SIGH OUT LOUD

BIBLIOGRAPHY

1. **Every breath we take. The lifelong impact of air pollution**
   Clin Med (Lond). 2017 Feb; 17(1): 8–12, doi: 10.7861/clinmedicine.17-1-8

Air pollution has become one of the major risks to human health because of the progressive increase in the use of vehicles powered by fossil fuels. While the risks of air pollution to health were thought to have been brought under control by the Clean Air Acts of the 1950s and 1960s, the situation of air pollution in the UK has now deteriorated to a point where it is contributing to 40,000 excess deaths each year. Here the findings of the RCP/RCPCH's 2015/16 Working Party on Air Pollution and Health are described and what actions now need to be taken. The UK needs to take a lead and introduce a new Clean Air Act that deals with the vehicle sources of pollution recognising that the toxic particles and gases emitted are effecting individuals from conception to death. This mandates urgent action by government both central and local, but also by all of us who have now become so dependent on road transport.

- The evidence is now overwhelming that primary and secondary small and ultrafine particles (PM10, PM2.5 and PM0.1) in particular, are linked to increased all-cause mortality (29,000 deaths each year in the UK) and especially deaths from cardiovascular and respiratory disease.
- Recent research shows that oxides of nitrogen (NOx: NO, NO2 and N2O4) and specifically NO2 emitted in vehicle exhaust are not as benign as previously thought, increasing the number of associated deaths by up to 40,000 each year.3
- Air pollution has adverse effects across the life course – from conception to old age. Air pollution impairs overall fetal growth, especially lung growth; this persists across childhood, increases the risks of developing new asthma, which might not occur in its absence, and affects the heart and lungs throughout life by direct toxicity and via epigenetic mechanisms that mediate gene/environmental interactions.
- Beyond respiratory and cardiovascular disease, air pollution has adverse impacts on the development of impaired cognition, type 2 diabetes, cancers, skin aging and even acts (co-relate) as a risk factor for obesity. Since the report, new evidence has become known on the adverse effects of pollution on neurodevelopment.4–6
- Importantly, the toxic health effects of vehicle-related pollution are greater in those socioeconomically deprived, living closer to busy roads, in poor housing, with inadequate diet, accompanying tobacco smoking and in the presence of family stress.

2. **Fossil fuels are harming our brains: identifying key messages about the health effects of air pollution from fossil fuels,** John Kotcher**,** Edward Maibach**,** Wen-Tsing Choi**,** https://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-019-7373-1

Previous research suggests that providing generalized information about the health implications of air pollution from fossil fuels may be effective at promoting public support for a transition to cleaner sources of energy. We sought to extend that work by identifying the specific messages about the health implications of air pollution from fossil fuels that are most and least concerning to people, and whether rankings of concern vary among different audiences. We also hypothesized that reading the statements would influence people's attitudes and behavioral intentions in a manner supportive of a transition to cleaner sources of energy.

Methods
We conducted a survey with a diverse sample of U.S. adults (*n*=1644) from a non-probability internet panel. Using maximum difference scaling, participants ranked a set of ten statements that revealed which statements were the most and least concerning to them. We also measured attitudes about air pollution and energy use before and after the ranking exercise to assess changes in opinion caused by cumulative exposure to the messages.

Results
Across all sub-groups examined, participants were most concerned by a message about the neurological impacts of air pollution on babies and children. After the ranking exercise, participants expressed increases in perceived health harm of air pollution and fossil fuels, a desire for more clean energy, and intention to engage in consumer advocacy to support clean energy.

Conclusions
To our knowledge, this study is the first to assess how people respond to information about the neurological health harms of air pollution from fossil fuels. While efforts to communicate the cardio-pulmonary health harms of air pollution are well established, our study suggests that efforts should now be organized to communicate the neurological effects of air pollution from fossil fuels, especially the neuro-developmental effects on babies and children.

3. Did you know neurotoxicant pollutants released during incomplete combustion of fossil fuel, tobacco impact your social competences?

Polycyclic aromatic hydrocarbons (PAH), including benzo[a]pyrene (B[a]P), are neurotoxicant pollutants released during incomplete combustion of fossil fuel, tobacco, and other organic material (Bostrom et al., 2002), found in air and dietary sources. Human exposure to PAH is ubiquitous. Differential siting of outdoor pollution sources in low-income, urban, and minority communities produces striking disparities in exposure levels (Heritage, 1992; Metzer, Delgado, & Herrell, 1995; Olden & Poje, 1995; Pirkle et al., 1996; Wagenknecht, Manolio, Sidney, Burke, & Haley, 1993; Wernette & Nieves, 1992). Exposures to PAH and other environmental pollutants during the prenatal and early postnatal stages are of particular concern for child health and development (Grandjean and Landrigan 2006; National Research Council 1993; Perera et al. 2004). Early exposure may add risk because of the heightened susceptibility of the developing brain to these exposures (Nijland, Ford, & Nathanielsz, 2008; Rodier, 2004). During the fetal period and early childhood years, the brain is rapidly developing and vulnerable to neurotoxic insults that may manifest as adverse outcomes in childhood and adulthood (Shonkoff et al., 2012; Stein, Schettler, Wallinga, & Valenti, 2002). We evaluated the influence of prenatal exposure to widespread urban air pollutants on the development of self-regulation and social competence in a longitudinal prospective cohort of children born to nonsmoking minority women in New York City.

Methods
Air pollutant exposure was estimated categorically by level of polycyclic aromatic hydrocarbon (PAH)-DNA adducts in maternal blood collected at delivery, providing a biomarker of maternal exposure to PAH over a 2–3 month period. DESR was defined as moderate elevations on three specific scales of the Child Behavior Checklist (Anxious/Depressed, Aggressive Behavior, and Attention Problems). We used Generalized Estimating Equations to assess the influence of prenatal exposure to PAH on DESR in children at 3–5, 7, 9 and 11 years of age, adjusted for gender, and race/ethnicity. Next, we assessed the association of prenatal exposure to PAH with social competence, as measured by the Social Responsiveness Scale (SRS), the association of impaired self-regulation with social competence, and whether impairment in self-regulation mediated the association of prenatal exposure to PAH with social competence.

Results
We detected a significant interaction (at p=.05) of exposure with time, in which the developmental trajectory of self-regulatory capacity was delayed in the exposed children. Multiple linear

regression revealed a positive association between presence of PAH-DNA adducts and problems with social competence ($p<0.04$), level of dysregulation and problems with social competence ($p<0.0001$), and evidence that self-regulation mediates the association of prenatal exposure to PAH with SRS ($p<0.0007$).

Conclusions
These data suggest that prenatal exposure to PAH produces long-lasting effects on self-regulatory capacities across early and middle childhood, and that these deficits point to emerging social problems with real-world consequences for high-risk adolescent behaviors in this minority urban cohort.

Keywords: Polycyclic aromatic hydrocarbons, self-regulation, prenatal exposure, social competence

4. **Did you know living nearby the major roadways increased the incidence of dementia?**

Ref. Mark A S Laidlaw, Arthur E Poropat, Andy Ball, Howard W Mielke, Exposure to lead in petrol and increased incidence of dementia https://doi.org/10.1016/S0140-6736(17)31466-6
VOLUME 389, ISSUE 10087, p2371-2372, June 17, 2017

In *The Lancet*, Hong Chen and colleagues reported that higher exposures to nitrogen dioxide (NO_2) and airborne particulate matter were greatly associated with dementia. Unfortunately, Chen and colleagues were unable to directly measure these airborne pollutants, relying instead on the proxy measure of proximity to pollution sources, namely major roadways.
The use of proximity to pollution sources as a proxy measure for airborne pollution was supported by a range of alternative measures, including satellite and ground-based monitoring. For most of the 20th century, lead was used as a petrol additive in Canada and was only phased out in 1990. Given the toxicity of lead, the magnitude of lead exposure to populations was large. In 1982, in the province of Ontario alone, $2{\cdot}6 \times 10^9$ g of lead was consumed in automotive petrol, which subsequently declined by an average of $3{\cdot}6 \times 10^8$ g per year until 1990, to a rate of $1{\cdot}9 \times 10^7$ g of lead per year.

Between 1982 and 1990, the decline in blood lead levels were closely correlated (r=0·9323) with the decrease in previous years' consumption of leaded petrol. Historic atmospheric lead levels in Ontario were positively correlated with blood lead levels (r=0·99) and average concentration of lead in petrol. Consistent with the pollution evidence reported by Chen and colleagues, distance from roadways has been shown to be inversely associated with lead concentrations in soils and in human blood. Likewise, the magnitude of lead deposited in the roadside ecosystem is positively associated with traffic flow volumes, and blood lead levels are positively associated with traffic flow rates on nearby roads. Consequently, Chen and colleagues' proxy measure for exposure to NO2 and particulate matter is also a proxy measure for past blood lead levels in populations. Unlike NO2, lead persists in the human body, with more than 95% of the adult body burden of lead stored in bones, where it can remain for decades. Stored lead is mobilised when bone turnover is higher than the normal turnover rate—ie, in patients with osteoporosis. Consequently, mobilisation of stored lead coupled with decline of bone mineralisation in elderly Canadian residents means that their blood lead levels can remain elevated many years after road-borne lead exposure. Measures of lead exposure have been linked with dementia in cross-sectional research and bone lead levels predict cognitive decline in elderly men and women.

Background
Emerging evidence suggests that living near major roads might adversely affect cognition. However, little is known about its relationship with the incidence of dementia, Parkinson's disease, and multiple sclerosis. We aimed to investigate the association between residential

proximity to major roadways and the incidence of these three neurological diseases in Ontario, Canada.

Methods
In this population-based cohort study, we assembled two population-based cohorts including all adults aged 20–50 years (about 4·4 million; multiple sclerosis cohort) and all adults aged 55–85 years (about 2·2 million; dementia or Parkinson's disease cohort) who resided in Ontario, Canada on April 1, 2001. Eligible patients were free of these neurological diseases, Ontario residents for 5 years or longer, and Canadian-born. We ascertained the individual's proximity to major roadways based on their residential postal-code address in 1996, 5 years before cohort inception. Incident diagnoses of dementia, Parkinson's disease, and multiple sclerosis were ascertained from provincial health administrative databases with validated algorithms. We assessed the associations between traffic proximity and incident dementia, Parkinson's disease, and multiple sclerosis using Cox proportional hazards models, adjusting for individual and contextual factors such as diabetes, brain injury, and neighbourhood income. We did various sensitivity analyses, such as adjusting for access to neurologists and exposure to selected air pollutants, and restricting to never movers and urban dwellers.

Findings
Between 2001, and 2012, we identified 243 611 incident cases of dementia, 31 577 cases of Parkinson's disease, and 9247 cases of multiple sclerosis. The adjusted hazard ratio (HR) of incident dementia was 1·07 for people living less than 50 m from a major traffic road (95% CI 1·06–1·08), 1·04 (1·02–1·05) for 50–100 m, 1·02 (1·01–1·03) for 101–200 m, and 1·00 (0·99–1·01) for 201–300 m versus further than 300 m (*p* for trend=0·0349). The associations were robust to sensitivity analyses and seemed stronger among urban residents, especially those who lived in major cities (HR 1·12, 95% CI 1·10–1·14 for people living <50 m from a major traffic road), and who never moved (1·12, 1·10–1·14 for people living <50 m from a major traffic road). No association was found with Parkinson's disease or multiple sclerosis.
Interpretation
In this large population-based cohort, living close to heavy traffic was associated with a higher incidence of dementia, but not with Parkinson's disease or multiple sclerosis.

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Pollution is the largest environmental cause of disease and premature death in the world today. Diseases caused by pollution were responsible for an estimated 9 million premature deaths in 2015—16% of all deaths worldwide—three times more deaths than from AIDS, tuberculosis, and malaria combined and 15 times more than from all wars and other forms of violence

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