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The role of quantitative risk assessments

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Abstract

Risk is a multi-faceted concept, used in various ways within different academic disciplines. This paper discusses the relevance of quantitative risk assessments and other approaches to risk analysis in connection with decision making within the field of pollution abatement. It also discusses to what extent different decision frameworks actually require calculations of risks and dose-response relationships. Within toxicology and epidemiology risk usually refers to estimated values for the extent of damage on human health or corresponding individual risk estimates (averaged probability). However, in addition to beeing a question of calculated probabilities, the attitudes towards different kinds of risks are strongly conditioned by social, psychological and cultural factors, a fact that ought to be reflected in decision-support tools. Within economics risk may be analysed by transforming physical harm or other undesired effects into a common denominator, subjective utility, thus providing a technique to compare distinctly different types of benefits and risk. Finally, the paper also includes a summary of current knowledge on health effects of carcinogens and other toxic air pollutants, and calls attention to the need for further methodological development as regards risk analysis for toxic non-carcinogenic substances.

CONTENTS

1. INTRODUCTION

Man and the environment are subject to a variety of risks, one of which is anthropogenic pollution. Air pollution can hardly be avoided by any of us, beeing exposed to a mixture of gases and particulates from near-by and remote sources. How to reduce damage to health and environment due to air pollution is a debate that often is characterised by the existence of *multiple objectives, subjectivity and uncertainty* in the decision-making process. Decision-support tools in this field therefore ought to address these issues (Jones, 1989).

This paper deals with the concepts of quantitative risk assessment and damage estimation and their relevance for decision-making. It particularly focuses on damage to human health and also provides a summary of current knowledge on health effects of carcinogens and other hazardous air pollutants, which is important as input to decision-support tools. This is part of the the CAPE project ("Climate, Air Pollution and Energy: Cost-effective Strategies for Reduction of Emissions"), directed by CICERO, where the aim is to develop a comprehensive methodology and a decision-support tool for ranking of abatement measures regarding air pollution and greenhouse gases (see Aunan et al., 1993).

Generally, any rational decision process implies weighing of pros and cons of alternative choices leading to a recommendation of the alternative that altogether seems to meet the main objectives in the optimal way. To be able to assess the advantages, or benefits, of implementing abatement measures against air pollution, knowledge of the different steps in the impact pathway is required. These are i.a. the composition of emissions, how these are spread, transformed and deposited, to what extent people, vegetation and materials are exposed and what kind of damage this exposure can cause. Whether the benefits are monetized or not, they must be compared to the abatement costs and trade-offs will have to be made.

Within the quantitative risk assessment methods *risk* is defined as a function of two

components - probability and magnitude of damage - often with equal weight for both components. Within toxicology and epidemiology risk usually refers to modelled values for probability of damage on human health and environment. However, the notion may be used in various ways[1]. As an example, the psychological perspective on risk focuses on personal preferences and attempts to explain why individuals do not base their risk judgement on expected values. It is also concerned about the intuitive processing of uncertainty that causes biases in people's ability to draw inferences from probabilistic information (Renn, 1992)[2].

In the CAPE project risk estimates from epidemiological studies supplemented with estimates from toxicological studies will be the basis. As far as possible dose-response functions will be applied, to be able to estimate how changes in the pollution level contribute to altered risk levels concerning different end points and how the extent and magnitude of damage may change. When it comes to the valuation of effects, we will apply the economists' toolbox for risk analysis. Physical harm or other undesired effects are transformed into subjective utilities which serve as a common denominator, making it possible to compare options that have different benefit profiles and implementation costs. It is crucial that the major technical parameters are de-coupled from the major value judgements, which eventually should be made by decision-makers and not by the analyst (Simpson and Walker, 1987).

Uncertainty is a central element in risk analyses and applies to all steps in the anaysis. Moreover, lack of knowledge, concerning e.g. toxicity of components may imply that possible important hazards are not included. When it comes to the economic valuation of the effects connected to production and use of energy, existing studies suffer from large shortcomings (see Navrud, 1992b, for a survey of monetary estimates concerning environmental amenities). Additionally, as will be returned to, it is uncertainty and strong disagreement concerning what is really measured in the different valuation studies.

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[1]See e.g. Renn (1992) for a comprehensive classification of how the notion of risk is used within different disciplines.

[2]Karl Dake (1992) suggests that how peoples *perceive* probabilities of harm is best viewed as a matter of *shared cognition*, i.e it is not merely subjective, but rather resulting from intersubjective processes.

While uncertainty in most of the technical and scientific cause-effect relations may be elucidated by means of mathematical methods for simulation (see e.g. Morgan et al., 1985 and Trønnes and Seip, 1988), this does not necessarily apply to questions of more ethical nature. Still, it is possible to illustrate the influence of different ethical viewpoints e.g. by estimating how different objectives and alternative weighing of the analysis parameters influence the conclusion (see e.g. Shrader-Frechette, 1991).

In this ocean of uncertainty and subjectivity the value of performing analyses of this kind may seem limited. Maybe the process in itself is more useful than the results. However, getting a picture of the variety of effects, how different factors influence others, and performing sensitivity analyses for important parameters, undoubtedly contribute to more consistent decisions within the field of environmental protection.

2. CALCULATIONS OF THE "REAL RISK" - RELEVANCE FOR DECISION-MAKING

Decisions-makers are subject to pressure from various groups. The public opinion about a certain environmental issue is part of this pressure, and the eagerness to demonstrate efficiency may be stronger than the wish of making "rational" decisions. Implementing measures in such cases may be interpreted as a confirmation. However, a not really well-founded action may create groundless fear. Critical factors in risk management are the ability of authorities and experts of communicating knowledge about the different risks to the population, and also to what extent the authorities enjoy confidence in the population (see i.e. Morgan et al. (1992) and Slovic (1987) about risks and communication between experts and laity).

Calculation of different kinds of environmental risks is an area where the research accelerated in the 1970s. Quantitative risk assessment gradually became an independent scientific discipline, with USA as a pioneer country. By means of analytical methods like probabilistic assessments one set out to find the "real risk" connected to different hazards/technologies. The work partly had its motivation in the need for protecting the population and environment and partly in the needs of industrial and economical interests claiming that the constantly more stringent environmental legislation was

inconsistent, contradictory and counterproductive (Misa, 1990).

However, a certain scepticism evolved in the public opinion towards the risk analyses of experts and authorities, as expressions like "risk technocrats" indicate. There were many reasons for this. One of the problems with the early generations of risk analyses was that they often focused on mortality, or lost years of life, while people in general also are concerned about morbidity. From a socioeconomical point of view morbidity is of course also very important and should be incorporated in the analyses.

Traditional quantitative risk assessment regarding human health mainly build on toxicological studies. It is very difficult to use epidemiological data for verification since the analyses generally cannot cope with complex exposure situations and synergism between components (Silbergeld, 1993).

The most fundamental problem in basing decisions on quantitative risk assessments is, however, that it is not exclusively the "real risk" that matters to people. Attitudes towards different risks are not solely determined by expected numbers of deaths and people becoming ill per unit of time. Research indicates that attitudes towards risks are influenced i.a. by how well the causal connection is understood, in what way mortality and morbidity manifest (degree of dreadfulness), the possibilities one has to control the exposure to the hazard and whether exposure is voluntary or not (i.e smoking versus air pollution) (see i.a. Morgan, 1993 and Slovic, 1987). There are indications that people have considerably higher willingness to pay for reducing the risk of dying of cancer than dying in a traffic accident (Åkerman, 1987).

While some experts may complain of laymen's lack of knowledge and irrationality concerning perception of risk, others argue that the presumtively objective risk analysis is nonoperational when it comes to analysis of action strategies and policy analysis (Silbergeld, 1993). If the perception of risk in some cases is "wrong", according to the experts' calculations of probabilities and extent of damage, this *may* reflect that risk connected to this particular type of environmental hazard is especially unwanted. That is, minimizing this risk is highly valuated. In a democratic society it would be unsound to brush aside attitudes in the opinion as irrationale, because they may represent an

important ethical dimension that the scientific risk analyses have a tendency to disregard. This may consist of several elements, i.a. attitudes towards risk and uncertainty, and considerations of future generations, vulnerable groups in the population and vulnerable landscapes.

As will be returned to in the following, in cost-benefit analysis different methods are employed to evaluate amenities that are not traded on the market. To a certain degree this may be looked upon as an attempt to take into account this ethical dimension in the analyses.

3. PROTECTION OF HEALTH AND ENVIRONMENT; CHOICE OF DECISION FRAMEWORKS

In order to place the CAPE project in a wider perspective it is useful to take a look at different frameworks, or principles, for goal setting and action within the field of environmental protection. One question is whether and to what extent knowledge about risks and dose-response relationships is necessary within the various approaches.

The choice of principle is, of course, subjective. Which rules to follow depends on the attitudes and preferences of the citizens and the society as a whole. The different approaches nevertheless imply different guidelines for decision-makers and using different rules in different situations may lead to inconsistency in the environmental legislation.

In practice one usually finds a combination of several principles that can be formulated in a number of ways. Morgan (1993) uses the following categorization:

- technology based
- rights based
- utility based

Using the BAT-principle (best available technology), the target changes with the

technological developments. In some cases this principle appears to be defensive, in other cases the contrary, e.g. when the emission source in question apparently does not contribute significantly to the pollution problems. Basically, the BAT-principle does not address questions like the cost/benefit ratio and considerations for vulnerable groups in the population etc., but solely focus on the status of available technology. Indirectly, the implementation costs are obviously important when it comes to the discussion and definition of what is "best *available*". The technological development may, moreover, be pushed forward by applying economic and administrative incentives.

The BAT-principle, combined with a fixed percentage goal for reductions, is an important element in the first generations of international agreements on reduction of i.a. SO_2 and NOx. This type of agreement has no strong theoretical platform, strictly speaking, but has been employed because of its simple and pragmatic approach. (In the second generation of the SO_2 agreement, the principles of critical levels and target levels are central elements.)

Concerning radiation protection, the BAT-principle has its equivalent in the ALARA-principle (as low as reasonably achievable).

As can be seen, the BAT approach is largely independent of explicit calculations of risks connected to the different hazards and masks the inevitable value judgements and social trade-offs between risks and costs.

In Morgans terminology, the *rights-based* principle places justice in front of utility. This implies that decision-makers should consider the situation for vulnerable individuals and recipients simply because they have a *right* to protection from harm[3]. With this approach, the most important basis for decisions is established critical levels/loads, guidelines based on LOAEL (lowest observable adverse effect level) and estimates of acceptable doses.

The intention of protecting sensitive groups in the population, vulnerable vegetation, landscapes etc. is usually articulated in environmental legislation. However, one

[3]See also Shrader-Frechette (1991) p. 185 concerning ethical principles for decisions.

hardly finds the rights-based approach purified. In most cases some kind of utilitaristic approach is employed. Thus, in the Norwegian Pollution Control Act it is stated that "The Act shall be used to achieve satisfactory environmental quality on the basis of a total appraisal of health, welfare, the natural environment, costs related to control measures and economic considerations" (Ministry of Environment, 1981). As will be discussed later on, it is possible to incorporate, at least to some extent, considerations of sensitive groups etc., within the cost/benefit model by means of weighing and evaluation methods.

The *utility-based* approach has its origin in economical theory (welfare theory), where the principle of optimal allocation of resources and maximizing the total welfare in the society is central. Two main types of amenities are vital to the social welfare, private goods and public goods. The environment in which we live is a public good and pollution may deteriorate it. Additionally, pollution may also reduce private goods, for example if high ozone levels damage agricultural crops and thereby reduce the income of the farmer.

In traditional cost/benefit analysis one attempts to evaluate all the costs and benefits of specific projects on behalf of the society, and to quantify them in one unit - money. The interest for employing economic damage estimates in air pollution regulation has increased, and in the USA the claim for economic estimates of the consequences of larger projects is obligatory. In other countries, including Norway, this work so far has been more or less ad hoc (Markandya, 1993).

In reality it is of course very difficult, some would say impossible or not desirable, to monetize the value of damages to human health and environment. Valuation of environmental amenities has become a large research field where particularly economists have played a central role (often with critical psychologists in hot pursuit). There are two main approaches to valuation of environmental goods that have no market prices. Within the one direction one attempts to reveal *people's preferences* either by means of interview techniques as in willingness-to-pay studies or by observing their behaviour for example on the housing market (hedonic pricing techniques) (see i.a. Navrud, 1992a and 1992b and Strand, 1990). The other principal direction uses expert

panels. Assisted by interactive data programs a small group of well informed persons are interviewed about their opinions (see Wenstøp et al., 1994). Whatever evaluation method applied, knowledge about dose-response relations is crucial in analyses of optimal abatement strategies.

A fundamental question is whether one gets hold of the *social values* by measuring *people's preferences* via for example their willingness to pay and, in any case, what is the most relevant parameter of the two. Do for instance willingness-to-pay studies measure preferences that can be used to deduce how people will actually act, or is it rather their attitudes (to how one ideally should act) that is measured? Seip and Strand (1992) performed a study indicating that people do not necessarily act as could be predicted by this kind of studies, because they have a tendency to overstate their willingness to pay. It is also a question whether it poses an unrealistic cognitive demand upon people to ask them to state their preferences for environmental goods in monetary units (see i.a. Gregory et al. 1993, Andersen, 1993, Kahneman and Knetsch, 1992). Traditionally, natural scientists have been somewhat sceptical to monetization in general, and maintained that the physical description of effects should be in focus (NAPAP, 1991).

Principally, cost/benefit analysis can be used not only to assess concrete projects and action plans, but also in more general goal settings, for instance concerning air quality guidelines. There are, however, very few examples where air pollution standards and guidelines are based on cost/benefit analyses (Markandya, 1993). An exception, as far as Norway is concerned, is the ongoing work on establishing legally binding standards for local air quality and noise where cost/benefit ratios of alternative levels are estimated.

Different decision frameworks have different qualities in terms of economic efficiency, equity and administrative simplicity. According to Pearce et al. (1992) there is an antagonism between these qualities. While the cost-benefit (or risk-benefit) approach has a high degree of economic efficiency, it is encumbered with low administrativ simplicity and equity. Technology-based approaches and regulations and bans on the other hand, have a high degree of administrativ simplicity and equity, but suffer from a low economic efficiency.

4. THE RELATION BETWEEN EXPOSURE AND RISK TO HUMAN HEALTH DUE TO AIR POLLUTANTS

Knowledge about exposure-risk relations for air pollutants has been essential as a basis for establishing guidelines, acceptable exposure levels, emission standards etc. It is now a growing interest in utilizing this knowledge for economic valuation purposes.

Knowledge on dose-response relations builds on studies of the object concerned (humans, animal species, vegetation, materials) in its natural environment or under some form of controlled conditions. Moreover, it is usual that results from studies in one type of target object provide a basis for evaluating effects in others, e.g. extrapolation from experimental animals to humans (SFT, 1992). To estimate the effects of long-term exposure a combination of short-term tests on humans, long-term animal test and epidemiology is necessary. Expert panels can also be employed to estimate dose-response functions for long-term exposure based on the joint information available (see e.g. Trønnes et al., 1986).

Biological damage effects can be categorized in the following way:

	Morbidity	**Mortality**
Acute effects	E.g. increased susceptibility to infections in the respiratory system, asthmatic attacks [1)], impaired pulmonary function, chest pain	E.g caused by asthmatic attack, acute obstructive respiratory incidence, heart attack
Delayed effects	E.g. chronic bronchitis, chronic lung disease, promotion/initiation of cancer	E.g. caused by chronic bronchitis, cancer

[1)] Even though one has quite clear indications that air pollution may contribute to provoke asthmatic attacks, it is still not clear whether air pollution in itself contributes to the initial development of the disease.

Morbidity may be either reversible or irreversible. Some minor effects are better

categorized as irritations, e.g. eye irritations due to PAN.

Care must be taken to distinguish acute and delayed *effects* from acute and chronic *exposure*, because delayed effects may or may not require chronic exposure[4].

Delayed effects, or slow poisoning, can occur in several different ways (Rodricks, 1992). In some cases, chemicals or their metabolites accumulate in the body over time because the rates of excretion are less than rates of absorption. After some time the tissue and blood concentrations become sufficiently high to cause injury. In other cases, chemicals do not accumulate in the body, but attain to cause some small amount of damage before they are excreted. The sum of many small events will eventually manifest as larger observable damage. Finally, delayed effects may originate from acute or chronic exposure to chemicals that alter DNA in a way that causes disease, e.g. cancer.

Traditionally, approaches to health risk assessment from chemicals represent a dichotomy: Carcinogens and chemicals that cause other toxic effects (some components belong to both groups).

4.1 Carcinogens

In the case of carcinogenic substances, there is generally assumed to be no lower limit below which the dose has no effect. Overincidence decreases with decreasing dose towards zero. The dose-response relation is usually based on data for high doses and there are large uncertainties about the actual effects at small doses. For most substances it is not clear whether the dose-response relations are linear functions or not. They are often assumed to be linear, an assumption that, in connection with action planning, may be legitimized by the need for ensuring that the risk is not underestimated (conservative estimates).

For the purpose of environmental regulation, carcinogens are treated by much the same principles as radiation and in risk assessment of carcinogens radiative equivalents, resulting from e.g. comparative mutagenicity tests, are often used (Törnqvist, M. og

[4]We also have subchronical exposure. This is not precisely defined, but refers to repeated exposure through a certain part of life.

Ehrenberg, L., 1989). An "acceptable exposure level" based on calculated life time risks at definite expose levels is usually given. Establishing these acceptable levels implies considerations of what are acceptable levels of risks, considerations that are beyond the sphere of natural and medical sciences.

Concerning energy production and emission of air pollutants, carcinogenic components are mainly due to incomplete combustion of hydrocarbons. An important chemical group in this connection is polycyclic organic matter (POM), a collective term for i.a. polycyclic aromatic hydrocarbons (PAH), substituted aromatic hydrocarbons (e.g. nitrated PAHs) and heterocyclic aromatic compounds (e.g. aza-arenes) (Lewtas et al., 1992).

The carcinogenic potency of the different compounds varies a lot. In condensate from car exhaust and combustion of coal the 4-7 ring PAH fraction represents nearly the entire carcinogenic potential of PAH. PAH-components with 2 or 3 rings have not been found to be carcinogenic. Dibenzo(a,h)anthracene and benzo(a)pyrene, BaP, are the most potent PAH-components and BaP are often used as indicator of the carcinogenicity of PAH (SFT, 1992).

Processes in the atmosphere contribute to altering the chemical composition and mutagenicity of incompletely combusted hydrocarbons. Nitrated PAH-compounds are of special interest in this connection, and the mutagenic activities of NO_2-PAH and OH-1-NO_2-pyrene isomers are well documented (Nishioka and Lewtas, 1992, and references therein). The higher mutagenicity per ng that is observed for condensate from car exhaust compared to e.g. exhaust gasses from wood combustion (which contains *more* PAH), is probably due to atmospheric chemical processes (i.a. photochemical) where presence of NO_x is important for the formation of nitrated PAHs in the motor vehicle emissions. (Lewtas et al., 1992).

In the body PAH is metabolized by the oxidizing enzyme system P450, to epoxides and/or phenols. It is probably the oxidized products that are the actual carcinogens. The oxidizing process may, however, also convert the metabolites into soluble substances which are eliminated in urine and faeces, and PAH show little tendency to

accumulate in fatty tissue (SFT, 1992).

Due to the long latence time in development of cancer diseases, for lung cancer up to 30 years, it is most difficult to clarify cause-effect relations by means of epidemiology. During such a long period of time the exposure may vary enormously because of possible changes in occupation, smoking habits, place of residence etc. Comparisons of urban and rural nonsmokers indicate that urban lung cancer death rates are approximately twice those found in rural areas. The results of a series of studies, however, cannot confirm nor invalidate that air pollution *per se* causes the increase in lung cancer incidence that actually is observed in the cities (Godish, 1991). However, studies in the Eastern Europe, e.g. in the surroundings of Katowice (Silesia region) where the concentrations of PAH may be 10-30-fold higher than in most Western European countries[5], demonstrate significant increases in molecular markers as carcinogen-DNA adducts (PAH-DNA and aromatic adducts) (Perera et al., 1992).

Estimates of urban cancer risk have previously focused primarily on the particulate fraction, because inhaled PAH is mainly adsorbed to particles of soot from combustion. However, it is now an increasing attention towards volatile and semivolatile components. A quantitative important component in this connection is ethene from automobile exhaust. Törnqvist and Ehrenberg (1992) have estimated a lifetime risk of cancer from ethene to 1.4×10^{-4} per $\mu g\ m^{-3}$ and from PAH (BaP) to 12×10^{-4} per $ng\ m^{-3}$ ([6]). The exposure of the population to ethene in urban air, is however, far higher than for PAH, resulting in respectively 30 (ethene) and 100 (PAH as BaP) estimated cancer incidences in Sweden caused by the two components. It is assumed that other quantitative important components are butadiene, formaldehyde and benzene. The authors, however, emphasize that the data do not exclude that the risk is zero.

Törnqvist and Ehrenberg (1989) have also made an attempt to estimate the total cancer risk (all types of cancer) from urban air pollution in Sweden and arrived at a figure of

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[5]In Gliwice, a town near Katowice, a mean concentration for BaP of approximately 60 ng/m^3 has been measured during the winter months (Perera et al., 1992).

[6]As a comparison WHO (1987) indicates a life time cancer risk of 9×10^{-5} at an exposure of 1 ng BaP/m^3 in 70 years. The discrepancy indicates the large uncertainties in the estimates.

at least 700 cases annually, corresponding to an average risk in the order of 10^{-4} per year in the population of 8.4 million. One great uncertainty in this estimate is due to insufficient understanding of the action mechanisms of some semivolatile components, such as fluoranthene (Törnqvist and Ehrenberg, 1990).

4.2 Other toxic components

In the case of components as SO_2, NO_x, particulates, CO, ozone and acid aerosols, it is generally assumed that there are threshold values for health effects. It should therefore be possible to elaborate values for "LOAEL" - lowest observable adverse effect level. In reality it is also here, as for carcinogens, great uncertainty considering what happens as the dose approaches zero.

While it has been payed much attention to dose-response relationships for carcinogens, the methods for risk analysis for toxic non-carcinogenic substances are primitive and nonspesific with respect to the biology of target organ systems (Silbergeld, 1993). To be able to compare risk associated with carcinogens directly to risk associated with non-carcinogens one needs to calculate the dose-response functions down to the lowest effect level. No well-accepted approaches to such calculations are currently available (Farland and Dourson, 1993). The explanation to this includes uncertainty concerning the relation between effects due to different exposure patterns, e.g. short-term exposure to high concentrations versus exposure to constantly repeating peaks or long-term exposure to lower concentrations. Furthermore, synergism may strongly complicate calculations of dose-response functions.

For carcinogenic substances it is principally a well-defined end point that is studied, namely cancer incidence, and it is generally assumed that the *accumulated* dose in relevant target organs is of main interest concerning development of the different cancer deseases (the time lag for development of the different types does, however, vary). When it comes to the non-carcinogenic components the picture is different. A number of end points can be observed, this means the *dose-effect* relation of an individual may exhibit different types of effects. Taking CO as an example, possible effects are headache, reduced vigilance and physical endurance at lower doses (measured by the carboxyhaemoglobin-percent in the blood) and nausea and heart

attack in angina patients at higher doses. NO_2 may increase the susceptibility to infections in the respiratory tract, it may contribute to provoke an asthma attack and it is also suspected to have a cocarcinogenic/promotive effect, at least at higher exposure levels (SFT, 1992, Törnqvist and Ehrenberg, 1992).

Traditionally, it has been payed most attention to acute effects of peak concentrations, or episodes. The classical example in epidemiology is the incident in London, the winter of 1952, where it was registred about 4000 more deaths than expected following a severe smog episode (Ministry of Health, 1954). A study in Crakow (Krzyzanowski og Wojtyniak, 1991/1992) indicates a clear correlation between air pollution episodes and daily mortality in the following four days. Concerning e.g. SO_2 and suspended particulate matter (important components in winter smog) increased acute mortality is associated with daily mean concentrations above 500 $\mu g/m^3$ for both components (SFT, 1992). As a comparison this is far beyond the concentrations in Norwegian cities today, where the mean values in the winter months are in the area 5-20 $\mu g/m^3$ (Grønskei et al., 1992)[7]. However, several research papers the last years show increasingly more concern about particulates, especially the inhalable fraction PM_{10}, indicating increased mortality associated with rather low concentration levels. It has been suggested that for every 10 $\mu g/m^3$ increase in the concentration of PM_{10}, there is a 1% increase in the number of deaths (see Bown, 1994).

For substances like SO_2, suspended particulate matter, NO_2 and ozone, the acute health effects primarily manifest as irritations and constriction in the respiratory tract (cough), increased susceptibility to infections (both because of reduced clearance and influence on the immune system) and reduced pulmonary function that may lead to reduced physical capacity. The condition of persons with respiratory diseases may be aggravated (SFT, 1992).

Lately, there has been an increasing attention towards the effects of long-term continuous exposure to lower concentrations and to repeating episodes over a lower background concentration. Concerning ozone, for instance, studies (i.a. experiments in

[7] An exception is the impact area for the Russian town Nikel, where mean hour concentrations beyond 1600 $\mu g/m^3$ have been measured at two stations in the northernmost Norway.

animals) indicate that long-term exposure may cause irreversible damage due to inflammations that induce formation of scars (synthesis of connective tissue) which then may cause development of chronical pulmonary disease. Some studies also indicate accelerated ageing of the lungs, which may be connected to living in areas with elevated ozone concentrations (i.a. SFT, 1992, McKee og Rodriguez, 1993). There is some evidence that long-term exposure to NO_2 and SO_2/suspended particulate matter is associated with a higher prevalence of respiratory diseases, e.g. bronchitis, in children and adults. Because the lungs have reatively few ways of responding and the fact that lung diseases generally develop as a result of many co-operating factors, it is usually hard to tell to what extent the effects are caused by air pollution. It is, of course, even harder to assess the importance of a single component.

Asthmatics and children are generally assumed to be vulnerable groups as regards air pollution. Asthmatics may be 10 times more sensitive to SO_2 than healthy people (SFT, 1992). Persons that exercise a lot in polluted air are also assumed to experience a higher risk, because the dose gets higher with increased inhalation and mouth breathing. Epidemiological studies indicate that elderly people are a vulnerable group, but it is not clear whether this is due to increased sensitivity to short-term elevated concentrations or a cumulative effect from long-term exposure (Gottinger, 1983).

Epidemiological studies do not always focus on the biological end points. The end points that are focused may be divided into three categories:

- *Biological end points,* e.g. mortality or lost years of life, reduced lung function, prevalence of bronchitis, asthma attacks, or eye irritation.
- *Subjective nuisance,* e.g. people afflicted by dust and soiling, smell, or reduced visibility.
- *Social performance,* e.g. occupational absence and hospital admission (due to given health symptoms).

In connetion with economic valuation of health damage due to air pollution, these categories have different advantages and drawbacks. The biological parameters are assumed to be relatively reliable. This specially applies to measurments of changes in

the lung function (CEC/US, 1993). The problem is that it is often difficult to predict the medical significance of these changes, e.g. in terms of affected physical capacity and life quality. Social performance, like emergency ward visits, is easier to assess in monetary units. On the other hand these parameters are highly dependent on social conditions, e.g. access to and costs of these services. When applying results from one country in an other these factors may imply great uncertainties.

CONCLUSIONS

Quantitative estimates of how environmental pollution contributes to risk concerning different types of damage are necessary as part of the basis for decisions on abatement measures. There are large uncertainties and lack of knowledge as regards the relation between exposure and damage, especially when it comes to long-term exposure and delayed health effects, and the methods for risk analysis for non-carcinogenic substances are still primitive. To be able to elaborate cost-effective abatement strategies it is essential to assess the different components and problem areas in an integrated context.

In model tools that are ment to support decision-makers in the process of choosing abatement measures and framing a policy, it is, however, not sufficient to estimate the extent of the damage and potential changes in the risk in a narrow technical-scientific perspective. It is also necessary to allow for considerations of attitudes and perceptions in the public opinion towards different risks. The question is not whether these are rationale, but how they may be included in the analysis.

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