Letters to the Editor

Inappropriate use of daily mortality analyses: A response

From BART D. OSTRO AND LAURAIN E G CHESTNUT

Sir—in a recent article in this journal, McMichael et al.1 present their interpretation of the proper inferences that can be drawn from epidemiological studies relating air pollution to mortality based on daily time-series analysis. They describe work we have done to estimate premature mortality reductions achievable with reductions in air pollution as an example of an improper inference from this literature. We disagree with their conclusions for two key reasons. First, they base their arguments on the time-series studies alone without taking into account the implications of other types of study that also show an association between mortality and air pollution. Second, they assert, without empirical support, that the results of the time-series studies represent an inconsequential mortality risk that should not be summed to annual premature mortality counts.

Three types of study have demonstrated an association between particulate matter air pollution and mortality. First, over two dozen time-series studies have demonstrated associations between daily fluctuations in pollution within a given city and fluctuations in all-cause or disease-specific mortality.2–4 These studies show that when daily pollution levels increase, mortality counts increase shortly after. Second, prospective cohort studies have compared survival rates across cities with varying long-term pollution levels, after controlling for many individual factors that may affect mortality such as age, sex, smoking and occupational exposure.5–6 Finally, cross-sectional studies have compared city-wide annual mortality rates against a continuum of average pollution levels—again controlling for a host of potential confounding factors. These latter two types of study are often interpreted as demonstrating the impact of long-term exposure to air pollution. Armed with this evidence, several analysts have developed concentration-response functions to predict the number of cases of premature mortality that might be reduced if existing air pollution levels were reduced.7 Some studies go on to estimate the economic value of these findings.8

McMichael et al. argue that it is not appropriate to use the results of time-series studies to calculate the annual number of excess deaths associated with air pollution.1 Their argument is based on two points. The first is that they assert that short-term fluctuations in air pollution are shifting expected deaths by only a few days. Their interpretation is that people dying on a given day from elevated pollution exposure are likely to be in poor health already and probably would have died within a few days anyway. This effect is sometimes called mortality displacement or harvesting. Their second point is that because one cannot estimate the number of life-years lost from premature deaths observed in the time-series studies, it is not appropriate to sum the excess daily deaths to achieve an annual count of premature deaths.

There are several reasons why these arguments are not supported. First, there is no empirical evidence that the time-series results are dominated by a harvesting effect. Direct tests of the harvesting hypothesis have not shown a significant harvesting effect and evidence from the long-term exposure studies suggests a significant air pollution effect on mortality risk, which contradicts the harvesting hypothesis. Finally, there is a difference between estimating the number of cases of premature mortality and determining the welfare significance of these cases.

Although it is reasonable that some amount of harvesting probably does occur, there is no evidence that it dominates the time-series results. McMichael et al. reference Spix in support of their argument for harvesting.9 Spix reported that the mortality effect was minimally reduced on days following a 15-day period of high daily average mortality. However, Spix reported that the hypothesis for no harvesting could not be rejected based on standard statistical tests of significance. In addition, more recent work does not support the harvesting-only hypothesis.10–11 For example, Schwartz10 reports that although pollution-related mortality for chronic bronchitis may involve displacement of about 3 months, there is little evidence for any short-term displacement for pneumonia, cardiovascular, or all-cause mortality related to air pollution exposure. Schwartz found that modelling longer exposure times generated larger, not smaller, daily mortality effects. If mortality displacement was occurring, the size of the effect would be reduced, not increased, by modelling longer exposure times.

Additional evidence that harvesting does not dominate the time-series results includes data showing a significant effect on mortality among various age groups. This does not prove that harvesting is not significant, but the harvesting hypothesis would be more strongly supported if the mortality effect were limited to the elderly population. For example, the reports of the 1950s smog episodes of London indicated elevated mortality among all age groups, including children under 5 years of age. More recently, a time-series study in Bangkok, Thailand found a statistically significant association between PM10 and mortality across all of the age groups, including children.5 Several cross-sectional studies have also demonstrated statistically significant mortality effects for children associated with long-term exposures to higher air pollution.12–13

In addition, long-term exposure studies show a significant difference in life expectancy across cities with different air pollution concentrations. If the only effect of air pollution exposure

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on mortality were to shift death by a few days, then no difference in life expectancy would be expected across cities with different pollution levels. Because annual average pollution concentrations are correlated with short-term concentrations, some of the mortality linked to long-term exposure is also likely to be reflected in the time-series studies. Brunekreef14 shows a difference in life expectancy between high versus low polluted cities over a one-year period. Lippmann and Thurston report that the US prospective cohort studies imply an average life span shortening of about 2 years between the most polluted and least polluted cities.13 They also note that these studies imply an amount of excess annual deaths that exceeds that implied by the cumulative results of the time-series studies.

Finally, McMichael et al. argue that because the number of expected life-years lost cannot be determined from time-series results it is inappropriate to calculate the annual number of cases of premature mortality based on the time-series results. This argument confuses the magnitude of the prematurity with the number of cases of prematurity. Whether it is days, months, or years, the cases should still be considered as premature deaths. It is the economic valuation of these cases, not the counts, that would be impacted by the degree of life shortening. Economic studies are underway to attempt to address the question of how the amount of life shortening affects the monetary value of the change in mortality risk. However, it is incorrect to criticize the estimates of additional cases of premature mortality on this basis.

References

Authors’ response

From AJ McMICHAEL, HR ANDERSON, B BRUNEKREEF AND AJ COHEN

Sir—Ostro and Chestnut state that they disagree with our conclusions for two key reasons. Both of their reasons are, in our opinion, wrong. They have also, in various respects, misrepresented the tenor of our arguments.

The central proposition in our paper is that, by their very nature, time-series analyses of daily fluctuations in air pollution and numbers of deaths can provide no direct information about the loss of person-time in the ‘extra’ deaths occurring on high-pollution days. Indeed, in the hypothetical and unlikely extreme, a population exposed to ambient air pollution may experience no more total deaths in a year of daily fluctuation exposures than it would in a year of non-fluctuating exposures. The only difference would be in the pattern of daily distribution of deaths. The annual death rate would then be unchanged, even though there would have been some marginal losses of person-time in deaths occurring on high-pollution days.

In reality, the distribution of lost person-time is more mixed. As we stated: ‘Every such extra death involves some degree of prematurity, from as little as a day to many years.’ But, neither knowing which actual individuals die because of transient high exposure nor having counterfactual information about them, it is, we argued, ‘not possible to estimate directly the loss of person-time associated with the estimated number of extra deaths.’

Our second main proposition is that the important public health question about air pollution and mortality refers to estimating the impact upon death rates of long-term exposure. We briefly described the three extant cohort studies that provide information about this longer-term relationship.

Ostro and Chestnut argue, first, that we have overlooked evidence from studies other than daily time-series analyses. This, as indicated above, is not correct. More importantly, it is not relevant. The fact that long-term exposure to air pollution manifestly affects long-term death rates provides no basis for quantitative inference about the life-shortening effect of acute exposure effects. We do not dispute the substantial effect of air
pollution on mortality. Rather, we argue (see the title of our paper) that it is inappropriate to use daily mortality analyses to estimate longer-term mortality effects.

Their second argument is that we assert both that the mortality impact of acute exposures is ‘inconsequential’ (their word, not ours) and that those impacts ‘should not be summed to annual premature mortality counts’. On the former issue we remained agnostic, not judgemental, pointing only to the inability to estimate actual life-shortening from time-series data. On the latter, we strongly reject the implication that ‘there is a difference between estimating the number of cases of premature mortality and determining the welfare significance of these cases.’ The notion that estimating the loss of person-time is a task for welfare agencies, while epidemiologists can simply add up the annual total of daily deaths is a serious misreading of the epidemiologist’s task. The denominator for calculating annual and longer-term death rates is measured in units of person-time, not just persons.

A recent paper by Zeger et al. has proposed a way of decomposing daily time-series data into ‘frequency domains’ of increasing time scales. The modelling differentiates short-term fluctuations in pollution and deaths from fluctuations occurring at longer time scales. The latter components provide clearer information about the life-shortening effects of fluctuations in air pollution levels. Perhaps we are beginning to find solutions to this difficult problem.

Reference


From WW HOLLAND

Sir—The paper by Treurniet et al. in the April issue of the Journal is an elegant analysis of the contribution of incidence variations to ‘available mortality’. It is, however, unfortunate that the authors do not consider that this is an aspect of quality-of-care. When we worked on this subject we considered a variety of different components of ‘avoidable’ mortality and emphasized that it should be used as a starting point for in-depth analysis of the possible reasons for any variation found. However, it is important to emphasize that it should be used as a starting point for in-depth analyses of the possible reasons for any variation found, and that health services are intended to serve populations. Thus, if a particular condition is common in an area, then the provision for that condition should be commensurate with the need. By some this is considered as access. A high mortality in an area of high incidence where the condition is ‘avoidable’ is as much a measure of the quality or performance of the service as, for example, a high operative case-fatality rate.

References
