Environmental pollution and the global burden of disease

David Briggs
Small Area Health Statistics Unit, Department of Epidemiology and Public Health, Imperial College, London, UK

Exposures to environmental pollution remain a major source of health risk throughout the world, though risks are generally higher in developing countries, where poverty, lack of investment in modern technology and weak environmental legislation combine to cause high pollution levels. Associations between environmental pollution and health outcome are, however, complex and often poorly characterized. Levels of exposure, for example, are often uncertain or unknown as a result of the lack of detailed monitoring and inevitable variations within any population group. Exposures may occur via a range of pathways and exposure processes. Individual pollutants may be implicated in a wide range of health effects, whereas few diseases are directly attributable to single pollutants. Long latency times, the effects of cumulative exposures, and multiple exposures to different pollutants which might act synergistically all create difficulties in unravelling associations between environmental pollution and health. Nevertheless, in recent years, several attempts have been made to assess the global burden of disease as a result of environmental pollution, either in terms of mortality or disability-adjusted life years (DALYs). About 8–9% of the total disease burden may be attributed to pollution, but considerably more in developing countries. Unsafe water, poor sanitation and poor hygiene are seen to be the major sources of exposure, along with indoor air pollution.

Introduction

Despite the major efforts that have been made over recent years to clean up the environment, pollution remains a major problem and poses continuing risks to health. The problems are undoubtedly greatest in the developing world, where traditional sources of pollution such as industrial emissions, poor sanitation, inadequate waste management, contaminated water supplies and exposures to indoor air pollution from biomass fuels affect large numbers of people. Even in developed countries, however, environmental pollution persists, most especially amongst poorer sectors
of society\textsuperscript{1,2}. In recent decades, too, a wide range of modern pollutants have emerged—not least, those associated with road traffic and the use of modern chemicals in the home, in food, for water treatment and for pest control. Most of these pollutants are rarely present in excessively large concentrations, so effects on health are usually far from immediate or obvious. As Taubes\textsuperscript{3} has noted, few of the problems of environmental exposure that concern us today imply large relative risks. Detecting small effects against a background of variability in exposure and human susceptibility, and measurement error, poses severe scientific challenges.

The progressively larger number of people exposed to environmental pollution (if only as a result of growing population numbers and increasing urbanization) nevertheless means that even small increases in relative risk can add up to major public health concerns. The emergence of new sources of exposure and new risk factors, some of them—such as endocrine disruptors—with the capacity to have lifelong implications for health, also means that there is a continuing need for both vigilance and action. As the impact of human activities and issues of environmental health become increasingly global in scale and extent, the need to recognize and to address the health risks associated with environmental pollution becomes even more urgent. Effective action, however, requires an understanding not only of the magnitude of the problem, but also its causes and underlying processes, for only then can intervention be targeted at where it is most needed and likely to have greatest effect. As background to the other chapters in this volume, therefore, this chapter discusses the nature of the link between environmental pollution and health and considers the contribution of environmental pollution to the global burden of disease.

Links between environmental pollution and health

Environmental pollution can be simply, if somewhat generally, defined as the presence in the environment of an agent which is potentially damaging to either the environment or human health. As such, pollutants take many forms. They include not only chemicals, but also organisms and biological materials, as well as energy in its various forms (e.g. noise, radiation, heat). The number of potential pollutants is therefore essentially countless. There are, for example, some 30,000 chemicals in common use today, any one of which may be released into the environment during processing or use. Fewer than 1% of these have been subject to a detailed assessment in terms of their toxicity and health risks\textsuperscript{4}. The number of biological pollutants is truly unquantifiable. They include not only living and viable organisms, such as bacteria, but also the vast array of endotoxins that can be released from the protoplasm of organisms after death. There is, therefore, no shortage of potential environmental risks to health. What
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is lacking, for the most part, is an understanding of the nature and mechanisms of these risks.

The source–effect chain

The link between pollution and health is both a complex and contingent process. For pollutants to have an effect on health, susceptible individuals must receive doses of the pollutant, or its decomposition products, sufficient to trigger detectable symptoms. For this to occur, these individuals must have been exposed to the pollutant, often over relatively long periods of time or on repeated occasions. Such exposures require that the susceptible individuals and pollutants shared the same environments at the same time. For this to happen, the pollutants must not only be released into the environment, but then be dispersed through it in media used by, or accessible to, humans. Health consequences of environmental pollution are thus far from inevitable, even for pollutants that are inherently toxic; they depend on the coincidence of both the emission and dispersion processes that determine where and when the pollutant occurs in the environment, and the human behaviours that determine where and when they occupy those same locations.

The whole process can simply be represented as a causal chain, from source to effect (Fig. 1). As this indicates, most pollutants are of human origin. They derive from human activities such as industry, energy production and use, transport, domestic activities, waste disposal, agriculture and recreation. In some cases, however, natural sources of pollution may also be significant. Radon, released through the decay of radioactive materials in the Earth’s crust, arsenic released into groundwaters from natural rock sources, heavy metals accumulating in soils and sediments derived from ore-bearing rocks, and particulates and sulphur dioxides released by wildfires or volcanic activity are all examples.

Release from these various sources occurs in a wide range of ways, and to a range of different environmental media, including the atmosphere, surface waters, groundwaters and soil (Fig. 2). Estimates of emission by source and environmental medium are inevitably only approximate, for they can rarely be measured directly. Instead, most emissions inventories derive from some form of modelling, either based on emission factors for different processes or source activities or on input–output models (i.e. by calculating the difference between quantities of the material input into the process and quantities contained in the final product).

Atmospheric emissions

Emissions to the atmosphere tend to be more closely modelled and measured, and more generally reported, than those to other media, partly...
because of their greater importance for environmental pollution and health (emissions to the atmosphere tend to be more readily discernible and to spread more widely through the environment), and partly because of the existence of better established policy and regulation. Figure 3 shows the main sources of emissions of selected pollutants in the European Union. As this shows, combustion represents one of the most important emission processes for many pollutants, not only from industrial sources, but also
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from low-level sources such as motorized vehicles and domestic chimneys, as well as indoor sources such as heating and cooking in the home or workplace. Emissions from industrial combustion or waste incineration tend to be released from relatively tall stacks, and often at high temperature, with the result that they are dispersed widely within the atmosphere. Emissions from low-level sources such as road vehicles and low-temperature combustion sources such as domestic heating, in contrast, tend to be much less widely dispersed. As a result, they contribute to local pollution

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Fig. 2 Sources and pathways of emission into environmental media.
hotspots and create steep pollution gradients in the environment. In urban environments, for example, traffic-related pollutants such as nitrogen dioxide and carbon monoxide typically show order-of-magnitude variations in concentration over length-scales of tens to a few hundred metres. Evaporation and leakage are also important emission processes contributing to local variations in environmental pollution. In the UK, releases from filling stations account for ca. 1.8% of benzene emissions; leakages from gas pipelines contribute ca. 13.7% of methane emissions to the atmosphere; evaporation and leakage of solvents during processing and use produce ca. 40% of atmospheric emissions of non-methane volatile organic compounds (NMVOCs). In addition, abrasion, corrosion and corrosion release significant quantities of emissions to the atmosphere. Wear and tear of catalytic converters during operation is a major source of platinum emissions, for example, whereas tyre wear (corrosion) and road wear (abrasion) account for about 16% of particulate emissions from road transport and almost 97% of zinc emissions from road transport—and perhaps more where studded tyres are used.

These fugitive and local emissions are often overlooked in epidemiological and other studies that use modelling techniques to estimate exposures, but they can be extremely important, both because they are frequently responsible for the highest concentrations of environmental
pollution, and because—unlike high-level emissions—they remain close to source and show marked dilution gradients with distance from source. In many cases, they may therefore be the real sources of variation being considered when distance is used as a proxy for exposure around point industrial sources in epidemiological studies.

**Emissions to surface water, groundwater and soil**

Releases to other media, such as surface waters, groundwaters and soil, also occur through a range of processes. Deliberate discharge, spillage (e.g. from storage, during transport, or during processing and usage), leakage and runoff (e.g. of agricultural chemicals) are all important in terms of aqueous pollutants. Legal limits for discharges to streams are set for many industries, aimed at keeping levels of contamination within accepted limits. Illegal discharges, or accidental spillage, however, sometimes occur and accounted for the majority of reported surface water pollution incidents in the UK in 2001, for which the cause is known. Dumping (both legally in landfill sites and illegally) represents a major source of emission of solid wastes, though final release into the wider environment may only occur when these materials decompose or break up. Landfill sites may thus be responsible for emissions of a wide range of pollutants, via different pathways, especially when these sites are inadequately sealed or poorly maintained. The contribution of informal and illegal dumping to environmental pollution is, inevitably, only poorly known.

**Environmental fate**

Once released into the environment, pollutants may be transported via many different processes and pathways, often moving from one medium to another, and undergoing a wide range of modifications in the process. Chemical reactions, physical abrasion, sorting by size or mass and deposition all change the composition of the pollutants and alter the pollution mix. Dilution occurs as pollutants spread outward into a wider volume of space; concentration may occur as pollutants accumulate in local ‘sinks’ or in the bodies of organisms, as they pass along the food chain.

In general, these processes tend to result in some degree of distance-decay in environmental concentrations, if only because the opportunity for dilution, decomposition and deposition increases with increasing distance of transport. It is largely on this basis that distance is often used as a surrogate for exposure in many epidemiological studies. The realities of environmental patterns of pollution are, however, often much more complex than these simple distance-based models imply. They also vary greatly between different pollutants and environmental media, because
of the different transportational behaviours that are involved. In addition, dispersion processes and resulting pollution concentration fields may vary substantially depending on the prevailing (e.g. meteorological) conditions at the time. Patterns of atmospheric dispersion, for example, differ not only in relation to windspeed and direction but also atmospheric stability (e.g. between stable and unstable weather conditions, or when there is a temperature inversion)\(^6\). Movement of many pollutants through soils occurs mainly as mass flow in water passing through larger pore spaces and fissures: the irregular distribution of these within highly structured soils means that dispersion often follows highly discrete pathways\(^12,13\). Gaseous pollutants may follow similar preferred pathways. Releases from landfill sites may thus travel relatively long distances in the soil or bedrock, before emerging at the surface, where they can cause local hazards including explosions\(^14\). Radon shows the same discrete and complex pattern, such that concentrations may vary by orders of magnitude from one home to another in the same district\(^15,16\). Modelling these locally variable pathways poses severe challenges.

To a large extent, the increased opportunity for mixing means that dispersion of pollutants in surface and groundwaters is more regular, leading to more uniform patterns of contamination, at regional scales. In developed countries, also, considerable water mixing often occurs during treatment and distribution, so that water quality is relatively uniform across large areas and populations. Local variations may occur, however, because of contamination within the distribution system or differences in the length of the network, and thus in the time available for contamination and decomposition of the disinfectants incorporated at treatment\(^17\). In developing countries, especially, considerable variations may also occur between waters in shallow wells, particularly where these are affected by local pollution sources, such as badly sited latrines or agricultural activities. Again, this makes exposure assessment difficult, without the ability to collect data on water quality for individual wells.

Similar difficulties occur in tracking and modelling transport of pollutants in the food chain. Whilst the general pathways followed by pollutants are often clear in natural (and some farmed) food chains, in that persistent compounds tend to accumulate as they pass from one trophic level to another, the detailed patterns of contamination are often far more complex. Many animals have very restricted feeding behaviours: even in areas of open grazing land, for example, sheep tend to focus on distinct home ranges from which they rarely stray\(^18\). As a result, marked variations may occur in contaminant uptake by livestock, even over short distances, as illustrated by patterns of contamination from the Chernobyl incident in the UK\(^19\). Significant accumulation of these contaminants in humans likewise tends to occur only where small groups of individuals rely on local food sources. On the other hand, in many modern food supply
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systems, industrial-scale processing and distribution operations mean that foodstuffs often travel large distances before consumption and are drawn from far-flung sources. In the UK, as in most developed countries, therefore, the average distance travelled by foodstuffs before consumption has increased markedly, from an average of about 82 km in 1978 to 346 km in 1998\textsuperscript{20}. In the light of these changes, several attempts have been made in recent years to calculate the distance travelled by ingredients in common food products or meals (so called ‘food-miles’). In Iowa, USA, for example, ingredients for a standard meal of stir-fry and salad were estimated to have been transported 20,000 km\textsuperscript{21}; in the UK, Sustain, a pressure group on food and agriculture, estimated that ingredients for a traditional turkey dinner had been transported some 38,620 km\textsuperscript{22}! Apart from implications for increased energy consumption and environmental pollution, such extended distribution networks clearly mean that it can be difficult to track and control potential contamination between source and consumption.

By whatever pathways and processes pollutants pass through the environment, four related factors are especially important in determining the potential for exposure and health effects: their persistence, their mobility, their decomposition products and their toxicity. The problems associated with the release of persistent pollutants into the environment were highlighted many years ago with recognition of the global extent of contamination, and wide-ranging environmental and health effects, caused by DDT and other organochlorine pesticides\textsuperscript{23}. The story is in many ways now being repeated in relation to chlorofluorocarbons and other atmospheric pollutants that act as greenhouse gases or scavengers of stratospheric ozone\textsuperscript{24}, and perhaps also in relation to endocrine disruptors\textsuperscript{25}. Persistence, however, is not necessarily the most important issue, for where they persist in inert yet inaccessible forms, pollutants may pose relatively limited risks. Thus, whereas inorganic mercury is persistent, it is less toxic and less readily bioavailable than methyl mercury, to which it is naturally converted through chemical reactions and the action of soil and aquatic microorganisms\textsuperscript{26,27}. Equally, many solid wastes represent little risk to health so long as they remain in their original form. The problems in these cases often come when decomposition occurs, either because the decomposition products are inherently more toxic or because they are more mobile, and thus are more likely to result in human exposure.

**Exposure and dose**

Whilst the potential for health impairment initially depends upon the existence and concentrations of pollutants in the environment, for health effects to occur exposures must take place that lead to a dose sufficient to have adverse health consequences. Exposure in this context is defined as the contact between a hazardous agent (in this case a pollutant) and an
organism. Dose refers to the quantity of the substance in the body. The absorbed dose refers to the amount of the substance entering the body as a whole; target organ dose refers to the amount reaching the specific organs that are affected.

Exposure can take place in many different ways. Three main forms of exposure are generally recognized: dermal contact, inhalation and ingestion. In some cases, however, it may also be useful to recognize a fourth— injection—for example, when pollutants are transmitted by animal bites or by deliberate injection. In each of these cases, exposure may occur in a range of different environments. Whilst some exposures occur in the outdoor (ambient) environment, most people spend the majority of their time indoors, either at home or at their place of work or learning. Indoor exposures therefore often make up a major proportion of total exposure—although they tend to be rather neglected in many epidemiological studies. Food and drinking water are likewise important routes of exposure for many pollutants.

What determines levels of exposure is consequently not just the distribution of pollution within the environment, but also human behaviours and lifestyles, and thus the sorts of exposure environments in which people spend their time. By the same token, exposure is not only an environmental process, it is also a social, demographic and economic one. Indeed, because of the myriad ways in which socio-economic and demographic factors influence and interact with environmental conditions, exposures, human susceptibility and health outcome, they may often appear to outweigh the effects of the environment per se in associations between pollution and health. One expression of these complex interactions is the patterns of what is often called environmental injustice that are seen throughout the world: namely, the tendency for environmental pollution and poverty or other forms of disadvantage to be strongly correlated, such that poorer people tend to live in more polluted environments. Whilst the reasons for this association are not fully understood, and may be more subtle than often assumed, the double jeopardy that it represents seems to be generally reflected in terms of health inequalities as well. The problem, however, comes in trying to separate the contributions to these adverse health outcomes from socio-economic and environmental factors—and thus to quantify the attributable effects of pollution.

It also has to be recognized that pollutants rarely occur in isolation; more typically they exist in combination. Exposures are therefore not singular. Instead we are usually exposed to mixes of pollutants, often derived from different sources, some of which may have additive or synergistic effects. Unravelling the effects of individual pollutants from this mix is a challenging problem that has yet to be adequately resolved in many areas of epidemiology.
Health effects: dose–response relationships, latency and attributable risk

One of the underlying tenets of environmental epidemiology is that, for the health effects of interest, a relationship exists between the level of exposure (or dose) and the degree of effect. Effects can, in fact, be represented in two different ways: by the type of effect or by its severity or the probability of its occurrence (often termed the ‘response’). In either case, these associations are generally assumed to be broadly linear, such that the effect or response increases with each increment of exposure to a pollutant (Fig. 4A). For many pollutants and many health effects, this assumption seems to hold true at least over a wide range of exposures and responses. Some, however, appear to be characterized by more complex associations. Thresholds may exist, for example, below which no detectable health effects occur (Fig. 4B). At high levels of exposure, responses may weaken, so that the dose–response relationship is essentially curvilinear-convex (Fig. 4C) or S-shaped (Fig. 4D). In a few cases, there is some evidence that \( \cap \)- or, more rarely, U-shaped relationships may exist—for example, in relation to solar radiation or vitamin intake. One of the main purposes of epidemiology is to demonstrate and, if possible, quantify these relationships, where they exist.

Just as exposures may be long- or short-term, so health effects can be short-lived (acute) or prolonged (chronic). Health effects may also be delayed to a greater or lesser extent after initial exposure, either because

![Fig. 4 Common forms of exposure–response relationships.](https://academic.oup.com/bmb/article-abstract/68/1/1/421204)
it takes time for exposures to reach a critical level, or because the disease itself takes time to develop and become apparent (latency). Many acute effects are almost immediate, and have latencies typically of no more than a few minutes to a few days. Many chronic effects, on the other hand, can have latencies of several years—up to 20 years or more, for example, in the case of some cancers and diseases such as asbestosis. Dealing with these latencies is problematic in epidemiological studies, both because they often imply the need for information on past (in some cases long-past) exposures, and because the degree of latency may vary from one individual to another, depending on factors such as the level of exposure, the age at which exposure occurred and pre-existing health status. In some cases, too, a so-called harvesting effect may occur, such that, following a brief increase in disease rates as more vulnerable people are affected, rates of illness fall because there are fewer vulnerable people left (Fig. 5). This, too, can complicate epidemiological studies, for the scale (and even the direction) of the dose–response relationship may vary depending on the length of latency allowed for in the analysis.

Long-term legacies for health may also occur as a result of sensitization and predisposition of individuals to the effects of exposure in early life. Sensitization to house dust mite and other allergens both in the diet and the indoor environment during the first few months of life, for example, appears to increase risks of allergic airway disease later in childhood. Similarly, inverse associations have been found between birth weight and the incidence of a range of diseases including hypertension, type-2 diabetes

![Fig. 5](https://academic.oup.com/bmb/article-abstract/68/1/1/421204)
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and cardiovascular disease in adults. Environmental exposures, such as air pollution, that contribute to these predisposing conditions may thus have long-term (and in some cases lifelong) implications for health.

Health outcomes may also be more or less specific to exposures to particular pollutants. Very few diseases are, in practice, pollutant-specific. Amongst these are asbestosis and mesothelioma (due to exposures to asbestos) and bagassosis, through exposure to organic dusts (the most common example, of which, is ‘Farmer’s lung’). Far more commonly, individual health effects may arise as a result of exposures to a number of different risk factors, either individually or in combination, whereas individual exposures can give rise to a range of different health effects. Environmental health is thus characterized by many-to-many relationships; understanding these is, again, a major challenge for epidemiology. Partly for this reason, it is often extremely difficult to assess the health burden attributable to an individual pollutant. Over-estimation may occur due to double-counting (or multiple attribution) of health effects; under-estimation may arise due to the failure to recognize some contributions to the disease burden as a result of masking by other risk factors.

In addition, of course, all epidemiological studies—and other studies that contribute to the establishment of dose–response relationships, such as laboratory experiments and clinical trials—are subject to error and uncertainty. These arise for many different reasons: because of errors in exposure assessment or classification, because of errors in diagnosis or reporting of health outcome, because of inadequate sample size, because of inadequate adjustment for confounding or effect modification by other factors, because of biases in sampling and statistical analysis, and because of the underlying indeterminacy of some of the associations of interest. As a result, dose–response reported by different studies often shows substantial differences, and many separate studies may be needed before a clear pattern of association emerges. Even then, problems may be encountered in deriving reliable dose–response relationships (e.g. through some form of meta-analysis), because of inconsistencies in study design (e.g. in methods of exposure classification, target populations or specification of health outcome). Most dose–response relationships are thus accompanied by a relatively large degree of uncertainty.

Models of pollutant pathways

As the discussion above has indicated, the relationships between pollution and health are both complex and often indirect. Considerable difficulties are thus encountered in quantifying the associations involved. It is largely for this reason that many of the health effects of environmental pollution are still uncertain, and that problems arise in attempting to attribute health outcome to environmental causes—for example, when trying to confirm or explain apparent spatial clusters in health.
These subtleties and complexities highlight the importance of examining critically any hypothesis about a relationship between a pollutant and an apparent health effect, and of setting such hypotheses within a wider environmental context. Assumptions about simple, singular cause–effect relationships often need to be eschewed; in their place we need to recognize the possibility for multifactorial effects in which single health outcomes are attributable to a wide variety of (possibly inter-related) environmental and other risk factors; and in which individual exposures may contribute to a range of different health effects. The contingent and historical nature of many of these associations also needs to be appreciated: health effects seen now in many cases owe their existence to exposures, sensitization or some process of predisposition far in the past. Because environmental conditions, and even the very nature of the risk factors involved, may change quite considerably over time, uniformitarianist principles may not hold true, *i.e.* the present cannot always be seen as the key to the past.

Against this background, the use of models to conceptualize the possible interplay of different risk factors and exposure pathways, and how they might have evolved over time, represents an important tool for attempts to understand associations between pollution and health. One example is illustrated in Figure 6, which shows possible sources and pathways of exposure of environmental pollution associated with landfill sites. Several important lessons can be drawn from this example. First, it is evident that the pathways of exposure are highly varied and complex. Which is the most important may well differ from one situation to another. The possibility of contributions from each and all of them needs to be allowed. Second, it is evident that landfill sites leave a legacy which may persist long after they are no longer operational. Present-day land use and activity may therefore not account for current exposures. Third, related to this, sources and pathways of exposure change markedly over time—and, indeed, many of the risks associated with what are now landfill sites may well predate the sites themselves (*e.g.* from prior land use). Perhaps it is for this reason that several studies of health risks around landfill sites have found that raised levels of risk existed before the landfill sites were opened.\(^35,36\)

### The contribution of environmental pollution to the global burden of disease

#### Estimating the global burden of disease

For all the reasons outlined above, estimating the contribution of environmental pollution to the burden of disease is far from easy. In general, too little is known either about the causal links between environmental
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pollution and health, or about the levels of exposure across the population, to make reliable assessments of the proportion of disease or mortality attributable to pollution. These difficulties are severe in developed countries, where disease surveillance, reporting of mortality, environmental monitoring and population data are all relatively well established. In most developing countries they become all but insurmountable, because of the generally impoverished state of routine monitoring and reporting. Given that controls on emissions and exposures in the developing world are often limited, it is in these countries that risks from environmental pollution are likely to be greatest. Such uncertainties thus render any attempt to quantify the environmental burden of disease highly approximate at best.

Assessments of the disease burden attributable to different forms and sources of pollution are nevertheless worth the effort. They are needed, for example, to raise awareness about some of the risks associated with environmental pollution, and as a basis for advocacy—to ensure that those most in need have a voice. They are needed to help motivate and prioritize action to protect human health, and to evaluate and monitor the success of interventions. They provide the foundation, therefore, for extremely powerful indicators for policy support, and a means of pricking the global conscience about inequalities in health.

Fig. 6 A model of emission processes and exposure pathways from landfill sites.
Over recent years, therefore, many attempts have been made to assess the health status of the population, both nationally and globally, and to deduce the contribution made by pollution and other environmental factors. In Europe, for example, more than 50 national environmental health action plans have been developed, following the Helsinki Conference in June 1994, setting out strategies to tackle problems of environmental health. Although these differ substantially in terms of their content and scope, many have involved attempts to make formal assessments of the disease burden attributable to different environmental hazards, and to rank these in terms of their public health significance. Various methods were used for this purpose, though most relied on some form of expert judgement, informed where available by quantitative data on mortality or disease rates. Whatever the weaknesses of these assessments, their practical importance is evident, for they have contributed directly to policy prioritization and development in the countries concerned.

The same need has arisen to support the development of environmental health indicators. Since the early 1990s, largely motivated by WHO, increasing attention has been given to constructing indicators on environmental health at all levels from the local to the global scale, and a number of indicator sets have been created (and to a lesser extent used). Environmental pollution is, inevitably, a major focus of concern in these indicator sets. By definition, also, environmental health indicators provide measures that link environmental hazards and health effect. As such they depend upon an understanding of the association between pollution and health, either in the form of what have been called ‘exposure-side indicators’, which use information on exposures to imply degrees of health risk, or ‘health-side indicators’, which use information on health outcome to suggest attributable effects. In both contexts, the ability to make at least semi-quantitative interpretations of the link between pollution and health, and thus to assess the contribution to the burden of disease, is assumed.

**Mortality**

The most explicit attempts to quantify these links, however, have come in recent years through work to estimate the global (and to some extent regional) burden of disease. Earlier efforts in this direction were targeted specifically at making broad-scale enumerations of the total disease burden across the world. The traditional measure used for such assessments was mortality, both because data on deaths tended to be more reliable and widely available, and because mortality is directly comparable in terms of health outcome, unlike morbidity which implies differences in severity of effect. Even so, results from the various efforts differed somewhat, largely because of the ways in which gaps and uncertainties in the available data were dealt with. Overall, however, cardiovascular
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Diseases were seen to be the major cause of mortality, accounting for between 19% (based on the World Health Survey estimate) and 28% (based on Murray and Lopez’s Global burden of disease study44) of total deaths worldwide. Cancer (an estimated 12% in each case), acute respiratory diseases (8.1, 8.7%, respectively), unintentional injuries (5.7, 6.4%), diarrhoeal diseases (ca. 5.8%), chronic respiratory diseases (ca. 5.7%) and perinatal conditions (6.2, 4.8%) were other major killers.

Years of life lost and disability adjusted life years
Crude estimates of the number and proportion of deaths due to different diseases, of this nature, obviously give only a distorted picture of the true burden of disease, for they take no account of the age of death or the duration of any preceding illness and disability, nor the amount of suffering involved. In an attempt to redress this, Murray and Lopez48 also computed estimates of the ‘years of life lost’ (YLL) and ‘disability adjusted life years’ (DALYs). Years of life lost are estimated as the difference between age at death and the life expectancy in the absence of the disease, based on an advanced developed country (82.5 years for women and 80 years for men at that time). DALYs also incorporate an allowance for the number of years lived with a disability due to disease or injury, weighted according to its severity (based on expert assessments of the relative impact of some 500 different conditions and disease sequelae). The years of disability or life lost are also discounted according to the age of onset (since it is assumed that future years of life lost contribute less to the burden of disease than current ones).

Results of these calculations are summarized and discussed by WHO49. Estimates of YYL and DALYs provide a somewhat different ranking of disease compared to crude mortality, since they give additional weight to early-onset diseases and chronic illness. Cardiovascular diseases are thus seen as somewhat less important (making up ca. 13% of YYL and 9.7% of DALYs). Acute respiratory diseases (12% and 8.5%, respectively), diarrhoeal diseases (10% and 7.2%) and unintentional injuries (9.3% and 11%) all become proportionally more significant. Mental health conditions also figure as a major source of ill health in terms of DALYs, contributing 11% of the total burden of disease worldwide.

Variations in the global burden of disease
By whichever method they are computed, marked variations are evident in the burden of disease between different sectors of the population. Children are seen to be especially at risk—and young children most of all. More than 30% of all deaths for all diseases in the Global burden of disease study occurred to children under 15 years of age; in the case of diarrhoeal diseases they accounted for 88% of deaths, and for acute respiratory illness 67% of deaths. Malaria also struck children disproportionately
(82% of deaths), whilst mortality as a result of perinatal diseases and vaccine-preventable diseases was inevitably almost wholly of children. When measured in terms of DALYs, the overwhelming burden of all these diseases falls on children49.

Similar inequalities occur both socially and geographically. The World Bank, for example, compared mortality rates and DALYs between poor and rich nations in the world50. Clear differences were shown. Whereas ischaemic heart disease, for example, was responsible for 23.4% of deaths in the rich countries, it accounted for only 7.3% in the poor countries; malignant neoplasms were responsible for 22.6% of deaths among rich countries, but only 5.6% among the poor. Conversely, respiratory infections and diarrhoeal diseases accounted for 13.4% and 11.3% of deaths, respectively in poor nations, compared with 4.0% and 0.3% in rich countries; for childhood cluster diseases, the proportions were 7.8% and 0.1%, respectively. Similar disparities have been shown in a comparison of ‘less developed’ and ‘more developed’ countries by Smith et al51. As these examples show, generalizations about the burden of disease thus need to be interpreted with care. Beneath the often stark global figures lie even starker indications of health inequalities that cry out to be addressed.

**The environmental burden of disease**

Whilst the original estimates of the global burden of disease made by Murray and Lopez and WHO during the mid 1990s were a major step forward in terms of providing comparable data on health status across the world, they gave information only on health outcomes and did not for the most part attempt to attribute these outcomes to specific causes. Smith et al51 did, however, make an attempt to assess the environmental contribution to the global burden of disease, using Murray and Lopez’s data. This suggested that environmental factors accounted for between 25% and 33% of the total burden of disease, but with a disproportionate share of this falling on children under 5 years of age. Diarrhoeal diseases (for which some 90% of DALYs were attributed to the environment), malaria (ca. 88%) and acute respiratory illness (60%) were seen as outcomes for which the environment was especially influential. Murray and Lopez52 also made preliminary assessments of the relative importance of different risk factors for the global burden of disease, based on their 1990 data. Malnutrition stood out as the most important factor considered, accounting for ca. 11.7% of deaths and 15.9% of DALYs worldwide. Poor water and sanitation was estimated to be responsible for ca. 5.3% of deaths and 6.8% of DALYs, whereas air pollution contributed 1.1% and 0.5%, respectively. Subsequently, a more specific attempt was made...
by Prüss et al\textsuperscript{53} to assess the effects of water, sanitation and hygiene, using a combination of exposure-based risk assessment and outcome-based disease attribution\textsuperscript{54}. Based on available information, they estimated that these environmental factors were responsible for ca. 4\% of global mortality and 5.7\% of total DALYs. These estimates are somewhat lower than those implied by the original Global burden of disease study\textsuperscript{52}, partly perhaps because of differences in methodology and partly because of a decline in mortality in the intervening years.

All these attempts to partition the global burden of disease by causative risk factor have faced, and admitted, a number of major difficulties. These relate not only to uncertainties in the available data on health outcome, but also to problems of how to attribute any single death to a single cause or risk factor. Two main approaches have been proposed for disease attribution\textsuperscript{55}. Categorical attribution assigns each death to a specific disease or risk factor, according to a defined set of rules (e.g. the ICD system). The advantage of this approach is that it is relatively straightforward and consistent, and avoids double-counting; the disadvantage is that it ignores the multi-factorial nature of many diseases and still leaves unresolved the problem of how to define appropriate rules. Counterfactual attribution involves comparing the current level of disease or mortality with that which might be expected to occur in the absence of the risk factor (or at some other reference level). One of the main difficulties with this approach is how to define this reference level. Several possibilities exist: for example, the complete absence of the risk factor, the level of risk in some reference population or area, or the achievable level of risk with current technologies. Each will tend to give a rather different measure of the attributable burden of disease. In this context, another difficulty also arises, \textit{i.e.} how to assess the likely change in disease burden under the selected scenario, in the absence of empirical data.

Notwithstanding these difficulties, a revised assessment of the global burden of disease has recently been carried out, involving explicit attempts at attribution by risk factor or hazard\textsuperscript{56}. A counterfactual approach was used, with the reference level for each disease being defined as that which would occur under conditions of a minimum theoretical exposure distribution (\textit{i.e.} that which would achieve the lowest population risk, irrespective of whether this is achievable in practice). Assessments were carried out by a series of expert groups, who first undertook a detailed review of relevant literature, and derived estimates of the exposures and relative risks for specific age and gender groups, for each of 14 sub-regions. Based on these data, estimates were then made of the population impact fraction of the disease or death in each region, for each risk factor:
where $PIF$ is the population impact fraction, $RR(x)$ is the relative risk at exposure level $x$, $P(x)$ is the population distribution of exposure, $P'(x)$ is the counterfactual distribution of exposure and $m$ is the maximum exposure level.

Results from this assessment for a number of environmental risk factors are summarized in Table 1. More detail on the methods and results are available on the WHO website for a number of the disease groups, including chronic obstructive pulmonary disease, malnutrition and injuries, and others will be published as available. The sources of environmental and occupational pollution listed in Table 1 account for 8–9% of the total global burden of disease, measured either in terms of mortality or DALYs. Amongst these risk factors, water, sanitation and hygiene and indoor air pollution are seen to be the most important; health effects of outdoor air pollution are comparatively small, although to some extent this may reflect differences in methodology between this and the other expert groups. It is also evident that a range of other sources of pollution, not included in this table, might be implicated in the global burden of disease, such as exposures to ionizing and non-ionizing radiation, food contamination, pesticides, household hazardous chemicals, wastes and other forms of indoor air pollution. The overall burden of disease attributable to pollution, therefore, cannot yet be assessed.

As with previous estimates of the burden of disease, marked variations can also be recognized between different parts of the world. As is to be

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Deaths</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>%</td>
</tr>
<tr>
<td>Total (all risk factors)</td>
<td>55,861</td>
<td>1,455,473</td>
</tr>
<tr>
<td>Water, sanitation and hygiene</td>
<td>1730</td>
<td>3.1</td>
</tr>
<tr>
<td>Urban outdoor air pollution</td>
<td>799</td>
<td>1.4</td>
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<tr>
<td>Indoor smoke from solid fuels</td>
<td>1619</td>
<td>2.9</td>
</tr>
<tr>
<td>Lead</td>
<td>234</td>
<td>0.4</td>
</tr>
<tr>
<td>Occupational carcinogens</td>
<td>118</td>
<td>0.2</td>
</tr>
<tr>
<td>Occupational airborne particulates</td>
<td>356</td>
<td>0.6</td>
</tr>
<tr>
<td>Occupational noise</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Total (pollution-related)</td>
<td>4856</td>
<td>8.7</td>
</tr>
</tbody>
</table>

Source: based on Ezzati et al.66.

Table 1 Global burden of disease (thousand and percent) attributable to selected sources of environmental and occupational pollution
expected, the developing countries bear the major proportion of the burden. Problems of unsafe water, sanitation and hygiene, for example, account for an estimated 6.6% of DALYs in Africa, and 4.7% in south-east Asia, compared with 0.5% in Europe. Indoor air pollution accounts for 4.4% of DALYs in Africa and 3.6% in south-east Asia, compared to 0.4% in Europe. In absolute terms the differences are even more stark. The total number of DALYs per head of population attributable to these two risk factors in Africa are 29.1 per thousand for unsafe water, sanitation and hygiene and 19.3 per thousand for indoor air pollution; in south-east Asia they are 12.8 and 9.9 per thousand, respectively; in Europe they are 0.8 and 0.6 per thousand, respectively. Risks attributable to environmental pollution in the developing world are thus 15–35 or more times greater than in developed countries.

Conclusions

The complexities involved in the link between environmental pollution and health, and the uncertainties inherent in the available data on mortality and morbidity, in existing knowledge about the aetiology of diseases, and in environmental information and estimates of exposure, all mean that any attempt to assess the environmental contribution to the global burden of disease is fraught with difficulties. The estimates produced to date must therefore be regarded as no more than order-of-magnitude estimates. Despite these limitations, however, several conclusions seem beyond refute.

The first is that environmental pollution plays a significant role in a number of health outcomes, and in several cases this adds up to a serious public health concern. Water pollution, sanitation and hygiene, indoor air pollution, and to a lesser extent outdoor air pollution and exposures to chemicals in both the indoor and outdoor environment are all important risk factors in this respect. Ionizing and non-ionizing radiation and noise are also causes for concern in many cases.

Secondly, it is clear that the distribution of risks from these factors is not equal across the world. The global burden of disease may be difficult to quantify, but stark contrasts in that burden are evident between the developed and the developing world, between rich and poor, and often between children and adults. The developed world is not risk-free, and development is no panacea for all environmental health ills. On occasions, in fact, the opposite is true: developments, such as increased reliance on road transport, increased use of chemicals in agriculture, and increased proportions of time spent in modern, hermetically sealed buildings surrounded by chemically-based fabrics and furnishings may actually increase exposures and exacerbate health risks. But overall the developing
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world is far more severely affected by pollution, and in many instances becoming more so, as pressures from development add to traditional sources of exposure and risk.

Thirdly, and perhaps most importantly, many of these risks and health effects are readily avoidable. Rarely does the solution lie in advanced technologies or even expensive drugs. Instead, the need is for preventive action to reduce the emission of pollutants into the environment in the first place—and that is largely achievable with existing know-how. Indeed, in many cases it has already been implemented in many of the richer countries. Science, therefore, certainly has a role to play in addressing these issues. More research is undoubtedly needed on a range of emerging environmental health issues. But the deficit of action that has allowed environmental pollution still to take its toll on health derives not so much from failures in science or technology as from the lack of political will and economic empowerment. It is from that direction that salvation needs ultimately to come for those at the mercy of environmental pollution.

References

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16 Henshaw DL. Radon exposure in the home: its occurrence and possible health effects. Contemp Phys 1993; 34: 31–48
18 Beresford NA, Barnett CL, Crout NMJ, Morris CC. Radioaesium variability within sheep flocks: relationships between the 137Cs activity concentrations of individual ewes within a flock and between ewes and their progeny. Sci Total Environ 1996; 177: 85–96
22 Bro-Rasmussen F. Contamination by persistent chemicals in food chain and human health. Sci Total Environ 1996; 188: S45–S60
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40 Corvalán CF, Kjellström T, Smith KR. Health, environment and sustainable development. Identifying links and indicators to promote action. Epidemiology 1999; 10: 656–60


57 WHO. http://www.who.int/peh/burden/globalest.htm (date last accessed 30 June 2003)