METHODOLOGIES OF HEALTH IMPACT ASSESSMENT AS PART OF AN INTEGRATED APPROACH TO REDUCE EFFECTS OF AIR POLLUTION

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Abstract

Quantification of average frequencies of health effects on a population level is an essential part of an integrated assessment of pollution effects. Epidemiological studies seem to provide the best basis for such estimates. This paper gives an introduction to a methodology for health impact assessment. It also gives results from some selected parts of a case-study in Hungary. This study is aimed at testing and improving the methodology for integrated assessment and focuses on energy production and consumption and implications concerning air pollution. Using monitoring data from Budapest, estimates of excess frequencies of respiratory illness, mortality and other health end-points are given. Concerning a number of health end-points, particles probably may serve as a good indicator component. Stochastic simulation is used to illustrate the uncertainties which are embedded in the exposure-response functions applied.

KEY WORDS: Health effects; air pollution; case-study; Hungary; stochastic simulation.

1. INTRODUCTION

The economic damage due to adverse effects on health and environment caused by air pollution may be very large in some countries. It is also very difficult to assess, because there often are no market prices for the qualities that are damaged, for instance for human health and cultural assets. Concerning the possible effects of emissions of greenhouse gases, the damage may be especially large, and so are the uncertainties. The size of the investments needed to reduce emissions may on the other hand also be very large and have marked effects on the economy of a country.

In the context of analysing abatement strategies an *integrated approach*, across different pollution components and environmental problems, is needed to develop optimal, cost-effective strategies. A ranking of measures based on several effects may turn out to be quite different from that obtained considering only one effect such as climate change. Under the bilateral environmental agreement between Hungary and Norway a joint project between Hungarian and Norwegian researchers is used as a case study aimed at testing and improving the methodology concerning integrated damage assessment. The study partly draws upon experiences from a project carried out in Norway - the Oslo Air Study. In this study, initiated and organised by the Norwegian State Pollution Control Authority, an analysis of air pollution abatement measures related to the city of Oslo was conducted (SFT, 1987; Trønnes and Seip 1988).

The following gives an introduction to the methodology and results from some selected parts of the Hungary study (see also Seip et al., 1995 and Aunan et al., 1995a).

2. INTEGRATED ASSESSMENT

Basically two approaches may be used to find cost-effective abatement strategies against pollution damages (Aaheim, 1995; Aunan et al., 1995b). In the "top-down approach" (T-D) the assessment is done by the use of macroeconomic models, which are particularly suitable for analysing the impact of general measures, as taxes, on main macroeconomic variables. From the predicted changes in economic activity the emission reductions are deduced, and in principle the benefits from these reductions can be fed back into the macroeconomic variables. In the "bottom-up approach" (B-U) specific abatement measures, for instance emission standards for vehicles, considered appropriate for solving a problem are explored in detail. Their potentials for reducing adverse exposure of recipients (people, crops, forests, materials etc.) and damage are estimated. Assessments of the values of the costs and benefits are then made according to observed or estimated market prices. To a large extent monetization of environmental and health qualities depends on subjective valuation and various methods have been applied for this purpose, all of them have weaknesses and problems (OECD, 1989; Navrud, 1994; Wenstøp et al., 1994). The social net benefit provides the basis for a ranking of measures.

Both approaches do, however, have major weaknesses: While T-D analyses tend to oversimplify the biogeochemical relations, the B-U analyses tend to oversimplify, or simply leave out, macroeconomic consequences.

Our approach is the B-U, because the main focus is the *damage assessment*, i.e. the relations between emission sources, concentration levels, exposure and effects on health, vegetation, materials and climate. We also believe that this approach has advantages in explicit valuation of environmental amenities. Besides, the special transient economic situation in post-communist countries at present would be extremely difficult to model. It is, however, necessary to analyse the political, institutional and socio-economic environment within which abatement strategies will be chosen, in order to make the analysis realistic.

3. THE SEVERITY OF AIR POLLUTION IN DIFFERENT AREAS

The emissions of air pollutants have been decreasing the last decade in many areas in Eastern and Central Europe, but there are still some areas where the pollution load is very high. In Hungary the main reasons for the reduced emissions are a general economic recession, increased use of nuclear energy, some fuel switching from coal to oil and gas, some specific abatement measures, and structural changes in the economy. In many ways the changes display the same pattern as in Western Europe a couple of decades ago, industry becoming less important as a pollution source and the transportation sector becoming more important. A predominant feature is that the private transportation does not seem to be prevented from growing even by deep economic recession. Traffic has become the fastest growing urban air pollution source in the past 15 years. In the period 1985-1992 the reductions in green-house gases and air pollutants in Hungary were approximately: CO_2: 26%, SO_2: 40%, TSP (total suspended particulates): 60%, NO_x: 28%, CO: 21%, nmVOC (non-methane VOC): 45%. The uncertainties in the emission data given are estimated to ±15% (see also Tajthy, 1993 and Tajthy et al., 1990).

Whereas the per capita emission of SO_2 has been decreasing in Hungary, as in Polen, since the mid-1980s, the trend in China is the opposite. The per capita emission of SO_2 is considerably lower than in Eastern and Central Europe, but is steadily increasing (see fig. 1). In some areas in China the average concentration level is several times higher as compared to e.g. Crakow, one of the more heavily polluted areas in Eastern Europe.

In Eastern and Central Europe large relative and absolute declines in health status since the mid-1960s are well documented (Feachem, 1994). It is believed that the main causes are lifestyle factors (diet, abuse of tobacco or alcohol), but pollution may also play a role (up to 9%). In some parts of China, where the pollution situation is far more severe, large effects on human health and environment are likely to occur.

Fig. 1. Per capita SO_2-emissions in China, Polen, Hungary and Norway.

4. ASSESSING HEALTH EFFECTS

A rough, semi-quantitative, indication of the seriousness of the pollution situation as regards health effects can be obtained by estimating the number of people living in areas where concentration levels exceed air quality guidelines. In the Oslo Air Study such estimates were the criteria by which the impact of an abatement measure was quantitatively assessed (Trønnes and Seip, 1988). Estimates are provided for Hungary in Sections 5.1 and 5.2.

However, the prevalence of effects will increase in the population as the concentration increases above the guideline and it has also been shown that the exposure-response curves for some substances seem to continue below guideline levels as given e.g. by the WHO (1987) (see e.g. Schwartz et al., 1988; Sunyer et al., 1991; Schwartz, 1992; Braun-Fahrländer et al., 1992; Brunekreef et al., 1995).

In this study we utilize findings on exposure-response relations in epidemiological studies to estimate public health effects, realising that the functions entail large uncertainties. The functions indicate the increased risk of experiencing an effect when the concentration level rises to a higher level, expressed as relative risk (RR) or the odds ratio (OR):

$RR = p_1/p_0$
$OR = p_1(1-p_0)/p_0(1-p_1)$

p_1 and p_0 are the probabilities of a person getting the effect, or seen the other way, the average prevalence or incidence frequency[1] in a population, given a higher or lower concentration level, respectively.

General concerns with epidemiological studies are misclassification of exposure, biases in the sample population, correlated variables (confounders) and omitted variables. For instance, misclassification of exposure has been found to result in a downward bias of the observed association between air pollution and health effects (Roemer et al., 1993, and Krupnick et al., 1990). On the other hand, the great advantage of epidemiological studies versus experimental studies in animals and clinical studies in humans, is the fact that effects are studied in a population with all degrees of susceptibility present, and even if the concentration levels measured may be far from the actual exposure, it is probably a good indicator of the relative pollution load. Meta-analyses, analyses based on results from several studies by use of a formalised statistical technique, have become an important tool to establish exposure-response functions on a broader basis (Mann, 1990). It is a matter of course that the results are seen in the light of toxicological studies to ensure that assumed cause-effect relations are biologically plausible.

When interpreting results from epidemiological studies, it should be held in mind that the confidence limits only take into consideration random variation in the data, not the systematic errors, the biases and confounders. Some epidemiologists hold, as a general rule of thumb, that the relative risk found to be associated with a certain increase in a risk factor should be three or more to be taken seriously. In air pollution epidemiology this is very rarely the case, and other epidemiologists say that an association with an increased risk of tens of percents may be believed if it shows up consistently in many different studies (see Taubes, 1995). Concerning for instance associations of particulate air pollution and daily mortality, numerous epidemiological studies show quantitatively similar and consistent findings on exposure-dependent increases in risks, and as noted by Schwartz (1994) the burden of proof has now shifted. It is incumbent on those who disbelieve the causality to provide evidence showing how the associations are confounded. As long as such evidence is absent, it is prudent to treat these associations as causal.

The association between health effects and different components may be difficult to disentangle in epidemiological studies. In our project the different health effects are attributed to one *indicator component*, primarily for pragmatic reasons. There are strong indications that *particles* are a key agent for many effects, partly, but not solely, as a vector for other components.

A number of different particle mass measures are employed in different epidemiological studies. This complicates an evaluation in terms of one single indicator component and is an obstacle for using any particle measure as indicator. Common measures are TSP (total suspended particles), PM_{10}, $PM_{2.5}$, black smoke (BS), and various measures of visual range and particle optical reflectance, e.g. Coefficient of Haze (CoH). The carboneous part of the particles may have various organic chemicals, some of which may be mutagenic, adsorbed

[1] Prevalence is the percentage of the population which has the effect at any given point of time (point prevalence). Incidence is the percentage of the population for which a new episode of the effect occurs during a specific period. If a symptom has an average duration of 2 days, the average daily prevalence will be twice the average daily incidence.

onto its surface and particles may also contain heavy metals involved in carcinogenic processes.

Whereas the coarser fractions, up to as large as 100 μm, indeed are important for some upper airway effects, the finer fractions, below 10 μm, are assumed to be better indicators for airway effects in general and effects in the small airways and alveoli in particular. However, often only TSP is measured and an exposure-response function for TSP is estimated, regardless of what kind of effect is looked at. TSP then acts as an indicator of PM_{10} or some other particle-related fraction, e.g. acid aerosols as discussed by Fairley (1990). In a study by Özkaynak and Thurston (1987), particle measures closely related to the respirable fraction ($PM_{2.5}$) and/or the toxic fraction (e.g. SO_4^{2-}), seemed to be better predictors of health risks linked to mortality than both TSP and PM_{10}.

Particles, SO_2 and NO_2 are both chemically and biologically interrelated because the gases participate in the formation of particles, and because the components partly are associated with the same type of effects. O_3, on the other hand, is chemically more independent from reactions leading to particle formation and is partially associated with other types of effects, mainly in the lower respiratory system. In light of the consistent evidence for health impacts of O_3 independent from simultaneous exposure to particles, O_3 should be regarded as an indicator component in addition to particles. However, since O_3 formation depends on the concentration of NO_2, nitrate formation may be accelerated by O_3, and since acidic components may potentiate the respiratory effects of O_3, this indicator is not fully independent of what could be called the particles/SO_2/NO_2 complex (see e.g. Lübkert-Alcamo and Krzyzanowski, in press).

Epidemiological studies focus on different end-points, that can be divided into two categories:

- *Biological end-points*, e.g. mortality or lost years of life, reduced lung function, prevalence of bronchitis, asthma attacks, and eye irritation. Biological end-points may be self reported or measured/reported by professionals.
- *Consequential end-points*, e.g. occupational and school absenteeism and hospital admissions (due to given health symptoms).

In connection with economic valuation of health damage due to air pollution, these categories have different advantages and drawbacks. Some of the biological parameters are assumed to be relatively reliable, e.g. measurements 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, respiratory morbidity and life quality (SFT, 1992). Consequential end-points, like emergency ward visits, are easier to assess in monetary units. On the other hand these parameters are highly dependent on social conditions, for instance access to and costs of these services. When results from one country are to be applied in another, these factors may imply great uncertainties. Studies reporting biological end-points are focused in the following. However, the relative response in the two categories of end-points should in most cases correlate.

A number of different effects are associated with air pollutants and classification of the different effects is a matter of balancing the need to define effect categories easily valuated in economic terms, and the need to render the information given in the studies as precisely as possible.

The following categories of health effects are focused here:

Reversible effects:
- Acute respiratory symptoms in children and in adults
- Chronic respiratory symptoms in children and in adults
- Asthma episodes in children and in adults
- Eye irritations
- Headache

Irreversible effects:
- Irreversible lung damage in children
- Excess mortality
- Cancer incidence

In addition to these effects an association between reduced pulmonary function and air pollutants is shown in many epidemiological studies (e.g. Moseler et al. 1994; Mohan Rao et al, 1992; Hoek and Brunekreef, 1994; Berry et al., 1991). Here reduced pulmonary function is not included as an effect category, because the medical consequences are unclear, as discussed above.

Different mathematical models are used in the studies to approximate exposure-response relationships in a population. Many studies solely report the relative increase *within* the concentration range that is observed (or estimated), whereas in other studies a function assumed to be valid outside the range is adjusted to the data. Logistic regression models, which are discrete regression models well suited for 0-1 data (no effect - effect), are often applied in epidemiological studies of for instance respiratory effects (e.g. Schwartz et al., 1988; Dockerey et al., 1989; Schwartz and Zeger, 1990; Krupnick et al., 1990). A logistic function is given by:

$$f(x) = \frac{\exp(V)}{1+\exp(V)} \qquad \text{where} \qquad V(x) = \alpha + \beta x + \varepsilon$$

and α is the intercept of $V(x)$, β is the regression coefficient, x is the independent variable and ε is the error component.

It is very difficult to demonstrate a specific no-effect level in a population by means of epidemiology. Clinical studies on humans may give indications of a threshold level, but the most sensitive individuals are, naturally, not represented in such studies. Air quality guidelines based on a no-effect level criterion may therefore merely give an indication of the level where, in simple statistical analysis, the role of air pollution is swamped by the background of much larger causal factors. Concerning the exposure-response relation in a population the S-shaped curve is biologically more plausible than the “hockey stick” (threshold of zero response followed by a linear exposure-response curve), because it reflects a normally distributed variation in susceptibility in a population (the logistic distribution is very close to the cumulative normal distribution).
Another feature of the logistic model, which makes it particularly attractive within epidemiology, is that the odds ratio is easily calculated:

$$OR_i = e^{\beta \Delta Ci} \quad (1a)$$

where β is the regression coefficient and ΔC_i is the change in concentration. The same applies to Poisson regression, often used in studies of mortality rates and other low frequency events, where:

$$RR_i = e^{\beta \Delta Ci} \quad (1b)$$

Since $e^{\beta C} = 1 + \beta C + {}^1\!/_2 (\beta C)^2$ .....,

a *linear percent point increase in response* can easily be calculated to be

1000β per 10 μg/m^3, if βC <<1.

When linearised functions are used there is a danger of biases by overestimating at lower concentrations and underestimating at higher. Within the concentration ranges usually observed in ambient air, however, nonlinearity in exposure-response functions is often of little practical relevance.

The functions make possible estimates of the relative change in effect frequency, and not e.g. excess cases per population unit. This is because a relative estimate probably is more appropriate than an absolute when the function is to be employed in a new geographical context, where the frequency of the effects in question may be different for other reasons than air pollution. As far as possible one should employ data on actual prevalence of health effects when estimating for instance reduced prevalence associated with reduced concentration levels. However, often one does not have data for frequency of different symptoms and diseases. To make possible estimations in such cases, a baseline prevalence, here called *hypothetical zero-concentration prevalence,* denoted p_0, is estimated for each effect. These are calculated from the data for actual prevalence (or incidence rates multiplied with average duration) and concentration levels given in the studies, and are extrapolated down to zero concentration by use of the OR or RR functions. Data on prevalence may differ substantially depending on survey methodology, e.g. whether symptoms are self-reported or self-reported with subsequent clinical consultation. This implies that prevalence figures should be employed with some discernment. The hypothetical zero-concentration prevalence is given by:

$$p_0 = \frac{p^{obs}}{OR_i - OR_i \cdot p^{obs} + p^{obs}} \quad (2a)$$

or

$$p_0 = \frac{p^{obs}}{RR_i} \quad (2b)$$

where OR_i and RR_i are, respectively, the odds ratio and relative risk estimated (by use of the regression coefficient β) for an increase in the concentration level from zero to C_i, and C_i is

the concentration level at which the observed prevalence is p^{obs}. The rationale for this procedure is that the observed prevalence to some extent is caused by air pollution.

For any concentration level, C_i, the prevalence, p_i, can be calculated:

$$p_i = \frac{OR_i \cdot p_0}{1 - p_0 + OR_i \cdot p_0} \tag{3a}$$

or

$$p_i = p_0 \cdot RR_i \tag{3b}$$

For *short-term health effects* the exposure-response functions given in the following are usually derived from the relation between daily mean concentrations, C_i, and the daily prevalence of the effects, $p(C_i)$. To estimate the annual number of symptom days ("person-days"), S_{sum} , in a population N one ideally should calculate the daily number of affected persons in the population and sum up over the year:

$$S_{sum} = \sum_{i=1}^{365} \left(p(C_i) \cdot N \right) = \sum_{i=1}^{365} \left(\frac{\exp(\beta \cdot C_i) \cdot p_0}{1 - p_0 + \exp(\beta \cdot C_i) \cdot p_0} \cdot N \right) \tag{4a}$$

or

$$S_{sum} = \sum_{i=1}^{365} \left(p(C_i) \cdot N \right) = \sum_{i=1}^{365} p_0 \cdot \exp(\beta \cdot C_i) \cdot N \tag{4b}$$

However, this procedure is time-consuming and in some cases one does not have data for daily means. An approximation of S_{sum}, called $S_{averaged}$, can be obtained by using an estimated average prevalence as a function of the annual mean concentration, $\overline{C}_{ann}$. By doing this one assumes linearity in the response function around $\overline{C}_{ann}$:

$$S_{averaged} = p(\overline{C}_{ann}) \cdot N \cdot 365 = \frac{\exp(\beta \cdot \overline{C}_{ann}) \cdot p_0}{1 - p_0 + \exp(\beta \cdot \overline{C}_{ann}) \cdot p_0} \cdot N \cdot 365 \tag{5a}$$

or

$$S_{averaged} = p(\overline{C}_{ann}) \cdot N \cdot 365 = p_0 \cdot \exp(\beta \cdot \overline{C}_{ann}) \cdot N \cdot 365 \tag{5b}$$

The difference between S_{sum} and $S_{averaged}$ depends mainly on the linearity in the response function in the concentration range of concern and to some extent on the distribution of the daily concentration levels. The difference gets larger if large variations in daily C_i's concur with a $\overline{C}_{ann}$ in a non-linear part of the curve. However, an approximate correction, which

usually will be satisfactory, may be calculated if the standard deviations, *SD*, of the concentration levels are available or can be estimated. The correction factor, C_f, is given by:

$$C_f = \frac{p(\overline{C}_{ann} - SD) + p(\overline{C}_{ann} + SD)}{2\,p(\overline{C}_{ann})} \qquad (6)$$

The adjusted number of symptom-days, S_{adjust} , is then given by:

$$S_{adjust} = S_{average} \cdot C_f \qquad (7)$$

With the concentration levels and distributions usually found in ambient air, S_{adjust} will be very close to S_{sum}, and considering other uncertainties connected to e.g. exposure assessment this procedure should provide a satisfactory approximation.

Finally, the number of excess symptom-days, S_{excess}, is calculated by subtracting the baseline number of symptom-days (calculated from p_0) from S_{adjust}.

In table 1 exposure-response functions for the effects listed above are shown. The functions are based on a review of a substantial number of epidemiological studies and meta-analyses. In some cases the studies did not provide exposure-response functions, but e.g. indicated a statistically significant variation in prevalence associated with different concentration levels. These were then used to construct an OR function, assuming the data would fit an S-shaped curve (see Aunan, 1995). In 5.3 some examples of estimated health effects in Budapest are given.

Table 1. Exposure-response functions for health effects. OR or RR = $e^{\beta \cdot C}$, β: regression coefficient, C: concentration level (from Aunan, 1995).

Health end-point	**Relative risk model**	**β (95% CI)**	**C unit (averaging time)**	**p_0[1]**
Acute resp. symptoms:				

Acute URS, children	OR	0.0045 (0.0011 - 0.0080)	$\mu g/m^3$ TSP (daily)	0.064
Pseudocroup, children	RR	0.1244 (0.0638 - 0.1850)	log $\mu g/m^3$ TSP (daily)	0.0000122
Acute URS, adults	OR	0.0014 (0.0010 - 0.0017)	$\mu g/m^3$ NO_2 (daily)	0.046
Chronic resp. symptoms:				
Chronic RS, children	OR	0.0145 (0.0015 - 0.0285)	$\mu g/m^3$ TSP (annual)	0.03
or	OR	0.0055 (0.0026 - 0.0088)	$\mu g/m^3$ NO_2 (annual)	
Chronic RS, adults	OR	0.029 (0.015 - 0.054) [2]	$\mu g/m^3$ TSP (annual)	0.013
Asthma:				
Symptoms, children	OR	0.016 (0.002 - 0.030) [2]	$\mu g/m^3$ NO_2 (annual)	0.02
Symptom incidence, asthmatic adults	Absolute[3]	0.06 (0.001 - 0.119)	$\mu g/m^3$ $PM_{2.5}$ (daily)	
Chronic lung damage:				
Children	Absolute[4]	0.0028 (0.0015 - 0.0040) [2]	$\mu g/m^3$ O_3 (seasonal)	
Other effects:				
Eye irritations, adults	OR	0.0009 (0.0006 - 0.0011)	$\mu g/m^3$ NO_2 (daily)	0.032
Headache, adults	OR	0.0109 (0.0066 - 0.0152)	mg/m^3 CO (daily)	0.073
Mortality:				
Total mortality	RR	0.0007 (0.0003 - 0.0010) [2]	$\mu g/m^3$ TSP (daily)	0.0000251
or		0.0009 (0.0006 - 0.0012	$\mu g/m^3$ SO_2 (daily)	0.0000218
>65 y	RR	0.0009 (0.0004 - 0.0014) [2]	$\mu g/m^3$ TSP (daily)	0.0000160
<65 y	RR	0.0003 (0.0001 - 0.0004) [2]	$\mu g/m^3$ TSP (daily)	0.0000091
Infant mortality (0-1 y)	OR	0.014 (-0.001 - 0.029) [2]	$\mu g/m^3$ PM_{10} (annual)	4.7 per 1000 live births
or				
Infant resp. mortality	OR	0.02 (0.00 - 0.04)[5]	$\mu g/m^3$ SO_2 (annual)	0.2 per 1000 -"-
Lung cancer incidence	OR	0.006 (-0.003 - 0.014)	$\mu g/m^3$ TSP (long-term)	0.0003 per y

[1] Hypothetical zero-concentration prevalence, daily or annual depending on whether the function refers to daily or annual average concentration, see Section 4.
[2] Estimated uncertainty interval
[3] Daily incidence = $\beta \cdot \ln C$
[4] Prevalence = $\beta \cdot C$
[5] The function has a lower cut-off at 35 $\mu g/m^3$. When calculating the prevalence, subtract 35 $\mu g/m^3$ from the actual concentration level.

5. THE CASE OF HUNGARY

In Hungary really critical areas are less frequent compared to other Central and Eastern European countries, and only a few "hot spots" can be named, e.g. the Sajo valley, the Transdanubian industrial districts and Budapest. Besides, the difference between these areas and other parts of the country with more typical levels of pollution is less pronounced than in other countries in this part of Europe (REC, 1994). However, since the most polluted areas are also the most densely populated many people are exposed to adverse concentrations, 44% of the population according to the Hungarian Ministry of Environment and Regional Policy (1992).

5.1 Health effects on a national scale

According to the Hungarian Ministry for Environment and Regional Policy (1992) detrimental effects of air pollutants to health include mainly an increase in acute and chronic upper and lower respiratory diseases among children and adults (see also Tar and Tajthy, 1991).

Based on average local concentrations for the summer and winter season in 1992/-93 in 94 cities/towns in the 19 counties in Hungary plus Budapest we have made a rough estimate of the number of people living in areas where the air quality guidelines (6 months' mean) are violated, see table 2. The uncertainties in these estimates are, however, very large i.a. because the concentration levels are only measured at a few points in each city. The estimation is based on concentration data for the majority of the larger and medium sized cities in Hungary where ca. 5.4 million people live, i.e. 52% of the population. According to Hungarian Central Statistical Office (1993), 6.5 million people live in towns while 3.8 million people live in villages and rural areas. To estimate the total population exposed to values above the guidelines, we have added respectively 10% and 20% to the number achieved from these 94 cities, representing low and high estimates respectively. We are assuming that violations of 6 months air quality guidelines mainly occur in cities and towns. Especially for TSP this may imply that we are underestimating the number, as discussed below.

Table 2. Estimated number of people living in cities where air quality guidelines (6 months) were violated in 1992/-93 (million people). Total population in Hungary is 10.3 mill. (See the text concerning TSP).

	In cities with measuring stations	Total for Hungary	
		Low estimate	High estimate
TSP (> 100 $\mu g/m^3$ *)	3.72	4.1	4.5 (5.6)
NO_2 (> 50 $\mu g/m^3$)	2.36	2.6	2.8
SO_2 (> 40 $\mu g/m^3$)	0.97	1.1	1.2
Settling dust (> 16 g/m^2/30 days)	0.58	0.6	0.7

*TSP was measured only in 20 cities/towns, exceeding 100 $\mu g/m^3$ in all of them.

We have used the guidelines recommended by the Norwegian Pollution Control Authority (SFT, 1992), except for TSP and settling dust, where we have used the Hungarian guidelines (protection class I). The guidelines recommended by SFT lie between the two most strict classes in the Hungarian system.

TSP is the component causing most violations of the guidelines. Nine of the cities have seasonal averages around or above 300 $\mu g/m^3$, far above the levels associated with increased frequency of health effects as impaired lung performance, chronic pulmonary disease and exacerbation of bronchitis. The fact that particulates were measured only in some of the cities and that in all these cities at least one of the 6 months' averages was above 100 $\mu g/m^3$, indicates that the population exposed to TSP levels above the 50 $\mu g/m^3$ guideline may be significantly higher than the estimates made by the above method. We have therefore made an extra estimate (adding 50% to the calculated number) which is given in parenthesis in table 2.

Concerning NO_x only eight cities (including Budapest) have 6 months' mean concentrations above the guideline (50 $\mu g/m^3$) and the violations are not very pronounced. The SO_2 guideline (40 $\mu g/m^3$) is violated in 20 of the cities and with few exceptions these cities are situated in two counties, namely Komárom-Esztergom and Borsod-Abauj-Zemplén. The five cities where the concentrations are highest ($\geq$ 70 $\mu g/m^3$) are all situated in Komárom-Esztergom. Power plants are an important air pollution source in this area. The SO_2 guideline is not violated in the summer period in any city.

5.2 Air pollution in Budapest and violations of Air Quality Guidelines

In Budapest two monitoring systems are operating. One is discontinuous and records twice a week at 53 stations throughout all the 22 city districts (here called data set I). These data represent 4-6 hours' measurements and are usually recorded in the rush hours. The other system is continuous and records at 8 stations in inner Budapest (called data set II). Probably due to location and measuring techniques there are in some cases large discrepancies between the two sets, especially for SO_2.

The air quality in Budapest, where ca. 20% of the population live, has changed during the last decades. The most distinct changes in this period have been for *SO_2:* The 6 months' average in the heating season in 1984/-85 was 70 $\mu g/m^3$, while it had decreased to 27 $\mu g/m^3$ in 1993/-94 (data set I). The concentration levels are considerable lower in the non-heating season and the reductions in this season are not very pronounced. The main source of SO_2 pollution in Budapest is households (34%).

For *NO_2* the pattern seems to be opposite to that for SO_2 although the trend is not as clear: In 1980 the yearly average concentration was 43 $\mu g/m^3$, while it was about 52 $\mu g/m^3$ in 1992 (data set I). Transportation is the dominant source (45%).

There are no data for the changes in the concentration of *TSP* during the last decade, but the emissions from the households, which is the largest source of particulates (50% in 1992), were reduced by about 42% during the period 1985-1992. This must obviously have improved the air quality concerning TSP, even though increased emissions from the transportation sector may to some extent have counteracted this.

Based on data set I (average for 1989 and 1990) we have estimated the number of people living in districts where the air quality guideline for NO_2 is violated. 1.85 million people live in districts where the air quality guideline is violated once or more during the year. The guideline is quite frequently violated in some districts and about half a million of people live in districts where it is violated more than ca. 20% of the days. The maximum concentration (4-6 hours mean) recorded at any station was about 400 $\mu g/m^3$ both in 1989 and in 1990 (at heavily trafficked stations). The long-term average concentration in inner Budapest has been above the guideline (50 $\mu g/m^3$, SFT, 1992) the last years. The population exposure level distribution for the whole city is shown in fig. 2.

Data set II, which provides data for *TSP*, indicates that suspended particulates may be one of the main air pollution problems in Budapest. Short-term peak concentrations of TSP may represent a health risk in Budapest. The Hungarian guideline for 30 min. is 200 $\mu g/m^3$ (Protection class I). The 30 minutes monthly maxima are often about 400-600 $\mu g/m^3$, for some stations the value sometimes exceeds 1000 $\mu g/m^3$.

According to data set I, the 24 h *SO_2* guideline as given by Hungarian authorities, is violated, but not frequently, and only at very few stations. These data are, however, in strong disagreement with data set II, where the recorded concentrations are significantly higher. In data set II the yearly average in inner Budapest (in 1992 and 1993) was 46 $\mu g/m^3$ which is below the Hungarian guideline (Protection class I: 70 $\mu g/m^3$), but above the Norwegian guideline (40 $\mu g/m^3$).

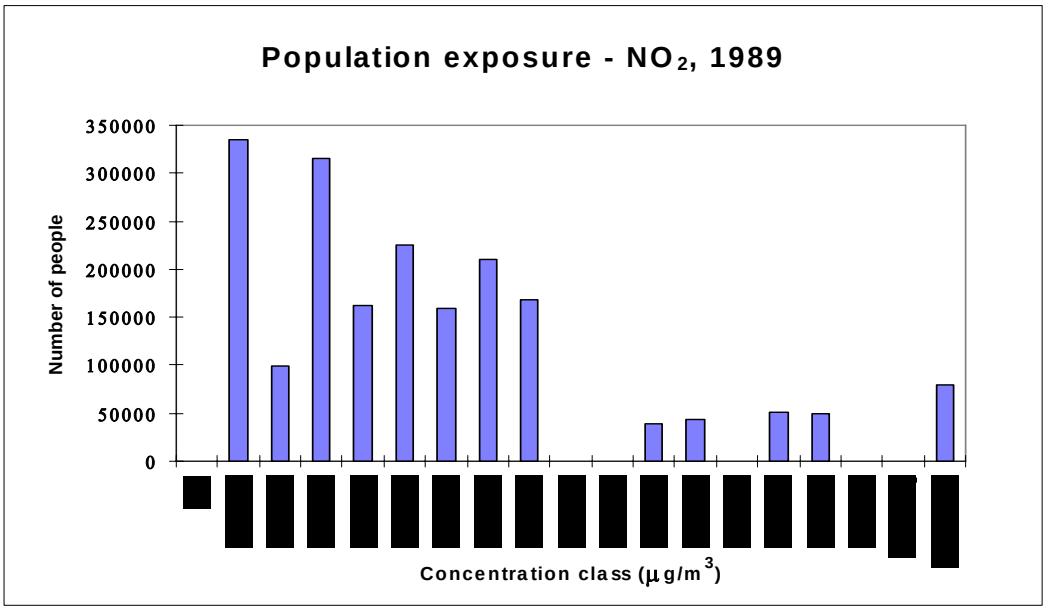

Fig. 2. Number of people living in districts where the average NO_2-concentration during rush hours is in the given intervals.

Concentration levels of CO and O_3 are given only in data set II. The *CO* concentration level is usually well below the guideline, especially in summer time, except for the stations close to the main roads and the data indicate that CO pollution does not represent a major health problem. However, it is likely that elevated CO concentrations to some extent may represent a health risk in the vicinity of the main roads.

There are frequent violations of the 1-h guideline for *O_3* (100 µg/m^3, SFT, 1992) in the summer period, but very few during the winter (O_3 is measured at only two stations). Generally, the ozone level is expected to be lower in the city, where there are high NO_x emissions, than in the air plume from the city, thus indicating that violations of the O_3 guidelines probably may be rather frequent in summer in the areas affected by this plume (of course, the population density is lower there).

5.3 Estimates of health effects in Budapest

Based on concentration data for 1992 and 1993 in inner Budapest, we have made estimates of the excess prevalence of some health effects due to air pollution.

The annual average of TSP in inner Budapest was 69 µg/m^3. Using the exposure-response function and p_0 for *chronic respiratory illness in children* given in table 1, an estimated excess prevalence of 0.047 (4.7% of the children) is obtained (95% CI, 0.002 - 0.19). The large uncertainties in this estimate may be illustrated by doing a stochastic (Monte Carlo) simulation, as shown in fig. 3. Random numbers are drawn from the uncertainty distribution of β, and a standard deviation of 0.01 is assumed for p_0. For instance, the simulation indicates a 63% probability that the excess prevalence is between 1% and 7% of the children, a 51% probability that it is between 3% and 9%, and a 14% probability that it is above 10%. Fig. 4 shows a corresponding simulation for chronic respiratory illness in adults. The excess prevalence of chronic illness in adults estimated from the exposure-response function given in

table 1 is 0.076 (95%CI, 0.014 - 0.414). (The standard deviation assumed for p_0 in the simulation is 0.005). The tails are very long, especially for the adult function, because the upper confidence limits for the coefficients result in the functions being strongly non-linear in the prevailing concentration range.

A preliminary risk assessment based on expert judgements of prevalence of mild or moderate lung lesion in children (assumed to be an appropriate indicator of potential chronic lung injury) indicates that the prevalence associated with a seasonal average of 47 $\mu g/m^3$ (average level in inner Budapest in summer) is 0.13 (95% CI, 0.07 - 0.19) (McKee and Rodriguez, 1993, see also tab.1).

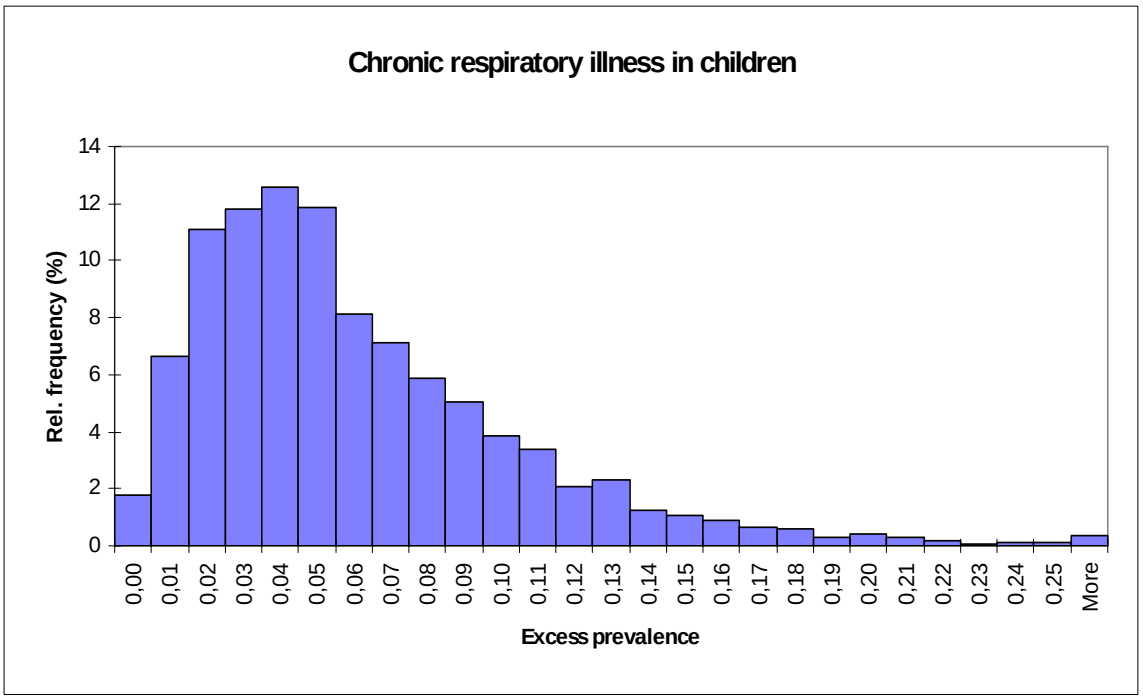

Fig. 3. Frequency distribution of estimates of excess prevalence of chronic respiratory symptoms in children in inner Budapest. (A column represents the interval from the number under the column to the number under the previous column inclusive the upper).

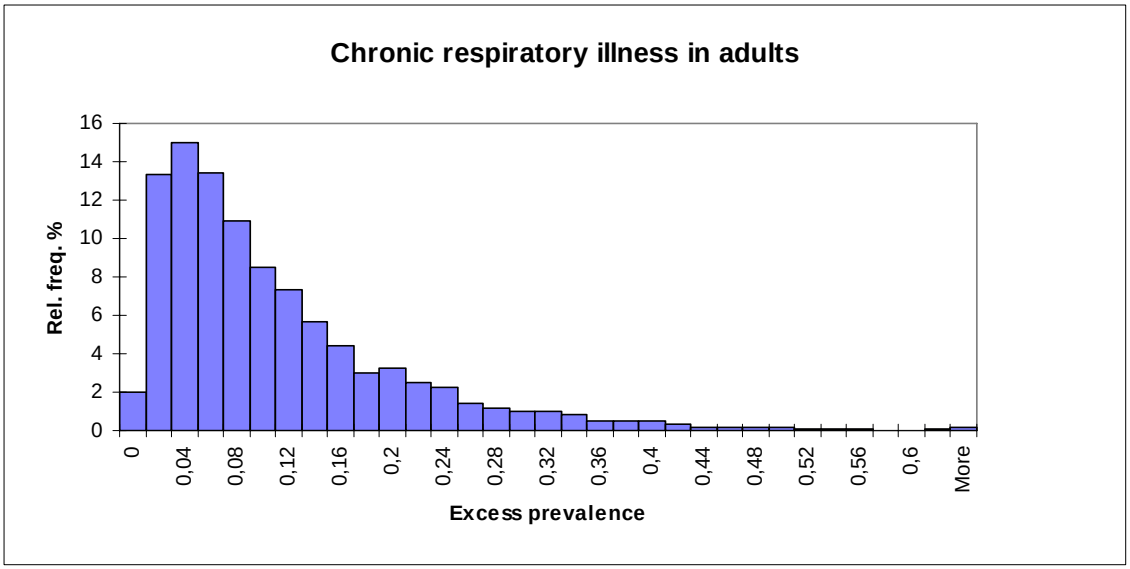

Fig. 4. Frequency distribution of estimates of excess prevalence of chronic respiratory symptoms in adults in inner Budapest. (A column represents the interval from the number under the column to the number under the previous column inclusive the upper).

To get an illustration of the magnitude of excess prevalence of acute respiratory symptoms in children, we have used the daily concentration levels (in 1993) at a typical inner Budapest measuring station. The estimated excess symptom-days per child per year due to air pollution then is 8.0. Using the annual average concentration and the adjustment procedure as described in Section 4, we also get 8.0, indicating that the approximation is very good. Figure 5 shows the result of a stochastic simulation. For instance there is a 54% probability that the number of annual excess symptom-days per child is between 4 and 10.

Figure 6 shows the result of a corresponding simulation for respiratory symptoms in adults. Using the actual daily concentrations or the yearly average (with the adjustment procedure) both give an estimate of 1.3 excess symptom-days per adult per year.

There are large uncertainties about the contribution of outdoor air pollution to asthma symptoms. However, there are some indications that the NO_2-concentration, maybe as an indicator of traffic pollution, can be used to assess the prevalence of asthma symptoms in children (see Aunan, 1995, and tab. 1). We have used the annual NO_2-concentration in inner Budapest (55 $\mu g/m^3$) to make an estimate of excess prevalence in this area. The annual excess prevalence is 0.03 (3% of the children) (uncertainty interval 0.001 - 0.109). Fig. 7 shows a stochastic simulation, where random numbers are drawn from the uncertainty distribution of β, and a standard deviation of 0.01 is assumed for p_0. For instance, there is a 69% probability that the excess prevalence is between 1% and 6%.

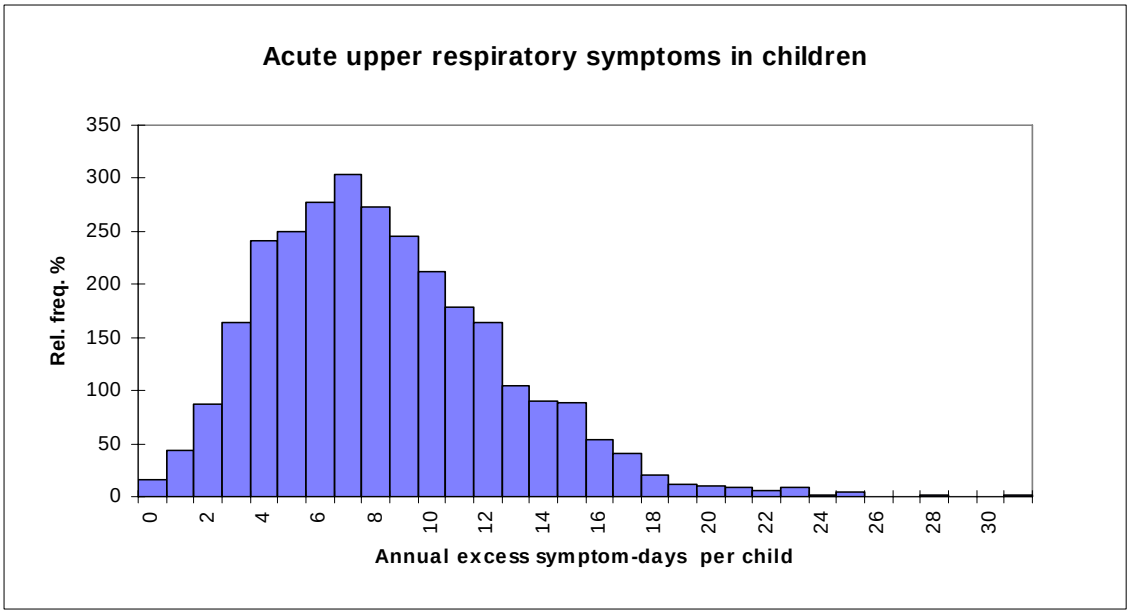

Fig. 5. Frequency distribution of estimates of excess annual number of symptom-days per child in inner Budapest.

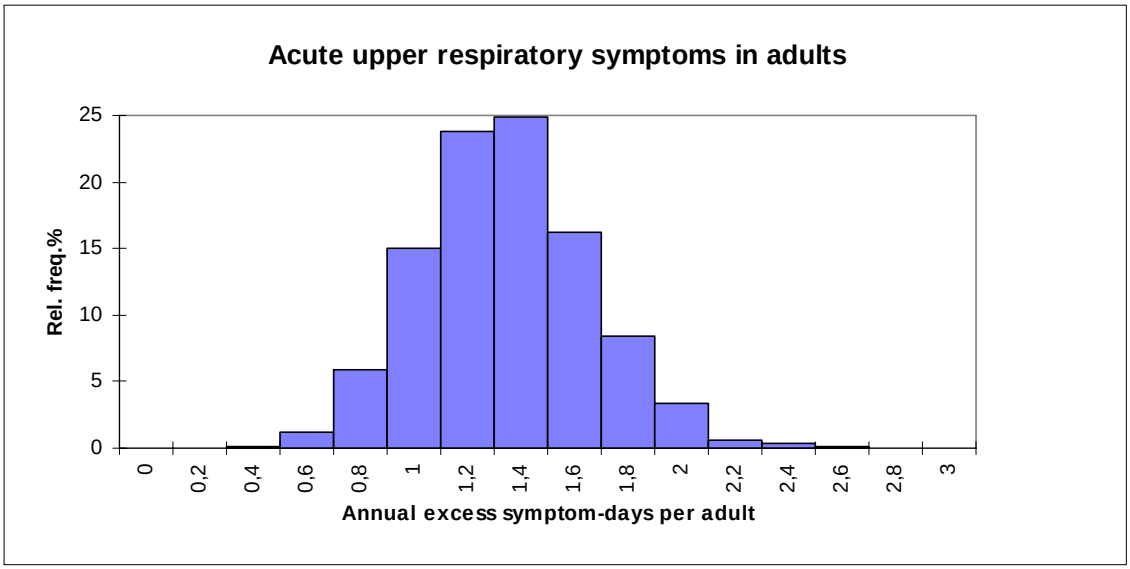

Fig.6. Frequency distribution of estimates of excess annual number of symptom-days per adult in inner Budapest.

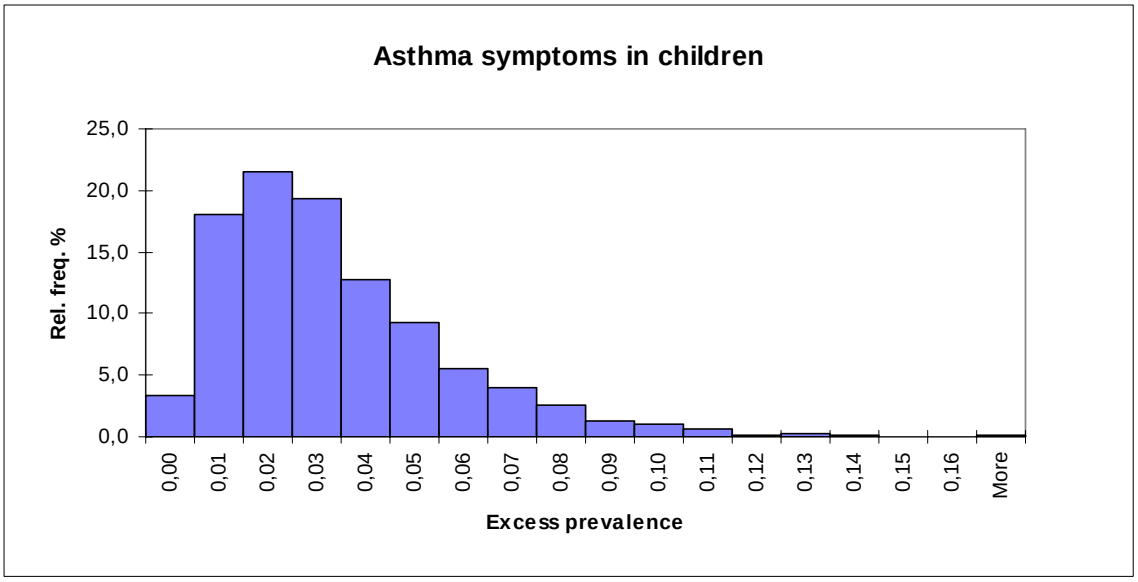

Fig.7. Frequency distribution of estimates of excess prevalence of asthma symptoms in children in inner Budapest.

The basis for establishing exposure-response function for air pollution and mortality is more firm than for numerous other air pollution health effects. Quantitatively similar estimates for the effect of particles on mortality have been reported over a large range of concentrations, in a variety of communities, with varying mixtures of pollutants and different climatology.

Using the same typical inner Budapest measuring station (annual average of TSP is 67 $\mu g/m^3$) we have estimated the excess number of deaths per year per 100.000 (all ages) in persons elder than 65 y and younger than 65 y, respectively. The estimates obtained by using the day to day concentrations and by using the annual average concentration and the adjustment procedure as described in Section 4 are the same. For the elder group the estimated annual excess number of deaths is 37 (95% CI, 8 - 86). A stochastic simulation is shown in Fig. 8 (we have assumed a SD in p_0 of $8 \cdot 10^{-6}$). For instance, there is a 64% probability that the number is between 20 and 60.

The vulnerability in persons <65 y of age is significantly lower than in the elder group. The estimated annual excess deaths per 100.000 in the inner Budapest area is ca. 7 (95% CI, 2 - 10). The uncertainties in this estimate are illustrated in fig. 9.

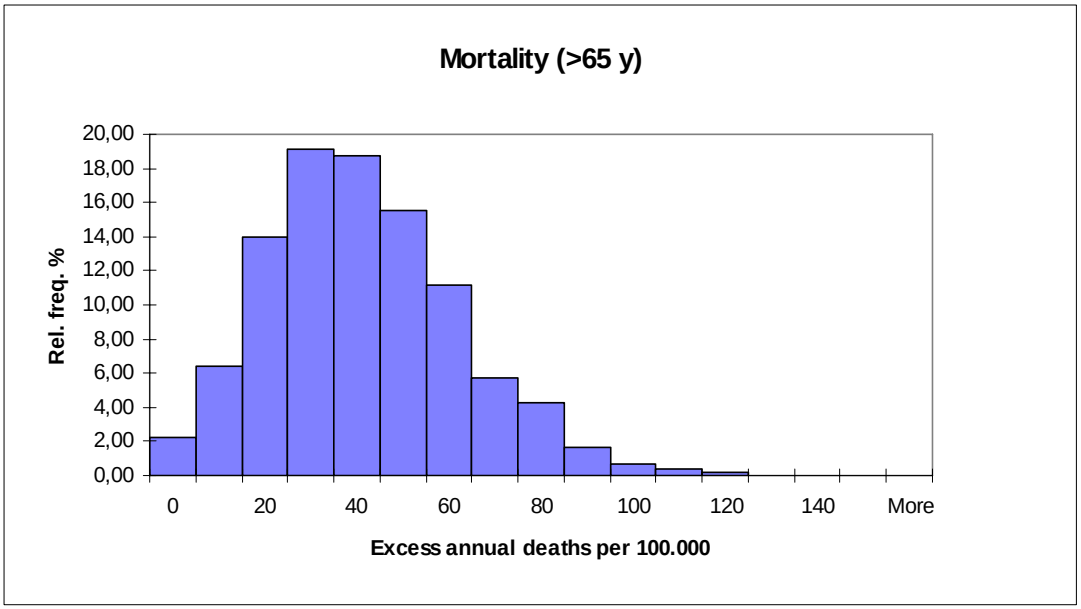

Fig. 8. Frequency distribution of estimates of excess annual deaths of persons > 65 y of age in inner Budapest, per 100.000 (total population - all ages).

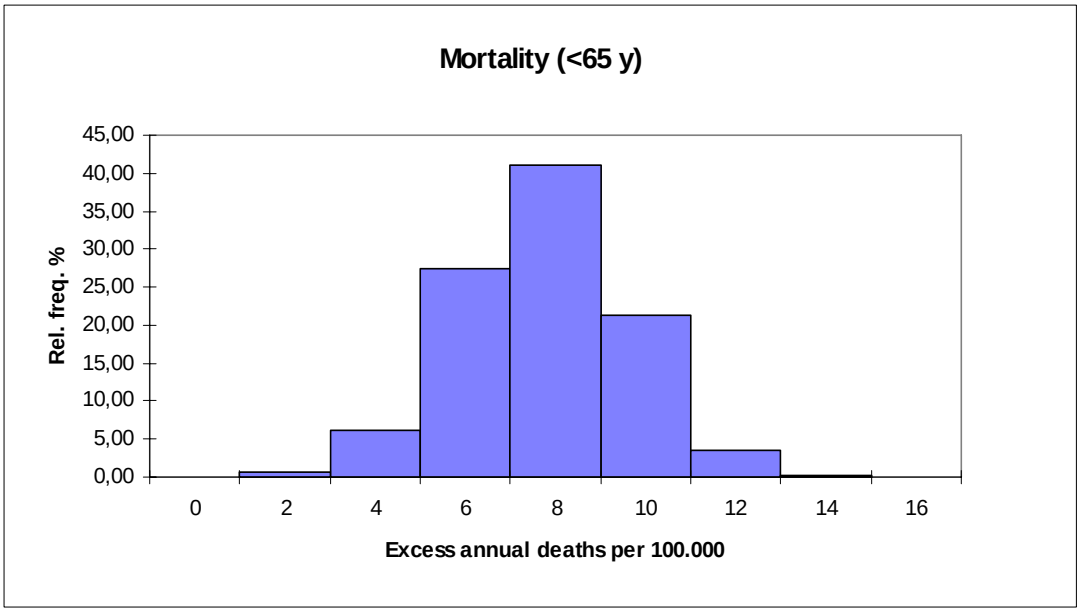

Fig.9. Frequency distribution of estimates of excess annual deaths of persons < 65 y of age in inner Budapest, per 100.000 (total population - all ages).

We may also go the other way around to calculate excess mortality, by looking at the actual mortality rates in Budapest. The total daily mortality rate (violent deaths excluded) in 1992 was $3.8 \cdot 10^{-5}$. Using the regression coefficient estimated for all ages (see tab. 1), the estimated number of excess deaths is 64 (95% CI, 28 - 90), as compared to 44 (95% CI, 11 - 89) in the above estimations. (The uncertainty interval is less in the first estimate because no uncertainty is assumed in p_1).

There are rather few studies on exposure-response relations for *total infant mortality*. However, Bobak and Leon (1992) in a study in the Czech Republic used logistic regression to indicate the increase in risk ratio associated with certain intervals of the annual concentration of PM_{10}, SO_2 and NO_2. The effect of a single air pollutant component was stronger for PM_{10} than for SO_2 and NO_2. We have used the function derived from this study (given in table 1) to estimate an excess infant mortality in inner Budapest of 3.2 per 1000 live births (95% CI, -0.2 - 9.1). Using the actual infant mortality in Hungary the corresponding number is 4.8 per 1000 live births (96% CI, -0.4 - 7.9).

In Budapest there are yearly 95 new cases of lung cancer per 100.000 inhabitants (1992). The average figure for Hungary as a whole is 70 per 100.000. There are of course very large problems connected to the exposure-response approach for lung cancer, i.a. because it takes long time to develop cancer. Using the exposure-response function given in tab.1 the estimated number of excess annual cases due to air pollution in inner Budapest is 32 cases per 100.000 (95% CI, -22 - 59). Fig. 10 shows a stochastic simulation and illustrates the large uncertainties in this estimate.

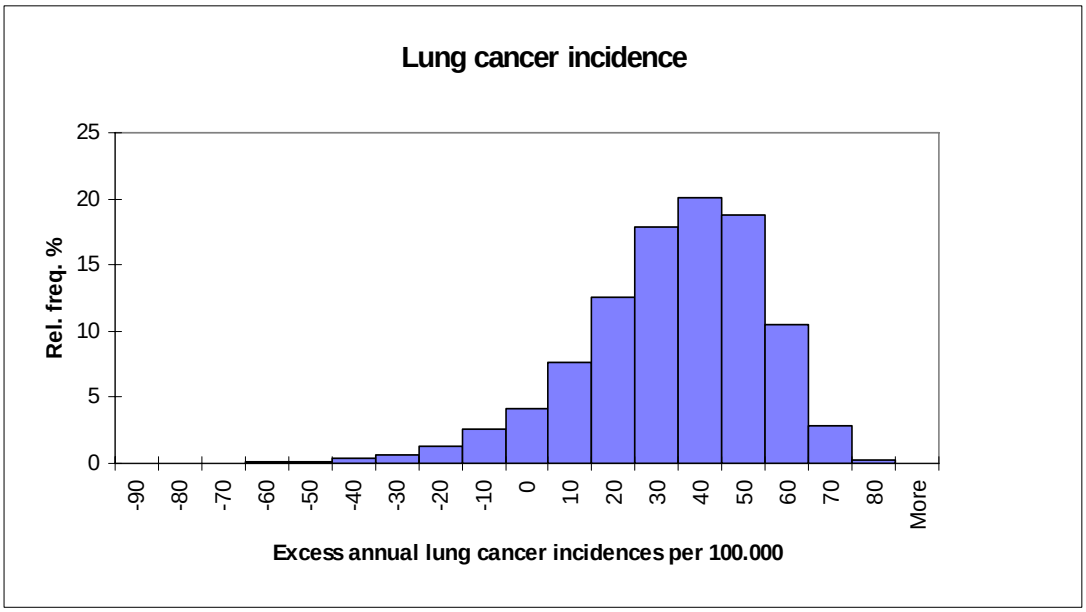

Fig.10. Frequency distribution of estimates of excess annual lung cancer incidences per 100.000 in inner Budapest. (A column represents the interval from the number under the column to the number under the previous column inclusive the upper).

5.4 Mortality in Chongqing

As a comparison to the estimates made for Budapest, we have also made some estimates for Chongqing, one of the most polluted cities in China. Excess deaths per year are estimated using the SO_2-function given in table 1. The mean SO_2-concentration the last years was 330 $\mu g/m^3$, and we have assumed that the SD in daily concentration is ca. 55% of the mean (as for the Budapest-station). The estimated excess annual deaths per 100.000 is 289 (95% CI, 134 - 475). Fig. 11 shows a stochastic simulation based on the data (the assumed SD in p_0 is $5 \cdot 10^{-6}$).

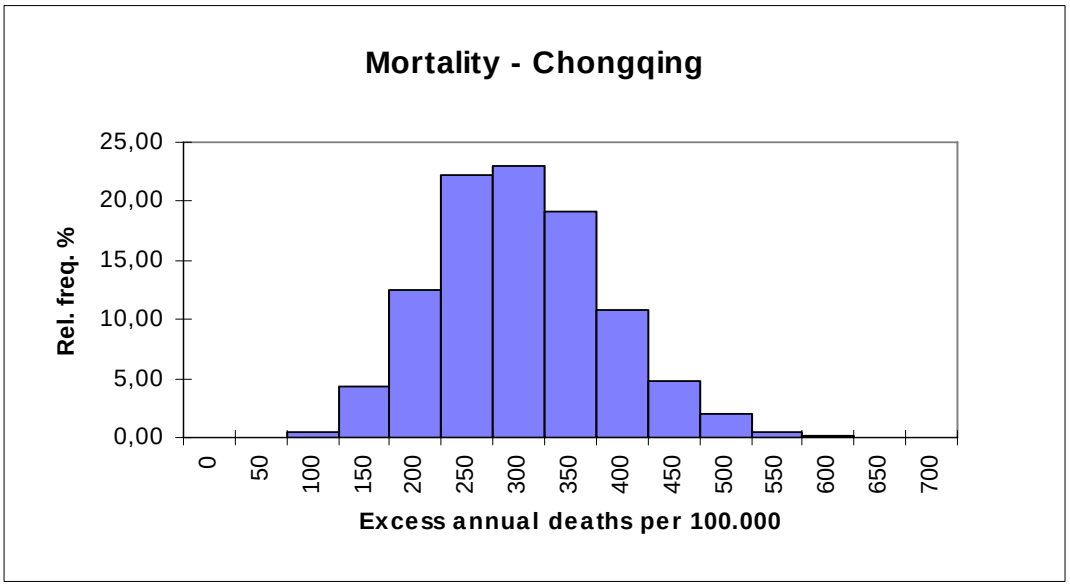

Fig.11. Frequency distribution of estimates of excess annual deaths per 100.000 (total population - all ages) in Chongqing, China. (A column represents the interval from the number under the column to the number under the previous column inclusive the upper).

There is no epidemiological basis for a function for SO_2 and total infant mortality, and since we do not have data for particles in Chongqing, an estimate of total infant mortality can not be made. However, the study by Bobak and Leon (1992) allows a rough estimate of the relation between SO_2 and *infant respiratory mortality* (the function applies to situations where the annual SO_2-concentration is above ca. 35 $\mu g/m^3$, below this level a positive association was not found). The function is very uncertain and it seems likely that it gives an overestimate at high concentration levels. The estimated excess infant respiratory mortality in Chongqing is roughly 75 per 1000 live births. This is in the range of the mean total infant mortality in countries like for instance Indonesia and Namibia. The mean total infant mortality in China today is 38 per 1000 live births (in 1970 it was 69) (World Bank, 1993).

5.5 Other effects of air pollution in Hungary.

Optimal measures against air pollutants can only be found by considering all important effects. In addition to health we have considered effects on vegetation and materials. The mean daily maximum concentration of ozone in the growing season (April - September) has been estimated to 120 - 140 $\mu g/m^3$ (Simpson, 1993), which indicates a 5 - 10 % loss in cereal crops (see Seip et al., 1995 for further discussion).

Forests in large parts of Europe are probably adversely affected by air pollution although the understanding of the causes and mechanisms is poor except in the most polluted areas where direct effects are plausible. The damage in Hungary is much less than in Poland, and the Czech and Slovak republics and has shown no significant trend since 1990 (UN/ECE/CEC, 1995). We have therefore not tried to quantify the forest damage due to air pollution in Hungary.

The costs of damage to buildings in Europe due to air pollution are huge. The savings in Europe if the Second Sulphur Protocol was fully implemented within UN-ECE have been estimated to US$ 9 500 millions/year by Kucera and Fritz (1995). Estimates of the savings in corrosion costs if average SO_2 levels were reduced to less than 20 $\mu g/m^3$, are about US$ 110/(inhab. year) for Prague and US$ 20/(inhab. year) for Stockholm (Kucera and Fritz, 1995). A preliminary estimate for Budapest indicates about US$ 50/(inhab. year), which means a total yearly saving of 100 millions US$ (Kruse, 1995).

5.6 Final remarks and further work

The demand for economic growth in Hungary and other post-communist countries may sharpen the conflicts between different goals, thus cost-effectiveness in environmental management is essential. The main goal with our project is to give advice to decision makers concerning measures against pollution. So far it has only been possible to make some general and tentative recommendations (Seip et al., 1995): Great concern should be directed towards problems occurring from expanding sectors such as the transport sector, generation of electricity and private consumption. Care must be taken not to reduce the importance of public transport. Better maintenance of buses and cars, in particular diesel vehicles, is likely to have a high benefit/cost ratio. Energy saving measures are important as the efficiency gap in Hungary has been estimated to 15-30%.

Further work in this study will include monetization of benefits from reductions of emissions, description of abatement measures and their costs, and estimations of net benefits for these measures.

CONCLUSIONS

Quantification of health effects is clearly an essential part of an integrated assessment. Recent research has greatly improved our understanding of the relationships between air pollution and health effects. However, the application of exposure-response functions is often hampered by large uncertainties in the concentration data, especially concerning particles. The need for high quality concentration data should be emphasised. This can only be obtained by reliable and comparable methods for sampling and analyses.

In spite of large uncertainties in every step in the analysis, an integrated assessment of costs and benefits of different abatement measures is valuable. It clarifies the main objectives of an abatement policy and explicitly describes the adverse impacts of different activities and their relative importance. This gives excellent opportunities for communication with those affected by the decisions and also reveals the areas where the need for more insight is strongest. Collaboration between different disciplines is necessary to achieve results that can be helpful to decision-makers.

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