Important Knowledge Gaps in Studying Health Effects of Traffic Exposure

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Air pollution is killing us.

Death from air pollution would be cut if UK hits walking and cycling targets
4 Dec 2017

Banning bikes from Oxford Street is a disaster for London cycling
Andrew Gilligan
7 Nov 2017

London's £10 T-charge comes into effect in fight against toxic car fumes
23 Oct 2017

Oxford Street could become 'traffic-free boulevard' next year
6 Nov 2017
Traffic-related air pollution
Exposure considerations

- Traffic-related air pollution (TRAP) exposure refers to exposure to primary (tailpipe and non-tailpipe) emissions from motor vehicles, not to the more broadly dispersed secondary pollutants such as ozone ($O_3$) or secondary organic aerosols (SOAs) that are derived from these emissions.
Exposure considerations

Exposure assessment of TRAP is challenging

- TRAP is characterized by high spatial and temporal variability.
- No exposure metric identified yet that uniquely indicates TRAP.
Exposure considerations

- Commonly used exposure metrics are measured or modeled concentrations of single pollutants considered indicators of TRAP (e.g., BC, NO$_2$), and simple indicators of traffic (e.g., distance to roadways/traffic density at nearest road).
Exposure considerations

Karner et al., 2010
• **Direct brake wear**: the fraction of pad, disc, and clutch wear particles that are directly airborne.

• **Direct tire wear**: the fraction of tire wear particles (TWPs) that are directly airborne.

• **Direct road wear**: the fraction of road wear particles (RWP) that are directly airborne.

• **Road dust suspension**: any particle on (paved) road surface that is suspended to air by vehicles or wind, including deposited brake/tire/road wear particles and other deposited.
The context of new studies on health effects of traffic-related air pollution

- Change in air pollution levels and general decrease of emissions
- **Change in population structure and mobility**
  - More elderly and fewer children in some cities
  - Migration of young adults
  - Migration of populations with different ethnic background
- **Changes in urban structure**
  - Low emission zones and congestion charges
  - More green areas
  - Inner cities versus outskirts (change in SES)
- **Change in traffic composition**
  - Larger vehicles, congestion
  - Changes in public transport
  - Bike sharing and bike lanes
  - Car sharing
Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects

HEI Panel on the Health Effects of Traffic-Related Air Pollution
Summary of the evidence HEI 2010

- «Sufficient evidence to infer a causal association»
  - Exacerbation of respiratory symptoms in children with asthma

- Between «Sufficient» and «Suggestive but not Sufficient»
  - Asthma incidence in children
  - Asthma prevalence in children

- «Suggestive but not Sufficient to infer a causal association»
  - All cause mortality
  - Cardiovascular morbidity
  - Exacerbation of respiratory symptoms in adults
  - Lung Function

- «Inadequate and Insufficient to infer a causal association»
  - Exacerbation of respiratory symptoms in children without asthma
  - Health care utilization for respiratory problems in children
  - Asthma in adults
  - Health care utilization for respiratory problems in adults
  - Chronic obstructive pulmonary disease (COPD)
  - Non-asthmatic respiratory allergy
  - Childhood cancer
  - Lung cancer and other cancers in adults
“The cumulative body of evidence indicates that short-term exposure to NO₂ can cause respiratory effects, in particular, effects related to asthma exacerbation. Recent results also strengthen the evidence that the respiratory effects of short-term NO₂ exposure are independent of the effects of many other traffic-related pollutants. There is now stronger evidence for a relationship between long-term exposure to NO₂ and respiratory effects, particularly the development of asthma in children. Results suggest that short-term exposure to NO₂ may be associated with cardiovascular effects and premature mortality and that long-term exposure may be associated with cardiovascular effects, diabetes, poorer birth outcomes, premature mortality, and cancer; however, it is uncertain whether NO₂ exposure has an effect on these health outcomes that is independent from the effects of other traffic-related pollutants”.
NO$_2$ per se….or…. not per se
A Systematic Review on the Health Effects from Traffic-Related Air Pollution

• To systematically evaluate the evidence for the associations of exposure to traffic-related air pollution with adverse human health outcomes (up to mid-2019).

• Results will be quantitatively combined, where appropriate, which may be useful for future risk and health impact assessments of traffic-related air pollution.

• The focus of the systematic review is the epidemiologic literature, especially on long-term effects.

• Non-tailpipe PM emissions will be specifically addressed, as well as the influence of traffic noise on traffic-related air pollution associations.
ERS / ATS: Statement on Adverse Health Effects (Thurston et al, 2017)

- Respiratory Disease Mortality
- Respiratory Disease Morbidity
- Lung Cancer
- Pneumonia
  - Upper and lower respiratory symptoms
  - Airway inflammation
  - Decreased lung function
  - Decreased lung growth
- Insulin Resistance
- Type 2 diabetes
- Type 1 diabetes
- Bone metabolism
- High blood pressure
  - Endothelial dysfunction
  - Increased blood coagulation
  - Systemic inflammation
  - Deep Venous Thrombosis
- Stroke
- Neurological development
- Mental Health
- Neurodegenerative diseases
- Cardiovascular Disease Mortality
- Cardiovascular Disease Morbidity
- Myocardial Infarction
- Arrhythmia
- Congestive Heart Failure
- Changes in Heart Rate Variability
- ST-Segment Depression
- Skin Aging
- Premature Birth
  - Decreased Birth Weight
  - Decreased foetal growth
  - In uterine growth retardation
  - Decreased sperm quality
  - Preclampsia

ERS = European Respiratory Society
ATS = American Thoracic Society
Selected examples of recent studies

- Mortality long-term effects
- Mortality short-term effects
- Respiratory symptoms in adults
- Lung function in children
- Respiratory symptoms in children
- Neurological degenerative diseases
- Diabetes
- Low-birth weight
- Cognitive function in children
Long-term effects on mortality, Ostro 2015

Associations of Mortality with Long-Term Exposures to Fine and Ultrafine Particles, Species and Sources: Results from the California Teachers Study Cohort

Bart Ostro, Jianlin Hu, Debbie Goldberg, Peggy Reynolds, Andrew Hertz, Leslie Bernstein, and Michael J. Kleeman

Figure 2. Association of PM$_{2.5}$ constituents and sources with IHD mortality (HRs and 95% CIs using inter-quartile range. Abbreviations: comb, combustion; comp, components.
The strongest positive associations were for EC and BC adjusted for particle mass and respiratory mortality, 2.66% (95% confidence interval: 0.11, 5.28) and 2.72% (0.09, 5.42) per 0.8 and 1.0 μg/m³, respectively. These associations were robust to adjustment for other traffic metrics and regional pollutants, suggesting a degree of specificity with respiratory mortality and diesel exhaust containing EC/BC.

EC = elemental carbon
BC = black carbon
Results for particulate matter (PM) components were adjusted for PM mass.
Short-term exposure to traffic pollution prevents the beneficial cardiopulmonary effects of walking.
Lifetime Exposure to Ambient Pollution and Lung Function in Children, Rice et al, AJRCCM 2016

“similar effects for FEV1 and FVC, i.e. a restrictive pattern”

FEV1 = Forced expiratory volume in one second; FVC = forced vital capacity;
AJRCCM = American Journal of Respiratory and Critical Care Medicine
Changes in mean NO₂ and changes in prevalence of bronchitic symptoms in children with asthma and without asthma.
Dementia – Chen 2017

Living near major roads and the incidence of dementia, Parkinson’s disease, and multiple sclerosis: a population-based cohort study


<table>
<thead>
<tr>
<th>Distance† by category</th>
<th>Incidence of dementia (n=243611)#</th>
<th>Incidence of Parkinson’s disease (n=31577)#</th>
<th>Incidence of multiple sclerosis (n=9247)#</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>95% CI</td>
<td>Pval</td>
</tr>
<tr>
<td>&lt;50 m</td>
<td>1.07</td>
<td>1.06-1.08</td>
<td>0.0349</td>
</tr>
<tr>
<td>50-100 m</td>
<td>1.04</td>
<td>1.02-1.05</td>
<td>.</td>
</tr>
<tr>
<td>101-200 m</td>
<td>1.02</td>
<td>1.01-1.03</td>
<td>.</td>
</tr>
<tr>
<td>201-300 m</td>
<td>1.00</td>
<td>0.99-1.01</td>
<td>.</td>
</tr>
<tr>
<td>&gt;300 m</td>
<td>Reference</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>Log(distance)$</td>
<td>0.91</td>
<td>0.89-0.93</td>
<td>.</td>
</tr>
</tbody>
</table>

Cox proportional hazards model with age as the time-scale, stratified by an indicator for living in the Greater Toronto Area or not, adjusted for sex, history of diabetes, hypertension, coronary heart disease, stroke, congestive heart failure, arrhythmia, and traumatic brain injury, income quintile, urban/rural indicator, census subdivision-level unemployment rate, education, recent immigrants, as well as the subtraction of these variables at the census dissemination level from their census division. For multiple sclerosis, the model was also adjusted for latitude. †Major traffic roads include primary urban roads and arterial roads whereas highways include expressways and primary and secondary highways, as defined by Ontario Government Road Network Data Standards. §Incidence of dementia and Parkinson’s disease was assessed among all adults aged 55–85 years (dementia/Parkinson’s disease cohort) whereas incidence of multiple sclerosis was assessed among all adults aged 20–50 years (multiple sclerosis cohort). $Distance was fitted as a continuous variable, using natural logarithm of distance. Hazard ratios (HRs) expressed per IQR increase in distance (dementia/Parkinson’s disease cohort: 310 m and multiple sclerosis cohort: 320 m).

Table 2: Hazard ratios and 95% CIs for the associations between residential proximity to major roadways in 1996 and the risks of incident dementia, Parkinson’s disease, and multiple sclerosis in Ontario, during the follow-up period 2001-12.
Exposure to Ambient Ultrafine Particles and Nitrogen Dioxide and Incident Hypertension and Diabetes

Li Bai, Hong Chen, Marianne Hatzopoulou, Michael Jerrett, Jeffrey C. Kwong, Richard T. Burnett, Ray Copes, Randall V. Martin, Keith Van Ryswyk, Hong Lu, Alexander Kopp, and Scott Weichenthal

Table 3. HRs and 95% CIs for the Associations of Incident Hypertension and Diabetes with Long-term Exposure to Ultrafine Particles and NO$_2$ Using IQR$_w$ Increases and Quintiles of Exposures

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Hypertension HR (95% CI)</th>
<th>Diabetes HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ultrafine particles (per IQR$_w$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stratified by age and sex</td>
<td>1.04 (1.03, 1.05)</td>
<td>1.09 (1.08, 1.11)</td>
</tr>
<tr>
<td>Adjusted for medical comorbidities$^a$</td>
<td>1.04 (1.03, 1.05)</td>
<td>1.09 (1.07, 1.10)</td>
</tr>
<tr>
<td>Adjusted for neighborhood-level covariates$^b$</td>
<td>1.03 (1.02, 1.04)</td>
<td>1.06 (1.05, 1.08)</td>
</tr>
<tr>
<td>Adjusted for PM$_{2.5}$</td>
<td>1.03 (1.02, 1.04)</td>
<td>1.06 (1.05, 1.08)</td>
</tr>
<tr>
<td>Adjusted for NO$_2$</td>
<td>1.03 (1.02, 1.04)</td>
<td>1.04 (1.02, 1.05)</td>
</tr>
<tr>
<td>Adjusted for PM$_{2.5}$ and NO$_2$</td>
<td>1.03 (1.02, 1.04)</td>
<td>1.04 (1.02, 1.05)</td>
</tr>
<tr>
<td>Ultrafine particles (by quintiles)$^c$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Q2</td>
<td>1.01 (1.00, 1.03)</td>
<td>1.02 (1.00, 1.04)</td>
</tr>
<tr>
<td>Q3</td>
<td>1.04 (1.02, 1.05)</td>
<td>1.07 (1.05, 1.09)</td>
</tr>
<tr>
<td>Q4</td>
<td>1.03 (1.02, 1.05)</td>
<td>1.10 (1.08, 1.13)</td>
</tr>
<tr>
<td>Q5</td>
<td>1.04 (1.02, 1.06)</td>
<td>1.10 (1.07, 1.13)</td>
</tr>
<tr>
<td>NO$_2$ (per IQR$_w$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stratified by age and sex</td>
<td>1.03 (1.02, 1.03)</td>
<td>1.10 (1.09, 1.11)</td>
</tr>
<tr>
<td>Adjusted for medical comorbidities$^a$</td>
<td>1.02 (1.02, 1.03)</td>
<td>1.09 (1.08, 1.10)</td>
</tr>
<tr>
<td>Adjusted for neighborhood-level covariates$^b$</td>
<td>1.01 (1.00, 1.02)</td>
<td>1.06 (1.05, 1.07)</td>
</tr>
<tr>
<td>Adjusted for ultrafine particles</td>
<td>1.00 (0.99, 1.01)</td>
<td>1.05 (1.04, 1.06)</td>
</tr>
<tr>
<td>Adjusted for PM$_{2.5}$</td>
<td>1.01 (1.00, 1.02)</td>
<td>1.06 (1.05, 1.07)</td>
</tr>
<tr>
<td>Adjusted for ultrafine particles and PM$_{2.5}$</td>
<td>1.00 (0.99, 1.01)</td>
<td>1.05 (1.04, 1.06)</td>
</tr>
<tr>
<td>NO$_2$ (by quintiles)$^c$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Q2</td>
<td>1.01 (1.00, 1.02)</td>
<td>1.02 (1.00, 1.04)</td>
</tr>
<tr>
<td>Q3</td>
<td>1.03 (1.02, 1.04)</td>
<td>1.08 (1.05, 1.10)</td>
</tr>
<tr>
<td>Q4</td>
<td>1.04 (1.02, 1.05)</td>
<td>1.11 (1.09, 1.13)</td>
</tr>
<tr>
<td>Q5</td>
<td>1.02 (1.00, 1.04)</td>
<td>1.16 (1.13, 1.18)</td>
</tr>
</tbody>
</table>

*For hypertension, we adjusted for comorbid diabetes, COPD, asthma, and never smoking; for diabetes, we adjusted for age, sex, education, family history of diabetes, and smoking status.

Editorial:
Too much TRAFFIC at the crossroads of diabetes and endothelial dysfunction
Zsolt Bagi, 2017
Impact of London's road traffic air and noise pollution on birth weight: retrospective population based cohort study

What this study adds

- There is an increased risk of LBW specifically in relation to the air pollution profile of London.
- Exposure to local air pollution from road traffic is associated with increased risk of LBW in London, but there is little evidence for an independent exposure-response effect of traffic related noise on birth weight.
- Reducing exposure to traffic related air pollution could reduce the burden of LBW, SGA, and subsequent morbidity, and ultimately give babies in urban environments a healthier start in life.

Rachel B Smith research associate\textsuperscript{1,2}, Daniela Fecht research fellow\textsuperscript{3}, John Gulliver senior lecturer\textsuperscript{1}, Sean D Beevers senior lecturer\textsuperscript{4}, David Dajnak deputy manager\textsuperscript{4}, Marta Blangiardo senior lecturer\textsuperscript{1}, Rebecca E Ghosh research associate\textsuperscript{3}, Anna L Hansell assistant director\textsuperscript{23}, Frank J Kelly professor\textsuperscript{24}, H Ross Anderson emeritus professor\textsuperscript{45}, Mireille B Toledano reader\textsuperscript{1,2}
Fig 2. Working memory development by high- or low-traffic-air-pollution school.

http://journals.plos.org/plosmedicine/article?id=info:doi/10.1371/journal.pmed.1001792
Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children


Table 3. Fully Adjusted Association Between Air Pollution Exposure During Fetal Life and Cortical Thickness at 6–10 Years of Age

<table>
<thead>
<tr>
<th>Hemisphere</th>
<th>Size Brain Region (mm²)</th>
<th>Coefficient</th>
<th>95% CI</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fine Particles Exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Precuneus region</td>
<td>Right</td>
<td>936</td>
<td>-0.045</td>
<td>-0.062 to -0.028</td>
</tr>
<tr>
<td>Pars opercularis region</td>
<td>Right</td>
<td>753</td>
<td>-0.024</td>
<td>-0.033 to -0.014</td>
</tr>
<tr>
<td>Pars orbitalis region</td>
<td>Right</td>
<td>651</td>
<td>-0.028</td>
<td>-0.043 to -0.012</td>
</tr>
<tr>
<td>Rostral middle frontal region</td>
<td>Right</td>
<td>2995</td>
<td>-0.029</td>
<td>-0.041 to -0.018</td>
</tr>
<tr>
<td>Superior frontal region</td>
<td>Right</td>
<td>722</td>
<td>-0.029</td>
<td>-0.043 to -0.016</td>
</tr>
<tr>
<td>Cuneus region</td>
<td>Left</td>
<td>843</td>
<td>-0.022</td>
<td>-0.035 to -0.009</td>
</tr>
<tr>
<td>Coarse Particles Exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lateral orbitofrontal region</td>
<td>Right</td>
<td>565</td>
<td>-0.037</td>
<td>-0.059 to -0.016</td>
</tr>
<tr>
<td>Absorbance of Fine Particles Exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fusiform region</td>
<td>Left</td>
<td>532</td>
<td>-0.105</td>
<td>-0.160 to -0.049</td>
</tr>
</tbody>
</table>

CI, confidence interval.

*Beta coefficient (95% CI) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indices and heights, maternal parity, family status, maternal psychological distress, maternal IQ, and child gender, age, and genetic ancestry. Coefficients represent the differences in thickness (mm) per each increase of 5 µg/m³ of fine particles, 5 µg/m³ of coarse particles, and 10⁻⁵ m⁻³ of absorbance of fine particles.
An excess risk of ischemic heart disease was observed among subjects aged less than 50 years (SMR = 1.63), corresponding to workers with a short duration of employment and a short latency since first employment.

Air pollution, Noise or Stress?
Relationship between traffic air pollution and health

TRAFFIC

- Strain-stress
- Noise
- TRAP

- Stress-related mechanisms
- Inflammation

- Green areas

Vulnerability, eg. SES, pre-existing diseases

Health effects
Fig. 2. Spatial distribution of noise ($L_{Aeq,24h}$) and air pollution ($PM_{2.5traffic}$) levels in central London. Estimates are for postcode centroids, median across the study period 2003–2010. Graphs show $L_{Aeq,24h}$ (left) and $PM_{2.5traffic}$ (right) levels across a tr...

Daniela Fecht, Anna L. Hansell, David Morley, David Dajnak, Danielle Vienneau, Sean Beevers, Mireille B. Toledano, Frank J. Kelly, H. Ross Anderson, John Gulliver

**Spatial and temporal associations of road traffic noise and air pollution in London: Implications for epidemiological studies**
Environment International, Volume 88, 2016, 235–242
http://dx.doi.org/10.1016/j.envint.2015.12.001
Air pollution and noise

- Separating effects of air pollution and noise.
- Meteorology more associated with air pollution than noise.
- Noise varies less day-to-day than air pollution.
- Differences in dispersion: Quiet sides of buildings, barriers may affect noise exposure more than air pollution.
- There may be different biological pathways for noise and air pollution effects.
Green Space

• Possible mediating effects of green space on the health effects of air pollution and noise.
• Green areas, less traffic and noise.
• Improved both physical and mental health and well-being: Reduced mortality.
• Reduced hospital admissions.
• Improved mental health.
• Better pregnancy outcomes.
• Enhanced brain development.
Conclusions

- There is no unique exposure metric for traffic related air pollution.
- Changes in emissions and population characteristics over the years.
- Several health effects under study but few with definitive conclusive statements regarding the body of the evidence.
- Need for a systematic approach.
- The role of other factors (noise, stress, SES) should be evaluated.
Thanks!!!

Acknowledgments

- Hanna Boogaard, HEI