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LETTERS

Urban Air Pollution and Mortality

Dear Editor:


In the above article, Burnett et al. reported that 8.2% of all non-accidental deaths from 1980-1991 were attributed in a time-series analysis to four gaseous air pollutants, nitrogen dioxide, sulphur dioxide, ozone and carbon monoxide. Such a high risk of premature mortality from contemporary levels of the four gases is surprising, as is the absence of risk from PM_{2.5} (respirable particulate matter less than 2.5 μm in average diameter). These surprising results, if confirmed, make this paper very important in a public health context by suggesting that a measurable, perhaps even dramatic improvement in population health can be obtained by reducing gaseous air pollution. However, these findings must be critically evaluated.

The high mortality indicated in Burnett et al.’s paper is inconsistent with other studies, and the authors do not acknowledge those differences. Time-series studies, in which area monitors serve as surrogate data for individual exposures and study populations, are assumed to be homogeneous (i.e., ecological study designs) and are useful in detecting associations, such as that in the present study, but such hypothetical associations require validation by other methods using other data sets. The authors’ analysis ignores a massive amount of traditional epidemiology, human clinical and animal toxicology research that finds no mortality resulting from exposures to the four gases at the low levels typical in Canada.

Burnett et al. do not provide sufficient details about their models to allow critical analysis. A host of investigator-specific decisions are incorporated into the statistical models and analyses, and many of these decisions influence the reported associations. These same authors have previously stated, “detecting an air pollution effect with time-series methodology requires the appropriate use of complex statistical techniques in order to tease out a relatively small signal from a forest of noise. The art of the analysis is to remove the proper amount of unwanted temporal variability in the time series, thus revealing the ‘true’ air pollution signal.” A means of confirming the validity of the “signal” in the present study is needed.

Among specific concerns, the selection of averaging time, days lagged and filters should be made a priori and should be based on some objective statistical criteria, such as what variable reduces autocorrelation to a minimum or causes the largest decrease in the log likelihood function, not based on maximizing relative risks. The use of biological criteria would be even more persuasive as a basis of modeling decisions and might suggest who (e.g., what sub-population) is at risk, which is a critical issue. The paper fails to provide statistical criteria used to select various model parameters, omits exposure-response functions, and fails to seriously treat biological plausibility. Rather, the authors note that different gases contribute to the overall risks differently in each city with no consistent pattern within regions. For example, Table IV shows the “increased risk” from nitrogen dioxide varies in different cities from 0 to 9.4% and that of sulphur dioxide from -0.3 to 2.9%. Is such variation indicative of a real, consistent effect, or is this an argument against causality?

Unfortunately, Burnett et al. do not address these critical issues, yet they appear to have succeeded where other investigators have failed, using the time-series approach alone to identify a combination of the four air pollutants (most of which are correlated) as a major cause of increased mortality at low ambient levels.

The strength of the opinions expressed in the article is disquieting for the additional reason that the four gases were ...see Letters, page 238
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shown to “explain” all the mortality previously associated with PM (particulate matter) or its surrogate, sulphate. These same authors have previously attributed significant adverse health effects to sulphate. Their present concern about the four gases is based on an inappropriate comparison of risk reductions derived from two very different models: results from a multi-pollutant model (for 5 Canadian cities) incorporating the four gases but not PM, are compared to results from a single pollutant model (in 6 U.S. cities) for PM$_{2.5}$ and sulphate during a different time period. This comparison does not appear to be appropriate if PM does not improve the predictive power of the multi-pollutant model for the air pollution mix, on morbidity and mortality. Similarly, risks due to different pollutants should be compared using comparable units of measure that take into account differences in means and variances.

Given the inconsistent city-to-city patterns for gaseous pollutants observed by Burnett et al., as well as the inconsistency of their results with others, more research appears to be warranted. For example, it would seem that any examples of risk reductions should be based on data for gaseous pollutants, PM and sulphate in the same cities, over the same time frame, and using the same multi-pollutant model. In addition, validation of these findings by other methods and using other populations are critically needed to reconcile the results from ecological time-series studies with the body of evidence supporting current air quality objectives.

If these surprising results are confirmed, the methods of Burnett et al. should be applied to a variety of control strategies in a comprehensive and planned process in order to contribute to gains in population health. The present study raises an interesting hypothesis which begs questions as to the relative importance to human health of gaseous and particulate matter, and who is affected; it is inappropriate as the basis for a specific risk management decision of the type in their example.

G.C. Granville, Manager
Toxicology and Materials Safety
Shell Canada Limited
400 - 4th Avenue, S.W.
Calgary, AB T2P 2H5

L.A. Gephart, Group Head
Exxon Biomedical Sciences, Inc.
Mettlers Road CN2350
East Millstone, NJ 08875-2350
U.S.A.

R.T. Keefe, Sr. Staff Toxicologist
Imperial Oil Limited
111 St. Clair Avenue West
Toronto, ON M5W 1K3

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Authors’ response

We would like to thank the Journal for giving us the opportunity to further clarify our work and to respond to the letter submitted by Granville, Gephart and Keefe.

The authors of the letter suggest that detecting an association between urban air pollution and daily mortality rates in Canadian cities is surprising. We suggest that not detecting such an effect would be more surprising since such effects have been observed in similar epidemiological studies throughout the world with little evidence to suggest a population threshold. This implies that there exists an association between relatively low concentrations of air pollution and mortality. We had access to large databases and used sensitive methods of analysis to detect effects.

The authors suggest that the relatively low levels of air pollution in Canada would preclude a mortality effect of the size reported in our paper (8.2% increase in mortality). However, this percentage was based on the effects of four pollutants in combination, not just a single pollutant...
reported other drugs (mainly speedballs and PCP). At T1, we found a slight increase in both cocaine (41%) and heroin (58%) use apparently due to a shift from other drugs (which decreased to 1%). On an individual level, however, there was much more variation. Indeed, a total of 28 youth (28%) had changed their choice of drug most frequently injected between the two interviews.

DISCUSSION

The high rate of initiation to injection as well as the high rate of relapse show that injection patterns are unstable among youth. This is also true concerning the drug most frequently injected. The fact that more than a quarter of "stable" users shifted from one drug to another between T0 and T1 underlines the risk, when planning detoxification services, of inappropriately labelling young IDU as cocaine or heroin addicts. The reasons for this shift are still unknown. Availability, peer pressure or individual preferences are possible factors.

Needle sharing is common among young IDU and it seems to begin soon after initiation. This behaviour occurs in a city where syringes are easily available through five needle exchange programmes and numerous pharmacies.

A better understanding of the reasons why young IDU change the drug they most frequently inject as well as why they share injection material is needed in order to make preventive efforts more effective.

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We reconfirm our ‘disquieting’ conclusion that the four gases can explain all the association with mortality in major urban locales with no additional impact of particulate matter. This is not to say that particulate matter is not a predictor of mortality, a conclusion supported by a host of studies. Particulate matter itself is a complex mixture of organic and inorganic matter, whose composition can vary by location and time. Much of the fine particulate matter in urban environments is generated either by the same sources of pollution as the gases, industrial activity and transportation, or from secondary formation in the atmosphere from primary gaseous emissions. Concentrations of the gases and particulate matter are correlated in time and as such, not all these variables are required to explain the total effect of the pollution mixture on health.

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measure like particulate matter. A complete review of the vast literature on this topic is not possible within the context of this response. We agree that our risk estimates are somewhat higher than those reported for ozone, nitrogen dioxide, sulphur dioxide, and carbon monoxide. However, these studies have not considered all these pollutants in combination and thus the reported size of their effects were not as large as those given in our recent study.

Our risk estimates were based on multiple day averages of pollutant concentrations which were shown to be a greater risk factor for mortality than single day measures. This finding implies that the pollution effect is distributed over several days. Some deaths occur on the day of high pollution exposure, other deaths are on the day after, and some additional deaths are observed two days after exposure. We believe that this distributed effect model is a more biologically plausible scenario than a single day effect model.

Granville et al. also suggest that we have ignored the 'massive amount of traditional epidemiology, human clinical and animal toxicology research that finds no mortality resulting from exposures to the four gases at low levels.' Traditional epidemiology and human clinical studies are not necessarily the only appropriate means of assessing effects on mortality. The new methodology of utilizing administrative health and air pollution information with innovative methods of robust statistical analyses of time series data have allowed us to detect adverse health effects at lower concentrations than previously thought possible. Statistical approaches to the analysis of parallel time series of health and environmental information have been well documented and shown to be robust against the type of statistical method employed.
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The inconsistency of risks among cities for each of the pollutants examined separately is similar to that of other studies examining multiple locales. One would expect more variation in the association of the more reactive and/or spatially heterogeneous pollutants such as carbon monoxide, sulphur dioxide, and nitrogen dioxide, with mortality compared to particulate mass since outdoor monitoring data are a poorer surrogate for personal exposure to the former versus latter pollutants.

Furthermore, there are different circumstances in each city potentially leading to differing relationships between the monitoring site data and the population exposures. These differences point out that given the present study it is premature to dwell on differences in risks among cities. Instead we should take note of the fact that positive associations were found under a wide variety of circumstances, as represented by different Canadian cities, and that there was reasonable consistency among cities in the combined effect of the pollutant mixture.

We included the example on the sulphur reductions in gasoline to illustrate the importance of considering the pollution mixture on health when assessing the benefits of pollution reduction strategies and not to rely on the impact of a single pollutant measure such as particulate matter. In a recent review of the health benefits of reductions in sulphur in gasoline, an expert panel was faced with using data from a US study on the association between mortality and fine particulate matter or sulphates, even though several air pollutants were predicted to decrease if sulphur levels in gasoline were reduced.

We attempted to improve on the current state of knowledge by examining Canadian data in multiple locations and including all the available information on pollution concentrations. The example was intended to demonstrate the importance of considering the health effects of urban pollution mixtures. We did not suggest that Canadian air pollution policy be independently developed based on this single study.

We are pleased that this paper has initiated an informed debate on this important public health issue and reiterate that further research may shed some light on these difficult scientific issues.

Richard T. Burnett, Sabit Cakmak, Jeffrey R. Brook

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Value of ecological analysis

Dear Editor:


The paper by Elliott and Dean is a welcome addition to the literature. It highlights the role of the ecological approach in the understanding of disease etiology in ... see Letters, page 273
researchers interested in issues of importance to public health, health care policy, and health care system management, its utility with respect to understanding the determinants of population health will also be enhanced, as other databases are ‘linked in’.

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both human populations and individuals.
As noted by the authors, among others, studies including ecological or group-level variables are necessary to examine the causal link between social environments, human behaviour, and disease development.

It was therefore surprising to read the conclusions of this paper, which seem to indicate that the ecological approach has limited utility in understanding disease etiology. Elliott and Dean write: "The difficulties of studying health issues…indicate that caution must be exercised when interpreting these results. Most important, this is ultimately an ecological level analysis. In order to link heart disease and psychosocial stress, the ideal dataset would contain measures of heart disease outcomes and indicators of stress, all preferably measured at the individual level [emphasis added], allowing conclusions to be made about individual level [emphasis added] relationships.”

Presumably the basis for this view is the "ecological fallacy", “a logical fallacy inherent in making causal inferences from group data to individual behaviours.”

In the epidemiologic literature this fallacy has discredited the ecological approach, with ecologic or group-level studies being viewed as crude attempts to determine individual-level associations.

Ecologic studies, however, are not simply substitutes for individual-level correlational studies. Very often a variable measured at the group level reflects a different construct or phenomenon than the same variable measured at the individual level. To illustrate, Schwartz writes: “The construct referenced on the ecological level may be the context or social environment in which individuals live, distinct from the attributes of those individuals. Thus, poverty as an individual characteristic and poverty as a neighbourhood characteristic may exert different, independent effects on health.”

By viewing ecological studies as mere substitutes of individual-level studies, the potential independent effects (or causal influences) of the broader social environment (e.g., families, peer groups, communities, and legislation) are ignored or neglected. The utility of the ecologic approach is that it provides a unique opportunity to ascertain the social or group contexts which contribute to disease risk.

In the case of heart disease (and most other illnesses), it is likely that a multi-level analysis including both individual-level variables (e.g., physiologic risk factors) and ecologic-level variables (e.g., job characteristics) will lead to a better understanding of etiology, in particular the mechanisms by which environmental stressors influence perceptions of stress and disease risk in individuals. Multi-level analysis will also indicate whether interventions to reduce risk should focus on the individual level, or the ecological level, or some combination of both.

Janice Husted, PhD
Associate Professor, Department of Health Studies and Gerontology, University of Waterloo, Waterloo, ON

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