Comment: (on Lancet Commission on pollution and health)

Implications for the science of air pollution health effects.

H.Ross Anderson MD

Institute of Population Health Sciences, St George’s, University of London and Environmental Research Group, King’s College London.

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. It aims “to raise global awareness of pollution, end neglect of pollution-related disease and mobilise resources and political will 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, 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 wider (e.g. greenhouse gas mitigation) benefits in doing so. This comment will focus on some aspects of 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 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 is a major task for comprehensive reviews such as those led by WHO (2, 3). For ambient pollutants this process is fraught with uncertainties on account of the somewhat subjective nature of the assessment process, the greatly varying pattern of evidence between pollutants and the lack of satisfactory ways of statistically separating the effects of individual pollutants within the pollution mixture to which study populations are exposed. For example, while it is widely accepted that associations with particulate matter with aerodynamic diameter less than 2.5 µm (PM2.5)are causal the methods of quantifying the effects of individual components of the PM2.5 mixture or of sources are poorly developed thus hampering policies for targeted interventions. For some other regulated and widely monitored pollutants such as nitrogen dioxide, the causality of associations between long-term exposure and mortality remains less clear (4).

For quantification of burden, pollution-outcome pairs need to be selected according to the availability of population exposure measurements of the specific pollutant and baseline rates for the specific outcome in the target population. Other considerations will include the transferability of coefficients from the evidential to the target population and the potential for converting the outcome into useful units of outcome for burden estimation and for monetization. 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 paper relies largely on the results of the Global Burden of Disease (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. The health burden is expressed by a common metric, the disability adjusted life-year (DALY), 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 is estimated to be 16% of premature deaths in 2015.

The methods of estimating the burden of ambient air pollution are have been described in detail (6). There was a prima facie case for selecting PM2.5 associations with mortality as long-term exposure studies of cohorts were available and the causality of associations was accepted. In the absence of adequate routine data on global PM2.5 concentrations, the project developed a method for estimating population weighted exposure to PM2.5 from satellite estimates combined with 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 GBD required cause-specific estimates. The need to have credible risk estimates for countries with air pollution levels 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 PM2.5 from diverse sources of PM2.5 (household air pollution, ambient air pollution, second hand smoke and tobacco smoking) into a single curve from which risks at higher concentrations can be obtained (8). Interestingly, this curve tends to flatten at higher concentrations which suggests that abatement strategies in high pollution areas may have relatively less health benefits for a given pollution decrement.

It is notable that the Commission, while recommending research into emerging health effects (diabetes, neurological disease, reproductive), does not make research recommendations concerning the “well characterized” outcomes used for the 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 burden estimates rely on small numbers of studies most of which are based in low pollution high income countries and which display considerable heterogeneity. While 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. Currently the estimate for cardiovascular disease (about 50% of the total mortality burden) is based only on the IER there being no substantial direct evidence of an association (9).

In spite of the vast literature on the health effects of air pollution we are seriously short on 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 may be difficult to aggregate for meta-analysis because of heterogeneity in study design, the ways in which pollution exposure is specified, definition of outcome and analysis of co-pollutants. Many good studies, while providing insights into hazard or mechanisms do not lend themselves to meta-analysis or burden estimation. These problems can be partly overcome by research consortia which facilitate the standardization of methods and thus increase the potential for aggregation and avoidance of publication bias.

For the vast majority of pollutants the direction of policy is straightforward, which is to reduce exposure. But in practice resource constraints require prioritization which will often include the use of economic tools. The Commission provides a good review of economic costs and pollution benefit-cost analysis and draws attention to the need for more evidence concerning morbidity. The Commission also deals at length with the issue 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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