Human exposure and health cannot be overlooked in environmental monitoring programmes

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Introduction

Anthropogenic damage to the earth's biosphere has potentially serious implications for human health [1]. For instance, global warming as a consequence of the emission of greenhouse gases leads to more frequent and more intense heatwaves and may directly result in an increased cardiovascular and cerebrovascular mortality among older individuals. The projected indirect effects of a global rise in temperature range from changes in vector-borne diseases, crop production and fresh water supplies to social and economic impacts [1]. However, many of the theoretical effects of global warming rest on extrapolations from theoretical models. Threats to human health acting on a global scale must be differentiated from environmental hazards, which operate locally in each person's microenvironment. At the local or microenvironment level, toxic agents from industrial or natural origin may be absorbed via the skin, or after inhalation or ingestion.

Vast amounts of public and private money are currently invested in environmental monitoring programmes, ecotoxicological studies and animal experiments, which aim to disentangle possible routes of exposure and mechanisms underlying the suspected or proven associations between noxious agents and health effects. These approaches make it possible to test working hypotheses and provide often essential information. However, whatever the scale of the exposure, the most important assessment of a potential environmental hazard must rest on the direct examination of the internal exposure of the people exposed and on the measurable repercussions on human health. The latter are illustrated by recent experience related to environmental exposure to lead and inorganic arsenic.

Low-level lead exposure and cardiovascular disease

The possible relation between low-level lead exposure and cardiovascular disease, in particular hypertension, has been debated [2]. A recent article reviewed 21 animal studies that

investigated the association between lead exposure and blood pressure [2]. One study was carried out in dogs, one in pigeons and the remainder in various rat strains. In 15 studies, in which the lead dose in drinking water or food exceeded 1 p.p.m., the association between blood pressure and exposure was found to be positive in seven, consistent in three, absent in four and negative in one. Of the six studies at lower exposure levels ($\leq$1 p.p.m.), five found a pressor effect attributable to lead. However, whether the lead doses in the animal studies are equivalent to the human exposure levels and the extent to which one is entitled to extrapolate from genetically heterogenous animals to humans are not known.

In a meta-analysis, in which 23 studies including 33 141 subjects were pooled, a twofold increase in the blood lead concentration was associated with a 1.0 mmHg rise in systolic blood pressure [95% confidence interval (CI) 0.4–1.6 mmHg; P = 0.002)] and a 0.6 mmHg increase in diastolic blood pressure (CI 0.2–1.0 mmHg; P = 0.02) [2]. Under the assumption of a causal and reversible relationship between blood pressure and the blood lead concentration, the potential health risks of lead exposure have previously been examined in white men by extrapolation from the multiple logistic regression models obtained in the Pooling Project [3] and in the Framingham Study [4]. These calculations suggested that a 37% decline in the blood lead concentration, as observed in the USA from 1976 to 1980 [5], would result in a 5% fall in the incidence of fatal and non-fatal myocardial infarction, in a 7% decrease in the rate of fatal and non-fatal strokes and in a 5–6% decrease in total mortality over a 10-year period [6]. However, these extrapolations have been criticized by investigators who had analysed the same database [7,8].

No prospective data are as yet available to substantiate the claim that a reduction of lead exposure would actually result in a decrease in cardiovascular disease in the general population. Mortality studies in heavily exposed workers did not demonstrate any excess of cardiovascular mortality [9]. Furthermore, theoretical considerations often disagree with subsequent observations. For instance, contraceptive pill intake in middle-aged women is known to increase blood pressure by an average of 5 mmHg systolic and 1–2 mmHg diastolic [10] and to increase the risk of overt hypertension three- to sixfold [10]. Nevertheless, prospective studies failed to show any marked excess of cardiovascular risk attributable to the contraceptive pill [11]. Moreover, from the observational reports on the relationship between hypertension and ischaemic heart disease, a 5 mmHg decrease in diastolic blood pressure induced by antihypertensive treatment was expected to diminish the incidence of coronary heart disease by 20–25% [12], whereas in reality the intervention trials showed a reduction by no more than 14% [13]. In contrast to that which has been suggested [6], these findings demonstrate that one cannot infer from previous unrelated studies [5,10] whether and to what extent a reduction of lead exposure would alter the incidence of cardiovascular diseases in the general population.

Exposure to inorganic arsenic

The general population is exposed to inorganic arsenic via inhalation of contaminated dust particles, which may be emitted by primary smelters, glass manufacturing or coal burning plants [14]. People may also be exposed via the consumption of arsenic-containing foodstuffs, via tap or bottled water pumped from arsenic-containing geological layers or via the use of medications such as Fowler's solution. The relative importance of these sources may vary depending on the area of residence and life-style habits. Long-term exposure of the general population to arsenic may affect various organs such as the peripheral vascular system and the nervous system, but chronic exposure is mainly associated with a higher incidence of skin cancer and malignant tumours of various organs [14]. Among chronically exposed workers, it is mainly mortality from lung cancer that increases.

The exposure of the population to inorganic arsenic has been assessed in Belgium by measuring the 24 h urinary arsenic excretion in the CadmiBel (1985–1989) and PheeCad (1991–1995) surveys [15]. The baseline results (1985–1989), adjusted for sex, age and body mass index, showed that the 24 h arsenic excretion was considerably elevated in one area ($\geq$ 600 nmol/24 h), in which the tap water was enriched by inorganic arsenic, but which was also located in the vicinity of non-ferrous smelters and other potential industrial sources emitting inorganic arsenic. The follow-up examination (1991–1995) demonstrated that the exposure to inorganic arsenic had dropped nearly fivefold in the area with the highest 24 h urinary excretion, whereas the decreases in the other areas ranged approximately from a 50% reduction to a threefold decline. Industry was identified as a possible source of the increased exposure in the area with the highest exposure at baseline because (1) the urinary arsenic excretion did not follow the regional and temporal differences in the arsenic content of the drinking water, (2) in individual subjects the urinary arsenic excretion was inversely correlated with the distance to the nearest smelter, (3) an increased exposure was only observed downwind from the industrial sites, and (4) the fall in the urinary arsenic excretion over time was preceded by a decrease in the industrial activity. An important observation was that the official network monitoring the arsenic concentrations in airborne and precipitating dust had not detected the increased exposure. These findings [15] again highlight the necessity to validate environmental monitoring programmes by directly estimating the internal exposure of the population and, if possible, the suspected health effects.

Conclusions

The awareness that environmental issues should be viewed from the perspective of human health is growing. On whatever scale the environment constitutes a risk for human health, traditional epidemiological monitoring of human exposure and follow-up of disease and mortality may admittedly have limitations, because the association between exposure and health effects may be non-linear with a critical threshold above which health effects occur, or because there

may be undesirable delays before changes in chronic diseases are detected. Other approaches must therefore also be implemented, such as monitoring of the concentrations of toxic agents in air, water and soil, ecotoxicological studies and animal experiments. Although often costly, such studies are relatively easily to perform, usually provide results within a short time-span and reassure the public that something is being undertaken. However, as illustrated above, the internal exposure of people and the repercussions upon human health should be integrated as the most important outcome measures into existing and planned environmental monitoring programmes.

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