaccidental mortality (*ICD-9* codes < 800) and cardiovascular mortality (*ICD-9* codes 390–448.9) in this study. Summary statistics for the mortality outcomes are presented in Table 1.

We obtained $PM_{2.5}$, PM_{10}, PM_{CF}, and $PM_{2.5}$ chemical composition data from the EPA NERL platform in central Phoenix. Chemical composition was only available for $PM_{2.5}$. The monitoring platform is approximately 10 km west-northwest of downtown Phoenix at a state and local air monitoring station. Standard meteorologic parameters such as wind speed and direction, temperature, and relative humidity were continuously measured. The average temperature in Phoenix from 1995 to 1997 was 23.7 ± 8.1°C. The average relative humidity was 32 ± 15%.

NERL investigators made hourly $PM_{2.5}$ and PM_{10} measurements each day using two colocated tapered element oscillation microbalance (TEOM) monitors (Rupprecht & Patasnick Co., Albany, NY). The TEOM-PM_{10} was fitted with an EPA-approved federal reference method PM_{10} impactor inlet (model 246b; Andersen Instruments, Smyrna, GA). The TEOM-$PM_{2.5}$ was fitted with a $PM_{2.5}$ cyclone inlet (University Research Glassware, Chapel Hill, NC). The $PM_{2.5}$ cyclone on the TEOM was replaced with a well-impactor ninety-six (WINS) inlet on 20 December 1996. The WINS inlet has a sharper cut point as compared to the cyclone. We averaged the hourly concentrations to create a 24-hr average (0700–0700 hr), and we calculated the concentration of coarse fraction (TEOM PM_{CF}) as TEOM PM_{10} minus TEOM $PM_{2.5}$.

NERL investigators collected the daily gravimetric integrated 24-hr (starting at 0700) fine particle filter samples using a dual fine particle sequential sampler (DFPSS; University Research Glassware). The DFPSS was fitted with a cyclone that was identical to the cyclone on the TEOM-$PM_{2.5}$. The DFPSS collected daily samples on both Teflon and quartz filters. The Teflon filter was used for mass and elemental analysis, whereas the quartz filter was used for carbon analysis. In addition to the DFPSS, NERL investigators operated a dichotomous sampler (Andersen Instruments, Inc.) every third day beginning 17 June 1996. Both the $PM_{2.5}$ and PM_{CF} samples were collected on Teflon filters. The investigators measured elemental concentrations at the EPA (Research Triangle Park, NC) with energy dispersive X-ray fluorescence. OC and elemental carbon (EC) were measured by Sunset Laboratory (Forest Grove, OR) using thermal optical transmittance (*21*).

PM and gaseous pollutant concentrations (range and mean ± SD) from 1995 to 1997 are presented in Table 2. We obtained gaseous criteria pollutant data for CO, NO_2, O_3, and SO_2 from the EPA Aerometric Information Retrieval System (AIRS) database (*22*) for residential sites in the Phoenix region. We averaged CO values over four monitoring sites and we averaged NO_2 over two sites. Only one residential monitoring site was available for SO_2. We averaged the hourly averages for CO, NO_2, and SO_2 over 24 hr from 0700 to 0700. We used the maximum hourly O_3 (O_3 max) concentration in the same 24-hr period in the analysis.

The $PM_{2.5}$ constituents that we evaluated for effects on mortality were sulfur, zinc, lead, soil-corrected potassium (K_S) (*23*), OC, EC, total carbon (TC), and reconstructed soil. Soil was reconstructed by summing the oxides of Al, Si, Ca, Fe, and Ti using the formula recommended by Malm et al. (*24*). We also considered $PM_{2.5}$ that was corrected for soil content (nonsoil $PM_{2.5}$ = $PM_{2.5}$ - reconstructed soil). Table 3 presents the percent of the total mass of $PM_{2.5}$ accounted for by each component. The elements aluminum, silicon, calcium, titanium, and iron were not evaluated separately in the mortality analysis because they are the major elemental components of soil.

Statistical analysis. In our zip-code regions, we analyzed a total of 9,276 nonaccidental deaths from 1995 to 1997. Poisson regression was used to evaluate the association between the air pollutant exposure variables and the mortality outcomes (*2,5*).

We used Poisson regression because mortality data are discrete counts and death is a rare event. Poisson regression assumes the variance is equal to the mean. When the variance exceeds the mean, the variance is overdispersed. We adjusted standard errors for overdispersion; however, the amount of overdispersion was small. The overdispersion parameter was 1.05 and 1.00 for nonaccidental and cardiovascular mortality, respectively. We calculated all relative risks (RRs) for an interquartile increase (25th–75th percentile) in pollutant concentration.

The effect of air pollution on mortality is small and can be influenced by confounders. Therefore, base models for total mortality and cardiovascular mortality were constructed by adjusting for day of the week with indicator variables, and time trends, temperature, and relative humidity with smoothing functions (*25*). We determined degrees of freedom (*df*) for the function used to smooth time trend by minimizing autocorrelation as well as the Akaike information criterion (AIC) (*26*). We chose the *df* and lag for the smoothing functions for temperature and

Table 1. Mortality counts for individuals ≥ 65 years of age in Phoenix.

Year	Total nonaccidental	Average nonaccidental deaths/day	Total cardiovascular	Average cardiovascular deaths/day
1995	3,072	8.45	1,391	3.86
1996	3,201	8.74	1,473	3.98
1997	3,003	8.45	1,318	3.73
1995–1997	9,276	8.55	4,182	3.85

Table 2. Annual range of pollutant concentrations (1995–1997).

Particulate matter pollutant, year	Range	Gaseous pollutant	Range
$PM_{2.5}$ (DFPSS)		CO (ppm)	
1995	4–37		0.5–4.0
1996	3–39		0.3–4.0
1997	2–35		0.3–3.7
3-year mean	12.0 ± 6.6		1.5 ± 0.8
PM_{10} (TEOM)		NO_2 (ppb)	
1995	9–129		8–64
1996	5–213		9–59
1997	7–186		8–61
3-year mean	46.5 ± 22.3		30 ± 10
$PM_{2.5}$ (TEOM)		O_3 max (ppb)	
1995	1–40		10–131
1996	0–42		14–112
1997	1–34		14–104
3-year mean	13.0 ± 7.2		57.0 ± 17.7
PM_{CF} (TEOM)		SO_2 (ppb)	
1995	5–104		0–11
1996	5–187		1–17
1997	5–159		2–12
3-year mean	33.5 ± 17.3		3.1 ± 2.2

relative humidity to minimize the AIC. The base model for total mortality used indicator variables for day of the week, 10 *df* for time trends, 2 *df* for temperature with 2 days lag, and 2 *df* for relative humidity with 0 days lag. The base model for cardiovascular mortality used indicator variables for the day of the week, 10 *df* for time trends, 2 *df* for temperature with 1 day lag, and 2 *df* for relative humidity with 0 days lag.

We included continuous daily data from 1995 to 1997 (1,097 days) in the study. Each day was coded and included in the model to adjust for time trends. Little autocorrelation was observed after adjusting for day of week, time trends, temperature, and relative humidity. The autocorrelation for days 1–25 for both total and cardiovascular mortality were within the 95% confidence interval for an independent series.

We evaluated air pollution exposure variables by adding them individually as linear terms to the base model. The air pollution exposure metrics that were evaluated in this analysis included CO, NO_2, O_3, SO_2, TEOM PM_{10}, TEOM $PM_{2.5}$, TEOM PM_{CF}, $PM_{2.5}$ (DFPSS), S, Zn, Pb, soil, K_S, nonsoil PM, OC, EC, and TC. Lag days ranging from 0 to 4 were investigated. We evaluated the assumption of a linear relationship using a smooth function. This assumption was met if a straight line could be placed within the 95% confidence intervals (CIs). A p-value < 0.05 associated with the pollution exposure variable was considered significant. We conducted Poisson regression analyses using S-PLUS 4 (Mathsoft, Inc., Seattle, WA)

Factor analysis. We conducted a factor analysis on the daily concentrations of the chemical components of $PM_{2.5}$ from samples collected by the DFPSS (Al, Si, S Ca, Fe, Zn, Mn, Pb, Br, K_S, OC, and EC). The analysis also included the daily averages of the gaseous species emitted by combustion sources (CO, NO_2, and SO_2). Factor analysis is a technique used to explain the correlations between variables in terms of underlying factors that are not directly measurable. Each factor is a linear combination of the original variables and all such factors are orthogonal to each other. The factors were extracted using principal component analysis with a varimax rotation. We conducted factor analysis using SAS (SAS Institute Inc, Cary, NC). We used the resultant factor scores as surrogate exposure variables in predicting mortality outcomes with the Poisson regression model. Each factor was evaluated in a single source model. However, because the factor scores formed a set of orthogonal variables, we performed a separate regression analysis with all of the scores included in one multifactor model.

We also conducted a factor analysis on the daily concentrations of the chemical components of PM_{CF} from samples collected by the dicot (Al, Si, Cl, S, K Ca, Mn, Fe, Zn, Br, Pb, Sr, Cu, and Rb). We did not use the scores from this analysis in the time–series analysis because the sampling period started in June 1996 and samples were only collected every third day.

Results

Table 4 shows the correlation coefficients between PM, gaseous pollutants, temperature, and relative humidity for Phoenix in 1995–1997. $PM_{2.5}$ (obtained from the DFPSS) was highly correlated with CO (r = 0.85) and NO_2 (r = 0.79), but less so with SO_2 (r =0.43). $PM_{2.5}$ from the DFPSS was highly correlated with that measured with the TEOM (r = 0.93). Table 5 shows the

Table 3. Percent of total mass of $PM_{2.5}$ accounted for by each component.

Component	$PM_{2.5}$ (%)
S[a]	3.69
Mn	0.05
Zn	0.15
Br	0.03
Pb	0.06
OC[a] 1.4	38.37
EC	10.78
K_S	0.52
Soil[b]	17.50

[a]If S is assumed to be in the form of $(NH_4)_2SO_4$, the mass percent would be 15.2%. [b]2.20% Al + 2.49% Si + 1.63% Ca + 2.42% Fe + 1.94% Ti (*23*).

Table 4. Correlation coefficients between PM, gaseous pollutants, temperature, and relative humidity (RH) for Phoenix, 1995–1997.

	$PM_{2.5}$[a]	Temp	PM_{10} (TEOM)	RH	$PM_{2.5}$ (TEOM)	PM_{CF} (TEOM)	CO	NO_2	O_{3max}	SO_2
$PM_{2.5}$[a]	1.00	-0.31	0.69	0.16	0.93	0.50	0.85	0.79	-0.24	0.43
Temp	–	1.00	-0.08	-0.55	-0.25	0.00	-0.49	-0.40	0.71	-0.38
PM_{10} (TEOM)	–	–	1.00	-0.12	0.77	0.97	0.53	0.53	-0.12	0.41
RH	–	–	–	1.00	0.09	-0.19	0.23	0.08	-0.54	0.10
$PM_{2.5}$ (TEOM)	–	–	–	–	1.00	0.59	0.82	0.77	-0.20	0.48
PM_{CF} (TEOM)	–	–	–	–	–	1.00	0.34	0.37	-0.08	0.33
CO	–	–	–	–	–	–	1.00	0.87	-0.40	0.53
NO_2	–	–	–	–	–	–	–	1.00	-0.24	0.57
O_3	–	–	–	–	–	–	–	–	1.00	-0.37
SO_2	–	–	–	–	–	–	–	–	–	1.00

[a]Measured with the DFPSS.

Table 5. Correlation coefficient matrix of air pollutants.

	S	Zn	Pb	OC	EC	TC	K_S	PM_{10}	$PM_{2.5}$	PM_{CF}	Nonsoil $PM_{2.5}$	Soil	CO	NO_2	O_3	O_{3max}	SO_2
S	1.00	0.14	0.25	0.12	0.04	0.10	0.02	0.19	0.27	0.13	0.26	0.25	0.01	0.04	0.13	0.31	-0.07
Zn	–	1.00	0.63	0.62	0.71	0.65	0.30	0.46	0.61	0.33	0.63	0.49	0.65	0.62	-0.49	-0.27	0.26
Pb	–	–	1.00	0.69	0.69	0.71	0.39	0.48	0.67	0.34	0.71	0.49	0.71	0.63	-0.51	-0.30	0.33
OC	–	–	–	1.00	0.91	0.99	0.65	0.58	0.89	0.38	0.96	0.52	0.89	0.81	-0.57	-0.32	0.49
EC	–	–	–	–	1.00	0.95	0.57	0.58	0.84	0.40	0.89	0.52	0.90	0.82	-0.64	-0.41	0.46
TC	–	–	–	–	–	1.00	0.64	0.59	0.90	0.39	0.96	0.53	0.91	0.83	-0.60	-0.35	0.49
K_S	–	–	–	–	–	–	1.00	0.34	0.59	0.19	0.64	0.26	0.52	0.45	-0.27	-0.14	0.25
PM_{10}[a]	–	–	–	–	–	–	–	1.00	0.79	0.97	0.62	0.72	0.55	0.56	-0.25	-0.11	0.42
$PM_{2.5}$[a]	–	–	–	–	–	–	–	–	1.00	0.60	0.91	0.64	0.82	0.77	-0.44	-0.19	0.47
PM_{CF}[a]	–	–	–	–	–	–	–	–	–	1.00	0.41	0.66	0.37	0.39	-0.14	-0.07	0.35
Nonsoil $PM_{2.5}$	–	–	–	–	–	–	–	–	–	–	1.00	0.54	0.87	0.80	-0.54	-0.29	0.46
Soil	–	–	–	–	–	–	–	–	–	–	–	1.00	0.48	0.49	-0.17	0.05	0.09
CO	–	–	–	–	–	–	–	–	–	–	–	–	1.00	0.87	-0.68	-0.39	0.51
NO_2	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	-0.60	-0.24	0.56
O_3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	0.81	-0.46
O_3 max	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00	-0.37
SO_2	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1.00

[a]Based on TEOM measurements.

correlation coefficients between selected chemical composition components of $PM_{2.5}$ and the other air pollutants. TEOM PM_{10} was correlated with fine soil ($r = 0.72$), OC ($r = 0.58$), EC ($r = 0.58$), and TC ($r = 0.59$). TEOM $PM_{2.5}$ was highly correlated with OC ($r = 0.89$), EC ($r = 0.84$), TC ($r = 0.90$), and to a lesser extent with Zn ($r = 0.61$), Pb ($r = 0.67$), and K_S ($r = 0.59$). The high correlation coefficients between carbon and $PM_{2.5}$ indicate that the majority of the variation in $PM_{2.5}$ is due to combustion products. PM_{CF} was correlated with soil ($r = 0.66$).

OC and EC concentrations follow a seasonal pattern—they are high in the colder months and low in the warmer months. This pattern is due to increased com-bustion emissions from space heating and the decreased mixing height during the winter months. Particulate sulfur concentrations peak in the warmer months. Soil concentration also follows a seasonal trend, with higher concentrations in the spring and fall. Measured soil concentrations decreased after 20 December 1996 because of the use of the WINS inlet.

Summaries of the RR between the exposure variables and both total and cardiovascular mortality are presented in Tables 6 and 7, respectively. Because of space limitations, we only present statistically significant ($p < 0.05$) and marginally significant ($p < 0.10$) results in the tables, although models were run using all of the pollutants listed in Table 5. Tables of all of the nonsignificant results are available from the authors by request. We evaluated the associations between total and cardiovascular mortality and the gaseous pollutants, PM mass metrics, and PM composition metrics using single-pollutant models.

We found significant associations between both mortality outcomes and selected gaseous air pollutants. CO and NO_2 were positively associated with total mortality at 0- and 1-day lags. There was evidence of a weak association with SO_2 at 0 days lag ($p < 0.10$). We found several strong associations with cardiovascular mortality. Cardiovascular mortality was positively associated with CO (0–4 days lag). This was the most consistent association because the association was significant for all 5 lag days. Statistically significant associations ($p < 0.05$) were also evident with NO_2 on lag days 1 and 4, although the association was weaker on lag days 2 and 3. Cardiovascular mortality was also associated with SO_2 (lag days 2, 3, and 4).

We also found significant associations between the mortality outcomes and particulate mass. The associations between PM_{10} and total mortality, and between PM_{CF} and total mortality, were marginal ($p < 0.10$). Total mortality was not significantly associated with $PM_{2.5}$; however, the RR was 1.02 (CI, 1.00–1.05). All PM mass metrics were associated with an excess risk of cardiovascular death. The strongest associations were with $PM_{2.5}$ (TEOM), followed by PM_{10} and PM_{CF}. $PM_{2.5}$ adjusted for soil content (nonsoil $PM_{2.5}$) was also related with cardiovascular mortality with 1 day lag ($p < 0.10$). Table 7 lists all of the statistically significant associations with cardiovascular mortality. Cardiovascular mortality showed a more consistent association with particulate mass concentrations than total mortality. We further investigated the associations between the mortality outcomes and PM by evaluating the association between the mortality outcomes and the PM composition. The $PM_{2.5}$ composition data analysis revealed that EC and TC were significantly associated with cardiovascular mortality (1 day lag). Weaker associations were also evident with OC at 1 and 3 days lag and TC at 3 days lag. K_S had a significant positive association with cardiovascular mortality (3 day lag).

Table 6. RR for total mortality in Phoenix from an interquartile range (IQR) increase in pollutants.

Pollutant	Lag days	β	SE	*t*	IQR	RR	LCI	UCI
CO	0	4.50×10^{-2}	1.48×10^{-2}	3.05	1.19	1.06	1.02	1.09
	1	4.15×10^{-2}	1.48×10^{-2}	2.81	1.19	1.05	1.01	1.09
NO_2	0	2.64×10^{0}	1.15×10^{0}	2.31	0.02	1.05	1.01	1.10
	1	3.29×10^{0}	1.13×10^{0}	2.91	0.02	1.07	1.02	1.12
	3	1.80×10^{0}	1.06×10^{0}	1.69	0.02	1.04	0.99	1.08
	4	2.20×10^{0}	1.07×10^{0}	2.05	0.02	1.04	1.00	1.09
SO_2	0	1.17×10^{-2}	6.37×10^{-3}	1.84	2.78	1.03	1.00	1.07
S	3	-1.38×10^{-4}	6.24×10^{-5}	-2.21	280.60	0.96	0.93	1.00
	4	-1.10×10^{-4}	6.10×10^{-5}	-1.80	279.90	0.97	0.94	1.00
Soil	1	-1.75×10^{-5}	8.67×10^{-6}	-2.01	1,767.45	0.97	0.94	1.00
	2	-1.76×10^{-5}	8.59×10^{-6}	-2.05	1,769.33	0.97	0.94	1.00
	3	-1.75×10^{-5}	8.56×10^{-6}	-2.04	1,772.48	0.97	0.94	1.00
	4	-1.47×10^{-5}	8.54×10^{-6}	-1.72	1,775.62	0.97	0.95	1.00
PM_{10} (TEOM)	0	1.06×10^{-3}	5.35×10^{-4}	1.98	24.88	1.03	1.00	1.05
PM_{CF} (TEOM)	0	1.17×10^{-3}	6.99×10^{-4}	1.68	18.39	1.02	1.00	1.05
Pb	3	-2.70×10^{-3}	1.59×10^{-3}	-1.69	6.00	0.98	0.97	1.00

Abbreviations: β, regression coefficient; LCI, lower 95% confidence interval; *t*, *t*-statistic from the regression model; UCI, upper 95% confidence interval.

Table 7. RR for cardiovascular mortality from an interquartile range (IQR) increase in pollutants.

Pollutant	Lag days	β	SE	*t*	IQR	RR	LCI	UCI
CO	0	4.49×10^{-2}	2.14×10^{-2}	2.10	1.19	1.05	1.00	1.11
	1	7.66×10^{-2}	2.07×10^{-2}	3.71	1.19	1.10	1.04	1.15
	2	5.79×10^{-2}	2.00×10^{-2}	2.89	1.19	1.07	1.02	1.12
	3	5.32×10^{-2}	2.03×10^{-2}	2.63	1.19	1.07	1.02	1.12
	4	6.43×10^{-2}	2.06×10^{-2}	3.12	1.19	1.08	1.03	1.13
NO_2	1	4.88×10^{0}	1.59×10^{0}	3.08	0.02	1.10	1.04	1.17
	2	2.53×10^{0}	1.54×10^{0}	1.64	0.02	1.05	0.99	1.12
	3	2.76×10^{0}	1.55×10^{0}	1.78	0.02	1.06	0.99	1.12
	4	5.74×10^{0}	1.57×10^{0}	3.66	0.02	1.12	1.05	1.19
SO_2	2	1.63×10^{-2}	8.64×10^{-3}	1.88	2.78	1.05	1.00	1.10
	3	1.85×10^{-2}	8.65×10^{-3}	2.14	2.79	1.05	1.00	1.10
	4	2.49×10^{-2}	8.58×10^{-3}	2.90	2.79	1.07	1.02	1.12
K_S	3	5.81×10^{-4}	2.96×10^{-4}	1.97	55.62	1.03	1.00	1.07
PM_{10} (TEOM)	0	1.88×10^{-3}	7.66×10^{-4}	2.46	24.88	1.05	1.01	1.09
	1	1.47×10^{-3}	7.56×10^{-4}	1.95	24.88	1.04	1.00	1.08
$PM_{2.5}$ (TEOM)	0	3.91×10^{-3}	2.38×10^{-3}	1.64	8.52	1.03	0.99	1.08
	1	6.85×10^{-3}	2.36×10^{-3}	2.90	8.52	1.06	1.02	1.10
	3	4.86×10^{-3}	2.35×10^{-3}	2.07	8.51	1.04	1.00	1.08
	4	5.43×10^{-3}	2.35×10^{-3}	2.31	8.47	1.05	1.01	1.09
PM_{CF}	0	2.50×10^{-3}	9.88×10^{-4}	2.54	18.39	1.05	1.01	1.09
	1	1.62×10^{-3}	9.78×10^{-4}	1.66	18.39	1.03	0.99	1.07
Nonsoil $PM_{2.5}$	1	5.56×10^{-6}	3.12×10^{-6}	1.78	6,601.06	1.04	1.00	1.08
OC	1	1.46×10^{-5}	6.82×10^{-6}	2.15	2,976.50	1.04	1.00	1.09
	3	1.39×10^{-5}	6.89×10^{-6}	2.02	2,960.00	1.04	1.00	1.08
EC	1	4.40×10^{-5}	1.82×10^{-5}	2.42	1,165.50	1.05	1.01	1.10
TC	1	1.15×10^{-5}	5.05×10^{-6}	2.28	4,169.00	1.05	1.01	1.09
	3	9.71×10^{-6}	5.10×10^{-6}	1.90	4,170.00	1.04	1.00	1.09

Abbreviations: β, regression coefficient; LCI, lower 95% confidence interval; *t*, *t*-statistic from the regression model; UCI, upper 95% confidence interval.

We also found that soil, S, and Pb were negatively associated with total mortality. That is, these exposure variables were associated with a decrease in excess deaths.

We further evaluated the associations between the mortality outcomes and sources of both particulate and gas-phase pollutants using the scores from a factor analysis in place of the individual pollutant concentrations. The results from the analysis with five factors are presented in Table 8. Factor 1 probably represents the influence of motor vehicle exhaust and resuspended road dust with high loadings (loading > 0.5) on Mn, Fe, Zn, Pb, OC, EC, CO, and NO_2. Factor 2 represents soil with high loadings on Al, Si, and Fe. Factor 3 represents vegetative burning with high loadings on OC and K_S. Factor 4 represents a local source of SO_2 with a high loading on SO_2. Factor 5 represents predominately regional sulfate with a high loading on S. The RRs associated with an interquartile range increase in each factor are presented in Table 9. Total mortality had both a positive and a negative association with the factor representing regional sulfate, positive on lag day 0 (same day) and negative on lag day 3. The factor representing SO_2 had a negative association with total mortality. We also found a significant negative association for fine soil on lag days 1 and 2, and a nearly significant negative association on lag days 3 and 4. Cardiovascular disease was significantly associated with the factors representing motor vehicles (lag day 1) and vegetative burning (lag day 3). Regression analysis with all of the factors included in a multisource model produced similar results.

Table 10 presents the results from the factor analysis on the daily concentrations of the chemical components of PM_{CF} from samples collected by the dichotomous sampler. Factor 1 represents soil with high loadings on Al, Si, K, Ca, Mn, Fe, Sr, and Rb. Factor 2 represents a source of coarse fraction metals with high loadings on Zn, Pb, and Cu. Factor 3 represents a marine influence with a high loading on Cl. These three factors explain 91.8% of the variance in the PM_{CF} data.

Sensitivity analysis. As a sensitivity analysis, we analyzed temperature as a cofactor rather than a confounder. That is, we evaluated the effects of temperature on mortality as an independent variable rather than adjusting for it in the model as a confounding variable. We evaluated the significance of temperature after adjusting for day of the week, time trends, and relative humidity. For total and cardiovascular mortality, we found that temperature was not associated with excess deaths. Temperature was not correlated with either PM_{10} (r = -0.08) or $PM_{2.5}$ (r = -0.25). A second analysis examined the effect of extreme temperatures. If the average daily temperature was greater than or equal to the 95th percentile (35.4°C), we assigned a 1 to the predictor variable; otherwise we assigned a 0. We did not find an association between extreme temperature and total mortality. However, with cardiovascular mortality, extreme temperature was associated with excess deaths at 0 and 2 days lag ($p < 0.1$). To further assess the importance of the high temperature days to our analysis, we evaluated the association between $PM_{2.5}$ and cardiovascular mortality after excluding the days when the temperature was above the 95th percentile. The effect of eliminating the high temperature days was negligible. The RR for cardiovascular mortality associated with $PM_{2.5}$ (1 day lag) including all days was the same as that excluding the hottest days (RR = 1.06; CI, 1.02–1.10).

We also conducted a sensitivity analysis with relative humidity as a cofactor, with the model controlling for time trends and temperature. As a cofactor, relative humidity was not associated with either total mortality or cardiovascular mortality. To further assess the effects of extreme relative humidity, we eliminated the driest days (relative humidity < 25th percentile) from the data. We then found that the coarse fraction was no longer associated with total mortality. The association between cardiovascular mortality and coarse fraction was statistically significant ($p < 0.05$) on the concurrent day, but nonsignificant with 1-day lag.

We also used dew point rather than relative humidity in the base model. Controlling for dew point rather than relative humidity did not alter our results. We obtained similar regression coefficients.

Table 8. Loadings from factor analysis.

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
Al	0.14	0.96	0.08	-0.01	0.07
Si	0.19	0.96	0.11	-0.01	0.10
S	0.04	0.15	0.01	-0.03	0.96
Ca	0.26	0.93	0.15	-0.01	0.09
Mn	0.66	0.62	0.05	0.13	0.07
Fe	0.57	0.76	0.19	0.19	0.05
Zn	0.86	0.24	0.03	-0.03	0.03
Br	0.46	0.31	0.59	0.01	0.28
Pb	0.74	0.21	0.25	0.12	0.26
OC	0.66	0.23	0.55	0.33	0.01
EC	0.76	0.25	0.42	0.28	-0.08
K_S	0.20	0.08	0.92	0.08	-0.04
CO	0.76	0.20	0.39	0.35	-0.09
NO_2	0.69	0.24	0.31	0.45	-0.05
SO_2	0.24	-0.04	0.09	0.93	-0.02
Percent variance explained by factor	30.5	27.5	13.7	9.7	7.4

Table 9. RR for total and cardiovascular mortality from an interquartile range (IQR) increase in each factor.

Outcome, factor	Lag days	β	SE	*t*	IQR	RR	LCI	UCI
Total mortality								
Factor 2	1	-0.03	0.01	-2.03	1.26	0.96	0.93	1.00
	2	-0.04	0.01	-2.45	1.26	0.96	0.92	0.99
	3	-0.02	0.01	-1.67	1.26	0.97	0.94	1.01
	4	-0.02	0.01	-1.74	1.26	0.97	0.94	1.00
Factor 4	2	-0.03	0.01	-2.01	1.09	0.97	0.94	1.00
	4	-0.03	0.01	-1.72	1.09	0.97	0.94	1.00
Factor 5	0	0.03	0.01	2.23	1.38	1.04	1.01	1.08
	3	-0.03	0.01	-2.22	1.39	0.96	0.92	0.99
Cardiovascular mortality								
Factor 1	1	0.05	0.02	2.59	1.11	1.06	1.01	1.10
Factor 3	3	0.05	0.02	2.67	1.02	1.05	1.01	1.09
Factor 5	0	0.04	0.02	2.03	1.38	1.06	1.00	1.12

Abbreviations: β, regression coefficient; LCI, lower 95% confidence interval; *t*, *t*-statistic from the regression model; UCI, upper 95% confidence interval.

Table 10. Factor analysis results for PM_{CF}.

Element	Factor 1	Factor 2	Factor 3
Al	0.91	0.33	0.22
Si	0.90	0.36	0.24
Cl	0.25	-0.35	0.82
S	0.59	0.55	0.41
K	0.91	0.33	0.23
Ca	0.84	0.41	0.31
Mn	0.88	0.42	0.17
Fe	0.84	0.50	0.19
Zn	0.47	0.83	0.07
Br	0.23	0.30	0.85
Pb	0.40	0.80	-0.02
Sr	0.83	0.42	0.28
Cu	0.41	0.82	-0.02
Rb	0.91	0.27	0.17
Percent variance explained by factor	51.1	26.5	14.2

To assess the effect of replacing the $PM_{2.5}$ cyclone on the TEOM with the WINS, we evaluated the association between soil and total mortality from 1 January 1995 to 31 December 1996 and from 1 January 1997 to 31 December 1997. The latter period represented the WINS inlet measurements. The association between soil and mortality was not significant for the cyclone measurements alone. Analysis with only the WINS data revealed that the association between soil and mortality was positive and significant at 0 days lag, but not significant for any of the other days.

We estimated soil-related potassium using a correction ratio = K/Si (*23*). We then reevaluated the RR for cardiovascular mortality associated with K_S using K_S calculated from three slightly different values of K/Si. This correction ratio is dependent on where the soil was obtained: $PM_{2.5}$ paved road dust (K/Si = 1.85/13.69), an agricultural field (K/Si = 1.98/14.35), or Phoenix desert soil (K/Si = 1.89/14.00) (*27*). We found similar RRs for cardiovascular mortality associated with K_S when we used any of these three approaches. In contrast, total potassium was not associated with either total or cardiovascular mortality.

Discussion

To our knowledge this is the first time–series analysis that has looked at the association between PM chemical composition and mortality and the association between the underlying factors influencing that composition and mortality. Ozkaynak and Thurston (*28*) combined source apportionment and epidemiologic methods to assess the effects of air pollution on mortality. However, their study was a cross-sectional analysis rather than a time–series analysis. The present study found significant associations between air pollutants and total nonaccidental and cardiovascular mortality. The association between PM_{10} and cardiovascular mortality is consistent with previous studies. Zmirou et al. (*17*) reported an RR for cardiovascular mortality from a 50-μg/m^3 increase in PM_{10} (RR = 1.04) in a study of air pollution in 10 large Western European cities. Pope et al. (*5*) found an association between respiratory disease death and cardiovascular deaths with PM_{10} in Utah. Schwartz (*2*) also found that on high-pollution days (increased total suspended particulates) there was an increased risk of death from cardiovascular disease (RR = 1.09) in Philadelphia, Pennsylvania, and Birmingham, Alabama (*1*). Furthermore, Anderson et al. (*29*) found that black smoke was associated with a 0.58% increase in cardiovascular deaths in London.

The association between $PM_{2.5}$ and cardiovascular mortality is similar to that of Schwartz et al. (*3*), who found that a 10-μg/m^3 increase in $PM_{2.5}$ was associated with a 1.5% increase in total mortality and 2.1% increase in mortality from ischemic heart disease in a study of six eastern U.S. cities. In contrast to Schwartz et al. (*3*), the present study also found a significant association between PM_{CF} and total and cardiovascular mortality. Although Schwartz et al. (*3*) did not find a significant association between coarse fraction and mortality when the results from all six cities were combined, there was an association in Steubenville, Ohio, alone. Such observed differences may have been due to differences in regional coarse fraction composition. In Spokane, Schwartz et al. (*12*) also found no association between coarse particle concentration and total mortality. However, that study only looked at high episodes of coarse particle concentrations resulting from dust storms. Our findings are in agreement with Ostro et al. (*13*), who found a significant association between daily PM_{10} dominated by coarse particles and mortality.

We investigated the possibility that PM_{CF} was a surrogate for dryness by eliminating the days with humidity less than the 25th percentile. Although the association with total mortality was no longer significant, we found a significant association with cardiovascular mortality.

The reason for the negative association between soil and total mortality is unclear. One possible explanation for this observation is related to the fact that the $PM_{2.5}$ cyclone on the DFPSS was replaced with a WINS on 20 December 1996. The sharper cut point reduced the amount of soil intrusion into the $PM_{2.5}$ sample, which could produce soil data that are essentially different between the 1995–1996 period and 1997. To assess this effect, we eliminated all 1997 soil data and reevaluated the RR for total mortality. After removing the WINS data, the association between reconstructed soil and total mortality was not significant. These observations are similar to that of Ozkaynak and Thurston (*28*), who in a study of the association between U.S. mortality rates and particle pollution levels in 1980 found that soil was the least significant predictor of mortality. We also evaluated the association between soil and mortality with only soil data obtained with the WINS. The association was positive and significant ($p < 0.05$) on the concurrent day, but not significant on any other lag days. However, this observation may be due to the low number of days used to evaluate the association between WINS $PM_{2.5}$ soil and total mortality ($n = 377$).

With respect to the elemental components of $PM_{2.5}$, we found that EC was significantly associated with cardiovascular mortality. EC is found in combustion-derived particles, most notably diesel exhaust (*21*). We found that K_S [potassium from vegetative burning (*23*)] was also associated with cardiovascular mortality.

We found several associations that are potentially spurious. The associations with these variables were found with only total mortality and not with cardiovascular mortality. Lead was negatively associated with total mortality at lag day 3, although this may be reflective of the moderate correlation between Pb and soil in Phoenix ($r = 0.49$). Pb may have accumulated in the soil or in road dust from the past use of leaded gasoline. S was also negatively associated with total mortality on lag day 3. At present, the reason for the negative association with S is unclear. However, S accounts for a rela-tively small percentage of the mass of $PM_{2.5}$ (15%). The significant negative associations between total mortality and Pb and S were not consistent with the lack of association between these exposure variables and cardiovascular mortality.

For the gaseous species, we found that total nonaccidental mortality and cardiovascular mortality were strongly associated with CO and NO_2. These observations are similar to those of Burnett et al. (*30*), who found associations between CO and NO_2 and total nonaccidental mortality in Toronto, Canada. Burnett et al. (*30*) also found that cardiac mortality was associated with CO. CO exacerbates cardiac conditions (*10*). CO concentrations are also associated with hospital admissions for cardiovascular disease (*31*). In Phoenix the primary sources of CO and NO_2 are motor vehicles.

The association between SO_2 and cardiovascular mortality was similar to that of Zmirou et al. (*17*), who also found that an increase in SO_2 was associated with an increase in cardiovascular deaths (RR = 1.04). In addition, Zmirou et al. (*17*) found weak but significant association between 1-hr maximum O_3 concentrations and cardiovascular mortality (RR = 1.02). Hoek et al. (*32*) also found an association between total mortality and O_3 in the Netherlands. We found no significant associations with O_3.

The present study demonstrated the use of factor analysis in an epidemiologic study. Using factor analysis, we were able to identify those underlying factors of measured air pollution composition variability that were associated with excess mortality. Poisson regression with factor scores as exposure variables revealed that combustion-related pollutants associated with motor vehicles and vegetative burning as well as fine particulate SO_4 concentrations were significantly associated with cardiovascular mortality. The soil factor, however, was associated with fewer than expected total deaths. These results are

consistent with our time–series results for individual pollutants, specifically CO, NO_2, K_S, EC, OC, and reconstructed soil. It is interesting to note that the factor repre-senting S was significantly associated with cardiovascular mortality, whereas S alone in an individual pollutant model was not associated with cardiovascular mortality. This may be reflective of the contribution of Pb and Br to the S factor.

A unique aspect of this study was the use of the chemical composition data of $PM_{2.5}$ Using such data, we found positive associations between cardiovascular mortality and K_S, OC, and EC as well as the more traditionally measured pollutants CO, NO_2, SO_2, PM_{10}, $PM_{2.5}$, and PM_{CF}. Significant associations were also found with factors associated with incomplete combustion products and particulate S compounds. A limitation of this study is that the factor analysis results are only in terms of the variance explained by each factor, rather than in terms of the quantitative contribution from a specific source category. Although methods are available to include quantitative source apportionments in a time–series framework (*33*), such an analysis is beyond the scope of this initial investigation.

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Attachment A16

Daily Mortality and Air Pollution in Santa Clara County, California: 1989–1996

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Daily Mortality and Air Pollution in Santa Clara County, California: 1989–1996

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Since the last revision of the national particulate standards, there has been a profusion of epidemiologic research showing associations between particulates and health effects—mortality in particular. Supported by this research, the U.S. Environmental Protection Agency promulgated a national standard for particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$). Nevertheless, the San Francisco Bay Area of California may meet this new standard. This study investigates the relationship between daily mortality and air pollution in Santa Clara County (a Bay Area county) using techniques similar to those utilized in earlier epidemiologic studies. Statistically significant associations persist in the early 1990s, when the Bay Area met national air pollution standards for every criteria pollutant. Of the various pollutants, the strongest associations occur with particulates, especially ammonium nitrate and $PM_{2.5}$. The continuing presence of associations between mortality and air pollutants calls into question the adequacy of national standards for protecting public health. *Key words:* air pollution, ammonium nitrate, carbon monoxide, epidemiology, National Ambient Air Quality Standards (NAAQS), ozone, $PM_{2.5}$, Poisson regression. *Environ Health Perspect* 107:637–641 (1999). [Online 25 June 1999]
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The past decade has seen a burgeoning of epidemiologic research investigating the relationship between air pollution and health effects. Dozens of these studies have analyzed the relationship of daily mortality to various air pollutants, especially particulates. The U.S. EPA analyzed many of these studies in Chapter 12 of *Air Quality Criteria for Particulate Matter* (*1*). This criteria document and the later staff report (*2*) concluded that the preponderance of evidence supports a causal connection between fine particulate levels and various health effects, including mortality. This led to the establishment of national standards for particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$).

A previous study (*3*) showed that an association existed between particulates [measured as coefficient of haze (COH)] and mortality in Santa Clara County (SCC), California, during the years 1980–1986. Since that time, the Bay Area Air Quality Management District (BAAQMD) has monitored particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}), and since 1990, the California Air Resources Board has operated $PM_{2.5}$ monitors, including one in SCC. An analysis of SCC $PM_{2.5}$ data shows that SCC would have met the $PM_{2.5}$ standard between 1991 and 1996. The present study is motivated by the concern that, although SCC may attain the new $PM_{2.5}$ standard, particulates there may still cause substantial health effects.

Air quality in SCC. Most of the studies of mortality and air quality have been based on eastern or midwestern U.S. cities, whose air quality dynamics differ markedly from those of the San Francisco Bay Area. Among the gaseous pollutants, ozone and carbon monoxide levels are similar, but Bay Area sulfur dioxide levels are an order of magnitude lower than in the eastern United States. In fact, sulfur dioxide is so low that it is no longer measured in SCC, but nearby San Francisco's 24-hr design value is < 0.01 ppm, compared with typical design values of approximately 0.05 ppm in many eastern cities (*4*).

SCC's particulate composition, dynamics, and sources also differ markedly from those of eastern cities. In eastern cities, ammonium sulfate represents approximately 45% of $PM_{2.5}$ (*1*), whereas in SCC it represents 5%. For many eastern and midwestern cities, particulate levels peak in the summer months (*1*). For SCC, however, particulates (especially fine particulates) are higher in winter. Specifically, mean San Jose, California, $PM_{2.5}$ levels in November, December, and January averaged 25 μg/m^3 in 1990–1996, but < 10 μg/m^3 during the rest of the year.

Wood-burning and ammonium nitrate each contribute approximately 40% of SCC's wintertime $PM_{2.5}$ (*5*). These sources, combined with wintertime stagnation periods, are the main causes of SCC's elevated wintertime particulate levels. As a result of this seasonality, the new SCC 15 μg/m^3 annual standard appears no more stringent than the 65 μg/m^3 24-hr standard (*6*). This is in spite of the EPA's stated intention to make the annual average the more stringent controlling standard (*7*).

Particle size also varies by season. During the winter, SCC $PM_{2.5}$ averages approximately 70% of PM_{10} compared with 50% for the year as a whole. Wintertime PM_{10} is dominated by combustion sources, with approximately 10% coming from geological dust. During the rest of the year, geological dust makes up a larger fraction, marine sea salt becomes significant, and the amount of ammonium nitrate decreases by half.

For several years during the early 1990s, SCC and, in fact, the entire Bay Area, had air quality that complied with air quality standards for all criteria pollutants. Moreover, the Bay Area would have attained the new 8-hr ozone standard and, based on research monitoring results, would have attained the new $PM_{2.5}$ standard, had those standards been in effect. In contrast, in the early 1980s, when the previous study was done, SCC violated the 8-hr CO standard, the 1-hr ozone standard, and would have violated the 8-hr ozone standard had that been in effect. Although PM_{10} and $PM_{2.5}$ were not measured in the 1980s, SCC violated the 150 μg/m^3 total suspended particulate (TSP) standard almost every year.

Methodology

This study attempts to draw on the extensive experience of previous studies to determine a modeling approach. The sensitivity of conclusions to model choice, meteorological adjustment, and covariates has been extensively investigated [e.g., (*1,8–10*)]. These studies have reached similar conclusions, namely that the choices of (reasonable) model and (reasonable) meteorological adjustment do not appear to greatly affect conclusions on the relationship between mortality and particulates, but the inclusion of other air contaminants ofren causes a substantial increase in the standard error of the particulate regression coefficient and sometimes a drop in the level of the coefficient. In other words, there can be substantial confounding of these variables.

Based on these considerations, various models were tried, including Poisson regression with either linear predictors or generalized additive models (GAMs) for temporal and weather variables, and models with an overdispersion fit using quasi likelihood.

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The disadvantage of the GAM approach is that it does not provide simple coefficients. Because the focus is on pollutant variables, however, this lack is not of great concern. The advantage is that the GAM approach is less likely to induce lack of fit. Thus, we will use the GAM approach. Models with an overdispersion parameter are useful for certain deviations from the Poisson model. However, if the Poisson model appeared adequate, it would be used.

The modeling strategy follows that of Samet et al. (*10*), first fitting terms for season and trend, then adding terms for meteorology, and finally adding pollutant terms, with the number of seasonal, trend, and meteorology terms determined by optimizing Akaike's information criterion (AIC).

Tests of goodness of fit. A goodness of fit test of the Poisson model was performed based on deviance. Under the null hypothesis that the data derive from this model, the deviance has an approximately χ^2 distribution with the residual degrees of freedom. Specifically, the χ^2 test is a likelihood ratio test versus a saturated model, where each day is fitted with a different mean. Serious lack of fit would result in unusually large values of the deviance.

Residuals were checked for extreme values. The GAM approach minimizes problems with any nonlinearity between the response and the temporal and weather variables.

To test the sensitivity of the results to the use of GAM, a parallel modeling approach was performed using sine and cosine terms for time and day of year and polynomials in minimum and maximum temperature.

A simulation of the model-fitting process. The statistical significance level for testing a parameter in a model is based on the assumption that the selection of the model was made before the data were gathered. In practice, this is rarely the case, so the *de facto* assumption is that the process of model-building has a minimal effect on the significance level.

An approach to finding more realistic significance levels is to simulate the model-building process itself. To that end, an S-Plus function was developed to simulate the following approximation of model-building. The idea was to simulate data from a true model that contains no pollutant term, then simulate the building up of the model and the fitting of a pollutant variable. The set of pollutant variable coefficients thus obtained should form a more realistic distribution than the simple one where model-building is ignored.

The steps of the simulation were as follows. Initially, a vector of Poisson means was generated by fitting daily mortality data to the seasonal, trend, and weather variables using the GAM approach. An S-Plus function was then invoked repeatedly with different random seeds that performed the following steps:

1. The function simulates a vector of Poisson variates from the initial mean vector.
2. It fits this simulated variate vector to GAM terms for time and day of year in a Poisson regression, increasing the degrees of freedom until there is no improvement in AIC from the addition of another degree of freedom in either GAM term.
3. It uses the optimal number of degrees of freedom for time and day of year from step 2, and adds GAM terms for minimum and maximum temperature, again adding terms until there is no improvement in AIC.
4. It fits the simulated variates to $PM_{2.5}$ in addition to the optimal number of time, day of year, and minimum and maximum temperature GAM terms found in steps 2 and 3, with the fitted $PM_{2.5}$ coefficient output.

The coefficient found from the actual data is then compared to the resulting distribution of simulated coefficients, providing what may be a more realistic *p*-value.

The data. California mortality data were obtained from the California Department of Health Services (Sacramento, CA) for the years 1989–1996. Counts of daily total nonaccidental mortality (henceforth described as mortality), respiratory mortality, and cardiovascular mortality were extracted for SCC residents who died in-county, using the same *International Classification of Diseases, Ninth Revision* (*11*) codes as in the previous study (*3*).

Pollutant data were obtained from the BAAQMD pollutant database. Long-term PM_{10} data were available for only one SCC site—San Jose 4th Street. These data cover the full period on an every-6-day schedule, with an every-other-day schedule during the first 3 years. This site also provided PM_{10} constituents nitrate and sulfate on the 6-day schedule and daily COH values. $PM_{2.5}$ and $PM_{10-2.5}$ were also available from a research model dichotomous sampler that operated at this site from 1990 through 1996 on the same 6-day schedule.

Ozone, carbon monoxide, and nitrogen dioxide data were also obtained for the 4th Street site. Although data for ozone were available from some other SCC sites, these were not included in the interests of simplicity. Because national standards are health-based it seemed reasonable to include variables with averaging times as defined in the standards, namely maximum 8-hr ozone, maximum 8-hr CO, and 24-hr NO_2. Nevertheless, 24-hr CO and ozone were also considered.

Comparisons of 4th Street ozone with other SCC sites consistently show correlations above 0.8 in seasonally adjusted ozone concentrations. Thus, 4th Street ozone concentrations represent a reasonably good surrogate for outdoor ozone exposure in SCC. Based on data from the late 1970s when the district operated a number of COH monitors in SCC, correlations with the 4th Street site were quite high. The correlation between season- and trend-adjusted $PM_{2.5}$ for Fremont and 4th Street was 0.86.

Weather data were obtained from the BAAQMD meteorological database for San Jose Airport. Previous studies have found nonlinear relationships between mortality and weather variables. Because mortality can be affected by both hot and cold weather, it seemed reasonable to consider both minimum and maximum temperature as variables. Therefore, both daily maximum and minimum temperature as well as 24-hr average relative humidity data (rh) were obtained. Missing values were filled in by regressing against temperature and rh values at other nearby BAAQMD meteorological sites—Alviso and Union City.

Comparison with previous results. To compare the results for 1989–1996 with the previous 1980–1986 results it was necessary to reanalyze the earlier results paralleling the new analysis as closely as possible.

For the 1980–1986 reanalysis, $PM_{2.5}$ and PM_{10} and its species were not available. COH was used along with NO_3 from the TSP filter. The other pollutants—NO_2, O_3, and CO—were measured, although the results were read from strip charts and recorded with one less significant digit. San Jose Airport data were not available for this time period; therefore, San Jose city temperatures were used.

Results

Table 1 provides summary statistics for $PM_{2.5}$ related to the new $PM_{2.5}$ standards. Note that from 1993 through 1996 the 4th Street site would have met the new $PM_{2.5}$ standards, based on results from the dichotomous sampler. Although the new $PM_{2.5}$ network will include many sites, the 4th Street site has historically had the highest particulate (PM_{10}) levels in the Bay Area.

Table 1. $PM_{2.5}$ design values (μg/m³): San Jose, CA, 4th Street, 1990–1996.

	1990	1991	1992	1993	1994	1995	1996
98th percentile	88	51	48	50.0	44	32	25
3-Year average			62.4	50.0	47.2	42.0	33.6
Annual mean	18.4	15.5	13.8	12.9	12.6	10.3	9.5
3-Year average			15.9	14.1	13.1	11.9	10.8

$PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter.

Thus, there is a good chance that the Bay Area (including SCC) would have met the standards had $PM_{2.5}$ samplers been in operation at other BAAQMD monitoring sites.

Table 2 presents summary statistics for all the variables considered. Note that $PM_{2.5}$ and $PM_{10-2.5}$ do not sum to PM_{10}, and that the number of observations for the PM_{10} fractions was 408 compared with 823 for PM_{10}. This is because the fractions were measured with a dichotomous sampler whereas the PM_{10} was measured with the district high-volume sampler. Although the dichotomous sampler averages lower than the district sampler, the two PM_{10} measurements have a correlation of 0.94.

Season and trend fits. Rather than predicting mortality from a single temporal GAM term, separate GAM terms in time and day of year were fit because a good-fitting model could be obtained using many fewer degrees of freedom. Terms were added sequentially until there was no further improvement in AIC. The best model contained a GAM term for time with 7 degrees of freedom (*df*) and a day of year term with 12 *df*. The resulting deviance was 3,038.5, with AIC 3,078.5.

Meteorological variables. GAM terms were fit for minimum and maximum temperature in addition to a 7-degree term for time and a 12-degree term for day of year, yielding an optimum AIC with 3 *df* for minimum temperature and 2 *df* for maximum. (Subsequently, this set of GAM terms will be referred to as the optimal GAM terms.) The inclusion of a minimum–maximum cross-product term did not improve the AIC, nor did the inclusion of relative humidity. The model had a deviance of 2,998.1 on 2,897 *df*, and an AIC of 2,897 *df*.

To test for lack of fit, a quasi likelihood model was fit. The overdispersion parameter of 1.02 is barely larger than 1, the value for the Poisson. The *p*-value for a χ^2 of 2,998.1 with 2,897 *df* is 0.09, so that the Poisson model cannot be rejected at the 0.05 level.

Pollutant variables. Table 3 presents partial correlations between mortality and the pollutant variables. Specifically, mortality and each pollutant variable were regressed against the optimal GAM terms, and the residuals saved. Several of the Poisson regressions did not converge, so least squares regressions were used. The table presents the correlations among these residuals.

Of the pollutant measures, $PM_{2.5}$ and NO_3 have the highest partial correlations with mortality. There are also reasonably high correlations with PM_{10} and SO_4. Interestingly, in contrast to other studies, there are actually negative correlations between mortality and the lags (previous day) of these variables. Another change is that COH is only weakly correlated with mortality, although there is a statistically significant correlation with lagged COH. The relationship with 24-hr CO is similar to that of COH. NO_2 is also highly correlated with COH, but lag NO_2 has a lower partial correlation with mortality than unlagged NO_2. The correlation between ozone and mortality is weak, although the correlation with 8-hr ozone is borderline significant.

Except for ozone, there are positive correlations between the other pollutant variables, with high correlations between some of the particulate measurements ($PM_{2.5}$ and PM_{10}, PM_{10} and COH, and $PM_{2.5}$ and NO_3). There are also high correlations between NO_2 and PM_{10}, and NO_2 and COH.

Various combinations of pollutants were tried in Poisson regressions that also included the optimal GAM terms. The results are shown in Table 4. As with the partial correlations, both NO_3 and PM_{10} were highly significant. $PM_{2.5}$, SO_4 and 8-hr ozone were also marginally statistically significant. Among lagged variables, COH and CO were highly significant and NO_2 was marginally so. $PM_{10-2.5}$ was not significant, nor was its lag.

Because NO_3 and $PM_{2.5}$ had the highest partial correlations with mortality, these were included in regressions with other pollutants.

Table 2. Summary statistics for mortality, weather variables, and pollutant concentrations, San Jose CA, 1989–1996.

Variable[a]	Obs (no.)	Mean ± SD	Min	Lower quartile	Med	Upper quartile	Max
Mortality (count)	2,922	20 ± 5	6	17	20	24	40
Respiratory mortality (count)	2,922	2.5 ± 1.7	0	1	2	3	14
Cardiovascular (count) mortality	2,922	8.7 ± 3.2	0	6	9	11	24
Min temperature (°F)	2,922	50 ± 8	22	45	51	56	70
Max temperature (°F)	2,922	70 ± 10	39	62	70	77	101
Relative humidity (%)	2,922	70 ± 12	24	63	70	77	100
PM_{10}[b] (μg/m^3)	823	34 ± 23	6	19	27	40	165
$PM_{2.5}$[b] (μg/m^3)	408	13 ± 13	2	6	9	14	105
$PM_{10-2.5}$[b] (μg/m^3)	408	11 ± 6	0	7	11	15	45
COH (COH units)	2,780	0.5 ± 0.4	0.0	0.2	0.3	0.5	3.1
NO_3 (μg/m^3)	523	3.0 ± 3.7	0.0	0.9	1.8	3.7	33.1
SO_4 (μg/m^3)	534	1.8 ± 1.3	0.0	0.9	1.5	2.5	7.9
O_3 (ppb)	2,856	16 ± 9	0	10	16	22	51
8-hr O_3 (ppb)	2,908	29 ± 15	0	19	29	37	105
CO (ppm)	2,849	1.4 ± 1.0	0.0	0.8	1.1	1.7	7.6
8-hr CO (ppm)	2,865	2.1 ± 1.6	0.2	1.0	1.5	2.5	12.0
NO_2 (ppb)	2,888	28 ± 13	5	19	25	34	96

Abbreviations: COH, coefficient of haze; Max, maximum; Med, median; Min, minimum; Obs, observations; $PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter; SD, standard deviation.
[a]Values are 24-hr averages unless otherwise noted. [b]The fine and coarse fraction of PM_{10} do not add to total PM_{10} because they derive from the dichotomous sampler, whereas total PM_{10} was collected with a separate high-volume sampler.

Table 3. Partial correlations among mortality and pollutant variables.[a]

Variable	Mortality	Mortality vs. lagged variable	PM_{10}	$PM_{2.5}$	$PM_{10-2.5}$	COH	NO_3	SO_4	O_3	8-hr O_3	CO	8-hr CO
PM_{10}	0.116	-0.026										
$PM_{2.5}$	0.142	-0.084	0.849									
$PM_{10-2.5}$	0.037	-0.031	0.649	0.508								
COH	0.026	0.041	0.827	0.772	0.476							
NO_3	0.141	-0.061	0.689	0.830	0.496	0.493						
SO_4	0.100	-0.040	0.509	0.499	0.444	0.318	0.614					
O_3	0.025	-0.013	-0.064	-0.122	-0.026	-0.289	-0.103	0.102				
8-hr O_3	0.037	-0.016	-0.102	-0.153	-0.070	-0.330	-0.108	0.085	0.844			
CO	0.033	0.048	0.609	0.435	0.326	0.736	0.270	0.146	-0.215	-0.210		
8-hr CO	0.029	0.024	0.620	0.542	0.378	0.750	0.285	0.155	-0.236	-0.277	0.810	
NO_2	0.036	0.022	0.800	0.662	0.590	0.826	0.459	0.293	-0.228	-0.219	0.651	0.643

Abbreviations: COH, coefficient of haze; $PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter.
[a]Correlations of residuals from regressions on generalized additive model terms of degree 12 for day of year, degree 7 for time, degree 3 minimum temperature, and degree 2 for maximum temperature.

NO_3 was statistically significant paired with every other pollutant except $PM_{2.5}$. $PM_{2.5}$ was statistically significant paired with every pollutant except NO_3 and PM_{10}. No other pollutant was statistically significant in regressions with either of these pollutants. Two runs of all four categories of pollutant were made (particulate, NO_2, CO, and ozone), using either $PM_{2.5}$ or NO_3 as the particulate variable. In these regressions, the particulate variable was highly statistically significant and the others were not.

Goodness of fit. In the fitted models in Table 4 that included $PM_{2.5}$, the deviance is actually 364.7—less than its degrees of freedom (382). This implies p-values of 0.5 or higher, i.e., there is no indication of lack of fit. Similarly, for the model with NO_3, the deviance is 503.4, with 497 *df*, the p-value is 0.31.

There are no large y-outliers, the largest daily mortality value being 40, approximately 4 standard deviations (SDs) above the mean. Several of the pollutant variables are right-skewed—$PM_{2.5}$ and NO_3 in particular. However, taking the log of $PM_{2.5}$ (after first adding 5 $\mu g/m^3$ to reduce undue influence of small $PM_{2.5}$ values) eliminates much of the skewness, and the transformed variable is still statistically significant in a Poisson regression with the trend, seasonal, and meteorological terms.

The raw mortality values have an autocorrelation of 0.18, but the residuals from the multiple regression of mortality on trend, season, and weather terms has an autocorrelation of only 0.04. Thus, autocorrelation of residuals is not a significant issue.

The fact that the deviance is approximately equal to the value expected under the null hypothesis suggests that it would be difficult to improve the fit substantially. The lack of y-outliers, the lack of influential x-values, and the lack of autocorrelation suggest that the Poisson model fits reasonably well.

Analysis using a parametric approach. To check the adequacy of the GAM approach, a parallel analysis was performed using sine/cosine functions for season and trend, and polynomials for weather variables. The results were similar both qualitatively and quantitatively to those found in Table 4.

A simulation of the model-fitting process. The simulation described in "A Simulation of the Model-Fitting Process" in "Methodology" was repeated 1,000 times. It yielded four fitted coefficients greater than that observed so that, based on the simulation, the p-value is approximately 0.004. This p-value is, if anything, smaller than that found using statistical theory, where the p-value was 0.012.

Comparison with 1980–1986 results. Table 5 presents a reanalysis of the 1980–1986 data using methods paralleling those of Table 4. In particular, the same variables for season, trend, and weather were used (although they were refit with 1980–1986 data). To make coefficients comparable, the same deltas are used; that is, $50 \times SD(p)/SD(PM_{10})$, where the SDs are from the 1989–1996 data.

Generally, the results for the 1980–1986 period are similar to those of 1989–1996. In particular, with the exception of ozone, the coefficient for every pollutant or the lagged pollutant is statistically significant. In pairwise models with lagged COH, the other pollutants are no longer statistically significant. Lagged COH remains highly significant in combination with NO_2 and ozone; with CO, it is not statistically significant, but its regression coefficient is little changed. NO_3 is borderline significant ($p = 0.06$) with the ozone, but not significant with CO or NO_2. Oddly, in combination with NO_3, NO_2 was significant. Note that the sample size for NO_3 is only 354, compared with over 2,000 for the other pollutants. The small sample size makes it more difficult to detect an effect. When both lag COH and NO_3 are in the model, the COH coefficient is smaller and no longer statistically significant, whereas the NO_3 coefficient changes only slightly and is borderline significant ($p = 0.09$).

One difference with the 1989–1996 results is that the 1980–1986 COH coefficient is highly significant, with a relative risk of 1.06, compared with 1.03 for 1989–1996. A comparison of the two coefficients—taking their difference and dividing by the square root of the sum of their sample variances—yields a value of $z = 1.36$, not statistically significant. Pooling the two periods, fitting the same coefficient for season, trend, and weather, but with different COH slopes and intercepts did not result in a statistically significant difference in COH coefficients.

One possible reason that the COH coefficient might have changed is that COH has diminished from the early 1980s to the 1990s. Thus, if the effect of COH is not linear, this could result in different coefficients. However, neither a quadratic nor a hockey-stick function of COH was significant in the pooled regressions for either period.

Respiratory and cardiovascular regressions. Table 6 shows relative risks from Poisson regressions using each pollutant or their lags (depending on which had the greater risk based on Table 4). SO_4 and CO were significantly associated with respiratory mortality.

Table 4. Pollutant relative risks[a] for models[b] with pollutant alone, lagged, and with other pollutants, Santa Clara County, CA, 1989–1996.

Pollutant	PM_{10}	$PM_{2.5}$	$PM_{10-2.5}$	COH[c]	NO_3	SO_4	NO_2[c]	CO[c]	8-hr O_3
Alone	1.08**	1.09*	1.02	1.03	1.07**	1.05*	1.03	1.02	1.03*
Lagged	0.99	0.96	0.98	1.05**	0.98	0.98	1.03*	1.04**	0.99
With									
PM_{10}	–	1.13	·	–	1.07*	–	–	-	
$PM_{2.5}$	0.96	–	0.97	0.99	1.09	1.00	0.96	0.98	1.04
$PM_{10-2.5}$	–	1.13*	–	·	1.11**	–	–	–	
Lag COH[c]	–	1.11*	·	–	1.07**		–	·	–
NO_3	1.02	1.00	0.95	1.01		1.01	0.99	1.01	1.05
SO_4	–	1.10*	·	–	1.07*	–	·	–	–
Lag NO_2[c]	–	1.12**		–	1.08**	–	–	·	–
Lag CO[c]	–	1.11**	–	·	1.07**	–	–	–	–
8-hr O_3	–	1.10**	–	–	1.08**	–	–	–	–
Four-pollutant[d]	–	1.13**		·	–	–	0.96	1.00	1.05
Four-pollutant[d]	–	–	–	–	1.09**	–	0.95	1.06	1.07

Abbreviations: SD, standard deviation; COH, coefficient of haze; $PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter.
[a]Relative risks calculated by $\exp(b \times \Delta p) - 1$, where b is the pollutant coefficient from the Poisson regression, and $\Delta p = 50$ for PM_{10}, corresponding to the increment used in the criteria document (*1*). For other pollutants, p, the increment was $50 \times SD(p)/SD(PM_{10})$; e.g., $SD(PM_{2.5}) = 13$, $SD(PM_{10}) = 23$, so for $PM_{2.5}$, $\Delta p = 50 \times 13/23 = 28$. [b]All models include 7 generalized additive model terms for trend, 12 for season, 3 for minimum temperature, and 2 for maximum temperature. [c]Lagged variables were used if they appeared to fit better lagged than unlagged. Thus, lagged CO and lagged COH were used when fitting jointly with other pollutants. [d]Pollutants are lagged CO, lagged NO_2, 8-hr ozone, and either $PM_{2.5}$ or NO_3.
*Statistical significance at the 0.05 level. **Statistical significance at the 0.01 level.

Table 5. Pollutant relative risks[a] for models[b] with pollutant alone, lagged, and with other pollutants, Santa Clara County, CA, 1980–1986.

	COH	NO_3	NO_2	CO	8-hr O_3
Alone	1.05**	1.04*	1.04**	1.04**	1.01
Lag 1[c]	1.06**	1.00	1.03**	1.05**	1.02
Lag COH[c]	–	1.03	1.01	1.00	1.02
NO_3	1.03		1.08*	1.03	1.03
NO_2	1.05**	1.01			
Lag CO[c]	1.06	1.03	–		
Lag O_3[c]	1.05**	1.04	·		
Four-poll[d]	1.06	–	1.01	1.00	1.02

Abbreviations: COH, coefficient of haze; poll, pollutant; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter; SD, standard deviation.
[a]Relative risks calculated by $\exp(b \times \Delta p) - 1$, where b is the pollutant coefficient from the Poisson regression, and $\Delta p = 50 \times SD(p)/SD(PM_{10})$ for the pollutant, p, where $SD(p)$ and $SD(PM_{10})$ are computed on 1989–1996 data. [b]All models include 7 generalized additive model terms for trend, 12 for season, 3 for minimum temperature, and 2 for maximum temperature. [c]Lagged variables were used if they appeared to fit better lagged than unlagged. Thus, lagged CO, COH, and 8-hr O_3 were used when fitting jointly with other pollutants. [d]Pollutants are lagged CO, NO_2, lagged 8-hr O_3, and lagged COH.
*Statistical significance at the 0.05 level. **Statistical significance at the 0.01 level.

Table 6. Respiratory and cardiovascular mortality relative risks[a] for models[b] with pollutant[c] alone, Santa Clara County, CA, 1989–1996.

	PM_{10}	$PM_{2.5}$	$PM_{10-2.5}$	Lag COH	NO_3	SO_4	Lag NO_2	Lag CO	8-hr O_3
Respiratory	1.11	1.13	1.16	1.07	1.10	1.15*	1.07	1.08*	0.96
Cardiovascular	1.09*	1.07	1.03	1.03	1.09*	1.04	1.02	1.04*	1.02

Abbreviations: COH, coefficient of haze; $PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter.
[a]Relative risks calculated by $\exp(b \times \Delta p) - 1$, where b is the pollutant coefficient from the Poisson regression and $\Delta p = 50$ for PM_{10} and $50 \times SD(p)/SD(PM_{10})$ for other pollutants, p; e.g., $SD(PM_{2.5}) = 13$, $SD(PM_{10}) = 23$, so for $PM_{2.5}$, $\Delta p = 50 \times 13/23 = 28$. [b]All models include 7 generalized additive model terms for trend, 12 for season, 3 for minimum temperature, and 2 for maximum temperature. [c]Lagged variables were used if they appeared to fit better lagged than unlagged. Thus, lagged CO and lagged COH were used when fitting jointly with other pollutants.
*Statistical significance at the 0.05 level.

and $PM_{2.5}$, NO_3, and CO were associated with cardiovascular mortality. For PM_{10}, $PM_{10-2.5}$, NO_3, and CO the point estimates for risk were higher than those in Table 4.

Analyses by season. Analyses were performed by season for pollutants with the highest partial correlations with mortality (Table 7). In most cases, the change in relative risk is not statistically significant. Based on Tukey's studentized range distribution, the risks differ significantly from season to season for NO_3. For the other pollutants, the differences in risk between seasons are not statistically significant.

Discussion

One striking result of the analysis is that although the Bay Area met every air quality standard in the early 1990s (and would have met the new 8-hr ozone and $PM_{2.5}$ standards had they been in effect), there is a statistically significant correlation between each pollutant considered (except coarse fraction PM_{10}) and mortality. Second, the regression coefficients of other pollutants that are correlated with particulates—CO and NO_2—drop to nonsignificance in a regression that also includes some measure of fine particulates (either $PM_{2.5}$ or NO_3), whereas there is little change in the fine particulate coefficients. This suggests that fine particulates (or what fine particulates may be a surrogate for) may be the real culprits. The result that NO_3 had the strongest association with mortality is clearly of practical importance and worth investigating for other areas.

The level of PM_{10} effect found—a relative risk of 1.08 for an increase of 50 μg/m^3 PM_{10}—is larger than that found in many other studies [see the EPA's Table 12-37 and Figure 12-43 (*1*)]. This may reflect a better correlation between monitored values and exposure in SCC. Part of the explanation may be that buildings in SCC are not as tight because of its mild climate, which could lead to a higher correlation of indoor and outdoor particulate levels. A second point is that the correlation between particulate values measured at the San Jose 4th Street monitor and other SCC monitors is high. Particulare levels at the 4th Street monitor exceed those of other SCC monitors (*12*); therefore, the relative risk as a function of SCC average levels could be higher than 1.08.

No evidence for a threshold was found. Although the COH coefficient was substantially lower for the 1989–1996 period than for 1980–1986, the result did not appear to be due to the lower particulate levels in the later period. One point that is important to keep in mind is the role of chance in these comparisons; because there is marginal power to detect effects of this magnitude, some data sets may yield nonsignificant results whereas others yield highly significant results.

Although the results for respiratory and cardiovascular mortality showed fewer significant results than for mortality as a whole, the level of effects appeared somewhat higher. The number of cardiovascular and respiratory deaths is considerably smaller than all deaths (Table 2), so the power to detect an effect is less unless the effect is much larger.

The results by season were ambiguous. The lack of statistical significance of most of the coefficients can be attributed to lack of power. The criteria document (*1*) found that a minimum sample size of 400 was necessary to achieve reasonable power in epidemiologic studies such as this. For $PM_{2.5}$, NO_3, and SO_4, there were approximately 100 observations per season, far below the 400 observations necessary to achieve reasonable statistical power. Nevertheless, a statistically significant difference in effect was found for NO_3, with positive effects for winter, spring, and summer, and a negative effect for fall; although only the winter coefficient was statistically significant, the range of coefficients was larger than expected by chance.

This analysis has found associations between air pollution variables and mortality—especially with fine particulate variables—similar to the levels of associations found in the studies that were used to justify the new $PM_{2.5}$ standards. Yet the Bay Area probably meets these new standards. The new $PM_{2.5}$ standards may be protective in other areas where seasonal PM variations are not as great. In the Bay Area, however, the seasonal variation in $PM_{2.5}$ is large, with winter concentrations averaging more than double that of summer concentrations. The results of this analysis suggest that current national air quality standards, specifically those for particulates, may not be protective of public health for the Bay Area.

Table 7. Relative risks by season.[a,b]

Season	PM_{10}	$PM_{2.5}$	Lag COH	NO_3	SO_4
Spring[c]	1.08	1.07	1.02	1.07	1.06
Summer[d]	1.10	1.05	1.13	1.32*	1.11*
Fall[e]	1.07	1.04	1.08	0.87	1.03
Winter[f]	1.06	1.05	1.04*	1.07*	1.00

Abbreviations: COH, coefficient of haze; $PM_{2.5}$, particulate matter ≤ 2.5 μm in aerodynamic diameter; PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter.
[a]Relative risks calculated by $\exp(b \times \Delta p) - 1$, where b is the pollutant coefficient from the Poisson regression and $\Delta p = 50$ for PM_{10} and $50 \times SD(p)/SD(PM_{10})$ for other pollutants, p; e.g., $SD(PM_{2.5}) = 13$, $SD(PM_{10}) = 23$, so for $PM_{2.5}$, $\Delta p = 50 \times 13/23 = 28$. [b]All models include 7 generalized additive model terms for trend, 12 for season, 3 for minimum temperature, and 2 for maximum temperature. [c]February, March, April. [d]May, June, July. [e]August, September, October. [f]November, December, January.
*Statistical significance at the 0.05 level.

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Attachment A17

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Health Effects from Chronic Low-Level Exposure to Hydrogen Sulfide

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Health Effects from Chronic Low-Level Exposure to Hydrogen Sulfide

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ABSTRACT. The acute toxic effects of hydrogen sulfide have been known for decades. However, studies investigating the adverse health effects from chronic, low-level exposure to this chemical are limited. In this study, the authors compared symptoms of adverse health effects, reported by residents of two communities exposed mainly to chronic, low-levels of industrial sources of hydrogen sulfide, to health effects reported by residents in three reference communities in which there were no known industrial sources of hydrogen sulfide. Trained interviewers used a specially created, menu-driven computer questionnaire to conduct a multi-symptom health survey. The data-collection process and questions were essentially the same in the reference and exposed communities. The two exposed communities responded very similarly to questions about the major categories. When the authors compared responses of the exposed communities with those of the reference communities, 9 of the 12 symptom categories had iterated odds ratios greater than 3.0. The symptoms related to the central nervous system had the highest iterated odds ratio (i.e., 12.7; 95% confidence interval = 7.59, 22.09), followed by the respiratory category (odds ratio = 11.92; 95% confidence interval = 6.03, 25.72), and the blood category (odds ratio = 8.07; 95% confidence interval = 3.64, 21.18). Within the broader health categories, individual symptoms were also elevated significantly. This study, like all community-based studies, had several inherent limitations. Limitations, and the procedures the authors used to minimize their effects on the study outcomes, are discussed. The results of this study emphasize the need for further studies on the adverse health effects related to long-term, chronic exposure to hydrogen sulfide.
<Key words: chronic exposure, hydrogen sulfide, industrial sources, low level>

HYDROGEN SULFIDE (H_2S) is a highly toxic gas that occurs naturally in the environment (e.g., in volcanic gases, marshes, swamps, sulfur springs, decaying matter) and is a byproduct of many industrial processes, such as paper manufacturing. Ambient levels of H_2S are not measured routinely,[1] but available information indicates levels in the low parts per billion (ppb) range.[1,2] Levels of H_2S in industrial settings can be in the range of 0.5–10 parts per million (ppm).[3] Spills, leaks, malfunctions, or the accumulation of H_2S in enclosed

workplaces or low-lying areas can result in much higher levels that can be highly toxic and quickly lethal. Exposure to H_2S is one of the leading causes of sudden death in the workplace.[1] In the United States, investigators attributed at least 5,563 exposures and 29 deaths to this chemical between 1983 and 1992.[4] In 1977, the National Institute for Occupational Safety and Health (NIOSH) estimated that approximately 125,000 employees in 73 industries were potentially exposed to H_2S in the United States.[5] Atmospheric release of H_2S represents the most significant public health concern of the growing geothermal energy industry.[6]

Many occupational and community studies, as well as studies of laboratory animals, have documented the adverse health effects of acute exposure to relatively high levels of H_2S.[2,7–9] The mechanism of action of H_2S involves interaction with a number of enzymes and other macromolecules, including hemoglobin and myoglobin. A critical target enzyme of H_2S is cytochrome oxidase.[10] Most organ systems are susceptible to the effects of H_2S; therefore, this toxic gas has often been regarded as a broad-spectrum toxicant. Organs and tissues most susceptible to H_2S toxicity, however, are those with exposed mucus membranes and those with high oxygen demands.[9] Neurotoxicity of the central nervous system (CNS) and pulmonary edema are well-documented consequences of acute H_2S poisoning.[7,9] Cardiovascular toxicity and gastrointestinal disturbances are also associated with exposure to this chemical.[7,9]

Although many individuals are exposed regularly to relatively low levels of H_2S through their jobs, or because they live in communities near industrial emissions of H_2S, studies investigating the adverse health effects of chronic, low-level exposures to this compound are limited. In one study, former workers and citizens who lived downwind from the processing of crude oil had neurophysiological abnormalities. Residents who lived near the plant were exposed to H_2S at 10 ppb—with periodic peaks to 100 ppb.[11] In a study of sewer workers, evidence indicated that low-level exposure to H_2S may be associated with reduced lung function.[3] Residents in an area of Finland located near paper mills reported an excess of adverse health symptoms (e.g., respiratory, eye, nasal symptoms) in comparison with a reference community,[12,13] although results in children did not reach statistical significance.[14] H_2S accounted for two-thirds of the total released sulfur compounds monitored in the reported studies.[15] Recent evaluations of hospital discharge records in Rotorua, New Zealand, which is situated near a geothermal field, showed an elevated prevalence of diseases of the nervous system and eye.[16] The results of several additional community and occupational studies indicate a considerable variety of adverse health effects from low-level, chronic exposure to H_2S.[17]

In this study, we compared symptoms of adverse health effects, reported by residents of two communities exposed mainly to chronic, low levels of industrial sources of H_2S, to the health effects reported by residents in three reference communities in which there were no known industrial sources of H_2S.

Materials and Method

We used a multi-symptom health survey, with multiple descriptive variables, to compare self-reported symptoms in communities exposed to low levels of H_2S to self-reported symptoms in reference communities with no known exposure to H_2S. The same questionnaire and the same data-collection and data-evaluation techniques were used for all communities.

Communities exposed to H_2S

Odessa, Texas. Odessa is a community of approximately 96,000 persons, located in the heart of the Permian Basin in West Texas. Beginning in about 1967, certain areas of this community were exposed to H_2S produced as a result of microbial action in solar ponds of industrial wastewater containing sulfate ions. Air modeling, performed in conjunction with a lawsuit based on citizen complaints, indicated that, in 1992, the levels of H_2S 1.6 km (1 mi) from the ponds were (a) 500–750 $\mu g/m^3$ (335–503 ppb), for a maximum 8-hr measurement; (b) 150–300 $\mu g/m^3$ (101–201 ppb), for a maximum 24-hr measurement; and (c) 3–40 $\mu g/m^3$ (7–27 ppb), for an annual average (Jim Tarr, personal communication). The ponds in this community were closed and reconstructed between 1995 and 1996.

Puna, Hawaii. Puna is a district located on the island of Hawaii, which is situated on the Kilauea East Rift Zone. This area is volcanically active and is a site for geothermal energy production. Since 1976, geothermal wells in the Puna area have been drilled for the purpose of generating electrical power. The Puna Geothermal Venture (PGV) currently generates up to 25% of the electricity on the island of Hawaii.[18] There are several fixed, elevated air monitors currently operating in the Puna area. Complete data from the fixed monitors, however, are not easily accessible, thus making it difficult for investigators to establish a clear and precise documentation of the recorded levels of H_2S exposure in this community. Available monitoring data from the 1990s indicate numerous H_2S hourly averages documented in the low-ppb range in the area near the PGV plant. Measurements of monitors run by the Hawaii Department of Health in the area during this same time period also revealed levels in the low-ppb range in 1996–1997, although most hourly measurements were less than 1 ppb or were not detectable.[18] As part of an investigation of the H_2S exposures in the area surrounding PGV in 1997, the Agency for Toxic Substances Disease Registry[18] (ATSDR) was provided with 29 incident reports that spanned the time period from June 1996 to July 1997. In these reports, the peak concentration detected was 301.7 ppb.[18] Periodic releases of H_2S in the range of 200–500 ppb have also been reported during other years.

Reference communities

Hilo, Hawaii. Hilo is a community located approximately 33.8 km (20 mi) to the northwest of the PGV plant. Hilo is exposed to natural volcanic emissions that

are similar to those of Puna, but there are no geothermal power plants or known sources of industrial H_2S in the Hilo area. Therefore, we would expect that exposure to H_2S in this community would be much less severe than exposures in Puna or Odessa.

Midlothian, Texas. Midlothian is a community of approximately 5,100 people, located approximately 48 km (30 mi) south of the Dallas-Fort Worth metropolitan area in Texas. The local economy is made up of both agricultural and industrial enterprises. Industries in the area include three cement companies and a secondary steel mill. There are no known industrial sources of H_2S in the area.

Waxahachie, Texas. Waxahachie, a community of about 19,000 people, is located 45 km (28 mi) south of Dallas, Texas. The local economy is mainly agricultural and small business or industrial enterprises. There are no known industrial sources of H_2S in the Waxahachie area.

Selection of subjects. Selection of subjects in the communities potentially exposed to H_2S was conducted as follows. In Odessa, a random selection was made from a list of plaintiffs (about 600) compiled by a legal firm involved in a community investigation. Participants in Odessa had to have lived in the community for 5 or more y, and only those who lived within 1.6 km (1 mi) of the H_2S source were selected. We recruited participants in Puna by phone; we used the local phone book to identify persons who lived within 8 km (5 mi) of the PGV source. Eligible participants had to have lived in the community for at least 5 y. Flyers, which were posted in Puna at various places in the community, also informed persons about the health survey. The nature of the study, or the fact that the study involved industrial exposures, was not revealed or discussed with potential participants. The one exception to this was that several flyers, posted at only one location, encouraged persons to participate in the study "if they believed PGV was making them sick." This error was made by one of the individuals in the community who helped with the study. These flyers were discovered and were removed by the study coordinator after being posted less than 1 full day.

Volunteer participants in the reference communities had to have lived in their community for at least 5 y, and they had to live within 8 km (5 mi) of a specific zip code selected as a point of reference. Participants in Midlothian and Waxachachie were selected by random phone calls within a certain geographic area. Participants in Hilo were also recruited by this method, as well as by posted flyers that were similar to those used in Puna.

All participants signed a consent form evidencing their agreement to participate in the study. The exposed group included 126 participants from the Odessa community and 97 participants from the Puna community ($n = 223$). The reference group included 58 participants from Midlothian, 54 participants from Waxahachie, and 58 participants from Hilo ($n = 170$).

Survey instrument and data collection. The survey instrument was a specially created, menu-driven questionnaire that was extensive and very structured. The questionnaire covered a broad spectrum of symptoms within 12 different categories of physical health or function. The questionnaire also included detailed information on demographics, life style, reproductive history, cancer history, and other medical information. In all 12 categories, reported symptoms had to originate or worsen after the person moved into the community, and symptoms had to occur with sufficient frequency to constitute a significant health problem. For certain symptoms, a predetermined number of occurrences were necessary (e.g., persistent bronchitis [2 or more episodes per year], persistent cough [daily], and frequent headache [1 or more per week]). Copies of the complete questionnaire are available from the authors upon request.

Trained interviewers administered the questionnaire during face-to-face interview sessions. Interviewers entered data directly into laptop computers, and the data were later downloaded to a central computer for storage and analysis. We used coded identification numbers for each subject to protect the confidentiality of the participants of the study.

The carefully constructed and refined structure of the questionnaire used for this study diminished the potential for error. Several internal checks served as indicators of data quality and, at several places in the questionnaire, respondents had to meet certain criteria before the interviewer could proceed from that point with questioning. In general, information had to be entered in the correct format for all activated data fields before the program would advance to the next field. The study coordinator also personally checked responses from each interview to be sure that they were completed, that respondents met any specific criteria required for certain areas of questioning, and that information was entered correctly in the proper format.

Interviewer selection and training. The interviewers for the 5 community surveys were previously trained graduate students and staff from the University of Texas Medical Branch, and community residents who were recruited and specially trained to assist with the study. The study coordinator personally trained each interviewer in the precise methods that had been developed for the proper use of the computer program. The coordinator also supervised mock interviews between interviewers for training purposes, and the coordinator observed each interviewer as he or she conducted the actual interviews. The study coordinator was accessible at all times during interview sessions to address any questions or potential problems.

Statistical analyses. Preliminary analysis included a 3×2 chi-square cross-comparison of the three reference communities, as well as a 2×2 chi-square cross-comparison of the two potentially exposed communities. We conducted these comparisons to determine if there were any significant differences between the two exposed communities and the three reference communities that would prevent them from being combined into the larger exposed and reference groups. Prevalence rates for total symptoms within each of the 12

Table 1.—Comparison of Symptom Categories in the Reference and Exposed Groups

Symptom/category	Reference population: Midlothian, Texas (*n* = 58) *n*	%	Waxahachie, Texas (*n* = 54) *n*	%	Hilo, Hawaii (*n* = 58) *n*	%	Reference groups (*n* = 170) χ^2 (*df* = 2)	Exposed population: Odessa, Texas (*n* = 126) *n*	%	Puna, Hawaii (*n* = 97) *n*	%	Exposed groups (*n* = 170) χ^2 (*df* = 1)
Central nervous system	25	43	19	35	14	24	0.1	112	89	82	85	0.45
Ear/nose/throat	31	53	26	48	24	41	0.43	110	87	84	87	0.96
Respiratory*†	19	42	5	12	8	14	< .01	58	66	54	70	0.68
Muscle/bone	17	29	25	46	16	28	0.07	79	63	58	60	0.76
Skin	15	26	10	19	13	22	0.65	58	46	56	58	0.11
Immune	15	26	11	20	11	19	0.64	79	63	55	57	0.44
Cardiovascular	23	40	27	50	20	34	0.24	78	62	53	55	0.34
Digestive	10	17	8	15	8	14	0.87	55	44	40	41	0.82
Teeth/gums	6	10	1	2	8	14	0.07	55	44	31	32	0.10
Urinary	10	17	9	17	4	7	0.19	37	29	27	28	0.92
Blood	3	5	3	6	1	2	0.53	37	29	24	25	0.54
Endocrine‡	10	17	11	20	1	2	0.01	29	23	15	15	0.22

*Reference group comparison, excluding Midlothian; χ^2 *p* value is .64.
†We excluded smokers from this category because confounding was possible.
‡Control comparison, excluding Hilo; χ^2 *p* value is .86.

health categories covered in the questionnaire, as well as prevalence rates for individual symptoms within certain categories, were tabulated for the combined reference group (*n* = 170) and the combined exposed group (*n* = 223). For certain categories, comparisons of the two exposed groups were made individually with the combined reference group. For all comparisons of the study and reference groups, we used Fisher's exact test to establish *p* values. Odds ratios (ORs) and their 95% confidence intervals (CIs) were also estimated.

Given the potential for a strong confounding effect of smoking on respiratory problems, smokers were removed from all analyses of respiratory symptoms. Also, as noted below, and in lieu of the findings reported in Table 1, Midlothian was removed from the reference group for analyses of respiratory symptoms, and Hilo was removed from the reference group for analysis of endocrine-system-related symptoms.

Results

Demographic characteristics of the samples drawn from the 5 exposed and reference populations are listed in Table 2. The percentages of subjects in each of the categories are similar for the exposed and reference groups, except with respect to ethnicity and age. The Odessa population was primarily African-American and Hispanic, whereas Puna and the three combined reference communities were 76% and 84% white and non-Hispanic, respectively. The mean age of the Waxahachie group (63.7 y) was higher than that of any other group, but the overall mean for the reference population was 56.2 y, which was only slightly higher than the mean of 50 y for the exposed population.

To determine if the reference groups could be combined in the analyses of individual symptoms, chi-square *p* values were calculated for a cross-comparison between each of the three reference communities (Table 1). Symptoms in the two exposed populations were also compared, and they were not significantly different in any category. The three reference populations were different ($p < .05$) in only two categories: respiratory and endocrine. The Midlothian population reported a high number of respiratory symptoms (Table 1). This was an expected finding because the population was near several cement kilns, thus resulting in exposure to emissions containing potential respiratory irritants.[19] When we excluded Midlothian from the control comparison for the respiratory category, the chi-square *p* value for respiratory symptoms between the remaining two reference communities was .64. Similarly, and as noted in Table 1, the Hilo population had only 1 subject who reported endocrine symptoms. When we removed Hilo from the reference group comparisons for endocrine symptoms, the chi-square *p* value between the remaining two reference groups became .86. In light of these differences, we excluded Midlothian from the respiratory category and excluded Hilo from the analysis of endocrine symptoms. For all other symptom categories, the three reference groups were not significantly different from each other and, therefore, were combined for analysis.

The numbers of symptoms reported and corresponding percentages in each of the 12 symptom categories for the two combined exposed groups and for the three combined reference groups are shown in Table 3. Fisher's exact *p* values, odds ratios, and 95% confidence intervals are also listed. As noted in the table, the reported numbers of symptoms in the exposed group were significantly higher than in the reference group ($p < .001$) for every symptom category, with the exception of the endocrine categeory. When we compared the exposed communities individually to the combined ref-

Table 2.—Demographic Characteristics of Exposed and Reference Populations

Characteristic	Exposed population						Reference population							
	Odessa, Texas (*n* = 126)		Puna, Hawaii (*n* = 97)		Total exposed population (*n* = 223)		Midlothian, Texas (*n* = 58)		Waxahachie, Texas (*n* = 54)		Hilo, Hawaii (*n* = 58)		Total reference population (*n* = 170)	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Age (y)														
Mean	51.7		48.2		50		49.6		63.7		55.2		56.2	
Range	14–83		17–78		14–83		16–76		29–86		18–86		16–86	
Under 40	33	27*	22	23	55	25	15	26	5	9	5	9	25	15
40–59	39	32*	63	65	102	47	27	46	16	30	33	57	76	45
60+	50	41*	12	12	62	28	16	28	33	61	20	34	69	41
Male	46	37	36	37	82	37	20	34	24	44	22	38	66	39
Female	80	63	61	63	141	63	38	66	30	56	38	62	104	61
Ethnicity														
White-Non-Hispanic	2	2	74	76	76	34	55	95	54	100	33	57	142	84
African-American	87	69	0	0	87	39	0	0	0	0	1	2	1	0.6
Hispanic	37	29	4	4	41	18	2	3	0	0	1	2	3	2
Other	0	0	19	20	19	9	1	2	0	0	23	40	24	14
Years at residence														
< 10	2	2*	53	55	55	25	17	29	3	5	27	47	47	28
10–29	33	28*	42	43	75	35	26	45	16	30	20	34	62	36
30+	85	71*	2	2	87	40	15	26	35	65	11	19	61	36
Smokers†	38	30	20	21	58	26	11	20†	9	17†	12	21	32	19

*Age was unavailable for 4 Odessa participants, and years at residence for 6 Odessa participants were unavailable.
†Smoking information was unavailable for 2 participants in Midlothian and for 2 participants in Waxahachie.

Table 3.—Self-Reported Symptoms in Various System Categories: Total Exposed (*n* = 223) Versus Total References (*n* = 170)

System categories	Total exposed Puna/Odessa		Total reference Midlothian/Waxahachie/Hilo		Iterated OR	95% CI	Fisher's Exact Probability
	n	%	*n*	%			
Central nervous system	194	87	58	34	12.7	7.59, 22.09	< .001
Ear/nose/throat	194	87	81	48	7.24	4.37, 12.42	< .001
Respiratory*	112	68	13	15	11.92	6.03, 25.72	< .001
Muscle/bone	137	61	58	34	3.06	1.99, 4.77	< .001
Skin	114	51	38	22	3.6	2.27, 5.82	< .001
Immune	134	60	37	22	5.35	3.36, 8.74	< .001
Cardiovascular	131	59	70	41	2.03	1.33, 3.12	< .001
Digestive	95	43	26	15	4.05	2.44, 6.96	< .001
Teeth/gums	86	39	15	9	6.31	3.46, 12.32	< .001
Urinary	63	28	23	14	2.48	1.44, 4.42	< .001
Blood	60	27	7	4	8.07	3.64, 21.18	< .001
Endocrine†	44	20	21	19	1.06	0.58, 1.98	.88

Notes: OR = odds ratio, and CI = confidence interval.
*In the respiratory comparison, Midlothian was excluded from the reference group; data from smokers were excluded from the respiratory comparison; and smoking information was unavailable for 2 participants in Midlothian and for 2 participants in Waxahachie.
†In the endrocrine comparison, Hilo was excluded from the reference group.

erence communities, similar differences were reported, with the endocrine category being the only category that was not statistically different. Nine of the 12 symptom categories resulted in odds ratios that exceeded 3.0. Symptom categories with the highest odds ratios were the CNS category (OR = 12.7; 95% CI = 7.59, 22.09), the respiratory category (OR = 11.92; 95% CI = 6.03, 25.72), and the blood category (OR = 8.07; 95% CI = 3.64, 21.18). In Figures 1–3, a breakdown of these three categories is presented.

The percentages for the individual symptoms within the CNS category are shown in Figure 1. Statistical anal-

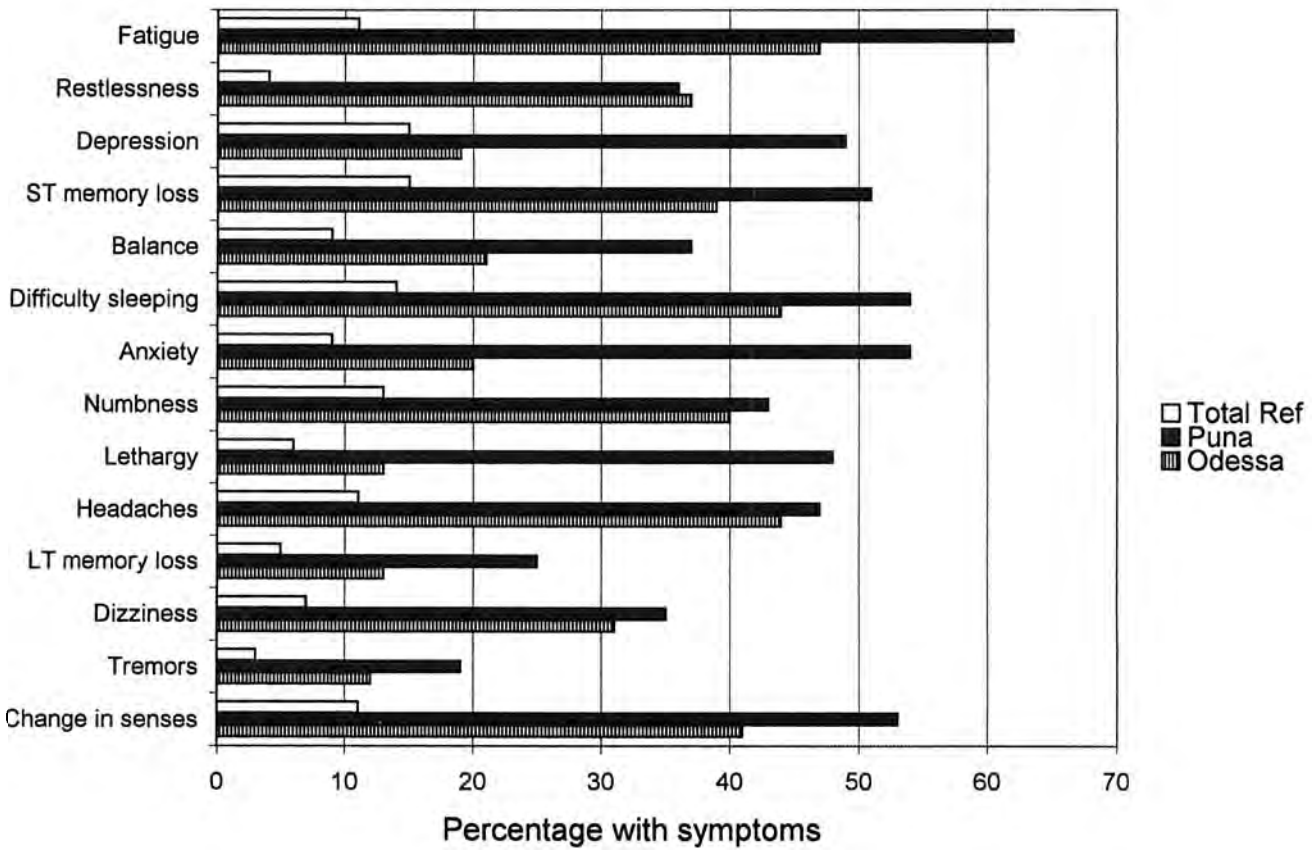

Fig. 1. Comparison of percentage of central nervous system symptoms among the combined reference populations and the hydrogen-sulfide-exposed Puna and Odessa populations. ST = short-term, and LT = long-term.

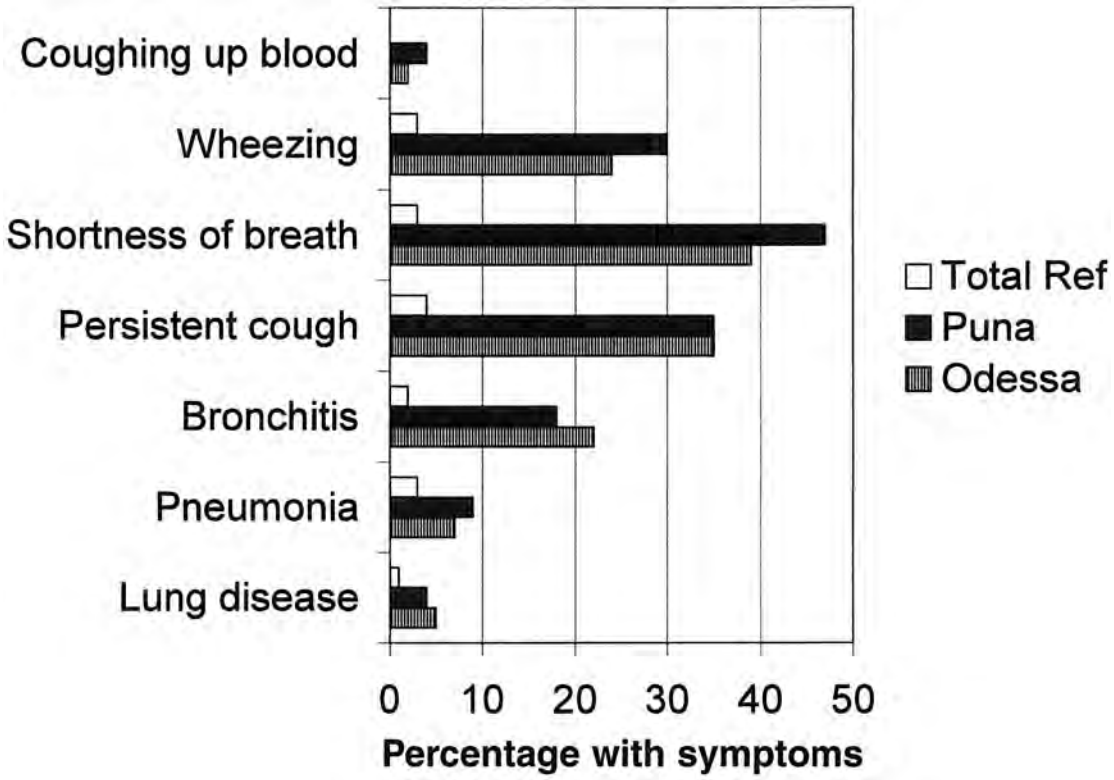

Fig. 2. Comparison of percentage of respiratory symptoms among the combined reference populations and the hydrogen-sulfide-exposed Puna and Odessa populations.

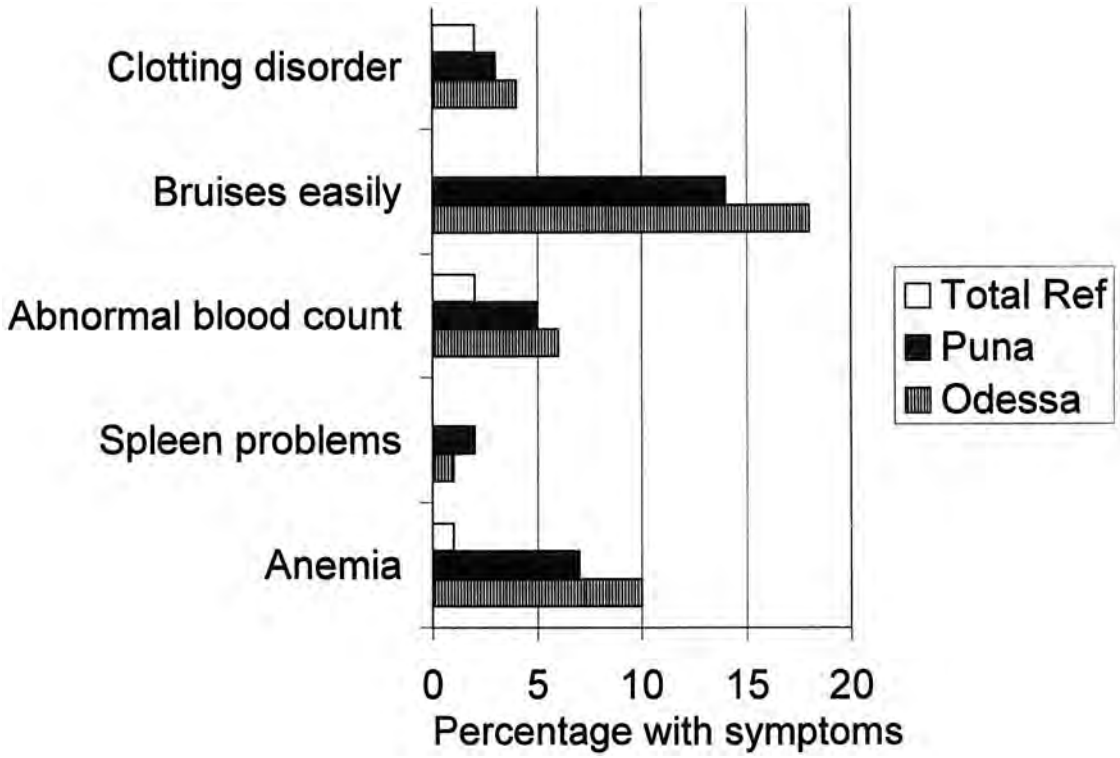

Fig. 3. Comparison of percentage of blood-related symptoms among the combined reference populations and the hydrogen-sulfide-exposed Puna and Odessa populations.

ysis of the data revealed that when we compared the Odessa and Puna communities, they were significantly different ($p < .05$) in 6 of 14 individual categories (i.e., long-term memory loss, lethargy, anxiety, balance, depression, and fatigue). The two exposed communities were compared separately to the combined reference communities. For the exposed population in Odessa, the odds ratio for each of the individual symptoms was elevated significantly over the symptoms reported by the combined reference populations ($p < .05$)—except for depression. For the exposed population in Puna, the odds ratios for all individual symptoms were significantly elevated over the combined reference populations ($p < .01$). It should be noted that neuropsychological evaluations were also conducted on the participants from Puna. These objective measurements supported and confirmed the CNS findings of this health-symptom survey.[20]

Individual respiratory symptoms are compared and summarized in Figure 2. For both exposed populations, the same four respiratory symptoms (i.e., bronchitis, persistent cough, shortness of breath, and wheezing) were significantly different from the combined reference populations ($p < .001$). In a comparison of the two exposed communities, the odds ratios were not significantly different for any of the individual categories.

The comparisons of individual symptoms in the blood category are shown in Figure 3. In both exposed communities, the same two symptoms (i.e., anemia and bruises easily) were significantly different from the reference group ($p < .05$). All other individual blood symptoms were not significantly different. The odds ratios for the two exposed communities were not significantly different from each other for any of the individual categories.

Discussion

In this multi-symptom health survey, we identified a variety of adverse health symptoms that appear to be associated with chronic exposure to low levels of H_2S. In this study, self-reported, adverse effects in every category analyzed, except the endocrine system, are documented. Within the broader health categories, a number of specific symptoms were reported, with significantly higher frequencies in the two exposed groups than in the reference communities. Although some differences were noted between the two exposed populations, the findings in these two independent groups were generally very similar. Additional strengths of this study include the consistency of the interviewing process and the use of identical questionnaires for all subjects in our study. There were also several objective neuropsychological tests and measurements that were performed on subjects in the exposed Puna and the reference Hilo communities.[20] The results of these tests support findings within the important CNS symptom category.

There were also limitations in our study. Surveys of self-reported symptoms may be susceptible to response enhancement bias (i.e., an increase in reported symptoms resulting from the fact that respondents are aware of, and sensitized to, the fact that they are exposed). Our study was vulnerable in this area given the rather intense political controversy that characterized the history of

H_2S exposure in the Puna community. In Odessa, the community was involved in a lawsuit related to H_2S exposure, and this involvement may have contributed to a potential response bias for this group. However, the results of a recent study indicate that plaintiffs in a legal case may be no more likely to "enhance" answers than the general population.[21] Also, the survey results for Puna and Odessa were very similar, even for symptoms that the community respondents would likely have not expected to be related to H_2S exposure.

A standard problem in studies that use subjective questionnaires is the potential for recall bias. In our study, we used a detailed questionnaire with internal checks to help control for this bias. It is impossible, however, with only subjective data, to completely avoid this problem, but we minimized the effect by using identical questionnaires for all of the subjects in all groups. There is also the potential for interviewers to introduce systematic errors in the data if there is a special way they ask certain questions, encourage certain responses, or if there are differences in the ways they interpret and transcribe answers. The design and methodology of this survey minimized the potential for this type of error or bias in at least two important ways. First, the use of computerized, menu-driven questionnaires, which allowed for direct input of data into the computers, enhanced the accuracy and consistency of the interviews. Second, the extensive training of the interviewers included supervised mock interviews, and the study coordinator also carefully supervised the actual subject interviews.

There were several possible confounders that could affect the findings of our study. Differences were observed in age, smoking behavior, and ethnicity between the exposed and reference groups. As noted in Table 1, Puna (a 76% white, non-Hispanic community) and Odessa (primarily an African-American and Hispanic community) differed significantly in terms of their ethnicity. However, there were no statistically significant differences in the responses of the subjects in these two communities in the broader symptom categories. It appears, therefore, that any potential ethnic effects were not sufficiently great to influence the major findings of this investigation. With respect to age, the mean age for the reference group (56.2 y) was slightly higher than that of the exposed group (50 y). Given that older individuals might have a higher number of adverse health symptoms, we would expect that any age effect in this study would have resulted in an underestimate of the true differences between the exposed and reference groups. As noted previously, to minimize the possible confounding effects of smoking in this study, we removed smokers from the analyses of all respiratory symptoms.

A final limiting factor that should be noted is often present in environmental studies of this type. In the Puna community, only a portion of the monitoring data from the fixed, elevated monitors was available for this investigation, thus making exposure difficult to analyze. Concurrent exposures to other chemicals, in addition to H_2S, in the Odessa community also clouded the exposure picture. Hence, it was impossible for us to know what the "true" exposure was for each individual, or even for groups of individuals, who lived in a certain area.

Despite the listed limitations, the two potentially exposed communities were very similar in terms of adverse health symptoms reported within each broad category. They were also very similar in the subcategories of respiratory and blood symptoms. Some differences were noted in individual symptoms in the CNS category, but the exact reason for this difference is unclear. Perhaps the difference may result, in part, from the stressful atmosphere in Puna that is related to the intense political dissension about geothermal development in that area.

On the basis of information contained in the available literature about acute toxic effects of H_2S exposure, increased health effects in the exposed populations (e.g., CNS, respiratory, and ear, nose, and throat symptoms) could be anticipated.[11–13,22] Other categorical symptoms in the exposed groups are less commonly associated with H_2S exposure. Information exists, however, that supports some of our unique findings. For example, changes in enzymes involved in heme metabolism have occurred in individuals exposed to H_2S.[23,24] Alterations in iron metabolism have also been noted.[25] There is also evidence from studies in rats that H_2S interferes with bacterial inactivation in the lung.[26] Studies in humans have also indicated that, as levels of sulfur compounds[13] decrease, the incidence of respiratory infections in individuals also decreases.[27]

This study, like all community-based observations, requires extension and confirmation. However, our results, providing evidence of the elevated prevalence of adverse health symptoms in communities potentially exposed to low levels of H_2S, emphasize the need for further studies on the effects of this toxin.

Given that we still do not know whether adverse effects are associated with low-level exposure to industrial chemicals in residential settings, studies such as this multi-symptom, community-health survey should prove valuable. However, community-based epidemiological studies, even conducted under the best of circumstances, do have important limitations, as discussed above. Despite these limitations, we anticipate that studies such as this one will play an ever-increasing role in identifying potential health problems related to chronic exposures to toxic substances. A committee of the National Academy of Science recognized the limitations of traditional environmental epidemiological studies, but it asserted that a community symptom survey, coupled with pertinent knowledge gained from laboratory studies, could lead to "causal inference" about the effect of potentially toxic chemicals on human populations.[28] The findings in our study, taken together with previously reported data concerning adverse responses to H_2S, strongly mandate the need for continued research on the possible detrimental effects of chronic exposure to this toxic agent. This is of decided public health significance, given the relatively large segment of the population that is regularly exposed to low levels of H_2S.

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Attachment A18

Symptomatic Effects of Exposure to Diluted Air Sampled from a Swine Confinement Atmosphere on Healthy Human Subjects

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Aerial emissions from a swine house at North Carolina State University's field laboratory were diluted to a level that could occur at varying distances downwind from a confined animal feeding operation (CAFO) both within and beyond the property line, and these emissions were delivered to an environmental exposure chamber. The study design consisted of two 1-hr sessions, one in which 48 healthy human adult volunteers were exposed to diluted swine air and another in which they were exposed to clean air (control). Objective measures of blood pressure, temperature, heart rate, respiratory rate, lung function, nasal inflammation, secretory immunity, mood, attention, and memory were correlated with objective measures of air quality. Ratings of perceived (self-reported) health symptoms were also obtained. The mean levels of airborne constituents in the swine air condition were hydrogen sulfide (24 ppb), ammonia (817 ppb), total suspended particulates (0.0241 mg/m^3), endotoxin (7.40 endotoxin units/m^3), and odor (57 times above odor threshold). No statistical differences on objective measures of physical symptoms, mood, or attention resulted from the 1-hr exposure to swine emissions in the environmental chamber when compared with clean air for healthy human volunteers. However, subjects were 4.1 (p = 0.001) times more likely to report headaches, 6.1 (p = 0.004) times more likely to report eye irritation, and 7.8 (p = 0.014) times more likely to report nausea in the swine air (experimental) condition than in the control condition. These results indicate that short-term exposure in an environmental chamber to malodorous emissions from a swine house at levels expected downwind can induce clinically important symptoms in healthy human volunteers. *Key words:* airborne emissions, attention, environmental chamber, memory, mood, nasal inflammation, pulmonary function, secretory immunity, spirometry, swine. *Environ Health Perspect* 113:567–576 (2005). doi:10.1289/ehp.6814 available via *http://dx.doi.org/* [Online 9 February 2005]

The rapid proliferation of confined animal feeding operations (CAFOs) that house thousands of animals at a single facility has raised public health concerns for workers as well as neighbors who live in adjacent communities (Schiffman et al. 1995; Thu et al. 1997; Wing and Wolf 2000). One focus of this concern has been potential human health effects from airborne agents that emanate from livestock houses, waste storage systems, and manure application sites. Aerial emissions from CAFOs are composed of a mixture of hydrogen sulfide (H_2S), ammonia, volatile organic compounds (VOCs), and particulates including bioaerosols that arise during biodegradation of manure (Sweeten 1988). VOCs, ammonia, and H_2S in the emissions are found in the gas phase as well as adsorbed to particulates (Schiffman 1998; Schiffman et al. 2001a).

Occupational studies of workers at CAFOs have documented a variety of health complaints as well as objective health effects including respiratory inflammation and dysfunction. Common health complaints among workers at animal production facilities include sinusitis, chronic bronchitis, nasal mucous membrane inflammation, nasal and throat irritation, headaches, and muscle aches and pains (University of Iowa Study Group 2002). Objective measures of lung function using spirometry have shown both acute cross-shift decline in lung function as well as chronic respiratory impairment in workers at confined swine and poultry feeding operations (Donham 1993; Donham et al. 1977; Schwartz et al. 1992, 1995). Progressive decline in lung function among CAFO workers occurs over a period of years. Furthermore, acute exposures to high levels of H_2S from agitated manure can lead to reactive airway distress syndrome, permanent neurologic damage, and even death (Centers for Disease Control and Prevention 1993; Schiffman et al. 2001a).

Studies of potential health risks to community residents living in the proximity of CAFOs have been more limited than investigations of occupational risks. Several controlled studies in North Carolina and Iowa, however, have found that self-reported health symptoms are elevated in communities adjacent to intensive swine facilities. A field study in Iowa found that persons residing within a 2-mile radius of a 4,000-head swine operation reported higher frequencies of 14 out of 18 physical health symptoms, especially respiratory symptoms, than did a control group in an area with no intensive livestock operations (Thu et al. 1997). Residents of a rural North Carolina community with a 6,000-head hog operation reported increased symptoms of headache, runny nose, sore throat, excessive coughing, diarrhea, burning eyes, and reduced quality of life compared with residents in rural communities with intensive cattle operations or without livestock facilities (Wing and Wolf 2000). Furthermore, residents near swine facilities in North Carolina reported more tension, more depression, more anger, more fatigue, and more confusion at the time when the odors were strongest than did a control group of unexposed persons (Schiffman et al. 1995). No objective medical tests of physical health symptoms, however, were obtained in these community studies near CAFOs.

The purpose of the present investigation was to build upon previous occupational health and epidemiologic studies that have reported health symptoms associated with exposure to swine emissions. In this study, we used an exposure chamber to systematically investigate the physiologic and psychological responses in human volunteers that result from an exposure to a known level of emissions of swine confinement air in a controlled environment. The environmental chamber was built next to a swine facility, and air from a swine house was diluted to a level that could occur downwind from a confined swine operation both within and beyond the property line. This method of exposure was novel in that it enabled an assessment of the symptomatic effects of an environmentally relevant mixture of well-characterized pollutants in a group of self-selected healthy volunteers. The objective was to determine whether healthy human subjects voluntarily exposed to diluted air from a swine confinement house in a controlled environment (e.g., environmental chamber) experienced altered lung function, nasal inflammation, psychological changes, or other health symptoms related to such an exposure. Use of the human chamber allowed direct dose–response assessment of potential acute health effects from a specified level of airborne emissions.

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Materials and Methods

Overview

The study design consisted of two 1-hr sessions, one in which human subjects were exposed to diluted swine air (experimental condition) and another in which they were exposed to cleaned air (control condition). Objective measures of lung function, nasal inflammation, secretory immunity, mood, respiratory symptoms, attention, and memory were correlated with objective measures of air quality. The concentration of odor, particulates, H_2S, ammonia, VOCs, and endotoxin in exposure chamber air were monitored throughout the study. The maximum exposure duration of 1 hr was requested by the Duke Institutional Review Board because health complaints have been reported to North Carolina agencies from ≤ 1 hr of exposure.

Subjects

Forty-eight healthy adults, ranging in age from 19 to 49 years (mean age = 26.0 ± 9.46 years), participated in this study. Half of the subjects were males and half were females. The group consisted of 33 Caucasians, 11 African Americans, 2 Hispanics, and 2 Asian Americans. The subjects were volunteers recruited by advertisements that were posted in workplaces throughout the Research Triangle community of North Carolina (Durham, Chapel Hill, and Raleigh). Potential subjects were prescreened by telephone to determine their eligibility for study participation. Those who met the inclusion criteria were enrolled sequentially in the order that they called. Enrollment stopped when 24 males and 24 females who met the inclusion criteria were enrolled.

To meet the criteria for the study, subjects were required to be healthy adults. Exclusion criteria were history of asthma (present or past), allergies for which they took prescription medications, smoking (not current smokers and never smoked > 10 packs of cigarettes in their lifetime), use of chronic prescription medications (except birth control pills), history of heart or lung disease, significant occupational exposure, and pregnancy. The mean height of the group was 67.4 ± 3.97 inches; the mean weight of the group was 171.4 ± 38.5 lbs. Subjects were paid $500.00 for their participation. All subjects signed a consent form approved by the Duke University Institutional Review Board that indicated their willingness to participate in "an experiment on exposure to air from swine operations." All 48 subjects completed the study, and none experienced a serious adverse event.

Exposure Facility

An exposure facility was constructed at the Swine Unit of North Carolina State University's field laboratory. The exposure chamber (12 × 16 ft) was adjoined by a medical examination room 8 × 12 ft (Figure 1). The exposure chamber accommodated eight subjects who were seated at a table with dividers so that they could not speak to or observe each other (Bottcher et al. 2002). The ventilation system was custom designed so that it could deliver either totally cleaned air drawn from outside through an air cleaning unit (control condition) or emissions drawn from the exhaust fans of an adjacent swine building diluted with cleaned air (experimental condition). The cleaned air was generated from outside air processed by a packaged air-cleaning unit (model 6500; Allerair Industries, Laval, Québec, Canada) that consisted of an in-series arrangement of a prefilter and HEPA filter for particulate matter removal and two cartridges containing activated charcoal granules specially formulated for removal of gaseous pollutants expected from swine facilities. Particulates were not directly filtered or removed from the swine building air stream so that they would be incorporated into the exposure room airflow. The indoor airflow pattern within the exposure chamber was symmetrical in order to eliminate variability in air quality in the microenvironments of each of the eight subjects. The walls of the facility were insulated and paneled with waterproof materials that did not absorb odors and could be completely cleaned between trials to eliminate residual odorant compounds and particulates from surfaces. The air conditioning system was sufficient to maintain the chamber at a constant temperature (70°F) with eight subjects in the room.

Exposure Conditions and Monitoring of Airborne Constituents

Subjects participated in two 1-hr exposures, one in which they were exposed to diluted swine air (experimental condition) and another in which they were exposed to cleaned air (control condition). The levels of gases, VOCs, particulates, endotoxin (a cell wall component of gram-negative bacteria), and odor in the experimental condition simulated concentrations that could occur downwind of swine production facilities both within and beyond the property line. Although higher concentrations than those tested here can potentially occur intermittently during sporadic spraying of fields with aerosolized liquid from the lagoons that hold decomposing waste, the levels used in this study are representative of air emissions both within and beyond the property line in the absence of spraying. Simulation of downwind exposure was achieved by the custom-designed air flow system that provided a variable method of mixing clean air with swine building air to allow a range of dilution ratios. The mean levels of the H_2S, ammonia, particulates, endotoxin, and odor in the two exposure conditions are given in Table 1. All means in the experimental condition were significantly different from those in the control condition as determined by *t*-tests.

Techniques to Quantify Airborne Emissions

H_2S was measured continuously with a Jerome 631-X H_2S analyzer (Arizona Instrument, Tempe, AZ) that uses a gold film sensor selective for H_2S without interference from sulfur dioxide, carbon dioxide, carbon monoxide, and water vapors. Ammonia was measured continuously with the model 17C chemiluminescence ammonia analyzer (Thermo Environmental Instruments, Franklin, MS). Total suspended particulate concentrations were measured in real time by the HAZ-DUST EPAM-5000 environmental particulate air monitor (Environmental Devices Corporation, Haverhill, MA) that uses aerodynamic particle sizing and an in-line filter cassette for gravimetric sampling. Endotoxin

Figure 1. Exposure facility. Reprinted from Bottcher et al. 2002 with permission.

Table 1. Mean levels of the H_2S, ammonia, particulates, endotoxin, and odor in the two conditions along with the instruments used for air quality measurements.

Emission	Instrument used for measurement	Control condition	Experimental condition
H_2S	Jerome meter	0 ppb	24 ppb
Ammonia	Chemiluminescence analyzer	46.4 ppb	817 ppb
Total suspended particulates	HAZ-DUST	0.0136 mg/m^3	0.0241 mg/m^3
Endotoxin	LAL assay	0.63 EU/m^3	7.40 EU/m^3
Odor	Scentometer and nasal ranger	0.3 D/T[a]	56 D/T
Odor	AC'SCENT olfactometer	—	57 D/T

[a]D/T (dilutions to threshold) indicates the dilution ratio at which the sample has a probability of 0.5 of being detected under the conditions of the test.

was collected on fiberglass filters placed in a 47-mm in-line filter holder (model 2220; Gelman Sciences, Pall Corporation, East Hills, NY) connected to a piston pump that was calibrated at 46 L/min (Rietschle Thomas, Sheboygan, WI). The endotoxin was eluted from the filters with 15 mL deionized water. Endotoxin on the filters was quantified using a *Limulus* amebocyte lysate (LAL) assay (Bio-Whittaker, Walkersville, MD), and the concentration in endotoxin units (EU) was calculated (EU per milliliter). The concentration was multiplied by the elution volume to get the total EUs in the sample (total per filter). The concentration of endotoxin in the air was then calculated using the pump speed (46 L/min) and collection period (60 min). Odor levels in the chamber were measured in two ways. Real-time monitoring of odor levels was performed with the Scentometer (Barnebey-Sutcliffe, Columbus, OH) and the Nasal Ranger (St. Croix Sensory, Lake Elmo, MN). The Scentometer and Nasal Ranger are portable devices that can determine the number of dilutions necessary to reach threshold (i.e., odor dilution-to-threshhold; D/T). In addition, air samples from the exposure chamber were obtained in Tedlar bags during each trial, and odor thresholds were determined in the laboratory by a trained panel using an AC'SCENT olfactometer (St. Croix Sensory). The mean value for each of the above variables in a given condition was maintained within 8% of the overall mean in Table 1 for each test session. Variability within a session was also limited to 8% using a plenum in the inlet with data integrated over 5-min intervals.

VOCs were measured in two ways. First, real-time monitoring of VOCs at ppb levels was performed with the ppbRAE VOC monitor PGM-7240 (RAE Systems, Sunnyvale, CA) that uses a photoionization detector that can detect VOC concentrations down to a few parts per billion. Second, air samples were obtained in canisters and analyzed by gas chromatography and mass spectrometry (GC/MS) and gas chromatography/flame ionization detection (GC/FID) at the U.S. Environmental Protection Agency (EPA; Research Triangle Park, NC). Mean total VOCs were numerically elevated in the experimental condition compared with the control condition using both the ppbRAE and GC techniques, but this did not reach statistical significance. The mean exposure in the experimental condition as determined by GC/FID was 344.2 ± 27.6 ppbC (parts per billion carbon) and in the control condition, 322.7 ± 21.3 ppbC.

Study Design

Each subject participated in two separate sessions that were at least 10 days apart. In one session, subjects were exposed to filtered air pumped into the exposure chamber for 1 hr (control session); in the other session, subjects were exposed for 1 hr to air from the swine house that had been diluted with uncontaminated air (experimental session). Eight subjects were tested at a time, resulting in 12 total sessions for all 48 subjects. Half the subjects participated in the experimental session first, and the other half participated in the control session first.

A series of physiologic and psychological measurements were obtained at four time points on each of the two exposure days: just before exposure, during the 1 hr exposure (at 30 min into the exposure), directly after exposure (at 1 hr), and 2 hr after the end of exposure (3 hr after beginning the exposure). The measurements assessed vital signs (blood pressure, temperature, heart rate, respiratory rate), pulmonary function (spirometry), nasal inflammation (using nasal lavage), total salivary IgA, mood [Profile of Mood States (POMS) scale (McNair et al. 1992)], attention, memory, and other symptoms. The timeline for these measurement types is given in Table 2.

Vital signs. Blood pressure and heart rate were measured using a Dinamap Pro 100 monitor (GE Healthcare—Critikon Division, Jupiter, FL). A Welch Allyn SureTemp thermometer (model 679; Welch Allyn Medical Products, Skaneateles Falls, NY) with an oral probe and a disposable Welch-Allyn probe cover were used to measure temperature. Respiratory rate was determined by counting the number of breaths each subject took in a 30-sec time interval and then multiplying that number by 2.

Table 2. Timelines for physiologic and psychological measurements.

Just before exposure	30 min into exposure	1 hr (end of exposure)	2 hr after end of exposure
Vital signs[a]		Vital signs	Vital signs
Spirometry		Spirometry	
Nasal lavage			Nasal lavage
Salivary IgA		Salivary IgA	
Mood	Mood	Mood	Mood
Attention and memory	Attention and memory	Attention and memory	Attention and memory
Odor ratings	Odor ratings	Odor ratings	
EEHQ		EEHQ	EEHQ

EEHQ, Environmental Exposures and Health Questionnaire.
[a]Blood pressure, temperature, heart rate, respiratory rate.

Spirometry. Forced vital capacity (FVC), forced expiratory volume at 1 sec (FEV_1), and averaged forced expiratory flow between the full expiration of 25 and 75% of the total FVC (FEF 25–75%) were assessed in triplicate using a KoKo Portable Spirometer and KoKo Pulmonary Function Testing Software (PDS Instrumentation, Louisville, CO). FVC is the maximal volume of air (in liters) released during the forced maximal expiration. FEV_1 is the volume of air that was expired in the first second of the forced maximal expiration. FEF 25–75%, measured in liters per second, gives an indication of the condition of the subject's smaller airways. The pulmonary function testing software indicated which of the three trials was the best for each subject. The best trial from the preexposure testing was compared with the best trial from the postexposure testing to determine if there were any changes in the subjects' pulmonary functioning. Subjects' height and weight were measured and recorded at the first visit because this information was necessary to analyze the pulmonary function data.

Nasal lavage. The nasal passages of study subjects were lavaged with 10 mL saline (0.9% sodium chloride; Abbott Laboratories, Chicago, IL), before and 3 hr after initiation of exposure (2 hr after completion of exposure) to swine facility air and to cleaned air. Subjects sat in a chair with their heads tilted back. They were given a plastic straw and instructed to blow into the straw while blocking the other end of the straw with a finger to close the passageway between the nose and the throat. Five milliliters of saline solution (warmed to body temperature) were introduced into each naris using a needleless 10-cc syringe and were held in the nares for 10 sec. The contents of the nares were then expelled into a 120-mL sterile specimen container. The sample was then transferred from the specimen container to a 15-mL centrifuge tube. The samples were put immediately on ice and transferred to the laboratory for analysis. Lavage fluids were treated with *N*-acetyl cysteine to disrupt mucus, and the cells were pelleted by centrifugation. Total cell counts were also determined by enumeration using a hemacytometer. Cytospin preparations of cells were stained for differential analysis. The nasal lavage supernatants were frozen at –70°C for cytokine analysis. The levels of the proinflammatory cytokines interleukin (IL)-1β and IL-8 were quantified because of their recognized importance in lipopolysaccharide-induced airway responsiveness (Jagielo et al. 1996; Wang et al. 1998). Both polymorphonuclear cells (PMN) and IL-8 are also known to increase dramatically in the lungs of persons who spend several hours inside of swine buildings (Larsson et al. 1997; Senthilselvan et al. 1997). Undiluted specimens of nasal lavage fluid were analyzed using

Quantikine enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, Minneapolis, MN) for the proinflammatory cytokines IL-8 and IL-1β.

Salivary IgA. Unstimulated saliva samples were collected using a sterile 2.0-mL vial and one-third of a plastic straw. Subjects uncapped the vial, placed the straw into the vial, and passively drooled down the straw for 90 sec. The samples were then collected and immediately placed in a freezer. They were later sent to Salimetrics LLC (State College, PA) on dry ice, where they were analyzed for salivary IgA. These measurements were obtained because Avery et al. (In press) found that persons exposed to strong swine odors had reduced levels of salivary IgA. All samples were assayed for salivary IgA in duplicate using a highly sensitive enzyme immunoassay (EIA) developed by Salimetrics. The test used 25 μL saliva, has a lower limit of sensitivity of 2.5 μg/mL, a range of sensitivity from 2.5 to 540 μg/mL, and average intra- and interassay coefficients of variation 5.6 and 8.79%, respectively. Method accuracy, determined by spike recovery, and linearity, determined by serial dilution, are 108 and 101%. Intermethod correlations for salivary IgA levels from saliva samples (n = 21) assayed using the Salimetrics EIA protocol and a radial diffusion assay, and the Salimetrics EIA protocol and a commercially available salivary IgA ELISA, were r(19)-values = 0.94 and 0.91 (p-values < 0.0001), respectively. The salivary IgA levels returned by the Salimetrics EIA protocol (mean ± SD = 379.39 ± 261.47 μg/mL) and the comparison ELISA (mean ± SD = 365.81 ± 311.53 μg/mL) were not statistically distinct. Salivary IgA levels returned by radial immunodiffusion were significantly higher (mean ± SD = 675.21 ± 467.94) than levels from both immunoassay protocols.

Mood. The POMS questionnaire was used to assess mood. The POMS is a highly sensitive standardized scale that, based on subjects' responses, measures six different aspects of transient mood: anger–hostility, confusion–bewilderment, depression–dejection, fatigue–inertia, tension–anxiety, and vigor–activity. The POMS has been used previously to evaluate mood changes in response to odors by neighbors of swine operations (Schiffman et al. 1995). The POMS questionnaire has been extensively tested and validated; it has been widely used to evaluate the degree to which behavioral and treatment interventions as well as environmental factors affect mood. The form of the scale used here consists of 30 different feelings (Appendix 1) on which subjects rated "how they are feeling at the present time" on scales coded from 0 (not at all) to 4 (extremely).

Attention and memory. We used a digit span test to measure levels of attention and memory. The test was a modified version of the digit span test used on the Weschler Adult Intelligence Scale, in which a researcher reads strings of simple numbers to a subject, and the subject repeats the numbers back to the researcher in the correct order. The test was presented visually in the present study rather than orally so that the results were not affected by the qualitatively different voices of several researchers who administered the test. Each subject was presented with strings of simple numbers (from 1 to 9) using flashcards, beginning with a string of two digits and presenting one digit per second. After each string of numbers, the subject was shown a flashcard that read, "recall numbers." The subject then recalled the digits in the order in which they were presented by writing them down. Each subject was given 10 sec between the time that they saw the "recall numbers" flashcard and the time that they were presented the next string of numbers to recall and write down the string of digits. After each recall, a new string of digits was presented, with each successive string increased by one digit until the subject recalled the last string consisting of 9 digits. Because the digit span test was administered to the subjects four times at each visit, four different sets of cards were made using random number generation. The same four sets were used at the subjects' second visit, but the sets were presented to the subjects at different time points at the second visit. The subject's score was the length of the last string of numbers accurately recalled.

Odor ratings. The perceived odor was rated on three global 9-point line scales numbered from 0 to 8. These included odor intensity, irritation intensity, and hedonic ratings. For odor and irritation intensity, the scale was labeled as follows: 0, none at all; 1, very weak; 2, weak; 3, moderate weak; 4, moderate; 5, moderate strong; 6, strong; 7, very strong; and 8, maximal. The descriptors for pleasantness/unpleasantness were 0, extremely pleasant; 1, very pleasant; 2, moderately pleasant; 3, slightly pleasant; 4, neither pleasant nor unpleasant; 5, slightly unpleasant; 6, moderately unpleasant; 7, very unpleasant; and 8, extremely unpleasant. Subjects also rated an additional five scales to characterize the odor using the intensity scale above: "musty, earthy, moldy," "fecal," "like urine," "sewer odor," and "sweaty."

Environmental Exposures and Health Questionnaire. Subjects indicated how much, if at all, they were affected by 48 different symptoms on this questionnaire (Appendix 2). The Environmental Exposures and Health Questionnaire (EEHQ) was developed by the U.S. EPA Health Effects Research Laboratory and has been used previously to assess health symptoms from odors (Schiffman 1998). Subjects made their ratings on four different categories: don't have symptom at all (0), mildly affected (1), moderately affected (2), severely affected (3).

Description of statistical methods. For all but one outcome, we estimated two equations of the general form:

$$y_2 = \tau_1 \text{ exposure} + \tau_2 \text{ period} + \tau_3\, y_1 + e \quad [1]$$

$$y_2 = \tau_1 \text{ exposure} + \tau_2 \text{ period} + \tau_3\, y_1 + \tau_4 \text{ exposure} \times \text{period} + e, \quad [2]$$

where y_1 and y_2 are the pre- and postexposure scores on an outcome, "exposure" is a dummy variable coded 1 for swine air and 0 otherwise, and "period" is a dummy variable coded 0 for those who received clean air first, and 1 for those who received swine air first. In Equation 1, the coefficient for exposure (τ_1) estimates its effect on y_2 with preexposure score and period-related differences controlled. As shown by Kessler and Greenberg (1981), this coefficient is equivalent to the effect of exposure on (time 2 – time 1) change in the dependent variable controlling for other independent variables in the equation. Our significance tests for the effect of exposure on each dependent variable are based on this coefficient from Equation 1. The (exposure × period) product term in Equation 2 was used to test whether the effect of exposure differed according to whether swine air was administered first or second. On all but one dependent variable (discussed below), this testfor the presence of a carryover effect was negative.

The analysis focused on potential effects of exposure on seven general classes of outcome variables: vital signs, pulmonary function (spirometry), nasal inflammation (cytokines and cell counts), saliva measures (salivary IgA), mood (POMS), memory/attention (digit span), and self-reported symptoms. Several of these classes, including vital signs, self-reported symptoms, mood, and digit span, contained multiple measures after exposure commenced. Because we did not hypothesize delayed effects of exposure on these specific outcomes, we tested whether exposure-related differences were present at multiple time points after exposure only if a significant effect was present for the first measurement after exposure. Given the exploratory nature of the study, we did not correct for multiple tests. However, given the p-values and magnitudes of most significant effects, the positive findings are not the result of chance. We return to this issue in the discussion of the findings.

All outcomes other than respiratory symptoms were analyzed as continuous dependent variables. We used SAS PROC MIXED (SAS Institute, Cary, NC) to obtain generalized least squares estimates of the coefficients (τ) in Equations 1 and 2, with between-subject

variance treated as a random effect and removed from the error term in significance testing. As discussed by Verbeke and Molenberghs (1997), generalized least squares estimators are more "efficient" (have smaller variance) compared with corresponding ordinary least squares estimators.

On self-reported symptom measures, nearly all respondents had scores of 0 or 1. Therefore, each self-reported symptom measure was coded as a (0/1) variable scored 1 for the presence of any symptoms, and SAS PROC GENMOD was used to estimate Equations 1 and 2 as logistic generalized estimating equations. Between-subject variance was again treated as a random effect, making these models the logistic equivalent of those estimated in PROC MIXED for the continuous outcomes. To examine potential nonproportionality (nonequivalence) of effects between those with and those without self-reported symptoms at baseline, we performed two analyses for self-reported symptoms.

Results

Results of significance testing for effects of exposure on change in an outcome. First, Equations 1 and 2 were estimated for all respondents. Then respondents reporting any preexposure symptoms were dropped, and our models were re-estimated excluding preexposure score (y_1) as a control. [An average of four respondents was excluded across self-reported symptom outcomes (maximum = 12) in the second set of analyses]. Results were essentially the same for both logistic analyses. In Table 3 we report those based on the full sample of respondents. For each dependent variable, we present *p*-values for whether change in an outcome is significantly different in the exposure group compared with the control group. When significant differences are present, we give regression coefficients estimating the effect of exposure (vs. control) on change in a dependent variable. Unlike the raw group differences in the descriptive tables, these coefficients are estimated controlling for initial (preexposure) status and for period of exposure.

None of the measures of vital signs, pulmonary function (spirometry), nasal lavage, salivary IgA, mood, or digit span score was significantly related to exposure. Two nasal lavage measures were related to exposure. Compared with controls, the (time 1–time 2) decrease in percentage of epithelial cells was greater among those exposed to swine air. The exposure group also had a larger increase in percentage of lymphocytes but not in absolute numbers of lymphocytes. Three (of 11) measures of the self-reported symptoms were significantly related to exposure. Based on the logistic odds ratio, when subjects were exposed to swine air, they were 4.1 (p = 0.001) times more likely to report headaches, 6.1 (p = 0.004) times more likely to report eye irritation, and 7.8 (p = 0.014) times more likely to report nausea than in the control condition. Significant exposure-related differences on headache were still present at time 3. None of the pulmonary or mood measures was related to exposure.

Descriptive statistics. The means ± SDs for physical measures (vital signs, nasal lavage, salivary measures, and pulmonary function) over time are given in Table 4. Results of pulmonary function studies are presented as percentage of predicted values based upon population norms. It is customary to report the magnitude of change as percent change from baseline. Means ± SDs for scores on POMS at four time points are shown in Table 5. Means ± SDs for scores on digit span at four time points are shown in Table 6. Table 7 gives the number of persons who self-reported specific symptoms.

Odor perception. All subjects perceived an odor in the experimental condition and very little odor in the control condition, with no overlap of ratings in the two conditions by any subject. The mean odor intensity during the experimental exposure was 5.29 (moderate strong to strong) compared with 1.46

Table 3. Results of significance testing for effects of exposure on change in an outcome (effect coefficients are given for significant effects only).

	Group differences on time 2 – time 1 change		*p*-Value, group differences at time 3[a]
	p-Value	Coefficient	
Vital signs			
Heart rate	0.78		
Respiratory rate	0.57		
Temperature	0.27		
Systolic blood pressure	0.70		
Diastolic blood pressure	0.29		
Blood pressure ratio (systolic to diastolic)	0.52		
Spirometry			
Percent change FEV_1	0.98		
Percent change FVC	0.80		
Percent change FEF 25–75%	0.88		
Salivary measure			
Salivary IgA (μg/mL)	0.57		
Mood scores (POMS)			
Anger	0.97		
Confusion	0.83		
Depression	0.45		
Fatigue	0.52		
Anxiety	0.39		
Vigor	0.52		
Total mood	0.55		
Digit span test			
Digit span score	0.35		
Nasal lavage			
IL-8 (pg/mL)	0.11		
IL-1β (pg/mL)	0.38		
Cell counts	0.76		
Percent epithelial cells	0.02	(b = –21.1)[b]	—[c]
Percent lymphocytic cells	0.008	(b = 23.0)	—[c]
Percent PMNs	0.22		
Absolute epithelial cells	0.15		
Absolute lymphocytic cells	0.78		
Absolute PMNs	0.27		
Self-reported symptoms			
Headache	0.001	(OR = 4.1)[d]	0.01
Sore throat	0.27		
Itchy throat	0.12		
Eyes irritated	0.004	(OR = 6.1)	0.07
Eyes tearing	—[e]		
Nasal congestion	0.76		
Nasal secretion	0.22		
Nasal irritation	0.34		
Difficulty breathing	—[e]		
Cough	0.66		
Nausea	0.014	(OR = 7.8)	0.57

[a]The *p*-value for time 3 is based on a test of whether the (time 2 – time 1) group differences persist at time 3. The time 3 test was performed only when group differences on (time 2 – time 1) were statistically significant. [b]The b-coefficient obtained from SAS PROC MIXED represents the metric effect of exposure on an outcome at time 2 controlling for period and preexposure (time 1) differences. [c]No time 3 measures were obtained for these outcomes. [d]The odds ratio (OR) coefficient estimated with SAS PROC GENMOD is the exponentiated logistic effect of exposure on the odds of any symptom at time 2 controlling for period and preexposure differences. [e]Model did not converge because of low prevalence at time 2.

(very weak to weak) in the control condition. The mean irritation intensity during the experimental exposure was 3.77 (moderate weak to moderate) compared with 0.73 (very weak) in the control condition. The mean unpleasantness during the experimental exposure was 6.21 (moderately unpleasant to very unpleasant) compared with 4.12 (neither pleasant nor unpleasant to slightly unpleasant) in the control condition. The rank order of the mean intensities on the odor adjectives in the experimental condition was fecal > sewer odor > musty, earthy, moldy > like urine > sweaty.

Discussion

The results of this study indicate that a 1-hr exposure to odorous swine air in an environmental chamber (at levels that could occur downwind from a swine facility both within and beyond the property line) has no significant acute effects on vital signs, lung function, nasal inflammation, salivary IgA, mood, attention, or memory in healthy volunteers. That is, there were no statistical differences on objective measures of physical symptoms, mood, or attention that resulted from a 1-hr exposure to air emissions from a swine facility when compared with clean air in persons who self-selected to participate in the exposure study. However, self-reported symptoms of headaches, eye irritation, and nausea were significantly more prevalent in these healthy volunteers exposed to swine air for 1 hr compared with clean air. The rapid onset of exposure-related avoidance symptoms reported by our subjects in response to diluted swine air is consistent with epidemiologic studies (Thu et al. 1997; Wing and Wolf 2000) in which persons "downwind" from swine facilities report similar symptoms.

The underlying mechanism responsible for the headaches, eye irritation, and nausea is not known, but it is unlikely that a single constituent of the emissions induces these effects. As explained below, no single component in the airborne emissions was present at a high enough concentration to be wholly responsible for these symptoms. However, additivity or synergy among the combined components may be the cause of these physical symptoms (Schiffman et al. 2000). That is, the symptoms may be caused by the combined load of some or all of the components in the air (H_2S, ammonia, VOCs, particulates, and endotoxin). Another possibility is that these self-reported symptoms are innate or learned warning signals of potential health effects at higher concentrations or with prolonged exposure.

Endotoxin. Headache, eye irritation, and nausea have been reported in previous studies by persons exposed to endotoxin (Crook et al. 1991; Melbostad and Eduard 2001; Poulsen et al. 1995a, 1995b; Thorn and Kerekes 2001). Endotoxin is also known to contribute to airway inflammation and airflow obstruction (Kline et al. 1999). However, it is unlikely that the endotoxin levels experienced by the subjects in this study are wholly responsible for these self-reported symptoms. The levels of endotoxin to which the subjects were exposed in the chamber were orders of magnitude lower than levels inside swine buildings (e.g., 3,984 EU/m^3 reported by Zhang et al. 1998). Furthermore, the levels used in the experimental condition are far lower than ambient air endotoxin in office buildings (0.25–0.4 μg/m^3) that have been associated with health complaints (Teeuw et al. 1994). (If one assumes that the biologic activity per mass unit of endotoxin is 10 EU/ng in this study, the exposure is approximately 0.06 ng/m^3 in the clean air condition and 0.74 ng/m^3 in the experimental condition.)

The cumulative exposure to endotoxin over 1 hr in the experimental condition of this study is also far below the level expected to cause physiologic symptoms. Assuming a

Table 4. Means ± SDs for vital signs, salivary measures, nasal lavage, pulmonary function, and the digit span test over time.

	Condition	Before exposure	1 hr (end of exposure)	2 hr after end of exposure
Vital signs				
Heart rate	Experimental	70.85 ± 14.61	65.02 ± 13.36	65.79 ± 11.95
	Control	69.96 ± 11.49	64.73 ± 13.39	64.81 ± 12.07
Respiratory rate	Experimental	17.50 ± 4.24	17.25 ± 3.86	16.63 ± 4.84
	Control	17.04 ± 3.67	16.75 ± 3.19	16.88 ± 3.25
Temperature	Experimental	97.97 ± 0.74	97.85 ± 0.64	97.63 ± 0.50
	Control	97.83 ± 0.61	97.72 ± 0.66	97.57 ± 0.58
Systolic blood pressure	Experimental	122.27 ± 15.27	120.44 ± 15.67	123.88 ± 14.61
	Control	121.63 ± 15.32	119.73 ± 14.72	121.85 ± 15.35
Diastolic blood pressure	Experimental	66.44 ± 10.23	66.33 ± 10.02	67.52 ± 11.42
	Control	64.15 ± 10.71	65.33 ± 10.60	69.13 ± 9.62
Nasal lavage				
IL-8 (pg/mL)	Experimental	396.1 ± 448.4	NA	190.6 ± 213.0
	Control	385.0 ± 321.7	NA	268.4 ± 310.2
IL-1β (ng/mL)	Experimental	10.6 ± 21.8	NA	3.5 ± 8.3
	Control	4.6 ± 6.5	NA	4.6 ± 10.3
Cell counts	Experimental	205541.7 ± 442500.2	NA	240364.6 ± 505983.6
	Control	146937.5 ± 332148.5	NA	277354.2 ± 1155336.9
Percent epithelial cells	Experimental	55.6 ± 38.1	NA	35.7 ± 35.6
	Control	67.2 ± 40.1	NA	56.7 ± 40.6
Percent lymphocytic cells	Experimental	44.1 ± 38.0	NA	64.9 ± 35.6
	Control	32.6 ± 40.2	NA	42.0 ± 41.5
Percent PMNs	Experimental	0.1 ± 0.5	NA	0.0 ± 0.3
	Control	0.0 ± 0.0	NA	1.2 ± 6.0
Salivary measures				
Salivary IgA (μg/mL)	Experimental	193.42 ± 112.17	191.94 ± 116.57	NA
	Control	194.68 ± 120.39	179.89 ± 116.88	NA
Pulmonary function				
Percent change FEV_1	Experimental	NA	0.02 ± 0.04	NA
	Control	NA	0.00 ± 3.31	NA
Percent change FVC	Experimental	NA	0.05 ± 0.03	NA
	Control	NA	–0.13 ± 3.78	NA
Percent change FEF 25–75%	Experimental	NA	1.02 ± 0.12	NA
	Control	NA	0.78 ± 7.63	NA

NA, not applicable.

Table 5. Means ± SDs for scores on POMS at four time points.

Group	Mood scale	Just before exposure	30 min into exposure	1 hr (end of exposure)	2 hr after end of exposure
Experimental	Anger–hostility	0.96 ± 1.86	1.42 ± 2.86	1.35 ± 3.27	0.94 ± 2.15
	Confusion–bewilderment	3.19 ± 1.83	3.79 ± 2.26	4.19 ± 2.47	3.60 ± 1.87
	Depression–dejection	0.83 ± 1.72	1.10 ± 2.43	1.02 ± 2.34	0.69 ± 1.69
	Fatigue–inertia	3.21 ± 3.96	4.79 ± 3.83	5.13 ± 4.29	4.15 ± 4.44
	Tension–anxiety	1.94 ± 2.93	1.73 ± 2.52	1.29 ± 2.20	0.79 ± 1.58
	Vigor–activity	8.27 ± 4.74	3.60 ± 3.75	3.29 ± 3.35	3.79 ± 3.87
	Total mood score	1.85 ± 11.71	9.23 ± 12.55	9.69 ± 12.56	6.38 ±10.80
Control	Anger–hostility	0.50 ± 1.46	1.00 ± 2.79	0.94 ± 2.68	1.17 ± 3.14
	Confusion–bewilderment	2.75 ± 1.45	3.48 ± 1.84	3.52 ± 1.82	3.46 ± 1.56
	Depression–dejection	0.65 ± 1.59	1.08 ± 2.36	0.58 ± 1.61	0.62 ± 1.79
	Fatigue–inertia	3.10 ± 4.55	4.38 ± 4.67	4.15 ± 4.83	4.08 ± 4.66
	Tension–anxiety	1.48 ± 2.10	1.23 ± 2.10	0.83 ± 2.12	0.92 ± 2.22
	Vigor–activity	7.98 ± 5.50	3.73 ± 4.03	3.54 ± 3.67	3.52 ± 3.92
	Total mood score	0.50 ± 11.44	7.44 ± 12.68	6.48 ± 11.43	6.73 ±11.65

tidal volume of 0.5 L (a single breath in normal quiet breathing) and 15 breaths/min, this translates to 450 L in 1 hr. Because there are 1,000 L in 1 m^3, the cumulative dose in this study is 0.332 ng. This dosage is far below the 15–20 µg dose at which airway responsiveness is altered in sensitive populations (Michel et al. 1989) and the 40 µg dose at which airway resistance is altered in healthy, nonatopic, nonasthmatic controls (Kline et al. 1999).

Ammonia. The mean concentration of ammonia in the experimental arm of this study was 817 ppb, a concentration that is below the published eye irritation threshold (irritation just barely noticeable) for ammonia of 4 ppm (World Health Organization 1986). It is also far below the short-term (15 min) exposure limit of for ammonia of 35 ppm set by the Occupational Safety and Health Administration (OSHA 2003). Average concentrations of ammonia in swine housing have been reported to range from 5 to 18 ppm; maximum concentrations in sow buildings are 43.7 ppm and in finishing barns are 59.8 ppm (Koerkamp et al. 1998), but these levels decrease rapidly downwind as they are diluted in ambient air.

H_2S and VOCs. H_2S is a colorless, flammable gas that smells like "rotten eggs" at low concentrations. The mean concentration of H_2S during the 1-hr exposure in this study was 24 ppb. This level is above the odor detection threshold (0.5 ppt to 8 ppb) but far below the irritant threshold, which ranges from 2.5 to 20 ppm (Amoore 1985; Collins and Lewis 2000). Thus, the H_2S level in this study was 3–4 orders of magnitude (i.e., 10^3 and 10^4 times) below the level that causes classical irritant symptoms. The scientific literature on H_2S, however, suggests that health symptoms can occur at H_2S concentrations far below the levels at which irritation or toxicity occur. For example, community investigations near paper mills, refineries, geothermal sources, and meatpacking plants indicate that sustained exposure to low levels of H_2S or other reduced sulfur compounds (below the irritant threshold) can cause health symptoms (Campagna et al. 2000; Jaakkola et al. 1990, 1991; Kilburn and Warshaw 1995; Legator et al. 2001). In two of these community studies, health symptoms were found from an average daily exposure to 10–11 ppb H_2S (Jaakkola et al. 1990; Kilburn and Warshaw 1995).

GC/MS was performed on air samples from both the experimental and control conditions in our study, and many diverse compounds were identified in both the control and experimental conditions. The vast majority of these compounds were present at concentrations far below published odor thresholds; furthermore, all of the compounds for which irritation thresholds were available in the literature were below these levels (Schiffman et al. 2001b). Yet human assessments indicated that odors as well as irritant sensations were perceived in the exposure condition of this study. Comparison of the findings from chemical and human assessments in this study with previous studies (Cometto-Muñiz et al. 1997; Schiffman et al. 2001b) points to the importance of the cumulative effects of hundreds of compounds in producing odor and irritant sensations.

Self-reported headaches, eye irritation, and nausea. The underlying cause of the significant increase in self-reported headaches, eye irritation, and nausea in the experimental condition of this study is not known. As described above, no single component in the airborne emissions was present at a high enough concentration to be wholly responsible for these symptoms. It is possible, however, that synergy among the combined components may induce these physical symptoms. That is, the symptoms may be caused by the combined load of all or some of the components in the air (H_2S, ammonia, VOCs, particulates, and endotoxin). Donham and Cumro (1999) have previously found that ammonia and particulates are synergistic with one another in their impact on human health. Furthermore, low concentrations (even subthreshold levels) of individual VOCs can add together when delivered in a mixture to produce noticeable sensory irritation (Cometto-Muñiz et al. 1997, 1999; Korpi et al. 1999). Another possibility is that these self-reported symptoms are innate or learned warning signals of potential health effects at higher concentrations or with more prolonged exposure. The symptoms may carry more significance for health effects in studies of vulnerable populations, such as children and elderly, and patients with cardiovascular or respiratory diseases.

Vital signs. The finding that no significant changes in respiratory rate, blood pressure, or pulmonary function were found here suggests that a single 1-hr exposure to unpleasant swine odor typical of downwind concentrations does not impair these health parameters in healthy volunteers tested in an environmental chamber. Previous studies have shown that exposure to unpleasant odors can in some cases lead to an inhibited breathing pattern (Schiffman et al. 2000). Stress, independent of unpleasant odors, also produces sustained inhibited breathing patterns that in turn can elevate blood pressure (Anderson 1998; Anderson and Chesney 2002). The mediating mechanism for elevated blood pressure from sustained inhibition of respiration is acidification of the plasma with subsequent increases in sodium/hydrogen exchange in kidneys and blood vessels. If inhibited breathing did occur during the 1-hr exposure in this study, it was not sustained after exposure, nor was the breathing frequency sufficiently altered to affect blood pressure. Future studies may employ additional measures of cardiovascular function such as alteration in heart rate variability, a finding that is associated with adverse effects in relationship to air pollution. More sensitive markers of airway inflammation, such as increased exhaled nitric oxide or increased epithelial permeability, may yield clues to long-term health effects of swine air exposure.

Mood (POMS scales). The finding that a 1-hr exposure to odorous swine air had no significant effects on mood scores on the POMS scale of healthy volunteers tested in an environmental chamber contrasts with a previous community study in which neighbors were frequently exposed to swine odor (Schiffman et al. 1995). In that study, neighbors of swine facilities in North Carolina experienced significantly more tension, depression, anger, fatigue, and confusion and less vigor on POMS scales when odors were present than when odors were absent (Schiffman et al. 1995).

Table 6. Means ± SDs for scores on the digit span test at four time points.

	Just before exposure	30 min into exposure	1 hr (end of exposure)	2 hr after end of exposure
Experimental	6.92 ± 1.30	6.90 ± 1.34	7.33 ± 1.40	7.46 ± 1.20
Control	6.92 ± 1.40	7.08 ± 1.25	7.46 ± 1.11	7.31 ± 1.36

Table 7. Number of persons self-reporting symptoms.

	Experimental			Control		
Symptom	Just before exposure	1 hr (end of exposure)	2 hr after end of exposure	Just before exposure	1 hr (end of exposure)	2 hr after end of exposure
Total headaches combined (migraine, sinus, other)	4	23	15	5	10	6
Eyes dry, irritated	2	11	7	2	3	2
Nausea	0	12	1	0	2	2
Throat sore, irritated	2	9	3	3	6	5
Throat itching inside	0	6	3	0	2	2
Tearing eyes	1	1	1	0	1	2
Sinus/nasal congestion	6	5	5	7	6	6
Nasal secretions	3	1	3	4	4	3
Nasal irritation, burning	0	1	2	0	3	1
Difficulty breathing	0	1	2	0	0	1
Cough	4	6	2	4	5	3

The difference in these findings can be explained by the differences in the exposure situations and the persons exposed. In the present study using a chamber, subjects were healthy volunteers who knew that the exposure would be time-limited and that the exposure levels were controlled by the investigators and approved for human subjects by the Duke University Medical Center Institutional Review Board. Furthermore, they were financially compensated and could withdraw at any time. Neighbors of swine operations, however, have no advanced warning about the timing, magnitude, or duration of the exposure. The intermittent presence of unavoidable, and unpredictable malodors can engender feelings of lack of control and negative affect when neighbors cannot use their home and property as they want. Unpleasant odors in the home can affect overall quality of life. Unconscious odor conditioning may also play a role in impaired mood of neighbors. When odors are associated with stressful or unpleasant situations, this odor can elicit subsequently alter mood, attitudes, and behavior (Kirk-Smith et al. 1983).

Salivary IgA. The finding of no changes in salivary IgA concentrations in this study is probably due to the short duration of the exposure period as well as the fact that the subjects were healthy volunteers who were financially compensated. Participants in this experimental trial as volunteers had more control over the odor exposure than do persons actually living downwind of a swine facility. Previous studies have shown that unavoidable stress and passive coping can produce decrements in salivary IgA within 10–15 min, whereas active coping and controllable stressors can increase salivary IgA (Bosch et al. 2001; Ring et al. 2002; Willemsen et al. 2002). Real-life stressful events and negative emotions can also decrease salivary IgA (Carins and Booth 2002; Yang et al. 2002). A recent study in North Carolina of neighbors of swine facilities found that their salivary IgA decreases significantly upon exposure to moderately strong swine odors (Avery et al., in press). This indicates that unavoidable and unpredictable odors from swine facilities that are not time-limited can have psychophysical impacts. The long-term health significance of alterations in salivary IgA levels is not well understood at present.

Odor ratings. The mean intensity ratings of 5.29 for odor (moderate strong to strong) and 3.77 for irritation (moderately weak to moderate) given by naive subjects in the experimental condition (for an odor 56 times above threshold) are higher than those given for the same level of swine odor by trained panelists who have extensive experience rating swine odor both on and off of farms in a natural environment. Trained panelists rate an odor 56 times above threshold at a mean odor intensity of 4.21 (moderate to moderately strong) with an irritation intensity of 3.01 (moderately weak) (Schiffman and Graham 2004). The mean unpleasantness ratings given by naive subjects during the experimental condition to an odor of 56 odor units was 6.21 (moderately unpleasant to very unpleasant). Trained panels give this odor a mean rating of 5.76 (moderately unpleasant). The probable reason why trained panelists give lower numbers is context specific. Trained panelists are exposed to very intense odors at odor sources next to the barns and lagoons as well as odors downwind. That is, scores of trained panelists are based on a wider range of intensities.

Conclusion

In this study that evaluated healthy volunteers, no statistical differences on objective physical measures, mood, or attention were found from a 1-hr exposure in an environmental chamber to air emissions from a swine house when compared with clean air. However, self-reported symptoms of headaches, eye irritation, and nausea were significantly higher in the swine air (experimental) condition than the clean air (control) condition. The underlying cause of self-reported headaches, nausea, and eye irritation in the experimental condition is not known but may be due to the combined load of some or all of the components in the air (H_2S, ammonia, VOCs, particulates, and endotoxin). Another possibility is that these self-reported symptoms are innate or learned warning signals of potential health effects at higher concentrations or with prolonged exposure.

The self-reported headaches, nausea, and eye irritation in this controlled study using healthy volunteer subjects without occupational exposure are a subset of a larger number of symptoms reported in community studies by individuals exposed to environmental odors (Shusterman 1992; Thu et al. 1997; Wing and Wolf 2000). The greater number of health symptoms reported by neighbors of swine operations compared with our healthy volunteers may be due to inclusion of vulnerable populations (e.g., persons with asthma), previous exposure history, higher levels of exposure in certain communities (both swine and nonswine sources), involuntary and prolonged exposure, and quality of life issues. In addition, persons living downwind are exposed to emissions from lagoons and spray fields as well as swine houses, although the former two sources tend to contain similar but less varied compounds than those emitted from the houses (Schiffman et al. 2000).

More research is necessary to determine the mechanism responsible for self-reported symptoms and their elevated number in neighbor exposures relative to this experimental exposure. First, controlled studies in the environmental chamber should be expanded in the future to include volunteers from vulnerable populations (e.g., persons with asthma). Most scientific literature (Nieto et al. 2003; Nolte and Berger 1983; Sant'Ambrogio 1987; Shusterman 2002), but not all (Levi et al. 1990; Opiekun et al. 2003), suggests that persons with asthma have sensory hyperresponsiveness to irritants. These conflicting findings may be due to medical status at the time of testing; activation of afferent neurons in the airways is not a static property but rather appears to change rapidly in response to inflammation (Carr and Undem 2001). Asthmatic subjects with active symptoms may not volunteer for an exposure experiment.

Appendix 1. The 30 feelings that were rated on the POMS.

Tense	Unworthy	Gloomy
Angry	Uneasy	Sluggish
Worn out	Fatigued	Weary
Lively	Annoyed	Bewildered
Confused	Discouraged	Furious
Shaky	Nervous	Efficient
Sad	Lonely	Full of pep
Active	Muddled	Bad tempered
Grouchy	Exhausted	Forgetful
Energetic	Anxious	Vigorous

Appendix 2. The 48 symptoms on the EEHQ.

Hives, itching skin	Feeling angry, irritable	Feeling anxious, panicky
Skin rash	Feeling depressed	Wheeze, chest tightness
Skin redness, flushing	Eyes dry, irritated	Shortness of breath
Feeling feverish, chills	Tearing eyes	Chest pain
Migraine headache	Blurred vision	Heart racing, pounding
Sinus headache	Sinus/nasal congestion	Difficulty breathing
Other headache	Nasal secretions	Cough
"Spacy" feeling	Nasal irritation, burning	Cough up sputum, phlegm
Brain fog	Difficulty concentrating	Hoarseness
Cold hands or feet	Memory problems	Nausea
Throat sore, irritated	Inappropriate emotions	Vomiting
Throat itching inside	Ear redness, flushing	Diarrhea
Coordination problems	Ears itching inside	Abdominal bloating, pain
Muscle weakness	Daytime sleepiness	Constipation
Muscle aches, joint pain	Undue fatigue	Heartburn
Numbness of legs, arms	Trembling, body shaking	Pelvic pain

Second, the contribution of stress must be incorporated in controlled experimental paradigms because stress responses can sensitize various neuronal, hormonal, and behavioral responses that could potentially affect the parameters tested in the present controlled exposure study (Johnson et al. 2004). Neighbors who are involuntarily exposed to unpredictable swine emissions report significantly more tension, depression, anger, fatigue, and confusion and less vigor on POMS scales (Schiffman et al. 1995) than did the subjects in the present experiment, whose exposure was voluntary. Although it is not possible to design a study that precisely replicates the involuntary and unpredictable exposure to malodorous swine emissions (potential stressor) in a natural setting, symptoms can be studied during a prolonged intermittent (and thus unpredictable) but time-limited exposure under controlled experimental paradigms. In addition, symptoms during exposure to swine air while performing a stressful activity (e.g., mental arithmetic) versus symptoms while performing a nonstressful activity (control) can be compared.

Controlled exposure studies as well as further epidemiologic studies should include subjects with a broad range of exposure history to swine emissions to determine the prevalence of sensitization as well as tolerance for (or adaptation to) odorous emissions. Several experimental studies suggest that increased sensitivity to an odor can develop with repeated exposure (Wysocki et al. 1989), and that the effect is pronounced in women (Dalton et al. 2002). Yet tolerance to swine confinement air (with fewer symptoms) has been reported to occur in some chronically exposed workers (Von Essen and Romberger 2003), although it is not known whether tolerance to aerial emissions develops in an analogous manner at lower concentrations that occur at neighbors downwind of swine facilities. Both controlled and epidemiologic research studies will help clarify the impact of sporadic exposure to swine emissions on health symptoms of persons who are involuntarily exposed intermittently to malodors.

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Odor from Industrial Hog Farming Operations and Mucosal Immune Function in Neighbors

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Odor from Industrial Hog Farming Operations and Mucosal Immune Function in Neighbors

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ABSTRACT. The authors evaluated whether exposure to malodor from industrial hog farming operations has a psychophysiologically mediated immunosuppressive effect on secretory immunoglobulin A (sIgA) in neighbors. Fifteen adults living within 2.4 km (1.5 mi) of at least one hog farming operation rated odor intensity on a 9-point scale and provided saliva samples twice daily for two weeks. The authors used hierarchical regression to model the association between reported odor and sIgA; study participants were their own controls. The natural log of sIgA concentration and secretion rate declined, on average, 0.058 (0.032) and 0.116 (0.103), respectively, for each incremental 1-unit increase in reported odor from 4 to 9, adjusted for time of day, suggesting reduced levels of sIgA in response to moderate or high odor. Findings support the hypothesized immunosuppressive effect of malodor on mucosal immunity and provide preliminary data useful in understanding health effects related to malodor from industrial hog farming operations.
<Key words: CAFOs, community-based participatory research, hog production, secretory IgA, swine odor>

IN RECENT DECADES, pork production has shifted from small farms to industrialized operations with intensive confinement of animals and concentration of wastes. Although many occupational and human challenge studies have shown that dusts, gases, and pathogens inside swine confinement houses can affect the health and respiratory function of workers and naive volunteers,[1–16] we know less about potential health effects in neighbors. Industrial hog operations emit malodorous mixtures of gases and particulates from confinement houses, waste lagoons, and spray fields. Organic particulates that carry endotoxin from dander, feed, and dried feces are vented from the confinement houses to the ambient environment by large fans. Feces and urine held in waste lagoons decompose anaerobically, releasing ammonia, hydrogen sulfide, and volatile organic compounds into the air.[17] Broadcast of feces and urine on nearby fields by industrial irrigation systems produces aerosols that drift downwind.

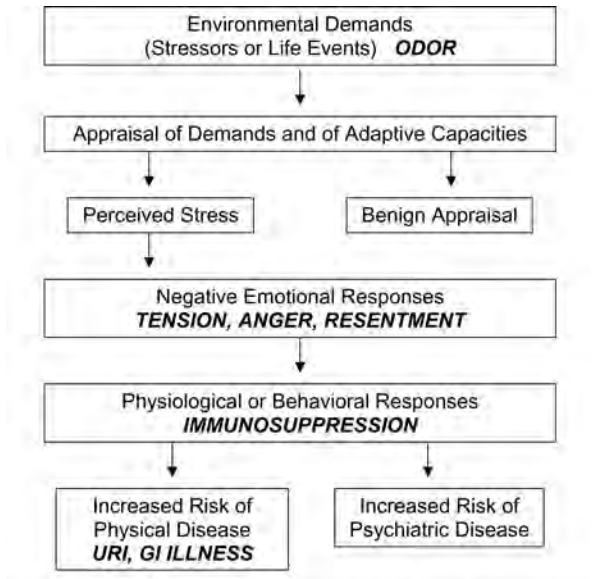
Two surveys of physical health symptoms conducted in North Carolina and Iowa have suggested that neighbors of industrial hog operations report headache, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes more frequently than do demographically comparable persons who do not live near such operations.[18,19] Researchers have also postulated effects of exposure on mood. A study in North Carolina suggested that neighbors experienced significantly more tension, depression, anger, fatigue, and confusion, and less vigor, when odors were present than in the absence of odor.[20]

Health effects of exposure to airborne emissions may occur through toxicologic and nontoxicologic mechanisms. Toxicologic mechanisms include physiological responses to irritants and pathogens present in airborne plumes, whereas nontoxicologic mechanisms involve psychophysiological responses to odor. Nontoxicologic mechanisms include "innate odor aversions; innate pheromonal phenomena; odor-related exacerbation of

underlying conditions; odor-related aversive conditioning; [and] odor-related, stress-induced illness."[21] Concentrations of individual compounds in the gaseous mixture produced by confinement houses and lagoons tend to be lower than those at which irritant effects are expected to occur.[22] Occurrence of symptoms at low levels would suggest a nontoxicologic mechanism[21] (Figure 1[23]).

In beginning to explore potential mechanisms through which odor may affect the health of neighbors, we evaluated the hypothesis that exposure to noxious odor from industrial hog operations has a psychophysiologically mediated effect on the secretory immune system; specifically, that odor as a stressor has an immunosuppressive effect on secretory immunoglobulin A (sIgA). In a recent review of the literature on stress and secretory immunity, Bosch et al.[24] summarized the biological rationale for a potential effect of stress on salivary secretory immune function:

> Salivary glands, as with other mucosal glands, are largely under autonomic nervous system control. The preganglionic autonomic centers in the brain stem that regulate salivary gland activity receive direct inhibitory and excitatory inputs from neural structures in the forebrain that are part of recognized "stress circuits" and centers for homeostatic regulation. The salivary glands form a highly sophisticated endpoint in the CNS control of local immune defenses, capable of responding instantly and with a high level of specificity to potential source of harm (e.g., stress, inflammation). This remarkable ability, together with their strategic location at the portal of entry to the respiratory and gastrointestinal tract, make these glands ideally suited to provide the host with a first line of defense.[24]



[a] Adapted from Cohen, S., R. Kessler, and L. Gordon. *Strategies for measuring stress in studies of psychiatric and physical disorders*, in *Measuring Stress: A Guide for Health and Social Scientists*, S. Cohen, R. Kessler, and L. Gordon, Editors. 1997. Oxford University Press: New York.

Fig 1. Conceptual framework for physiologic response to odor.

Repeated exposure to intense malodor from hog operations in people who are unwillingly exposed in their homes can be considered stressful via impacts on quality of life, negative feelings, and triggering of physical symptoms, such as coughing, mucous membrane irritation, nausea, and vomiting.[18–20,25,26] Neighbors of industrial hog operations, who live in rural and semirural communities, report that they cannot enjoy outdoor activities when odors are present, keep windows closed even in nice weather, worry about planning outdoor recreational activities that could be ruined by odor, and worry about potential for declining property values. The unpredictability of odor episodes implies a lack of control over the use of home and property. Animal and human studies suggest that the psychophysiologic impacts of stress can be greater when stressors are unpredictable and uncontrollable.[27–29] Other researchers have associated exposure to malodor with negative affect, motivation to escape, annoyance, and increased feelings of helplessness and depression.[21,30,31] Furthermore, intensive sensory stimuli, such as strong light and noise, are known to be stressors.[32]

In previous studies, researchers have found that other types of stressors and negative mood can suppress salivary sIgA. Daily hassles and undesirable life events,[33,34] negative mood,[35–37] dental work,[38] prolonged academic stress,[39–41] cold stress,[42] viewing a stressful video,[29] and perceived work-related stress[43] are all associated with reduced concentrations of sIgA in saliva. Generally, acute stress is associated with increases in sIgA, whereas chronic stress is associated with decreased levels of sIgA. However, there appears to be further differentiation in the effect of acute stress on sIgA by type of stressor. Active coping stressors are consistently associated with increased sIgA secretion, but passive coping stressors tend to be associated with decreased secretion.[24,29] Hog odor is a unique stressor in the literature on stress and secretory immunity; it is a chronic stressor that persons experience for repeated acute episodes. That it is a chronic, repeatedly experienced passive coping stressor suggests the hypothesized immunosuppressive effect on sIgA that we examined here.

This research reflects concerns over health and well-being widely expressed by people who live near industrial hog operations. The study builds on a program of community-driven research into the impacts of industrial hog production in North Carolina.[17,18,44–46] Because of the political contentiousness of industrial hog

production in North Carolina and a widespread distrust of government, industry, and universities in the low-income and African American communities most affected by industrial swine production,[47] collaboration with community organizations was a key aspect of the recruitment and retention of study participants, and the collection of reliable data for this study.

Method

To study the effect of exposure to hog odor on sIgA secretion, we used a longitudinal repeated-measures design in which study participants served as their own controls. Each morning and evening for 14 days, persons living within 2.4 km (1.5 mi) of an intensive hog operation rated the intensity of odor and provided 2-min, unstimulated, whole saliva samples for sIgA analysis. Concentration of sIgA (micrograms per milliliter) in saliva was determined by enzyme-linked immunosorbent assay (ELISA), and we calculated the sIgA secretion rate by multiplying the concentration by the salivary flow rate. We then evaluated the relationship between reported odor and sIgA concentration and secretion rate in stratified analyses and hierarchical modeling.

Study participants and data collection. In conjunction with local community organizations, we identified exposed communities and recruited study participants in five geographic clusters. Participants were nonsmoking adults who lived within 2.4 km (1.5 mi) of an intensive hog operation and had at least one neighbor within 0.4 km (0.25 mi) of their home who was also willing to participate. One person from each household participated. Clusters included two to four households. Three clusters were near a single hog operation, one was near two hog operations, and one was near four operations. The permitted number of animals in each operation ranged from 1,000 to 12,000.

Participants in each cluster agreed on two times, approximately 12 hr apart (for example, 7:00 AM and 7:00 PM), at which they would collect data for 14 days. Prior to data collection, participants gave informed consent and provided basic demographic information and additional information on residence, employment, and medical history. At the specified times in the morning and evening, study participants were asked to spend 5 min outside. They then recorded the intensity of hog odor at that time on a scale from 1–9 on which 1 represented no odor and 9 represented very strong odor. Participants also completed other required activities, which included collection of 2-min saliva samples in 50-ml centrifuge tubes numbered to correspond to the individual and time of collection. To prevent the degradation of protein by proteolytic enzymes, we filled the tubes with 200 ml of a salivary enzyme inhibitor cocktail containing 10 mM ethylenediamine tetraacetic acid, 30 mM ε-aminocaproic acid, and 6 mM benzamidine hydrochloride. Participants were instructed to void their mouths of saliva by swallowing before beginning collection, and to spit into the collection tube after 1 min and again after 2 min. Saliva samples were stored in the participant's freezer and collected at the end of the study. On the 15th day, participants provided information on illness during the data collection period and problems performing the required tasks. The Institutional Review Board of the School of Public Health of the University of North Carolina at Chapel Hill reviewed and approved all activities performed by study participants.

Laboratory assay. Each participant provided a maximum of 28 samples: 2 samples (morning and evening) per day for a total of 14 days. We analyzed the samples without knowledge of reported odor levels. Samples were thawed at 37°C and incubated at 56°C for 1 hr to inactivate proteolytic enzymes. Tubes were individually weighed before and after collection of the saliva sample to determine the mass of the saliva. Salivary flow rates in milliliters per minute were determined by dividing the volume of saliva (equal to the mass of saliva, assuming a density of 1 g/ml) by the 2-min collection time.[48] Samples were centrifuged at 11,000 rpm for 5 min to remove food and other debris, and the supernatant was then analyzed for sIgA content by ELISA. sIgA concentrations were calculated in micrograms per milliliter; sIgA secretion rates in micrograms per minute were calculated by multiplying the sIgA concentration by the salivary flow rate to control for variation in rates of salivary flow.[48] Total sIgA concentrations above the upper limit of detection of the assay (29 samples of 410) were assigned the value of the upper limit of detection, either 800 or 1600 μg/ml, depending on assay performance.

Statistical analysis. We assumed missing data to be noninformative with respect to an association between reported odor and sIgA, and these data were excluded from the analysis. Because each person was both unexposed and exposed to odor at different points in time, factors associated temporally with both odor and sIgA were potential confounders. Time-independent factors, such as age or gender, are unlikely confounders because their association with odor and sIgA, would have to vary over the 2-wk period of data collection. We considered two time-dependent potential confounders in this analysis: day of data collection (1–14), coded as a continuous variable; and time of day (AM versus PM), coded as a binary variable.

Given the clustered design of repeated measures on people over time, we modeled data using hierarchical mixed models. Two levels were modeled: day (within person) and person (within cluster). The outcome variables were log transformed because sIgA was lognormally distributed. We considered fixed effects for odor, time of day, and day, and random effects for cluster, person within cluster, odor, and time of day. We dropped day of data collection (coded 1–14) from the

model in preliminary analyses because there was very little variation in ln(sIgA) concentration by day, and, therefore, it was unlikely to be a confounder of the effect of reported odor on ln(sIgA). We also excluded a random effect for odor because the estimated variances in the effect of odor on ln(sIgA) between cluster and between person within cluster were negligibly small. Although graphs of each participant's sIgA values over the 14 days suggested individual differences in diurnal sIgA variability, we considered time of day to be a fixed rather than a random effect due to the small study size.

The final model included fixed effects for odor and time of day and random effects for cluster and person within cluster. A first-level, within-person regression model was specified as follows:

$$\ln Y_{(ijk)} = \beta_{0(jk)} + \beta_{1(jk)}(\text{odor}) + \beta_{2(jk)}(\text{time of day}) + r_{(ijk)}; \quad r_{(ijk)} \sim N(0,\sigma^2)$$

where Y_{ijk} is the sIgA measurement on person j in cluster k at timepoint i;

k^{th} cluster: $k = 1, 2, 3, 4, 5;$
j^{th} person: $j = 1, 2, 3, \ldots 15;$
i^{th} timepoint: $i = 1, 2, 3, \ldots 28;$

$\beta_{0(jk)}$ is the estimated average individual ln(sIgA) level at reference levels of reported odor and time of day; $\beta_{1(jk)}$ is the estimated change in ln(sIgA) for each unit increase in odor above a rating of three (or the estimated difference between medium/high odor compared to no/low odor); $\beta_{2(jk)}$ is the mean difference in sIgA levels between AM and PM time points, and $r_{(ijk)}$ is the residual within-person variation in ln(sIgA).

A second-level, between-person, and between-cluster regression model was specified as follows:

$$\beta_{0(jk)} = \gamma_{00} + \gamma_{01}\text{cluster}_k + \gamma_{02}\text{person}_j(\text{cluster}_k) + \mu_{0(jk)}; \quad \mu_{0(jk)} \sim N(0,\tau_{00})$$

where $\beta_{0(jk)}$ is the person-specific intercept, γ_{00} is the mean of person-specific intercepts ($\beta_{0(jk)}$) for ln(sIgA), γ_{01} is the proportion of the variation in person-specific intercepts explained by cluster, γ_{02} is the proportion of variation in person-specific intercepts explained by person, and $\mu_{0(jk)}$ is the residual between-person variation in ln(sIgA).

Results

Mean participant age was 55.3 yr, with a standard deviation of 13.4 yr. The median age was 53 yr, and the range was from 33 to 77 yr. One-third of the participants were men and two-thirds were women. Two participants were African American and 13 were white. Seven participants were employed outside the home during the data collection period and 7 grew up around some type of livestock.

Of 420 potential observations (2 measurements/day × 14 days × 15 participants), 12 (2.9%) odor reports were missing, and 10 (2.4%) saliva samples were missing, resulting in 407 data points available for analysis. For the majority of time points no odor was present, although some clusters experienced more odor episodes than others. Specifically, there was no odor reported at 60% of morning time points and 50% of evening time points. Figure 2 shows the distribution of odor ratings for all participants across all time points. The mean odor rating for all participants across both morning and evening time points was 2.2, with a standard deviation of 1.8. Reported odors tended to be higher in the evenings, higher for the younger age group, and higher for men than women (Table 1). All study participants reported no (odor rating = 1) or low (2–3) odor, 14 reported moderate odor (4–6), and 9 reported high odor (7–9) at least once during the data collection period.

Figures 3 and 4 show the distribution of sIgA concentrations and secretion rates for all participants across all time points. The average concentration of sIgA for all participants across all time points was 374.5 μg/ml, with a standard deviation of 365.1. The average concentration was higher in the morning than in the evening, similar by age, and higher in men than women. The average secretion rate was 138.9 μg/min, with a standard deviation of

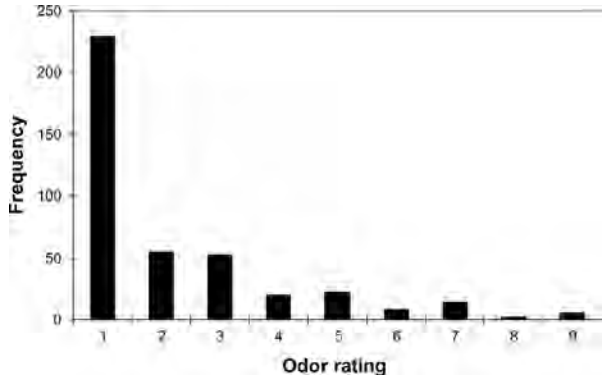


Fig. 2. Distribution of odor ratings across time points.

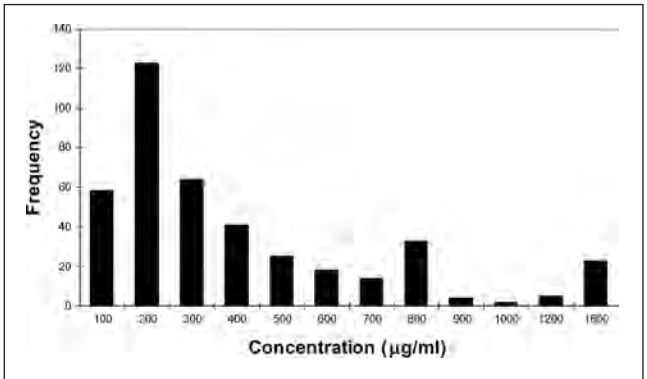


Fig. 3. Distribution of sIgA concentration across time points.

123.1, higher in the morning than evening, slightly lower in the older age group, and similar in men and women (Table 1). For morning and evening times combined, sIgA concentrations and secretion rates were lower at higher levels of reported odor (Table 2, top panel). Concentrations declined from approximately 400 μg/ml at times of no or low odor to 274.7 μg/ml at moderate odor and 212.1 μg/ml at high odor. Secretion rates were 154.5, 140.2, 86.9, and 87.7 μg/min at no, low, moderate, and high odor levels, respectively. The pattern of lower sIgA levels at moderate and high odor levels holds for both morning and evening times considered separately (Table 2, middle and lower panels).

Table 3 shows results from hierarchical models including fixed effects for reported odor and time of day and random effects for cluster and person within cluster. We included odor as a continuous variable in Model 1. Because mean sIgA levels were similar during no-odor and low-odor conditions (Table 2), odor was coded 1 at odor ratings below 4, and 2–7 at odor levels 4–9. Log sIgA concentration declined, on average, 0.058 (*SD* = 0.032), and log secretion rate declined 0.054 (*SD* = 0.034) for each unit increase in odor above a rating of three on the original scale. This represents an approximate 28 to 29% decline in estimated sIgA concentration and secretion rate at the highest odor level compared with no or low reported odor. Because sIgA levels—in particular, secretion rates—were similar at moderate and high odor, we also evaluated the sIgA response to exposure using an indicator variable to compare moderate (4–6) and high (7–9) odor to low or no odor (1–3) (Model 2). Model 2 estimates suggest a 10% to 11% decline in sIgA concentration and secretion rate at moderate and high odor, compared with low or no reported odor. The *t*-values, indicating the goodness of fit of the models, were modest in size, but suggest a slightly better fit for Model 1 than Model 2.

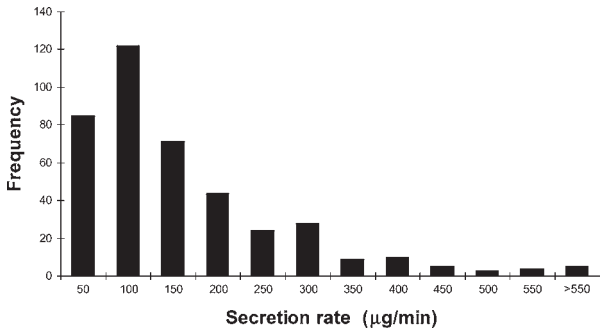


Fig. 4. Distribution of sIgA secretion rate across time points.

Discussion

The study findings provide evidence for an effect of exposure to malodors from industrial hog operations on the functioning of the mucosal immune system. Salivary IgA concentrations and secretion rates were lower following exposure to moderate or high, compared with no or low, reported odor. Although sIgA concentrations and secretion rates were lower, and odor ratings were higher, on average, during evening than morning hours, we observed the association between odor level and sIgA for both morning and evening time periods, and the association persisted after controlling for time of day. Using hierarchical regression to take into account the clustered repeated-measures design, and to adjust for time of day, we estimated that log sIgA concentration decreased, on average, 0.058 (*SD* = 0.032) and log sIgA secretion rate decreased, on average, 0.054 (*SD* = 0.034) for every unit increase in odor above a rating of 3 on a 9-point scale. Although the number of study participants was limited, the longitudinal design in which participants serve as their own controls lends strength to

Table 1.—Mean Odor Ratings, Secretory Immunoglobulin A (sIgA) Concentrations, and sIgA Secretion Rates according to Time of Day, Age, and Gender

Variable	*n* people	*n* reports	Odor (*SD*)	sIgA concentration* (*SD*)	sIgA secretion rate† (*SD*)
Time of day					
Morning	15	204	1.9 (1.3)	453.1 (427.6)	160.0 (138.8)
Evening	15	203	2.5 (2.1)	293.7 (268.6)	117.8 (101.1)
Age					
⩽ 53 years	8	213	2.4 (2.1)	372.4 (323.8)	148.0 (135.1)
> 53 years	7	194	2.0 (1.5)	375.0 (407.4)	129.0 (108.0)
Gender					
Male	5	139	2.8 (1.9)	466.1 (420.9)	143.2 (110.5)
Female	10	268	1.9 (1.7)	325.6 (323.9)	136.7 (129.3)
Total	15	407	2.2 (1.8)	373.6 (365.6)	138.9 (123.1)

*In micrograms per milliliter.
†In micrograms per minute.

Table 2.—Mean sIgA concentration and secretion rate according to time of day and odor level

Time of day and odor level	*n*	sIgA concentration*	*SE*	sIgA secretion rate†	*SE*
Both times of day					
None (1)	228	395.2	25.4	154.5	8.7
Low (2–3)	108	405.2	38.1	140.2	11.9
Moderate (4–6)	50	274.7	32.2	86.9	10.4
High (7–9)	21	212.1	22.7	87.7	15.5
Morning					
None (1)	124	480.5	39.4	180.8	14.0
Low (2–3)	58	460.3	60.5	143.3	13.8
Moderate (4–6)	18	278.4	41.6	84.4	14.4
High (7–9)	4	285.9	45.1	95.5	42.9
Evening					
None (1)	104	293.5	27.0	123.1	8.2
Low (2–3)	50	341.3	41.9	136.6	20.2
Moderate (4–6)	32	272.6	45.1	88.3	14.3
High (7–9)	17	194.8	24.6	85.8	17.0

*In micrograms per milliliter.
†In micrograms per minute.

Table 3.—Estimates of Association between Odor and Secretory Immunoglobulin A (sIgA) from Hierarchical Regression

Dependent variable	β	*SE*	*t*
Model 1*			
log sIgA concentration (μg/ml)	−0.058	0.032	−1.81
log sIgA secretion rate (μg/min)	−0.054	0.034	−1.62
Model 2†			
log sIgA concentration (μg/ml)	−0.116	0.103	−1.13
log sIgA secretion rate (μg/min)	−0.131	0.107	−1.22

*Odor coded as a seven-level continuous variable (nine-level variable recoded: 1–3, 4, 5, 6, 7, 8, 9).
†Odor coded as indicator variable (1 = odor rating 4–9; 0 = odor rating 1–3).

the results because we did not have to control for a range of between-person confounding factors.

Errors in measurements could have led to bias in measures of association, or decreased their precision. First, random imprecision in the assay that is unrelated to exposure would be expected to decrease the precision of the estimates of odor–sIgA association but not their magnitude. We performed assays blinded to exposure classification to reduce any potential association between measurement error and odor rating. Second, the ELISA assays of sIgA produced readings above the detection limit for 29 (7.1%) of the samples. We assigned these samples the upper limit of detection of the assay, either 800 or 1600 μg/ml. Of the 29 samples, 28 were collected at times at which the participant reported no or low odor. Because the actual concentrations of the samples assigned the upper limit of detection were probably higher than their assigned values, the true difference in sIgA levels between no or low reported odor and moderate or high odor is likely greater than we observed here. Third, because we calculated secretion rates based on sIgA concentration, salivary mass, and the assumption that each participant collected saliva for exactly 2 min, errors in measurement for secretion rate are a product of errors in each of these values. Therefore, it is probable that the error in the sIgA secretion rate data is greater than the error in the sIgA concentration data. Nonetheless, the error associated with the assay is probably the predominant source of error for the secretion rate.

A fourth potential source of measurement error is the rating of odor. Given the hypothesized psychophysiologic mechanism whereby odor as a repeated stressor affects levels of sIgA, participant rating of perceived odor intensity is preferable to measures of chemical and physical constituents of odor plumes. Rating of perceived odor intensity, however, does not directly assess whether the participant actually perceives the odor episode as stressful, although future work should attempt to do so. Furthermore, we were not able to

evaluate the repeatability of participants' ratings of known odorants in this study. An additional source of error in odor classification occurs as a result of variation in exposure to odor in the period prior to sIgA collection. Odor reports reflect an assessment of odor present a short time period before saliva collection, whereas sIgA levels in saliva may also reflect exposure to odor prior to the odor report. Future work should more explicitly evaluate time course.

Overall, these data suggest that sIgA, which serves as a first line of defense against invading organisms at mucosal surfaces,[49] is suppressed in neighbors of hog operations after exposure to moderate to intense odors from these facilities. Malodor is a common environmental exposure that occurs in many industrial settings, including sewage treatment plants, landfills, incinerators, paper and petrochemical plants, and livestock production. Malodor is often considered to be merely a nuisance, an exposure that is unpleasant but has no physical or physiological effects. Our study suggests that malodor from industrial swine operations can affect the secretory immune system, although the reduced levels reported here are still within normal range.[48] Additional research is needed to clarify psychophysiologic mechanisms, the time course over which responses to odor occur, individual differences in responses, clinical implications of reduced sIgA levels, and the generalizability of the response to other stressful odorants.

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Attachment A20

Race, Poverty, and Potential Exposure of Middle-School Students to Air Emissions from Confined Swine Feeding Operations

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Previous studies suggest that airborne effluent from swine confined animal feeding operations (CAFOs) may affect the health and quality of life of adults and the prevalence of asthma symptoms among children. To investigate the extent to which public school students may be exposed to airborne effluent from swine CAFOs and to evaluate the association between schools' demographic characteristics and swine CAFO exposures, we assessed the proximity of 226 schools to the nearest swine CAFO and conducted a survey of school employees to identify schools with noticeable livestock odor. We used publicly available information describing the enrollment of each school to assess the association between race and socioeconomic status (SES) and swine CAFO exposure. Odor from livestock was noticeable outside (n = 47, 21%) and inside (n = 19, 8%) school buildings. Schools with < 63% enrollment of white students and ≥ 47% of students receiving subsidized lunches at school were located closer to swine CAFOs (mean = 4.9 miles) than were the remaining schools (mean = 10.8 miles) and were more likely to be located within 3 miles of an operation than were schools with high-white/high-SES enrollment (prevalence ratio = 2.63; 95% confidence interval, 1.59–4.33). The prevalence of reported livestock odor varied with SES (low SES, 25%; high SES, 17%). These analyses indicate that the potential for in-school exposure to pollution arising from swine CAFOs in North Carolina and the environmental health risks associated with such exposures vary according to the racial and economic characteristics of enrolled students. *Key words:* **adolescent health, children's health, confined swine feeding, environmental epidemiology, environmental justice, industrial hog operations, school health.** *Environ Health Perspect* **114:591–596 (2006). doi:10.1289/ehp.8586 available via** *http://dx.doi.org/* **[Online 10 November 2005]**

Confined animal feeding operations (CAFOs) house large numbers of animals, flush animal wastes into open-air waste pits, and apply partially decomposed wastes to land, releasing pollutants into soil, air, and water (National Research Council 2003). Odor and local air pollution—including ammonia (Reynolds et al. 1997; Subramanian et al. 1996; U.S. Environmental Protection Agency 2002), hydrogen sulfide (Reynolds et al. 1997), methane (Sharpe and Harper 1999), residues of veterinary antibiotics (Hamscher et al. 2003), total bacteria (Radon et al. 2001), fungi (Radon et al. 2001), and endotoxin (Reynolds et al. 1997)—arise from CAFO buildings and waste pits and are of particular concern to CAFO neighbors because of their documented impacts on the health and quality of life of livestock farm workers and neighbors (Cole et al. 2000; Merchant et al. 2005; Schiffman 1998; Schiffman et al. 1995; Thu et al. 1997; Vogelzang et al. 1999, 2000; Wing and Wolf 2000). A study of the mental and physical health of swine CAFO neighbors in Iowa found elevated rates of respiratory symptoms among CAFO neighbors compared with residents not living near livestock production (Thu et al. 1997). One study of swine CAFO neighbors in North Carolina reported negative impacts of odor on tension, depression, and anger among individuals living near operations (Schiffman et al. 1995), and another in North Carolina reported "increased occurrences of headaches, runny nose, sore throat, excessive coughing, diarrhea and burning eyes" and decreased quality of life among residents living near swine CAFOs (Wing and Wolf 2000). In an investigation of possible stress-mediated impacts on immune function, swine CAFO neighbors had lower average concentration and secretion of salivary immunoglobulin A during periods of moderate to high odor (Avery et al. 2004). Among children, increased prevalence of asthma symptoms has been associated with proximity to swine CAFOs (Chrischilles et al. 2004; Merchant et al. 2005).

In the United States, race and socioeconomic status (SES) are closely intertwined and have been widely associated with health, including chronic disease morbidity and mortality (Borrell et al. 2004; Roux et al. 2001; Winkleby et al. 1998), infectious diseases (Centers for Disease Control and Prevention 2005), immunization (Egede and Zheng 2003), health care services (Gaskin and Hoffman 2000; Monheit and Vistnes 2000; Weinick et al. 2000), and environmental exposures (Evans and Kantrowitz 2002; Guidry and Margolis 2005; Northridge et al. 2003). Swine CAFOs are disproportionately located in communities of color and regions of poverty (Edwards and Ladd 2000; Wilson et al. 2002; Wing et al. 1996, 2000) and are thus located among populations that may be more susceptible to the airborne exposures and more likely to experience detrimental health consequences of such exposures (Bradley and Corwyn 2002; Williams and Jackson 2005). The literature published to date about health impacts of CAFO-related exposures focuses on health impacts of exposures among adults; however, knowledge about the growth and development of the human respiratory tract suggests that the children in these exposed communities may be at increased risk of respiratory health effects because of their size, behavior, and developmental stage (Dietert et al. 2000; Kim et al. 2004; Peden 2000).

In light of recent research about health effects of CAFO-related exposures, children's susceptibility to environmental pollutants, and concern about the conditions of school buildings, we sought to assess the extent to which adolescents attending public schools may be exposed to swine CAFO emissions. Based on a large sample of public schools in North Carolina, we estimated potential exposure using both record-based and survey-based exposure indices and examined racial and economic differences in potential exposure.

Materials and Methods

During the 1999–2000 school year, seventh- and eighth-grade students from 499 public schools in North Carolina participated in a statewide school-based survey designed to assess the prevalence of asthma-related symptoms among adolescents (North Carolina Department of Health and Human Services 2001; Sotir et al. 2003). During the 2003–2004 school year, we conducted a follow-up survey of employees in the participating schools to collect

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information about environmental health conditions inside the schools and for an evaluation of the relationship between sources of environmental pollution located near schools and students' self-reported respiratory health symptoms. From the 499 participating schools and based on the aims of our study, we excluded 160 schools from further data collection because of school locale and level of participation in the asthma survey. Specifically, we excluded schools located in counties with no swine CAFOs and none in neighboring counties (n = 45), schools with < 25 students surveyed (n = 34), schools located within 5 miles of a state border (n = 17), schools physically located within a city with population > 100,000 (n = 61), and schools that had closed or relocated to a new building since the 1999–2000 school year (n = 10). The remaining 339 schools composed our final target population of public schools.

We used publicly available records about the geographic positions of schools (North Carolina Center for Geographic Center and Analysis 2002) and swine CAFOs (Wing et al. 2000, 2002) to generate location-based estimates of in-school exposure for each school. We calculated distance to the nearest operation using the formula given by Goldberg et al. (1999) and categorized proximity as within or beyond 3 miles of the nearest operation. A 3-mile radius was selected as a suitable zone of potential exposure because elevated prevalence of asthma has been reported among children attending schools within 3 miles of a swine CAFO (Mirabelli et al., in press). Furthermore, although previous studies about the impacts of swine CAFOs on health and quality of life use a 2-mile radius (Thu et al. 1997; Wing and Wolf 2000), odors are sometimes reported at distances > 2 miles, and a radius of 3 miles yields a more balanced distribution of schools in our data. Swine CAFOs typically store animal waste in open waste pits, whereas other types of livestock operations in North Carolina tend not to employ such practices. Details about the locations of operations not using this liquid waste management system are not publicly available and could not be included in these analyses.

For a second metric of in-school exposure, we conducted a four-page, 21-item pencil-and-paper–style survey about environmental health conditions inside and surrounding the school buildings. In October 2003, we mailed the surveys to school principals and asked each to distribute four surveys to potential respondents in the following jobs: administrator, teacher, maintenance or custodial staff, and school nurse or health care provider. During a 9-month survey collection period, respondents from 267 (79%) of the surveyed schools returned 801 of the 1,632 surveys, whereas the remaining schools either actively (n = 1) or passively (n = 71) declined to participate. After receiving completed surveys, we excluded two additional schools based on updated information about the location of one school and because respondents from another school indicated that the school had closed and reopened in a new building since the 1999–2000 school year.

Respondents were asked whether odors from livestock farms were noticeable outside or inside the school buildings never, once per month or less, two to three times per month, about once per week, or more than once per week, and were asked to rate the odor, at its worst, on a five-point scale: 1, very faint; 2, faint; 3, moderate; 4, strong; 5, very strong. We assigned an odor rating of zero for respondents who indicated that they never noticed livestock farm odor at the school, and we created final school-level exposure indicator variables based on whether any survey respondent reported ever noticing livestock farm odor outside or inside the school building. For schools with livestock odors reported by any respondent, the odor ratings assigned to that school and used in the analyses are averages of the ratings provided by all survey respondents for the school. Because of publicity about the effects of industrialized swine production in North Carolina, we were concerned that survey questions specifically about swine CAFOs would cause response bias; therefore, respondents were asked about livestock odor in general. Public concern about odor from swine CAFOs has generated more reports to the health department than have other types of livestock operations in the state (Cline JS, personal communication). However, survey respondents did report odor from other livestock, primarily poultry; this was sometimes noted as a comment on the survey form. To avoid misclassifying these schools as being exposed to swine CAFO odor, we excluded from analysis 39 schools located > 5 miles from a swine CAFO for which respondents indicated the presence of livestock odor. Our final population for analysis was 226 public schools. Approximately 11% (145,704 of 1,315,363) of all students in North Carolina were enrolled in our population of schools during the 2003 school year.

To assess survey response within demographic and economic categories, we used data from the State of North Carolina (National Center for Education Statistics 2004; North Carolina Department of Public Instruction 2003) describing each school's racial and ethnic composition and enrollment in the National School Lunch Program, used here as a proxy for SES. Students participating in the National School Lunch Program receive lunches for free or at reduced price, with the level of subsidy determined by the income of each child's family. Children from families with incomes ≤ 130% or between 130 and 185% of the poverty level are eligible for fully or partially subsidized lunches, respectively (Child Nutrition and WIC Reauthorization Act 2004; U.S. Department of Agriculture 2004). We classified schools into race and SES categories using the median values of white enrollment (median, 63%) and subsidized lunch (median, 47%). The resulting matrix of race and economics was used to identify schools as high white/high SES (96 schools), low white/high SES (16 schools), high white/low SES (18 schools), and low white/low SES (96 schools).

We assessed the association between the race, economics, and both metrics of school-based swine CAFO exposure using binary regression in a log-linear model to estimate the prevalence of the exposures. Regression models were adjusted for rural school locale using data from the National Center for Education Statistics (2004), which uses information about proximity to metropolitan areas and population size and density to assign a locale code to each school. We categorized schools as rural if they were identified as "not within a consolidated metropolitan statistical area (CMSA) or metropolitan statistical area (MSA) and designated as rural" or "within a CMSA or MSA and designated as rural." All remaining categories, including location within large or mid-size central cities, urban locations, or small towns with populations of at least 2,500, were categorized as nonrural. All independent variables in the models are school-level variables, and the resulting measures of association are prevalence ratios (PRs). We used SAS statistical software (version 8.2; SAS Institute Inc., Cary, NC) for all analyses.

Results

Across the 226 schools, mean enrollments of black and white students, respectively, were 26% and 63%. The mean enrollments of Asian students (< 1%), Hispanic students (3%), and Native-American students (< 1%) were low, and none of the schools had majority enrollment of Asian or Hispanic students. The percentage of enrolled students receiving fully or partially subsidized lunches was highly correlated with white, non-Hispanic enrollment (Figure 1).

For the 226 schools, distances between schools and the nearest swine CAFO ranged from 0.2 to 42 miles (mean ± SE, 8.3 ± 0.5), and mean distances increased across tertiles of white enrollment (low, 4.9; medium, 7.0; high, 12.7 miles) and SES (low, 4.6; medium, 8.4; high, 12.1 miles). Sixty-six (66) schools were located within 3 miles of one or more operations (Figure 2). Livestock odor was reported outdoors at 47 (21%) of the surveyed schools. In 19 schools (8%), the livestock odor was noticeable indoors, including in classrooms and hallways of the school buildings

and in temporary, portable classroom buildings. Overall, the average livestock odor rating was 2.2 (SE = 0.2), which corresponds to an odor rating between "faint" and "moderate" on the scale used for the survey. The average rating of odor at schools with odor noticeable inside the school building was 2.8 (SE = 0.3). The percentage of schools reporting livestock odor and ratings of the strength of the odor each decreased with increasing distance to the nearest swine CAFO (Figure 3). The percentage of schools located within 3 miles of a swine CAFO was lowest (16%) in high-SES schools. A similar percentage (17%) was observed when exposure was considered using reported livestock odor.

Table 1 shows estimates of the relationship of race and SES with distance to the nearest swine CAFO. Having a swine CAFO within 3 miles was most prevalent in schools with low-white/low-SES enrollment [PR = 2.93; 95% confidence interval (CI), 1.79–4.80] compared with schools in the highest category of white enrollment and SES. Restricting the outcome to school location within 2 miles (n = 44) showed a similar trend of higher prevalence among low SES schools. A swine CAFO within 2 miles was more prevalent in schools with low-white/low-SES enrollment (n = 26; PR = 2.62; 95% CI, 1.38–4.97), high-white/low-SES enrollment (n = 5; PR = 2.43; 95% CI, 0.97–6.06), and low-white/high-SES enrollment (n = 2; PR = 1.39; 95% CI, 0.34–5.71) compared with schools with high-white/high-SES enrollment (n = 11). When exposure was considered using survey-based reports of livestock odor, the highest prevalences of noticeable odors outside or inside the school buildings were in schools with low SES enrollment (high white/low SES: n = 5, 28%; low white/low SES: n = 23, 24%), and the lowest prevalence of such odor was observed in schools with high-white/high-SES enrollment (n = 16, 17%) (Table 2). The mean (± SE) odor rating declined across tertiles of percent white, non-Hispanic enrollment (low, 2.1 ± 0.3; medium, 2.5 ± 0.4; high, 1.9 ± 0.4) and SES (low 2.4 ± 0.3; medium, 2.1 ± 0.3; high, 2.0 ± 0.5).

By excluding 39 schools for which survey respondents reported livestock odor, but located beyond 5 miles of a swine CAFO, we intended to reduce misclassification of schools located near nonswine CAFOs. Among the excluded schools, 33 had high enrollments of white students (high white/high SES, 25 schools; high white/low SES, 8 schools). Inclusion of these 39 schools approximately doubled the prevalence of reported odor (outside or inside, 34%; outside only, 18%; outside and inside, 19%) and resulted in marked attenuation of the effect of low white enrollment (outside or inside: PR = 0.89; 95% CI, 0.59–1.32; outside only: PR = 0.88; 95% CI, 0.48–1.61; outside and inside: PR = 0.86; 95% CI, 0.45–1.64).

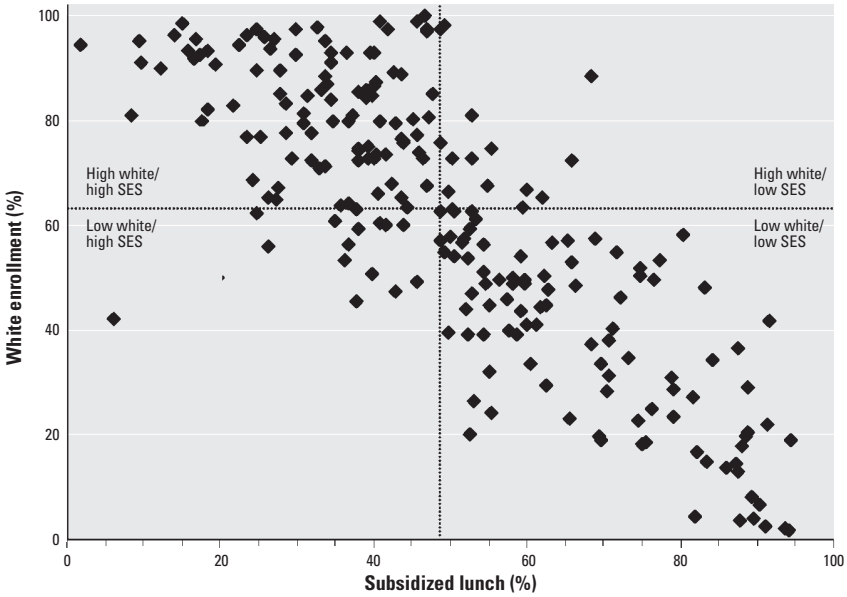

Figure 1. Distribution of white race and economic disadvantage in 226 public schools in North Carolina. Percentages are based on the population students enrolled during the 2003–2004 school year identified as white, non-Hispanic, and receiving subsidized lunches through the National School Lunch Program.

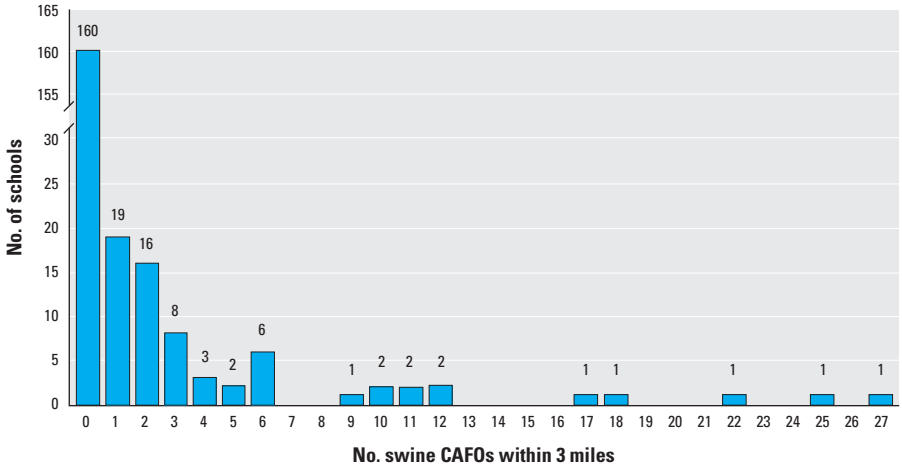

Figure 2. Number of schools by number of swine CAFOs within 3 miles.

Discussion

In 2002, there were approximately 56,000 crop and livestock farms in North Carolina, and nearly 30% of the state's land was used for agricultural production, including the cattle, hog, and poultry industries that significantly contribute to the state's agricultural economy (North Carolina Department of Agriculture and Consumer Services 2003). Previous research about the presence of swine CAFOs shows a disproportionately high concentration of the industry in communities of color despite the declining number of black farmers in the southeastern United States (Wilson et al. 2002; Wing et al. 1996, 2000). In this study we examined the relationship of the racial and economic characteristics of students enrolled in public schools in North Carolina with estimated exposure to airborne effluent from nearby swine CAFOs and found that economic disadvantage was associated

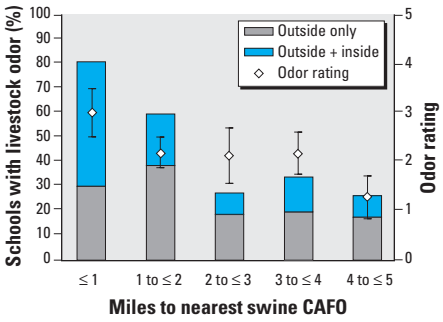

Figure 3. Percentage of schools with noticeable livestock odor and mean ± SE odor ratings for schools with reported odor, by distance between the school and the nearest swine CAFO.

with proximity to the nearest swine CAFO and with strength of the odor. These findings suggest that swine CAFO emissions and any inhalable exposures, including odorant and nonodorant chemicals and respirable organic dusts, that correlate with odor disproportionately affect a population of children and adults who, regardless of their livestock-related exposures, may be predisposed to asthma-related health outcomes and other illnesses for reasons largely attributable to their economic disadvantage (Gee and Grimpayne-Sturgesalt 2004).

Odorous plumes arising from livestock farms contain a variety of gaseous and particulate elements, including inhalable dusts, bacteria, mold, hydrogen sulfide, ammonia, methane, pharmaceutical residues, and animal dander (Reynolds et al. 1997; Subramanian et al. 1996). In this study, the composition of the air present when livestock-related odors were reported and the specific agents responsible for the odor are both unknown. Without information about the extent to which odorous plumes from CAFOs contain respiratory irritants or odorants capable of inducing health effects (Shusterman 1992), we cannot draw conclusions about exposures relevant for respiratory health of the enrolled students, school employees, or neighbors. However, livestock-related odor at public school buildings indicates the presence of airborne livestock effluent beyond the agricultural land from which it arose and in the surrounding community. Reports of livestock odor outside and inside school buildings raise concern not only about health risks resulting from swine CAFO effluent but also about educational and behavioral consequences such as classroom disruptions that might occur when livestock odor reaches the classroom, anxiety associated with the students' and staff members' inability to avoid the odor or change their environments, and concerns or precautions for students who have a history of acute respiratory reactions. Our results clearly suggest that livestock odor is a more common problem for schools with lower SES enrollment. Livestock odors at public schools, particularly those in economically disadvantaged areas, may have broad implications for schools and communities if such schools are unappealing to new teachers and staff or if odors affect the retention of current employees, influence parent and volunteer involvement, or affect the use of school facilities for recreational and community purposes.

Because nonodorous pollutants arising from swine CAFOs may also be present in these communities, our analysis included distance as a measure of potential exposure to airborne swine CAFO effluent. Overall, we observed increased frequency of swine CAFOs near schools with above-median enrollment in the National School Lunch Program. Distances between schools and swine CAFOs were estimated using publicly available data about the locations of public school buildings and hog operations that raise more than 250 animals using a liquid waste management system. Smaller confinement-based operations, smaller "family farms," and confined livestock operations that produce chickens, turkeys, or other animals are not included in our distance comparisons but may be included in reports of odor from livestock farms. We excluded 39 schools for which respondents reported livestock odor but that were located > 5 miles beyond a swine CAFO. Inclusion of the excluded schools approximately doubled the prevalence of reported odor, and the effect of low white/low SES, compared with high white/high SES, changed from elevated risk to reduced risk in each of the three livestock odor models, suggesting that schools with high white enrollment are disproportionately exposed to odors from other types of livestock operations. Among the excluded schools, 13% (n = 5) returned surveys with specific mention of livestock odor from poultry, compared with 3% (n = 7) of the schools included in our main analysis. In both populations of schools, reports of poultry odor were more common among schools with ≥ 63% enrollment of white students (excluded schools, 15%; schools in main analysis, 4%) than among schools with < 63% enrollment of white students (excluded schools, 0%; schools in main analysis, 2%); a less pronounced division was observed across categories of SES. Information about odor from poultry operations was not directly solicited on our surveys, so our supposition that poultry operations are located near schools with higher white enrollment, based on the demographics of schools with survey-reported poultry odor, is uncertain. We were unable to evaluate the proximity of schools to poultry operations because few poultry operations in North Carolina require government-issued liquid waste management permits from which location data can be abstracted. The State of North Carolina does not currently release information about the locations of poultry CAFOs because of state regulations about confidentiality of agricultural data (State of North Carolina 2002).

Study limitations. Our survey-based reports of livestock odor are vulnerable to several sources of potential bias. If respondents at schools with higher enrollment of white students are more likely to report livestock odor on our survey than are respondents at schools with higher nonwhite enrollment, then this finding may be the result of biased survey response. If ventilation or window use correlate with enrollment, then differences in the odor reports may be due to differences in indoor odor levels. School SES may be correlated with the size, age, technology, or other features of livestock operations that affect odor. And, although in the distance-based analyses we categorized proximity to a swine CAFO as within 3 miles of at least one swine

Table 1. Associations between distance to the nearest swine CAFO and public school enrollment in North Carolina.

Enrollment[a]	All schools (no.)	Distance to nearest swine CAFO: > 3 miles No. (%)	≤ 3 miles No. (%)	≤ 3 miles PR (95% CI)[b]
All schools	226	160 (70.8)	66 (29.2)	
High white/high SES	96	80 (83.3)	16 (16.7)	1.00
High white/low SES	18	12 (66.7)	6 (33.3)	1.95 (0.90–4.25)
Low white/high SES	16	14 (87.5)	2 (12.5)	0.95 (0.24–3.72)
Low white/low SES	96	54 (56.3)	42 (43.8)	2.93 (1.79–4.80)

[a]Enrollment categories: high white, ≥ 63% enrollment of white, non-Hispanic students; low white, < 63% enrollment of white, non-Hispanic students; high SES, < 47% of students receiving free or reduced price lunch at school; low SES, ≥ 47% of students receiving free or reduced price lunch at school. [b]Adjusted for rural school locale.

Table 2. Associations between noticeable livestock odor and public school enrollment in North Carolina.

Enrollment[a]	All schools (no.)	No odor No. (%)	Outside or inside No. (%)	Outside or inside PR (95% CI)[b]	Outside only No. (%)	Outside only PR (95% CI)[b]	Outside + inside No. (%)	Outside + inside PR (95% CI)[b]
All schools	226	179 (79.2)	47 (20.8)		28 (12.4)		19 (8.4)	
High white/high SES	96	80 (83.3)	16 (16.7)	1.00	9 (9.4)	1.00	7 (7.3)	1.00
High white/low SES	18	13 (72.2)	5 (27.8)	1.63 (0.70–3.80)	4 (22.2)	2.42 (0.86–6.84)	1 (5.6)	0.89 (0.12–6.62)
Low white/high SES	16	13 (81.3)	3 (18.8)	1.44 (0.48–4.30)	2 (12.5)	1.87 (0.45–7.79)	1 (6.3)	1.12 (0.15–8.49)
Low white/low SES	96	73 (76.0)	23 (24.0)	1.58 (0.90–2.78)	13 (13.5)	1.63 (0.74–3.61)	10 (10.4)	1.66 (0.66–4.15)

[a]Enrollment categories: high white, ≥ 63% enrollment of white, non-Hispanic students; low white, < 63% enrollment of white, non-Hispanic students; high SES, < 47% of students receiving free or reduced price lunch at school; low SES, ≥ 47% of students receiving free or reduced price lunch at school. [b]Adjusted for rural school locale.

CAFO, the number of swine CAFOs located near a school and the distances and geographic directions between the school and each of the nearby swine CAFOs are each reflected in the survey-based estimates of swine CAFO exposure. The surveys provided an estimate of total exposure, whereas the analysis based solely on distance may have underestimated the burden of exposure on schools located near more than one swine CAFO. These components of exposure would be important to consider in an assessment of health impacts of swine CAFO–related exposures.

Our sample size was determined largely by whether employees in each of the surveyed schools participated in our environmental health survey. If the presence of livestock odor at the school or the presence of the livestock industry in the community systematically influenced employees' decisions to complete and return surveys, then our sample of schools may not be representative of the surveyed population. If embarrassment, denial, or exaggeration of the odor problem affected respondents' odor ratings, or if respondents' adaptation to the odor affected their ratings, then the distribution of odor reported on our surveys may not reflect the presence of odorant chemicals in this population of schools. For example, if respondents in farming communities and who routinely smell livestock odor rate the odor as less severe than do survey respondents who are not routinely exposed outside of the school, then the exposures of more exposed schools may be underestimated in these data.

To assess potential bias in survey response, we evaluated school-level survey participation and found that response rates increased across tertiles of increasing percent enrollment of white, non-Hispanic students (< 51% white, 75% participation; 51% to < 78%, 77%; ≥ 78%, 85%). Lower participation among schools with larger nonwhite populations may reflect a broad pattern of nonparticipation in research activities initiated by predominantly white institutions (Corbie-Smith et al. 1999, 2004; Gamble 1993; Shavers-Hornaday et al. 1997). Among participating schools, our classification of the presence of livestock odor based on employees' responses to the survey question may have introduced additional bias in our results. We mailed more than one survey to each school and received up to seven completed surveys per school; for each survey question, we assigned the exposure to a school if any respondent indicated the presence of livestock odor at the school. Consequently, our exposure assignments were sensitive to the number of surveys completed and returned from each school. With each additional survey returned from a single school, and with each additional respondent providing a new opportunity for the school to be classified as exposed, the likelihood of a school's classification as having noticeable livestock odor increased. To assess the impacts of our use of all survey responses and our method of classifying exposure, we estimated the effect of race and economic characteristics on livestock odor using data from one randomly selected survey from each of the participating schools. We repeated this sampling 50 times to generate a range of estimates. On repeated sampling and estimation of the effect of race and economics on any noticeable livestock odor, variation in PRs was low, with 76% (38 of 41) of low-white/high-SES estimates, 80% (40 of 50) high-white/low-SES estimates, and 42% (21 of 50) of low-white/low-SES estimates being closer to the null than the results we report.

Conclusions

Our results provide evidence that North Carolina's swine CAFOs are located closer to schools enrolling higher percentages of nonwhite and economically disadvantaged students and that livestock odor is a more common problem for schools with lower SES enrollment. By considering the environmental exposures that adolescents attending school near the facilities may experience, our findings support and extend previous research about the density of swine CAFOs in nonwhite and poor communities (Wilson et al. 2002; Wing et al. 2000), and the association between environmental exposure and race and poverty in communities located near industrial sources of air pollution (Perlin et al. 1999). Understanding the vulnerability of populations bearing the burden of swine CAFO exposures is of public health importance because of the health risks associated with swine CAFOs and swine odor in other studies (Avery et al. 2004; Merchant et al. 2005; Thu et al. 1997; Wing and Wolf 2000) and the likelihood that hazardous air pollutants arising from swine CAFOs affect the health of children in similar ways. Our findings may have implications for school personnel, particularly those in economically disadvantaged communities, who are concerned about this common exposure and its potential impact on adolescents' respiratory health and should be used to address existing racial and economic disparities in exposure to environmental health hazards.

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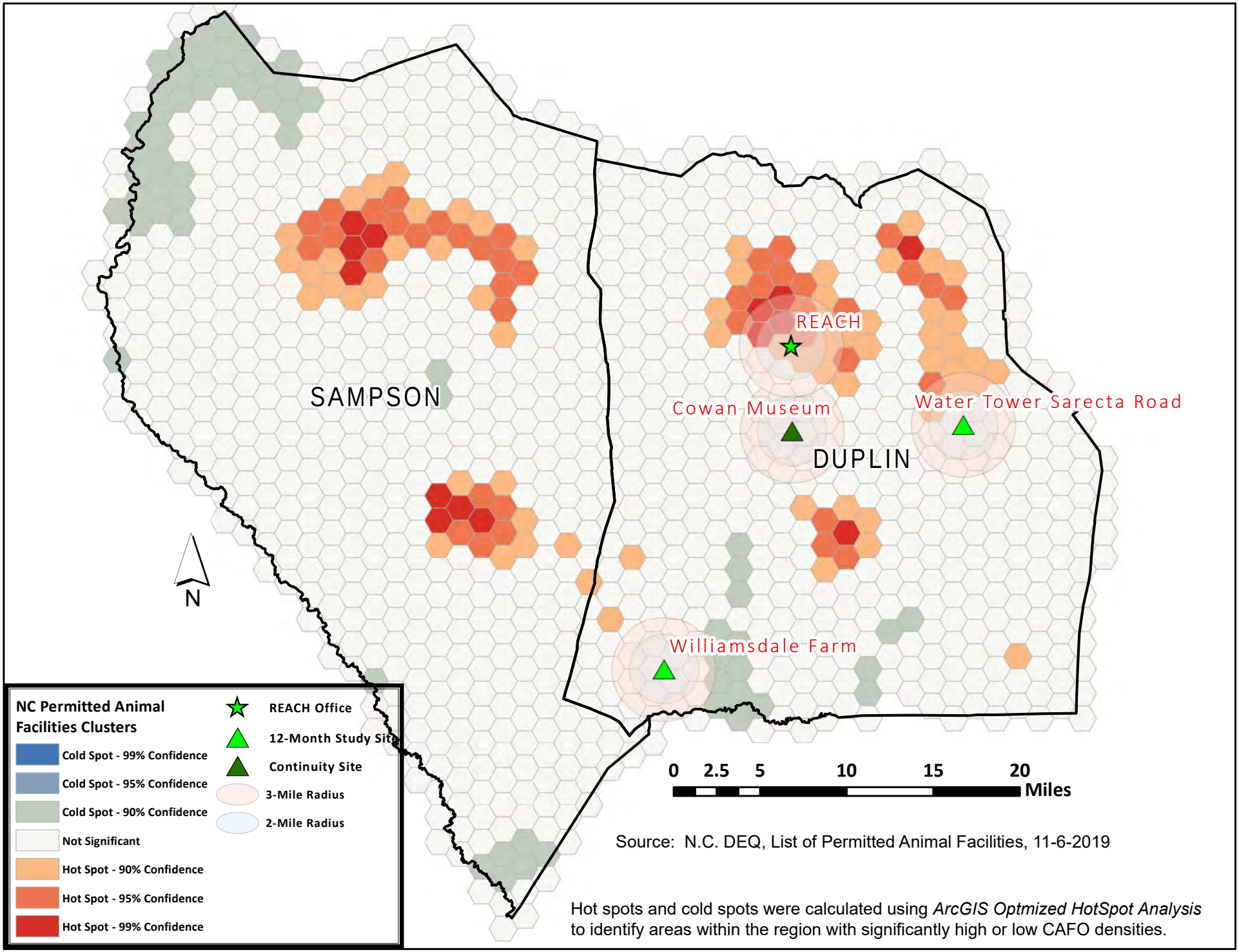
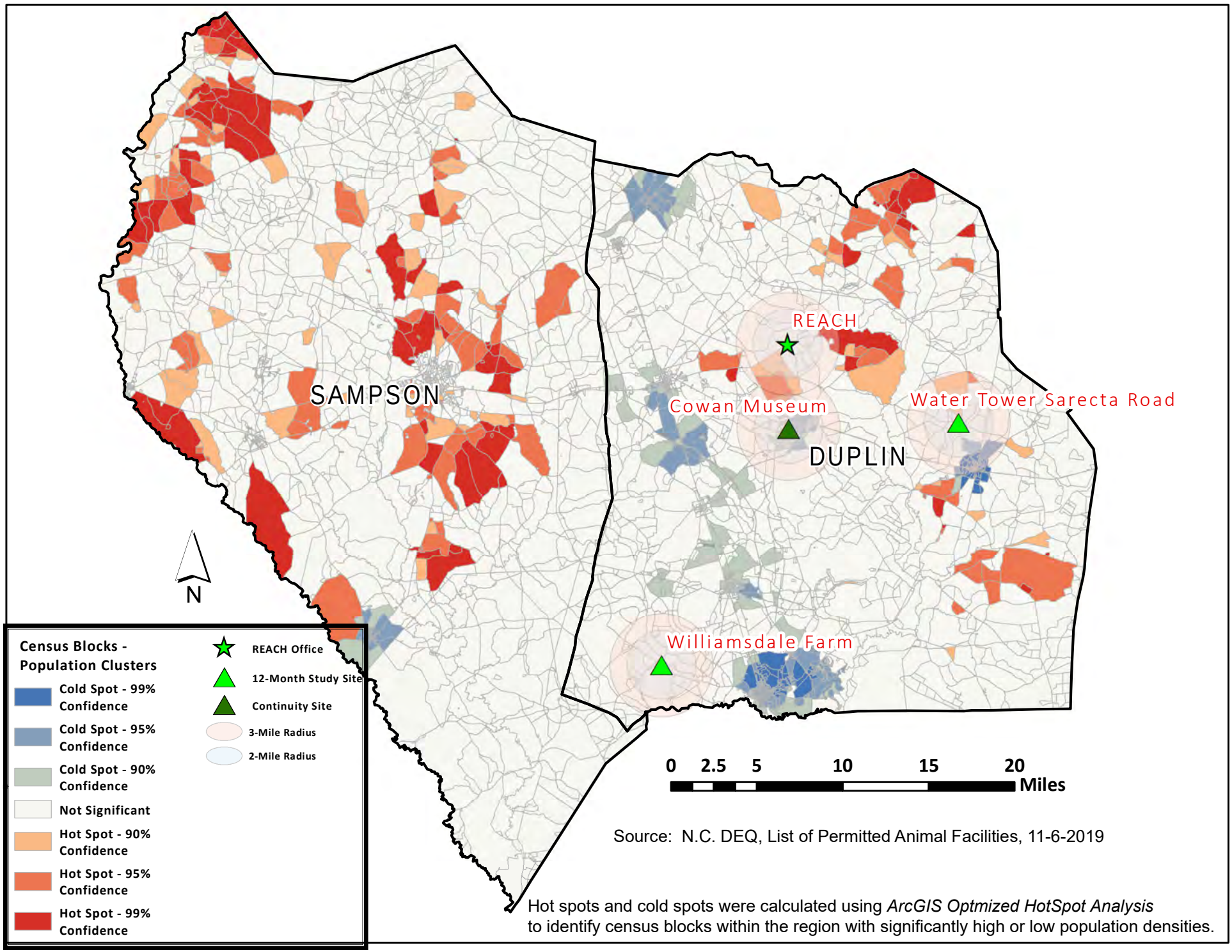
"Hot Spots" and "Cold Spots" of CAFOs in Sampson and Duplin County



Source: N.C. DEQ, List of Permitted Animal Facilities, 11-6-2019

Hot spots and cold spots were calculated using *ArcGIS Optmized HotSpot Analysis* to identify areas within the region with significantly high or low CAFO densities.

Population Clusters by Census Block in Duplin and Sampson Counties



Source: N.C. DEQ, List of Permitted Animal Facilities, 11-6-2019

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