

# Ambient Air Pollution and Population Health: Overview

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In November 2003 approximately 200 researchers, stakeholders, and policymakers from more than 40 countries gathered to discuss the science and policy implications of air pollution and human health as part of the AIRNET/NERAM Strategies for Clean Air and Health initiative. The purpose of this paper is to review the more than 35 research posters presented at the conference, including exposure, toxicological, and epidemiological studies of air pollution. Collectively, these papers support previous evidence that both short- and long-term exposures to particulate air pollution have adverse population health impacts, including effects on children. Cellular studies also suggest that air pollution can cause mutagenic and oxidative effects, raising concerns about carcinogenicity and cellular regeneration. Studies of biomarkers, such as Clara-cell proteins and lymphocyte damage assessment, provide further evidence of air pollution effects at the cellular level. Other studies have focused on improvements to measurement and sources of air pollution. These studies suggest that particle mass rather than particle composition may be a more useful indicator of potential human health risk. It is well known that emissions from transportation sources are a major contributor to ambient air pollution in large urban centres. Epidemiologic researchers are able to reduce bias due to misclassification and improve exposure assessment models by allocating air pollution exposure according to distance from traffic sources or land-use patterns. The close association between traffic patterns and air pollution concentrations provides a potential basis for the development of transport policies and regulations with population health improvements as a primary objective. The results of the research presented here present opportunities and challenges for the development of policies for improvements to air quality and human health. However, there remains the challenge of how best to achieve these reductions.

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European-funded thematic network on pollution and health. NERAM is the Canadian Network for Environmental Risk Assessment and Management, which fosters the integration of scientific knowledge and expertise to support efficient environmental protection practices and decision making. The purpose of this paper is to review the more than 35 research posters presented at the AIRNET/NERAM conference. The studies have been organized into the following areas: studies of air pollution exposure, studies of the effects of air pollution on human and animal toxicology, epidemiological studies, and studies providing valuable insights using alternative methodologies and approaches. We conclude with a summary of the research findings and speculate on some of the implications for the development of policy to prevent population health impacts from air pollution exposures.

## EXPOSURE ASSESSMENT

### Sources of Variation

As part of the HEAPSS project (Health Effects of Air Pollution on Susceptible Subpopulations), Aalto et al. (2003) used an ultrafine particle database with information from five European cities, to quantify the risk of hospitalization and of death due to air pollution. Particle measurement sites were selected to represent urban background concentrations. Initial reviews of the particle data suggest an increase in ultrafine particle concentrations is associated with decreasing latitude in the winter months and with temperature inversion events. The Aarnio et al. (2003) adjunct research on traffic-specific particle exposures improves estimates of the actual concentrations experienced while commuting in urban areas. Particle samples were drawn from locations specific to commuting by train, metro, bus, or by foot. Preliminary conclusions reveal significant differences between urban background and traffic-specific locations in terms of concentration exposures and particle size.

### Particle Composition

Exposure studies have also focused on the hypothesis that the physiological reaction to particulates will vary depending on particle composition. Cassee et al. (2003) examined the

qualitative differences among particles collected from different sites throughout Europe. Aside from the usual increase in particle concentrations during the winter months, the study also found significant contrasts in chemical composition as a function of location. For example, metal concentrations were higher in Rome, whereas higher concentrations of magnesium and vanadium were found from samples collected in Amsterdam. In a similar study, De Berardis et al. (2003) studied the composition and physicochemical characteristics of particles less than  $2.1 \mu\text{g}/\text{m}^3$  in Rome using scanning electron microscopy (SEM) and ion chromatographic techniques. Initial conclusions indicate a predominance of particles originating from vehicular sources, except during winter, when particles are released through combustion of heating oil or methane gas for heating purposes. Exposure to specific chemicals associated with carbonaceous particles will typically vary with climatic and transportation patterns.

### Source Apportionment

Urban populations experience heavier exposures to traffic-related air pollution concentrations. Frequency of exposures increases with the density of traffic and with traffic congestion, a trend common in many large urban centers, and magnitude of exposure increases among higher density populations. However, it is interesting to note that a majority of exposure to traffic and combustion related by-products takes place indoors. Iacqua and Jantunen (2003) reported that volatile organic compounds (VOCs) from tailpipe emissions comprise the largest contributor to personal exposure, with more than two-thirds of the exposure taking place indoors. Transportation is also a source of particulate air pollution and is highly variable, depending on weather conditions and traffic flow densities. In the United Kingdom, for example, research has shown that an average of 25% of particulate pollution originates from road transportation and that this figure rises to 75–80% on high pollution days (O'Connell & Matthews, 2003).

### Temporal Trends

Epidemiological assessment of the population health risks from air pollution could not be performed without the construction of high-quality, long-term data sets. Of particular interest are ultrafine particles, as research is beginning to reveal the inverse relationship between particle size and risk to health. Particle concentrations are time and location dependent. For example, Marconi et al. (2003) found that ultrafine particle concentrations were much greater in winter than in summer, were highly correlated with hours of high traffic volumes, and were lower on weekends than on weekdays. They also found that proximity to high traffic locations plays an important role in determining exposure. Background sites characteristically have particle concentrations up to 50% lower than with sites located near high traffic areas. Also of population health significance is the development of methods for the derivation of

long-term air pollution concentration series. Metz (2003) correlated source emission contribution data with information on available air quality data to develop a model useful for forecasting future particle emissions and ambient concentrations. These types of series will be useful for estimating the impact of population health interventions associated with technological improvements, transportation policies, and strategies employed to reduce human exposure, especially among susceptible populations.

## HUMAN TOXICOLOGY

### Biomarkers

Biomarkers are used to improve assessments of biological plausibility in the relationship between air pollutants and human health effects. Clara-cell proteins, a promising biomarker, have been identified as sensitive markers of increased permeability of the lung epithelial barrier, and an increase in serum levels has been associated with ambient ozone concentrations. Ozone exposures under controlled laboratory conditions among healthy adults resulted in a significant increase in serum levels of Clara-cell proteins up to 6 h after initial exposure (Blomberg et al., 2003). However, the influence of serum baseline diurnal variations is uncertain and further research is warranted. Research has also focused on identification of specific biological reactions from exposure to fine particulates. Research has also shown that fine particles may have direct or indirect effects on blood coagulation processes and thus physiological impacts affecting cardiac function. To evaluate the biological effects of fine particles Tarrona et al. (2003) examined a cohort of healthy adults between the ages of 20 and 55 yr working in clean industrial environments, such as the semiconductor and pharmaceutical industries, and living in urban areas. Markers of blood coagulation were assessed at three points—before the work shift, after the work shift, and after exposure to outdoor ambient air during commuting or social and domestic routines—with the goal of providing a plausible biological explanation for the ecologic link between exposure to particulate air pollution and myocardial infarction.

### Genotoxicity

Genotoxic chemicals are able to cause damage to DNA but do not inevitably lead to the creation of cancerous cells (PTCL, 2004). Inorganic and organic compounds that dominate the composition of fine particulate matter air pollution may cause genotoxic health effects, although the extent of genotoxicity is not well known. Brits et al. (2003) collected fine particulate matter from urban and industrial sites in Belgium to evaluate the mutagenic and cytotoxic properties. Particle mass concentrations of both  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  were significantly higher in the industrial area. Human alveolar epithelial cells were exposed

in vitro to particulate matter for a 48-h period to investigate toxicological potential. Although particles from both size fractions showed no significant toxicity, the micronucleus frequencies in binucleated cells were significantly increased, indicating potential mutagenetic effects.

Gábelová et al. (2003) assessed the genotoxicity of the organic components associated with particulate air pollution. The organic components of particulate matter were extracted from particulates collected at three sites from three countries. The organic components induced a seasonal and dose-dependent in vitro increase of DNA damage in human cells. Effects were most pronounced for coarse particles in the 2.5–10  $\mu\text{m}$  diameter range.

Guastadisegni et al. (2003) investigated the relationship between the chemical composition of particulate matter air pollution and toxicity. An analysis of 32 samples collected from a variety of European metropolitan and rural areas sought to assess the oxidative activity of particles. The investigators created a synthetic model of the respiratory tract lining fluid and measured the depletion rates of ascorbate, urate, and reduced glutathione following a 4-h exposure to 50  $\mu\text{g}/\text{ml}$  particle concentration. Inflammatory response was measured by evaluating the release of arachidonic acid, tumor necrosis factor alpha, and interleukin-6 from a macrophagic cell line. The oxidative potential of particulate matter air pollution seems strongly related to a capacity to induce arachidonic acid from the macrophages and can be explained by the bioavailability of iron specific to every particle. However, these relationships were only apparent in the coarse fraction.

The aim of the EXPAH studies (Effects of polycyclic aromatic hydrocarbons [PAHs] in environmental pollution on exogenous and endogenous DNA damage) is to evaluate the role of PAHs as a source of genotoxic activities of organic mixtures associated with particulate air pollution. The effect of DNA damage is monitored by determination of chromosome aberration, and susceptibility in populations is studied by investigating the effect of metabolic polymorphisms in carcinogen metabolism and DNA repair. A case-control design was adopted to evaluate genotoxicity in exposed and less-exposed groups in the cities of Prague, Kosice, and Sofia. The exposed group consisted of police (as well as bus drivers in Sofia), and the control groups were volunteers who spent more than 90% of their time in indoor environments. The role of competing risks factors such as diet and other lifestyle factors was also evaluated.

As part of the EXPAH studies, Cebulska-Wasilewska et al. (2003) investigated cellular susceptibility to polycyclic aromatic hydrocarbons (PAHs). Damage to DNA was evaluated as a function of lymphocyte repair competence. No significant differences were detected between the referent and exposed groups, although residual damage to DNA and kinetic repair is less efficient among those with greater exposure to PAHs. Lymphocyte repair rates were also lower for those with less education, implying a possible role of education as

an effect modifier in the relationship between exposure to PAHs and genetic injury. Studies of the potential for oxidative damage to DNA were also performed to investigate the differences between exposed and less exposed groups. Some significant results were detected between cities, although most conclusions were mixed (Singh et al., 2003). Sram et al. (2003a, 2003b) used DNA adducts analysis and a common molecular cytogenetic technique called fluorescence in situ hybridization (FISH) to assess genotoxic risk.

The results are of interest for two reasons. First, the FISH technique seems to be an efficient tool to detect chromosomal aberrations and thus useful as a sensitive biomarker of exposure to PAHs. Second, the analysis detected significantly increased FISH cytogenetic parameters in nonsmoking policemen and bus drivers (exposed group), indicating an association between increased exposure to particulate air pollution with PAHs and potential genotoxic effects.

### Source Toxicity

Exposure to air pollution, and fine particulate pollutants, can originate from either point or line (mobile) sources. As opposed to typical epidemiological investigations designed to assess health risk within a population, toxicological studies will assess the level and variability of chemicals within the bloodstream or serum to determine the level of exposure to a specific pollutant source. From these studies it is then assumed that these chemicals will lead to measurable health impacts or possible mortality. Individuals in a toxicological study are generally grouped according to level of exposure, for which distance can be used as a suitable proxy.

For example, Fierens et al. (2003) conducted a study to quantify the amount of dioxins, polychlorinated biphenyls (PCBs), and heavy metals in the bloodstreams of residents living in the vicinity of a municipal solid waste incinerator. A comparison or control group living in an unpolluted area was used for statistical comparison, and information on lifestyle, dietary, and occupational characteristics was also collected. The study concluded that exposure to emissions from a municipal solid waste incinerator can increase the body burden of dioxin and PCBs but that the increase is dependent on the consumption of animal fat products from the local food chain. The proposed interaction between exposure from the point source and dietary intake provides an indication of the difficulty involved when assessing toxic effects of pollution in a localized ecosystem.

Point sources of pollution may also have significant impacts on highly susceptible subpopulations as well as in the population. Lolova et al. (2003) investigated the relationship between air pollution emissions from a smelter plant and blood lead levels in children, as well as in placenta, maternal, and cord blood. Air quality and ambient lead and cadmium levels were determined from three fixed monitoring stations. Blood lead levels were analyzed from children residing close to the monitoring sites and from a control group not exposed to a specific

point source, as well as from 76 maternal/infant pairs. The investigators found that lead and cadmium concentrations in the vicinity of the smelter were above national guidelines, suggesting that the population is at risk of increased exposure to these chemicals. The researchers also found that blood lead levels in children living within the smelter area were significantly higher than among children living outside the area. Furthermore, the results also indicate a significant relationship between abnormal birth outcomes (preterm births) and significantly higher than normal lead levels in maternal and cord blood.

Mobile sources of air pollution, such as particulate pollution from road traffic, also pose serious health risks. Since much of the air pollution in urban areas arises from motor vehicle emissions, recent research has begun to take interest in the variation of health effects associated with distance from road traffic sources, as well as the variation in particle toxicity associated with density of road traffic. Gerlofs-Nijland et al. (2003) investigated how the toxic potency of particulate pollution varied with levels of traffic exhaust emissions. Particles were assessed for their toxic potential and grouped according to particle size and reactivity. Tests of toxicity were performed *in vitro* (in rats) and *in vivo* (in humans) using indicators of ascorbate depletion, arachidonic acid release, interleukin-6, and damage to DNA (genotoxicity). The researchers found that toxicity varies with particle size, although both fine and coarse particles have adverse health effects. They also found that particles from locations with high-traffic areas induce more effects than those from lower traffic areas.

New research is also probing into the toxicological characteristics of antioxidants as potential protection from vehicle-related air pollution. de Burbure et al. (2003) investigated antioxidant status as a measure by selenium levels and lung response to NO<sub>2</sub> in rats. Selenium-normal and selenium-deficient rats were differentially exposed to NO<sub>2</sub> concentrations ranging from normal concentrations to acute exposures. The conclusions suggest that antioxidants play an important protective role in respiratory response to both acute and long-term challenges from air pollutants such as NO<sub>2</sub>. The results also support previous research of Clara-cell proteins (CC16) as excellent markers of increased lung permeability. Ironically, selenium may interact with CC16 antioxidants, resulting in decreased antioxidant production, thus depriving the lung of its natural capacity to protect against foreign pollutants.

## EPIDEMIOLOGY

### Cohort Studies

Cohort study designs afford an opportunity to investigate the long-term health effects from exposure to air pollution. In North America, large cohort studies such as the Harvard Six Cities study (Dockery et al., 1993), the American Cancer Society studies (Pope et al., 1995; Krewski et al., 2003), and

the ongoing Adventist Smog and Health study (Abbey et al., 1999), have consistently demonstrated associations between long-term exposure to particulate matter and mortality and have played a pivotal role in the establishment of national air quality objectives in both the United States and Canada (Greenbaum et al., 2001). Many newly developed European cohort studies have adopted advanced spatial techniques and will make an important contribution to improving the estimation of human health risk from ambient air pollution.

The European Community Respiratory Health Survey (ECRHS) consists of a 9-yr follow-up prospective survey of more than 13,000 adults including data from 29 research centres located in 14 countries. As part of the air pollution working group, Götschi et al. (2003) are currently investigating correlations between surrogates of exposure to air pollution across 21 European centres and evaluating the influence of air pollution sampler location on air pollution exposure assessment. The SAPALDIA 2 (Swiss Cohort Study on Air Pollution and Lung Diseases in Adults) Swiss cohort study, part of the ECRHS study, is assessing associations of long-term air pollution exposure with the 10-yr course of respiratory and atopic symptoms, diseases, and markers of respiratory health and of atopy and cardiovascular health among 9651 adults. Participants have been followed for address changes and mortality since 1991 and 83% of the original cohort is available for continued study. A geographic information systems (GIS)-based approach was used to derive individual exposure assignments from traffic-related air pollution exposures while also taking into consideration historical ambient pollutant levels and participant mobility patterns (Bayer-Oglesby et al., 2003). Although results are still forthcoming, the SAPALDIA 2 study constitutes a novel approach to the creation of a cohort from an original cross-sectional study and represents a novel approach for detecting the long-term health effects of low-level air pollution.

Cohorts have also been created to assess the impacts of air pollution on morbidity outcomes. A study called HEAPSS (Health Effects of Air Pollution on Susceptible Subpopulations) was designed to assess whether air pollution increases the risk of first acute myocardial infarction (MI) or the subsequent risk of cardiac events among patients who have survived their first MI (Lanki et al., 2003). Using a common protocol, the cohort will consist of approximately 27,000 cases of nonfatal MI derived from hospital admission registries located in Germany, Spain, Sweden, Italy, and Finland. Although pooled associations were generally weak, the researchers concluded that acute exposure to carbon monoxide (CO) and ultrafine particles (PM<sub>2.5</sub>) increases risk of hospitalization for new MI even after controlling for the potential confounding effects of weather and personal health characteristics.

Many cohort studies of health impacts from air pollution are limited by the quality and extent of exposure data available. For example, many older air pollution monitoring stations were activated to monitor specific point-source pollutants resulting

from manufacturing or industrial processes and not for the measurement of ambient exposures. In Scotland, however, long-term (>50 yr) records exist for measurements of black smoke and sulfur dioxide (SO<sub>2</sub>). Yap et al. (2003) have linked this valuable exposure data set to health outcomes data derived from three existing cohorts consisting of more than 26,000 subjects; they are also able to link each subject to individual hospital admission data and/or to mortality records using the Scottish Health Record Linkage System. Multilevel modeling and GIS techniques will allow for the investigation of potential confounding and effect modification by individual and aggregate level factors, and long-term exposure data will enable an examination of exposure latency durations and temporal variation in pollutant concentrations. Results of this work are forthcoming.

### Cross-sectional Studies

Cross sectional studies measure the prevalence of health outcomes or determinants of health in a population at a point in time or over a short period. This type of information can be used to explore etiology, for example, the association between exposure to air pollution and any variety of health outcomes. Heinrich et al. (2003) in the Bitterfeld study utilized data from three cross-sectional surveys taken over a 7-yr period to determine temporal changes in the impact of ambient pollution on children's health. The study included a total of 5360 children ranging in age from 5 to 7 yr. The children were administered a questionnaire, and subsequent tests of spirometry, histamine response, and blood analysis were employed to assess physiological response. Children from more polluted areas showed poorer respiratory health, and ambient particles were correlated with increased risk of allergenic sensitization.

Another cross-sectional study has also reported acute effects of air pollution on respiratory conditions among susceptible populations. The AUPHEP (Austrian Project on Health Effects of Particulates) project investigated the short-term effect of particulate matter on lung function, morbidity, and mortality in three urban centers (Vienna, Linz, and Graz) and one less polluted rural control area (Neuberger et al., 2003). The investigators found fine particles to be associated with cardiovascular mortality among the elderly of Vienna and with an increase in respiratory-related hospital admissions, and there was an association with lung function impairment among children in Linz. Thus, despite overall improvements to air quality in Austria, particulate matter and traffic-related air pollution still pose human health risks, especially for the elderly and children.

More recently, epidemiological investigations have focused on health effects from traffic-related air pollution—a large source of urban ambient air pollution. The APHEIS (Air Pollution and Health: A European Information System) network has collected air pollution concentration information for 26 European cities, of which 2, in Madrid and Rome, have dedicated traffic-based monitors. Mean concentrations among all sites

ranged from 20 to 45  $\mu\text{g}/\text{m}^3$  for fine particles; however, values from the traffic-related monitors revealed concentrations in the upper end of the distribution at 37 and 43  $\mu\text{g}/\text{m}^3$  for Madrid and Rome, respectively. Cross-sectional analysis of health and air pollution data from 1999 reveal increased chronic and acute effects on mortality in cities with higher vehicular particulate concentrations (Mücke et al., 2003). Vehicular traffic is also a major contributing source of ambient urban air pollution in Tblisi, Georgia. To investigate the potential for health impacts, Samadashvili (2003) examined the leading morbidity outcomes of children between the ages of 7 and 17 yr living either in an area with high traffic related pollution concentrations or in a suburban area with much lower concentrations. Children from the more polluted district were more likely to have some form of respiratory illness and were 2.5 times more likely to contract some form of skin disease.

### Time-series Studies

Evidence of associations between air pollution and human health impacts is derived primarily from time-series studies. A time-series approach is used to associate daily fluctuations in ambient air pollution levels with daily rates of mortality or morbidity, and such studies have consistently demonstrated increased risk to health on or shortly following a day with increased ambient pollution levels, in urban centers worldwide. The analysis of time-series data also requires careful consideration of seasonal trends in the data, as well as adjustment for important covariates such as climate and gaseous copollutants (Burnett et al., 1995).

In Reggio Emilia, Italy, a time-series approach was adopted to evaluate the short-term effects of urban air pollution levels on children's emergency visits from respiratory causes (Bedeschi et al., 2003). Researchers used generalized additive regression models to develop risk estimates of emergency visits after controlling for temporal and weather-related influences. Statistically significant and positive associations were found for NO<sub>2</sub> (11% increase in emergency visits) and for PM<sub>10</sub> (3% increase). An analogous statistical approach was adopted by Vigotti et al. (2003) to investigate the association between ambient air pollution (CO, NO<sub>2</sub>, and PM<sub>10</sub>) and presentations to the emergency room for respiratory-related complaints by children and elderly patients in Pisa, Italy. A significant increase in emergency-room visits was confirmed for children and the elderly resulting from particulate pollution exposure, after controlling for weather and temporal variability.

An important but often overlooked consideration in the evaluation of time-series studies linking particulate air pollution levels with health outcomes is the influence of specific metal concentrations located on the particle surface compared to the particle mass concentration alone. To examine this issue more thoroughly, Beverland et al. (2003) conducted a long-term study to investigate the relationship between elevated respiratory and cardiovascular mortality and morbidity rates and

the variance of metal composition of  $PM_{10}$  in urban and rural locations in Edinburgh, Scotland. The researchers used a novel air mass trajectory approach to determine the particle characteristics of air masses common to the Edinburgh area. Air masses centered on the United Kingdom or originating from east/central Europe were associated with a 25% greater concentration of both  $PM_{10}$  and  $PM_{2.5}$  when compared to air masses from other regions. A forward stepwise multiple-regression modeling approach was adopted to examine the fraction of water-soluble metal concentrations (Fe, Cu, Ni, V, Zn) as a constituent of particle mass. Generalized additive models were used to investigate the associations between health outcomes and either particle mass or metal fractions after controlling for particle mass. A positive, significant association was found between cardiovascular admission and a  $10\text{-}\mu\text{g}/\text{m}^3$  increase in total  $PM_{10}$  concentrations; although similar associations were found for specific metal fractions, the risk estimates became statistically insignificant after adjustment for total particle mass in the model.

Another novel development in the use of time-series approaches to investigate air pollution-related health effects is the integration of genotyping to identify susceptible subgroups and to determine the role of gene variation as a determinant of inflammatory response. In a proposed study, Peters et al. (2003), as part of the AIRGENE Study Group, will use time-series methods to determine the relationship between inflammatory response and air pollutants among survivors of myocardial infarction in six cities in Europe. Genotyping techniques will be used to provide insight into the mechanisms leading to inflammatory response in an effort to identify and define susceptible subpopulations and prevent the exacerbation of cardiovascular disease from ambient particle concentrations.

Several issues associated with the adoption of time-series approaches are noteworthy. First, although time-series analyses provide important information about the effects of short-term exposure on mortality, they are unable to identify effects with a lag period longer than several weeks (Dominici et al., 2000, 2003). Second, research has recently shown that time-series approaches can lead to overestimation of risk as well as overstatement of the precision of risk estimates, due to convergence problems in the generalized additive model (Ramsay et al., 2003a, 2003b). A correction to this problem using more stringent convergence criteria confirms that mortality is associated with short-term fluctuations in particulate air pollution (HEI, 2003). Third, health effect estimates from time-series studies are sensitive to a number of assumptions, including model and parameter selection, comparison of health effect estimates to zero pollution levels, issues related to confounding from multipollutant mixtures, and nonstandardized reporting methods (Sahsuvargolu & Jerrett, 2003). It may be helpful in future research for investigators to explore more fully the consequences of uncertainty in the calculation and reporting of health risk estimates from exposure to air pollution.

## OTHER STUDIES

Recent research contributions have illuminated the health impacts of air pollution at subregulatory concentrations, and have highlighted the need for researchers to consider the roles of human activity patterns and meteorology as influential in the air pollution and health risk relationship. An extensive review of peer-reviewed published research on the health effects from ozone suggests positive significant associations to health outcomes below the 40–80 ppb 8-h average range (Walton, 2003). Ambient concentrations of ozone below current Belgian air quality standards have been associated with inflammatory changes in the airways of children (Nickmilder et al., 2003). Research has also revealed the importance of considering time-activity data and exposures to common chemicals as potential risks to the development of respiratory and cardiopulmonary diseases such as asthma (Bernard et al., 2003). Moreover, attention should be directed toward a more thorough consideration of *atmospheric* risk factors than just air pollution. Air mass analysis and other meteorological approaches to controlling for the health impacts from variations in weather will help to elucidate the potential for weather/air pollution interactions and will enable public health authorities to develop population health impact prediction systems based on atmospheric risk factors (Michelozzi et al., 2003).

## SUMMARY OF FINDINGS

The health effects of air pollution research posters represent important new findings from more than 41 cities and metropolitan areas across Europe. Consistent with previous air pollution and health research, many studies found children to be particularly susceptible to ambient concentrations of air pollution in urban areas, resulting in decreased respiratory function and related morbidities. It is also clear that population health impacts are evident from both short- and long-term exposures to PM (Glorennec & Monroux, 2003). At the cellular level, studies reveal air pollution to have potential mutagenetic and oxidative effects, which raises serious concern for the potential for oncogenicity and healthy cellular regeneration. Studies of biomarkers, such as Clara-cell proteins and lymphocyte damage assessment, present the possibility of identifying air pollution effects at the cellular level and contribute to the evidence of air pollution as a causal factor for human health impacts.

At the other end of the spectrum, studies have focused on improvements to air pollution measurement and sources. For example, a few studies have shown that it may be more prudent and efficient to assess particle mass rather than particle composition as an indicator of potential human health risk. Studies of the impact to health from air pollution are also moving upstream. It is well known that emissions from transportation sources are a major component of the total ambient air pollution concentration in large urban centers. From an epidemiological perspective, researchers are able to reduce bias due to

misclassification and improve exposure assessment models by allocating air pollution exposure according to distance from traffic sources and/or land-use patterns. From a policy perspective, the close association between traffic patterns (frequency and mode) and air pollution concentrations leads to the development of transport policies and regulations with population health improvements as a primary objective.

## POLICY IMPLICATIONS

The results of the research presented here provide many opportunities and challenges for the development of policies for improvements to air quality and human health. Policymakers interested in morbidity and mortality related to air pollution should be made aware that the moderate and prolonged exposures, rather than peak exposures, are responsible for a majority of human health effects. Of particular concern are the critical pollutants—respirable forms of particulate matter, carbon monoxide, nitrogen oxides, and volatile organic compounds—that have been linked expressly to vehicle and transportation-related emissions. The results from epidemiological research suggest there is insufficient evidence to depart from assumptions of linearity in associations between air pollution and human health. Benefits to health may be realized from any reductions in air pollution, inter alia pollution sources from transportation. Policymakers are thus presented with an opportunity to create population health improvements by giving priority to reducing ambient pollutant levels from traffic sources, especially in residential areas. The challenge remains, however, of how best to achieve these reductions.

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