Air Pollution and Health: A Never-Ending Story

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Program for Air Quality, Health, and Society,
University of Utah
April 14, 2014
Geneva Steel Mill, Utah Valley: 1980s
What I am going to talk about.

• Evidence and environmental policy.
• Evidence-driven air quality improvement.
• Further scientific challenges.
• Problems we need to address now.
Air Pollution Disasters of the 20th Century

The Fog Disaster in the Meuse Valley, 1930...led to the first scientific proof of the potential for atmospheric pollution to cause deaths and disease, and it clearly identified the most likely causes. 60 deaths that were attributed to the fog occurred on Dec 4 and 5. Nemery et al. Lancet 2001

Beginning on October 26, 1948, sparse air movement contributed to a temperature inversion in the atmosphere over western Pennsylvania, Ohio, and areas of neighboring states. A fog laden with particulates and other industrial contaminants saturated the air of Donora, a small industrial town on the banks of the Monongahela River, some 30 miles south of Pittsburgh. Visibility was so poor that even locals lost their sense of direction. An estimated 5000 to 7000 persons in a town of 14000 residents became ill, some 400 required hospitalization, and 20 died before rain dispersed the killing smog on October 30 and 31, 1948. Helfand et al.
“This is why the London Disaster of 1952 should be commemorated; the many efforts to limit ambient air pollution that have occurred in the past 50 years are the proper memorial to those who were its unheralded victims.”

David V. Bates

Environmental Health Perspectives • Vol 110 | No.12 | December 2002
Burden of disease attributable to 20 leading risk factors in 2010, expressed as a percentage of global disability-adjusted life years, both sexes.
What evidence?

• Evidence for causation?
  – Does A cause B?

• Evidence for the magnitude of risk?
  – By how much does exposure to A increase risk for B?

• Evidence for the consequences of removal of A for the occurrence of B?
  – How would the occurrence of B be affected by the removal of A?
FIGURE 2.2  SOURCE: Drawing by Richter; ©1988 The New Yorker Magazine, Inc.
The Yin and Yang of Evidence and Uncertainty

**Evidence**
- What we know
- Determine causation and risk
- Basis for action
- Developed with synthesis/integration

**Uncertainty**
- What we do not know
- Basis for more research
- Delay action
- Uncertainties will always persist
The Evidence Scale

Act

Evidence

Uncertainty

Not Act

Politics
Costs
Activists
Advocates
The Evidence Scale

Act

Not Act

Evidence

Politics
Costs
Activists
Advocates

Uncertainty
Equipoise and Evidence
What is it?

The balance point for strength of evidence on risk and causation
Word clouds generated using Wordle, with selected words commonly associated with either the Democratic Party or the Republican Party, political parties in the United States.
Ozone Decision in the News

Obama ozone decision blindsides enviros - and his own EPA

The White House deflected suggestions the president caved into GOP pressure. | AP Photos
Public Comments: O₃ Reconsideration

- The evidence is flawed; action not needed.
- The evidence is clear; action is needed.
- Regulation costs jobs.
- Regulation is complicated by background O₃ levels.
- Risk is not large.
Creating “Doubt”

Merchants of Doubt
How a Handful of Scientists Obscured the Truth on Issues from Tobacco Smoke to Global Warming
Naomi Oreskes & Erik M. Conway

Doubt Is Their Product
How Industry’s Assault on Science Threatens Your Health
David Michaels

The Bottom Line or Public Health
Tactics Corporations Use to Influence Health and Health Policy, and What We Can Do to Counter Them
Edited by William H. Wiist
EVIDENCE DRIVEN IMPROVEMENTS IN AIR QUALITY
LA’s Storied Past:
The Birthplace of Smog

July 26, 1943: L.A. Gets First Big Smog

Source: www.lasmogtown.com
A blanket of warm air covered the city yesterday, trapping pollutants and sending the air-pollution index close to the danger mark. Officials warned persons with heart, lung or respiratory ailments to stay indoors.

From 6 A. M. to 10 A. M., the amount of sulphur dioxide, carbon monoxide and dust-carrying haze was so high that Austin N. Heller, the Commissioner of Air Pollution Control, was on the verge of calling the first-alert stage of the city's air pollution warning system.

The air became cleaner in the late morning and early afternoon, but the pollution shot up again in the evening, with the index reaching a high of 60.6 between 8 and 9 P. M.

Commissioner Heller said the pollution count was possibly the highest in the city's history.
The Mandate: Clean Air Act

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as one “the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, is requisite to protect the public health.”
New NAAQS review process

April 2009

Peer-reviewed scientific studies

Integrated Review Plan: timeline and key policy-relevant issues and scientific questions

CASAC review and public comment

Integrated Science Assessment: concise evaluation and synthesis of most policy-relevant studies

CASAC review and public comment

Risk/Exposure Assessment: concise quantitative assessment focused on key results, observations, and uncertainties

Policy Assessment: staff analysis of policy options based on integration and interpretation of information in the ISA and REA

EPA proposed decision on standards

Interagency review

Agency decision making and draft proposal notice

Public hearings and comments on proposal

Agency decision making and draft final notice

Interagency review

EPA final decision on standards
Six Cities Study

Surveys
Ecological studies
Early time-series studies

1960
1970
1980
1990
2000
2010

Exposure assessment

Modern time-series studies
Multi-site studies

Research Approach

General Questions

What are the adverse effects of air pollution?

What are the exposure-response relationships?

What are the key sources? What determines mixture toxicity?
What are risks at current levels?

• For mortality:
  – Short-term increases in mortality
  – Longer-term increases in mortality

• For morbidity:
  – Diverse adverse respiratory effects
  – Adverse cardiovascular effects
  – Other effects: reproductive
  – Susceptible populations
The Clean Air Act Success Story

Cumulative benefits 1970-90
$6 to $50 trillion
Cumulative costs - $520 billion

Gross Domestic Product
Vehicle Miles Traveled
Energy Consumption
Population
NOx Emissions
VOC Emissions
CO Emissions
SO2 Emissions
The Clean Air Act and Health — A Clearer View from 2011

Jonathan M. Samet, M.D.

From my office, I have views of downtown Los Angeles and the San Gabriel Mountains. Air pollution infrequently obscures these views, and only rarely are my eyes and throat irritated by smog when I’m outdoors. The Los Angeles air of today is far better than that of the mid-20th century, when severe oxidant pollution, initially of unknown origins, threatened the health and welfare of the city’s residents. Severe smog was a common occurrence, leading to public health concerns and environmental damage.

The 20th-century pollution episodes and the pervasive smoke problem in cities motivated increasingly stringent and sweeping laws and programs to address air pollution. For more than 40 years, the CAA, aided by amendments passed in 1977 and 1990, ceased to be a widespread problem when it was removed from gasoline. These pollutants are referred to as “criteria pollutants,” thanks to a passage in the law that requires the administrator of the Environmental Protection Agency (EPA) to issue “air-quality criteria,” accurately reflecting the scientific evidence related to identifiable public health and environmental effects, for any substance designated as an air pollutant.
FURTHER SCIENTIFIC CHALLENGES
“However, we should expect that, as the severity of the dose increases, the effects will become more and more obvious, more and more evident, and more and more dramatic. If the intention of any legislation is prevention of morbidity, we necessarily have to deal with “weaker” evidence. ..Research to establish these elusive cutoff points has given way to a more sensible effort to define with greater precision the magnitude of the risk…”
The Narrowing Range of Exposure

National Ambient Air Quality Standards
http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_index.html
The Relationship between Air Pollution and Emergency Room Visits in an Industrial Community

Steubenville, Ohio, 1974-1977

<table>
<thead>
<tr>
<th></th>
<th>TSP</th>
<th>SO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 hr mean</td>
<td>156</td>
<td>90</td>
</tr>
<tr>
<td>Mean (µg/m³)</td>
<td>14-696</td>
<td>4-369</td>
</tr>
</tbody>
</table>

This study assessed the relationship between air pollution levels and numbers of emergency room visits in an industrial town. Records of emergency room visits to the major hospital during March, April, October, and November of 1974–1977 were abstracted. Air pollution and temperature data were collected from instruments located centrally, near the hospital. For the analyses, the morbidity indices were the deviations of daily visit numbers from the expected values based on day of week, season, and year. The diagnostic categories were collapsed to “all respiratory diseases,” “all diseases but trauma,” and “all diseases.” The mean deviations for each category were first grouped by pollutant quartile and by maximum temperature. These deviations did not vary in a fashion consistent with a pollutant effect for SO₂, TSP, NO₂, CO, or O₃—either unlagged or lagged by 24 hours. A linear regression model showed small but statistically significant effects of unlagged TSP and of unlagged SO₂ on the respiratory disease index. For TSP, however, days above the 24-hr federal standard were not associated with excess respiratory disease visits. These analyses demonstrate at most a limited association between air pollutant visits and respiratory disease emergency room visits.

APCA Journal 1981;31(3):236-40

<table>
<thead>
<tr>
<th></th>
<th>TSP</th>
<th>SO₂</th>
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</thead>
<tbody>
<tr>
<td>Mean (µg/m³)</td>
<td>67.3</td>
<td>46.8</td>
</tr>
<tr>
<td>Range (µg/m³)</td>
<td>14.5-222.0</td>
<td>0.8-282.0</td>
</tr>
</tbody>
</table>

24 hr mean

J. E. Kelsall, 1

Analyses of daily mortality data for Philadelphia, Pennsylvania, 1974-1988, were conducted with air pollution data for TSP (total suspended particles) and SO₂ (sulfur dioxide). The analyses were based on prior understanding of the effects of these factors on mortality and on consideration of model fit. The authors found moderate correlations of daily concentrations of total suspended particles (TSP), sulfur dioxide (SO₂), nitrogen dioxide (NO₂), and carbon monoxide (CO), and only slight correlations of ozone (O₃) with other pollutants. When included individually in the model, the means of current and previous days’ levels of TSP, SO₂, and O₃ had statistically significant effects on total mortality; pollutant increases of an interquartile range (34.5 µg/m³, 12.9 ppb, and 20.2 ppb, respectively) were associated with increases in mortality of around 1% for TSP and SO₂, and of around 2% for O₃. The effects of TSP and SO₂ were diminished when both pollutants were simultaneously included in the model, whether pairwise or in the full multi-pollutant model. These analyses confirm the association between TSP and mortality found in previous studies in Philadelphia and show that the association is robust to consideration of other pollutants in the model. Am J Epidemiol 1997;146:750-62.

air pollution; mortality; ozone; weather

Scott L. Zeger,1 Francesca Dominici,1 Aidan McDermott,1 and Jonathan M. Samet2

1Department of Biostatistics, and 2Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA

<table>
<thead>
<tr>
<th>U.S. Medicare cohort, 2000-2005 (annual average PM$_{2.5}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>U.S. Region</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Eastern</td>
</tr>
<tr>
<td>PM$_{2.5}$ (µg/m$^3$)</td>
</tr>
</tbody>
</table>

In order to reduce the potential for confounding in the study of the health effects of air pollution, we have developed a new retrospective cohort study, the Medicare Cohort Air Pollution Study. The study population comprises 13.2 million participants living in 4,568 ZIP codes having centroids within 6 miles of a PM$_{2.5}$ monitor. We estimated relative risks adjusted by socioeconomic status and smoking by fitting log-linear regression models.

RESULTS: In the eastern and central regions, a 10-µg/m$^3$ increase in 6-year average of PM$_{2.5}$ is associated with 6.8% [95% confidence interval (CI), 4.9–8.7%] and 13.2% (95% CI, 9.5–16.9) increases in mortality, respectively. We found no evidence of an association in the western region or for persons ≥ 85 years of age.

CONCLUSIONS: We established a cohort of Medicare participants for investigating air pollution and mortality on longer-term time frames. Chronic exposure to PM$_{2.5}$ was associated with mortality in the eastern and central regions, but not in the western United States.

Hierarchical oxidative stress model: response to redox cycling

The Johns Hopkins PM Center
National Risk-Based Approach

• Map variation in risk of PM across the country using mortality and Medicare hospitalization as outcomes
  – Select sites based on risk differential

• Collect and characterize PM in locations with contrasting PM risks
  – High vs. low risk sites

• Carry-out biological assays on the PM from the selected locations
Bulk Particulate Matter Sampling
Hopkins Sequential Cyclone System x 2

Integrated sample collection
  – QA/QC
    • leak test
    • flow test
    • daily inspection

  – Sample collection
    • mg quantities coarse
    • mg quantities fine
    • harvested weekly and at end

Platforms built by Doug Knowlton / Seattle
Asthmatic Mouse Model

AJ Mice

<table>
<thead>
<tr>
<th>Day 0</th>
<th>Day 14</th>
<th>Day 17</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ovalbumin Sensitization 0.4 mg/kg i.p.</td>
<td>Ovalbumin Challenge 30 mg/kg i.t.</td>
<td>PM exposure 1, 3, 10 mg/kg i.t.</td>
</tr>
</tbody>
</table>

Outcomes all at Day 4
- Airway Hyperreactivity (AHR)
- BAL (cell/protein)
- Cytokine
- Histology
- Toxicogenomic profile

BAL/Tissue Evaluation
- BAL protein/BAL cells
- BAL cytokines

Bio-Informatics & Molecular Signatures
- Lung microarray
Multi-organ involvement in PM-induced cardiac arrhythmias in CREB$_{A133}$ mice with CHF

Increased lung permeability compared to CD-1 wild type mice & increased ROS and cytokine generation

Altered ion channel expression
Increased inflammatory cytokines
Heightened CB sensitivity

INCREASED CARDIAC ARRYTHMIAS
What is the Form of the Relationship?

- Supra-linear
- Linear, No-threshold
- Linear, Threshold
- Sublinear
The Political Morphology of Dose-Response Curves

Proctor (1995)
Cancer Wars
PM$_{10}$-total mortality dose-response curve for the mean lag PM$_{10}$ and 95% pointwise CI

Dose-Response for PM$_{10}$ And Total Mortality

Source: Daniels et al. 2001
Epidemiology and Prevention

Cardiovascular Mortality and Exposure to Airborne Fine Particulate Matter and Cigarette Smoke

Shape of the Exposure-Response Relationship

C. Arden Pope III, PhD; Richard T. Burnett, PhD; Daniel Krewski, PhD; Michael Jerrett, PhD; Yuanli Shi, MD; Eugenia E. Calle, PhD; Michael J. Thun, MD

Background—Fine particulate matter exposure from both ambient air pollution and secondhand cigarette smoke has been associated with larger risks of cardiovascular mortality than would be expected on the basis of linear extrapolations of the relative risks from active smoking. This study directly assessed the shape of the exposure-response relationship between cardiovascular mortality and fine particulates from cigarette smoke and ambient air pollution.

Methods and Results—Prospective cohort data for >1 million adults were collected by the American Cancer Society as part of the Cancer Prevention Study II in 1982. Cox proportional hazards regression models that included variables for increments of cigarette smoking and variables to control for education, marital status, body mass, alcohol consumption, occupational exposures, and diet were used to describe the mortality experience of the cohort. Adjusted relative risks of mortality were plotted against estimated average daily dose of fine particulate matter from cigarette smoke along with comparison estimates for secondhand cigarette smoke and air pollution. There were substantially increased cardiovascular mortality risks at very low levels of active cigarette smoking and smaller but significant excess risks even at the much lower exposure levels associated with secondhand cigarette smoke and ambient air pollution.

Conclusions—Relatively low levels of fine particulate exposure from either air pollution or secondhand cigarette smoke are sufficient to induce adverse biological responses increasing the risk of cardiovascular disease mortality. The exposure-response relationship between cardiovascular disease mortality and fine particulate matter is relatively steep at low levels of exposure and flattens out at higher exposures. (Circulation. 2009;120:941-948.)

Key Words: air pollution ■ cardiovascular diseases ■ mortality ■ tobacco smoke pollution ■ smoking
Dose-Response Curves

An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure


http://dx.doi.org/10.1289/ehp.1307049

Received: 6 May 2013
Accepted: 7 February 2014
Advance Publication: 11 February 2014
Figure 1.

![Graphs showing relative risk and PM$_{2.5}$ concentrations for IHD, STROKE, COPD, and LC.](image-url)
Concentration-Response

Risk

Concentration

- Background Risk
- Optimum Risk
- Achievable Risk
- Unacceptable Risk
Using Concentration-Response

- Risk
- Concentration

- Actual
- Target
- Residual
- Benefit

Background
“If control legislation is written from the point of view of “protecting public health,” the question immediately arises as to what would constitute an “impairment of public health.”
One Person: Too Much Data
Air Pollution and Reproductive Effects

Record Count

Publication Year

PubMed search string: ("air pollution"[MeSH Terms] OR ("air"[All Fields] AND "pollution"[All Fields]) OR "air pollution"[All Fields]) AND ("reproduction"[MeSH Terms] OR "reproduction"[All Fields] OR "reproductive"[All Fields])

*partial year data
Review

Ambient air pollution, birth weight and preterm birth: A systematic review and meta-analysis

David M. Stieb a,b,*, Li Chen a, Maysoon Eshoul b, Stan Judek a

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Abstract

Low birth weight and preterm birth have a substantial public health impact. Studies examining their association with outdoor air pollution were identified using searches of bibliographic databases and reference lists of relevant papers. Pooled estimates of effect were calculated, heterogeneity was quantified, meta-regression was conducted and publication bias was examined. Sixty-two studies met the inclusion criteria. The majority of studies reported reduced birth weight and increased odds of low birth weight in relation to exposure to carbon monoxide (CO), nitrogen dioxide (NO2) and particulate matter less than 10 and 2.5 microns (PM10 and PM2.5). Effect estimates based on entire pregnancy exposure were generally largest. Pooled estimates of decrease in birth weight ranged from 11.4 g (95% confidence interval −6.9–29.7) per 1 ppm CO to 28.1 g (11.5–44.8) per 20 ppb NO2, and pooled odds ratios for low birth weight ranged from 1.05 (0.99–1.12) per 10 µg/m3 PM2.5 to 1.10 (1.05–1.15) per 20 µg/m3 PM10 based on entire pregnancy exposure. Fewer effect estimates were available for preterm birth and results were mixed. Pooled odds ratios based on 3rd trimester exposures were generally most precise, ranging from 1.04 (1.02–1.06) per 1 ppm CO to 1.06 (1.03–1.11) per 20 µg/m3 PM10. Results were less consistent for ozone and sulfur dioxide for all outcomes. Heterogeneity between studies varied widely between pollutants and outcomes, and meta-regression suggested that heterogeneity could be partially explained by methodological differences between studies. While there is a large evidence base which is indicative of associations between CO, NO2, PM and pregnancy outcome, variation in effects by exposure period and sources of heterogeneity between studies should be further explored.

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Traffic-Related Air Pollution, Particulate Matter, and Autism

Heather E. Volk, PhD, MPH; Fred Lurmann; Bryan Penfold; Irva Hertz-Picciotto, PhD; Rob McConnell, MD

Context: Autism is a heterogeneous disorder with genetic and environmental factors likely contributing to its origins. Examination of hazardous pollutants has suggested the importance of air toxics in the etiology of autism, yet little research has examined its association with local levels of air pollution using residence-specific exposure assignments.

Objective: To examine the relationship between traffic-related air pollution, air quality, and autism.

Design: This population-based case-control study includes data obtained from children with autism and control children with typical development who were enrolled in the Childhood Autism Risks from Genetics and the Environment study in California. The mother’s address from the birth certificate and addresses reported from a residential history questionnaire were used to estimate exposure for each trimester of pregnancy and first year of life. Traffic-related air pollution was assigned to each location using a line-source air-quality dispersion model. Regional air pollutant measures were based on the Environmental Protection Agency’s Air Quality System data. Logistic regression models compared estimated and measured pollutant levels for children with autism and for control children with typical development.

Setting: Case-control study from California.

Participants: A total of 279 children with autism and a total of 243 control children with typical development.

Main Outcome Measures: Crude and multivariable adjusted odds ratios (AORs) for autism.

Results: Children with autism were more likely to live at residences that had the highest quartile of exposure to traffic-related air pollution, during gestation (AOR, 1.98 [95% CI, 1.20-3.31]) and during the first year of life (AOR, 3.10 [95% CI, 1.76-5.77]), compared with control children. Regional exposure measures of nitrogen dioxide and particulate matter less than 2.5 and 10 μm in diameter (PM$_{2.5}$ and PM$_{10}$) were also associated with autism during gestation (exposure to nitrogen dioxide: AOR, 1.81 [95% CI, 1.37-3.09]; exposure to PM$_{2.5}$: AOR, 2.08 [95% CI, 1.93-2.25]; exposure to PM$_{10}$: AOR, 2.17 [95% CI, 1.49-3.16]) and during the first year of life (exposure to nitrogen dioxide: AOR, 2.06 [95% CI, 1.37-3.09]; exposure to PM$_{2.5}$: AOR, 2.12 [95% CI, 1.45-3.10]; exposure to PM$_{10}$: AOR, 2.14 [95% CI, 1.46-3.12]). All regional pollutant estimates were scaled to twice the standard deviation of the distribution for all pregnancy estimates.

Conclusions: Exposure to traffic-related air pollution, nitrogen dioxide, PM$_{2.5}$, and PM$_{10}$ during pregnancy and during the first year of life was associated with autism. Further epidemiological and toxicological examinations of likely biological pathways will help determine whether these associations are causal.

Air Pollution and Neurological Effects

PubMed search string: ("air pollution"[MeSH Terms] OR "air"[All Fields] AND "pollution"[All Fields]) OR "air pollution"[All Fields]) AND ("nervous system"[MeSH Terms] OR "nervous"[All Fields] AND "system"[All Fields]) OR "nervous system"[All Fields]) OR neurological[All Fields] OR ("neurology"[MeSH Terms] OR "neurology"[All Fields]))
Neurobehavioral effects of ambient air pollution on cognitive performance in US adults

Jiu-Chuan Chen a,b,*, Joel Schwartz b,c

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Cognitive aging
Epidemiology
Memory

ABSTRACT

Background: In vivo animal experiments demonstrate neurotoxicity of exposures to particulate matter (PM) and ozone, but only one small epidemiological study had linked ambient air pollution with central nervous system (CNS) functions in children.

Objectives: To examine the neurobehavioral effects associated with long-term exposure to ambient PM and ozone in adults.

Methods: We conducted a secondary analysis of the Neurobehavioral Evaluation System-2 (NES2) data (including a simple reaction time test [SRTT] measuring motor response speed to a visual stimulus; a symbol-digit substitution test [SDST] for coding ability; and a serial-digit learning test [SDLT] for attention and short-term memory) from 1764 adult participants (aged 37.5 ± 10.9 years) of the Third National Health and Nutrition Examination Survey in 1988–1991. Based on ambient PM10 (PM with aerodynamic diameter <10 μm) and ozone data from the EPA Aerometric Information Retrieval System database, estimated annual exposure prior to the examination were aggregated at the centroid of each census-block group of geocoded residences, using distance-weighted averages from all monitors in the residing and adjoining counties. Generalized linear models were constructed to examine the associations, adjusting for potential confounders.

Results: In age- and sex-adjusted models, PM10 predicted reduced CNS functions, but the association disappeared after adjustment for sociodemographic factors. There were consistent associations between ozone and reduced performance in NES2. In models adjusting for demographics, socioeconomic status, lifestyle, household and neighborhood characteristics, and cardiovascular risk factors, ozone predicted high scores in SDST and SDLT, but not in SRTT. Each 10-ppb increase in annual ozone was associated with increased SDST and SDLT scores by 0.16 (95%CI: 0.01, 0.23) and 0.56 (95%CI: 0.07, 1.05), equivalent to 3.5 and 5.3 years of aging-related decline in cognitive performance.

Conclusions: Our study provides the first epidemiological data supporting the adverse neurobehavioral effects of ambient air pollutants in adults.

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Review Article

Air Pollution, Oxidative Stress, and Alzheimer’s Disease

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Alzheimer’s disease (AD) is the most common form of dementia affecting millions of people worldwide and will continue to affect millions more with population aging on the rise. AD causality is multifactorial. Known causal factors include genetic predisposition, age, and sex. Environmental toxins such as air pollution (AP) have also been implicated in AD causation. Exposure to AP can lead to chronic oxidative stress (OS), which is involved in the pathogenesis of AD. Whereas AP plays a role in AD pathology, the epidemiological evidence for this association is limited. Given the significant prevalence of AP exposure combined with increased population aging, epidemiological evidence for this link is important to consider. In this paper, we examine the existing evidence supporting the relationship between AP, OS, and AD and provide recommendations for future research on the population level, which will provide evidence in support of public health interventions.
PROBLEMS WE NEED TO ADDRESS NOW
Fine particles in the air:
NASA satellite data
Deaths Attributable to Ambient PM$_{2.5}$ by Cause in the US, India, and China in 2010

Deaths Attributable to Ambient Particulate Matter Pollution in 2010

- **US**
  - Total attributable deaths = 103027
  - Pie Chart illustrating the distribution of attributable deaths for various causes.

- **India**
  - Total attributable deaths = 627426
  - Pie Chart illustrating the distribution of attributable deaths for various causes.

- **China**
  - Total attributable deaths = 1233891
  - Pie Chart illustrating the distribution of attributable deaths for various causes.

Slide attributed to Aaron Cohen, Health Effects Institute, Co-Chair of GBD 2010 Ambient Air Pollution Expert Group
IARC Scientific Publication No. 161
Air Pollution and Cancer
Editors: Kurt Straif, Aaron Cohen, and Jonathan Samet
Available at: http://www.iarc.fr/en/publications/books/sp161/index.php
Neighborhood Pollution: indoor and outdoor

China
Air pollution data stirs debate over holiday fireworks

Xinhua, February 11, 2013

Fewer fireworks have helped to decrease air pollution in some Chinese cities, but high readings of air pollutants have made many wonder if greater efforts need to be made.

An air quality index issued by municipal environmental authorities in Shanghai on Sunday showed a reading of 238 for PM2.5, or particulate matter less than 2.5 microns in diameter, indicating that the air was severely polluted.

In Beijing, the air quality was even worse, with the city's PM 2.5 density peaking at midnight to roughly 500 micrograms per cubic meter and subsequently easing to less than 300 micrograms per cubic meter.

Although the figure was dwarfed by last year's readings thanks to a mild north wind and the restrained use of fireworks, Beijing's environmental authorities has reported the clean negative impact of fireworks on air quality.

Zhang Dawei, director of the Beijing Municipal Environmental Monitoring Center, said the city’s PM2.5 readings started going up around 5 p.m. Saturday, when people started going outdoors to set off fireworks for Lunar New Year's Eve.

In Nanjing, capital of east China's Jiangsu Province, local environmental authorities urged local residents to refrain from setting off fireworks Saturday night.

Nanjing's daytime PM2.5 density reading stood at 39 micrograms per cubic meters on Saturday, representing fairly clean air in comparison to most other cities.
Traffic-Related Air Pollution
Climate change is the biggest global health threat of the 21st century.

Executive summary
Climate change is the biggest global health threat of the 21st century. Effects of climate change on health will affect most populations in the next decades and put the lives and wellbeing of billions of people at increased risk. During this century, Earth’s average surface temperature rises are likely to exceed the safe threshold of 2°C above preindustrial average temperature. Risks will be greater at higher latitudes, with medium-risk scenarios predicting 2–3°C rises by 2050 and 4–5°C rises in northern Canada, Greenland, and Siberia. In this report, we have outlined the major threats—both direct and indirect—to global health from climate change through changing patterns of disease, water and food insecurity, vulnerable shelter and human settlements, extreme climatic events, and population growth and migration. Although vector-borne diseases will expand their reach and death tolls, especially among elderly people, will increase because of heatwaves, the indirect effects of climate change on water, food security, and extreme climatic events are likely to have the biggest effect on global health.

A new advocacy and public health movement is needed urgently to bring together governments, international agencies, non-governmental organisations (NGOs), communities, and academics from all disciplines to adapt to the effects of climate change on health. Any adaptation should sit alongside the need for primary mitigation: reduction in greenhouse gas emissions, and the need to increase carbon biosequestration through reforestation and improved agricultural practices. The recognition by governments and electorates that climate change has enormous health implications should assist the advocacy and political change needed to tackle both mitigation and adaptation.

Management of the health effects of climate change will require inputs from all sectors of government and civil society, collaboration between many academic disciplines, and new ways of international cooperation that have hitherto eluded us. Involvement of local communities in monitoring, discussing, advocating, and assisting with the process of adaptation will be crucial. An integrated and multidisciplinary approach to reduce the adverse health effects of climate change requires at least three levels of action. First, policies must be adopted to reduce carbon emissions and to increase carbon biosequestration, and thereby slow down global warming and eventually stabilise temperatures. Second, action should be taken on the events linking climate change to disease. Third, appropriate public health systems should be put into place to deal with adverse outcomes.

While we must resolve the key issue of reliance on fossil fuels, we should acknowledge their contribution to large improvements in global health and development over the past 100 years. In the industrialised world and richer parts of the developing world, fossil fuel energy has contributed to a doubled longevity, dramatically
COULD THE LONDON FOG OF 1952 HAPPEN AGAIN?
Delhi

Credit: Jonathan M. Samet
Smog envelops city. Pollution to blame?

**HT Correspondent**

**NEW DELHI:** A thick blanket of smoke and haze sheathed Delhi on Saturday, reducing visibility to 500-800 metres.

While the meteorological department described this as a normal winter phenomenon, environmental groups ascribed it to rising pollution levels.

“This kind of smog is not unusual for this time of the year but such thick smog is also not seen frequently,” said S.C. Bhan, director, Indian Meteorological Department. The smog here had nothing to do with the oil depot fire in Jaipur, he added.

The Centre for Science and Environment (CSE), a research and advocacy organisation, however said Saturday’s “unprecedented smog” was a clear indicator Delhi had lost the gains of converting its buses and auto-rickshaws to Compressed Natural Gas (CNG). “The annual average level of pollution in Delhi has been going up since 2007 and is back to pre-2000 levels”, said Anumita Roychowdhury, Associate Director, CSE. The Met office said locally generated smoke and pollutants aren’t dispersing because there’s no wind. Such condition may continue till Sunday, the weatherman said.

**THICK WITH POLLUTION, P4**
Delhi, Kolkata have worst air quality in India: Report

Vishwa Mohan, TNN | Oct 19, 2013, 06:25 AM IST

NEW DELHI: With the World Health Organization’s (WHO) International Agency for Research on Cancer declaring air pollution as a major cause of cancer, its findings have put the focus on Indian hotspots like Delhi, West Bengal, Maharashtra and Jharkhand which showed high concentration of life-threatening air pollutants.

Air quality data of Central Pollution Control Board for 2010 shows that Kolkata and Delhi are among the worst affected cities in terms of air pollution.

In fact, figures obtained by this correspondent indicate what the world health body has already warned: outdoor air pollution is as bad as smoking tobacco, if not worse.

Quoting the ICMR, figures released during 2009-11 indicate that the prevalence of lung cancer was highest in Delhi, Mumbai and Kolkata.

Kolkata, Delhi and areas around these two cities have most polluted air in country.

Analysis of three pollutants (SO2, NO2 and particulate matter) at 450 air monitoring locations in 190 cities/towns across the country shows

One-third of urban population lives in cities/towns with PM10 levels classified as ‘critical’

Lung cancer cases peaked in ‘09-’11

Delhi, Jharkhand, West Bengal and Maharashtra have worst air quality

Half of urban population breathes air that exceeds the accepted standard of PM10

She added, “It's not that we can't do it. Taking the message from world to the Indian public, we need to immediately make Euro-IV norms in vehicles mandatory across the country as the worst transport in a big way to minimize vehicular air pollution on priority.”

One-third of urban population lives in cities/towns with PM10 levels classified as ‘critical’. The standards are not being met in 40 per cent of urban places across the country, and delinquent action to reverse the situation is required, she said.
Air pollution data stirs debate over holiday fireworks

Xinhua, February 11, 2013

Fewer fireworks have helped to decrease air pollution in some Chinese cities, but high readings of air pollutants have made many wonder if greater efforts need to be made.

An air quality index issued by municipal environmental authorities in Shanghai on Sunday showed a reading of 238 for PM2.5, or particulate matter less than 2.5 microns in diameter, indicating that the air was severely polluted.

In Beijing, the air quality was even worse, with the city's PM 2.5 density peaking at midnight to roughly 500 micrograms per cubic meter and subsequently easing to less than 300 micrograms per cubic meter.

Although the figure was dwarfed by last year's readings thanks to a mild north wind and the restrained use of fireworks, Beijing's environmental authorities have reported the clear negative impact of fireworks on air quality.

Zhang Dawei, director of the Beijing Municipal Environmental Monitoring Center, said the city's PM2.5 readings started going up around 5 p.m. Saturday, when people started going outdoors to set off fireworks for Lunar New Year's Eve.

In Nanjing, capital of east China's Jiangsu Province, local environmental authorities urged local residents to refrain from setting off fireworks Saturday night.

Nanjing's daytime PM2.5 density reading stood at 39 micrograms per cubic meters on Saturday, representing fairly clean air in comparison to most other cities.
Streets and buildings are seen under heavy smog in Harbin, northeast China's Heilongjiang province, on Oct. 21, 2013. (STR/AFP/Getty Images)
Mission China
Beijing

Beijing - PM2.5

Past 24-hour AQI was **Good**

**Most Recent AQI**
Mar 21, 2014 12 AM

25 AQI
Good

if at this level for 24 Hours

Concentration: 6.0 µg/m³

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The U.S. EPA has developed a formula to convert PM 2.5 readings into AQI scores.
March 14, 2014

Does Paris have worse air pollution than Beijing?

On Friday, levels of pollution in Paris were higher than in many of the world’s most notoriously polluted cities. With your help, Karl Mathiesen, investigates how the City of Light became the City of Smog.

Paris lies beneath a blanket of chemicals last week. For the first time since 1997, the French government has issued an alternating driving ban to deal with emergency levels of pollution. Photograph: Patrick Kovarik/AFP/Getty Images
High Levels of Pollution Spur Paris to Action

By ALISSA J. RUBIN  MARCH 14, 2014

A view of the Eiffel Tower seen through thick smog on Friday.
Credit Patrick Kovarik/Agence France-Presse — Getty Images
Do anti-pollution car bans actually work?
By Bryan Pirolli, for CNN
updated 1:23 AM EDT, Wed March 19, 2014

(CNN) -- Running along the banks of the Seine Sunday, I take a lap around the giant hot air balloon in the south of Paris hovering gently over the Parc André Citroen.

Essentially a giant weather balloon, its LEDs sparkled green -- Parisian air quality is just fine, a significant change from just two days before, Friday March 14, when the light flashed ominously red.

Friday's visibility levels were so bad that most Parisians couldn't even see the balloon -- let alone the Eiffel Tower.

Warmer temperatures and scarce winds caused particulate levels to reach record highs, spurring the city into action.

Paris' City Hall launched unprecedented policies aimed at reducing traffic in and around the city.

According to Laure Bencheik of the RATP, Paris's transit authority, for the first time ever public transportation including the metro, regional trains, trams and buses were all free.

Supplementary trains added 600,000 to 1 million seats across the various lines.

It cost the city around €4 million ($5.6 million) per day.
"Prediction is extremely difficult. Especially about the future."

- attributed to Niels Bohr
GLOBAL BURDEN OF DISEASE

Summary
Background Quantification of the disease burden caused by different risks informs prevention by providing an account of health loss different to that provided by a disease-by-disease analysis. No complete revision of global disease burden caused by risk factors has been done since a comparative risk assessment in 1990, and no previous analysis has assessed changes in burden attributable to risk factors over time.

Methods We estimated deaths and disability-adjusted life years (DALYs) for years lived with disability (YLD) and years of life lost (YLL) attributable to the independent effects of 67 risk factors and clusters of risk factors for 21 regions in 1990 and 2010. We estimated exposure distributions for each year, region, sex, and age group, and relative risks per unit of exposure by systematically reviewing and synthesising published and unpublished data. We used these estimates, together with estimates of cause-specific deaths and DALYs from the Global Burden of Disease Study 2010, to calculate the burden attributable to each risk factor exposure compared with the theoretical minimum-risk exposure. We incorporated uncertainty in disease burden, relative risks, and exposures into our estimates of attributable burden.

Findings In 2010, the three leading risk factors for global disease burden were high blood pressures (7·0% [95% uncertainty interval 6·2–7·7] of global DALYs), tobacco smoking including second-hand smoke (5·9% [5·1–6·7]), and alcohol use (5·3% [4·5–6·5]). In 1990, the leading risks were childhood underweight (7·5% [6·3–8·6]), household air pollution from solid fuels (6·4% [5·4–7·4]), and tobacco smoking including second-hand smoke (5·9% [5·0–6·8]). The burden of disease attributable to each risk factor increased between 1990 and 2010, with the most prominent dietary risks being diet in fruits and vegetables in southeast Asia, and fewest dietary risks being in high-income countries, reflecting a wide range of dietary intakes.
Estimating the Global Burden of Disease due to Ambient Air Pollution

Slide attributed to Aaron Cohen, Health Effects Institute, Co-Chair of GBD 2010 Ambient Air Pollution Expert Group
A model for estimating the global attributable burden: Integrated exposure-response function (IER)

- All cohort studies of PM$_{2.5}$ and mortality from chronic disease have been conducted in the US and Western Europe

- New models needed to estimate exposure-response functions at high levels of PM in Asia, other regions

- IERs estimate E-R functions using results of studies of second-hand smoke (SHS), household air pollution (HAP), and active tobacco smoking (ATS) (Burnett R et al. 2013 Submitted)

- Key model assumptions:
  - Risk is a function of PM$_{2.5}$ inhaled dose regardless of source (Pope et al. 2009; 2011)
  - Consistent with risk observed in current cohort studies
  - Predict risk for highest PM$_{2.5}$ concentrations consistent with risks from SHS, HAP, active smoking

From: Pope CA et al. EHP 2011

Slide attributed to Aaron Cohen, Health Effects Institute, Co-Chair of GBD 2010 Ambient Air Pollution Expert Group
• Global estimates of PM$_{2.5}$ at 10km x 10km scale

• Combined estimates from satellites (AOD), chemical transport models and ground-level measurements

• Estimates include contribution of all sources of PM$_{2.5}$
Exposure: Ambient PM Pollution

- Satellite-based measures of aerosol optical depth (AOD)
- TM5 chemical transport models
- Calibrated against ground-based PM$_{2.5}$ sensors

PM$_{2.5}$ (µg per m$^3$)
- 0 - 2.5
- 2.5 - 5
- 5 - 7.5
- 7 - 10
- 10 - 15
- 15 - 20
- 20 - 25
- 25 - 30
- 30 - 40
- 40 - 50
- 50 - 60
- 60 - 70
- 70 - 80
- 80+

Slide attributed to Stephen Lim, Global Burden of Disease Project, Institute for Health Metrics and Evaluation, University of Washington
Exposure - Ambient PM Pollution

Annual average PM2.5 (μg/m3)

- Measured
- Estimated from PM10

Slide attributed to Stephen Lim, Global Burden of Disease Project, Institute for Health Metrics and Evaluation, University of Washington
Burden of Disease Attributable to 20 Leading Risk Factors in 2010, % DALYs, both sexes

- High blood pressure
- Tobacco smoking, including second-hand smoke
- Alcohol use
- Household air pollution from solid fuels
- Diet low in fruits
- High body-mass index
- High fasting plasma glucose
- Childhood underweight
- Physical inactivity and low physical activity
- Diet high in sodium
- Diet low in nuts and seeds
- Iron deficiency
- Suboptimal breastfeeding
- High total cholesterol
- Diet low in whole grains
- Diet low in vegetables
- Diet low in seafood omega-3 fatty acids
- Drug use
- Occupational risk factors for injuries

Slide attributed to Stephen Lim, Global Burden of Disease Project, Institute for Health Metrics and Evaluation, University of Washington
### Regional variation in leading risks, 2010

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<th>Western Europe</th>
<th>Australia</th>
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</table>

Slide attributed to Stephen Lim, Global Burden of Disease Project, Institute for Health Metrics and Evaluation, University of Washington
Air Pollution Linked to 1.2 Million Premature Deaths in China

By EDWARD WONG

BEIJING — Outdoor air pollution contributed to 1.2 million premature deaths in China in 2010, nearly 40 percent of the global total, according to a new summary of data from a scientific study on leading causes of death worldwide.

Figured another way, the researchers said, China’s toll from pollution was the loss of 25 million healthy years of life from the population.

The data on which the analysis is based was first presented in the ambitious 2010 Global Burden of Disease Study, which was published in December in The Lancet, a British medical journal. The authors decided to break out numbers for specific countries and present the findings at international conferences. The China statistics were offered at a forum in Beijing on Sunday.

“We have been rolling out the India- and China-specific numbers, as they speak more directly to national leaders than regional numbers,” said Robert O’Keefe, the vice president of the Health Effects Institute, a research organization that is helping to present the study. The organization is partly financed by the United States Environmental Protection Agency and the global motor vehicle industry.

What the researchers called “ambient particulate matter pollution” was the fourth-leading risk factor for deaths in China in 2010, behind dietary risks, high blood pressure and smoking. Air pollution ranked seventh on the worldwide list of risk factors, contributing to 3.2 million deaths in 2010.

By comparison with China, India, which also has densely populated cities grappling with similar levels of pollution, had 620,000 premature deaths in 2010 because of outdoor air pollution, the study found. That was deemed to be the sixth most common killer in South Asia.

The study was led by an institute at the University of Washington and several partner universities and institutions, including the World Health Organization.

Calculations of premature deaths because of outdoor air pollution are politically threatening in the eyes of some Chinese officials. According to news reports, Chinese officials cut out sections of a 2007 report called “Cost of Pollution in China” that discussed premature deaths. The report’s authors had concluded that 350,000 to 400,000 people die prematurely in China each year because of outdoor air pollution. The study was done by the World Bank in cooperation with the Chinese State Environmental Protection Administration, the precursor to the Ministry of Environmental Protection.
Estimated population-weighted ambient air pollution levels - PM$_{2.5}$ increased worldwide and in China 1990-2010

1990 → 2010:
10% increase in global population-weighted PM$_{2.5}$

50% increase in population-weighted PM$_{2.5}$

Slide attributed to Aaron Cohen, Health Effects Institute, Co-Chair of GBD 2010 Ambient Air Pollution Expert Group
# Deaths Attributable to Air Pollution, Worldwide, 1990 and 2010

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
<th>Both sexes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambient PM pollution</td>
<td>1,549,448</td>
<td>1,850,428</td>
<td>1,360,712</td>
<td>1,373,113</td>
<td>2,910,161</td>
<td>3,223,540</td>
</tr>
<tr>
<td>Household air pollution from solid fuels</td>
<td>2,251,932</td>
<td>1,867,043</td>
<td>2,221,558</td>
<td>1,611,730</td>
<td>4,473,490</td>
<td>3,478,773</td>
</tr>
<tr>
<td>Ambient ozone pollution</td>
<td>77,087</td>
<td>86,335</td>
<td>66,274</td>
<td>66,100</td>
<td>143,362</td>
<td>152,434</td>
</tr>
</tbody>
</table>

*Lancet 2012; 380: 2224–60*
AIR POLLUTION AND CANCER: THE IARC CLASSIFICATION
Air Pollution and Cancer
Editors: Kurt Straif, Aaron Cohen, and Jonathan Samet
Available at: http://www.iarc.fr/en/publications/books/sp161/index.php
IARC Monograph meetings held subsequent to 2004

- Volume 93 (February 2006): Carbon black, titanium dioxide, and talc (IARC, 2010b).
- Volume 95 (October 2006): Household use of solid fuels and high-temperature frying (IARC, 2010c).
- Volume 105 (June 2012): Diesel and gasoline engine exhausts and some nitroarenes (IARC, 2013).
- Volume 109 (October 2013): Outdoor air pollution
IARC: Outdoor air pollution a leading environmental cause of cancer deaths

Lyon/Geneva, 17 October 2013 – The specialized cancer agency of the World Health Organization, the International Agency for Research on Cancer (IARC), announced today that it has classified outdoor air pollution as carcinogenic to humans (Group 1).  

After thoroughly reviewing the latest available scientific literature, the world’s leading experts convened by the IARC Monographs Programme concluded that there is sufficient evidence that exposure to outdoor air pollution causes lung cancer (Group 1). They also noted a positive association with an increased risk of bladder cancer.

Particulate matter, a major component of outdoor air pollution, was evaluated separately and was also classified as carcinogenic to humans (Group 1).

The IARC evaluation showed an increasing risk of lung cancer with increasing levels of exposure to particulate matter and air pollution. Although the composition of air pollution and levels of exposure can vary dramatically between locations, the conclusions of the Working Group apply to all regions of the world.

A major environmental health problem
Air pollution is already known to increase risks for a wide range of diseases, such as respiratory and heart diseases. Studies indicate that in recent years exposure levels have increased significantly in some parts of the world, particularly in rapidly industrializing countries with large populations. The most recent data indicate that in 2010, 223 000 deaths from lung cancer worldwide resulted from air pollution.
Air pollution is a leading cause of cancer, says WHO

More lawsuits expected after WHO links air pollution and cancer

Lawyers expect employees with work-related cancer to use WHO declaration of health hazard to build case on effects of being outdoors

Emily Tsang
emily.tsang@scmp.com

More workers are likely to sue their employers for work-related cancer, lawyers say, after the World Health Organisation officially classified outdoor air pollution as cancer-causing.

Lawyers Albert Luk Wai-hung and Vitus Leung Wing-hung both said the number of claims would surge as outdoor workers suffering from cancer related to air quality had a better chance of winning lawsuits and bigger payouts.

The WHO International Agency for Research on Cancer on Thursday classified outdoor air pollution as a leading cause of cancer. It is more dangerous even than second-hand tobacco smoke, according to the WHO.

The only way to address the problem is to improve air quality, they said.

The WHO International Agency for Research on Cancer classified outdoor air pollution as a leading cause of cancer. Photo: David Wong

Motorists travel through haze in Pekanbaru, Indonesia. Air pollution has been named as the main cause of lung cancer by the World Health Organisation

MAIN SOURCES OF OUTDOOR INDUSTRIAL AND AGRICULTURAL

In residential buildings
Lung Cancer and PM2.5 Meta-Analysis

<table>
<thead>
<tr>
<th>Study ID</th>
<th>Country</th>
<th>Relative Risk Estimate (RR) 95% CI</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hart et al. 2011 USA</td>
<td>1.18 (0.95, 1.48)</td>
<td>1.97</td>
<td></td>
</tr>
<tr>
<td>Hystad et al. 2013 Canada</td>
<td>1.29 (0.95, 1.76)</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>Jerrett et al. 2013 USA</td>
<td>1.12 (0.91, 1.37)</td>
<td>2.41</td>
<td></td>
</tr>
<tr>
<td>Krewski et al. 2009 USA</td>
<td>1.09 (1.05, 1.13)</td>
<td>75.26</td>
<td></td>
</tr>
<tr>
<td>Lepeule et al. 2012 USA</td>
<td>1.37 (1.07, 1.75)</td>
<td>1.63</td>
<td></td>
</tr>
<tr>
<td>Lipsett et al. 2011 USA</td>
<td>0.95 (0.70, 1.28)</td>
<td>1.08</td>
<td></td>
</tr>
<tr>
<td>McDonell et al. 2000 USA</td>
<td>1.39 (0.79, 2.46)</td>
<td>0.30</td>
<td></td>
</tr>
<tr>
<td>Pope et al. 2002 USA</td>
<td>1.14 (1.05, 1.24)</td>
<td>14.02</td>
<td></td>
</tr>
<tr>
<td>Turner et al. 2011 USA</td>
<td>1.19 (0.97, 1.46)</td>
<td>2.28</td>
<td></td>
</tr>
<tr>
<td>Subtotal (I-squared = 0.0%, p = 0.509)</td>
<td>1.11 (1.07, 1.14)</td>
<td>100.00</td>
<td></td>
</tr>
<tr>
<td>Europe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beelen et al., 2008b NL</td>
<td>1.06 (0.82, 1.38)</td>
<td>2.52</td>
<td></td>
</tr>
<tr>
<td>Carey et al. 2013 UK</td>
<td>1.11 (0.86, 1.43)</td>
<td>2.69</td>
<td></td>
</tr>
<tr>
<td>Cesaroni et al. 2013 Italy</td>
<td>1.05 (1.01, 1.10)</td>
<td>93.84</td>
<td></td>
</tr>
<tr>
<td>Raaschou-Nielsen et al 2013 EU</td>
<td>1.39 (0.91, 2.13)</td>
<td>0.94</td>
<td></td>
</tr>
<tr>
<td>Subtotal (I-squared = 0.0%, p = 0.607)</td>
<td>1.05 (1.01, 1.10)</td>
<td>100.00</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cao et al. 2011 China</td>
<td>1.03 (1.00, 1.07)</td>
<td>53.50</td>
<td></td>
</tr>
<tr>
<td>Kataneda et al. 2011 Japan</td>
<td>1.24 (1.12, 1.37)</td>
<td>46.50</td>
<td></td>
</tr>
<tr>
<td>Subtotal (I-squared = 91.0%, p = 0.001)</td>
<td>1.13 (0.94, 1.34)</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Weights are from random effects analysis
Figure 1. The empirical relationship between the mutagenic potency of organic extracts of atmospheric PM in TA98 without exogenous metabolic activation and atmospheric PM level. The data represent observations from 26 countries on 5 continents. All values were log transformed to equalize the variance over the range of observations.

\[ r^2 = 0.08, F \text{ ratio} = 40.0 \]
\[ n = 466, p < 0.0001, \text{RMSE} = 0.54 \]
Figure 2. The empirical relationship between the mutagenic potency of organic extracts of atmospheric PM in TA98 with exogenous metabolic activation and atmospheric PM level. The data represent observations from 26 countries on 5 continents. All values were log transformed to equalize the variance over the range of observations.

\[ r^2 = 0.10, F\text{ ratio} = 56.8 \]

\[ n = 501, p < 0.0001, \text{RMSE} = 0.54 \]
THE PROBLEM OF TRAFFIC
Traffic-Related Air Pollution
China: Increasing numbers of vehicles

Civil vehicle fleet (per 10000)

1978-2007

Private vehicle fleet (per 10000)

1990-2007

REGULATING AIR QUALITY
The Evidence Scale

Act

Evidence

Uncertainty

Not Act

Politics

Costs

Activists

Advocates
The National Ambient Air Quality Standards (NAAQS) review process

- **Peer-reviewed scientific studies**
- **Workshop on science-policy issues**
- **Integrated Review Plan**
  - Timeline and key policy-relevant issues and scientific questions
- **CASAC review and public comment**
- **Integrated Science Assessment (ISA)**
  - Concise evaluation and synthesis of most policy-relevant studies
- **Risk/Exposure Assessment (REA)**
  - Concise qualitative assessment focused on key results, observations, and uncertainties
- **CASAC review and public comment**
- **Policy Assessment**
  - Staff analysis of policy options based on integration and interpretation of information in the ISA and REA
- **Agency decision making and draft proposal notice**
- **Interagency review**
- **Agency decision making and draft final notice**
- **Public hearings and comments on proposal**
- **EPA proposed decision on standards**
- **EPA final decision on standards**
The NAAQS - getting better all the time

Bachmann 2007;57:652-97
The PM Dilemma

• In higher-income countries, current PM levels are still associated with adverse health effects.

• And, in low- and middle-income countries, PM is present at concentrations above EPA NAAQS and WHO guidelines.
Annual Average PM$_{2.5}$ levels ($\mu$g/m$^3$)

PM$_{2.5}$ Ranges in Epi studies

- Delhi, India
- Beijing, China
- Hong Kong
- Los Angeles, US
- ACS Cancer Prevention Study
- Six Cities Study
- ESCAPE

0 10 20 30 40 50 150
Air Quality Guidelines

Global Update 2005

Particulate matter, ozone, nitrogen dioxide and sulfur dioxide
WHO AQG: Particulate Matter

Guidelines

\[ \text{PM}_{2.5}: \begin{align*} &10 \, \mu g/m^3 \text{ annual mean} \\ &25 \, \mu g/m^3 \text{ 24-hour mean} \end{align*} \]

\[ \text{PM}_{10}: \begin{align*} &20 \, \mu g/m^3 \text{ annual mean} \\ &50 \, \mu g/m^3 \text{ 24-hour mean} \end{align*} \]

Proportion of Ischemic Heart Disease DALYs Attributable to Individual Risk Factors, 2010

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Disability-adjusted life-years (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological risk factors</td>
<td></td>
</tr>
<tr>
<td>High blood pressure</td>
<td>53%</td>
</tr>
<tr>
<td>High total cholesterol</td>
<td>29%</td>
</tr>
<tr>
<td>High body-mass index</td>
<td>23%</td>
</tr>
<tr>
<td>High fasting plasma glucose</td>
<td>16%</td>
</tr>
<tr>
<td>Alcohol use</td>
<td>5%</td>
</tr>
<tr>
<td>Tobacco smoking, including second-hand smoke</td>
<td>31%</td>
</tr>
<tr>
<td>Dietary risk factors and physical inactivity</td>
<td></td>
</tr>
<tr>
<td>Diet low in nuts and seeds</td>
<td>40%</td>
</tr>
<tr>
<td>Physical inactivity and low physical activity</td>
<td>31%</td>
</tr>
<tr>
<td>Diet low in fruits</td>
<td>30%</td>
</tr>
<tr>
<td>Diet low in seafood omega-3 fatty acids</td>
<td>22%</td>
</tr>
<tr>
<td>Diet low in whole grains</td>
<td>17%</td>
</tr>
<tr>
<td>Diet high in sodium</td>
<td>17%</td>
</tr>
<tr>
<td>Diet high in processed meat</td>
<td>13%</td>
</tr>
<tr>
<td>Diet low in vegetables</td>
<td>12%</td>
</tr>
<tr>
<td>Diet low in fibre</td>
<td>11%</td>
</tr>
<tr>
<td>Diet low in polyunsaturated fatty acids</td>
<td>9%</td>
</tr>
<tr>
<td>Diet high in trans fatty acids</td>
<td>9%</td>
</tr>
<tr>
<td>Diet high in sugar-sweetened beverages</td>
<td>2%</td>
</tr>
<tr>
<td>Air pollution</td>
<td></td>
</tr>
<tr>
<td>Ambient particulate matter pollution</td>
<td>22%</td>
</tr>
<tr>
<td>Household air pollution from solid fuels</td>
<td>18%</td>
</tr>
<tr>
<td>Other environmental risks</td>
<td></td>
</tr>
<tr>
<td>Lead exposure</td>
<td>4%</td>
</tr>
</tbody>
</table>

Table 2: Proportion of ischaemic heart disease disability-adjusted life-years attributable to individual risk factors, worldwide, 2010

*Lancet 2012; 380: 2224–60*
Exposure to particulate matter with an aerodynamic diameter of 10 μm or less (PM10) in urban areas*, 2003–2010

* The mean annual concentration of fine suspended particles of less than 10 microns in diameters is a common measure of air pollution. The mean is a population-weighted average for urban population in cities above 100,000 inhabitants of a country.

http://www.who.int/gho/phe/outdoor_air_pollution/burden/en/
Exposure to particulate matter with an aerodynamic diameter of 10 μm or less (PM10) in 1100 urban areas*, 2003–2010

Annual mean PM10 (ug/m3)
- <20
- 20–29
- 30–49
- 50–99
- 100–149
- ≥150
- Not applicable

* The mean annual concentration of fine suspended particles of less than 10 microns in diameters is a common measure of air pollution. The mean is a population-weighted average for urban population in cities above 100,000 inhabitants of a country.

Data Source: World Health Organization
Map Production: Public Health Information and Geographic Information Systems (GIS)
World Health Organization

http://www.who.int/gho/phe/outdoor_air_pollution/burden/en/
Air pollution data stirs debate over holiday fireworks

Xinhua, February 11, 2013

Fewer fireworks have helped to decrease air pollution in some Chinese cities, but high readings of air pollutants have made many wonder if greater efforts need to be made.

An air quality index issued by municipal environmental authorities in Shanghai on Sunday showed a reading of 238 for PM2.5, or particulate matter less than 2.5 microns in diameter, indicating that the air was severely polluted.

In Beijing, the air quality was even worse, with the city's PM 2.5 density peaking at midnight to roughly 500 micrograms per cubic meter and subsequently easing to less than 300 micrograms per cubic meter.

Although the figure was dwarfed by last year's readings thanks to a mild north wind and the restrained use of fireworks, Beijing's environmental authorities has reported the clear negative impact of fireworks on air quality.

Zhang Dawei, director of the Beijing Municipal Environmental Monitoring Center, said the city's PM2.5 readings started going up around 5 p.m. Saturday, when people started going outdoors to set off fireworks for Lunar New Year's Eve.

In Nanjing, capital of east China's Jiangsu Province, local environmental authorities urged local residents to refrain from setting off fireworks Saturday night.

Nanjing's daytime PM2.5 density reading stood at 39 micrograms per cubic meters on Saturday, representing fairly clean air in comparison to most other cities.
WHO GUIDELINES FOR INDOOR AIR QUALITY

SELECTED POLLUTANTS
<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Critical outcome(s) for guideline definition</th>
<th>Guidelines</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>• Acute myeloid leukemia (sufficient evidence on causality)</td>
<td>• No safe level of exposure can be recommended</td>
<td>The concentrations of airborne benzene associated with an excess lifetime risk of 1/10000, 1/100000 and 1/1000000 are 17, 1.7 and 0.17 μg/m³, respectively</td>
</tr>
<tr>
<td></td>
<td>• Genotoxicity</td>
<td>• Unit risk of leukemia per 1 μg/m³ air concentration is 6.6 × 10⁻¹².</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute exposure-related reduction of exercise tolerance and increase in symptoms of ischemic heart disease (e.g. ST-segment changes)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute respiratory effects</td>
<td>• 15 minutes – 100 mg/m³</td>
<td>The guideline (valid for any 30-minute period) will also prevent effects on lung function as well as non-hodgkin lymphoma and myeloid leukemia</td>
</tr>
<tr>
<td></td>
<td>Sensory irritation</td>
<td>• 1 hour – 35 mg/m³</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Respiratory tract lesions leading to inflammation and malignancy in animal studies</td>
<td>• 8 hours – 10 mg/m³</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Respiratory symptoms, bronchoconstriction, increased bronchial reactivity, airway inflammation and decreases in immune defence, leading to increased susceptibility to respiratory infection</td>
<td>• 24 hours – 7 mg/m³</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sensory irritation</td>
<td>0.1 mg/m³ – 30-minute average</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Respiratory tract lesions leading to inflammation and malignancy in animal studies</td>
<td>0.01 mg/m³ – annual average</td>
<td>The long-term guideline is also assumed to prevent potential malignant effects in the airways</td>
</tr>
<tr>
<td></td>
<td>Respiratory symptoms, bronchoconstriction, increased bronchial reactivity, airway inflammation and decreases in immune defence, leading to increased susceptibility to respiratory infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>200 μg/m³ – 1 hour average</td>
<td></td>
<td>No evidence for exposure threshold from epidemiological studies</td>
</tr>
<tr>
<td></td>
<td>40 μg/m³ – annual average</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lung cancer</td>
<td></td>
<td>Bis(paraben) is taken as a marker of the PAH mixture</td>
</tr>
<tr>
<td>Poly cyclic aromatic hydrocarbons</td>
<td>Lung cancer</td>
<td></td>
<td>Bis(paraben) is taken as a marker of the PAH mixture</td>
</tr>
<tr>
<td></td>
<td>Suggestive evidence of an association with other cancers, in particular leukemia and cancers of the extrathoracic airways</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>The excess lifetime risk of death from radon-induced lung cancer (by the age of 75 years) is estimated to be 0.6 × 10⁻¹² per Bq/m³ for lifelong non-smokers and 1.3 × 10⁻¹² per Bq/m³ for current smokers (5–24 cigarettes per day); among ex-smokers, the risk is intermediate, depending on time since smoking cessation</td>
<td>WHO guidelines provide a comprehensive approach to the management of health risk related to radon</td>
<td></td>
</tr>
<tr>
<td></td>
<td>The radon concentrations associated with an excess lifetime risk of 1/100 and 1/1000 are 67 and 6.7 Bq/m³ for current smokers and 1570 and 167 Bq/m³ for lifelong non-smokers, respectively</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radon</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Trichloroethylene</td>
<td>Carcinogenicity (liver, kidney, bile-duct and non-Hodgkin’s lymphoma), with the assumption of genotoxicity</td>
<td>• Unit risk estimate of 4.3 × 10⁻⁵ per μg/m³</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• The concentrations of airborne trichloroethylene associated with an excess lifetime cancer risk of 1/10000, 1/100000 and 1/1000000 are 230, 23 and 2.3 μg/m³, respectively</td>
<td></td>
</tr>
<tr>
<td>Tetrachloroethylene</td>
<td>Effects in the kidney indicative of early renal disease and impaired performance</td>
<td>0.25 mg/m³ – annual average</td>
<td>Carcinogenicity is not used as an endpoint as there are no indications that tetrachloroethylene is genotoxic and there is uncertainty about the epidemiological evidence and the relevance to humans of the animal carcinogenic data</td>
</tr>
</tbody>
</table>