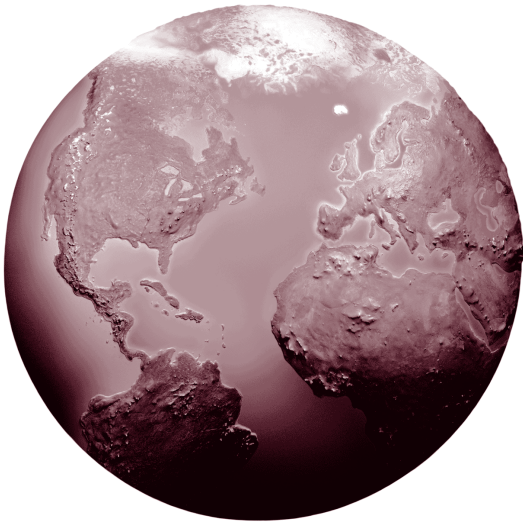

HEALTH EFFECTS INSTITUTE

ANNUAL CONFERENCE 2019

Program and Abstracts

May 5–7, 2019

W Seattle Hotel
1112 4th Avenue
Seattle, Washington

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Trusted Science • Cleaner Air • Better Health

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HEALTH EFFECTS INSTITUTE

2019 Annual Conference

May 5–7
W Seattle Hotel
Seattle, Washington

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Please Note: With the speakers' permission, presentation slides will be posted at *www.healtheffects.org/annual-conference* after the conference.

AT A GLANCE

Sunday May 5

9:30 Registration Opens

11:30 Lunch

1:00 Welcome and Conference Opening

1:10 New Mobility: Changing Health?

4:00 Poster Session 1

6:00 Opening Reception, Dinner, and Awards Presentation

Monday May 6

7:00 Breakfast

8:00 Health Effects of Early-Life Exposure to Air Pollution

10:30 Poster Session 2

12:15 Lunch

1:15 Global Health: Building Science for Informed Action

3:30 Where There's Wildfire, There's Smoke

5:30 Adjourn for Free Evening

Tuesday May 7

7:00 Breakfast

8:30 How Low? Testing Health Effects at the Lowest Levels of Air Pollution

11:30 Boxed Lunch

12:00 HEI Strategic Plan 2020–2025

1:30 Conference Adjourns

HEI Annual Conference 2019 Program

Sunday, May 5

11:30 am	Lunch

1:00 pm **Welcome and Conference Opening**

Dan Greenbaum, Health Effects Institute

1:10 pm **New Mobility: Changing Health?**

Chairs: *David Foster*, University of Wisconsin, Madison, and HEI Research Committee; and *Frank Kelly*, King's College London, United Kingdom, and HEI Review Committee

Mobility systems are undergoing a paradigm shift and have the potential to significantly change the way people travel. Rideshare businesses are expanding, and innovations and investments in electric and autonomous vehicles are increasing. In response, the relationship between transportation and air quality is also expected to change, but the actual path that change will take is not yet known. This session will explore the likely impact of emerging mobility trends on current and future air quality and health.

1:10	Introduction *Frank Kelly*
1:15	Global Overview of the Transportation Revolution *Dan Sperling*, University of California, Davis
1:45	Connection Between Changing Mobility and Infrastructure *Charlene Rohr*, RAND, United Kingdom
2:15	Effects of Technologies and Their Deployment on Air Pollution *Marianne Hatzopoulou*, University of Toronto, Canada
2:45	Potential Health Implications of New Mobility *Haneen Khreis*, Texas A&M University
3:15	Panel Discussion Moderator: *David Foster* Discussants: *Shannon Walker*, City of Seattle Department of Transportation, *Britta Gross*, General Motors, and the speakers

3:45 pm	Break

4:00 pm **Poster Session 1**

6:00 pm **Opening Reception, Dinner, and Awards Presentation**

Conference Program continues next page

Monday, May 6

7:00 am Breakfast

8:00 am **Health Effects of Early-Life Exposure to Air Pollution**

Chairs: *Barbara Hoffmann*, University of Düsseldorf, Germany, and HEI Research Committee; and *David Savitz*, Brown University and HEI Research Committee

Evidence for potential impacts of prenatal and early-life air pollution exposure on health is rapidly increasing, and some birth outcomes are currently being considered for inclusion in Global Burden of Disease estimates. This session will provide an overview of the state of knowledge on various birth and other childhood health outcomes, consider methodological issues unique to this topic, and discuss longer-term consequences of early-life exposures for adult health.

8:00 Introduction
Barbara Hoffmann

8:10 Epidemiological Evidence for Adverse Birth Effects Associated with Prenatal Exposure to Air Pollution
Marie Pedersen, University of Copenhagen, Denmark

8:35 Respiratory Effects and Asthma in Children
Rosalind Wright, Icahn School of Medicine at Mount Sinai

9:00 Neurodevelopmental Effects
Sharon Sagiv, University of California, Berkeley

9:25 Obesity and Type 2 Diabetes in Children
Tanya Alderete, University of Colorado, Boulder

9:50 General Discussion

10:00 am Break

10:30 am **Poster Session 2**

12:15 pm Lunch

1:15 pm **Global Health: Building Science for Informed Action**

Chairs: *Kiros Berhane*, University of Southern California and HEI Review Committee; and *Aaron Cohen*, Health Effects Institute and Institute for Health Metrics and Evaluation, University of Washington, Seattle

In regions where air pollution levels are high, local data on exposures, sources, and related health effects are often limited. While current health burden estimates draw primarily from studies conducted in North America and Europe, efforts are underway to generate locally relevant air quality and health data in developing countries. This session will explore data, methods, and technology developments to characterize air pollution and its sources and health effects in these countries.

1:15 Introduction and Overview of HEI Global Health Program
Katy Walker, Health Effects Institute

1:25 Harmonizing Disparate Global and Local Air Quality Data to Support Research and Communication
Christa Hasenkopf, OpenAQ

1:45 New Developments and Opportunities in Global Satellite Technology for Air Pollution and Health Research
Bryan Duncan, National Aeronautics and Space Administration

2:05 Improving Understanding of Concentration Response Functions in Countries with High Ambient Particulate Matter Exposure: China Cohort Studies of Air Pollution and Health
Haidong Kan, Fudan University, Shanghai, China

2:25 GBD 2017 State-Level Burden of Disease from Air Pollution in India: Findings and Future Research Needs
Lalit Dandona, Public Health Foundation of India

2:45 General Discussion

3:15 pm Break

3:30 pm Where There's Wildfire, There's Smoke

Chairs: *Jennifer Peel*, Colorado State University and HEI Review Committee; and *Allen Robinson*, Carnegie Mellon University and HEI Research Committee

Wildfire smoke is increasingly recognized as an important source of air pollution, and frequency and intensity of wildfires are likely to increase with climate change. Wildfires and wildfire smoke composition are complex and dynamic, making exposure characterization difficult. Increasing evidence links air pollution from wildfire smoke to adverse health effects, in particular respiratory morbidity. This session will explore perceptions and realities about wildfires and their global impacts.

3:30 Why Study the Health Effects of Wildfires?
Jennifer Peel

3:50 Where There's Wildfire, There's Smoke: An Epidemiological Perspective
John Balmes, University of California, San Francisco

4:10 Modeling, Monitoring, and Messaging Wildfire Smoke for Air Quality and Public Health
Sim Larkin, U.S. Department of Agriculture, Forest Service

4:30 Hands-On Experience Mitigating Wildfire Impacts
Sarah Coefield, Missoula City-County Health Department

4:50 U.S. EPA's Research Perspectives on the Health Impacts of Wildfires and Wildfire Smoke
Alan Vette, U.S. Environmental Protection Agency

5:10 General Discussion

5:30 pm Free Evening

Tuesday, May 7

7:00 am Breakfast

8:30 am How Low? Testing Health Effects at the Lowest Levels of Air Pollution

Chairs: *Amy Herring*, Duke University and HEI Research Committee; and *Sverre Vedal*, University of Washington, Seattle

Although ambient air pollution levels are declining in high-income regions, epidemiological studies report associations with health effects at levels below current standards, raising questions about even lower standards. HEI is in the midst of funding three studies investigating the health effects of low-level exposure in very large populations in the United States, Canada, and Europe. This session will present the results currently available from those studies and their strengths and weaknesses identified by an independent HEI Review Panel, and discuss implications for future risk assessment and regulation.

8:30 What Are the Risk Assessment and Policy Decisions to Be Informed?
Dan Greenbaum, Health Effects Institute

8:40 Introduction to HEI's Program to Assess Health Effects of Long-Term Exposure to Low Levels of Ambient Air Pollution
Hanna Boogaard, Health Effects Institute

8:50 Results from the European Analysis Using ESCAPE Cohorts and Various Large Administrative Data Sets
Maciej Strak, Utrecht University, the Netherlands

9:10 MAPLE: Mortality–Air Pollution Associations in Low Exposure Environments in Canada
Dan Crouse, University of New Brunswick, Canada

9:30 Results from the U.S. Study Using Medicare Data
Qian Di, Harvard T.H. Chan School of Public Health

Conference Program continues next page

	9:50	General Discussion
10:00 am		Break
	10:30	Comments from the HEI Review Panel *Sverre Vedal*
	10:45	The Current Knowledge on Adverse Effects of Low-Level Air Pollution: Have We Filled the Gap? *Jon Samet*, Colorado School of Public Health
	11:00	General Discussion
11:30 am		Boxed Lunch

12:00 pm **The HEI Strategic Plan 2020–2025**

Chairs: *Dan Greenbaum*, *Robert O'Keefe*, and *Rashid Shaikh*; Health Effects Institute

The Institute's draft blueprint for the future, the HEI Strategic Plan for 2020–2025, will be presented and discussed. Conference participants are encouraged to suggest and comment on upcoming policy decisions for which enhanced science will be needed and on priorities for HEI's research programs and other activities during the next five years.

1:30 pm **Conference Adjourns**

POSTER SESSION 1

Sunday, May 5, 4:00–5:45 PM

GLOBAL HEALTH

HEI's Global Health Program — supported by additional funding from foundations and others — includes various initiatives that will be presented. **Dr. Brauer** will evaluate disease burden estimates from ambient $PM_{2.5}$ across seven source sectors at the national level for 195 countries and territories included in the Global Burden of Disease study. **Drs. Pant** and **Walker**, representing the HEI Panel on Air Pollution in Ghana, evaluated the current evidence on the contributions of household air pollution to emissions, ambient air pollution, and health in Ghana. **Dr. Vermeulen** is assessing the association between long-term exposure to outdoor $PM_{2.5}$ and NO_2 and all-cause and cause-specific mortality in a pooled analysis of Asian cohorts. **Dr. Zhang** investigated the impacts on air quality and health of recently implemented and proposed controls on near-shore emissions of air pollutants from ships around one of the busiest port clusters in China.

In related work (not funded by HEI), **Dr. Achakulwisut**, a Postdoctoral Travel Award Recipient, will provide the first global estimate of the burden of pediatric asthma incidence attributable to ambient NO_2 pollution at 250 m spatial resolution; **Dr. Burkart** assessed the global burden of adverse reproductive health outcomes attributable to air pollution; and **Ms. Causey** estimated the global burden of type 2 diabetes attributable to ambient $PM_{2.5}$ and household air pollution from 1990 to 2017 within the comparative risk assessment framework of the Global Burden of Disease project.

Global, National, and Urban Burdens of Pediatric Asthma Incidence Attributable to Ambient NO_2 Pollution*
Ploy Pattanun Achakulwisut (Presenter and Travel Award Recipient), Michael Brauer, Perry Hystad, and Susan C. Anenberg

Global Burden of Disease–Major Air Pollution Sources — a GLOBAL Approach
Michael Brauer, Erin E. McDuffie, Randall V. Martin, Joseph Spadaro, and Richard T. Burnett

Estimating the Burden of Adverse Reproductive Health Outcomes Attributable to Air Pollution*
Katrin Burkart, Rakesh Ghosh, Kate Causey, Richard Burnett, Aaron Cohen, and Michael Brauer

The Global Burden of Diabetes Mellitus Type 2 Due to Indoor and Outdoor Sources of Particulate Matter Pollution*
Kate Causey, Katrin Burkart, Michael Brauer, Richard Burnett, Aaron Cohen, Stephen S. Lim, Devashri Salvi, and Jeffrey Stanaway

Contribution of Household Air Pollution to Ambient Air Pollution in Ghana: Using Available Evidence to Prioritize Future Action
Pallavi Pant (Presenter), Katy Walker, and the HEI Panel on Air Pollution in Ghana

Long Term Outdoor Air Pollution and Cause-Specific Mortality in a Pooled Analysis of Asian Cohorts
George S. Downward, Gerard Hoek, Perry Hystad, Eiko Sato, Manami Inoue, Sarah K. Abe, Md. Shafiur Rahman, Nat Rothman, Qing Lan, Zhu Tong, Lutzen Portengen, and Roel C.H. Vermeulen

* Study not funded by HEI.

Impacts of Shipping on Air Pollutant Emissions, Air Quality, and Health in Yangtze River Delta/Shanghai, China
Junlan Feng, Yan Zhang, Cong Liu, Junri Zhao, Weichun Ma, Cheng Huang, Jingyu An, Yin Shen, Qingyan Fu, Shuxiao Wang, Dian Ding, Wangqi Ge, Freda Fung, Kethural Manokaran, Allison P. Patton, Katy Walker, and Haidong Kan

HEALTH EFFECTS OF TRAFFIC-RELATED AIR POLLUTION AND NOISE

Three studies were funded in 2017 under RFA 17-*1, Assessing Adverse Health Effects of Exposure to Traffic-related Air Pollution, Noise, and their Interactions with Socio-Economic Status*. **Drs. Dadvand** and **Sunyer** are assessing the effects of maternal exposure to traffic-related pollution on birth weight, fetal growth trajectories, and placental function in a new pregnancy cohort in Barcelona. **Dr. Franklin** is evaluating environmental factors affecting stress in children and their contribution to health effects in the most recent cohort of the Children's Health Study in southern California. **Dr. Raaschou-Nielsen** is evaluating myocardial infarction, stroke, diabetes, and related biomarkers diabetes in three large Danish cohorts. All three studies are estimating exposure to several pollutants and transportation noise, and evaluating the roles of socioeconomic status, green space, physical activity, diet, and stress.

Traffic-Related Air Pollution and Birth Weight: The Roles of Noise, Placental Function, Green Space, Physical Activity, and Socioeconomic Status (FRONTIER)
Payam Dadvand, Jordi Sunyer (Presenter), Maria Dolores Gómez-Roig, Elisa Llurba, Mar Alvarez, Gustavo Arévalo, Mariona Bustamante, Xavier Basagaña, Maria Foraster, Mireia Gascon, Michael Jerrett, Jose Lao, Edurne Mazarico Gallego, Teresa Moreno, Tim Nawrot, Mark J. Nieuwenhuijsen, Xavier Querol, Joel Schwartz, and Cathryn Tonne

Environmental Factors Affecting Stress in Children: Interrelationships Between Traffic-Related Noise, Air Pollution, and the Built Environment
Meredith Franklin, Xiaozhe Yin, Robert Urman, Rebecca Lee, Scott Fruin, and Rob McConnell

Health Effects of Air Pollution Components, Noise, and Socio-Economic Status ("HERMES")
Ole Raaschou-Nielsen, Theis Lange, Matthias Ketzel, Ulla Hvidtfeldt, Henrik Brønnum-Hansen, Thomas Münzel, Lise M. Frohn, Jesper Christensen, Ulas Im, Ole Hertel, Jørgen Brandt, and Mette Sørensen

IMPROVING ASSESSMENT OF EXPOSURE TO AIR POLLUTION

HEI supports research and advancement regarding the assessment of exposure to air pollution, including the use of mobile sampling platforms, for application in health studies. **Dr. Apte**, a 2017 recipient of HEI's Walter A. Rosenblith New Investigator Award, is comparing and contrasting insights regarding fine-scale variation in black carbon gained from mobile monitoring and fixed measurement techniques. **Mr. Cook** estimated the potential contribution of highway vehicles and other mobile source sectors to secondary concentrations of carbonyl compounds in the atmosphere (not funded by HEI). **Dr. Koutrakis** used a mobile sampling laboratory to collect particle samples in the greater Boston area to measure levels of particles in tailpipe and non-tailpipe emissions and resuspended road dust, as well as factors affecting their levels. **Dr. Sheppard** designed an approach to optimize a mobile monitoring campaign to obtain unbiased annual average estimates of multiple pollutants in an epidemiological cohort (not funded by HEI).

Mapping Air Pollution at High Spatial Resolution: Comparing Mobile and Fixed Sensing Approaches
Sarah E. Chambliss and Joshua S. Apte

Contribution of Mobile Sources to Secondary Formation of Carbonyl Compounds*
Rich Cook, Sharon Phillips, Madeleine Strum, Alison Eyth, and James Thurman

Chemical and Physical Characterization of Non-Tailpipe and Tailpipe Emissions at 100 Locations Near Major Roads in the Greater Boston Area
Petros Koutrakis, Brent Coull, Marco Martins, Joy Lawrence, and Stephen Ferguson

Design and Implementation of a Mobile Monitoring Campaign for Epidemiology*
Magali Blanco, Amanda Gassett, Edmund Seto, Tim Larson, and Lianne Sheppard

POTENTIAL BENEFITS OF AIR QUALITY IMPROVEMENTS

HEI supports accountability and health impact research to evaluate whether regulations and other actions taken to improve air quality result in the intended public health benefits, and to predict likely benefits of future actions. **Dr. Hakami** will estimate the location-specific marginal benefits of reducing air pollution through integration of air quality modeling, concentration–response functions, and economic valuation. **Dr. Meng** evaluated the impact of the 2006 Emission Reduction Plan for Ports and Goods Movement issued by the California Air Resources Board to improve air quality in the goods movement corridors. Following Phase 1 to evaluate air quality improvement, Dr. Meng will present Phase 2 findings on changes in health outcomes among California Medicaid enrollees with chronic diseases who are living in areas affected by the Goods Movement Plan compared to those living in control areas. In related work (not funded by HEI), **Dr. Garcia**, a Postdoctoral Travel Award Recipient, examined the effect on asthma incidence from hypothetical air pollution interventions in a population of Southern California children using g-computation, a method based in a framework that uses counterfactuals — that is, compares the regulation to a scenario under "business as usual."

Effects of Policy-Driven Hypothetical Nitrogen Dioxide and Particulate Matter Interventions on Asthma Incidence in Southern Californian Children*
Erika Garcia (Presenter and Travel Award Recipient), Robert Urman, Kiros T. Berhane, Rob McConnell, and Frank D. Gilliland

Quantifying Societal Health Benefits of Transportation Emission Reductions in the United States and Canada
Amir Hakami, Armistead Russell, Alan Krupnick, and Howard Chang

Goods Movement Actions Improved Air Quality and Health Outcomes Among California Medicaid Enrollees (Phase 2 Health Effects Study)
Ying-Ying Meng, Dahai Yue, Jason G. Su, Michael Jerrett, Xiao Chen, and John Molitor

* Study not funded by HEI.

POSTER SESSION 2

Monday, May 6, 10:30 AM–12:15 PM

HEALTH EFFECTS AT LOW AMBIENT CONCENTRATIONS

The three studies funded under HEI RFA 14-3, *Assessing Health Effects of Long-Term Exposure to Low Levels of Ambient Air Pollution*, are investigating health effects in millions of people exposed to low levels of air pollution in North America and Europe. The studies aim to shed light on the observation that some epidemiological studies have reported associations of air pollution with health effects at levels below current air quality standards, raising questions as to whether current standards are protective of the general population. **Dr. Brauer** is investigating mortality effects of low levels of air pollution in Canada using Canadian Census data from about 10 million people. **Dr. Brunekreef** is investigating health effects of low levels of air pollution in Europe using pooled data from 11 ESCAPE (European Study of Cohorts for Air Pollution Effects) cohorts and seven large administrative cohorts, resulting in a study population of about 35 million people. **Dr. Dominici** is examining health effects of low levels of air pollution in the United States using data from about 61 million people enrolled in Medicare and Medicaid and developing new causal modeling methods to characterize the shape of the exposure–response function. Two posters from each of these large studies will be presented.

MAPLE: Mortality–Air Pollution Associations in Low Exposure Environments
Michael Brauer, Jeffrey R. Brook, Yen Li Chu, Tanya Christidis, Dan L. Crouse, Anders Erickson, Perry Hystad, Chi Li, Randall V. Martin, Jun Meng, Amanda J. Pappin, Lauren L. Pinault, Michael Tjepkema, Aaron van Donkelaar, Scott Weichenthal, and Richard T. Burnett

MAPLE: Mortality–Air Pollution Associations in Low Exposure Environments. Evaluating the Sensitivity of $PM_{2.5}$–Mortality Association to the Spatial and Temporal Scale of Exposure Assessment and the Inclusion of Immigrant Populations
Dan L. Crouse, Anders C. Erickson (Co-presenter), Tanya Christidis, Lauren Pinault, Aaron van Donkelaar, Chi Li, Jun Meng, Randall V. Martin, Michael Tjepkema, Perry Hystad, Rick Burnett, Amanda Pappin, Michael Brauer, and Scott Weichenthal (Co-presenter)

Mortality, Morbidity, and Low-Level Air Pollution in a Pooled Cohort of 392,826 in Europe in the ELAPSE Project
Bert Brunekreef, Maciej Strak (Presenter), Kathrin Wolf, Ulla A. Hvidtfeldt, Kees de Hoogh, Sophia Rodopoulou, Evi Samoli, Klea Katsouyanni, Marjan Tewis, Jie Chen, and Gerard Hoek, on behalf of the ELAPSE Project Team

Mortality, Morbidity, and Low-Level Air Pollution in a Population of 35 Million in Europe: Analysis of Administrative Cohorts in the ELAPSE Project
Bert Brunekreef, Danielle Vienneau (Presenter), Nicole Janssen, Massimo Stafoggia, Mariska Bauwelinck, Klea Katsouyanni, Evi Samoli, Sophia Rodopoulou, Maciej Strak, and Gerard Hoek, on behalf of the ELAPSE Project Team

Assessing the Short-Term Effect of $PM_{2.5}$ on Cardiovascular Hospitalizations in the Medicaid Population: A Case-Crossover Study
Priyanka deSouza, Danielle Braun (Co-presenter), Francesca Dominici, and Marianthi-Anna Kioumourtzoglou (Co-presenter)

An Ensemble-Based Model of $PM_{2.5}$ Concentration Across the Contiguous United States with High Spatiotemporal Resolution
Qian Di (Presenter), Heresh Amini, Itai Kloog, Rachel Silvern, James Kelly, M. Benjamin Sabath, Christine Choirat, Petros Koutrakis, Alexei Lyapustin, Yujie Wang, Joel Schwartz, and Francesca Dominici

HEALTH EFFECTS OF AMBIENT AIR POLLUTION ON SUSCEPTIBLE POPULATIONS

Several ongoing studies at HEI are evaluating different aspects of associations between air pollution exposure and various neurological and respiratory outcomes in children, and the influence of socioeconomic factors and genetic backgrounds on cardiovascular outcomes in adults. **Dr. Clougherty** is quantifying combined effects of spatial and temporal trends in multiple pollutants and stressors, including socioeconomic susceptibility, on 1.1 million cardiovascular events in New York City that resulted in emergency department visits. **Dr. Guxens**, the 2016 recipient of HEI's Walter A. Rosenblith New Investigator Award, is investigating the association between autism spectrum disorders and prenatal air pollution exposure, as well as between brain changes in children and prenatal and postnatal air pollution exposure. **Dr. Pedersen**, a 2017 recipient of HEI's Walter A. Rosenblith New Investigator Award, is evaluating whether early-life exposure to air pollution is associated with development of asthma in children and adolescents. In related work (not funded by HEI), **Ms. Hoskovec**, a Student Travel Award Recipient, will present results from a simulation study that evaluates five contemporary statistical methods for estimating health effects associated with multipollutant mixtures in a cohort of asthmatic children; and **Dr. Ward-Caviness** will present environmental exposure data from EPA CARES and the SmokeSense mobile application to demonstrate how research and messaging can be combined to effect change in public health.

Susceptibility to Multiple Air Pollutants in Cardiovascular Disease
Jane E. Clougherty, Jamie L. Humphrey, Ellen J. Kinnee, Laura D. Kubzansky, Colleen E. Reid, Leslie A. McClure, and Lucy Robinson

Air Pollution, Autism Spectrum Disorders, and Brain Imaging Among CHildren in Europe — the APACHE Project
Mònica Guxens, Małgorzata J. Lubczyńska, Laura Perez, Albert Ambrós, Matteo Renzi, Matteo Scortichini, Maciej Strak, Xavier Basagaña, Ryan Muetzel, Antònia Valentín, Itai Kloog, Gerard Hoek, Joel Schwartz, Francesco Forastiere, Tonya White, Jordi Sunyer, Henning Tiemeier, Bert Brunekreef, Massimo Stafoggia, and Hanan El Marroun

Statistical Methods for Estimating the Association Between Multipollutant Mixtures and Health Outcomes: A Simulation Study*
Lauren Hoskovec (Presenter and Travel Award Recipient), Wande Benka-Coker, Rachel Severson, Sheryl Magzamen, and Ander Wilson

Maternal Smoking During Pregnancy and Asthma in Children and Young Adults
Marie Pedersen, Katrine Facius, Zorana J. Andersen, Anne-Marie Nybo Andersen, Xavier Basagaña, Hans Bisgaard, Jørgen Brandt, Esben Budtz-Jørgensen, Klaus Bønnelykke, Casper-Emil Pedersen, Lise Frohn, Leslie Stayner, Matthias Ketzel, Bert Brunekreef, and Steffen Loft

The Impacts of Environmental Exposures on Public Health Outcomes*
Cavin Ward-Caviness, Tom Long, Lisa Baxter, Kristen Rappazzo, Stephanie Deflorio-Baker, Anne Weaver, Ana Rappold, David Diaz-Sanchez, and Wayne Cascio

* Study not funded by HEI.

HEI REVIEWS OF THE SCIENTIFIC LITERATURE

The Health Effects Institute has several scientific literature reviews underway. **Dr. Boogaard** will present progress on an update to HEI's well-cited 2010 critical review of traffic-related air pollution. The current review will evaluate the epidemiological evidence for associations of long-term exposure to traffic-related air pollution with selected adverse human health outcomes. Additionally, **Dr. Rosofsky** will present two critical reviews being conducted for HEI's Energy Research Program on potential exposures to, and health effects from, chemical or non-chemical agents from oil and natural gas derived from shale and other unconventional resources across the United States.

Systematic Review on the Health Effects of Long-Term Exposure to Traffic-Related Air Pollution
Hanna Boogaard, Allison P. Patton, Rashid Shaikh, and the HEI Panel on the Health Effects of Long-Term Exposure to Traffic-Related Air Pollution

Review of the Literature on Potential Exposures and Health Effects from Unconventional Oil and Gas Development
Anna Rosofsky (Presenter), Kathryn Liziewski, Donna Vorhees, Dan Greenbaum, Robert O'Keefe, and Rashid Shaikh

OZONE AND PARTICULATE MATTER MECHANISMS

HEI is funding two experimental studies investigating oxidative responses and chemical cellular changes in response to air pollution. **Dr. Gowdy**, the 2015 recipient of the Walter A. Rosenblith New Investigator Award, is assessing whether vascular injury after exposure to ozone is mediated through changes in the lung and blood of levels of oxidized phospholipids. **Dr. Shiraiwa**, the 2018 recipient of the Walter A. Rosenblith New Investigator Award, is examining reactive oxygen species formation in epithelial lining fluid by ambient particulate matter deposition.

Additionally, final results will be presented from the Multicenter Ozone Study in oldEr Subjects (MOSES). Phase 1 investigated the effects of controlled exposure to ozone on the respiratory and cardiovascular systems (see Research Report 192, 2017). For Phase 2, **Drs. Rich** and **Frampton** assessed whether personal and ambient ozone and other pollutant exposures during the days prior to the participants' visits to the laboratory may have affected the results reported in Phase 1.

Dietary DHA Mitigates Ozone Induced Pulmonary Inflammation and Reductions in Specialized Pro-resolving Mediators
Kymberly M. Gowdy, Brita J. Kilburg-Basnyat, Elizabeth Browder, Sky Reece, Myles Hodge, Bin Luo, Michael J. Yaeger, Michael Odom, Christine Psaltis, Jonathan Manke, Michael L. Armstrong, Johanna L. Hannan, N. Reisdorph, Robert M. Tighe, and S. Raza Shaikh

Multicenter Ozone Study in oldEr Subjects (MOSES), Part 2: Impacts of Personal and Ambient Concentrations of Ozone and Other Pollutants on Cardiopulmonary Biomarkers
David Q. Rich (Presenter), Mark Frampton, Sally W. Thurston, Kelly Thevenet-Morrison, Wojciech Zareba, John Balmes, Mehrdad Arjomandi, Peter Ganz, Philip Bromberg, Milan Hazucha, and Neil Alexis

Formation of Reactive Oxygen Species in Epithelial Lining Fluid by Particle Deposition and Comparisons with Oxidative Potential Measurements
Ting Fang, Pascale S. J. Lakey, and Manabu Shiraiwa

ABSTRACTS

(In Alphabetical Order by Principal Investigator)

Global, National, and Urban Burdens of Pediatric Asthma Incidence Attributable to Ambient NO_2 Pollution*

Ploy Pattanun Achakulwisut[1] (Presenter and Travel Award Recipient), Michael Brauer[2], Perry Hystad[3], and Susan C. Anenberg[1]

[1]George Washington University, Washington, DC, USA; [2]University of British Columbia, Vancouver, BC, Canada; [3]Oregon State University, Corvallis, OR, USA

Background A confluence of evidence from epidemiological, toxicological, and gene-environment studies indicates that there is likely a causal relationship between long-term exposure to traffic-related air pollution (TRAP) and pediatric asthma development. The evidence is most robust for nitrogen dioxide (NO_2) a major component and common proxy of the complex TRAP mixture. At present, the Global Burden of Disease does not include air pollution as a risk factor for asthma incidence or prevalence, and the only previous study to have estimated the global burden of pediatric asthma incidence attributable to ambient air pollution relied on exposure datasets at resolutions too coarse to resolve the intra-urban NO_2 variability driven primarily by TRAP.

Methods In this study, we provide the first global estimate of the burden of pediatric asthma incidence attributable to ambient NO_2 pollution at a spatial resolution (250 m x 250 m) sufficient to resolve intra-urban and near-roadway exposure gradients. Results are provided for 194 countries and 125 major cities. We use 2010-2012 annual average surface NO_2 concentrations derived from land-use regression, 2015 national incidence rates from the Institute for Health Metrics and Evaluation, 2015 global gridded population from the European Commission's Joint Research Center Global Human Settlement Layer, and relative risks from a multi-national meta-analysis of epidemiological studies.

Results We estimate that, while only 3% of children are exposed above the World Health Organization guideline for annual average NO_2 concentration (21 ppb), 3.9 million (95% uncertainty interval 1.8-5.1 million) new pediatric asthma cases are attributable to NO_2 pollution annually, 63% of which occur in urban centers. This burden accounts for 13% (6-17%) of global incidence. Regionally, the largest NO_2-attributable burdens per 100,000 children are estimated for Andean Latin America (330 cases/year), high-income North America (310), and high-income Asia Pacific (300). The largest national burdens are estimated for China (760,000), India (340,000), and the USA (230,000). Within cities, the largest burdens per 100,000 children are estimated for Lima, Peru (690), Shanghai, China (650), and Bogota, Colombia (580). Among 125 cities, NO_2 accounts for 6% (Orlu, Nigeria) to 48% (Shanghai, China) of pediatric asthma incidence. This contribution exceeds 20% in 92 cities.

Conclusions Our findings suggest that current levels of ambient NO_2 pollution are a significant risk factor of pediatric asthma incidence in both developed and developing countries. We also find that countries and cities with higher greenhouse gas emissions from fossil fuel combustion tend to have higher NO_2 exposures, providing further support that alignment of policy initiatives to mitigate air pollution and climate change can have multiple public health benefits.

* Study not funded by HEI.

Mapping Air Pollution at High Spatial Resolution: Comparing Mobile and Fixed Sensing Approaches

Sarah E. Chambliss and Joshua S. Apte

University of Texas at Austin, USA

Background Urban air pollution concentrations can vary sharply over short distances. High spatial resolution surfaces of urban air quality data are needed for a variety of purposes, including for exposure assessment in health studies. Two recent advances in measurement technology enable high resolution pollution mapping: on one hand, lower-cost sensors suitable for fixed deployment, and on the other, mobile sampling platforms suitable for extensive deployment on city streets.

Objectives This poster utilizes measurements from an unusually rich field experiment to compare and contrast the insights about fine-scale air pollution variability that can be gained from sampling using mobile and fixed sampling techniques. We investigate the following research questions: (i) can repeated mobile air sampling reproduce the same time-averaged spatial patterns that are observed by fixed-site observations, and (ii) can intermittent mobile sampling recover similar time-resolved pollutant concentration data as fixed-site monitoring?

Study Design For 100 days in summer 2017, a dense network of 100 custom-built black carbon (BC) sensors (ABCD, Aerosol Black Carbon Detector) was deployed in West Oakland, California. The ABCD sensor network provided BC information at 1 minute time resolution for each site. In parallel, we repeatedly sampled on-road air quality in the same neighborhood using two Google Street View cars custom-equipped with photoacoustic extinctiometer instrumentation for measuring BC at 1 Hz time resolution. The normal sampling program consisted of repeatedly driving every street in the ~5 km^2 domain, resulting in ~300 daytime hours of on-road BC measurements. Approximately 2% of the time-resolved in-motion BC measurements were obtained within 25 m of one of the 100 ABCD sampling sites. On average, the Google cars passed each ABCD monitor ~33 times over the course of the campaign, accumulating an average of 6 seconds of measurements while passing each monitor. In addition, we parked the Google cars within close proximity to 30 of the ABCD monitors for 10–15 minute sessions to create stationary "colocation" events.

Results The overall spatial patterns detected by both the mobile and fixed sampling approaches aligned well. We observed no meaningful systematic difference in BC concentration between the fixed ABCD sensors and the in-motion mobile measurements. The average on-road concentration collected within 25 m of each ABCD monitor was moderately well correlated with the daytime average concentration reported by the BC sensor, with an R^2 of 0.57. In contrast, the ~15-minute BC concentration resulting from a small number (median = 2-3) of parked colocations at each of 30 sites was only weakly correlated (R^2 ~ 0.2) between mobile and fixed sensors. This comparison suggests that highly dynamic fine-scale transport of conserved pollutants may cause substantial disagreement in real-time concentrations among even closely spaced monitors, although these concentration differences may substantially be reduced at longer time averaging scales. Results from this unique experiment can inform the design of future studies that wish to compare mobile and fixed observations.

Systematic Review on the Health Effects of Long-Term Exposure to Traffic-Related Air Pollution

Hanna Boogaard, Allison P. Patton, Rashid Shaikh, and the HEI Panel on the Health Effects of Long-Term Exposure to Traffic-Related Air Pollution

Health Effects Institute, Boston, MA, USA

Background The health effects of traffic-related air pollution continue to be of important public health interest, with highest exposures in urban settings and residences in proximity to busy roadways.

Following the Health Effects Institute's well-cited 2010 critical review, in early 2018, the HEI Board of Directors appointed an expert HEI panel again to review the traffic and health literature because much new science has been published. The Panel consists of 13 experts in epidemiology, exposure assessment and biostatistics, and is chaired by Francesco Forastiere (King's College London, UK) and Fred Lurmann (Sonoma Technology, Inc., Petaluma, California). In addition, HEI has obtained the services of a team at Swiss Tropical and Public Health Institute, Switzerland, to execute bibliographic searches, data extraction, and parts of the data synthesis, in close collaboration with HEI staff and Panel members.

The overall objective is to systematically evaluate the epidemiologic evidence for the associations of long-term exposure to traffic-related air pollution with selected adverse human health outcomes. Long-term exposure is defined as a duration of months to years. Results will be quantitatively combined, where appropriate, for potential use in future risk and health impact assessments of traffic-related air pollution.

Methods A systematic approach is being followed to search the literature, assess study quality, summarize results, and reach conclusions about the body of evidence. To this end, a review protocol has been developed. The literature in Pubmed will be searched with publication dates between January 1980 and June 2019.

The Panel has formulated criteria for selection of health outcomes in the review, including relevance for policy and public health. Selected health outcomes include all cause and cause-specific mortality, respiratory effects, cardiovascular effects, diabetes, childhood leukemia, birth and pregnancy outcomes, neurodevelopmental outcomes and neurocognitive outcomes.

In addition, the Panel has developed an exposure framework to guide the selection and evaluation of epidemiological studies on traffic-related air pollution, building on the 2010 critical review. The Panel will consider studies both within and outside the near-road environment because the impact of traffic-related air pollution extends beyond the near-road environment and contributes to ambient air pollution at neighborhood and urban scales. To define which studies in the latter category would qualify is particularly challenging because there needs to be evidence that the spatial contrast in exposure is mainly related to traffic sources. The Panel is also finalizing a risk of bias assessment to assess the quality of each included study.

Expected Results The review is currently well underway, with preliminary results expected in late 2019. The systematic review will undergo peer review in 2020 and publication is aimed for late 2020.

MAPLE: Mortality–Air Pollution Associations in Low Exposure Environments

Michael Brauer[1], Jeffrey R. Brook[2], Yen Li Chu[1], Tanya Christidis[3], Dan L. Crouse[4,5], Anders Erickson[1], Perry Hystad[6], Chi Li[7], Randall V. Martin[7,8], Jun Meng[7], Amanda J. Pappin[3], Lauren L. Pinault[3], Michael Tjepkema[3], Aaron van Donkelaar[7], Scott Weichenthal[9], and Richard T. Burnett[1]

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Background Fine particulate matter ($PM_{2.5}$) is associated with mortality, though uncertainty exists regarding the shape of the concentration response function at low levels. Since nearly the entire population of Canada lives in areas with ambient concentrations below 12 $\mu g/m^3$, it is ideal for the study of low $PM_{2.5}$ concentration mortality impacts.

Objectives To apply novel satellite-based estimates of $PM_{2.5}$ exposure to several large population-based cohorts, and characterize the shape of the relationship between $PM_{2.5}$ exposure with mortality.

Methods We developed annual satellite-based $PM_{2.5}$ exposure estimates at 1 km resolution across Canada for 1998-2016. Estimates were back-casted to 1981 using remote sensing, chemical transport models and historical ground monitoring data. Estimates for NO_2 and ozone were also developed for the same period. Exposures were estimated for ~8.5 million subjects in three Canadian Census Health and Environment Cohort (CanCHEC) cohorts, and 540,900 participants in the Canadian Community Health Survey (CCHS). Subjects were linked to annual tax records to establish residential histories, and to mortality until 2016. Sensitivity and sub-analyses evaluated (i) adjustment for individual, contextual, and geographic risk factors, (ii) adjustment for gaseous co-pollutants, (iii) exposure time window, and (iv) exposure spatial scale. Models were adjusted for a range of individual and contextual covariates. Estimates were evaluated across strata of age, sex and immigrant status and the shape of the association between $PM_{2.5}$ and mortality was examined with the Shape Constrained Health Impact Function (SCHIF).

Results The mean 3-year average $PM_{2.5}$ level was 7.4 $\mu g/m^3$ and 5.9 $\mu g/m^3$ over all person-years of follow-up in pooled CanCHEC and CCHS analysis, respectively. We estimated mortality hazard ratios (HR) of 1.04 (95% CI: 1.04-1.05) and 1.11 (95% CI 1.04-1.18) per 10 $\mu g/m^3$ change in outdoor $PM_{2.5}$ for the CanCHEC and CCHS analyses, respectively. In CanCHEC the SCHIF model predicted a sublinear concentration-mortality curve with little increase in risk below 5 $\mu g/m^3$, and monotonically increased HR predictions above this concentration. In CCHS we estimated a supra-linear relationship extending to concentrations below 2 $\mu g/m^3$. In both cohorts, HRs were larger for females, younger ages and non-immigrants and attenuated by inclusion of gaseous pollutants.

Conclusions In large population-based cohorts exposed to low levels of air pollution with extended follow-up, we found evidence of associations between $PM_{2.5}$ and mortality for concentrations as low as 5 $\mu g/m^3$ suggesting health benefits of reductions in air pollution where higher concentrations exist.

MAPLE: Mortality–Air Pollution Associations in Low Exposure Environments. Evaluating the Sensitivity of $PM_{2.5}$–Mortality Association to the Spatial and Temporal Scale of Exposure Assessment and the Inclusion of Immigrant Populations

Dan L. Crouse[1], Anders C. Erickson[2] (Co-presenter), Tanya Christidis[3], Lauren Pinault[3], Aaron van Donkelaar[4], Chi Li[4], Jun Meng[4], Randall V. Martin[4], Michael Tjepkema[3], Perry Hystad[5], Rick Burnett[6], Amanda Pappin[3], Michael Brauer[2], and Scott Weichenthal[7] (Co-presenter)

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Background Few studies have examined the sensitivity of $PM_{2.5}$-mortality associations to the spatial and temporal scale of exposure assessment or the inclusion of immigrant populations. There are important issues, particularly at low $PM_{2.5}$ concentrations where the shape of the concentration response curve remains uncertain.

Objectives This study had two objectives: (1) To evaluate the impact of the spatial and temporal scale of exposure assessment on associations between long-term exposure to $PM_{2.5}$ and mortality at low mass concentrations; (2) To evaluate how the healthy immigrant advantage impacts the $PM_{2.5}$-mortality association in Canada.

Methods We used the 2001 Canadian Census Health and Environment Cohort (2.4 million people with 10-year follow-up), and fit standard Cox proportional hazards models to examine the associations between ambient $PM_{2.5}$ exposure and non-accidental and cause-specific mortality. Satellite-based estimates of $PM_{2.5}$ exposures were assigned annually to residential locations. Three different temporal moving averages (1, 3, and 8-years) and three spatial scales (1-, 5-, and 10-km^2) of exposure assignment were compared. In addition, we examined different spatial scales based on age, employment status, urban/rural location, and adjustment for O_3, NO_2, or their combined oxidant capacity (O_x). Separately, models were examined by stratifying or adjusting for immigrant status (n=764,000), year immigrated (>30 years, 21-30 years, 11-20 years, ≤ 10 years), or excluding the most recent immigrants. Effect modification by age at immigration, country of birth, and neighbourhood-level ethnic concentration was also explored.

Results Longer moving averages and smaller spatial scales resulted in improved model fit and stronger associations between $PM_{2.5}$ and mortality. Respiratory and lung cancer mortality were more sensitive to the spatial scale of exposure assessment than cardiovascular outcomes. All of the best fitting models included adjustment for oxidant gases; this attenuated associations between $PM_{2.5}$ and cardiovascular mortality and strengthened associations between $PM_{2.5}$ and lung cancer. A healthy immigrant effect was observed for all mortality outcomes with a clear gradient of reduced mortality risk with shorter duration in Canada. Stratified analyses indicated that $PM_{2.5}$ was positively associated with mortality in the two most recent immigrant groups; including all immigrants in the overall analysis increased $PM_{2.5}$ hazard ratios compared to models excluding most recent immigrants.

Interpretations and Conclusions The unique characteristics and settlement patterns of immigrants influence $PM_{2.5}$-mortality relationships in Canada, as does the spatial and temporal pattern of exposure assessment. Nevertheless, our findings provide further support for an important relationship between long-term exposure to $PM_{2.5}$ and mortality at low mass concentrations.

Global Burden of Disease–Major Air Pollution Sources — a GLOBAL Approach

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Background Identification of contributing sources is an important next step in research to address air pollution as a global health risk factor. Source identification can be useful to initiate air quality management and to evaluate different policy options. Recent work conducted in China and India used updated national emissions inventories and chemical transport model simulations to identify current and future contributions to exposure and disease burden from multiple source sectors. There is interest in conducting similar analyses for all countries included in the Global Burden of Disease (GBD). This project will estimate source sector contributions to disease burden from ambient $PM_{2.5}$ at the national level for all ~195 countries and territories included in the GBD.

Approach We will use global anthropogenic emissions from the Community Emissions Data System (CEDS) inventory together with additional inventories for biomass burning (GFED4) and other sources (e.g. wind-blown and anthropogenic dust) to examine sensitivity to 7 main source sector categories (Residential Energy, Industry, Power Generation, Agriculture, Transport, Open Fires, Other). The GEOS-Chem 3-dimensional chemical transport model will be used to determine the global distribution of $PM_{2.5}$ mass concentration and major chemical species. Ambient annual average concentrations from sensitivity simulations with perturbed emissions in GEOS-Chem will then be used to isolate the fractional contributions of these source sectors to $PM_{2.5}$ mass. Additional sensitivity simulations with 15 sub-sector categories (including coal [residential, industry, power]; residential heating, cooking; residential biomass; shipping, air transport, on-road, non-road/rail; agricultural waste; open waste burning at residential dumpsites; anthropogenic dust) will provide additional policy-relevant information. With the output of each simulation, we will bias-correct and downscale the simulated concentrations to the 0.1° x 0.1° GBD satellite-based exposure estimates for direct comparability to the GBD. We will further downscale these estimates to 0.01° x 0.01° resolution to facilitate analysis at an urban area scale.

Our primary analysis of disease burden will combine the downscaled and bias-corrected $PM_{2.5}$ concentrations for each source sector to Integrated Exposure Response (IER) functions to estimate the population attributable fraction for each disease (ischemic heart disease, stroke, lung cancer, COPD, acute lower respiratory infections, type 2 diabetes) included in the GBD. We will also conduct a sensitivity analysis to estimate disease burden using the newly-developed Global Exposure Mortality Model (GEMM) which estimates non-accidental mortality and cause-specific mortality in functions derived only from studies of ambient air pollution health impacts.

Mortality, Morbidity, and Low-Level Air Pollution in a Pooled Cohort of 392,826 in Europe in the ELAPSE Project

Bert Brunekreef[1], Maciej Strak[1] (Presenter), Kathrin Wolf[2], Ulla A. Hvidtfeldt[3], Kees de Hoogh[4,5], Sophia Rodopoulou[6], Evi Samoli[6], Klea Katsouyanni[6,7], Marjan Tewis[1], Jie Chen[1], and Gerard Hoek[1], *on behalf of the ELAPSE Project Team*

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Background Cohort studies have consistently found associations between long-term exposure to outdoor air pollution and morbidity and mortality endpoints. However, uncertainty about the shape of the concentration response function exists at low concentrations. Within the multicentre *Effects of Low-Level Air Pollution: A Study in Europe* (ELAPSE), we investigate associations between long-term exposure to low concentrations, defined as below current European Union, US Environmental Protection Agency (EPA), and World Health Organization limit values or guidelines, of several air pollutants and morbidity and mortality endpoints.

Methods We pooled data from eight ESCAPE cohorts and the Danish Nurse Cohort, creating a total study population of up to 392,826 participants. We assessed residential exposure to air pollutants as annual 2010 mean concentrations of fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2), ozone (O_3; including cold and warm season estimates) and black carbon (BC) based on estimates derived from Europe-wide hybrid land use regression models at a 100 m spatial scale. After harmonizing individual and area-level variables between cohorts, we applied Cox proportional hazard models with increasing adjustment for confounders to investigate the association between long-term air pollution exposure and natural mortality, coronary events and lung cancer incidence. We also assessed the shape of the concentration-response relationship using different methods, including subset analysis, threshold analysis, and spline approaches.

Results The total study population contributed 7,518,024 person-year at risk (average follow-up 19 years). Average exposure to air pollution was 15.4 (SD 3.5) $\mu g/m^3$ for $PM_{2.5}$, 25.3 (8.2) $\mu g/m^3$ for NO_2, 1.6 (0.4) $\mu g/m^3$ for BC, and 86.2 (9.4) $\mu g/m^3$ for (warm season) O_3. A 5 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a hazard ratio (HR) of 1.13 (95% CI: 1.11, 1.16) in natural mortality and a hazard ratio of 1.13 (95% CI: 1.05, 1.23) in lung cancer incidence, whereas the association with coronary events was not statistically significant (HR = 1.02; 95% CI: 0.95, 1.10). Associations with natural mortality remained significant when exposure was restricted to $PM_{2.5}$ concentrations lower than the European annual mean limit value of 25 $\mu g/m^3$ (HR = 1.13; 95% CI: 1.11, 1.16) and also below the EPA air quality standard of 12 $\mu g/m^3$ (HR = 1.30; 95% CI: 1.14, 1.47). Significant positive associations were also found between natural mortality and NO_2, natural mortality and BC, coronary events and NO_2. Threshold analyses did not identify thresholds. Splines indicated no strong deviation from linearity.

Conclusions Long-term exposure to outdoor air pollution is associated with morbidity and mortality, even at low concentrations.

Mortality, Morbidity, and Low-Level Air Pollution in a Population of 35 Million in Europe — Analysis of Administrative Cohorts in the ELAPSE Project

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Background Recent evaluations by the World Health Organization and the Global Burden of Disease study have suggested that associations between long-term exposure to outdoor air pollution and morbidity and mortality may persist at very low concentrations. ELAPSE aims to investigate the adverse health effects of long-term exposure to low levels of ambient air pollution in Europe. In addition to a pooled cohort of 392,826 participants, we analysed data from very large administrative cohorts geographically spread across Europe.

Methods Seven large administrative cohorts (>35 million subjects) were included. Estimates of fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2), ozone (O_3) and black carbon (BC) derived from Europe-wide hybrid land use regression models were linked to subjects in each cohort based on their address-level geocodes. Variables in all cohorts were harmonised by implementing a common ELAPSE codebook. Data from the administrative cohorts were analysed locally using the exact same statistical scripts. We applied Cox proportional hazard models with successively more detailed control for individual- and area-level confounders to explore associations between exposure to pollutants and mortality as well as CVD and cancer incidence. Shapes of the concentration-response relationships were assessed using subset analysis, threshold analysis and spline approaches. Based on the main models, we further (1) applied multi-pollutant models to disentangle role of individual pollutants, (2) used other exposure metrics such as time-varying exposure and estimates from local exposure models, (3) evaluated the impact of measurement error on the magnitude of the effect estimates, (4) used health survey data to support indirect adjustment for potential confounders. The results from individual cohorts will be combined by meta-analysis.

Results Five cohorts have completed the main analyses for natural mortality (Belgian, Danish, Dutch, Rome and Swiss; Norwegian and English in progress). As an example, the annual average concentrations were 20, 12, 1.0 and 67 μg/m^3, respectively for NO_2, $PM_{2.5}$, BC and O_3, in the Danish cohort (2.8 million adults). Long-term exposure to $PM_{2.5}$, BC and NO_2 was significantly associated with increased risk of natural mortality in this cohort, the hazard ratio was 1.15 (1.12, 1.19) per 5 μg/m^3 $PM_{2.5}$. We anticipate to present results of meta-analyses across most or all of the administrative cohorts at the conference.

Conclusions No conclusions can yet be drawn as the joint analyses are still proceeding.

Estimating the Burden of Adverse Reproductive Health Outcomes Attributable to Air Pollution*

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Background Reduced birthweight and gestational age are well-established risk factors for increased neonatal and infant mortality and morbidity from preterm birth, cardio-respiratory and other diseases, and are responsible for a substantial global burden of disease. A growing body of evidence also links exposure to air pollution to both reduced birthweight and gestational age and their adverse effects. This suggests that mortality and lost years of healthy life in neonates and infants may be increased due a mediated effect of air pollution exposure acting on birthweight and gestational age.

Method To begin to quantify this potential burden, we conducted a systematic review of epidemiological evidence on the effect of prenatal air pollution effects on birthweight (continuous), low birthweight (dichotomous, <2500 grams of weight at birth) and preterm birth (dichotomous, <37 completed weeks of gestation) combining observed risk estimates into a meta-analysis using a random effects model. We searched PubMed using a combination of outcomes ("birth weight", "low birth weight", "preterm birth", "gestational age" or "small-for-gestational age") and exposure ("particulate air pollution", $PM_{2.5}$, and PM_{10}"). Studies were included if they considered long-term exposure, i.e., the entire pregnancy or trimesters.

Findings We identified 35 studies assessing the effect of $PM_{2.5}$ exposure on birthweight, 31 studies assessing its effect on low birthweight, 34 studies assessing the effect on preterm birth and 21 studies assessing the effect on small for gestational age. The meta-regression revealed significant odds ratios for all three outcomes: birthweight decreased by 15 g (95%CI: 9-20g) per 10 $\mu g/m^3$ increase in $PM_{2.5}$. We found an odds ratio of 1.05 (95%CI: 1.02-1.08) for low birthweight and 1.08 (95%CI: 1.03-1.13) for preterm birth. We observed substantial heterogeneity across-studies with I^2 of 96.3% for birthweight studies, 82% for low birth weight estimates and 98.7% for pre-term birth studies.

Conclusion Although quantitative estimates displayed substantial heterogeneity, there is substantial evidence that air pollution affects reproductive health outcomes, and leads to lower birthweight, small for gestational age, and pre-term birth. However, it is unclear whether the excess relative risk per unit exposure changes over the global range of exposure to $PM_{2.5}$ or remains constant. Fitting an integrated exposure-response function covering the global exposure range of ambient and household $PM_{2.5}$ as well as the non-linear (spline) meta-regression (MR-BRT) will provide further insight and allow more accurate burden estimation.

* Study not funded by HEI.

The Global Burden of Diabetes Mellitus Type 2 Due to Indoor and Outdoor Sources of Particulate Matter Pollution*

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Background Diabetes is a growing public health concern across the globe. Recent epidemiologic evidence suggests a causal link between exposure to $PM_{2.5}$ and diabetes mellitus type 2 incidence and mortality. We estimated the global burden of type 2 diabetes attributable to ambient $PM_{2.5}$ and household air pollution from 1990 to 2017 within the comparative risk assessment framework of the Global Burden of Disease project.

Methods We systematically assembled the epidemiologic evidence connecting four sources of particulate matter pollution — ambient air pollution, household air pollution, environmental tobacco smoke, and cigarette smoking — to type 2 diabetes in order to develop an integrated exposure response function (IER). The IER included 14 cohort studies of ambient air pollution in five countries and one cohort study of household air pollution. We applied this function to modeled ambient and household $PM_{2.5}$ exposures to estimate attributable diabetes burden for 195 countries and territories and 355 subnational locations. We decomposed trends in attributable burden to account for changes in population size, age distribution, air pollution exposure, and other factors.

Results In 2017, over a quarter of type 2 diabetes burden, 276 thousand deaths (95% Uncertainty Interval (U.I.) 191-330) and 15.2 million disability-adjusted life years (DALYs) (95% U.I. 10.0-19.9), were attributable to particulate matter pollution. The attributable burden from ambient $PM_{2.5}$ in 2017 — 184 thousand deaths (95% U.I. 123-227) and 10.5 million DALYs (95% U.I. 6.7-13.9) — was roughly double that from household air pollution — 92 thousand deaths (95% U.I. 63-113) and 4.8 million DALYs (95% U.I. 3.1-6.2) — whereas the contributions were nearly equal in 1990. We saw the highest burden attributable to ambient $PM_{2.5}$ in North Africa and the Middle East; Latin America and the Caribbean; and Central Europe, Eastern Europe, and Central Asia. In contrast, the highest burden attributable to household air pollution was in Sub-Saharan Africa; Southeast Asia, East Asia, and Oceania; and South Asia. The number of particulate matter attributable DALYs more than doubled since 1990. Most of this increase is attributable to population growth and population aging, while on the global scale, increases in exposure to ambient $PM_{2.5}$ have roughly offset decreases in exposure to household air pollution.

Conclusions Ambient and household $PM_{2.5}$ are important risk factors for type 2 diabetes and should be considered in the development of environmental and public health policy.

* Study not funded by HEI.

Susceptibility to Multiple Air Pollutants in Cardiovascular Disease

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Background Cardiovascular disease (CVD), the leading cause of death in the U.S., has been linked to chronic and acute air pollution exposures. Research has identified stronger effects of air pollution in lower-socioeconomic position (SEP) communities, where exposures are also often higher. While specific factors underlying this socioeconomic susceptibility remain unknown, chronic psychosocial stress related to social adversity is hypothesized to be a key component.

Methods In this study, we use data on 1.1 million New York City (NYC) CVD emergency department (ED) visits, multiple air pollutants [fine particles ($PM_{2.5}$), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), summertime ozone (O_3)], and community-level social stressors (e.g., SEP, violence, race-based residential segregation) to examine susceptibility to multiple air pollutants in CVD. First, we used ecologic cross-sectional models to examine spatial relationships among pollutants, stressors, and age-adjusted community CVD incidence rates. Second, we examined associations between spatio-temporal pollution exposures and CVD events using case-crossover models, across lag days 0-6. Finally, we are testing modification in these associations by community-level social stressors.

Results In ecologic models, higher CVD rates were associated with NO_2, $PM_{2.5}$, and O_3 only in areas of higher violence and physical disorder; these associations were consistent for overall CVD and all sub-diagnoses. In case-crossover models examining associations between spatio-temporal air pollution and CVD, we found significant same-day associations between NO_2 and risk of any CVD event, ischemic heart disease, and heart failure; this association remained significant with any form of co-pollutant adjustment. Significant associations for $PM_{2.5}$ and SO_2 on all CVD, heart failure (for $PM_{2.5}$), and ischemic heart disease (for SO_2) were somewhat less robust to co-pollutant adjustment. We have implemented case-crossover models exploring effect modification by SEP, violence, and residential segregation. However, community stressor exposures are profoundly confounded by individual race/ethnicity in NYC; separating the simultaneous impacts of individual- and community-level modification may not be plausible in a case-crossover design with air pollution as the main exposure of interest. Because case-crossover inherently adjusts for age, the method obscures the critical fact that the median age of non-Hispanic blacks in our CVD case-only dataset is only 60 years old, versus 74 years for non-Hispanic whites. To more fully understand the role of social stressors in shaping racial differences in pollution response — more meaningfully capturing the vast differences in pollution susceptibility and CVD risk by age and race — we are now examining age at CVD event using methods which leverage longer (annual) time-scales, (e.g., survival analysis, Cox proportional hazards).

Discussion Preliminary Cox proportional hazard results suggest a dose-response relationship between chronic (annual-average) NO_2 exposure and age at CVD, with increasing levels of social stressors (i.e., segregation, violent crime, socioeconomic deprivation).

Contribution of Mobile Sources to Secondary Formation of Carbonyl Compounds*

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Background Carbonyl compounds are emitted directly into the atmosphere from anthropogenic and biogenic sources, as well as being formed secondarily by degradation of volatile organic compound (VOC) precursors. Previous research suggests that photochemical production accounts for most of ambient concentrations of carbonyl compounds such as formaldehyde and acetaldehyde, and a significant portion of ambient acrolein. All three of these pollutants are considered hazardous air pollutants (HAPs).

The 2014 National Air Toxics Assessment (NATA) estimated that, nationwide, about 75% of ambient formaldehyde and acetaldehyde, and about 18% of acrolein, is formed secondarily. Mobile sources may be significant contributors to these secondary concentrations. They are key sources of alkene and alkane emissions, which react to form formaldehyde and acetaldehyde, and 1,3-butadiene, which reacts to form acrolein.

A number of source apportionment techniques, including use of reactive tracers, have been used in conjunction with photochemical models to estimate source sector contributions to secondary concentrations of criteria pollutants, such as ozone and particulate matter. Development of such techniques for hazardous air pollutants has been very limited, however. Most recently, Luecken et. al. (Env. Sci. Technol. 2018, 52: 4668-4675) used direct decoupled method (DDM) in the Community Multiscale Air Quality (CMAQ) model to quantify the relative role of emission sectors and individual classes of VOCs on ambient formaldehyde. While the DDM provides sensitivities to changes in emissions, it does not provide source contributions for the predicted concentrations.

Methods We conducted several CMAQ runs, where emissions are set to zero for different mobile source sectors, to estimate their potential contribution to secondary formation of formaldehyde, acetaldehyde, and acrolein. Sectors analyzed include highway vehicles, commercial marine vessels, airports, locomotives, and other nonroad equipment. The analysis was conducted for calendar year 2014, based on input data and methods developed for 2014 NATA.

Results Our results suggest mobile sources are major contributors to secondarily formed carbonyl compounds in the atmosphere. The magnitude of the contribution varies significantly across the U.S. However, because the chemistry of aldehydes is very complex, and involves complex interactions among many compounds, zeroing out emissions from an individual sector can offer only a rough approximation of how that sector might contribute to overall secondary concentrations. Furthermore, since secondarily formed pollutants can result from many different reaction pathways, they are affected by uncertainties associated with specific pathways, as well as uncertainties in the emissions of their precursors. Despite these limitations, the modeling results provide useful insights into the role of mobile sources in secondary formation of carbonyl compounds.

Conclusions This study suggests that precursor compounds emitted by mobile sources are substantial contributors to secondary concentrations of carbonyl compounds in the atmosphere. Thus, reduction of precursor emissions from mobile sources should significantly reduce ambient levels.

Note: This abstract does not necessarily reflect U.S. EPA policy.

* Study not funded by HEI.

Traffic-Related Air Pollution and Birth Weight: The Roles of Noise, Placental Function, Green Space, Physical Activity, and Socioeconomic Status (FRONTIER)

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Background A substantial body of evidence has associated air pollution to impaired fetal growth; however, there are still substantial limitations in terms of applied exposure assessment methods, such as disentangling the role of co-exposures such as noise, and evaluating the modifiers, mediators, and mitigators of this association. FRONTIER aims to provide a robust and comprehensive evaluation of the impact of maternal exposure to traffic-related air pollution on fetal growth. Towards this aim, it will (i) disentangle the effects of noise; (ii) identify the relevant window(s) of exposure; (iii) evaluate its modification by socioeconomic status, stress, and physical activity; (iv) elucidate the role of placental function as an underlying mechanism; and (v) explore the potential of green spaces to mitigate it.

Methods FRONTIER will establish a new pregnancy cohort of 1,000 women in Barcelona, Spain. Fetal growth will be characterized by anthropometric measures at birth together with ultrasound-based trajectories of fetal development. Placental function will be evaluated using state-of-the-art Doppler ultrasound indicators. Hair cortisol levels will be used as an indicator of maternal stress during pregnancy. Time-activity patterns will be objectively characterized using a combination of smartphones and personal physical activity monitors. We will develop and validate an innovative exposure assessment framework integrating data on time-activity patterns with a hybrid modeling framework combining dispersion and land use regression models and campaigns of personal and home-outdoor air pollution monitoring to estimate maternal exposure level as well as inhaled dose of nitrogen dioxide (NO_2), fine particulate matter ($PM_{2.5}$), $PM_{2.5}$ light absorption, and $PM_{2.5}$ copper (Cu), iron (Fe), and zinc (Zn) contents (markers of non-tailpipe emissions) at the main microenvironments for pregnant women (home, workplace, and commuting routes). We will assess maternal exposure to noise by integrating measurements at participants' home-outdoor using noise monitors together with modeled microenvironmental levels of noise and data on noise sensitivity, annoyance, and protections against noise. We will apply detailed information on different characteristics of each tree canopy in our study region together with a high-resolution remote-sensing map of greenness to separately characterize the canopy and greenness surrounding maternal residential address. We will develop single- and multi-pollutant models to evaluate the impact of air pollution exposure and inhaled dose on fetal growth and the mediatory role of placental function.

Results & Conclusions FRONTIER is now in the recruitment phase. It will generate a vigorous evidence base for implementing finely-targeted regulations to tackle effects of air pollution on fetal growth.

Assessing the Short-Term Effect of $PM_{2.5}$ on Cardiovascular Hospitalizations in the Medicaid Population: A Case-Crossover Study

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Background Many studies have demonstrated an association between short-term exposure to fine particulate matter ($PM_{2.5}$) and cardiovascular disease (CVD). This association has been shown to vary by socioeconomic status (SES), in space (e.g., by region) and time (e.g., by season). We conducted a national epidemiologic analysis using US-wide Medicaid data to estimate the association between short-term $PM_{2.5}$ and CVD hospitalizations for the years 2010 and 2011 among low-income and disabled Americans.

Methods We applied a time-stratified case-crossover design to estimate the association between short-term $PM_{2.5}$ (measured as the average of $PM_{2.5}$ on the day of the event and the preceding day), and the rate of total and cause-specific CVD hospitalizations. We also examined this association for days with $PM_{2.5}$ levels lower than 25 $\mu g/m^3$, the current guideline for daily $PM_{2.5}$ concentrations by the World Health Organization (WHO). We used the Medicaid eligibility criteria to identify the subpopulation with a disability, and report the association between $PM_{2.5}$ and CVD hospitalization for this subpopulation. Finally, we compared the association between $PM_{2.5}$ and CVD in the Medicaid population with the association in the Medicare population for the same years.

Results We observed an increase of 1.4% (95% CI: 0.8, 2.0%) in total CVD hospitalizations, 1.1% (95% CI: -0.8, 3.1%) in acute myocardial infarctions, 2.6% (95% CI: -3.1, 8.7%) in ischemic heart diseases, 2.6% (95% CI: 1.1, 4.1%) in congestive heart failure, and 1.3% (95% CI: -0.5, 3.2%) in ischemic strokes for each increase of 10 $\mu g/m^3$ in $PM_{2.5}$. The estimated association between $PM_{2.5}$ and CVD hospitalizations among Medicaid enrollees with a disability was 1.3% (95% CI: 0.4, 2.3%). The estimated association between $PM_{2.5}$ and CVD hospitalizations, only considering days where $PM_{2.5}$ levels were less than 25 $\mu g/m^3$ was 1.7% (95% CI: 1.0, 2.4%). The estimated association between $PM_{2.5}$ and CVD hospitalizations for the Medicaid population over 65 years of age was 2.2% (95% CI: 1.1, 3.3%) The corresponding estimated association in the Medicare population was 1.0% (95% CI: 0.7, 1.3%).These associations were robust in sensitivity analyses.

Conclusions Our analyses showed an increased rate of CVD hospitalizations associated with short-term $PM_{2.5}$ exposure in the Medicaid population. This association in the Medicaid population older than 65 years old was greater than the association in the Medicare population, albeit the confidence intervals of the two estimates were overlapping. This finding indicates increased vulnerability among the low-income and disabled elderly. In addition, we found that this association was still statistically significant at $PM_{2.5}$ levels below the WHO guidelines for 24 hour $PM_{2.5}$ concentrations.

An Ensemble-Based Model of $PM_{2.5}$ Concentration Across the Contiguous United States with High Spatiotemporal Resolution

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Background Various approaches have been proposed to model $PM_{2.5}$ in the last decade, with satellite-derived aerosol optical depth, land-use variables, chemical transport model predictions, and several meteorological variables as major predictor variables. The training methods have been evolving from simple linear regressions to more complex machine learning algorithms.

Methods Our study used an ensemble model that integrated multiple machine learning algorithms and predictor variables to estimate daily $PM_{2.5}$ at a resolution of 1 km × 1 km across the contiguous United States. We used a generalized additive model that accounted for geographic difference to combine $PM_{2.5}$ estimates from neural network, random forest, and gradient boosting models. The three machine learning algorithms were based on multiple predictor variables, including satellite data, meteorological variables, land-use variables, elevation, chemical transport model predictions, and several reanalysis datasets.

Results The model training results from 2000 to 2015 indicate good model performance with a 10-fold cross-validated R^2 of 0.86 for daily $PM_{2.5}$ predictions. For annual $PM_{2.5}$ estimates, the cross-validated R^2 was 0.89. Our model demonstrated good performance up to 60 μg/m^3. Using the trained $PM_{2.5}$ model and predictor variables, we predicted daily $PM_{2.5}$ from 2000 to 2015 at every 1 km × 1 km grid cell in the contiguous United States. We also used localized land-use variables within 1 km × 1 km grids to downscale $PM_{2.5}$ predictions to 100 m × 100 m grid cells. To characterize uncertainty, we used meteorological variables, land-use variables, and elevation to model the monthly standard deviation of the difference between daily monitored and predicted $PM_{2.5}$ for every 1 km × 1 km grid cell.

Conclusions Our $PM_{2.5}$ prediction dataset, including the downscaled and uncertainty predictions, will allow epidemiologists to accurately estimate the adverse health effects of $PM_{2.5}$. Based on the model performance of individual learners, we also conclude that model performance of $PM_{2.5}$ models is based on context, and the best training algorithm to fit $PM_{2.5}$ globally does not exist.

Environmental Factors Affecting Stress in Children: Interrelationships Between Traffic-Related Noise, Air Pollution, and the Built Environment

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Background There is a growing body of research linking several aspects of the physical environment to children's mental and physical health. Stressors such as traffic-related noise have been associated with decrements in children's mental health, and air pollution has been shown to modify neurocognitive development and behavior. Exposure to artificial light in the built environment can lead to circadian disruption and stress, while studies have shown that living near greenspace reduces stress and has a wide range of other health benefits for children and adolescents. Chronic stress in childhood may contribute to direct and indirect health effects such as asthma, cardiovascular disease, and obesity.

Methods When aged 13-14 and 15-16 years, participants in the longitudinal Southern California Children's Health Study (CHS) responded to four questions pertaining to stress. An individual's combined responses to these questions resulted score from 0 to 16 on the Perceived Stress Scale (PSS-4). Residential exposures were derived for traffic-related noise from the Traffic Noise Model, freeway and non-freeway NO_x from the CALINE line dispersion model, greenspace from Moderate Resolution Imaging Spectroradiometer (MODIS) satellite observations of enhanced vegetation index (EVI), and artificial light at night (ALAN) from Visible Infrared Imaging Radiometer Suite (VIIRS) satellite observations in the day-night band. We assessed the marginal and joint associations with these exposures using mixed effects models with adjustments for subject-specific characteristics and confounders.

Results Overall, PSS-4 is significantly higher ($p<0.001$) for girls [mean (s.d.) = 5.8 (3.4)] than boys [mean (s.d.) = 4.9 (3.2)]. From ages 13-14 to 15-16, stress significantly increased in girls from 5.6 (3.3) to 6.0 (3.4), but for boys stress did not change. Marginally, all of the environmental factors were significantly associated with stress after adjustment for sex, height, weight and trouble initiating sleep. Greenspace showed a -0.33 (95% CI: -0.49, -0.18) decrease in stress per IQR (0.06) increase in EVI. All other environmental factors have positive associations with stress, including a 0.22 (95% CI: 0.05, 0.40) increase in stress per IQR (22.6 $nW/cm^2/sr$) increase in ALAN. Jointly, we found similar patterns with a -0.23 (95% CI: -0.43, -0.03) decrease in PSS-4 per IQR increase in EVI, together with a 0.15 (95% CI: 0.03, 0.27) increase in PSS-4 per IQR increase in freeway NO_x. In this model traffic-related noise was only statistically significant with PSS-4 at the community level, with some Southern California communities showing stronger associations than others. Furthermore, due to the strong negative correlation between EVI and ALAN ($r = -0.67$) it was difficult to tease out their joint association with stress.

Conclusions The physical environment consists of a complex mixture of factors that can both positively and negatively affect health. We find evidence that residential greenspace may mitigate the impact of traffic-related air pollution and noise on perceived stress in a longitudinal cohort of children in Southern California.

Effects of Policy-Driven Hypothetical Nitrogen Dioxide and Particulate Matter Interventions on Asthma Incidence in Southern Californian Children*

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Background Numerous studies have linked incident childhood asthma with air pollution, including traffic-related air pollutants such as nitrogen dioxide (NO_2) and particulate matter ≤2.5 μm ($PM_{2.5}$). To examine the benefits of different air pollution reduction policies we estimated the effect on asthma incidence of hypothetical air pollution interventions in a population of Southern California children using g-computation, a method based in the counterfactual framework. This method attempts to answer such policy-relevant questions as "What would have been the asthma incidence rate if participant exposure to NO_2 had been no higher than 30 ppb NO_2?"

Methods We used data from three cohorts in the Southern California Children's Health Study (recruited in 1993, 1996, and 2002) with prospectively identified incident asthma cases and continuously monitored NO_2 and $PM_{2.5}$ at community central sites. Multilevel Poisson regression modeled the relation between asthma incidence and baseline-year community-level annual mean pollution concentration, adjusting for potential confounders. For NO_2 and $PM_{2.5}$ individually, we estimated the effect on asthma incidence of three sets of interventions: (1) maintain air pollutant concentrations at their community-specific 1993 levels, which were the highest levels measured during the study period before observed air pollution declines in the 1990s-2000s; (2) a percent reduction in pollutant concentration (10%, 20%, and 30%); and (3) a dynamic intervention using a threshold whereby communities with concentrations above the threshold were set to the threshold value, mimicking adherence to a standard (30, 20, and 10 ppb for NO_2; 15, 12, and 10 $\mu g/m^3$ for $PM_{2.5}$). Selection of threshold values was informed by US EPA and WHO standards. We contrasted hypothetical interventions with observed exposures.

Results The three cohorts included 4,140 children with no history of asthma at baseline and mean follow-up of 5.9 years (mean baseline age: 9.5 years; 53% female; 59% White; 42% Hispanic). Remaining at 1993 NO_2 levels was estimated to increase asthma incidence by 19.3% (95% CI: 9-32%), whereas reductions of 10%, 20%, and 30% were estimated to reduce asthma incidence by 10.5%, 19.6%, and 27.6% respectively. Implementing hypothetical standards of 30, 20, and 10 ppb were estimated to reduce asthma incidence by 7.3%, 19.6%, and 39.2%, respectively. For $PM_{2.5}$, there was an increase of 9.8% (95% CI:0.9-20%) estimated for remaining at 1993 levels and reductions of 4.5%, 8.8%, and 12.8% for implementing reductions of 10%, 20%, and 30%, respectively. Implementing hypothetical standards of 15, 12, and 10 $\mu g/m^3$ $PM_{2.5}$ were estimated to reduce asthma incidence by 11.0%, 14.9%, and 17.6%, respectively.

Conclusions Our study demonstrated a large potential public health benefit of air pollution reduction — both realized and hypothetical improvements — in reduced childhood asthma incidence. These findings were observed in communities at NO_2 concentrations well below the current US EPA annual standard of 53 ppb, indicating potential public health benefits in reevaluating air quality standards.

* Study not funded by HEI.

Dietary DHA Mitigates Ozone Induced Pulmonary Inflammation and Reductions in Specialized Pro-resolving Mediators

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Background Ozone (O_3), a criteria air pollutant, causes significant pulmonary morbidity and mortality. Recent epidemiological studies suggest that diet may influence the health effects associated with air pollution, including O_3. Our previous studies indicate O_3 exposure decreases pulmonary docosahexaenoic acid (DHA)-derived specialized pro-resolving mediators (SPMs). SPMs are lipid mediators produced at the site of injury that support resolution of the immune response and return of tissue homeostasis. Therefore, we hypothesize pulmonary inflammation noted after O_3 exposure is due to a decrease in pulmonary SPM production that can be mitigated with dietary DHA supplementation.

Methods Five-week-old C57Bl/6J (WT) male mice were fed a normal diet (ND) or a DHA supplemented diet (ND+2% DHA). After 6 weeks, mice were exposed to filtered air (FA) or 1 ppm O_3 for 3h and necropsied 24h post-exposure. Bronchoalveolar lavage fluid (BAL) was assessed for cytokine production, cell counts/differentials, and protein. Pulmonary lipid mediators were quantified using LC-MS/MS.

Results In O_3-exposed groups, there were decreases in the pulmonary levels of DHA-derived SPM precursors 17-HDHA and 14-HDHA and the SPM Protectin DX. However, DHA supplementation prior to O_3 mitigated these reductions in SPM precursors/SPMs. In the ND group, O_3 exposure significantly increased BAL macrophages and neutrophils that was also reduced with dietary DHA supplementation. Compared to O_3+ND, DHA reduced CCL2, CCL3, IL-6, and IL-1β cyto/chemokine expression in lung tissue.

Conclusions Supplementing a diet with DHA increased pulmonary SPMs and their precursors resulted in a decrease in O_3-induced cellular inflammation and expression of select cytokines and chemokines. These data support the novel idea that a DHA supplemented diet may mitigate O_3-induced pulmonary responses.

Air Pollution, Autism Spectrum Disorders, and Brain Imaging Among CHildren in Europe — the APACHE Project

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Background We aim to investigate (1) the association between prenatal air pollution exposure at different time windows and the development of autism spectrum disorders (ASD) and (2) the association between prenatal and postnatal air pollution exposure at different time windows and brain structural and functional changes in children.

Methods We use data from two epidemiological studies: (1) a population-based case-cohort study of ASD in Catalunya (Spain), where children diagnosed with ASD identified through the Catalan mental health network are linked to the Catalan birth registry and (2) the Generation R population-based birth cohort study (the Netherlands), with existing longitudinal data on brain imaging in children at 6-10 years and at 8-12 years. For both study regions we compile existing land use regression models for a large number of pollutants. For the study of Catalunya, we also combine land use variables and satellite data remote sensing of aerosol optical depth to develop new $PM_{2.5}$ and PM_{10} models. We estimate air pollution levels at participants' home addresses at different time-windows during pregnancy (entire pregnancy, monthly, and weekly) and childhood (entire childhood, yearly, and monthly). We apply methods for measurement error and multi-pollutant models. We assess the association between air pollution exposure at different time windows during pregnancy and the development of ASD. We also assess the association between air pollution exposure at different time windows during pregnancy and childhood and structural and functional brain changes at 6-10 years old and at 8-12 years old.

Results and Conclusions We are setting up the case-cohort study on ASD and developing new air pollution models for Catalunya. There are no results from that study yet. Regarding the imaging study, we found that prenatal $PM_{2.5}$ exposure was associated with a thinner cortex in several brain regions in 6-10 years old children and these alterations partially mediated the association between prenatal $PM_{2.5}$ exposure and impaired child inhibitory control. Regarding white matter microstructure, in single pollutant analysis, higher prenatal and postnatal exposure to several air pollutants was associated with a decrease in fractional anisotropy and an increase in mean diffusivity in 9-12 years old children, indicating a developmental delay in white matter miscrostructure. In the multi-pollutant analyses and mutually adjusting between prenatal and postnatal exposures, higher prenatal levels of silicon content in $PM_{2.5}$ and higher postnatal levels of zinc content in $PM_{2.5}$ were associated with an increase in mean diffusivity (0.07 [95%CI 0.01; 0.13] and 0.03 [95%CI 0.01; 0.06] for each 10 ng/m^3 increase of airborne silicon and zinc, respectively). Measurement error correction methods were applied.

Quantifying Societal Health Benefits of Transportation Emission Reductions in the United States and Canada

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Background One measure of the efficacy of an air pollution control option is the Marginal Benefit (MB) associated with the option, or the monetized societal benefits of reducing one metric tonne of emissions through its implementation. We use adjoint modeling, a recent approach for quantifying source-receptor relationships, and integrate air quality modeling, epidemiology, and economic valuation to estimate location-specific MBs.

Objectives We aim to (a) create a database of location-specific MBs for transportation and other select sectors in Canada and the U.S., (b) quantify the uncertainty in estimated MBs, (c) examine the sensitivity to various assumptions made in estimation of MBs, (d) develop an associated database of co-benefits from combustion-based CO_2 reductions, and (e) present per-vehicle transportation MBs for various vehicle types and vintages.

Methods We use the adjoint version of the U.S. EPA's Community Multiscale Air Quality (CMAQ) model to estimate MBs at 12 km resolution for various locations and times in Canada and the contiguous U.S. The adjoint approach allows for the estimation of influences from individual sources on nationwide health outcomes. Our analysis will primarily focus on health impacts associated with chronic exposure to $PM_{2.5}$ and O_3, as well as exposure to NO_2 in Canada. We will examine the robustness of MB estimates with respect to various assumptions such as the choice of health and valuation metrics, simulated episodes, model resolution, shape of concentration-response functions, and the emission inventory level. We will combine estimated MBs with sector-specific CO_2 emission profiles to develop a related database of co-benefits.

Past Results Our past results suggest that there is great spatial (and temporal) variability in MBs for various emitted species, particularly at higher spatial resolutions. As expected, this spatial variability closely follows that of population distribution for primary pollutants (e.g., primary $PM_{2.5}$) but is more nuanced for precursor emissions (i.e., SO_2, NO_x, and NH_3). Our past findings also indicate that co-benefits of CO_2 reductions from transportation and electricity generation can be significantly larger than the social cost of carbon for many source locations in the U.S. and Canada.

Statistical Methods for Estimating the Association Between Multipollutant Mixtures and Health Outcomes: A Simulation Study*

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Background Populations are continuously exposed to complex mixtures of environmental pollutants. Evaluation of health effects associated with environmental mixtures is complicated by a high number of pollutants, multicollinearity, and possible non-linear or interaction effects. Numerous statistical methods have been proposed to address multipollutant mixtures, but a formal comparison among methods is lacking. We conducted a simulation study to evaluate five contemporary methods for estimating health effects associated with multipollutant mixtures.

Methods We evaluated the performance of five recently proposed statistical methods for multipollutant mixtures: nonparametric Bayes shrinkage with main effects only (NPBr), nonparametric Bayes shrinkage with pairwise interactions (NPB), unsupervised Bayesian profile regression (UPR), supervised Bayesian profile regression (SPR), and Bayesian kernel machine regression (BKMR). We also included traditional linear models with main effects only (LM) and pairwise interactions (LM-int) for comparison. We used existing exposure data on seven ambient air pollutants and agricultural pesticides from Central Valley, CA to simulate health responses for three distinct exposure-response scenarios. We evaluated each methods' estimation of the exposure-response function through root mean squared error (RMSE) and coverage, and identification of important mixture components and interactions through true and false selection rates. Finally, we illustrated each method by analyzing forced expiratory volume (FEV1) in a children's asthma cohort using the same exposures from the simulation.

Results BKMR had the highest true selection rates in all three scenarios and was a top-performing method in terms of RMSE and coverage for the exposure-response function. NPB estimated the exposure-response function well and had high true selection rates in most cases. NPB was the only method to accurately identify interactions between mixture components. LM-int had coverage closest to the nominal level (0.95) in all three scenarios but was consistently poor at identifying important mixture components. UPR and SPR performed poorly across all scenarios when using existing exposure data but performed better on simulated data with clearly defined exposure profiles and a strong signal. In the children's cohort analysis, most methods identified a negative association between NO_2 and FEV1, though exposure-response estimation differed across method classes.

Conclusions This study shows that sophisticated methods such as BKMR and NPB can estimate complex exposure-response functions and identify important mixture components in the presence of highly correlated data. Traditional linear regression techniques may be appropriate for estimating a linear exposure-response function; however, these methods are generally unable to identify important mixture components within highly correlated data. Bayesian profile regression may be more suitable to data sets with clear exposure profiles and a strong signal. The formal evaluation of these methods fills a critical gap in methods evaluation identified by both the National Institute of Environmental Health Sciences and the Environmental Protection Agency. Our results provide useful information for researchers to determine which methods are most appropriate in practice.

* Study not funded by HEI.

Chemical and Physical Characterization of Non-Tailpipe and Tailpipe Emissions at 100 Locations near Major Roads in the Greater Boston Area

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Background The purpose of the proposed research is to develop sampling and statistical analysis methods to improve assessment of exposure to traffic particles and pollutants for health effects studies, characterize direct (tailpipe and non-tailpipe) and indirect contributions, and identify parameters which influence them.

Methods Using a mobile sampling platform equipped with coarse and fine particulate matter (PM) concentrators we are collecting ambient ultrafine, fine minus ultrafine (accumulation mode), and coarse particle samples near roads in the Greater Boston. In addition, we are collecting $PM_{2.5}$ and PM_{10} samples of surface road dust aerosolized *in situ*, using our Road Dust Aerosolization sampler. We are collecting these road dust (RD) and ambient PM samples at three distance ranges (including background) from each of the 100 major (A1, A2, and busy A3) road sites included in this study.

Results We analyzed particle samples collected at 26 sites (nine A1, five A2, and twelve A3-type roads) using XRF and reflectometry and conducted preliminary data analysis.

For both coarse and fine ambient PM and RD samples, we observed significant differences in composition between those collected at the roadside, local background, and intermediate distances. Ambient PM mass concentrations were generally highest at the roadside and lowest at the local background site. Comparison of ambient fine and coarse PM sample composition between near major roads and background locations indicated that vehicular sources were significant contributors to the concentrations of certain elements (e.g., Cu, Ti, Ba, Zn, Zr, Mn and Fe). Concentrations of these elements were higher at the roadside as compared to those observed at locations 50 to 200 m from the roadway.

For RD samples, we normalized elemental composition of coarse and fine fractions relative to Al. While Si is the most abundant element in the earth crust and has been used as a soil tracer, we selected Al because ceramic brake pads, which have been used extensively in recent years, contain Si. As with ambient PM, comparison of specific elements in fine and coarse RD samples between near major roads and background locations indicates that vehicular sources are significant contributors. Also, concentrations of many elements were elevated at the roadside as compared to those observed at locations 50 to 200 m from the roadway.

While distance from road had a significant impact on composition of both RD and ambient PM, road type did not. There was little difference in composition of RD or ambient PM among A1, A2, and A3 roads.

Conclusions Composition of ambient and road dust samples near major roads compared to background indicated that vehicular sources are significant contributors for certain elements. Differences in composition between ambient PM and RD collected at roadside, intermediate, and local background sites were significant despite the small number of observations, and did not depend on road type.

Goods Movement Actions Improved Air Quality and Health Outcomes Among California Medicaid Enrollees

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Background This project aims to examine the impact of the "Emissions Reduction Plan for Ports and Goods Movement" of the California Air Resources Board (CARB) in 2006 on reductions in ambient air pollution and subsequent improvements in health outcomes among Medicaid fee-for-service (FFS) beneficiaries in 10 counties in California. Specifically, we examined whether air pollution reduction actions resulted in decreases in emergency department (ED) visits and hospitalizations among enrollees with chronic conditions.

Methods The study areas were grouped into goods movement corridors (GMCs) as locations within 500 m of truck-permitted freeways and ports; non-goods movement corridors (NGMCs) as locations within 500 m of truck-prohibited freeways or 300 m of a connecting roadway, and controls (CTRLs). We created annual air pollution surfaces for NO_2, $PM_{2.5}$ and ozone across California at a spatial resolution of 30 m, then assigned them to enrollees' home addresses. We used a retrospective cohort of 23,000 adults with six years of data (September 1, 2004 to August 31, 2010). We analyzed data using a multilevel generalized linear model with negative binomial distribution and random intercepts for enrollees. We estimated the predicted outcomes for enrollees in GMC with and without policy by using the control group as the counterfactual. To facilitate interpretation, we calculated difference-in-differences (DD) estimates in the first-, second- and third-year after the policy intervention, respectively. To verify the parallel assumption, we also visualized the empirical time trend and tested the differential trends statistically.

Results We observed significant reductions in pollutant exposures for enrollees in 10 counties with the enrollees in GMCs experiencing the greatest reduction from the pre- to post-policy periods. Furthermore, we observed statistically significant reductions in ED visits in the study population in post-policy years. For instance, the number of ED visits among those with asthma living in GMCs significantly decreased comparing with those living in CTRLs in the second year (DD=-0.08, $p<0.01$) and third year (DD=-0.08, $p<0.01$) post-policy. ED visits for those with chronic obstructive pulmonary disease (COPD) were reduced in post-policy years but the policy effects were only found to be statistically significant in the third post-policy year (DD=-0.09, $p<0.005$).

Conclusions Our findings add to empirical evidence that air pollution control actions reduce pollution exposures; and lead to health outcome improvements among people with chronic conditions. Our investigation also contributes to scientific methods for accountability studies assessing the health effects of long-term, large scale, and complex regulatory actions with routinely collected data, such as medical claims data.

Contribution of Household Air Pollution to Ambient Air Pollution in Ghana: Using Available Evidence to Prioritize Future Action

Pallavi Pant (Presenter), Katy Walker, and the HEI Panel on Air Pollution in Ghana

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Background In 2017, the Global Burden of Disease study estimated that nearly half of the global population, about 3.6 billion people, continued to rely on solid fuels for cooking, a major source of exposure to household air pollution (HAP) in the form of fine particulate matter ($PM_{2.5}$). Despite years of efforts to reduce exposures in the home, the health burden attributed to HAP remains high — an estimated 1.64 million deaths in 2017. What has not yet been fully appreciated by policymakers is that household reliance on solid or other polluting fuels is also a major source of ambient air pollution with health consequences for broader populations. Household air pollution's contribution to this larger health burden offers an additional policy lever to motivate transitions to cleaner energy solutions.

Objectives The primary objectives of this study were to evaluate the current evidence on the contributions of HAP to emissions, ambient air pollution and health in Ghana, implications for application and expansion of such work in Ghana, and the lessons for other low- to middle-income countries.

Approach HEI staff, in consultation with a group of experts working in Ghana and the sub-Saharan region, identified relevant studies and summarized the state of knowledge on the contributions of HAP to emissions, air quality and health in Ghana.

Results No studies to date have specifically estimated the contribution of HAP to health burden via its contribution to ambient $PM_{2.5}$ in Ghana; none was initially designed with this objective in mind. However, Ghana has been beneficiary of several independent, complementary, but not fully comparable efforts, using top-down and bottom-up methods for source apportionment. Results from the studies suggest that: household fuel use may account for nearly 65% of total primary $PM_{2.5}$ emissions in Ghana and that biomass combustion, as a broad category, explains 10 to 36% of ambient $PM_{2.5}$ concentrations. The studies also make clear that $PM_{2.5}$ air pollution is a regional problem, requiring regional solutions. Several ongoing studies and emerging resources were also identified and will pave the way for development of accurate estimates of household and other source contributions to ambient air pollution and health.

Opportunities This review identifies several near- and longer-term opportunities to advance both the capacity to understand and to address household air pollution's impact on population health in Ghana and the region: (1) communication of the current results showing the importance of household air pollution as a contributor to emissions and ambient air pollution in Ghana; (2) support for sustained development and improvement of emissions inventories in Ghana and the West African region; (3) support for expanded ground level air quality monitoring in Ghana; (4) harmonization of ongoing efforts and better coordination among researchers in the region; and (5) support for regional coordination and action on air pollution.

Maternal Smoking During Pregnancy and Asthma in Children and Young Adults

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Background Asthma is a common disease with a heterogeneous etiology. The role of ambient air pollution on asthma development is unclear, as multiple factors starting from fetal life may be involved. Some of these factors may be correlated or share sources and pathways resulting in joint effects that are greater than additive. Thus, the potential for confounding and modification of the air pollution exposure effects on asthma by maternal smoking during pregnancy is very high.

Objectives To prepare for our HEI funded study on air pollution and asthma, we examined the associations between maternal smoking during pregnancy and asthma. We examined different definitions of asthma since the severity can range from intermittent to severe disease.

Methods We included all live-born children born to women with singleton pregnancies in Denmark without infant death or emigration during 1997 to 2016. We used the unique personal identification number for data linkage and to extract information about asthma and personal characteristics from population-based registries. We defined a prescription case of asthma as (1) a child who had redeemed any type of anti-asthmatic drug except for beta2-agonists as liquid, inhaled beta2-agonists only once or inhaled steroid only once, and (2) a hospitalization case of asthma as a child who had a registered asthma diagnosis. For both definitions, only the first registered case was used. We also categorized asthma into three distinct phenotypes for children born before 2002; early transient as having asthma only during ages 4-6, but not thereafter, persistent as having asthma during 4-6 years of age and still having asthma during 14-19 years of age and adolescent-onset as having asthma after, but not before age 14. Odds ratios (ORs) for asthma associated with smoking were estimated using adjusted logistic regression with generalized estimating equations (GEE).

Results Out of the 1,156,878 children, 32.4% had asthma according to redeemed prescriptions and 6.4% had a diagnosis from the hospitalization registry. Overall, 14.0% of the mothers smoked and the prevalence of smoking decreased over time (21.4% in 1997-2001 vs. 8.4% in 2012-2016). Any smoking during pregnancy was associated with increased odds of asthma at any age (adjusted ORs and the 95% confidence intervals were 1.38 (1.35, 1.41) and 1.41 (1.40, 1.43) for prescription and hospitalization, respectively). Increased odds were evident for early transient and persistent asthma, but not for adolescent-onset asthma.

Conclusions These preliminary findings support existing evidence that links maternal smoking during pregnancy with increased risk of asthma development. We plan to examine the independent and combined effects of air pollution and smoking when the data are available.

Health Effects of Air Pollution Components, Noise, and Socioeconomic Status ("HERMES")

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Background Traffic-related air pollution (TRAP), traffic noise and low socio-economic status (SES) impair health, including cardiovascular disease and diabetes. However, knowledge gaps still remain including identification of the causal agent(s) in the complex TRAP, the degree of confounding or possible interaction between TRAP and traffic noise, and how socio-economic status (SES) and individual susceptibility interplay in this equation.

Objectives The objectives of the study are (1) to identify the specific TRAPs strongest associated with myocardial infarction (MI), stroke and diabetes; (2) to disentangle how TRAP and road traffic noise interact in relation to these endpoints; (3) to investigate how SES, green spaces, co-morbidity and stress confound or interact with the associations between TRAP and road traffic noise and risk of MI, stroke and diabetes; and (4) to investigate effects of TRAP and road traffic noise in relation to cardiovascular and metabolic biomarkers.

Experimental Design and Results At present (month 8 of the study) we focus on data collection and method development.

Data collection. We use data for the entire Danish population, the Diet, Cancer & Health cohort, and the DCH Next Generations cohort. We will link each individual to the nationwide Danish registries with information on residential address history, prevalent and incident MI, stroke and diabetes, vital status, indicators of stress and SES. We will present an overview of the registries and data to be combined in the HERMES database, and we will provide a status of the data collection process.

Modelling ultrafine particles (UFP). We have implemented the M7 UFP module into the Danish AirGIS dispersion modelling system. We will present the model set-up.

Development of a new statistical method to disentangle effect of correlated pollutants. This approach is based on a first step where machine learning techniques are used to capture all links between pollutants and outcomes without imposing linearity assumptions or similar. In a second step, the prediction model from the first step is used to predict events under specific pollutants scenarios. From a technical perspective, this can be viewed as using machine learning methods as the so-called Q-model when applying the G-formula, which is well-known from causal inference theory. Similar ideas have recently been implanted in the R-package Causal forest; however, this implementation cannot accommodate the time-to-event type outcomes that we analyse in HERMES. We have extended the method to also handle time-to-event outcomes.

Discussion/Interpretation The data collection is ongoing. We will present a first comparison of modelled and measured UFP concentrations at regional, urban background and street level. We will present our first experience with the performance of the new statistical model on simulated data.

Multicenter Ozone Study in oldEr Subjects (MOSES), Part 2: Impacts of Personal and Ambient Concentrations of Ozone and Other Pollutants on Cardiopulmonary Biomarkers

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Background The Multicenter Ozone Study in oldEr Subjects (MOSES) was a multicenter study evaluating whether short-term controlled exposure of older, healthy individuals to low levels of ozone induced acute changes in cardiovascular biomarkers. In MOSES-1, ozone exposure caused concentration-related reductions in lung function with evidence for airway inflammation and injury, without effects on cardiovascular function. However, other pollutant exposures before the study may have confounded or modified these effects.

Study Design In MOSES-2, we used a longitudinal panel study design, MOSES-1 cardiopulmonary biomarker data, and passive personal exposure samplers (PES) for ozone and NO_2, and ambient air pollution measurements in the 96 hours before the pre-exposure visit. Using mixed effects linear regression, we evaluated whether pollutant concentrations confounded the MOSES-1 controlled ozone exposure effects on pre- to post-exposure biomarker changes (Aim 1), modified these biomarker responses to the controlled ozone exposures (Aim 2), were associated with changes in biomarkers measured at the pre-exposure visit or morning of the exposure session (Aim 3); and whether they were associated with differences in the pre- to post-exposure biomarker changes independent of the controlled ozone exposures (Aim 4).

Results Controlled ozone exposure effects on pre- to post-exposure biomarker differences were little changed when including PES or ambient pollutant concentrations in the model (Aim 1). Controlled O_3 exposure effects on FEV_1 and FVC were modified by ambient NO_2 and CO, and PES NO_2, with reductions observed only when pollutant concentrations were "Medium" or "High" in the 72 hours before the pre-exposure visit. There was no modification of the controlled O_3 exposure effect on any other pollutant/biomarker association (Aim 2). Increased ambient O_3, $PM_{2.5}$, CO, and NO_2 concentrations were associated with decreased pre-exposure heart rate variability (HRV; Aim 3), but also associated with increases in HRV from pre- to post-exposure (Aim 4), likely reflecting a 'recovery' during the MOSES O_3 exposure sessions. A similar pattern was observed for FEV_1 and FVC and ambient $PM_{2.5}$, CO, and NO_2. Increased pollutant concentrations were not associated with adverse changes in other biomarker groups.

Conclusions Our MOSES-1 findings of controlled ozone exposure effects on pulmonary function, but effects on cardiovascular biomarkers, were not confounded by ambient or personal pollutant exposures before the pre-exposure visit. MOSES-1 ozone effects on pulmonary function were modified by ambient NO_2 and CO, and PES NO_2, with reductions observed only when these pollutant concentrations were elevated in the hours/days before the pre-exposure visit. Increased ambient O_3, $PM_{2.5}$, NO_2, and CO concentrations were associated with reduced HRV and pulmonary function, with "recovery" during exposure visits. Increased pollutant concentrations were not associated with changes in other cardiopulmonary biomarkers.

Review of the Literature on Potential Exposures and Health Effects from Unconventional Oil and Gas Development

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HEI's Energy Research Program The purpose of the Program is to identify and conduct high priority research on potential population exposures and health effects from development of oil and natural gas from shale and other unconventional resources (UOGD) across the United States. A source of high-quality, impartial science is needed to support policy decisions about how best to ensure protection of public health in the oversight and implementation of this development.

Literature Reviews to Inform Research Planning HEI's Energy Research Committee (the "Committee") has conducted impartial, critical reviews of the literature describing potential exposures and health effects among people living in areas where they might be exposed to chemical agents (e.g., benzene) or non-chemical agents (e.g., noise) from UOGD. These reviews will benefit those in society who are navigating the growing and sometimes conflicting body of information on this topic.

The Committee conducted the reviews following five general steps: (1) develop an understanding of UOGD operations across regions and time, (2) define questions to guide literature review, (3) solicit information and recommendations from a broad range of stakeholders, (4) gather and extract information from the literature, and (5) define knowledge gaps in the literature and research questions to address them. The health literature review consisted of a systematic review of twenty-five analytical epidemiology studies published between 2012 and 2018. The exposure literature review involved assessment of hundreds of reports and peer-reviewed scientific papers published in recent years, which report on the level of chemical or non-chemical agents in environmental media (e.g., air and water) to which people might be exposed and that might be attributable, at least in part, to UOGD.

Findings and the Path Forward to Exposure Research The epidemiology literature represents a substantial effort to understand potential health effects at a relatively early stage of UOGD expansion in the United States, with investigators making innovative use of available exposure and health data. Results are mixed and subject to some important sources of uncertainty that indicate a need for additional research that overcomes these limitations.

The exposure literature has grown rapidly in the last several years, focused primarily on air and water quality. While much progress has been made in understanding the potential for exposure, few studies successfully address source attribution among UOGD, conventional oil and gas development, other industries, and natural sources. Also, the generalizability of studies to date is not clear given important sources of variability across time and locations and evolving UOGD operational practices in response to changing regulations, community concerns, and technological innovation.

The Committee has begun discussing research priorities, informed by the literature reviews, a tour of oil and gas operations, workshops, and stakeholder consultations. HEI expects to fund population-level exposure research in two or three major oil- and natural gas-producing regions of the United States, beginning in 2019.

Design and Implementation of a Mobile Monitoring Campaign for Epidemiology*

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Background There is increasing evidence that traffic-related air pollutants (TRAP) such as ultrafine particles (UFP) and black carbon (BC) may have adverse neurologic effects. However, study of these effects is profoundly limited by the lack of fine-scale spatially varying measurements that are representative of human exposures. Our goal is to design and conduct an innovative mobile monitoring sampling campaign that will allow valid inference about the impact of long-term exposure to TRAP on the aging brain.

Application and Objective We are enriching the ongoing Adult Changes in Thought Air Pollution (ACT-AP) study with spatially varying TRAP data. This population-based cohort study has followed adults aged 65 and older in the Puget Sound region since 1994. Our objective is to obtain good estimates of annual average TRAP concentrations for 2019; use these to predict annual average spatial surfaces of TRAP, as individual pollutants (UFP, BC, nitrogen dioxide [NO_2], carbon monoxide [CO], and carbon dioxide [CO_2]) and as a multi-pollutant summary; and then by leveraging time-varying traffic information, extrapolate this surface back to 1990.

Network Design Considerations Preliminary design development analyses showed that the optimal approach to using a mobile platform to obtain an unbiased annual average estimate of multiple pollutants at a given location is to repeatedly sample for very short time periods multiple times over a year. Our analyses showed that it is essential to visit each location at all times of day, days of the week, and seasons of the year. To ensure spatial compatibility in our design, we compared distributions of key covariates for 300 proposed locations with the locations of ACT participant residences.

Approach We have outfitted a mobile platform to measure BC, UFP, $PM_{2.5}$, CO, CO_2 and NO_2. Throughout 2019 we are driving this platform 280 days on nine fixed routes in the Puget Sound region with approximately 35 designated two-minute stops on each route. The protocol incorporates appropriate quality control activities, including co-locations, regular calibrations, regular review of data for completeness and appropriateness, and make-up days to account for equipment failure. By following fixed driving routes, we will be able to address whether incorporating on-road mobile data will further enhance our ability to model TRAP spatially. We will use geostatistical models with R-LINE, the U.S. EPA's mobile source pollutant dispersion model, and other geocovariates to predict annual average TRAP. R-LINE will allow us to account for temporal changes in TRAP when extrapolating the surface back in time to align the estimated exposures to biologically relevant exposure time periods in the cohort.

Conclusion This innovative mobile monitoring study is the first of its kind to be designed specifically for exposure assessment in an epidemiologic cohort.

* Study not funded by HEI.

Formation of Reactive Oxygen Species in Epithelial Lining Fluid by Particle Deposition and Comparisons with Oxidative Potential Measurements

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Background Reactive oxygen species (ROS) play a central role in adverse health effects and oxidative stress of atmospheric air particulate matter. Respiratory particle deposition can lead to the release of ROS in the epithelial lining fluid due to catalytic reactions cycles of redox-active components including soluble transition metal ions and organic compounds with lung antioxidants.

Methods Size-segregated ambient particles were collected in Atlanta in 2016 and organic carbon and water-soluble metals were measured. A kinetic multi-layer model of surface and bulk chemistry in the lining fluid (KM-SUB-ELF) model was combined with a human respiratory tract model to estimate the concentrations and production rates of different types of ROS (hydroxyl radical [$OH^{\bullet}$], superoxide radical [$O_2^{\bullet-}$], hydrogen peroxide [H_2O_2]). The model considers ROS formation by redox reactions of metal ions and quinones as well as decomposition of organic hydroperoxides contained in secondary organic aerosols (SOA). The estimated ROS production rates were compared with oxidative potential measured with the dithiothreitol (DTT) and ascorbic acid assays.

Results The extrathoracic region was found to have higher ROS concentrations compared to the bronchial and alveolar regions due to higher particle deposition into epithelial fluid with lower volume. Iron (Fe) and copper (Cu) ions contribute mainly to H_2O_2 and O_2^- production rates, which show strong correlation with measured oxidative potentials. In contrast, oxidative potential does not exhibit significant correlations with OH production rates, which are mainly driven by SOA decomposition and Fenton(-like) reactions of metal ions.

Conclusions Oxidative potential is a good indicator of production of H_2O_2 and O_2^- but not OH in epithelial lining fluid. Combination of field measurements of chemical composition and oxidative potential with model simulation can provide critical insights into ROS formation by ambient particulate matter in the human respiratory tract.

Long-Term Outdoor Air Pollution and Cause-Specific Mortality in a Pooled Analysis of Asian Cohorts

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Background Air pollution represents one of the most important risk factors for human health and global burden of disease (GBD). Annual GBD studies attribute approximately 4 million deaths per annum to ambient (outdoor) air pollution with much of the world — especially those in low-and-middle-income nations subject to levels of ambient air pollution in excess of World Health Organization (WHO) guidelines. The bulk of studies from which disease burdens and policy efforts have been derived are performed in North America and Europe. However, the largest global impacts of air pollution are estimated to occur in Asia, where air pollution levels are typically higher than North America and Europe. It is therefore uncertain whether the linear associations between air pollution and health often found in relatively low pollution areas extend to the higher pollution levels found in Asia. This knowledge gap is further compounded by cultural and socio-economic differences between (and within) Asian nations.

Objectives The objectives of this project are to assess the association between long-term exposure to outdoor $PM_{2.5}$ and NO_2 and all-cause and cause-specific mortality in a pooled analysis of Asian cohorts. As a secondary objective, we aim to explore any observed heterogeneity in mortality risks in the context of cultural, social, economic, or infrastructural differences between cohorts and countries.

Study Design Cohorts are being recruited from the Asia Cohort Consortium — a multicenter consortium representing over 20 cohorts and over 1 million participants. Levels of ambient air pollution will be assigned to the residential address(es) of study participants via the application of satellite-derived global models of air pollution (specifically $PM_{2.5}$ and NO_2). The association between ambient air pollution and natural cause and cause-specific mortality will be assessed by the generation of Cox proportional hazards models. Specific mortality outcomes which will be evaluated include lung cancer, cardiovascular disease, metabolic disease, and non-malignant lung disease. Data will be analyzed as a single "meta-cohort" in addition to cohort-specific analyses with subsequent meta-analysis. In addition to linear analysis, spline analysis will be employed to further evaluate the shape of the exposure-response relationship, especially at the higher levels of exposure. Together, these findings will provide important new insight into the burden of disease attributable to air pollution.

The Impacts of Environmental Exposures on Public Health Outcomes*

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Background Environmental exposures are a significant determinant of health and wellness. Despite decades of air quality improvements, air pollution exposure remains a substantial contributor to morbidity and mortality in the United States. Tackling the public health burdens linked to environmental exposures, requires comprehensive and coordinated research into exposure risks, human behaviors, and environmental health messaging.

Methods Multiple research efforts are required to fully understand environmental health risks, exposure reducing behaviors (ERBs), and environmental health messaging. EPA CARES is a collaborative resource which enables the use of electronic medical records for environmental epidemiology. SmokeSense is a mobile application that provides information on wildland fires, collects smoke exposure and health symptom data, and encourages citizen science. These efforts, in combination with research into existing literature for exposure risks, allow for a comprehensive, translational approach to environmental health.

Results By studying $PM_{2.5}$ exposure in >30,000 heart failure patients from 2004 – 2016 and available in EPA CARES, researchers associated annual average $PM_{2.5}$ exposure with 0.86 years of life lost (95% confidence interval [CI] = 0.77 – 0.95) for patients with exposures above the 12 $\mu g/m^3$ national annual standard as opposed to below. Additionally, heart failure patients had differing mortality risks based on their socioeconomic environment. Patients residing in rural communities with middling (hazard ratio [HR] = 1.13, CI = 1.05 – 1.19) to low (HR = 1.08, CI = 0.99 – 1.15) socioeconomic indicators had increased mortality risks as compared to patients living in socioeconomically advantaged suburban communities.

Observations from EPA CARES complement data from SmokeSense. With a 92% user return rate, SmokeSense highlights the enthusiasm for an application which tracks smoke exposure, health symptoms, and provides information on wildland fires. SmokeSense users with pre-existing health conditions have a higher odds of reporting health symptoms when impacted by smoke exposure. The majority of SmokeSense users, even those with pre-existing conditions, report taking ERBs, e.g. wearing a dust mask, only after multiple days of symptoms, demonstrating a need for research and communication on the benefits of proactive ERBs. A literature review on the benefits of exercise versus air pollution exposure risks, further highlighted the need for more research and effective messaging for those vulnerable to environmental exposures. A literature review showed that while studies for the general population frequently reported that outdoor exercise may outweigh air pollution exposure risks, studies of more vulnerable populations, like children, indicated that exposure risks likely outweigh exercise benefits.

Conclusions Effecting change in the public health burdens arising from environmental exposures requires a complete research program incorporating epidemiological research, research into effective messaging, and increased understanding of ERBs. As such coordinated research efforts emerge, we will better understand health risks in vulnerable communities, behaviors surrounding exposure reduction, and how environmental health messaging may be improved and supported with research efforts.

Note: This abstract does not necessarily reflect U.S. EPA policy.

* Study not funded by HEI.

Impacts of Shipping on Air Pollutant Emissions, Air Quality, and Health in Yangtze River Delta/Shanghai, China

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Background Globally, air pollution from shipping contributes to approximately 60,000 to 135,000 lung cancer and cardiopulmonary deaths each year. Between 9,400 and 26,600 of those deaths are in China, near some of the world's busiest coastal and inland water ports. We investigated the impacts on air quality and health of recently-implemented and proposed emission control areas to reduce near-shore emissions of air pollutants from ships in the region of one of the busiest ports in China.

Methods We developed air pollutant emissions inventories of sulfur oxides, particulate matter, nitrogen oxides, and volatile organic carbon from coastal and oceangoing ships, inland-water ships, port machinery and trucks, and other land-based sources for the Yangtze River Delta (YRD) and Shanghai city. We considered scenarios with and without shipping under baseline (year 2015) conditions and in 2030 under current, anticipated, and aspirational policies. We used the Community Multiscale Air Quality model to predict fine particulate matter ($PM_{2.5}$) concentrations for each scenario. The impact on health was estimated using the Benefits Mapping and Analysis Program—Community Edition. Relationships of mortality and total hospital admissions with air pollution were estimated exposure response functions from the Global Burden of Disease study and local cohorts.

Results Shipping contributed up to 4.62 $\mu g/m^3$ of $PM_{2.5}$ in the YRD under the summer monsoon conditions. In the YRD, about half of the annual emissions of ship-related $PM_{2.5}$ and more than 60% of annual emissions of sulfur dioxide were from ships within 12 nautical miles of shore. However, ships as far as 100 nautical miles offshore contributed to $PM_{2.5}$ concentrations in the YRD. In Shanghai City, inland-water ships contributed 40% to 80% of the shipping impact on urban air quality and contributed more to population-weighted concentrations than other shipping-related sources. We attributed 3,500 excess deaths and 353,000 hospital admissions in the YRD to shipping emissions in 2015. About 200 of the estimated excess deaths were from shipping emissions in municipal waters of Shanghai City. Contributions of shipping to ambient $PM_{2.5}$ and health impacts were lower in all three 2030 scenarios than they were in 2015.

Conclusions This study explored the impact of shipping emissions and control policies on air quality and health in Shanghai and the broader YRD region. Our results provide scientific evidence to inform policy discussions for controlling future shipping emissions; in particular, stricter standards could be considered for the ships on inland rivers and other waterways close to residential regions.

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*Study not funded by HEI.

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