Implications for the science of air pollution and health

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The *Lancet* Commission on Pollution and Health[1] is a landmark in the history of attempts to improve health by reducing exposure to harmful environmental pollution, nearly all of which is created by human activity. This report aims "to raise global awareness of pollution, end neglect of pollution-related disease, and mobilise resources and the political will needed to effectively confront pollution". The first section of the report describes the health burden attributable to three types of environmental pollution: water pollution, air pollution and soil, heavy metal and chemical pollution, the latter including occupational pollutants. The remaining sections concentrate in some detail on the economic costs of pollution and pollution-related disease, links between pollution-related disease and poverty, and effective interventions. The optimistic message is that these problems can be addressed and that there are economic, social, and other, wider benefits, such as greenhouse gas mitigation, in doing so. This Comment will focus on some aspects of the methods underlying the estimates of burden of air pollution, with a view to identifying important gaps and challenges for science and policy.

An essential step in quantifying the health burden of pollution is to obtain a quantitative estimate of the risk of a health outcome that is causally associated with a pollutant. Causality is based on the evaluation of all available evidence (toxicology, clinical studies, and epidemiology), explicitly or implicitly using a range of criteria to arrive at a qualitative judgment. This evaluation is a major task for comprehensive reviews, such as those led by WHO.[2,3] For ambient pollutants, this process is fraught with uncertainty due to the somewhat subjective nature of the assessment process, the greatly varying pattern of evidence between pollutants, and the lack of satisfactory ways of disentangling the effects of the individual pollutants comprising the multipollutant mixture to which study populations are exposed. For example, although it is widely accepted that associations of particulate matter with aerodynamic diameter less than 2·5 μm ($PM_{2·5}$) cause disease, the methods of quantifying the effects of individual components of the $PM_{2·5}$ mixture are poorly developed, thereby hampering policies for targeted interventions. For some other regulated and widely monitored pollutants such as nitrogen dioxide, the causality of association between long-term exposure to pollution and mortality remains less clear.[4]

To quantify the associated burden of disease, pollution–outcome pairs need to be selected on the basis of availability of measurements of population exposure to the specific pollutant and of baseline rates of the specific outcome in the target population. Other considerations should include the transferability of the pollution–outcome functions to the target population and the potential for combining the outcomes from a range of diseases into a common metric for aggregation of the burden and monetisation. The details of this process will differ according to the purpose and context of the quantification. For a global assessment, this is a very challenging task.

The Commission relies largely on the results of the Global Burden of Disease study.[5] This large and ambitious project now provides estimates of health burden from diseases and risk factors on a global scale, at country level, for nearly 80 risk factors. This study expresses health burden by a common metric, the disability-adjusted life-year, which enables mortality and morbidity information to be combined and compared across risk factors, diseases, countries, and time. The combined burden of the three sources of pollution was estimated to be 16% of total global mortality in 2015.

The methods of estimating the burden of ambient air pollution have been described in detail.[6] There was a prima facie case for including associations of $PM_{2·5}$ concentrations with mortality, since long-term exposure studies of cohorts were available and the causality of associations was accepted. In the absence of adequate routine data on global $PM_{2·5}$ concentrations, methods were developed for estimating population-weighted exposure to $PM_{2·5}$ pollution from satellite estimates, chemical transport models, and surface measurements.[7] One constraint in study selection was that studies reporting only all-cause mortality could not be used because the Global Burden of Disease study required cause-specific estimates. The need to have credible risk estimates for countries with concentrations of air pollution above those of the range of study populations was addressed by the development of the Integrated Exposure Response curve (IER), which integrates relative risks of $PM_{2·5}$ pollution from diverse sources of $PM_{2·5}$ (household air pollution, ambient air

pollution, second hand smoke, and tobacco smoking) into a single curve, from which risk estimates of higher concentrations of pollution can be obtained.[8] Notably, this curve tends to flatten at higher concentrations, which suggests that abatement strategies in high-pollution areas might have relatively fewer health benefits for a given pollution decrement.

It is of note that the Commission, despite recommending research into emerging health effects (diabetes, neurological disease, and reproductive outcomes such as premature birth and low birth weight), does not make research recommendations concerning the well characterised outcomes used for existing burden estimates (lower respiratory infections, ischaemic heart disease, stroke, COPD, lung cancer). This omission could be questioned in view of the fact that the existing estimates of the burden of disease rely on small numbers of studies, most of which are based in high-income countries with relatively low pollution and that display considerable heterogeneity. Although the IER is a very useful tool, there remains a need for additional empirical studies in the relevant exposure scenarios to improve and validate it. This is especially relevant to the burden estimates for household pollution from solid fuels. The estimate for household $PM_{2·5}$ and cardiovascular disease (which represents about 50% of the burden from this source) assumes that it is plausible that household $PM_{2·5}$ from solid fuels will have cardiovascular effects.[9] Empirical studies to support this assumption are needed.

Despite extensive literature on the health effects of air pollution, we lack evidence that can be used for burden assessments of this type. This is not only because most cohorts are from countries with low pollution, but because existing studies are difficult to aggregate for meta-analysis because of heterogeneity in study design, in definitions of pollution exposure and outcomes, and in analyses of co-pollutants. Many good studies, although providing insights into hazards or mechanisms, do not lend themselves to meta-analysis or burden estimation. These problems can be partly overcome by research consortia that facilitate the standardisation of methods and thus increase the potential for aggregation and avoidance of publication bias.

For most pollutants, the direction of policy is straightforward: to reduce exposure. But, in practice, resource constraints require prioritisation, which will often include the use of economic tools. The Commission on Pollution and Health provides a good review of economic costs and pollution benefit–cost analysis and draws attention to the need for more evidence concerning morbidity. This Commission also deals at length with the issues of environmental justice and poverty. Research resources are expensive and scarce so must be directed as closely as possible to the ultimate public health benefit. The Commission report illustrates how important it is for science and policy to work together at all levels in the field of environmental pollution.

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I declare no competing interests.

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