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**Economic Damage of Air
Pollution**

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Economic Damage of Air Pollution

Abstract:
The paper considers available information on physical dose-response functions related to air pollution and damage of human health and important materials. The dose-response functions are translated into a form suitable for implementation in multi-sectoral computable general equilibrium models (CGEs) and simulations are carried out illustrating the direct and indirect (allocation) costs of environmental damage to human health and materials on economic growth in Norway. The model is further supplemented with a module relating the volume of road traffic to traffic accidents and their consequences on labour productivity and public health expenditures.

Keywords: Air pollution, externalities, valuation.

JEL classification: Q25, H51, D62.

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1 Introduction

Statistics Norway has previously made some tentative assessments of the cost of air pollution associated with damage to forests, fresh water lakes, human health and some important materials. The assessments were mainly based on opinions obtained from expert panels together with empirical studies carried out in the U.S. in the 1970's, see Glomsrød and Rosland, 1988 [19], Brendemoen et al., 1992 [7], Alfsen et al., 1992 [3], Alfsen, 1993 [2], and Alfsen and Glomsrød, 1993 [5]. These assessments were linked to economic and emission forecasts generated by integrated economic, energy and emissions models, in order to study future development in air pollution damage and the likely impacts of various policies (e.g. carbon taxes) on the level of the damage.

Lately a new consensus have emerged on some of the effects of air pollution on human health, and further studies have been carried out to measure the geographical distribution of building materials and the pollution induced corrosion of these materials. Based on this new knowledge it is now possible to take some further steps in assessing the economic costs of air pollution. The main aim of this paper is to outline what we currently are doing in Statistics Norway on the topic of health damage and damage to materials from excessive air pollution. This work is documented in detail (and - unfortunately - so far only in Norwegian) in Rosendahl (1996) [45], Glomsrød, Rosendahl and Hansen (1996) [18] and Glomsrød et al. (1996) [20]. The basis for the work is national and international studies on the physical effects of air pollution. We then convert these estimates into forms suitable for inclusion in economy wide models, in order to capture not only the direct cost of air pollution, but also the allocation or general equilibrium effects of the damage. Economic activity in turn determines emission to air and hence pollution concentration. The effects of pollution then closes the circle, see figure 1.
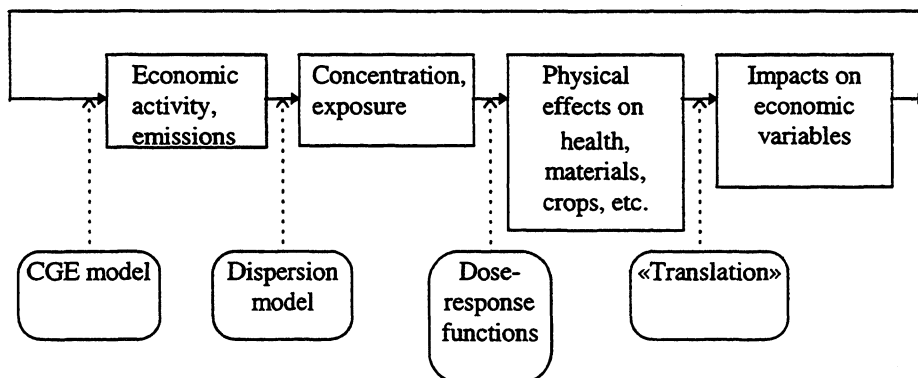

Figure 1: Framework for environmental damage assessment

Only the "pure" economic costs of air pollution, i.e. the effect of air pollution on labour supply and productivity, on public health expenditures and on maintenance and investments in capital, are considered. In addition we have included a module calculating the effects of road traffic on traffic accidents and their impact on labour productivity and public health expenditures (Glomsrød, Nesbakken and Aaserud, 1996 [17]). What is left out by this approach are the welfare effects (not captured by the market economy) of health damage due to air pollution and traffic accidents. Combined with the fact that we only cover a rather small fraction of the many economic effects of air pollution, it is clear that the numbers we arrive at represents a considerable underestimate of the total cost of air pollution and the use of fossil fuels. Nevertheless, the numbers are of interest since they provide a relatively secure or conservative lower limit to which policy makers and others can add their own judgement of the importance of welfare effects and other types of damage not currently well documented in the literature.

The paper is organised as follows (see table 1 and figure 1). In the first part, we consider the effects of air pollution on human health. We start by describing the calculation of the relevant exposure of people to concentration of PM_{10}, NO_2, O_3, and SO_2. We then go on to discuss the effects of this exposure on mortality and morbidity. These physical effects are then translated into economic variables, like loss of working hours and public health expenditures. These relations are

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implemented in a multi-sectoral general equilibrium model of the Norwegian economy in order to assess also the allocation costs of the induced health effects.

In the second part we follow a similar path for material damage of air pollution, now mainly related to the exposure of various materials to SO_2, but also to some extent covering the effects of O_3. The third part briefly describes the traffic accident module, while part four reports on some simulations carried out with an integrated economy-energy-environment model with and without the feedbacks described above.

	Health	Materials	Scenarios
Emissions	PM_{10}, NO_2, O_3, SO_2	SO_2, O_3	
Exposure	Population weigthed	Material density weighted	
Dose-response	Mortality: -Short term	Material loss	Physical
	-Long term	$\downarrow$	scenario
	Morbidity: -RADs (MRADs)	Economic life time	
	-Resp. symptoms		
	-COPD		
Economic	Labour supply	Maintenance or	
"damage"	Public expenditures	replacement cost	
	("Welfare")		
Effects	Macro model implementation→		Economic
	cost of factor use, productivity→		scenario
	emissions→ economic effects		

<div align="center">Table 1: Framework for assessing environmental damage of air pollution</div>

Part I

Health effects

2   Emissions

The polluting compounds considered for their effects on human health in this paper comprise particulate matter (PM_{10}), nitrogen dioxide (NO_2), ozone (O_3), and sulphur dioxide (SO_2)[1].

3   Exposure to air pollutants

There is disagreement and uncertainty about how to best measure the exposure of the population to air pollution. Some argue for measuring mean concentration levels over certain periods, while other measures the number of hours the concentration level is above a critical level. In most studies of dose-response functions, mean concentration levels over one or a few days are employed in studies of acute health effects, while chronic effects are related to the mean concentration level over semiannual or longer periods. In applied studies these measures are then converted to annual mean concentration levels. This is possible given that the dose-response functions are linear without any threshold levels. Most of the studies find this to be a reasonable approximation for the effects and pollutants covered in this paper, and we will follow this custom here. There are, however, evidence in some cases (e.g. in the case of NO_2 pollution) that shorter episodes with high concentration levels are of greater importance. In that case, our approach will underestimate the effects of these episode related events.

Oslo is the capital and largest city in Norway, and population exposure to air pollutants is treated in a detailed manner for this region. A dispersion model (EPISODE developed by The Norwegian Institute for Air Research – NILU) is used together with information on population density to derive population weighted annual mean concentration levels for particulate matter in the form of PM_{10}, i.e. particles with diameter less than 10 μm, and nitrogen dioxide (NO_2)

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[1]The presentation relies heavily on Rosendahl (1996) [45] where more details and discussions of the various assumptions used can be found.

(Walker, 1996 [55]). For other large population centres in Norway we have, in consultation with NILU and based on concentration measurements, extended the procedure to obtain the functional relationships reported in table 2 and 3. The urban areas covered represent some 30 per cent of the total population of Norway.

City	Annual mean concentration of PM_{10} ($\frac{\mu g}{m^3}$)	Level in 1992 ($\frac{\mu g}{m^3}$)
Oslo	$4.2 \cdot I_T + 5.3 \cdot I_F + 3.7 \cdot I_{KM} + 4.0 \cdot I_R + 6$	23.2
Bergen	$0.7 \cdot I_T + 7.0 \cdot I_F + 0.6 \cdot I_{KM} + 2.7 \cdot I_R + 4$	15
Trondheim	$1.1 \cdot I_T + 7.9 \cdot I_F + 1.0 \cdot I_{KM} + 2.0 \cdot I_R + 3$	15
Stavanger	$1.1 \cdot I_T + 5.2 \cdot I_F + 1.0 \cdot I_{KM} + 2.7 \cdot I_R + 5$	15
Drammen	$1.9 \cdot I_T + 5.4 \cdot I_F + 1.7 \cdot I_{KM} + 5.0 \cdot I_R + 6$	20
Skien	$1.3 \cdot I_T + 7.5 \cdot I_F + 1.2 \cdot I_{KM} + 4.0 \cdot I_R + 6$	20
Porsgrunn	$1.5 \cdot I_T + 7.2 \cdot I_F + 1.3 \cdot I_{KM} + 4.0 \cdot I_R + 6$	20
Bærum	$1.0 \cdot I_T + 2.1 \cdot I_F + 0.9 \cdot I_{KM} + 5.0 \cdot I_R + 6$	15

Table 2: Person weighted mean annual concentration of PM_{10} in 8 Norwegian cities.

City	Annual mean concentration of NO_2 ($\frac{\mu g}{m^3}$)	Level in 1992 ($\frac{\mu g}{m^3}$)
Oslo	$14.6 \cdot I_T + 2.1 \cdot I_F + 16.1 \cdot I_B + 13.2$	46
Bergen	$11.8 \cdot I_T + 2.1 \cdot I_F + 16.1 \cdot I_B + 10.0$	40
Trondheim	$10.8 \cdot I_T + 3.2 \cdot I_F + 13.0 \cdot I_B + 8.0$	35
Stavanger	$6.9 \cdot I_T + 1.0 \cdot I_F + 16.1 \cdot I_B + 11.0$	35
Drammen	$8.3 \cdot I_T + 0.5 \cdot I_F + 18.0 \cdot I_B + 13.2$	40
Skien	$9.8 \cdot I_T + 0.9 \cdot I_F + 16.1 \cdot I_B + 13.2$	40
Porsgrunn	$2.9 \cdot I_T + 7.8 \cdot I_F + 16.1 \cdot I_B + 13.2$	40
Bærum	$3.4 \cdot I_T + 0.4 \cdot I_F + 18.0 \cdot I_B + 13.2$	35

Table 3: Person weighted mean annual concentration of NO_2 in 8 Norwegian cities.

In the tables, I_T is an index of emissions from traffic, I_F is an index of other local emissions, I_{KM} is an index of the number of km driven (of relevance because of the use of studded tires in Norway), I_R is an index of Norwegian regional emissions (e.g. outside Oslo), while I_B is an index of the background concentration determined by foreign emissions. All indices take on the value of 1 in the year 1992.

4 Dose-response functions: effects on health

Several human health related effects of air pollutants are considered in this paper: From mortality, through restricted activity days (RADs) and days with respiratory symptoms (RS) to chronic obstructive pulmonary diseases (COPD). The occurrence of these effects will sometimes lead to hospital admittance with associated costs, and often to a reduction in the productivity of the labour force (depending among other things on the age structure of those affected). The general structure of the dose-response functions are as follows:

Mortality:

$$\frac{\Delta D}{D} = \delta \cdot \Delta C. \tag{1}$$

Chronic obstructive pulmonary diseases:

$$\frac{\Delta COPD}{COPD} = \nu \cdot \Delta C. \tag{2}$$

Effect on the number of days with respiratory symptoms and/or restricted activity days (RADs) per year:

$$\Delta LS = \lambda_S \cdot \Delta C \cdot P \tag{3}$$

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Effect on number of hospital admissions per year:

$$\Delta LSH = \eta_H \cdot \Delta C \cdot P \qquad (4)$$

Here D is the number of deaths per year and C designates the concentration of the relevant pollutants (measured in $\mu g/m^3$) averaged over a relevant period. We will report both short term (acute) and long term effects. Of these, the short term effect is the better documented. $COPD$ is the number of new diagnoses of chronic obstructive pulmoray diseases. LS is the number of days per year with respiratory symptoms or number of restricted activity days, and P is the number of people exposed to the pollutants. LSH is the number of hospital admissions with respiratory symptoms per year.

The parameters in the dose-response functions are taken from a number of studies. The most central are Ostro (1987) [32], Ostro (1993) [34], Krupnick et al. (1990) [27], Pope (1991) [39] and Abbey et al. (1993) [1]. ORNL/RFF (1994) [30], Rowe et al. (1995) [47] and the EC (1994) [13] studies are also mainly based on these reports. Below, we will report on the dose-response functions we have chosen and provide some brief remarks related to the specific parameter values. For further discussions and arguments for the particular choices, see Rosendahl (1996) [45]. Several of the parameter values are highly uncertain. Therefore only a restricted subset of the relations reported in this part of the paper will actually be implemented in the macroeconomic model. We will return to this in part four of the paper.

4.1 Particulate matter

Particulate matter comprise a large family of particles of varying sizes and chemical compositions. Studies of the health effects of air born particles have chosen to relate the observed effects to different measures of exposure, like for instance PM_{10} and $PM_{2.5}$ (concentration of particles with diameters smaller than 10 and 2.5 μm, respectively), TSP (total suspended particles - covering all size fractions), sulfate and soot (the latter defined by the way it is measured - blackening of filters). As already mentioned, the period of the measurements also varies, from a few hours to several years. In the literature it is common practice to convert all of these measures to a standardized variable; annual mean concentration of PM_{10} measured in $\frac{\mu g}{m^3}$. This is of course a simplification which introduce some additional uncertainty into the results, since the time profile and composition of particle pollution varies somewhat from place to place.

Table 4 provides the parameter values we employ in this paper together with references. The uncertainty ranges (given in parenthesis) must be understood as very tentative. Usually they refer to ±one standard deviation in the relevant studies.

Health effects of PM_{10}	Coefficient (per annual mean concentration of $PM_{10}(\frac{\mu g}{m^3})$)	Sources
Acute risk of death (short term exposure)	$0.96 \cdot 10^{-3}$ $(0.63\text{-}1.30) \cdot 10^{-3}$	Ostro (1993) [34]
Risk of death (long term exposure)	$6.5 \cdot 10^{-3}$ $(4.6\text{-}9.1) \cdot 10^{-3}$	WHO (1995) [56]
Annual number of restricted activity days (RADs) per person	$57.5 \cdot 10^{-3}$ $(36\text{-}90) \cdot 10^{-3}$	Ostro (1987) [32] ORNL/RFF (1994) [30]
Annual number of days with respiratory symptoms per person	0.18 $(0.09\text{-}0.27)$	Krupnick et al. (1990) [27] Ostro (1994) [35]
Annual hospital admittances with respiratory symptoms per person	$36 \cdot 10^{-6}$ $(12\text{-}102) \cdot 10^{-6}$	Various studies, see Rosendahl (1996) [45]
Risk for chronic obstructive pulmonary diseases (COPD)	$11 \cdot 10^{-3}$ $(5\text{-}17) \cdot 10^{-3}$	Abbey et al. (1993) Rowe et al. (1995)

Table 4: Dose-response functions for particulate matter (PM_{10}).

Of all the health effects of air pollution, *acute increase in mortality* from particulate matter is probably one of the best documented. Furthermore, there now seems to have emerged a consensus on the quantitative effect on mortality of short term exposure to particulate matter. There is evidence for claiming that it is the *size* of the particles, and not for instance the chemical composition, that is usually of most importance for acute changes in mortality (Pope *et al.*, 1992 [40], Fairley, 1991 [14], Ostro, 1995 [36]). The people most affected by short term increases in exposure to particle pollution are elderly people (i.e. above approximately 65 years old) (see e.g. Schwartz and Dockery, 1992 [49]).

Far fewer studies have focused on the *long term effects on mortality* of exposure to particulate matter. The parameter in table 4 is taken from WHO (1995) [56] who refer to two cohort studies (Dockery *et al.*, 1993 [10], and Pope *et al.*, 1995 [41]). These studies find that the long term effect of exposure to particulate matter is from 5 to 10 times larger than the short term effects. The results are, however, more uncertain than the corresponding results for the short term effects.

It is important to interpret the mortality coefficients in table 4 correctly. The coefficients are related to changes in *risk* of death within for instance a period of one year due to changes in the concentration of particulate matter. The actual *number of deaths* provoked by a change in the concentration level will thus also depend on the time profile of mortality in the population.

The effects of exposure to particulate matter on the number of *restricted activity days (RADs)* have been studied by Ostro (Ostro 1983 [31], 1987 [32], 1990 [33], Ostro and Rothschild, 1989 [37]). According to ORNL/RFF (1994) [30], 62 per cent of the RADs represent lost working days, while the remaining are designated as minor restricted activity days (MRADs). These are assumed here to reduce the labour productivity by 10 per cent.

Krupnick *et al.* (1990) [27] have carried out a study of the number of days with *respiratory symptoms* as a function of exposure to particulate matter. The result, as reported by Ostro (1994) [35] and Rowe *et al.* (1995) [47], is given in table 4, however corrected for the number of RADs in order to avoid double counting.

The dose-response function for *number of hospital admittances* as a function of exposure to particulate matter reported in table 4, is based on a number of studies. These are discussed in more detail in Rosendahl (1996) [45]. The final outcome is a dose-response function 3 times larger than the function reported in Pope *et al.* (1991) [39].

Abbey *et al.* (1993) [1] reports significant correlations between the long term exposure to particulate matter and the occurrence in the population of chronic bronchitis, asthma or emphysema, which are the three most common forms of chronic obstructive pulmonary diseases (COPD). The result, transformed to relations between PM_{10} concentration and COPD, is given in table 4. These results are further corroborated by other studies (see Rosendahl, 1996 [45]). We note that in principle there may be some overlap between short term and long term morbidity dose-response functions. However, in the case of COPD, the main and strongly dominant effect is due to the long term nature of the disease. Thus, the double counting of the acute effect represents only a very minor part of the total effect.

4.2 Nitrogen dioxide (NO_2)

In contrast to the case of particulate matter, relatively few epidemiological studies have found any significant association between (outdoor) NO_2 exposure and health effects. However, clinical trials and studies of indoor exposure to NO_2 clearly indicates that such exposure is harmful. It is possible, however, that the main damage by NO_2 is inflicted by short term exposure to high concentration levels. This could explain why it may be difficult to obtain significant results from epidemiological studies which mainly relies on longer term mean concentration levels as explanatory pollution related variable. In table 5 we list some dose-response functions found in the literature.

Of particular interest is the study of hospital admittances due to asthma which was carried out in Finland, a country with a similar climate to Norway. The results of this study is further confirmed by another Finnish study (Rossi *et al.*, 1993 [46]).

4.3 Sulphur dioxide (SO_2)

Most studies of the health effects of SO_2 have been carried out in areas with considerable higher concentration levels than found in Norwegian cities, which have typical concentration levels of

Health effects of NO$_2$	Coefficient (per annual mean concentration of NO$_2(\frac{\mu g}{m^3})$)	Sources
Risk for hospital admittance due to asthma	$26 \cdot 10^{-6}$	Pönkä (1991) [44]
Risk for hospital admittance due to croup	$4 \cdot 10^{-3}$	Schwartz *et al.* (1991) [48]
Annual number of days with respiratory symptoms per person	$9 \cdot 10^{-3}$ $(5\text{-}13) \cdot 10^{-3}$	Schwartz and Zeger (1990) [50]

Table 5: Dose-response functions for NO$_2$.

the order of 15 $\frac{\mu g}{m^3}$ or lower. Also, emissions of SO$_2$ leads to the creation of sulphate in the atmosphere which properly belong to the group of small particles (PM$_{2.5}$). In several studies the apparent health effects of SO$_2$ disappear when particle concentrations are measured correctly. It is thus unclear whether SO$_2$ has any major independent health effects. Although we list some dose-response functions in table 6, we will not use them further on in this paper.

Health effect of SO$_2$	Coefficient (per annual mean concentration of SO$_2(\frac{\mu g}{m^3})$)	Sources
Risk of death	$480 \cdot 10^{-6}$	Ostro (1994) [35], ORNL/RFF (1994) [30]
Annual number of days with respiratory symptoms per person	$10 \cdot 10^{-3}$	Ostro (1994) [35], ORNL/RFF (1994) [30]

Table 6: Dose-response functions for SO$_2$.

4.4 Ozone (O$_3$)

Ozone is a so called secondary air pollutants which is created in a reaction between NO$_x$, methane (CH$_4$) and non-methane volatile organic compounds (NMVOC) in the atmosphere. Contrary to the situation in many other countries, the O$_3$ concentration in Norway is mainly determined by transboundary transport from the European continent. In fact, higher emissions of NO$_x$ in Norwegian cities is likely to *reduce* the concentration of O$_3$ in the city centers, but increase the level somewhat in the surroundings. Since we in this paper are mainly concerned with the effects related to Norwegian activities, we disregard at this stage the effects of O$_3$ on health. However, table 7 reports some dose-response functions for ozone found in the literature.

The concentration level in the dose-response functions related to mortality refers to maximum mean hourly level over a day, while the level in the function for MRADs, respiratory symptoms and hospital admittances refer to the annual mean of these concentration values. The coefficient for MRADs is an average of the results found in the two refered studies.

4.5 Summary of dose-response function

Table 8 provides a summary of some of the parameters entering the dose-response functions for the health effects of air pollution.

5 Transformation to economic variables - labour productivity

The next step is to transform the physical information contained in the dose-response functions to economic information on the direct costs associated with the various health effects. We do this

Health effects of O_3	Coefficient (per mean concentration of $O_3(\frac{\mu g}{m^3})$)	Sources
Risk of death	$75 \cdot 10^{-6}$	EC (1994) [13], Kinney et al. (1994) [26]
Annual number of minor restricted activity days per person (MRADs)	$12 \cdot 10^{-3}$	Ostro and Rothschild (1989) [37], Portney and Mullahy (1986) [42]
Annual number of days with respiratory symptoms per person	$26 \cdot 10^{-3}$	EC (1994) [13]
Annual hospital admittances with respiratory symptoms per person	$3.9 \cdot 10^{-6}$	Thurston et al. (1992) [53]

Table 7: Dose-response functions for O_3.

Health effects	PM_{10}	NO_2	SO_2	O_3
Acute mortality (δ)	$0.96 \cdot 10^{-3}$		$0.48 \cdot 10^{-3}$	$75 \cdot 10^{-6}$
COPD (ν)	$11 \cdot 10^{-3}$			
RAD/MRAD (λ_S)	$57.5 \cdot 10^{-3}$			$12 \cdot 10^{-3}$
Resp. symptoms (λ_S)	$180 \cdot 10^{-3}$	$9 \cdot 10^{-3}$	$10 \cdot 10^{-3}$	$26 \cdot 10^{-3}$
Hosp. admittance (η_H)	$36 \cdot 10^{-6}$	$26 \cdot 10^{-6}$		$3.9 \cdot 10^{-6}$

Table 8: Summary of parameters in dose-response functions for health effects.

for each pollutant in turn.

5.1 Particulate matter (PM_{10})

5.1.1 Mortality

People who die because of *short term* increases in the concentration of particulate matter, are mainly older people who do not participate in the labour force (Schwartz and Dockery, 1992 [49] as cited in Rowe et al., 1995 [47]). Also small children are suscestible to short term exposure to particulate matter. This group represents, however, only a very minor part of all those who dies in Norway. For these reasons *we will not assign an economic productivity loss to increased risk of mortality from short term exposure to particulate matter.* We should not forget, however, the considerable welfare loss associated with this effect. We will briefly touch upon this issue later on.

The more recent studies of the effects of *long term exposure* to particulate matter on mortality find that also people in the labour force are affected. In a study by Dockery et al. (1993) [10], the population between 25 and 74 years of age was divided into groups each covering a 5 year span. Job related exposure to dust and gases was identified and controlled for. In the study, the effect of long term exposure was found to affect all ages more or less equally. Mostly, the mortality effect was through lung cancer and respiratory diseases. Below, we illustrate how the information on physical effects are translated into effects on some economic variables.

In Norway in 1993, 330 people died from malign tumors in the respiratory organs. This represented 3.6 per cent of all deaths in this year, and the mean age of this group was 57.6 years. Thus, they had on average 7.4 years left before retiring. In addition 1552 people in the 25-64 years age range died of diseases in the cardiovascular and respiratory systems, representing 56.4 per cent of all deaths. Their mean age were 56.2 years, leaving 8.7 years to retirement. Combining these two groups, we have 1882 deaths due to the illnesses mostly associated with particle pollution with an average time to go before retirement of 8.5 years. Dividing the number of hours worked in 1993 (2.87 billion) by the population in the age bracket from 20 to 70 years (2.70 million), we find that the average numbers of hours worked per person in 1993 was 1061. This is probably a reasonable estimate of the annual hours worked for the age group we are most concerned with (55-65 years old).

Total hours of work loss because of the conditions mentioned above then amounts to (1882 deaths)×(8.5 years to retirement)×(1061 hours worked per year per person) = 17 million hours.

Combined with the dose-response function in table 4, and taking into account that 60 per cent of the total number of deaths was related to the two classes of diseases mention above, and finally assuming that only these conditions are affected by exposure particulate matter, we find the following effect on hours worked due to changes in PM_{10} concentration:

$$\Delta L = -17 \cdot 10^6 \cdot \frac{6.5 \cdot 10^{-3}}{0.6} \cdot \Delta PM_{10} \cdot \frac{P}{B} = -187 \cdot 10^3 \cdot \Delta PM_{10} \cdot \frac{P}{B}, \tag{5}$$

where L is the numbers of hours worked, $\frac{P}{B}$ is the fraction of the total population exposed to particulate matter concentrations and ΔPM_{10} is the change in the long run PM_{10} level measured in $\frac{\mu g}{m^3}$. Dividing by the number of hours worked in 1993 (2.87 billion) we can write this relation[2] as

$$\frac{\Delta L}{L} = -65 \cdot 10^{-6} \cdot \Delta PM_{10} \cdot \frac{P}{B}. \tag{6}$$

Since there is considerable uncertainty to several of the steps in the above calculation, not least with respect to the age structure of the mortality increase due to an increase in the PM_{10} concentration level, the final effect on the labour supply is also highly uncertain. For this reason we have at this stage decided *against* including this relation in the macroeconomic model.

5.1.2 Restricted activity days (RADs)

According to ORNL/RFF (1994) [30], 38 per cent of the restricted activity days are so called minor restricted activity days (MRADs). In the same study the cost of a MRAD was estimated to somewhat more than $\frac{1}{4}$ of the wage. Given than this estimate also covers some welfare effects, we assume that the labour productivity loss associated with one MRAD is 10 per cent. The average productivity loss of RADs can then be calculated as $(0.62 \cdot 1 + 0.38 \cdot 0.1) = 65.8$ per cent. We further assume that the RADs are uniformly distributed among working days and holidays. In the latter case we do not experience any effects on the labour productivity. According to the dose-response function for RADs (table 4), a unit increase in PM_{10} concentration increases the average number of RADs per year per person by $57.5 \cdot 10^{-3}$. Measured per day, this translates into $\frac{57.5 \cdot 10^3}{365} = 0.16 \cdot 10^{-3}$. On a working day the productivity is, as mentioned, reduced by 65.8 per cent for each RAD. Thus the effect of a unit increase in PM_{10} concentration on labour productivity is given by $0.16 \cdot 10^{-3} \cdot 0.658 = 1.04 \cdot 10^{-4}$, i.e.

$$\frac{\Delta L}{L} = -104 \cdot 10^{-6} \cdot \Delta PM_{10} \cdot \frac{P}{B}. \tag{7}$$

5.1.3 Respiratory symptoms

The dose-response function for respiratory symptoms has already been adjusted to avoid overlap with the occurrence of restricted activity days. Eskeland (1995) [12] assumes that each day with respiratory symptoms leads to a loss of labour productivity of 6 per cent. Following the same procedure as in the previous paragraph, we then obtain

$$\frac{\Delta L}{L} = -29.6 \cdot 10^{-6} \cdot \Delta PM_{10} \cdot \frac{P}{B}. \tag{8}$$

5.1.4 Chronic obstructive pulmonary diseases (COPD)

This is the only chronic effect of long term exposure to particulate matter that we have included. We assume that the change in the occurrence of COPD (given by the dose-response function in table 4) leads to a proportional change in number of people on sick leaves[3], number of people in restitution programs and number of people permanently disabled with this diagnosis. From the Norwegian health authorities, we have obtained data on the money paid for, or the number of

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[2]It is also possible, by use of Norwegian life tables, to transform the above results into reductions in expected length of life. The result is $\Delta FV = \left(\begin{smallmatrix} -0.059 \\ -0.064 \end{smallmatrix}\right) \Delta PM_{10}$, where the upper (lower) coefficient refers to the female (male) part of the population and ΔFV is the change in expected life times measured in years due to changes in the concentration level of PM_{10}.

[3]Only sick leaves longer than 14 days are registered.

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people in, the three categories by diagnosis in 1993 and 1994. Using the average hourly wage (sick leaves and restitution) and the average number of hours worked per person in the age group 20-70 years (disableness), we obtain the following relations between changes in hours worked and the three conditions of COPD patients

$$\frac{\Delta L}{L} = -242 \cdot 10^{-9} \cdot \Delta PM_{10} \cdot \frac{P}{B}, \text{ due to sick leaves} \tag{9}$$

$$\frac{\Delta L}{L} = -1.34 \cdot 10^{-6} \cdot PM_{10} \cdot \frac{P}{B}, \text{ due to restitution}$$

$$\frac{\Delta L}{L} = -24.1 \cdot 10^{-6} \cdot \Delta PM_{10} \cdot \frac{P}{B}, \text{ due to disableness.}$$

The study used for the underlying dose-response function (Abbey et al., 1993 [1]) was conducted over a ten years period. Thus, ΔPM_{10} is interpreted as changes in the ten years average PM_{10} concentration. A change in particle emissions today will therefore affect the labour force for the next ten years.

5.2 Nitrogen dioxide (NO$_2$)

The dose-response function for respiratory symptoms from NO$_2$ exposure can be translated into effects on hours worked in a similar manner to the procedure applied to the PM$_{10}$ dose-response function. The result is

$$\frac{\Delta L}{L} = -1.51 \cdot 10^{-6} \cdot \Delta NO_2 \cdot \frac{P}{B}. \tag{10}$$

Here, ΔNO_2 is the change in annual mean level of NO$_2$ measured in $\frac{\mu g}{m^3}$. The NO$_2$ concentration level in Norwegian cities is approximately twice the level of PM$_{10}$, which implies that the effect of NO$_2$ on the labour force is very much smaller than the corresponding effect of PM$_{10}$. This makes the problem of potential double counting of effects small.

5.3 Sulphur dioxide (SO$_2$)

The dose-response function for days with respiratory symptoms is, as mentioned before, highly uncertain. Nevertheless, we – for completeness – transform it in a manner similar to the above procedure to obtain:

$$\frac{\Delta L}{L} = -1.64 \cdot 10^{-6} \cdot \Delta SO_2 \cdot \frac{P}{B}. \tag{11}$$

5.4 Ozone (O$_3$)

Although we will not utilize it further on, we – again for completness – list the effects of O$_3$ on the labour productivity. We have two relations. For minor restricted activity days (MRADs) we find

$$\frac{\Delta L}{L} = -3.4 \cdot 10^{-6} \cdot \Delta O_3 \cdot \frac{P}{B}, \tag{12}$$

assuming a 10 per cent decrease in productivity for each MRAD. Respiratory symptoms are assumed to give a 6 per cent decrease in productivity, leading to the following relation for loss of working hours:

$$\frac{\Delta L}{L} = -2.3 \cdot 10^{-6} \cdot \Delta O_3 \cdot \frac{P}{B}. \tag{13}$$

5.5 Summary of health effects on labour productivity

The above results on the effects of air pollution on labour productivity can be summarized as in table 9. The table shows values for the coefficient λ in the relations of the form

$$\frac{\Delta L}{L} = \lambda \cdot \Delta C \cdot \frac{P}{B}. \tag{14}$$

Note, however, that only a small subset of all possible effects are included in this table, and that only some of them as judges as being certain enough to include in the macroeconomic analysis further on.

Effects	λ	Comments
PM_{10} : Mortality	$-65 \cdot 10^{-6}$	Not implemented in macromodel
PM_{10} : RADs, MRADs	$-104 \cdot 10^{-6}$	
PM_{10} : Resp. symptoms	$-30 \cdot 10^{-6}$	
PM_{10} : COPD sick leaves	$0.24 \cdot 10^{-6}$	10 years average pollution level
restitution	$1.34 \cdot 10^{-6}$	
disableness	$24.1 \cdot 10^{-6}$	
NO_2 : Resp. symptoms	$-1.51 \cdot 10^{-6}$	
SO_2 : Resp. symptoms	$-1.64 \cdot 10^{-6}$	Not implemented in macromodel
O_3 : MRADs	$-3.4 \cdot 10^{-6}$	Not implemented in macromodel
O_3 : Resp. symptoms	$-2.3 \cdot 10^{-6}$	Not implemented in macromodel

Table 9: Summary of health effects of air pollution on labour productivity.

6 Transformation to economic variables - public health expenditures

In order to assess the changes in public expenditures due to changes in air pollution levels, we employ average figures for the length of hospital stays and the hospital cost per day. In 1993 the average length of a hospital admission due to a respiratory illness was 5.96 days (Statistics Norway, 1995 [52]). The (variable) cost of each day in a Norwegian hospital was 3187 1991-Nkr (Statistics Norway, 1993 [51]). Based on the dose-response function for PM_{10} in table 4, we then deduce the following relation

$$\Delta O_H = 0.675 \cdot \Delta PM_{10} \cdot P, \tag{15}$$

where O_H is the cost measured in 1993-Nkr. Since the base year for the macroeconomic model we are going to employ is 1988, we also give the relation when public expenditures are measured in 1988-Nkr.

$$\Delta O_H^{88} = 0.60 \cdot \Delta PM_{10} \cdot P. \tag{16}$$

According to Statistics Norway (1993) [51], 71 per cent of the variable cost of a hospital day represent wages. The average wage cost in the health sector was 130.1 Nkr per hour in 1988. From this we deduce the following function for changes in hours worked due to changes in exposure to particulate matter

$$\Delta L_H = 5.5 \cdot 10^{-3} \cdot \Delta O_H^{88} = 3.3 \cdot 10^{-3} \cdot \Delta PM_{10} \cdot P. \tag{17}$$

The rest of the hospital costs are due to use of materials. It is unclear how large the investments costs are in the hospital sector. We have chosen to use a moderate estimate equal to 5 per cent of the variable cost. We then obtain the following functions for these cost categories, where H denotes use of intermediates and J denotes investments

$$\Delta H_H = 0.29 \cdot \Delta O_H^{88} \tag{18}$$
$$\Delta J_H = 0.05 \cdot \Delta O_H^{88}.$$

If we assume that the increase in number of hospital days due to chronic obstructive pulmonary disease (COPD) varies linearly with the occurrence of COPD, and combining this with the information that in 1993 there were 28.3 hospital days per 1000 inhabitants with the diagnosis of COPD, we can deduce the following cost function related to COPD

$$\Delta O_H^{88} = 0.87 \cdot \Delta PM_{10} \cdot P. \tag{19}$$

Here, ΔPM_{10} is to be interpreted as change in the concentration level averaged over a 10 year period.

Regarding hospital admittance due to asthma, we found a 1.5 per cent increase per unit increase in NO_2 concentration (table 5). In 1993 there were on average 8.7 hospital days per 1000 people with the diagnosis of bronchitic asthma. Following the same procedure as above, we obtain the following cost function related to this diagnosis

$$\Delta O_H^{88} = 0.37 \cdot \Delta NO_2 \cdot P. \tag{20}$$

The effects on wage-, intermediates- and investment cost are as above (eq. 18).

The coefficients for the air pollution induced variable costs in the public health sector

$$\Delta O_H^{88} = \kappa \cdot \Delta C \cdot P \tag{21}$$

are summarized in table 10.

	κ
PM_{10}	
Acute	0.60
Chronic	0.87
NO_2	
Asthma	0.37

Table 10: Summary of effects on the variable costs in the public health sector.

7   Welfare effects

All the health effects explored above of course have important welfare effects in addition to the economic losses incurred. To value these welfare effects is, however, extremely difficult (and perhaps even impossible). For illustrative purposes it may nevertheless be of interest to mention some monetary values in this connection, although we would warn against using these numbers uncritically in a decision making process. Rather one should then use the results on the *physical* effects contained in the dose-response functions.

There exist an estimate of the value of a statistical life developed in connection with traffic accidents in Norway (Elvik, 1993 [11]) which is given as 10.5 million 1993-Nkr (approximately equal to 1.6 million US$). It may be discussed how relevant this value is for deaths due to air pollution, as for instance the age distribution of such deaths is very different from deaths due to traffic accidents. The value is, however, meant to capture only the welfare effects of the risk for deaths. International studies seem often to use higher values, see e.g. Pearce (1995) [38]. With the dose-response function for mortality associated with short term exposure to PM_{10}, and with a general mortality rate in the population of 1.04 per cent, we get the following expression for the welfare effects of PM_{10} mortality

$$\Delta V = 105 \cdot \Delta PM_{10} \cdot P, \tag{22}$$

where V is measured in 1993-Nkr.

We can also illustrate the welfare effect of higher risks for chronic respiratory diseases by noting that Rowe et al. (1995) [47] is using an estimate of 210 000 1992-US$ based on willingness to pay studies. This corresponds to an amount approximately equal to 1.5 million 1994-Nkr.

13

Part II

Material damage

Most materials placed in a polluted atmosphere will experience corrosion over and above what would occur in a (hypothetical) clean atmosphere. It is mainly the exposure to sulphur and ozone that creates the damage, but also other compounds (e.g. NO_x) may induce additional corrosion. Here we concentrate on the effects of SO_2 and O_3.

Pollution levels, climatic conditions and material densities all varies from region to region and thus induce differences in corrosion rates and the cost of corrosion. We have chosen to treat conditions in Oslo in a very detailed manner, and used a cruder methodology for other urban areas in Norway. In Oslo, we have, through dispersion modelling, available information on air pollution concentration levels within a 500×500 m grid. Also, we know the coordinates, size and use of every building in this area. From special surveys (to be discussed below), we know the typical material composition of buildings employed for different purposes, and can thus deduce the distribution of material densities across the grids of Oslo. In other urban areas, the material densities are deduced from other types of statistics, such as manufacturing, agricultural, population and employment statistics.

8 Exposure to climate, SO_2 and O_3

The concentration of SO_2 in the atmosphere is the sum of a background concentration level, S_b, and contributions from local emissions U

$$S = S_b + \varepsilon \cdot U, \tag{23}$$

where S_b is measured in $\frac{\mu g}{m^3}$, U is local emissions of SO_2 measured in metric tonnes, and ε is a parameter relating emissions to concentration levels.

Ozone concentration in the city centres, where most of the material is concentrated, is generally a declining function of the NO_2 concentration level (Kucera et al., 1995 [29]). We model the O_3 level in Oslo by the following function

$$O = 60.5 \cdot e^{-0.014 \cdot [NO_2]}, \tag{24}$$

where $[NO_2]$ is the concentration level measured in $\frac{\mu g}{m^3}$. In other regions than Oslo, the ozone levels are assumed constant and equal to typical observed values. Table 11 gives climatic conditions and concentration levels of SO_2 and O_3 in the regions of Norway. The values for Oslo are from a typical grid site in the city. In the table, $[SO_2]$ and $[O_3]$ are concentration levels of SO_2 and O_3, respectively, T_W is the time of wetness measured as the fraction of a year when relative humidity exceeds 80 per cent and the temperature is above $0°C$, R is the amount of precipitation measured in $\frac{m}{year}$, and H^+ is the acidity of the precipitation measured as $\frac{mgH^+}{l}$ rain.

9 Exposed material

The amount of exposed material is based on a study by Kucera et al. (1993) [28]. The study, which is the most comprehensive existing, surveyed in a detailed manner material densities of different types of buildings in Praha, Stockholm and the Norwegian town Sarpsborg. These results have been extrapolated to national numbers for Sweden (Anderson, 1994 [6]) and to Europe (Cowell and ApSimon, 1994 [9]). From these studies it is possible, together with national statistics and building registers, to estimate the amount of materials in urban areas in Norway. Figure 2 shows the result for Oslo. Glomsrød et al. (1996) [20] provides discussion of the methodology employed and detailed results for all Norwegian regions.

10 Material loss due to corrosion and the lifetime functions

Annual maintenance cost of materials (K) can be written

Regions	$[SO_2]$ $(\frac{\mu g}{m^3})$	$[O_3]$ $(\frac{\mu g}{m^3})$	H^+ $(\frac{mg}{l})$	R $(\frac{m}{year})$	T_w
Halden	5	55	0.040	0.80	0.38
Sarpsborg	16	50	0.032	0.88	0.39
Fredrikstad	7	50	0.032	0.79	0.38
Moss	5	55	0.032	0.81	0.40
Bærum	6	45	0.032	0.82	0.39
Asker	5	45	0.032	0.94	0.41
Oslo	15	30	0.025	0.60	0.32
Drammen	6	43	0.032	0.95	0.34
Porsgrunn	5	55	0.032	0.92	0.33
Skien	14	55	0.032	0.85	0.33
Bamble	3	53	0.032	0.87	0.33
Kr.sand	3	54	0.040	1.38	0.49
Stavanger	6	60	0.032	1.25	0.70
Bergen	7	62	0.020	2.25	0.53
Trondheim	5	55	0.008	0.93	0.41
Tromsø	2	55	0.010	1.03	0.24
Urban south	3	55	0.031	1.03	0.42
Urban north	2	55	0.010	1.03	0.24
Rest of the country	1	40	0.031	1.03	0.42

Table 11: Climatic conditions and pollution levels in Norwegian regions. The values for Oslo are from a typical grid site in the city center.
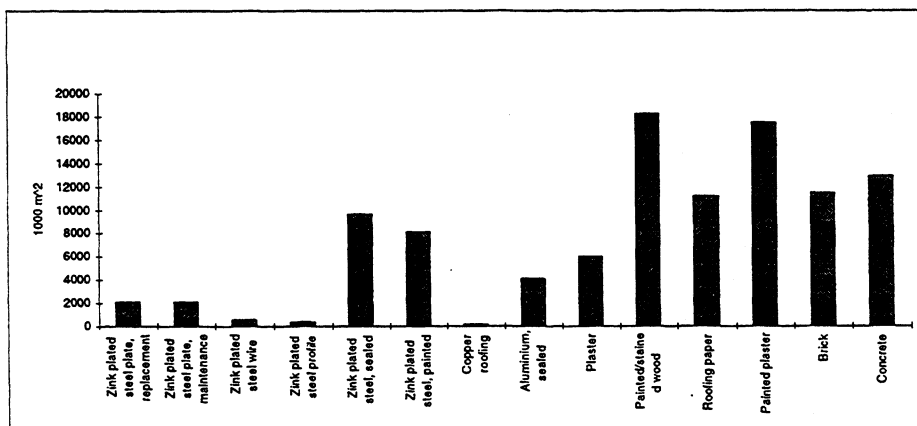
Figure 2: Materials in Oslo, 1000 m^2.

$$K = \frac{M \cdot P}{L},\qquad(25)$$

where M is the amount of material of a given type measured in m^2, P is the price of maintenance or replacement measured in $\frac{Nkr}{m^2}$, and L is the time period between maintenance or replacement. L is here also denoted the *lifetime* of the material.

The lifetime is a function of amount of material lost due to corrosion which in turn is dependent on the external environment, in particular the exposure to air pollution. Of particular importance in this respect is the exposure to sulphur dioxide (SO_2) and ozone (O_3). However, also climatic conditions play a role, in particular humidity, precipitation and the acidity of the precipitation. Extensive studies of material corrosion, as for instance summarized in Haagenrud and Henriksen (1995) [21], together with assumptions on when maintenance or replacement is necessary, makes it possible to derive lifetime functions. It is found that a functional form of the following form gives a good fit to the experimental data (see Glomsrød et al., 1996 [18] for further documentation, references and discussions):

$$L(S,O) = \frac{a}{(b + c \cdot T_W \cdot O) \cdot S + d \cdot H^+ \cdot R + e},\qquad(26)$$

where a, b, c, d and e are material specific constants, S and O are concentration levels of SO_2 and O_3, respectively, measured in $\frac{\mu g}{m^3}$. T_W, R and H^+ have the same meanings as before. Table 12 gives values for the constants in eq. 26 and also shows the types of materials covered.

Materials	a	b	c	d	e
Zink plated steel plate, replacement	30		0.0015	2.82	0.51
Zink plated steel plate, maintenance	20		0.0015	2.82	0.51
Zink plated steel wire	30		0.0015	2.82	0.51
Zink plated steel profile	60		0.0015	2.82	0.51
Zink plated steel, sealed	1000	0.155			38.6
Zink plated steel, sealed and painted	1000	0.37			64.3
Zink plated steel, painted	1000	0.803			84.5
Painted steel	1000	1.37			108
Copper roofing	100		0.00031	4.575	0.542
Aluminium, sealed	1000	0.107			32.6
Aluminium, sealed and painted	1000	0.37			64.3
Plaster	1000	0.124			15.7
Painted plaster	1000	0.278			19.9
Painted/stained wood	1000	1.03			91.4
Roofing paper	1000	0.327			48.9
Brick	If $S < 10\,\mu g/m^3$, then the lifetime is 70 years, else 65 years.				
Concrete	If $S < 10\,\mu g/m^3$, then the lifetime is 50 years, else 40 years.				

Table 12: Constants in the lifetime function.

Of the materials listed in the table, zink plated sealed and painted steel, painted steel and sealed and painted aluminium are not included in the macroeconomic simulations presented later in this paper.

11 The price of maintenance/replacement

Table 13 shows the maintenance or replacement costs employed in this study. The prices have been obtained by an informal survey of prices in the Oslo region in 1995.

12 The costs of corrosion

From the expression for the annual maintenance cost (eq. 25), the table 12 giving the lifetime functions and the prices in table 13, we can calculate the corrosion cost associated with air pollution

Material	Prices ($\frac{Nkr}{m^2}$)
Zink plated steel plate, replacement	275
Zink plated steel plate, maintenance	150
Zink plated steel wire	105
Zink plated steel profile	300
Zink plated steel, sealed	150
Zink plated steel, painted	300
Copper roofing	450
Aluminium, sealed	150
Aluminium, sealed and painted	150
Plaster	350
Painted/stained wood	80
Roofing paper	160
Painted plaster	250
Brick	300
Concrete	525

Table 13: Prices of maintenance or replacement exclusive of taxes. 1995-Nkr per m^2.

levels above "natural" background levels (S^b, O^b)

$$K^f = M \cdot P \left[\frac{1}{L(S,O)} - \frac{1}{L(S^b,O^b)} \right].$$ (27)

Marginal cost from changes in the *concentration level* of SO_2 is then given by

$$K_S \equiv \frac{\partial K}{\partial S} = M \cdot P \cdot \frac{b + c \cdot T_W \cdot O}{a},$$ (28)

while the marginal cost from changes in local *emissions* of SO_2 is given by

$$K_U \equiv \frac{\partial K}{\partial U} = \varepsilon \cdot M \cdot P \cdot \frac{b + c \cdot T_W \cdot O}{a}.$$ (29)

Due to the linearity of the functions, the marginal costs are independent of concentration and local emission levels. However, they depend on the interpretation of "local" or "natural" background, i.e. they depend on the parameter ε in eq. 23.

Two interpretations are natural. One is that the background concentration is due to emissions in all other regions than the one under consideration, e.g. including emissions in foreign countries and in other domestic regions. Although this is perhaps the most natural interpretation, it is difficult to make operational since we generally do not know the transport matrix a_{ij} between the regions used in this study. An alternative interpretation is to consider the background as that due only to foreign emissions. However, the concept of marginal costs then will have a rather special meaning since it in this case will reflect the increase in local costs due to a proportional increase in emissions *in the whole of Norway*.

Generally, we have that the concentration level in region i is determined by

$$S_i = \sum_{j \in \{regions\}} a_{ij} U_j = S_i^{b,u} + S_i^{b,i} + a_{ii} U_i$$ (30)

where $S_i^{b,u}$ is the background concentration due to foreign emissions and $S_i^{b,i}$ denotes the background due to emissions in other regions of Norway. The element a_{ii} corresponds to the parameter ε above. We know U_i, S_i and $S_i^{b,u}$, while $S_i^{b,i}$ and a_{ii} are unknown. As a simplification we in this study put $S_i^{b,i}$ equal to zero and determine $a_{ii} = \varepsilon$ by eq. 30:

$$\varepsilon = \frac{S_i - S_i^{b,u}}{U_i}.$$ (31)

By neglecting $S_i^{b,i}$, ε is biased upwards by $\frac{S_i^{b,i}}{U_i}$, and the marginal costs due to local emissions overestimated correspondingly. It should be noted, however, that SO_2 is generally not transported over long distances as such, and therefore the above approximation may not be too bad.

Table 14 gives the SO_2 emission levels, estimated total corrosion costs due to these emissions and the marginal costs associated with an increase in the emission of SO_2 based on the data and formulas above.

Region	Emission of SO_2 1994 (Metric tonnes SO_2)	Cost (Mill. 1995-Nkr)	Marginal cost $(\frac{1995-Nkr}{kg\,SO_2})$
Halden	68	1.77	26.17
Sarpsborg	1 669	5.36	3.21
Fredrikstad	885	2.41	2.73
Moss	664	1.56	2.34
Bærum	119	7.11	59.90
Asker	62	2.57	41.28
Oslo	1 051	74.40	70.81
Drammen	74	3.84	51.65
Porsgrunn	556	2.08	3.73
Skien	229	13.15	57.49
Bamble	24	0.48	19.91
Kristiansand	1 008	2.13	2.12
Stavanger	277	8.62	31.11
Bergen	262	22.61	85.98
Trondheim	674	9.23	13.70
Tromsø	108	0.80	7.48
Urban south	17 886	34.76	1.94
Urban north	5 798	3.56	0.61
Rest of Norway	13 120	1.29	0.10
Total	44 543	197.73	4.44 (averaged)

Table 14: Emissions, costs and marginal costs of material damage by regions.

It is seen from the table that the marginal costs varies widely between regions. This is not surprisingly, given that Norway is a sparsely populated country where much of the most polluting (power intensive) industries are located in remote areas. The national average marginal cost of 4.44 Nkr per kg SO_2 reported in table 14, is somewhat high compared to results from other studies, see Calthrop and Pearce (1996) [8]. However, several studies in their sample show marginal costs up to 13 Nkr per kg SO_2.

Table 15 shows how the marginal costs are distributed by type of emission sources. Process emissions, i.e. emissions not related to stationary combustion of fossil fuels, account for more than half of the total emissions in 1994, but are associated with only a quarter of the corrosion costs. This is due to the localisation of the power intensive industry in Norway in relatively remote areas. Emissions from mobile sources, mainly diesel powered automobiles, have the highest marginal cost.

	Emissions 1000 tonnes	Costs Mill. 1995-Nkr	Marginal costs Nkr/kg
Process	19.2	47.2	2.5
Stationary	7.9	63.5	8.0
Road	3.2	39.9	12.6
Rail	0.1	0.4	5.1
Airplanes	0.1	0.6	4.5
Ships	13.4	41.9	3.1
Other machinery	0.6	4.2	7.2
Total/average	44.5	197.7	4.4

Table 15: SO_2 emissions by source type and material corrosion costs in 1994.

Based on the marginal cost information, it is also possible to calculate the gains obtained over the 10 years period from 1985 to 1994 when Norway experienced strong reductions in SO_2 emissions. The result, excluding the allocation cost[4] associated with material damage, is given in

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[4]The allocation costs are calculated to have been 233 million 1994-Nkr in 1985 and 93 million Nkr in 1994, thus

table 16.

	Emission of SO$_2$ (1000 metric tonnes)	Total costs (Mill. 1994 Nkr)
1985	97.4	496
1994	44.5	198
Reduction 1985-1994	61.3	298

Table 16: Emissions and total material costs due to corrosion in 1985 and 1994.

Part III

Traffic accidents

Effects of a similar kind to those discussed above, but not directly associated with a deteriorated environment, are road traffic accidents. These are strongly correlated with the use of fossil fuels, and have predominantly effects on labour supply and productivity, and public health expenditures, given that we neglect welfare effects. Fridstrøm and Bjørnskau (1989) [15] have carried out a detailed empirical study of possible explanatory variables for traffic accidents with person injuries in Norway. Controlling for climatic variations, road standards, traffic density and special security measures, they find that a 10 per cent increase in gasoline consumption leads to 8-9 per cent increase in traffic accidents with injuries. Moreover, they find that higher road standards with less traffic density *increase* the number of accidents with injuries. This finding is also supported by an older Danish study (Vejdirektoratet, 1979 [54]). Thus there seems to be a trade off between congestion costs and the cost of injuries from traffic accidents. Here, we only consider the costs of accidents.

Table 17 gives some key numbers related to traffic accidents in Norway.

	Persons affected	Loss of man-years
Traffic accidents	33 900	
Deaths due to traffic accident (also in previous years)	332	7 254
Sick leaves first year after accident		1 350
Sick leaves due to children's accidents		167
Productivity loss due to traffic accidents in the last 10 years		1 346
50 per cent disabled due to traffic accidents in previous years	272	2 888
100 per cent disabled due to traffic accidents in previous years	477	10 146
Total		23 151

Table 17: Some key annual numbers related to traffic accidents in Norway in 1990.

Loss of man-years due to people killed in the traffic is of the order of 7 000. The average age of those killed in the traffic is quite low; the length of the expected active participation in the work force is 38 years for those killed. Sick leaves and productivity losses for those in work represent each about 1 300 man-years, while 13 000 man-years are lost each year due to varying degrees of invalidity following traffic accidents. Thus, over 23 000 man-years are lost each year due to traffic accidents in Norway. The breakdown of traffic accidents according to seriousness, sick leaves and disableness is described in Haukeland (1991) [23], while Hagen (1993) [22] has analysed the costs associated with the various categories of person injuries from traffic accidents.

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representing an additional annual saving of 104 million Nkr.

13 Traffic accidents in a macroeconomic model

The effects of traffic accidents are implemented in the macroeconomic models of Statistics Norway in the following manner.

Use of fuels (gasoline and diesel) are projected by the macroeconomic models. This is translated to kilometer driven by the following formulas

$$KM_t^G = m^G \cdot G_t \cdot e^{\theta^G t}, \tag{32}$$

$$KM_t^D = m^D \cdot D_t \cdot e^{\theta^D t}, \tag{33}$$

where $m^{G,D}$ are average number of kilometres driven per tonnes of gasoline and diesel, respectively, calibrated in the base year. The energy efficiency of vehicles increase by fixed annual rates $\theta^{G,D}$, while G and D represent the demand for the two types of fuels. Traffic density (CON_t) is determined by the ratio between kilometres driven and the total length of roads

$$CON_t = \frac{KM_t^G + KM_t^D}{ROADS_t}. \tag{34}$$

The growth of the variable $ROADS$ is exogenously determined as an exponentially decreasing function fitted to historical data from the period 1966 to 1993, and planned extensions for the period 1994-1997.

The number of persons injured in traffic accidents is modelled as follows:

$$S = K \cdot e^{\varepsilon t} \cdot \left(KM_t^G\right)^\gamma \cdot \left(KM_t^D\right)^\delta \cdot \left(\frac{CON_t}{CON_0}\right)^\sigma. \tag{35}$$

Here, K is a constant, while $e^{\varepsilon t}$ represents a trend due to factors not covered in this analysis. A fraction of the number of people injured in traffic accidents are productive workers. Thus the accidents leads to reduced supply or productivity of the labour force through sick leaves or permanent losses due to deaths or disabilities.

The temporary loss of manpower is modelled as follows:

$$\Delta L_t^T = \sum_{\tau=0}^{9} \beta_\tau S_{t-\tau} + (\zeta + \xi)(S_t - S_0). \tag{36}$$

The first term on the right hand side represents productivity loss in year t due to traffic accidents in the last 10 years. The last term represents loss due to own accidents and children's accidents this year.

Permanent loss of manpower due to deaths (ΔL_t^F) and invalidity (ΔL_t^D) are given by

$$\Delta L_t^F = \eta(S_t - S_0), \tag{37}$$

$$\Delta L_t^D = \mu(S_{t-1} - S_0). \tag{38}$$

The permanent reduction in the labour force accumulates according to

$$\Delta L_t^P = \Delta L_t^F + \Delta L_t^D + \Delta L_{t-1}^P. \tag{39}$$

Total reduction of the labour force in year t is calculated as the sum of temporary and permanent reductions:

$$\Delta L_t = \Delta L_t^T + \Delta L_t^P. \tag{40}$$

We let the total treatment cost of traffic victims vary with number of victims. Thus, in the model simulations the number of traffic accidents will influence on the otherwise exogenously determined health service production level. The demand for input factors in the health sector is then given by

$$G_{i,t} = \omega_i(S_t - S_0), \quad i = labour, \ materials, \ capital. \tag{41}$$

Glomsrød, Nesbakken and Aaserud (1996) [17] discuss and documents the coefficients appearing in the above equations.

Part IV

Simulations

By including the effects of air pollution discussed above (health effects, effects on public budgets and material corrosion) in a macroeconomic model (MSG-EE, see Alfsen et al., 1996 [4]), it is possible to capture the *allocation costs* associated with changes in relative prices and supply of resources due to the environmental feedbacks. In the private sectors, the effect of material corrosion is implemented as a change in the user cost of capital. The relation between changes in SO_2 emissions and the user cost of capital is determined by dividing the total corrosion cost, which varies between sectors due to differences in pollution levels and material densities, by the amount of capital of the relevant type (buildings, machineries, etc.) and a weighted average of SO_2 concentration. For the public sectors, where the production levels are exogenously determined, we have increased the depreciation of the building capital in order to incorporate the effect of material corrosion. In order to maintain the production levels, public investments must then be increased.

To be conservative and not incorporate too many uncertain results, we have only included the corrosion costs due to changes in SO_2 emissions and some of the health effects associated with PM_{10}. Thus, effects of changes in NO_2 or O_3 exposure is not included. Also, we have left out the potentially important effects of long term exposure of PM_{10} on mortality and morbidity.

14   The marginal costs of emissions

As a first exercise, we have calculated the marginal cost of increased pollution by adding one tonne of PM_{10} and SO_2 to the emissions in Oslo. Simulating the Norwegian economy by use of the MSG-EE model (Alfsen et al., 1996 [4]) without and with the additional emissions, we obtain the results reported in table 18, which also shows the marginal costs associated with a litre additional use of gasoline and diesel.

		Costs
Health	PM_{10}	1 128 Nkr/kg
	Gasoline	0.08-0.3 Nkr/l
	Diesel	2.2-4.1 Nkr/l
Materials	SO_2	175 Nkr/kg
	Gasoline	0.08 Nkr/l
	Diesel	0.18 Nkr/l

Table 18: Marginal costs of additional pollution in Oslo.

It should be noted that the results represent strictly lower limits to actual costs, since several potentially important health effects have been omitted. Also, the welfare effects of morbidity are neglected. Thus, if we includes the effect of long term exposure to PM_{10} on mortality, the marginal health cost is increased by a factor larger than 5. Similarly, including the welfare effects of chronic respiratory diseases will increase the marginal cost by some 60 per cent.

The cost of corrosion damage in other regions than Oslo can be obtained from the marginal cost figures in table 14 by multiplying by the factor 1.48, representing the allocation costs. Including the allocation costs, we find that the annual cost of corrosion increase from approximately 200 million Nkr to 290 million Nkr, increasing the average national marginal cost of SO_2 emissions from 5.59 to 8.20 $\frac{Nkr}{kgSO_2}$. Similarly, we find that the allocation cost of the restricted set of health effects studied, represents an additional 11 per cent to the direct productivity costs.

15 Secondary benefits of a carbon tax

We have also studied the impacts of the environmental feedbacks described above on policy analysis of carbon taxation. The starting point is a reference path to year 2020 generated with the MSG-EE model without the environmental feedbacks. Without going into neither a detailed description of the economic growth path, nor a discussion of the underlying model mechanisms, we note that from an environmental point of view, the path is characterized by slightly falling PM_{10}, NO_x and SO_2 emission levels and increasing CO_2 emission level, see figure 3.
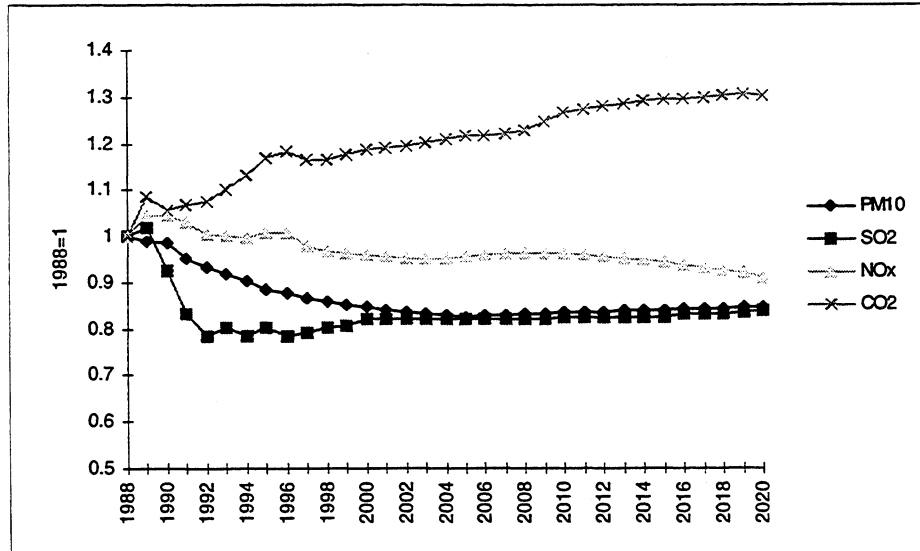
Figure 3: Emissions of PM_{10}, NO_x, SO_2 and CO_2 in the reference scenario. 1988=1.

With almost constant emission levels of local pollutants, the health and material corrosion costs are also almost constant. Since we interpret the labour supply, depreciation rates, etc. in the model as values where historical levels of health and material damage have been included, we will not see any effects of the environmental feedbacks in scenarios with more or less constant emission levels.

In order to study the effects of the feedbacks due to changes in emission levels, we have analysed an economic scenario where the current CO_2 tax is increased gradually from 150 Nkr per tonnes CO_2 (0.40 Nkr per litre oil) to 2 854 Nkr per tonnes CO_2 (7.60 Nkr per litre oil) in 2020. The tax on labour is reduced correspondingly to make the tax reform revenue neutral. Note, however, that within the framework of the general equilibrium model MSG-EE, total labour supply (disregarding the health effects) is exogenously given. The effects of the carbon tax on emission levels are reported in table 19.

	Changes in 2020
PM_{10}	-6%
NO_x	-13%
SO_2	-31%
CO_2	-35%

Table 19: Emission reductions in the tax scenario relative to the reference scenario in year 2020.

The relative low reduction in particle emission can be explained by the fact that household's use of wood is an important source for these emissions in Norway. The comparatively strong effect on SO_2 emissions, is related to the fact that the power intensive industry in Norway is an important contributor to these emissions, while at the same time contributing heavily to national CO_2 emissions. Lower emission levels reduce the concentration levels correspondingly. Thus, the PM_{10} and NO_2 concentrations in Oslo in year 2020 are reduced from 24.1 to 22.3 and 42.0 to

39.1$\frac{\mu g}{m^3}$, respectively. The weighted national concentration of SO_2 is reduced by 1.5$\frac{\mu g}{m^3}$ to a level marginal above 4$\frac{\mu g}{m^3}$.

The carbon tax increases the cost of production and lowers the level of economic activity. GDP is thus reduced by almost 20 billion 1994-Nkr, corresponding to 1.7 per cent, in year 2020, see table 20.

		2020
Loss of GDP	Without feedbacks	19 624
		(-1.7 %)
Gain in GDP	From feedbacks	216
Reduced damage to buildings (private sector)		203
Reduced damage to buildings (public sector)		50
Reduced public health expenditures		5
Total productive gains		474
Productive gains as percent of the first order GDP loss		2.4 %

Table 20: Effects of a CO_2 tax and reduced tax on labour. Million 1994-Nkr.

Including the feedbacks, reduces the GDP loss by 216 million 1994-Nkr. Total maintenance costs are reduced by some 250 million Nkr. Total gain in productivity is thus 474 million Nkr, representing 2.4 per cent of the initial GDP loss. As mentioned, these numbers represent minimum levels since only a few, well documented effects have been included among the feedbacks. Additional welfare effects are disregarded, but the importance of these can be illustrated as in table 21, which reports physical effects of the increased carbon tax and some tentative valuations of these physical effects.

Health effects	Physical numbers	Monetary values
Number of deaths	-17	177
Hospital days	-1 424	
New cases of COPD	-69	121

Table 21: Health effects of an increased carbon tax and tentative monetary values in Million 1994-Nkr.

If we include the effects on mortality of long term exposure to particulate matter, we come up with a reduction in the number of deaths in year 2020 of the order of 70-80, corresponding to an increase in welfare of the order of 800 million Nkr based on the value of a statistical life mentioned above. This illustrates that effects so far left out of the analysis may be very important for the overall importance of the feedbacks.

Another and related topic left out of the discussion so far, is the issue of external effects of road traffic. Including these effects along the lines discussed in part 3 above, we find additional productive gains from the increase in the carbon tax of the order of 1.1 billion 1994-Nkr in year 2020 with welfare effects of the same order of magnitude. Thus, at the moment these traffic related effects are more important than the pollution related health and material effects included in the above discussion, see table 22.

Other effects not included so far comprise the effects of traffic on noise levels, damage to roads and congestions, as well as pollutant related damage to fresh water lakes, forest and agricultural crops. Also damage to cultural and historic buildings and sites are excluded. It may be that these effects are of more importance than those included in this analysis, but currently the effects are less well documented than those included in this paper.

16   Summary

Valuing an environmental asset like clean air is at best a difficult task, and most probably impossible. The problems range from lack of technical knowledge of the physical effects of air pollution, to fundamental problems associated with the definition and aggregation of individual welfare effects. Probably, a substantial part of the valuation procedure cannot be carried out in an objective and

	2020
Reduced mortality from long term exposure to PM_{10}	800
Traffic accident related productivity effects	1 100
Wellfare effects from pollution	300
Wellfare effects from traffic accidents	1 000+
Total additonal gains	3 200+
Additional gains as percent of the first order GDP loss	16 %+

Table 22: Additional and more uncertain benefits of a CO_2 tax combined with a reduced tax on labour in year 2020. Million 1994-Nkr.

value free manner and should be left to the political arena for decision. We are, however, able to provide some well researched and objective input to the political process, and this paper is an attempt to document the state of the art in Norway in this regard.

The use of economy wide and disaggregated macroeconomic models in this process has several advantages. First, we are able to capture the macroeconomic allocation costs associated with the productivity losses imposed on the economy by air pollution and road traffic. Second, the model treats several air pollutants (and transport activities) and their effects on the economy simultaneously, thus providing a framework for assessing many of the cost components of several pollutants at once. Third, the model allows us to assess the effects of not only environmental control policies directed at curbing the emission of one or a few pollutants, but also to assess more general policies like for instance tax or trade policies. Finally, and perhaps special for Norway, the models are actively used by the policy makers. Thus, effects, linkages and results integrated in the models are taken note of in the policy making process.

Two aspects of the preliminary results reported in the paper are perhaps stricking. One is the relative smallness of the economic costs associated with air pollution in Norway. As noted many times, this is most probably due to the limited number of environmental effects we judge to be well enough documented to form part of an objective knowledge base. The second aspect has the opposite "flavour"; namely the seriousness of the physical impacts of air pollution. Thus, we find relative large marginal corrosion cost of SO_2 emissions in several large cities in Norway. These costs allows for far more stringent air pollution abatement policies than are in force today. Also, the number of deaths associated with short term exposure to particulate matter is probably well above what most people would have expected. That the people affected are old and unproductive in a limited economic sense, does not detract from the seriousness of the situation. This point is, of course, made even more important when one consider the possible effects of long term exposure to particulate matter and when we try to grasp the loss of welfare associated with all the health effects described in this paper. That we are unable to account for these effects in monetary terms, places a heavy responsibility on all of us as participants in the political processes.

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