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Case Study: Health effects of traffic-related air pollution in a small community

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About Case Studies

The Environmental and Occupational Health team provides scientific and technical advice and support to the health care system and the Government of Ontario. We have created the Case Study series to share the diverse environmental health issues we have encountered and encourage dialogue in these areas.

This response was originally produced in April 2014. The specifics about the location and requestor involved have been removed.

The following was selected as a Case Study because of the ubiquity of traffic-related air pollution as an environmental health issue.

Background to the request

This review of the health effects of traffic-related air pollution was prepared for an Ontario health unit regarding a small community, "the Community". The main road through the Community is "the Highway", which is also used by industrial vehicles. Members of the Community had voiced concerns regarding public safety, noise, and the vehicle exhaust due to this traffic. The health unit was asked for information on the health effects of air pollution related to motor vehicle use. This review provides an overview of this problem, with consideration to the roadway in question.

Methods

The PHO Library performed a MEDLINE search on the health effects of traffic-related air pollution, as well as a "snowball search" on the article: Brauer M, Reynolds C, Hystad P. Traffic-related air pollution and health in Canada. CMAJ. 2013;185(18):1557-8.[1] This involved retrieving articles labeled as "related" to the above PubMed citation. Relevant references from journal articles were retrieved. Toxicology and environmental health textbooks were consulted.

Air pollution overview

Outdoor air pollution is a complex mixture of gases and particulate matter (PM). Pollutants can be naturally produced or human made. They can be emitted directly from the source (primary) or formed by the chemical or physical conversion of precursors in the environment (secondary).[2] Natural sources of air pollution include forest fires, volcanic eruptions, dust storms and emissions from vegetation. Human activities that produce air pollution include fossil fuel combustion from motor vehicles and industrial processes, wood burning and home heating.[2] Common pollutants in outdoor air include carbon monoxide (CO), nitrogen oxides (NOx), sulfur dioxide (SO_2), ground level ozone and PM.[2,3] Inhalable coarse particles are between 2.5 µm and 10 µm in diameter and mostly deposit in the human nasopharynx.[4] Fine particles are 2.5 µm and smaller in diameter, that deposit in the bronchioles and in the alveoli of the human lung.[4] Ultrafine particles are less than 0.1 µm in diameter and can deposit in the alveoli and the bronchioles of the lung.[4]

Health effects of air pollution

Morbidity and mortality resulting from very high levels of PM air pollution have been known for decades. Major episodes of air pollution London in 1952 and Donora, Pennsylvania in 1948 are well known.[5] Evidence on the cardiopulmonary effects of longterm exposure to much lower levels of ambient air pollution emerged in the 1990s and 2000s, with no threshold level found for causing health effects.[6]

The International Agency for Research on Cancer (IARC) has assessed the human carcinogenicity of some air pollutants. Diesel engine exhaust was classified as carcinogenic to humans (Group 1) and gasoline engine exhaust was classified as possibly carcinogenic to humans (Group 2B) in 2012.[7] The conclusion for diesel engine exhaust was drawn from occupational exposures to miners, railroad workers, and workers in the trucking industry.[7] Outdoor ambient air pollution and particulate matter were also classified as carcinogenic to humans (Group 1) in 2013.[8]

Traffic-related pollution and population exposures

Traffic-related air pollution (TRAP) is a major contributor to air pollution in urban areas, although it may be difficult to isolate its contribution from other combustion sources.[9] Motor vehicles release a mix of combustion and non-combustion emissions.[9] Fuel combustion produces carbon dioxide (CO_2), CO, NOx, PM, and volatile organic compounds (VOCs).[9] Emissions vary by vehicle type, age, operation, maintenance, exhaust treatment, fuel, wear of parts, and engine lubricants. Major pollutants in gasoline engine exhaust include CO, hydrocarbons, and NOx.[9] Diesel engine exhaust is notable for NOx and particulates, especially ultrafine PM[10] in the absence of emission controls.[9] Non-combustion emissions include re-suspended road dust, tire wear, and brake wear.

While traditional air pollution monitoring networks are useful for detecting regional differences in air quality, they do not capture local variations in pollutant concentrations that may be important as risk factors for human health.[9] In urban areas, the chief source of pollutant variability is TRAP, making it a useful measure of individual human exposure to air

pollution.[9] Many studies have used proximity to traffic such as distance to the nearest major road or traffic volume on the nearest road as an index of human exposure. Over the last decade, methods to model the spatial variation of pollutants as a function of variables like location, surrounding land use, population density and traffic patterns have been developed.[11,12] These land-use regression (LUR) models have been able to provide relatively high-resolution views of the variation in specific pollutants in the areas mapped.

For instance, the highest levels of NO_2 in Windsor were found near major roadways, particularly the road leading to the Ambassador Bridge crossing to Detroit.[13] Annual concentrations higher than 18 ppb were found near some segments of roadway, while less than 10 ppb was seen in outlying areas.[13] LUR models are specific to the areas modeled and have been developed for Toronto,[14] Windsor,[13] Hamilton[15] and Sarnia[16]. All of these Ontario cities are urban centres with high-traffic roads and highways. Highway 402 in Sarnia near the Blue Water Bridge border crossing saw a relatively smaller amount of traffic, about 13,000 average annual daily traffic (AADT)[1] in 2010.[17] However, Sarnia's LUR model was significantly influenced by the chemical industrial centres.[16]

Traffic in the Community

In a report for Health Canada, Brauer et al[18] considered roads with AADT greater than 15,000 as important local sources of TRAP. The gradients of pollutants seen near large roads may not apply to roads with less traffic.[18] Where the Highway runs through the Community, it accommodates about half the volume of traffic cited by Brauer et al.[18] While the available literature focuses on areas with greater road traffic, the volume of traffic from industrial vehicles in the Community may provide a rough estimate of the additional pollution generated. The following table shows the AADT estimates for the Highway in the Community from 2001 to 2012.

Table 1: AADT estimates for the Highway in the Community from 2001 to 2012

Year	AADT estimates
2001	9,050
2002	8,200
2003	9,220
2004	9,360
2005	9,610
2006	8,560
2007	8,920
2008	8,900
2009	8,610
2010	8,840
2011	8,860
2012	8,440

The AADT estimates show a slight downward trend. Of this traffic, roughly 10%–15% is estimated to be due to industrial traffic in the Community. The industrial component of this traffic may contribute more diesel-related pollutants and coarse PM. However, its absolute contribution to pollutant levels is probably small compared to traffic volumes in larger centres where gradients have been studied.

Health effects of traffic-related air pollution

A comprehensive critical review of the weight of evidence on traffic-related emissions, human exposure and the associated health effects was done by the Health Effects Institute (HEI) in 2010.[9] The panel concluded a zone within 300–500 metres from a major road as the area most highly affected by traffic emissions. The further away from roads, the greater the influence of regional pollution concentrations, meteorologic conditions and seasonality.[9]

[1] AADT is the total volume of traffic on a road over one year divided by 365 days.

Table 2: The panel's conclusions with respect to asthma and other health effects

Sufficient evidence for a causal relationship with TRAP	• exacerbation of asthma
Suggestive but not sufficient evidence for a causal relationship with TRAP	• child asthma onset • non-asthma respiratory symptoms • impaired lung function • total and cardiovascular mortality • cardiovascular morbidity
Inadequate and insufficient evidence for a causal relationship with TRAP	• adult asthma onset • chronic obstructive pulmonary disease • allergy • birth outcomes • cancer

The panel concluded that the weight of evidence supported a causal relationship between TRAP and asthma exacerbations.[9] The panel's conclusions with respect to asthma and other health effects are presented in Table 2.

Since the HEI report in 2010, studies have continued to focus on TRAP's health effects. Brauer et al produced a report for Health Canada in 2012 reviewing TRAP, its health effects and potential mitigation strategies.[1,18] They reported that accumulating evidence now suggests TRAP as a cause of childhood asthma onset.[18] One of the supporting studies was a systematic review and meta-analysis of asthma onset from 17 cohort studies by Anderson et al.[19] The majority of studies looked at differences in exposure to air pollution within communities that were largely due to traffic. An increase of 10 $\mu g/m^3$ (about 5.2 ppb) in NO_2 was found in the meta-analysis to be a significant predictor of the incidence of asthma and wheeze symptoms, while the summary estimate for $PM_{2.5}$ was borderline but not significant.[19]

TRAP's impact on cardiovascular mortality also continues to be studied. A recent cohort study of residents in Hamilton, Toronto and Windsor found a significant relationship between a 5 ppb increase in exposure to NO_2 and death from all cardiovascular and ischemic heart diseases.[20] The authors found significant associations regardless of when NO_2 was measured –at baseline, in the year prior to the outcome, in the three years prior to the outcome, or over the long-term. No association was found between NO_2 increases and cerebrovascular disease.[20] Biological plausibility for the cardiovascular effects of TRAP has also been supported by studies. In one, the progression of artery disease was found to be associated with residential proximity to highways.[21]

Though IARC has classified diesel engine exhaust as carcinogenic, the association of TRAP with lung cancer is still being explored. A recent meta-analysis of 17 European cohorts combined data from over 300,000 people.[22] Using land-use regression models to estimate PM exposures at home addresses, the authors found that an increase in PM_{10} was associated with incidence of lung cancer. When analysis was restricted to subjects with adenocarcinomas and people who never moved for the duration of study, PM_{10} was even more strongly associated with lung cancer incidence.[22]

The health effects of the non-combustion component of TRAP have not been studied as well.[9] Much of this component consists of

coarse particles with diameters above 2.5 µm. Coarse particles generally arise from mechanical processes rather than combustion.[23] The composition of coarse PM can vary depending on the season, geography and nearby industrial activity. For instance, in arid areas, coarse particles comprise a greater proportion of PM_{10}.[23] A review of the health evidence on coarse particles found that many studies could not separate the health effects of coarse PM from fine PM, and the available data do not permit a meta-analysis.[23] Although some of the studies found associations between coarse particles and respiratory health effects, the evidence overall was inconsistent and difficult to interpret due to challenges such as accurate measurement of coarse PM.[23]

Conclusion

While urban gradients in combustion-related traffic pollutants have been shown to cause exacerbations and possibly the onset of asthma, many of these studies have been done in dense urban areas. Qualitatively, the health effects of traffic pollutants are probably the same in the Community as in cities where they have been studied. For example, the health effects of NO_2 should be the same in the Community as they are in large cities. However, the quantity of health effects produced will depend on the local volume of traffic and the change in pollutant concentrations away from the roadway. These factors are probably different in a rural location where the buildings, configuration of roadways, and land cover are different from urban centres. To apply the findings of TRAP studies to the Community and derive a measure of risk from vehicle use, the respective AADTs may be useful. The volume of industrial traffic would provide a rough benchmark of the incremental increase in TRAP from industrial versus non-industrial traffic. Because the industrial traffic is a relatively small component of total traffic, it would likely be difficult to measure any health benefits of limiting the current traffic volumes.

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