

HEI

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Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects

A Special Report of the HEI Panel on the Health Effects
of Traffic-Related Air Pollution

EXECUTIVE SUMMARY

ABOUT HEI

The Health Effects Institute is a nonprofit corporation chartered in 1980 as an independent research organization to provide high-quality, impartial, and relevant science on the effects of air pollution on health. To accomplish its mission, the institute

- Identifies the highest-priority areas for health effects research;
- Competitively funds and oversees research projects;
- Provides intensive independent review of HEI-supported studies and related research;
- Integrates HEI's research results with those of other institutions into broader evaluations; and
- Communicates the results of HEI research and analyses to public and private decision makers.

HEI receives half of its core funds from the U.S. Environmental Protection Agency and half from the worldwide motor vehicle industry. Frequently, other public and private organizations in the United States and around the world also support major projects or certain research programs. Additional work for this report was funded by the U. S. Federal Highway Administration.

HEI has funded more than 280 research projects in North America, Europe, Asia, and Latin America, the results of which have informed decisions regarding carbon monoxide, air toxics, nitrogen oxides, diesel exhaust, ozone, particulate matter, and other pollutants. These results have appeared in the peer-reviewed literature and in more than 200 comprehensive reports published by HEI.

HEI's independent Board of Directors consists of leaders in science and policy who are committed to fostering the public-private partnership that is central to the organization. The Health Research Committee solicits input from HEI sponsors and other stakeholders and works with scientific staff to develop a Five-Year Strategic Plan, select research projects for funding, and oversee their conduct. The Health Review Committee, which has no role in selecting or overseeing studies, works with staff to evaluate and interpret the results of funded studies and related research.

All project results and accompanying comments by the Health Review Committee are widely disseminated through HEI's Web site (www.healtheffects.org), printed reports, newsletters, and other publications, annual conferences, and presentations to legislative bodies and public agencies.

EXECUTIVE SUMMARY

Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects

INTRODUCTION

Motor vehicles are a significant source of urban air pollution and are increasingly important contributors of anthropogenic carbon dioxide and other greenhouse gases. As awareness of the potential health effects of air pollutants has grown, many countries have implemented more stringent emissions controls and made steady progress in reducing the emissions from motor vehicles and improving air quality. However, the rapid growth of the world's motor-vehicle fleet due to population growth and economic improvement, the expansion of metropolitan areas, and the increasing dependence on motor vehicles because of changes in land use has resulted in an increase in the fraction of the population living and working in close proximity to busy highways and roads — counteracting to some extent the expected benefits of pollution-control regulations and technologies.

This Special Report, developed by the Health Effects Institute (HEI) Panel on the Health Effects of Traffic-Related Air Pollution, summarizes and synthesizes information linking emissions from, exposures to, and health effects of traffic sources (i.e., motor vehicles). The term *traffic-related exposure* is used in this report to refer to exposure to primary emissions from motor vehicles, not to the more broadly dispersed secondary pollutants such as ozone (O₃) that are derived from these emissions. The report focuses on specific scenarios with a high aggregation of motor vehicles and people — that is, urban settings and residences in proximity to busy roadways.

EMISSIONS FROM MOTOR VEHICLES

Motor vehicles emit large quantities of carbon dioxide (CO₂), carbon monoxide (CO), hydrocarbons (HC), nitrogen oxides (NO_x), particulate matter (PM), and substances known as mobile-source air toxics (MSATs), such as benzene, formaldehyde, acetaldehyde, 1,3-butadiene, and lead (where leaded gasoline is still in use). Each of these, along with secondary by-products, such as ozone and secondary aerosols (e.g., nitrates and inorganic and organic acids), can cause adverse effects on health and the environment. Pollutants from vehicle emissions are

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related to vehicle type (e.g., light- or heavy-duty vehicles) and age, operating and maintenance conditions, exhaust treatment, type and quality of fuel, wear of parts (e.g., tires and brakes), and engine lubricants used. Concerns about the health effects of motor-vehicle combustion emissions have led to the introduction of regulations and innovative pollution-control approaches throughout the world that have resulted in a considerable reduction of exhaust emissions, particularly in developed countries. These reductions have been achieved through a comprehensive strategy that typically involves emissions standards, cleaner fuels, and vehicle-inspection programs. Recognizing the likely continued growth in the vehicle fleet and the remaining problems in traffic-related air quality, the United States, European countries, Japan, and other countries are continuing to push for even stricter emissions controls in coming years.

Resuspended road dust, tire wear, and brake wear are sources of noncombustion PM emissions from motor vehicles. As emissions controls for exhaust PM become more widespread, emissions from noncombustion sources will make up a larger proportion of vehicle emissions. Noncombustion emissions contain chemical compounds, such as trace metals and organics, that might contribute to human health effects. However, current estimates of these emissions are highly uncertain. Thus, although they are not regulated in the way exhaust emissions are, noncombustion emissions will need to be considered more closely in future assessments of the impact of motor vehicles on human health.

The quantification of motor-vehicle emissions is critical in estimating their impact on local air quality and traffic-related exposures and requires the collection of travel-activity data over space and time and the development of emissions inventories. Emissions inventories are developed based on complex emissions models (of which the U.S. Environmental Protection Agency's MOBILE6 has been the most widely used) that provide exhaust and evaporative emissions rates for total HC, CO, NO_x, PM, sulfur dioxide (SO₂), ammonia (NH₃), selected air toxics, and green house gases (GHGs) for specific vehicle types and fuels. The quality of the travel-activity data (such as vehicle-miles traveled, number of trips, and types of vehicles) and the complex algorithms used to derive the emissions factors suggest the presence of substantial uncertainties and limitations in the resulting emissions estimates (NARSTO 2005). It should be noted that estimates of PM emissions have had very limited field valuation and verification.

The actual measurement of motor-vehicle emissions is critically important for validating the emissions models. Studies that have sampled the exhaust of moving vehicles in real-world situations (specifically, in tunnels or on roadways) have contributed very useful information about the emissions rates of the current motor-vehicle fleet and also have allowed the evaluation of the impact of new emission-control technologies and fuels on emissions.

Receptor models have been used to estimate the contributions of various types of sources, including motor vehicles, to ambient air pollution. Some of the models (those defined as *chemical mass balance models*) require the knowledge of the chemical profile of both the emissions of all the area sources and the air at the receptor (that is, the impacted location). Other models (referred to as *principal components and factors analyses*) do not require a priori knowledge of the source profiles. The application of these models has yielded a wide range of results on the contribution of motor vehicles to ambient pollution, depending on the model, the location of the monitoring sites, and the other sources present. In U.S. cities, the results show that motor-vehicle contributions range from 5% in Pittsburgh, Pa., under conditions with very high secondary aerosol, to 49% in Phoenix, Ariz., and 55% in Los Angeles, Calif. Outside the United States, estimates of the motor-vehicle contribution to $PM_{2.5}$ ($PM \leq 2.5 \mu m$ in aerodynamic diameter) range from 6% in Beijing, China, to 53% in Barcelona, Spain.

Ultimately, an important goal of emissions-characterization studies is to improve our ability to quantify human exposure to emissions from motor vehicles, especially in locations with high concentrations of vehicles and people. Such characterization requires improving emissions inventories and a more complete understanding of the chemical and physical transformations on and near roadways that can produce toxic gaseous, semivolatile, and particle-phase chemical constituents.

ASSESSMENT OF EXPOSURE TO TRAFFIC-RELATED AIR POLLUTION

Traffic-related emissions contribute to primary and secondary local, urban, and regional (background) pollutant concentrations against a background of similar contaminants emitted from other sources. Traffic emissions are the principal source of intra-urban variation in the concentrations of air pollutants in many cities; thus, population-oriented central monitors cannot by themselves capture this spatial variability. Studies that have examined gradients in pollutants as a function of distance from busy roadways have indicated exposure zones for traffic-related air pollution in the range of 50 to 1500 m from highways and major roads, depending on the pollutant and the meteorologic conditions.

Because it is not practical or feasible to measure all the components of the traffic-pollutant mix, surrogates of traffic-related pollution have been used as a reasonable compromise for assessing the contribution of traffic emissions to ambient air pollution and for estimating traffic exposure. Surrogates can also help in the assessment of spatial and temporal distributions of ambient pollution related to motor vehicles and of traffic-mitigation control strategies.

Two broad categories of surrogates have been used in epidemiology studies to estimate traffic exposure: (1) measured or modeled concentrations of pollutant surrogates and (2) direct

measures of traffic itself (such as proximity, or distance, of the residence to the nearest road and traffic volume within buffers). The most commonly used traffic-pollutant surrogates include CO, NO_2 , elemental carbon (EC; or black carbon [BC] or black smoke [BS]), PM, benzene, and ultrafine particles (UFP). Exposure models include geostatistical interpolation, land-use regression, dispersion, and hybrid models (the latter combine time-activity data, personal measurements, and models). They incorporate numerous parameters (such as meteorologic variables, data on land use, traffic data, and monitoring data or emissions rates depending on the model) and can improve the spatial representation of the local impact of traffic against a background of regional and urban concentrations. However, the accuracy of the inputs is critical to the usefulness of any given model.

None of the pollutant surrogates considered in the report met all the criteria for an ideal surrogate. Data are not available to assess the ratios of the surrogates to emissions from all sources over time. CO, benzene, and NO_x (in this case NO_2), found in on-road vehicle emissions, are components of emissions from all sources, making it difficult to disentangle the contributions from motor vehicles from other sources (including some in indoor environments). Primary, on-road vehicle emissions of PM ($PM_{2.5}$ or PM_{10} [$PM \leq 10 \mu m$ in aerodynamic diameter]) represent only a small contribution to emissions from all sources, typically around 3%. EC has been used as a surrogate, primarily for diesel exhaust, although it is not a specific marker, unless other sources are ruled out. UFP concentrations are very high in vehicle-exhaust plumes but decrease rapidly with distance from the source, which poses a significant challenge for characterization of the spatial and temporal concentration gradients of UFP from roadway traffic.

With regard to exposure models, the Panel noted that, although proximity models (direct measures of traffic) are the easiest to implement, they are error prone because they ignore the parameters that affect the dispersion and physicochemical activity of the pollutants. Moreover, estimates based on proximity can be confounded by factors such as socioeconomic status and noise. Geostatistical interpolation models are best implemented in conjunction with dense, well-distributed monitoring networks; their chief limitations are the size of the network and the number of measurements needed over time to estimate the spatial distribution of pollution surrogates accurately. Land-use regression is appealing in that it can account for the diversity of sources that contribute to a surrogate; however, the true contribution (in terms of associated variance) of traffic to the regression is not always known or reported. Dispersion models utilize motor-vehicle-emissions and air-quality data and incorporate meteorologic data, but must be calibrated correctly to realize their advantages. These models are very data- and computation-intensive and depend on the validity of the model assumptions. Hybrid models that combine measurements of personal exposure to traffic surrogates or time-activity data with exposure models come closest to a logistically feasible “best” estimate of human exposure.

Factors influencing ambient concentrations of a traffic-pollutant surrogate are related to time–activity patterns, meteorologic conditions, vehicle volume and type, driving patterns, land-use patterns, the rate at which chemical transformations take place, and the degree to which the temporal and spatial distribution of the surrogate reflects the traffic source.

To improve assessment of exposure to traffic-related pollution, a potential solution is the deployment of a large number of monitors in places where concentrations of air pollutants are expected to be highly variable and the population density is high. The use of models that incorporate numerous spatial factors in order to estimate exposures that are more relevant to the individual's exposure situation can also be helpful.

The Panel concluded that the impact of vehicle emissions extends beyond the local scale to the urban and regional scales. What people are exposed to is influenced by their proximity to the sources, the presence of other ambient or microenvironmental sources, and time–activity patterns. If, as the evidence suggests, groups of lower socioeconomic status experience higher exposures than groups of higher socioeconomic status, this merits consideration in the interpretation of epidemiologic findings and in future regulatory actions.

Based on a synthesis of the best available evidence, the Panel identified an exposure zone within a range of up to 300 to 500 m from a highway or a major road as the area most highly affected by traffic emissions (the range reflects the variable influence of background pollution concentrations, meteorologic conditions, and season) and estimated that 30% to 45% of people living in large North American cities live within such zones.

HEALTH EFFECTS OF TRAFFIC-RELATED AIR POLLUTION: EPIDEMIOLOGY AND TOXICOLOGY

In reviewing the epidemiologic literature on the association between exposure to traffic-related air pollution and health outcomes, the Panel developed criteria for the inclusion of studies based on the characterization of traffic exposure. The Panel decided to include only studies that investigated associations between primary emissions from traffic and human health and that provided specific documentation of a traffic source and estimates of exposure on a local scale. Thus, studies that relied exclusively on measurements from a central monitoring site were not included unless the site was in proximity to traffic. The Panel also developed criteria for inferring whether associations between exposure and health outcome were causal by adapting the criteria used by the U.S. Surgeon General in the report *The Health Consequences of Smoking: A Report of the Surgeon General* (U.S. Department of Health and Human Services 2004). In order to deem the evidence sufficient to conclude that association between a metric of traffic exposure and an outcome was causal, it was necessary for the magnitude and direction of the effect estimates to be consistent across different populations and times and to rule out with reasonable confidence chance, bias in subject

selection, and confounding (in particular, socioeconomic status). The four inference criteria applied to this review are listed in Table 1. To these criteria the Panel added a traffic-specific coherence criterion (also included in Table 1) to account for the degree of validity of the traffic-specific exposure metrics. As noted earlier, the Panel concluded that not all traffic-exposure measures have equivalent validity and considered simple measures of proximity to roads or road length and of pollutant surrogates without specific traffic data to be the least specific. The proximity measures are also likely to introduce confounding.

Modeled estimates of exposure to traffic pollution were thought to be, a priori, more valid than traffic density estimates alone because they account for other factors that affect the exposure, such as geography, land use, and meteorology, when making estimates for particular locations. In addition, the validity of estimates can be enhanced by modeling strategies that separately estimate the contribution of traffic and background pollution to personal exposure.

The Panel developed qualitative and quantitative summaries (in tables and figures) for the estimates of the associations between traffic-related exposure and various health outcomes for the studies reviewed, but did not derive meta-analytic summaries by pooling associations estimates because of the lack of equivalence among the exposure measures and populations studied.

The Panel also reviewed the literature on the toxicology of traffic-related pollution. This included studies of direct exposures to traffic emissions (though there were very few in this category), studies that utilized laboratory atmospheres that replicate aspects of the traffic mix (such as concentrated ambient particles, or gasoline or diesel exhaust), and studies of specific components of emissions from motor vehicles. The aim was to identify possible mechanisms by which exposure to traffic pollutants may cause effects and provide an understanding of the role of traffic emissions in the effects being observed in epidemiology studies. While toxicology studies are limited in their ability to capture the full complexity of human exposure — because of the small number of subjects and, in animal studies, the relevance of the results to humans — they offer the opportunity to explore hypotheses on specific pathophysiologic mechanisms of action.

The Panel evaluated whether oxidative stress might be the underlying mechanism of action by which exposure to pollutants from traffic may lead to adverse health effects. Oxidative stress results from events occurring in any tissue in the body when the prooxidant–antioxidant balance is disturbed. This imbalance can happen when the generation of reactive oxygen species, or *free radicals*, exceeds the available antioxidant defenses and is characterized by the presence of increased cellular concentrations of oxidized lipids, proteins, and DNA. Oxidative stress can trigger inflammatory reactions, which lead to an increased production of oxidants by activated phagocytes recruited to the airways, perpetuating the cycle of oxidative injury.

The Panel concluded that, although the evidence supported the hypothesis that oxidative stress is an important determinant of health effects associated with ambient air pollution in general,

Executive Summary Table 1. Criteria for Assessing the Presence or Absence of Causal Associations in Studies of the Health Effects of Traffic-Related Air Pollution^{a,b}

A. Sufficient Evidence to Infer the Presence of a Causal Association

The evidence was deemed sufficient to conclude that an association observed between a metric of traffic exposure and a disease (or biomarker of disease) risk was causal in studies where chance, bias, and confounding could be ruled out with reasonable confidence, and the effect estimates were consistent in magnitude and direction.

Traffic-specific criterion. Classification A was applied:

When all studies were of the appropriate quality, at least one study measured traffic density or modeled traffic exposure^c, measures of socioeconomic status were taken into account in distance-only studies, and the studies' results were consistent.

B. Suggestive but Not Sufficient Evidence to Infer the Presence of a Causal Association

The evidence was deemed suggestive but not sufficient to conclude that an association between a metric of traffic exposure and a specific disease (or biomarker of disease) risk was causal in studies where chance, bias, and confounding could not be ruled out with reasonable confidence.

Traffic-specific criterion. Classification B was applied:

When all the criteria for Classification A were met except that only studies that used distance-based metrics were available

OR

When all the criteria for Classification A were met except that not all the studies that used distance-only metrics took into account measures of socioeconomic status or the studies took into account measures of socioeconomic status but the results were not consistent.

C. Inadequate and Insufficient Evidence to Infer the Presence or Absence of a Causal Association

The evidence was deemed inadequate and insufficient when the available studies were of insufficient quality, consistency, or statistical power to conclude whether a causal association was present or absent.

Traffic-specific criterion. Classification C was applied:

When the results from studies that used distance-only metrics were not consistent

OR

When the results of all studies using distance-only metrics were consistent but all those studies failed to include measures of socioeconomic status

OR

When the results from at least one study based on traffic density or modeled traffic exposure were inconsistent with those from distance-only studies

OR

When the number of distance-only studies was too small.

D. Evidence Suggestive of No Causal Association

The evidence was deemed suggestive of no causal association when there were several adequate studies, covering the full range of human exposure levels, that were consistent in not showing a positive association, at any level of exposure, between exposure to a metric of traffic exposure and a disease outcome. (Of course, a conclusion of “no association” is inevitably limited to the conditions, level of exposure, and length of observation covered by the available studies. In addition, the possibility of a very small elevation in risk at the levels of exposure studied cannot be excluded.)

Traffic-specific criterion. Classification D was applied:

When studies were of adequate quality (using distance-only metrics or at least some measures of traffic density or modeled traffic exposure) and were consistent in failing to find an association.

^a The Panel did not use exposure–response gradations as a criterion because, in virtually all epidemiologic studies, it is difficult to infer meaningful exposure–response gradations from the types of exposure metrics used or the forms of data presented.

^b This table was adapted from Tables 4.2a and 4.2.b in Chapter 4.

^c In some cases, this criterion was met when modeling or source-apportionment data were cited to show that a pollution surrogate in the study was reasonably accurate in representing the traffic sources in the study area.

the extent to which primary traffic-related pollutants contribute to the burden of reactive oxygen species experienced by humans near roadways remains undefined.

The Panel's main conclusions regarding the epidemiologic associations between exposure to traffic-related air pollution and health outcomes and the toxicologic evidence (when available) are presented below for each health outcome. A discussion of the extent to which toxicology studies do or do not provide general mechanistic support for the observations and inferences contributed by epidemiology studies is also provided.

ALL-CAUSE AND CARDIOVASCULAR MORTALITY

Epidemiology

Very few studies of all-cause mortality or cardiovascular mortality and long-term exposure met the criteria for inclusion in the report. Mostly because of the small number of studies, the evidence for an association of all-cause mortality with long-term exposure was classified as “suggestive but not sufficient” to infer a causal association. Additional factors that led to this classification were the substantial differences among populations, time periods, and confounders across studies.

Only four time-series studies of all-cause mortality associated with short-term exposure met the Panel's criteria; these, too, were classified as “suggestive but not sufficient,” largely on the strength of one well-done study (Maynard et al. 2007). Two time-series studies based on source-apportionment models were found to have a number of limitations that prevented a stronger statement about inferred causality.

Many of the issues that applied to studies of all-cause mortality applied as well to studies of cardiovascular mortality associated with long-term exposure and led, similarly, to a classification of “suggestive but not sufficient.” Only two time-series studies of cardiovascular mortality met the inclusion criteria, and although they both show positive associations, the Panel concluded that, given the overall paucity of studies, the evidence for effects of short-term exposure was “inadequate and insufficient.”

CARDIOVASCULAR MORBIDITY

Epidemiology

Studies that documented changes in cardiac physiology (such as heart-rate variability) after short-term exposure to traffic-related pollution (which was assessed using surrogates, source apportionment, or pseudo-personal monitoring) provided strong evidence for a causal association with the exposure. However, the failure of some studies to consider stress and noise as potential confounders led the Panel to classify them as “suggestive but not sufficient” to infer a casual association. Among the studies that evaluated cardiovascular morbidity, two well-executed studies on hospitalization for acute myocardial infarction were identified (Rosenlund et al. 2006; Tonne et al. 2007). In addition, a prospective study in a German cohort reported an association

between living near a major road and coronary-artery calcification as well as higher prevalence of coronary heart disease (Hoffmann et al. 2006, 2007). Collectively, these studies made a very strong case for an association between exposure to traffic-related pollutants and atherosclerosis. However, because of the small number of studies, the Panel classified them as “suggestive but not sufficient” to infer a causal association.

Toxicology

There have been a few toxicology studies that examined the cardiovascular effects of traffic emissions specifically. However, the Panel concluded that the recent toxicology literature provides suggestive evidence that exposure to pollutants that are components of traffic emissions, including ambient and laboratory-generated PM and exhaust from diesel and gasoline-fueled engines, alters cardiovascular function. There is also evidence, albeit inconsistent, for acute effects on vascular homeostasis and suggestive evidence in animal models that repeated exposures to ambient PM in general enhance the development of atherosclerosis. Some studies support the involvement of oxidative stress. Although the evidence from toxicology studies in isolation is not sufficient in terms of a causal association between traffic emissions and the incidence or progression of cardiovascular disease, when viewed together with the epidemiologic evidence, a stronger case could be made for a potential causal role for traffic-related pollutants in cardiovascular-disease morbidity and mortality. The extent to which these associations apply to individuals without underlying cardiovascular disease cannot be determined from the evidence available at this time.

ASTHMA AND RESPIRATORY SYMPTOMS

Asthma is an inflammatory disease of the lung airways characterized by episodic obstruction of the airways, which can lead to chronic obstructive lung disease. The most prevalent form of asthma in children and young adults is allergic asthma, which develops as an immune response to inhaled allergens. Individuals with asthma and other allergic conditions who have an increased tendency to develop immediate and localized reactions to allergens (such as pollens) that are mediated by immunoglobulin E (IgE) are referred to as “atopic.”

Epidemiology

In epidemiology studies, asthma is most frequently identified by means of responses to questionnaires that do not make use of a single, universally accepted set of questions, alone or in combination with other criteria. This is further complicated by the challenges of distinguishing factors that affect its onset from those (often the same factors) that lead to its episodic worsening. A history of asthma symptoms (such a wheezing) often is used in epidemiology studies as part of the definition both of asthma's onset (incidence) and of its prevalence and exacerbation.

Respiratory Health Problems in Children: Asthma Incidence and Prevalence Seven studies conducted in four separate

cohorts and one case-control study qualified as studies of asthma incidence in children. Eleven studies qualified as studies of asthma prevalence in children. From these studies, the Panel concluded that living close to busy roads appears to be an independent risk factor for the onset of childhood asthma. The Panel considered the evidence for a causal relation to be in a gray zone between “sufficient” and “suggestive but not sufficient.” The results found across the studies followed a pattern that would be expected under the plausible assumption that the pollutants really are causally associated with asthma development, if only among a subset of children with some accompanying pattern of endogenous or exogenous susceptibility factors. The conditions that underlie an increased risk for asthma development among children exposed to traffic-related pollutants are not known.

Exacerbation of Symptoms in Children with and without Asthma and Health-Care Utilization for Respiratory Problems

Among the more than 20 cohort and cross-sectional studies reviewed that examined the association between exposure to traffic-related pollution and wheezing (an important symptom in the expression and diagnosis of asthma) in children, there was a high degree of consistency in finding positive associations, many of which reached statistical significance (i.e., had reasonably precise point estimates of associations). This was true particularly for the large majority of studies that used models to assign estimates of local concentrations of pollutants, such as NO₂ or soot (the carbonaceous component of PM), to the place of residence of the study participants. Studies based on proximity or traffic density also indicated an association between exposure and wheezing. In addition, exacerbation of other asthma-related symptoms, such as cough or dry cough, was consistently associated with exposure across a variety of exposure measures. Although most studies were not restricted to children with asthma, all these symptoms were more prevalent among those with asthma, and it is very likely that the observed associations were driven by exacerbations of asthma in mixed groups of participants. The Panel concluded that the evidence is “sufficient” to infer a causal association between traffic exposure and exacerbations of asthma but that it is “inadequate and insufficient” to infer a causal association between exposure and respiratory symptoms in children without asthma.

Nine studies assessed the association between exposure to traffic-related pollution and the use of health-care services to treat respiratory problems in children. Most of the studies reported positive associations between exposure and hospital-admission rates, but the majority had methodologic problems that hampered their interpretation. The panel concluded that there is “inadequate and insufficient” evidence to infer a causal association.

Respiratory Health Problems in Adults: Asthma Onset and Respiratory Symptoms

The Panel noted that the evidence between exposure to traffic-related pollution and new adult asthma was “inadequate and insufficient” as this was investigated in only one study (Modig et al. 2006). The Panel reviewed 17 studies on respiratory symptoms, of which all but one relied on proximity to roads or traffic-density measures, and concluded that the evidence for a causal association is “suggestive but not sufficient.”

Toxicology

The few human studies in which subjects were exposed to realistic traffic conditions (a road tunnel or busy street) are supportive of the possibility that persons with asthma may be more susceptible to adverse health effects (such as decrements in lung function and enhanced responses to allergens) related to such exposure. The Panel’s evaluation of the toxicologic data on the respiratory system regarding the effects of components of traffic-related air pollution was that such exposures result in mild acute inflammatory responses in healthy individuals and enhanced allergic responses in allergic asthmatics and animal models.

When the epidemiologic and toxicologic data were viewed together, the Panel noted that a case could be made that there are likely to be causal associations related to exposure to traffic-related air pollution and asthma exacerbation and some other respiratory symptoms. However, given the lack of a large body of toxicologic data based on human and animal exposures to real-world traffic scenarios, the Panel noted that it was hazardous to conclude that causality has been established at this time for all respiratory symptoms at all ages.

LUNG FUNCTION AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Changes in lung function are considered reliable markers of health that reflect the effects of endogenous and cumulative exposure to exogenous factors that might have adverse health consequences. Reduced lung function is strongly associated with future morbidity from a variety of causes and is a predictor of life expectancy (Hole et al. 1996); however, the relevance to health of small, short-term changes has not been assessed. The Panel considered lung function and chronic obstructive pulmonary disease (COPD) together in this review, because the principal criterion for the diagnosis of COPD is based on lung-function measures.

Epidemiology

Lung Function in Children and Adults The studies reviewed were heterogeneous in their design, approach to exposure assessment, and lung-function measures. Given their limited comparability, the Panel concluded that the evidence is “suggestive but not sufficient” to infer a causal association between short- and long-term exposure to traffic-related pollution and decrements in lung function. However, in the case of long-term exposure, there was some coherence in the data, suggesting that (1) long-term exposure is associated with changes in lung function in adolescents and young adults; (2) lung-function measures are lower in people who live in more polluted areas; and (3) changing residence to a less-polluted area in one study is associated with improvements in lung function (Burr et al. 2004). The first and second points are consistent with longer-lasting effects on lung structure and/or function. The third point can be interpreted to indicate that some component of the apparent effects on lung function is reversible or is more the result of short-term exposure.

Chronic Obstructive Pulmonary Disease Because only two of the COPD studies fulfilled the criteria for inclusion in the review and their results were not consistent, the Panel concluded that there is “inadequate and insufficient” evidence for causal associations between exposure to traffic pollution and COPD.

Toxicology

A very limited database of controlled human exposure has shown short-term reductions in forced expiratory volume in 1 second (FEV₁) and increases in inflammation with exposure to traffic-related air pollution. However, the two end points have not been associated with each other. Virtually no data are available from animal models. There are no studies of traffic-related air pollution and COPD.

While the epidemiology studies do provide suggestive evidence for chronic exposure effects on lung function in adolescents and young adults, there are too few toxicologic data to indicate what mechanisms underlie these observations. The aggregate epidemiologic and toxicologic evidence on chronic exposure to traffic-related air pollution and altered lung function in older adults and the occurrence of COPD is too sparse to permit any inference with respect to causal association.

ALLERGY

Epidemiology

The 16 epidemiology studies on this outcome included in the review not only had to meet criteria for the quality of their exposure data but also had to report at least one of the following: (1) positive skin-prick testing for common aeroallergens; (2) serum-specific IgE to common aeroallergens; (3) a physician’s diagnosis of eczema or allergic rhinitis; or (4) use of questionnaires on the history of symptoms of hay fever, seasonal runny nose, rhinitis or conjunctivitis, or itchy eyes. With a few inconsistent exceptions, results based on the skin-prick test reactivity or allergen-specific IgE failed to show associations with any of the traffic-exposure surrogates. Inconsistent results with self-reported symptoms were also noted. The Panel concluded that there is “inadequate and insufficient” evidence to infer a causal association, or even a noncausal association, between exposure to traffic-related pollution and IgE-mediated allergies. Overall, the lack of consistency across epidemiology studies might have reflected a failure to identify susceptible subgroups.

Toxicology

The Panel noted that the toxicology data provide strong mechanistic evidence with respect to the diesel particle component of traffic-generated pollution and IgE-mediated allergic reactions and some evidence for NO₂ and late-phase response to allergen. However, the epidemiology studies were inconsistent. The relevance of the toxicology studies (often by nasal instillation with diesel exhaust particles) to the actual manifestations of non-asthmatic allergic phenotypes (e.g., allergic rhinitis or conjunctivitis, eczema, serum-specific IgE, and evidence of sensitization to aeroallergens) could not be determined.

BIRTH OUTCOMES

Epidemiology

Although a considerable body of data from around the world has identified consistent associations between exposure to ambient air pollution in general and various birth-outcome measures (low birth weight, small for gestational age, and perinatal mortality), only four studies of exposure to traffic-related pollution met the criteria for inclusion in this review. The small number of studies and their limited geographic coverage led the Panel to conclude that there is “inadequate and insufficient” evidence to infer causality.

Toxicology

The toxicology studies reported effects on reproductive organs and sperm functionality in animals, but these outcomes were not evaluated in the epidemiology studies. Among the challenges in interpreting these results are the data limitations and the almost-universal use of very high exposure concentrations that have questionable relevance to actual ambient concentrations. Due to their lack of overlap, the epidemiology and toxicology studies on reproductive health and birth outcomes do not lend themselves to any overall synthesis.

CANCER

Epidemiology

The Panel focused on general-population exposure studies and did not review the extensive epidemiologic literature on cancer from occupational exposure to traffic emission constituents (e.g., benzene and diesel exhaust). Among the studies reviewed, five were of childhood cancers (mainly leukemias, lymphomas, and cancers of the central nervous system), and four of adult cancers (two of lung cancer, one of female breast cancer, and one of several cancers combined). Data on childhood cancers were inconclusive in terms of overall consistency and of specific cancers. Too few data were available in adults. Overall the Panel concluded that the evidence was “inadequate and insufficient” to make inferences for causality between exposure to traffic pollution and cancer.

Toxicology

The toxicologic research summarized included in vitro mutagenicity studies of exposure of cells to PM from traffic pollution, diesel or biodiesel exhaust, and organic components of some of these mixtures, as well as animal carcinogenicity studies after exposure to exhaust from diesel and gasoline-fueled engines. Although studies in cells demonstrating the capacity of DEP to induce DNA-strand breaks, base oxidation, and mutagenicity provide a possible mechanism for the induction of carcinogenicity by traffic-related pollution, the applicability of in vitro mutagenicity studies to human risk assessment has been questioned.

Animal studies have demonstrated the ability of high concentrations of exhaust components in both diesel and gasoline-fueled engines to cause tumors in animals. However, caution must be exercised in extrapolating these data to people exposed to much lower concentrations of pollutants, as seen in the epidemiology studies. Therefore, the Panel concluded that any statement that tries to relate the toxicologic to the epidemiologic data is premature at this time.

OVERALL CONCLUSIONS

Studies have shown that traffic-related emissions affect ambient air quality on a wide range of spatial scales, from local roadsides and urban scales to broadly regional background scales. Based on a synthesis of the best available evidence, the Panel identified an exposure zone within a range of up to 300 to 500 m from a major road as the area most highly affected by traffic emissions (the range reflects the variable influence of background pollution concentrations, meteorologic conditions, and season).

Surrogates for traffic-related exposure have played, and are likely to continue to play, a preeminent role in exposure assessments in epidemiology studies. The optimal selection of relevant surrogates (especially surrogates that are single chemicals) depends on accurate knowledge of the degree to which they represent the chemical and physical properties of the actual primary traffic-pollution mixtures to which humans are exposed, which, in turn, depends on accurate knowledge of motor-vehicle-emissions composition and near-source transformation and dispersion. The Panel concluded that none of the pollutant surrogates (CO, NO₂, UFP, EC, and benzene) is unique to emissions from motor vehicles. Among the surrogates based on traffic-exposure models, the question remains as to the extent to which the proximity model (i.e., the simple distance-to-road measures) should be employed in future epidemiology studies because it is particularly prone to yielding measures potentially containing extraneous information that can lead to the confounding of associations between health effects and exposure. In the Panel's view, the hybrid model is the current optimal method of assigning exposures to primary traffic-related pollution.

Many aspects of the epidemiologic and toxicologic evidence relating adverse human health effects to exposure to primary traffic-generated air pollution remain incomplete. However, the Panel concluded that the evidence is sufficient to support a causal relationship between exposure to traffic-related air pollution and exacerbation of asthma. It also found suggestive evidence of a causal relationship with onset of childhood asthma, nonasthma respiratory symptoms, impaired lung function, total and cardiovascular mortality, and cardiovascular morbidity, although the data are not sufficient to fully support causality. For a number of other health outcomes, there was limited evidence of associations, but the data were either inadequate or insufficient to draw firmer conclusions. The Panel's conclusions have to be considered in the context of the progress made to reduce emissions from motor vehicles. Since the epidemiology studies

are based on past estimates of exposure from older vehicles, they may not provide an accurate guide to estimating health associations in the future.

In light of the large number of people residing within 300 to 500 m of major roads, the Panel concludes that the sufficient and suggestive evidence for these health outcomes indicates that exposures to traffic-related pollution are likely to be of public health concern and deserve public attention. Although policy recommendations based on these conclusions are beyond the scope of this report, the Panel has tried to organize, summarize, and discuss the primary evidence in ways that will facilitate its usefulness to policy makers in the years ahead.

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