Exposure to lead in petrol and increased incidence of dementia

We read with great interest the Article by Hong Chen and colleagues (Feb 18, p 718) on living near major roads and the incidence of dementia, Parkinson’s disease, and multiple sclerosis. We hypothesise that the link found between residential proximity to major roads and dementia incidence might be partly due to a more distal risk factor, past exposure to leaded petrol.

Three-quarters of Canadians aged 65 years and older own their homes and have many have lived in these homes most of their adult lives. Although exact data on the Canadian population’s duration of tenure were not readily available, the average US home owner aged 65 years and older had been in their home for 24.7 years. A substantial proportion of Canadians aged 65 years and older in the study by Chen and colleagues had probably lived in the same residence in the 1970s when leaded petrol was still in use. Individuals living closest to major roads would have had the greatest exposure to this lead neurotoxin. A 2006 review concluded that an association exists between adults’ cumulative lead exposure and the rate of decline in cognitive function. With respect to Mini-Mental State Exam scores, a prospective study of community-dwelling older men (mean age 67-4 years [SD 6-6]) reported that an increase of one interquartile range in cumulative lifetime exposure to lead (measured by bone lead levels) was similar to being 5 years older at baseline. The link between lead exposure and dementia is plausible because of lead’s ability to cross the blood-brain barrier and its effect on oxidative stress and neuronal death. Future studies examining the association between dwelling proximity to major roads and dementia incidence would benefit from inclusion of information on bone lead levels.

We declare no competing interests.

*Esme Fuller-Thomson, Sydney A Jopling esme.fuller.thomson@utoronto.ca

Department of Family and Community Medicine (EF-T), Factor-Inwentash Faculty of Social Work (EF-T, SAJ), and Institute of Health Policy, Management and Evaluation (SAJ), University of Toronto, Toronto, ON M5S 1V4, Canada


In The Lancet, Hong Chen and colleagues reported that higher exposures to nitrogen dioxide (NO₂) 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. However, nearness to major roadways is associated with other potential biological risks; one of the more potent risks being previous exposure to lead.

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, 2·6 × 10⁶ g of lead was consumed in automotive petrol, which subsequently declined by an average of 3·6 × 10⁶ g per year until 1990, to a rate of 1·9 × 10⁶ 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—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. By contrast, Chen and colleagues were unable to cite direct associations of NO₂ or particulate matter exposure with dementia, arguing instead on the basis of an indirect link provided by a single laboratory study.
Correspondence

and colleagues1 and other researchers lead exposure near roadides. The between dementia and environmental that is consistent with causal links causing dementia is either indirect or confounded by probable lead exposure. Consequently, we suggest that Chen and colleagues’ conclusions are unconfirmed and their results should be reconsidered as providing further evidence of the toxic effects of lead.

We declare no competing interests.

*Mark A S Laidlaw, Arthur E Poropat, Andy Ball, Howard W Mielke mark.laidlaw@rmit.edu.au

Centre for Environmental Sustainability and Remediation, School of Science, RMIT University, Bundoora, VIC 3083, Australia (MASL, AB); School of Applied Psychology, Griffith University, Brisbane, QLD, Australia (AAP), and Department of Pharmacology, Tulane University School of Medicine, Tulane University, New Orleans, LA, USA (HWM)


Authors’ reply

We appreciate the comments by Esme Fuller-Thomson and Sydney A Jopling, and Mark A S Laidlaw and colleagues on our cohort study,1 in which we investigated the association between living close to busy roadways and the incidence of dementia, Parkinson’s disease, and multiple sclerosis in Ontario, Canada. Both letters hypothesised that past exposure to leaded petrol might explain, at least partly, our observed association between living near roadways and higher incidence of dementia. Their proposition is an important reminder of the potentially long-lasting negative effects of many environmental factors on human health, even decades after exposures are dramatically reduced.

Living near roadways substantially increases exposure to ultrafine particles, heavy metals, particles from wear of tyres and friction materials, nitrogen oxides, volatile organic compounds, noise, and other factors. The idea that lead exposure near roadways might potentially affect dementia risk is plausible, but so do many other constituents of traffic-related pollution, such as ultrafine particles and black carbon. These other pollutants are known to exhibit steep, stable, and sharp gradients around major roadways which match what we observed for the incidence of dementia. However, whether blood lead levels also exhibit similar (sharp) spatial gradients remains uncertain.2

In addition to our finding relating nitrogen dioxide and fine particulate matter to dementia, increasing epidemiological, experimental, animal, and post-mortem studies have found an association between air pollution and dementia.3 For example, results of a recent study4 that comprised a prospective cohort study of older women in the USA and neurotoxicological substance inhalation experiments with mice, showed the contribution of particulate air pollutants to neurodegenerative changes and higher risk of Alzheimer’s disease. The converging lines of evidence are further reinforced by the emergence of studies that link traffic-related air pollution to impaired cognitive development in children.5 Taken together, strong evidence exists implicating that exposure to present-day air pollution might be a culprit in the development of neurodegenerative disorders such as dementia.6

We agree that further research is needed to improve the understanding of the different aspects of traffic that underlie the observed association of roadway proximity and dementia. Ultimately, though, to guide public health interventions to reduce the burden of dementia, epidemiological studies need to inform public actions towards reducing exposures, especially ongoing exposures that give rise to dementia. Past policy actions to phase out leaded petrol have substantially reduced lead exposure near roadways over the past decades, whereas global efforts are still required to reduce air pollution exposure. To this end, we would like to reiterate the urging of Calderón-Garcidueñas and Villarreal-Ríos from their commentary about our Article: “we must implement preventive measures now, rather than take reactive actions decades from now”.6

We declare no competing interests.

*Hong Chen, Barry Jessiman, Ray Copes, Paul J Villeneuve, Richard T Burnett hong.chen@oahpp.ca

Public Health Ontario, Toronto, ON, M5G 2V2, Canada (HC, RC), Institute for Clinical Evaluative Sciences, Toronto, ON, Canada (HC); Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada (HC); Population Studies Division, Health Canada, Ottawa, ON, Canada (BJ, RTB); and Department of Health Science, Carleton University, Ottawa, ON, Canada (PV)


Correspondence

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