Knut Einar Rosendahl (ed.)

Social Costs of Air Pollution and Fossil Fuel Use
– A Macroeconomic Approach

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Emneord
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Luftforurensning
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Abstract

Knut Einar Rosendahl (ed.)

Social Costs of Air Pollution and Fossil Fuel Use
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Economic activity and environmental conditions are related to each other in several ways. Production and consumption may pollute the environment, and at the same time the state of the environment may affect the production capacity of the economy. Thus, it follows that studying social costs of air pollution should be handled within an integrated model. Moreover, air pollution mostly stems from the use of fossil fuels, which also brings about other non-environmental externalities, particularly in the transport sector. It is therefore topical to include these externalities in a full social costs evaluation.

In this book we are concerned with social costs on a national level, although the environmental effects are evaluated on a more local level. We apply a general equilibrium model of the Norwegian economy, which is extended to integrate environmental and non-environmental effects of fossil fuel use. Moreover, the model includes feedback effects from the environment to the economy. In four independent studies, selected environmental and non-environmental externalities are analysed within this model. These are material damages, crop damages and health damages from air pollution, and finally health damages from traffic accidents.

Keywords: Air pollution, fossil fuel use, integrated economy-environment model, road traffic, social costs.

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Sammendrag

Knut Einar Rosendahl (red.)

Samfunnsøkonomiske kostnader av luftforurensning og fossile brensler

– En makroøkonomisk tilnærming

Sosiale og økonomiske studier 99 • Statistisk sentralbyrå 1998

Økonomisk aktivitet og miljøforhold er knyttet til hverandre på flere måter. Produksjon og konsum kan forurense miljøet, samtidig som miljøtilstanden kan påvirke produksjonskapasiteten i økonomien. Det er derfor viktig å studere samfunnsøkonomiske kostnader av luftforurensning i en integrert modell. Samtidig skyldes luftforurensning i hovedsak bruk av fossile brensler, som også medfører andre eksternaliteter, spesielt i transportsektoren. Det er derfor hensiktsmessig å inkludere disse eksternalitetene i en samlet evaluering av de samfunnsøkonomiske kostnadene.

Denne boka konsentrerer seg om samfunnsøkonomiske kostnader på et nasjonalt nivå, selv om miljøeffektene analyseres på et lokalt nivå. Vi benytter en generell likevektsmodell for den norske økonomien, som er utvidet til å inkludere miljøeffekter og andre effekter av fossile brensler. Modellen inneholder også tilbakevirkende effekter fra miljøet til økonomien. I fire uavhengige studier blir utvalgte miljø- og andre eksternaliteter analysert ved hjelp av denne modellen.

I kapittel 3 studeres korrosjonskostnader på bygningsmaterialer og biler som følge av luftforurensning. Basert på norske data for luftforurensning, materialbeholdning og vedlikeholdspriser, benyttes dose-respons funksjoner til å analysere vedlikeholdskostnader knyttet til nasjonale utslipp av SO_2. Beregningene for Oslo blir utført ved bruk av en spredningsmodell for luftforurensning, og bygningsregisteret GAB. For andre deler av Norge blir mer generelle metoder anvendt. Til tross for lave utslipp av SO_2 i Norge (i 1994), indikerer beregningene at årlige vedlikeholdskostnader som følge av denne forurensningen er omtrent 200 millioner kroner, hvorav en tredel rammer Oslo. Når disse resultatene blir implementert i den integrerte modellen, øker de samfunnsøkonomiske kostnadene til nesten 300 millioner kroner. Dette skyldes en høyere brukerpris på kapital, som fører til at kapitalnivået faller. Dermed avtar den økonomiske veksten.

Kapittel 4 presenterer beregninger av avlingsskader som skyldes bakkenær ozon i et år (1992) med høye ozon-nivåer i Norge. Kjennskap til ozon-eksponeringen i løpet av vekstsesongen (AOT40) fås på basis av spredningsmodeller og målestasjoner. Basert på geografiske data om plantearealer og avlinger, beregnes tap av hvete, potet og gress (fra dyrket eng). Siden jordbrukssektoren er svært regulert i Norge, er skyggeprisen på

avlingene avhengig av hvordan myndighetene reagerer. To ulike beregninger blir derfor utført. I den ene antas det at avlingstapet kompenseres ved økt import. De direkte kostnadene er da rundt 200 millioner kroner. Når disse resultatene implementeres i den integrerte modellen, blir de totale kostnadene nesten doblet. I den andre beregningen antas det at den innenlandske ressursinnsatsen økes for å opprettholde produksjonsnivået. I dette tilfellet blir de direkte kostnadene ca. 500 millioner kroner, mens de totale kostnadene øker til over 1,2 milliarder kroner. Forklaringen på denne store økningen er at ressurser blir trukket vekk fra andre og mer produktive sektorer i økonomien.

Kapittel 5 analyserer samfunnsøkonomiske kostnader av helseskader knyttet til luftforurensning. Den internasjonale litteraturen om dose-respons funksjoner blir gjennomgått, og det blir dokumentert hvordan disse funksjonene kan bli brukt til å analysere økonomiske virkninger av luftforurensning i Norge. Ved å benytte denne informasjonen blir en egen beregning av helseeffekter og samfunnsøkonomiske kostnader av luftforurensning gjennomført for Oslo. Dette er basert på sammenhenger mellom utslipp og konsentrasjon av partikler (PM_{10}) og NO_2, framkommet ved hjelp av en spredningsmodell. De totale samfunnsøkonomiske kostnader beregnes til 1,7 milliarder kroner. 90 prosent av disse kostnadene er imidlertid knyttet til verdsetting av ikke-produktive effekter (dvs. framskyndet dødelighet og kronisk sykdom). Videre er bare 1 prosent knyttet til tilbakevirkende effekter på økonomien (dvs. 10 prosent av de produktive effektene). Disse effektene er derfor ikke spesielt viktige for helseskader, i motseting til hva analysene i kapittel 3 og 4 konkluderer med.

I det siste kapitlet studeres eksternaliteter knyttet til trafikkulykker. Norske studier av sammenhengen mellom trafikkulykker og drivstofforbruk (og andre forklaringsfaktorer), samt detaljert kunnskap om ulykkeskostnader, blir brukt til å modellere samfunnsøkonomiske kostnader av drivstofforbruk. Virkninger av trafikkulykker på arbeidstilbudet og offentlige utgifter, som følge av dødsfall og personskader, blir analysert. Sammenhengene er videre implementert i den integrerte modellen. Det vises at framskrivninger av BNP i 2020 blir noe redusert, nærmere bestemt med 0,34 prosent, når tilbakevirkningene fra trafikkulykker blir tatt hensyn til. Dette skyldes at trafikkvolumet forventes å øke framover, noe som medfører flere ulykker og dermed en mindre arbeidsstokk enn ved uendret ulykkesfrekvens. Innføring av en CO_2-avgift som stabiliserer utslippene viser seg videre å være mindre kostbar for økonomien når tilbakevirkningene tas hensyn til. BNP blir redusert med 0,44 prosent i 2020, sammenlignet med 0,47 prosent når tilbakevirkningene ignoreres.

Emneord: Fossile brensler, helseeffekter, likevektsmodeller, luftforurensning, samfunnsøkonomiske kostnader, veitrafikk, økomomi-miljø modeller.

Prosjektstøtte: Miljøverndepartementet har gitt finansiell støtte til prosjektet.

Contents

1. Introduction*

1.1. Motivation

There has been a growing awareness over the last decades that economic activity in some respects leads to extensive negative externalities on environmental resources, implying a suboptimal deterioration of the environment. This has called for governmental actions to bring the economy on a more optimal path. Traditionally, economists have favoured market-based instruments like Pigouvian taxes (Pigou 1932), i.e., the polluter must pay a tax corresponding to the marginal damage inflicted on others.[1] Natural scientists, on the other hand, have usually advocated command and control policies, which have often been adopted by policy makers, too. Irrespective of instrument choice, in order to make right decisions one has to know the actual social costs associated with an environmental externality. Then these costs may be compared with the costs of control. In this study we present calculations of the social costs of certain environmental externalities, as well as other externalities related to the use of fossil fuels.

Current economic activity and the state of the environment are in many ways tightly connected. As pointed to above, production and consumption of goods and services may cause pollution, e.g., related to the use of energy. The evolution of the environmental quality therefore depends on the economic development. Simultaneously, pollution is responsible for human and non-human damages, which to some degree is detrimental to the resource base of economic activity. Hence, the economic development may be hampered if the pollution levels come out of control.

These interactions favour integrated analyses of economic and environmental aspects. This point is emphasised in our study of social costs of environmental externalities. Air pollution causes, e.g., various health effects, material corrosion and crop damages, which in turn reduce the actual supply of labour, increase the

* Thanks to Torstein Bye and Nils Martin Stølen for valuable comments on earlier drafts, and to Mona Irene Hansen for valuable research assistance related to all the four analyses in this book. Thanks to Peter Thomas for translating earlier versions (in Norwegian) of chapters 3, 4 and 5. As the chapters have been edited since, the editor is resposible for both the content and the language.

[1] In a seminal paper, Coase (1960) attacks the Pigouvian tradition by emphasizing property rights aspects.

user cost of capital and decrease agricultural productivity. These effects have macroeconomic implications which may be considerable. Hence, the social costs of air pollution may be miscalculated if these macroeconomic feedback effects are ignored.

Nevertheless, whereas the environmental impacts of economic activity are well comprehended, the opposite links are rarely taken into account in studies of environmental damages.[2] Major studies conducted for the European Commission (EC 1995) and the US Department of Energy (ORNL/RFF[3] 1994) analyse external costs of energy production thoroughly using partially integrated analyses, but do not consider the macroeconomic impacts pointed to above.

The environmental damages discussed in this book are all related to air pollution, which for the most part stems from the use of fossil fuels. At the same time, there are other important externalities related to fossil fuels, particularly in the transport sector (e.g., accidents, noise and congestion). Thus, it may be argued that an integrated analysis of air pollution should also focus on these non-environmental externalities, at least when it comes to policy recommendations. Moreover, several of these externalities have detrimental effects on the resource base of economic activity, just like the environmental externalities. E.g., both traffic accidents and transport noise may have negative consequences on the efficient supply of labour. Hence, in calculating social costs of transport-related externalities, one should take a macroeconomic approach.

This book is not aiming at including *all* environmental externalities, not to say all externalities from fossil fuel use. We present studies of four selected externalities, three of them are environmental externalities and the last one is related to traffic accidents. Moreover, even within the specific environmental areas we focus on, there are at all probabilities several effects that are ignored. The reason is that environmental impacts are a complex matter, so that the current scientific knowledge is insufficient to calculate the total social costs of environmental damages. Thus, the four externalities analysed in this book are not selected because they are the most important ones, but rather because of the applicable information that exists for these externalities. This is an important point when interpreting the results in this book.

1.2. Integrated analyses

Integrated analyses have become a popular scientific method, e.g. in the studies of climate change. By integrated analyses is meant bringing together analyses of various parts of a joint problem into one simultaneous analysis. In this book we shall restrict ourselves to discuss such analyses related to social costs of local and regional environmental externalities. In order to calculate these costs in a credible way it is necessary to integrate analyses of natural science and economics. Natural science may provide information about the natural links, whereas economics may provide information about the social costs of certain environmental damages. As the natural links are particularly complex, lack of scientific knowledge has for long time put a restraint on valuing environmental

[2] Bergh (1993) and Rosendahl (1997) are two theoretical exceptions.

[3] Oak Ridge National Laboratory and Resources for the Future.

externalities. Thus, earlier analyses have to some degree been based on expert judgements[4] and control costs[5], which have a more questionably scientific foundation, or on various valuation studies of, e.g., clean air, where the specific impacts are skipped.[6]

The rationale for using integrated analyses as indicated above, has increased considerably the last decade. New research has managed to estimate quantitative relationships between particularly air pollution and various human and non-human damages. These associations are commonly referred to as dose-response functions. Whereas expert judgements, control costs and valuation methods leave little information about the characteristics of the damages, dose-response functions help identifying the specific impacts, e.g., hospital admissions and reduced lifetime of various materials. These functions have been used by the two major studies mentioned above (EC (1995) and ORNL/ RFF (1994)) to calculate the direct external impacts of energy production. Furthermore, the dose-response functions make quantification of feedbacks to the economic resource base possible. Hence, they are natural links in a fully integrated economy-environment model.

Integrated analyses of environmental externalities, using dose-response functions, clearly call for a disaggregated approach. First, the level of emissions of various pollutants depends on the choice of energy use, the choice of combustion technology and substitution possibilities, which vary between different sectors of the economy. Second, the costs of environmental externalities vary with respect to both space and time. For instance, health damages from a certain emission of particulate matter are clearly higher in the middle of the day in a large city than at night or in the countryside. Thus, an integrated model for our purpose should be disaggregated both on the economic and the environmental part.

An important justification for applying dose-response functions is their transparency. However, Stirling (1996) claims that this methodology may not come up with even approximately correct numbers. There are several reasons for this. First, there is a number of uncertainties related to the dose-response functions applied; both to the interpretation of the original study and to the transferability of the results to other locations. However, this uncertainty is partly reduced as the number of original studies grows, and a consensus view is reached. Second, as mentioned above there will always be a chance of overlooking important associations which for some reason have not been demonstrated. Thus, there is an underlying risk of underestimating the total impacts of pollution. Third, given the physical information, an economic valuation will necessarily have to rely on some value judgements, like how to appraise risk, distributional aspects and non-economic impacts in general. However, this problem applies to all methods that intend to calculate social costs of environmental externalities (see section 1.4).

[4] E.g., the social costs of health damage in Alfsen et al. (1992).

[5] The social costs in Hohmeyer (1988) and PACE (1990) were partly based on control costs.

[6] The most common valuation methods are Contigent valuation method (CVM) and hedonic approach method (see Brookshire et al. (1982) for a comparison of these methods).

1.3. An integrated economy-environment model

Although this book presents four *separate* studies, they all apply the same integrated economy-environment model. This model is an extended version of MSG-EE (see Alfsen et al. 1996), which is an applied general equilibrium model for energy and environmental analyses of the Norwegian economy, with inter alia a detailed modelling of the transport sector. In a submodel MSG-EE calculates the national emissions of several air pollutants. The extension of MSG-EE is more or less based on results from the four studies presented in this book. Both MSG-EE and the extended version is further outlined in chapter 2 of this book. Below we give a brief description of how the economy and the environment are connected within the model.

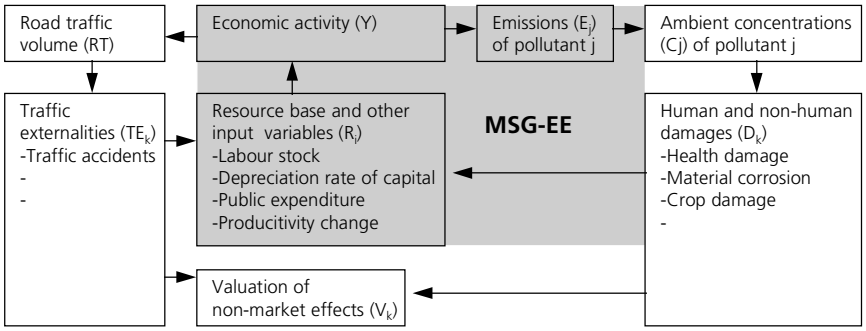
The extended model is illustrated in figure 1.1, where the shaded area is the original MSG-EE model. Economic activity is determined by inter alia the size of the resource base (labour and capital stock etc.) and other input variables. The size and allocation of economic activity determine, through the use of fossil fuels for transport, heating and industrial processes, the national emissions of the various pollutants. In the extended model the national emissions are partly distributed on various geographical locations (main cities etc.), and then the ambient concentrations of different pollutants are determined for these locations. Dose-response functions, as described in section 1.2, are then used to calculate the human and non-human damages of air pollution. Finally, these damages affect the resource base of the economy and other input variables. Thus, we have a simultaneous economy-environment model.

Similarly, economic activity and the transport level are tightly connected, and the extended model calculates the national road traffic volume. This and other variables determine the extent of non-environmental traffic externalities, which in turn affect the basis of the economy. Again, the circle is closed, and the traffic externalities (which in this book are restricted to accidents) and the economic activity are determined simultaneously.

The new information about social costs obtained with this analysis compared to most other externality analyses may originate from two effects. To see this, consider a marginal increase in the emissions of a specific pollutant. Through the concentration and dose-response functions, this increased emission brings about some damages that are valued at fixed prices in traditional analyses. In our model, on the other hand, the costs of the damages also depend on the effects on economic activity, i.e., how the economic equilibrium is changed on the margin through the changes in input variables. As will be seen in some of the chapters of this book, the resulting costs may differ significantly from the direct costs (from small increases to a doubling of the costs).

The other effect is of less importance, but should be included for the sake of completeness. As the economic equilibrium is changed, the total emissions are changed, too, and in the end we arrive at an equilibrium where all the links in figure 1.1 are fulfilled. Since economic activity is negatively affected by emissions as indicated above, and emissions are an increasing function of economic activity, a marginal increase in emissions has a negative feedback effect on total emissions. Thus, this effect dampens the social costs of emissions somewhat.

Figure 1.1. **An integrated economy-environmental model**



However, as the economy after all is very inelastic with respect to emissions, and the elasticity of emissions with respect to economic activity presumably is not higher than one, this effect turns out to be negligible.

1.4. Valuing environmental damages and other externalities

It is useful to separate the valuation of environmental damages and other externalities into market and non-market effects. This is illustrated in figure 1.1. Some damages, which affect elements of the economy, are treated within the model, which chooses the right valuation as well as the feedback effects on the economy. This could, e.g., be corrosion of building materials. Other effects, which do not (merely) have impacts on the economy, are valued in a subsequent model. This could, e.g., be reduced quality of life related to increased morbidity or mortality (which of course may have economic impacts, too). This separation provides that the externalities are treated consistently and transparently.

In most studies of environmental externalities (e.g. EC (1995)) the valuation of a specific damage is made without separating market from non-market effects of the damage. For health damages one either chooses results from a willingness to pay (WTP) study (or other contigent valuation studies), or uses results based on a cost of illness (COI) approach, which intends to measure the lost earnings and medical costs. As WTP estimates are generally assumed to capture the entire welfare cost of the damage, i.e., including the COI estimates, the latter estimates are usually corrected for by a factor of 2. This is based on the results of some empirical studies of specific morbidity endpoints (see the discussion by US Environmental Protection Agency in EPA (1995)). However, as this relationship may differ significantly between different health damages, this should not be done without caution. Moreover, treating COI as a portion of WTP may be wrong in calculating social costs in countries like Norway, where the economic losses of being ill is mainly born by the government. Thus, the two estimates may rather be partly additive.

Valuation methods of non-market effects have been subject to a lot of criticism. One main reason is that objective valuations of, e.g., increased mortality or biological

diversity may not be feasible. Ideally the valuation should therefore be placed on the decision-makers.[7] Moreover, several studies have pointed to major weaknesses of the existing valuation methods.[8] As placing the valuation on the decision-makers may not be practically feasible in all respects, the valuation estimates may be used as indicative numbers which are exposed to alterations. In any case the physical non-market effects should be pointed out.

1.5. Outline of the book

This book presents four separate works on the social costs of externalitities from fossil fuel use in a macroeconomic framework; three of them are concerned with environmental externalities, whereas the last one is concerned with externalities from traffic accidents. In the following a brief outline of each chapter is presented.[9]

Chapter 3, by Glomsrød, Godal, Henriksen, Haagenrud and Skancke, deals with corrosion costs of building materials and cars due to air pollution. Based on Norwegian data on air pollution, material stocks and maintenance prices, they apply dose-response functions to analyse maintenance costs due to national emissions of SO_2. The calculations for Oslo are carried out with the aid of a dispersion model for air pollution, and the GAB building register. For other parts of Norway more general methods have been used. Despite small emissions of SO_2 in Norway (in 1994), the calculations indicate that the annual maintenance costs due to this pollution is about Nkr 200 million, of which one third falls on Oslo. When these findings are put into the model illustrated in section 1.3, the social costs increase to almost Nkr 300 million. This is due to a higher user cost of capital, which implies that the desired capital stock decreases. Thus, the economic growth is dampened.

Chapter 4, by Tørseth, Rosendahl, Hansen, Høie and Mortensen, presents calculations of crop damages from ground level ozone in a year (1992) with high ozone levels in Norway. Information on ozone exposure during the growth seasons (AOT40) is found on the basis of dispersion models and measuring sites. Based on geographical data on crop areas and yields, total loss of wheat, potato and meadow is calculated. As the agricultural sector is very regulated in Norway, the shadow prices of the crops depend on how the government responds. Thus, two sets of calculations are carried out. In one calculation, it is assumed that the yield losses are compensated for by increased imports. Then total direct costs are found to be around Nkr 200 million. When integrating these links into the model above, the total social costs almost double. In the other calculation, it is assumed that the domestic resource use is increased in order to maintain the production level. In this case the direct costs are about Nkr 550 million, whereas the total costs found by using the integrated model is more than Nkr 1.2 billion. The explanation for this big increase is that resources are drawn away from other, and more productive, sectors of the economy.

Chapter 5, by Rosendahl, analyses social costs of health damages due to air pollution. The international literature on

[7] Nyborg (1996) discusses the information requirements that are needed to succeed in this attempt.
[8] Kahneman and Knetsch (1992) point to some important problems with contigent valuation methods (CVM). This is further analysed by Halvorsen (1996), using data from a Norwegian CVM survey. Her findings largely support the criticism.
[9] Alfsen and Rosendahl (1996) give a short presentation of the work behind chapter 3, 5 and 6.

dose-response functions are examined, and it is documented how these functions can be applied to analyse economic impacts of air pollution in Norway. Using this information, a specific calculation of annual health effects and social costs of local air pollution is carried out for Oslo. This is based on relationships between emissions and concentrations for particulate matter (PM_{10}) and NO_2, established by a dispersion model. The total social costs are found to be about Nkr 1.7 billion. However, 90 per cent of these costs are due to valuations of non-market effects (i.e. premature mortality and chronic illness), which may be viewed as particularly debatable as stated above. Moreover, only 1 per cent is attributed to the feedback effects on the economy (i.e., 10 per cent of the market effects). Thus, as opposed to the preceding chapters, this effect does not seem to be very important for health damages.

Finally, chapter 6, by Glomsrød, Nesbakken and Aaserud, considers externalities related to traffic accidents. Norwegian studies on the association between accidents and fuel consumption (and other factors), and a social accounting system for accident costs, are used to model the social costs of fuel consumption related to traffic accidents. Impacts of accidents on labour supply and public expenditure through deaths and injuries are analysed. The links are further implemented in the model illustrated in section 1.3. It is shown that projections of GDP in 2020 are slightly reduced, i.e. by 0.34 per cent, when the feedback effects of traffic accidents are taken into account. This is due to a projected increase in traffic volume, implying more accidents and thus a smaller labour stock than in the case of unchanged frequency of accidents. Moreover, introducing a CO_2 tax to stabilise emissions is found to be less expensive when these feedbacks are accounted for. GDP is reduced by 0.44 per cent in 2020, compared to 0.47 per cent when the feedbacks are ignored.

2. An integrated economy-environment model

Knut Einar Rosendahl

In this chapter we give a description of the integrated economy-environment model that is used in the four studies presented in this book. The core of this model is an applied general equilibrium model for the Norwegian economy called MSG-EE. This model is briefly outlined in section 2.1, emphasizing features that are important for the analyses in the following chapters. A more thoroughly description is given in Alfsen et al. (1996). Then in section 2.2 we describe a version of MSG-EE where the economic model is extended to include links to and from the environment. Figure 1.1 in the preceding chapter gives an illustration of the integrated model, where the shaded area covers the original MSG-EE model.

2.1. MSG-EE: An applied general equilibrium model[10]

MSG-EE (Multi-Sectoral-Growth – Energy and Environment) has been developed by Statistics Norway for *energy and environmental analyses* of the Norwegian economy.[11] Both the choice of industries, commodities and input factors in the model reflect the kind of use of the model. Thus, MSG-EE offers interesting studies of e.g. environmental effects of both various levels and compositions of economic activity.

As energy and environmental issues have a long-term perspective, MSG-EE is based on the theory of economic growth. Thus, increases in the primary input factors (e.g., capital stock and an exogenous labour supply) are the main determinants of the economic development, together with exogenous changes in productivity, see figure 1.1. Producer and consumer behaviour are explicitly modelled based on optimisation principles. Parameters in the utility and production functions are to a large extent based on estimation results from Norway, which are based on data from the National Accounts for the period from 1960 to 1989 (see chapter 3 in Alfsen et al. (1996)).

MSG-EE is a fairly disaggregated model, both with respect to commodities and

[10] This section is to a large extent based on Alfsen et al. (1996).

[11] MSG-EE is a special version of the fifth official generation of the MSG model, originally worked out by Leif Johansen (Johansen 1960). MSG-EE has been used in a wide range of energy and environmental studies, e.g. Glomsrød et al. (1992), Aasness et al. (1996) and Moum (1992).

industries.[12] As the sectors are not equally efficient, this disaggregated industry structure means that the sector composition also affects the aggregate production level. Moreover, the model includes a detailed description of the markets for energy and transport. The disaggregated approach with emphasis on environmentally important sectors is a clear advantage when studying environmental issues, as the emission intensities differ greatly between industries and commodities. Thus, changes in emissions can occur through changes in the input demand as well as changes in the industry structure. However, this requires that the substitution possibilities are well known, both within an industry and between various sectors of the economy.

The production structure for the industries in MSG-EE is illustrated in figure 2.1. At the top level there are five input factors, i.e., capital (other than transport equipment) (K), other materials (V), labour (L), transport (T) and engergy (U):

(2.1)
$$Y = f\{K, L, V, T(K_T, F_T), U(E, F_U)\}$$

These factors are determined according to a constant returns to scale flexible technology. The capital stock is a sector specific Leontief aggregate of eight capital goods, which again are Leontief aggregates of all the basic commodities in the model. Other material inputs are also Leontief aggregates of these commodities.

Transport is divided into five types of transport services, i.e., transport by road, air, rail, sea and post and telecommunication. Each of these services may be purchased in the market from a corresponding transport sector. In addition a significant share of road transport and some sea transport are produced directly by the industries themselves (own transport). The volume of own transport is approximated by the use of transport capital (K_T) and transport fuels (F_T). The amount of own transport in a sector is linked to the amount of commercial transport services by fixed coefficients. As rail transport and post and telecommunication are relatively clean transport technologies, a shift between the five transport sectors in favour of these will contribute to reduced emissions. However, due to data limitations, the compositition of transport services within the industries is exogenous. Still, changes in industry structure may lead to substitution effects at the macro level.

As transport fuels are modelled as input factors to the transport services, oil products used for transport are excluded from the energy aggregate U at the top level of the production function (see equation (2.1)) and figure 2.1. The energy aggregate is used for stationary combustion, and is divided into electricity (E) and fuel for heating purposes (F_U) according to a CES production function with constant returns to scale.

There are several household groups in the model. At the top level, each group allocates total consumption expenditure on 15 consumption goods. At the next level consumption of transport services is divided into private and public transport. Private transport is further divided into petrol and car maintenance, and the stock of cars, whereas public transport is allocated into five transport services. Energy is an aggregate of electricity and fuels (energy demand functions are based

[12] MSG-EE specifies 47 commodities, and the number of industries is 33.

on econometric studies in Norway). Thus, at the bottom line we end up with 22 consumption activities. We see that the choice of activities is clearly relevant for studies of environmental problems. Each of the consumption activities consists of a Leontief aggregate of all the basic commodities. There is no intertemporal behaviour among the households in the model, and total consumption expenditure is assumed to ensure full capacity utilisation in the economy.

In MSG-EE the government receives both direct and indirect taxes (or offer subsidies). The indirect taxes and subsidies vary across sectors and commodities, and affect prices and incomes. A carbon tax is specifically modelled. Moreover, employers' contribution to social security and National Insurance is also included. In addition governmental production is exogenously specified on health care and three other sectors, and the model distinguishes between local and central services.

In a long run equilibrium domestic producer prices are assumed to equal total unit costs. As the production functions have constant returns to scale, unit costs are independent of the scale of production. Thus, the domestic producer prices are only functions of so called primary cost components, which include the wage rate, the user cost of capital, import prices, technological change, indirect tax rates and prices of public services. Both the wage rate and the user cost of capital differ between sectors.

These two cost components are by nature endogenous. The same apply to the trade surplus and the capital stock. However, as the model is not intertemporal, in order to close the model, either the wage rate or the trade surplus have to be exogenous, and either the shadow price of capital or the capital stock have to be exogenous. This choice is left to the model user. In the analyses in this book the trade surplus and the shadow price of capital have been chosen as exogenous variables. According to Alfsen et al. (1996), this closure rule has "been frequently used in normative policy studies of welfare and resource allocation" as "one wants to exclude welfare gains that are financed by increasing foreign debt."

MSG-EE includes several subroutines, and one of them calculates the national emissions of 8 air pollutants based on the use of fossil fuels and material inputs in the various sectors of the economy (see figure 1.1). For our purpose, emissions of particulate matter, NO_x and SO_2 are particularly relevant. 6 different emission sources are identified for each of the production sectors and the private households. Four of them are related to transport combustion (F_T in equation (2.1) for the production sectors) and one is related to stationary combustion (F_U in equation (2.1)). The final source covers the remaining emissions, which are mainly from industrial processes (connected to V in equation (2.1)). The emission calculations are based on exogenous coefficients for each source in each sector. The coefficients are generally linked to certain economic variables in the model, and may change over time due to expected changes in emission intensities.

2.2. MSG-EE with feedback effects from the environment

The extensions in this version of MSG-EE are more or less based on results from the four studies presented in this book, and we will not anticipate these results here. However, we will give a formal description of the general links that are used, as

illustrated in figure 1.1 in the introductory chapter. First, we formalise the connections within the original MSG-EE, i.e. without feedback effects from the environment, with emphasis on variables that are important in this study. As pointed out in section 2.1, the economic development (Y) depends on the development of the resource base and other input factors (R_i), jointly denoted R:

(2.2) $Y = Y(R)$

Whereas the labour stock growth is exogenous, the growth in capital stock depends on the user cost of capital, which is a function of inter alia the shadow price of capital and the depreciation rate. Moreover, productivity changes and public expenditures are other exogenous input factors to MSG-EE. The size and structure of economic activity determine, mainly through the use of fossil fuels, the national emissions ($E^{s,e}_j$) of the 8 pollutants (j), distributed on sector (s) and source (e):

(2.3) $E^{s,e}_j = E^{s,e}_j(Y)$

Statistics Norway collects and calculates emission data for each municipality in Norway, and these emissions are also distributed on pollutants, sectors and sources, in the same manner as the national emissions. Thus, using fixed coefficients for each emission source in each economic sector, calculated in the base year, the extended version of the model distributes national emissions on various geographical locations (main cities etc.) in a fairly detailed way. Then, based on dispersion models and/or measuring sites, the ambient concentrations (C_j) of 4 different pollutants are determined for the same locations:

(2.4) $C_j = C_j(E^{s,e}_j)$

The concentrations of air pollutants lead to various human and non-human damages (D_k), such as health damages, material corrosion and crop damages:

(2.5) $D_k = D_k(C_j)$

These associations are based on dose-response functions, which were discussed in chapter 1. The functions are usually linear. Some of these damages affect central input factors to the economy, such as the labour stock and the depreciation rate of capital:

(2.6) $R_i = R_i(D_k)$

These functions are also generally assumed to be linear, and are based on various national statistics. Thus, summarising equations (2.2) to (2.6) we get:

(2.7) $Y = Y\{R[D(C(E(Y)))]\}$

where the variables must be viewed as vectors. That is, we have a simultaneous economy-environment model.

Similarly, the detailed transport modelling of MSG-EE gives a good foundation for calculating the road traffic volume (RT):

(2.8) $RT = RT(Y)$

which is a main determinant of several non-environmental externalities from road traffic (TE_k):

(2.9) $TE_k = TE_k(RT)$

In this book we only focus on traffic accidents. As for the environmental damages, these non-environmental externalities also affect the input of economic activity, such as the labour stock and public expenditures:

(2.10) $R_i = R_i\ (TE_k)$

Again, the circle is closed, and equation (2.7) may be extended to include equations (2.8) to (2.10):

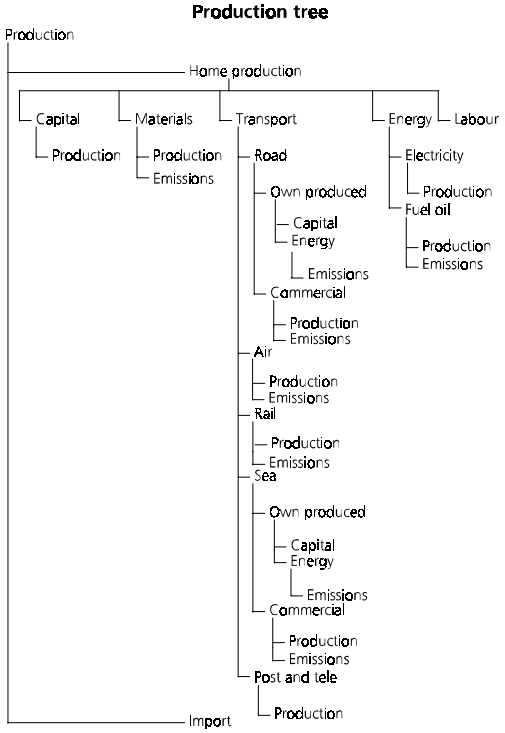
(2.11) $Y = Y\{R[D(C(E(Y))),\ TE\ (RT(Y))]\}$

Thus, in this model economic activity, environmental conditions and traffic accidents are determined simultaneously.

When the input factors of the economy are affected by environmental or traffic-related externalities, prices will change, too, and the structure of the economy changes. Consider e.g. that the labour supply is reduced due to increased sick leaves, either because of air pollution or traffic accidents. Then labour becomes a scarcer resource, and the wage rises. This implies that employers will hire fewer employees, so that the labour market clears. Each industry will generally become less labour intensive, and the industry structure will change. Labour intensive industries will experience higher cost increases than other industries, and will in general diminish. However, demand conditions and the selection of other input factors in production are also crucial, and the final outcome has to be found from the model. As the production of investment products also faces cost increases, the accumulation of capital declines, so that future production capacity is altered, too, even if future sick leaves are not taken into account.

To calculate social costs of externalities by employing this integrated model, we focus on changes in the present value of GDP in addition to the valuation of non-market effects. Using GDP only as a measure of economic costs may however give a biased

Figure 2.1. **Production structure in the MSG-EE model**



result, at least for two reasons in this case. First, when air pollution causes e.g. increased material corrosion and hospital admissions, more economic resources are used for maintenance and health care. However, compared to a situation without air pollution, the value added from this resource use is zero, and should be subtracted from GDP in calculations of social costs. Second, economic welfare is not a function of production, but of consumption. Thus, if investments are increased today at the expense of consumption, GDP will rise in the future, but economic welfare is not necessarily higher. This depends on the marginal utility of consumption today and in the future, and on the relevant discount rate.

Thus, changes in the present value of consumption or, even better, money metric utility, is a better indicator for economic welfare. Whereas the first point is easily handled within the model, the second point is not because the model is not intertemporal.[13] Thus, the relevant discount rate is unknown (see however the study by Aasness et al. (1996) using results from MSG-EE).

[13] In the latest version of MSG (MSG-6), the model is intertemporal (see e.g. Bye (1996)).

3. Corrosion costs of building materials and cars in Norway[14]

Solveig Glomsrød[15], Odd Godal[16], Jan Fr. Henriksen[17], Svein E. Haagenrud[17] and Torstein Skancke[18]

3.1. Introduction

Air pollution causes increased corrosion of building materials and motor vehicles. This entails higher maintenance outlays and increases the user cost of capital. New knowledge and new methodology now make it possible to compute these costs in some detail in Norway. In this study we do this for the year 1994.

The study is based on the use of geographical information systems (GIS), data on local air pollution and distribution of materials at risk. Internationally established relations between air pollution and degradation of various materials are also employed. Full use is made of GIS for Oslo. Estimates for the rest of the country are done by extrapolation adjusted for pollution levels and stocks of materials. The project quantifies both direct maintenance costs and the feedback-effects of such costs in the economy as a whole when building capital becomes more expensive for enterprises and households (indirect costs). The study is a following up on Glomsrød and Rosland (1988), who made similar calculations for the year 1985.

In later years new and improved descriptions of the link between concentration of air pollution and the decomposition rate for various materials have been developed internationally. Moreover, new data on building materials have emerged which enable more precise computation of material stocks at risk.

In section 3.2 we present the quantitative relations between concentrations of air pollution and materials degradation. Air pollution levels and the volume of materials involved are described in section 3.3 and 3.4 respectively, while in section 3.5 corrosion rates and total maintenance costs resulting from air pollution are computed. Section 3.6 explains the marginal costs of increased SO_2 emissions. The effects these have for the national economy are elucidated in section 3.7.

[14] This study was commissioned by the State Pollution Control Authority (SFT) and carried out jointly by the Norwegian Institute for Air Research (NILU), the NORGIT Centre and Statistics Norway. The artickle has earlier been published in Norwegian in Glomsrød et. al. 1996.

[15] Statistics Norway

[16] CICERO (Statistics Norway at the time of the study).

[17] Norwegian Institute for Air Research

[18] NORGIT-center

3.2. Dose-response and lifetime functions for some materials

3.2.1. Current knowledge of dose-response functions

Dose-response functions (see e.g. Lipfert (1987)) describe the physical/chemical relations between materials degradation and exposure to pollution. When calculating corrosion damage these must be translated to capital degradation in economic terms. The usual approach is to set a criterion for how far corrosion can proceed before maintenance or replacement of a building component has to be carried out. Use of dose-response functions enables us to calculate to which extent the lifetime of building elements is affected by increased pollution levels. The dose-response function is thus transformed into a *damage function*.

In the past decade numerous corrosion studies have been carried out with respect to dose-response and damage functions, material stocks and exposure conditions, see e.g. Haagenrud and Henriksen (1995). With respect to dose-response functions, three studies are particularly prominent:

Lipfert (1987) has performed a synoptic statistical analysis of environmental and corrosion measurements for important metals covered in eight international test programmes from up to 72 field stations. Lipfert has carried out a similar survey of calcareous stone materials. Dose-response functions are also given for types of paint coatings.

Two studies carried out by Henriksen et al. (1981) and Haagenrud et al. (1984) contain highly important basic data for Norway in terms of dose-response functions for metals. Good statistical analyses are available for two Norwegian towns (Sarpsborg and Fredrikstad), but more detailed and synoptic analyses of all data sets have yet to be carried out.

The most extensive and best documented database for dose-response functions is the ECE-ICP base. The 8 year research program on which it is based, is not yet completed, but preliminary results are available. Equations for corrosion development over time have not been developed. However, the ECE-ICP base contains descriptions of degradation as a function of SO_2, O_3 and H^+ within a geographical area covering the greater part of Europe. It also encompasses considerably more materials than previous surveys.

Examination of the dose-response functions shows that fairly reliable functions exist for many important building materials such as metals, painted metal, calcareous stone and the like. The functions contain terms describing the effect of SO_2, and where relevant also O_3, H^+ concentration in precipitation and climate variables expressed as time of wetness (TOW). Time of wetness is defined as the part of the year with relative humidity higher than 80 per cent and temperature higher than 0°C.

3.2.2. Lifetime functions for materials

When damage functions are elaborated, account is taken of how far degradation can proceed before maintenance or replacement is necessary. In practice there is a large difference between standard exposure tests and substantive effects on buildings. It is assumed that maintenance or replacement is only based on the state of the materials, and not on other factors such as economic value. Damage functions can be determined *directly* by field inspection through visual description of

the state of wear and tear and actual damage to buildings, or *indirectly* by recording maintenance performed at regular intervals. When the optimal interval for maintenance or replacement is determined, the damage function is usually termed the *lifetime function*.

Lifetime functions are as a rule dominated by the most aggressive pollutant. Several studies have developed lifetime functions for building materials. A comprehensive statistical sample of different houses in various pollution areas has been analysed by Kucera et al. (1993). This study, known as the MOBAK study, is the most comprehensive of its type and contains results from Prague, Stockholm and the Norwegian town Sarpsborg. Based on this study, results have been extrapolated to the national level in Sweden (Andersson 1994), and to the European level (Cowell and ApSimon 1994). Lifetimes and maintenance intervals as a function of various SO_2 levels are available for many building materials. Using extrapolation techniques, Andersson (1994) has also introduced acid precipitation sensitivity (H^+) in these functions when calculating material costs in Sweden.

Thus, lifetime functions may be arrived at either directly from inspection of buildings or from dose-response functions. In the latter case degradation (D) is described using linear dose-response functions including pollution parameters as a degradation factor. The general formula used in our calculations is:

$$(3.1) \quad D_1 = a_1 \cdot SO_2 + b_1,$$

or

$$(3.2) \quad D_2 = a_2 \cdot TOW \cdot SO_2 \cdot O_3 + b \cdot Rain \cdot H^+ + c_2$$

where a, b and c are constants, SO_2 and O_3 concentrations are measured in $\mu g/m^3$, H^+ concentrations in mg/l, and Rain is measured in meter precipitation per year. Degradation is here measured in thickness reduction per year.

To arrive at lifetime functions, we note that lifetime (L) is inversely proportional to degradation. For most materials a lifetime function of the following type is employed (based on the first dose-response function, equation 3.1):

$$(3.3) \quad L_1 = 1/[a \cdot 10^{-3} \cdot SO_2 + b \cdot 10^{-3}] = 1000/[a \cdot SO_2 + b]$$

These are taken directly from Anderson (1994). However, for zinc and copper the dose-response functions from the ECE project are employed (i.e., equation 3.2), and the following lifetime function is arrived at:

$$(3.4) \quad L_2 = m/[a \cdot TOW \cdot SO_2 \cdot O_3 + b \cdot Rain \cdot H^+ + c]$$

where m is reduction in thickness in micrometer (μm) before maintenance or replacement is recommended. Table 3.1 shows the selected or derived lifetime functions for 14 materials that are used in this study. In addition, lifetime functions exist for 3 other materials that are excluded because of lack of material stock data.

Regarding zink, for galvanised sheets and wire where the mean thickness of zinc is 30μm, the premise has been that repainting should be carried out after m=20μm has corroded, while replacement should take place when all zinc (m=30μm) has gone. For galvanised profiles with a mean thickness of 80μm, painting should take place when m=60μm has corroded.

Table 3.1. **Lifetime functions for materials at risk**

Material name	Lifetime function, year
Galvanised steel sheet, replacement	$L = 30 / (0.51 + 0.0015 \cdot TOW \cdot SO_2 \cdot O_3 + 2.82 \cdot H^+ \cdot Rain)$
Galvanised steel sheet, maintenance	$L = 20 / (0.51 + 0.0015 \cdot TOW \cdot SO_2 \cdot O_3 + 2.82 \cdot H^+ \cdot Rain)$
Galvanised steel wire	$L = 30 / (0.51 + 0.0015 \cdot TOW \cdot SO_2 \cdot O_3 + 2.82 \cdot H^+ \cdot Rain)$
Galvanises steel profile	$L = 60 / (0.51 + 0.0015 \cdot TOW \cdot SO_2 \cdot O_3 + 2.82 \cdot H^+ \cdot Rain)$
Copper roofing	$L = 100 / (0.54 + 0.00031 \cdot SO_2 \cdot O_3 + 4.58 \cdot H^+ \cdot Rain)$
Strip-lacquered aluminium	$L = 1000 / (0.107 \cdot SO_2 + 32.6)$
Strip-lacquered galvanised steel	$L = 1000 / (0.155 \cdot SO_2 + 38.6)$
Painted galvanised steel	$L = 1000 / (0.803 \cdot SO_2 + 84.5)$
Limestone/Cement plaster	$L = 1000 / (0.124 \cdot SO_2 + 15.7)$
Painted plaster	$L = 1000 / (0.278 \cdot SO_2 + 19.9)$
Felt roofing	$L = 1000 / (0.327 \cdot SO_2 + 48.9)$
Painted/Stained wood	$L = 1000 / (1.03 \cdot SO_2 + 91.4)$
Brick	IF (SO_2<10 then 70 years else 65 years)
Concrete	IF (SO_2<10 then 50 years else 40 years)

Sources: Anderson (1994), ECE-ICP base and Heinz et al. (1995).

The corresponding equation for copper is determined by how much a copper sheet can corrode and still be in functional order. Copper sheets for roofing and frontages are currently 0.5-0.7mm thick. Due to the unevenness of corrosion, and impaired strength resulting from corrosion, replacement is recommended when 0.1mm (m=100μm) of the sheet has been corroded.

Concrete has long been by far the most used construction material and is therefore of major economic significance. Concrete breaks down more rapidly in an industrial and urban atmosphere than in an unpolluted atmosphere. Since the reasons for this are very difficult to clarify, it has not been possible to arrive at good dose-response functions or damage functions. This is because concrete is a highly complex and complicated material. It is porous, contains a number of additives, and the water/cement mix is in itself of great significance. Several environmental variables and other mechanisms influence degradation. The environmental factors are carbonation, temperature fluctuations, dampness, chlorides (sea salt, road salt), atmospheric pollutants (SO_2 and NO_2) and solar radiation. Where reinforced concrete is concerned, carbonation is of greatest interest, i.e. the reaction between CO_2 and concrete.

Despite the lack of good dose-response functions, as concrete constitutes a substantial share of the materials in all building categories in the survey (see table A2 in appendix A), it has been considered more important to include concrete in the calculations than to omit it on grounds of uncertainty. The lifetime of concrete is specified as follows for background and corrosive atmosphere respectively (Heinz et al. 1995). Maintenance/lifetime in the background atmosphere (defined as SO_2 concentrations below 10 $\mu g/m^3$) is assumed to be 20-80 years, and in corrosive atmosphere (SO_2 concentrations above 10 $\mu g/m^3$) 10-70 years. This averages out to 50 and 40 years respectively. Hence in our context we have chosen to use a stepwise lifetime function (see table 3.1).

Brick is also a complicated material that is porous and contains many different ingredients in varying mixes. A number of degradation mechanisms are present, and, as in the case of concrete, it is difficult to determine dose-response or damage functions. In the same way as for concrete, the German study by Heinz et al. (1995) has carried out practical studies of lifetime, which is generally longer than for concrete. Using the same method as for concrete we arrive at the lifetime function in table 3.1.

3.3. Air quality

Calculating material corrosion due to air pollution requires a quantitative description of air quality. The concentration of an air pollutant depends not only on the emission level in that area but also on emissions in other areas combined with meteorological variables. The best method for establishing the concentration at a location is to measure it. Since there are practical constraints on measuring concentration at all locations, one is dependent on calculating concentrations away from measuring stations to obtain a good picture of the extent to which, for example, building materials are subjected to air pollution.

3.3.1. Modelling pollution in the Oslo area

The level of pollution in Oslo is modelled using NILU's (Norwegian Institute of Air Research) dispersion model AirQuis Models based on the emission database AirQuis Emissions (Grønskei and Walker 1993). The model calculates the concentration of SO_2 and NO_2 in grid comprising 44x36 (=1 548) squares of 500x500 m^2, over a selected time period, where account is taken of emissions from heating and vehicle traffic as well as factors such as wind and temperature. A regional contribution is also included which is based on measurements outside Oslo. Our calculation of material corrosion costs was done for the year 1994. Measurements and calculations from previous studies provide the basis for emission data in each square: Data for NO_2 emissions are based on 1991 figures (Gram 1994) and for SO_2 on 1979 figures (Gram 1982). The SO_2 data were subsequently adjusted in 1987 (without compiling new basic data) based on known energy consumption in Oslo and on known reductions in industrial point sources in the preceding few years.

When calculating the lifetime of materials, grid values must be transformed to average annual concentrations (annual means). In order to minimise uncertainties attached to transformation, the dispersion estimates must represent the mean distribution of pollutants over the year. This was done by selecting a scenario where meteorological conditions are representative for the year. With a basis in a reliable dispersion estimate, the transformation to annual values based on the results from two measuring stations in the centre of Oslo (i.e., Johannes Bruns gate and Hausmanns gate) will produce good annual values for all squares in the calculation. The mapping of individual sources is poorer for the SO_2 database than for the NO_2 database, whereas the total values for SO_2 emissions and NO_2 emissions are approximately equally accurate. Hence in the present work we employ total SO_2 emissions, while NO_2 emissions are distributed on the individual sources heating, vehicle traffic and background, and distributed on the grid on the basis of each group's contribution in the square.

Formation of ozone in the troposphere, i.e. ground-level ozone, is a complex

process containing several combinations of chemical reactions. The outcome of these reactions is ozone formed in the presence of hydrocarbons with NO_2 as a catalyst. In the presence of a large surplus of NO, ozone is reduced to oxygen. There is a large surplus of NO in areas of high vehicle traffic density. Hence in practice ozone levels are lower in town centres and higher in the areas surrounding towns. Kucera et al. (1995) present results of a four-year comparative measurement in several towns between NO_2 and ozone levels at the same sites. They find that the following equation gives the best description of this relation:

$$(3.5) \quad O_3 = 60.5 \exp^{-0.014 \cdot NO_2}$$

with concentrations measured in $\mu g/m^3$. This equation has been used to estimate an ozone value in each square in Oslo, based on the corresponding grid values for NO_2.

We assume that the variables rain, time of wetness (TOW) and acidity of precipitation (H^+) remain constant across various parts of Oslo. For these variables it has been decided to use data from a UN-ECE station in the centre of Oslo (i.e., Hausmannsgate) as a mean for the period 1987-1993 (see table A1 in appendix A).

3.3.2. Data for the rest of the country

In estimating material corrosion costs for the rest of the country, we have chosen to single out 15 towns and urban areas (in addition to Oslo). These are locations where either high SO_2 concentrations or a large building stock are present, and hence where a high proportion of the total material corrosion costs are likely to be incurred. The rest of the country is divided into three groups (see table 3.2).

For each area data for the annual mean SO_2 and ozone concentration and values for pH, precipitation and time of wetness (TOW) are required. These parameters are shown in table A1 in appendix A.

As opposed to the pollution data for Oslo, we only use one representative value for the other towns. Thus, the calculations for these towns are not as precise as the calculations for Oslo. However, the variation in air pollution is probably higher within Oslo than within most other towns. Moreover, an earlier study by Glomsrød and Rosland (1988) indicated that one third of the Norwegian material corrosion costs are incurred in the capital.

The values for SO_2 are annual means taken from NILU's monitoring programme for the SFT (Norwegian Pollution Control Authority) for towns and urban areas (Hagen 1994) and the SFT's own monitoring programme in the industrial region Grenland. Ozone and pH in precipitation are based on data from SFT's monitoring programme for long-range air pollution (SFT 1994). Data for precipitation are taken from The Norwegian Meteorological Institute's tables for the past 30 years' mean (Aune 1993; Førland 1993), and time of wetness data are recorded by NILU's own stations which measure temperature and relative humidity (Ofstad 1995). Where measurement data are not available, data are generated by extrapolation from the closest available data sets.

The three remaining groups are urban areas[19] in the southern part of Norway (i.e., from Sør-Trøndelag and southwards), urban areas in the northern part

[19] Urban areas with more than 2 000 inhabitants excl. those already included in the analysis.

of Norway, and the rest of the country (mainly rural). In the north the values for sulphur dioxide, ozone, rain, time of wetness and acidity of precipitation for Tromsø were used for all towns and urban areas. In the south, 3 $\mu g/m^3$ for SO_2 and 55 $\mu g/m^3$ for O_3 were chosen for all towns and urban areas. For rain, time of wetness and acidity of precipitation the arithmetic mean values from the 15 selected towns and urban areas in Southern Norway were used. Account has not been taken of the fact that precipitation is higher in western Norway, as this is only significant for calculations for zinc and copper. For the rest of the country, an SO_2 concentration of 1 $\mu g/m^3$ (which is equal to the background concentration – see the next section) is chosen. The other data are similar as those for urban areas in the south.

3.3.3. Local and long-range contributions to pollution

The long-range contribution to SO_2 concentration in Norway is at present about 1 $\mu g/m^3$. The remainder is due largely to local combustion of petroleum products. Only in the vicinity of certain industrial enterprises will process emissions dominate (e.g., in Sarpsborg and Skien).

Ozone formation is as stated more complex and the local contribution is difficult to estimate. Several calculations have been made to quantify ozone levels without (local and long-range) contributions from air pollutants, and 40 $\mu g/m^3$ appears to be a figure acceptable to a number of researchers (Bojkov 1986). However, most of the difference between this background level and the actual ozone concentrations in Norway is due to long-range contributions. Still, in lack of other estimates we will regard the exceeding of 40 $\mu g/m^3$ as a local contribution. This is clearly a major simplification. However, as will be seen in section 3.6, ignoring local contributions of ozone reduces the locally imposed costs by only 1.4 per cent. Thus, pollution data on SO_2 are clearly the most crucial.

For NO_2, which affects the ozone levels, background concentrations in Norway are 3-4 $\mu g/m^3$. The remainder is attributable to local emission sources where vehicle traffic dominates over heating. The percentage distribution between vehicle traffic and heating will vary from square to square in Oslo and from town to town in Norway.

For pH in precipitation the local contribution is minimal, and in the calculations all acidification has been assumed to be related to long-range pollutants.

3.4. Stock of materials at risk

3.4.1. Method

There exist no complete statistics on outdoor use of materials in Norway. We are therefore consigned to *estimate* the stock of various materials at risk. Several methods are available for this purpose. Glomsrød and Rosland (1988) tried to calculate the total amount of building materials based on the volume of domestic production over a given period adjusted for imports and exports. Since then, information has emerged that makes other types of analysis possible.

In this study we start out from investigations that have been made on the use of materials in varous buildings and infrastructure. An important contribution has been the results of the comprehensive MOBAK study undertaken in Stockholm, Prague and the Norwegian town Sarpsborg (Kucera et al. 1993). Thorough

fieldwork was carried out on surveying the condition and material-mix of various types of buildings. One of the conclusions was that the mix of materials for various types of buildings in Sarpsborg and Stockholm was almost identical whereas a different mix was found in Prague. One explanation given for this result was that the Nordic countries share a similar architectural tradition. This may in turn be due to the relatively uniform availability of various types of building materials and the similarity of climate. Hence it was considered that the mix of materials in buildings in Sarpsborg could be extrapolated to the rest of Norway without major margins of error.

A number of materials not classified as buildings are subjected to corrosion caused by air pollution. Part of this stock of materials can be classified under the term infrastructure. We have included contributions from wire fencing around buildings, high-voltage transmission lines for electricity and railways, lampposts, traffic signs and the motor vehicle population under this term. The material-mix for these items is also based on the MOBAK study. Table A2 in appendix A shows average size and material-mix for each building category.

Now, the following procedure shows how we will calculate the stock of materials at risk, where point 2 is based on the results of the MOBAK study:

1. Derive total area of building materials for various building categories and infrastructure (dwellings, manufacturing, agriculture, office buildings, service buildings and motor vehicles) in each location.
2. Make use of the recorded mix of materials for an average building of each main type.

In the following sections we will describe how we have derived at the figures outlined in point 1.

3.4.2. Stock of materials at risk in Oslo

In Oslo we have used the building register, GAB (the Norwegian acronym for: Properties, Addresses, Buildings), to obtain information on point 1 above. GAB records all buildings exceeding 15 sq.m. throughout the country. The register includes detailed information on the geographical location as well as the building's purpose. The register has made it possible to distribute the building stock on a grid covering Oslo whose square size matches that of the pollution parameters (500m x 500m). Within each square we have information on the number of buildings distributed by purpose.

Retrieving information from this register is highly resource-demanding. Hence we have had to confine ourselves to Oslo and then employ various extrapolation techniques to arrive at national estimates. As mentioned above, Oslo was a natural choice since previous estimates (Glomsrød and Rosland 1988) showed that a third of the national material costs are incurred in Oslo, where both the concentration of buildings and air pollutants are high.

3.4.3. Stock of materials at risk outside Oslo

In regard to geographical distribution outside Oslo, we follow the same division as in section 3.3.2. In order to extrapolate from Oslo to other regions, we have to know what proportions of the various types of building are to be found in Oslo

compared with the other municipalities. Such information can be retrieved from various statistics containing municipal data. Stocks of material in the various municipalities are then estimated as follows.

Dwellings, which include all private household buildings, are divided into small houses and apartment blocks, each showing a different geographical distribution. Apartment blocks are more heavily represented in towns than in peripheral districts. They will therefore, relatively speaking, incur higher corrosion costs. Figures from the Population and Housing Census 1990 (Statistics Norway 1992a) show the number of different types of housing distributed by municipality. The Survey of Housing Conditions 1988 (Statistics Norway 1990) shows the average size of each dwelling type. Using these statistics we have arrived at a municipal distribution of the housing capital.

For distribution of materials in industrial buildings (mining/manufacturing), figures have been taken from Manufacturing Statistics 1992 (Statistics Norway 1994a) stating the fire-insurance value of industrial buildings at municipal level. Our assumption is that the distribution of material stocks follows the distribution of fire-insurance values of the buildings. For office/commercial/transport, hotel and restaurant, and public and private services, the choice of distribution is by number of employees in the respective trades as stated in the Population and Housing Census 1990 (Statistics Norway 1992a).

In primary industries, i.e. agriculture, forestry, fishing etc., farm buildings predominate. We have therefore taken a basis in the Census of Agriculture and Forestry for 1989 (Statistics Norway 1992b) which states the floor area of outbuildings (1000 sq.m.).

Buildings that are not specified in the GAB register (called other buildings), are mainly attached to small houses. Hence these buildings have been assigned the same geographical distribution as small houses.

For infrastructure we have calculated the average stock of materials per capita and distributed the result on towns/regions in relation to population. For Oslo we have calculated the total stock of materials using the population figure and then distributed the result on the grid in proportion to the building area. In this way the city centre, which contains a relatively large amount of infrastructure per capita, is assigned a larger share of materials. Since it has not been possible to apply such considerations to the rest of the country, distribution of material stocks has been carried out directly based on population.

Distribution of material stocks for all types of buildings in the areas 'urban south', 'urban north' and the 'rest of the country' has been done using population figures. The resulting keys used to distribute building capital among regions are set out in table A3 in appendix A. Table 3.3 in section 3.5.4 shows the stocks of various building categories for the whole country. Based on this information, distribution of material stocks in the various regions and on various types of buildings are calculated (see tables A4 and A5 in appendix A).

3.5. Maintenance costs

3.5.1. Assumptions

Substantial maintenance is carried out on exterior surfaces of buildings since the materials making up the frontage undergo a continuous process of degradation. This process can, for our purposes, be split into two main components: Natural degradation which takes place irrespective of air quality, and degradation due to emission of pollutants into the atmosphere.

The owner of the building is him/herself in a position to decide when maintenance is to be carried out. A reasonable assumption is that owners wish to minimise total outlays on maintenance of the building over time and will therefore actively adapt maintenance frequency to maintenance needs. We also assume that owners know when maintenance should be carried out with a view to minimising costs.

When calculating costs of corrosion of building materials it is not enough to consider the actual cost of maintenance work. The maintenance that would have been required in a "clean" immediate environment with natural corrosion (background corrosion) must be subtracted to obtain a correct estimate of material costs due to air pollution.

Our calculations incorporate two main assumptions about agents' information and patterns of behaviour. First, we have assumed that the owner of the building knows when maintenance of the building should be performed in order to minimise costs in the long term and that he/she performs the work at this point. Second, as mentioned earlier, we disregard other motives behind replacement or maintenance work than material degradation. If the agent performs maintenance more often than required by the criterion of physical wear and tear, e.g. because of aesthetic reasons or because the *economic* lifetime is shorter than the *physical*, air pollution may not alter agents' maintenance frequency. If this frequency is higher than optimal maintenance frequency based on technical criteria, air pollution will not result in increased costs for the owner. On the other hand, for owners with an initially optimal maintenance frequency who do not adapt this frequency to the increased wear and tear caused by air pollution, the long-term costs may exceed our calculations. Information from the construction industry suggests that agents are more concerned with adapting building maintenance to budget constraints than to maintenance needs. The sum total of these sources of uncertainty resulting from lack of information gives no clear indication as to its effect on costs.

3.5.2. Model for calculating costs

We will define the material corrosion costs due to national air pollution (K^p) as the difference between the total corrosion costs (K^t) and the corrosion costs that would occur in the background atmosphere (K^b):

$$(3.6) \quad K^p = K^t - K^b$$

Moreover, we can state the corrosion costs of a given material j as an expression of the total stocks of the material (M_j), maintenance costs per square metre of the material (C_j), and the lifetime of the material (L_j). The lifetime of the various materials differ between various environments as discussed in section 3.2.2 (see table 3.1). Thus, we have the following two expressions for corrosion costs of material j in, respectively, the background

atmosphere (b) and the actual atmosphere (t):

$$K_j^b = \frac{M_j \cdot C_j}{L_j^b} \quad (3.7)$$

$$K_j^t = \frac{M_j \cdot C_j}{L_j^t} \quad (3.8)$$

The numerator indicates the cost of cutting the lifetime of the material by one year. Inserting (3.7) and (3.8) into (3.6) we obtain

$$K_j^p = M_j \cdot C_j \cdot \left(\frac{1}{L_j^t} - \frac{1}{L_j^b} \right) \quad (3.9)$$

This expression may be summed over all materials, and over all regions.

3.5.3. Maintenance prices

For most materials in the analysis maintenance is in the form of cleaning and repainting. Only galvanised steel wire, roofing felt and copper roofing are invariably replaced. For galvanised steel sheeting we assume that 50 per cent is maintained and 50 per cent replaced. The prices of maintenance and replacement of materials depend inter alia on the extent of damage, building size and shape, design, material quality and choice of contractor. The data, based on information provided by the industry, are presented in table A6 in appendix A.

Part of the building maintenance work done to remedy material corrosion is performed by the owner in person, and in such cases the maintenance work is not a service traded in the market. Still, we assume that the market price is the best estimate to use here, too.

Table 3.2. **Material corrosion costs by region in 1994. Mill. 1995-Nkr**

Region	Costs	Per cent
Halden	1.8	0.9
Sarpsborg	5.4	2.7
Fredrikstad	2.4	1.2
Moss	1.6	0.8
Bærum	7.1	3.6
Asker	2.6	1.3
Oslo	74.4	37.6
Drammen	3.8	1.9
Porsgrunn	2.1	1.1
Skien	13.2	6.7
Bamble	0.5	0.2
Kristiansand	2.1	1.1
Stavanger	8.6	4.4
Bergen	22.6	11.4
Trondheim	9.2	4.7
Tromsø	0.8	0.4
Urban-south	34.8	17.6
Urban-north	3.6	1.8
Rest of the country	1.3	0.7
Total	197.7	100.0

3.5.4. Cost calculations

From sections 3.3 and 3.4 we have data on, respectively, air quality and stocks of each individual material distributed by building type. These data are given for each of the 1 584 squares of Oslo as well as for the other 18 towns/areas. Actual lifetime and lifetime in background atmosphere of each material in each geographical location is then calculated as described in section 3.2.[20] By applying the maintenance prices shown above, material corrosion costs are calculated for each material and geographical location using equation (3.9). The results are set out in table 3.2 to table 3.4, where the total costs are distributed on region, building type and material type, respectively. More

[20] With 1 584 squares, 9 building types and 14 types of material, the total costs in Oslo is aggregated from 199 584 separate cost units.

Table 3.3. **Stocks and corrosion costs of building types for the whole country, 1994. Mill. 1995-Nkr**

Building	Area, mill. m^2	Per cent	Corrosion costs	Per cent
Small house	282.1	33.0	42.7	21.6
Apart.block	107.1	12.5	47.1	23.8
Manufact.	24.5	2.9	4.8	2.4
Office	191.2	22.3	46.4	23.5
Hotel	1.7	0.2	0.5	0.2
Service	27.6	3.2	6.7	3.4
Agriculture	128.0	15.0	10.8	5.5
Other	50.4	5.9	8.8	4.5
Infrastr.	43.1	5.0	30.0	15.2
Total	855.6	100.0	197.7	100.0

Table 3.4. **Total material stocks, material corrosion costs and costs per square meter, 1994. Mill. 1995-Nkr**

Material type	Total material stock, mill. m^2	Per cent	Total costs	Per cent	Average cost, Nkr/m^2
Galvanised steel, untreated					
sheet maint.	15.3	1.8	11.2	5.7	0.74
sheet repl.	15.3	1.8	13.7	6.9	0.90
wire	5.2	0.6	1.9	1.0	0.36
profile	3.0	0.4	1.6	0.8	0.52
Galvanised steel, treated					
str.-lacq.	64.9	7.6	5.0	2.5	0.08
painted	67.0	7.8	51.1	25.8	0.76
Aluminium, str.-lacq.	28.9	3.4	1.5	0.8	0.05
Copper	1.3	0.2	0.3	0.2	0.25
Wood, stained/painted	269.9	31.5	53.6	27.1	0.20
Plaster, untreated	28.4	3.3	5.0	2.5	0.18
Plaster, painted	71.9	8.4	22.3	11.3	0.31
Concrete	97.1	11.3	14.2	7.2	0.15
Roofing felt	82.4	9.6	14.6	7.4	0.18
Brick	105.1	12.3	1.6	0.8	0.02
Total	855.6	100.0	197.7	100.0	0.23

detailed results are shown in tables A7 and A8 in appendix A.

The geographical distribution of the costs shows that Oslo incurs more than one-third of the costs even though only 12 per cent of the stock of materials at risk is located here. This is due to a high concentration of SO_2. On the other hand, the category 'rest of the country' which has about 40 per cent of the stock of materials, carries less than one per cent of the costs because of the low pollution level in this category (the SO_2 concentration is set equal to the background concentration, and so the costs are only due to ozone concentration). The distribution of costs on building types shows that dwellings (small houses and apartment blocks) incur almost half the costs,

whereas e.g. the manufacturing buildings only incurs 3 per cent of the costs.

From the distribution of costs on materials we see that metals' share of total costs is larger than metals' share of total material stocks. The reason is that metals are the most sensitive materials to SO_2 pollution. This is particularly reflected in the category 'infrastructure' which accounts for 15 per cent of the costs but only 5 per cent of the material stock. A quarter of the costs can be ascribed to painted or stained wood. This is not a sensitive material, but the stock at risk is large. Brick and concrete incur costs in only three towns (see table A7 in appendix A). This is because the lifetime functions for these materials are assumed to be stepwise functions which are only triggered where SO_2 concentration is higher than 10 $\mu g/m^3$.

3.6. Marginal corrosion costs of SO_2 emissions

In public decision-making related to this issue it will be useful to know the marginal social costs of more emissions due to increased material corrosion, or in other other words the marginal gains of reducing emissions. Then this and other environmental gains may be compared to the marginal costs of implementing an emission reduction initiative. In this chapter we therefore calculate the marginal costs of SO_2 emissions due to material corrosion.

Systems for monitoring air quality and development of dispersion models show that for SO_2 the link between emission and concentration may be well described by a linear relationship. Furthermore, as the main part of SO_2 concentrations are currently caused by local emissions, we assume that, except for the background level, SO_2 concentration in a municipality depends exclusively on emissions in this municipality. Thus, we do not consider transport of emissions between municipalities. This is a reasonable simplification for most of the areas we study. Moreover, we shall assume that the contribution to SO_2 concentration of a tonne of SO_2 emitted in a municipality is independent of the source of the emission. This assumption is tenable if particularly large changes in point sources are disregarded. A percentage increase in emissions will then produce the same percentage increase in concentration adjusted for the background level.

A corresponding relation between ozone concentrations and NO_x emissions is far more complicated, since NO_x is involved both in the formation and decomposition of O_3. While decomposition predominates in town centres, ozone formation is the rule in the outskirts of towns away from the main traffic arteries. Since reliable quantitative descriptions of these effects are not available, we have been unable to calculate marginal costs for nitrogen emissions.

It is easily seen from equation (3.9) that for 7 of the 14 materials in table 3.1, which constitute 77 per cent of the total costs, the relation between SO_2 concentration and costs is linear. This is because material lifetime is inversely proportional to the concentration of SO_2, and costs are inversely proportional to lifetime. For concrete and brick, which constitute 8 per cent of the costs, the stepwise functions imply that the marginal costs are zero except at the point where concentrations pass 10 $\mu g/m^3$, where marginal costs are infinite. Of course, this is a pure simplification, and we will assume in this section that the marginal costs are equal to the average costs in each region. This is

equivalent to assuming that the derived costs were obtained from a linear function of the concentration. For the last 5 materials the multiplicative ozone concentration term in the lifetime functions complicates the matter as the marginal costs of SO_2 concentrations depend on the level of the ozone concentrations. In our calculations we shall assume that the ozone concentrations are fixed at the current level.[21] Then the relation between SO_2 concentrations and costs is linear for these materials, too.

The easiest way to calculate marginal costs of SO_2 emissions is now to split the total corrosion costs for each region into two components, where the first component is the corrosion costs with SO_2 concentrations at its background level. These costs are for some materials equal to zero, for others solely due to ozone concentrations. Then the second cost component is the contribution from SO_2 concentrations when ozone concentrations are at the current level. From the discussion above we established that this cost component is proportional to the concentration levels (in excess of the background levels). Since emissions are proportional to these same concentration levels, the second cost component can be directly divided by the emission levels for each region in order to calculate marginal costs, i.e. the marginal costs equal the average costs of this component.[22]

SO_2 emissions distributed at municipal level by emission source are available (Statistics Norway 1995a). The figures, set out in table 3.5, are exclusive of emissions from international air traffic above 1000 metres and sea traffic outside the Norwegian economic zone. Since emission figures distributed by municipality do not exist for 1994, national 1994 figures for each source are used combined with municipality distribution from 1992.

Except for Oslo and 'Rest of the country' the costs of SO_2 emissions are between 97 and 100 per cent of the total corrosion costs (compare table 3.2 with table 3.5). For Oslo the costs of SO_2 emissions are actually *higher* than the total costs. The reason is that the concentration level of ozone in Oslo is below its background level, so that the costs due to ozone is negative in the capital. In 'Rest of the country' the concentration level of SO_2 was assumed to equal its background level, which means that the whole cost is attributable to the ozone concentration.

The resulting marginal costs, also presented in table 3.5, show wide regional variations. However, the figures should be taken by caution as we have not considered transport of emission between the areas. This is apparent when comparing the results for e.g. Porsgrunn and Skien, which are neighbouring towns. The figures indicate that a significant amount of emissions in Porsgrunn are transported to Skien. The highest marginal costs are in areas where total costs are high compared to emissions, as for example in Bergen and Oslo. These are towns with a high density of buildings and infrastructure. On

[21] If we had rather fixed the ozone concentrations at the background level, the marginal costs of SO_2 emissions would have decreased by 2-3 per cent.

[22] Calculating the second cost component for Oslo is complicated by the fact that concentration levels for SO_2 and ozone vary across the grids. We have rather used the central value in the concentration intervals shown in table A1 in appendix A, i.e. 14.5 and 34 $\mu g/m^3$ for SO_2 and ozone, respectively. If we had chosen the most conservative estimates, i.e. the highest value for ozone and the lowest value for SO_2, the second cost component would have decreased by merely 0.5 per cent. Moreover, we do not consider that marginal costs of SO_2 emissions actually vary across the grids.

Table 3.5. **SO_2 emissions, total and marginal costs of SO_2 emissions by region, 1994. Mill. 1995-Nkr**

	SO_2 emissions tonnes	Costs of SO_2 emissions	Marginal costs Nkr/kg SO_2
Halden	68	1.8	25.9
Sarpsborg	1 669	5.4	3.2
Fredrikstad	885	2.4	2.7
Moss	664	1.5	2.3
Bærum	119	7.1	59.7
Asker	62	2.6	41.1
Oslo	1 051	74.5	70.9
Drammen	74	3.8	51.6
Porsgrunn	556	2.1	3.7
Skien	229	13.1	57.4
Bamble	24	0.5	19.6
Kristiansand	1 008	2.1	2.1
Stavanger	277	8.5	30.6
Bergen	263	22.3	84.8
Trondheim	674	9.1	13.5
Tromsø	108	0.8	7.3
Urban-south	17 886	34.0	1.9
Urban-north	5 798	3.5	0.6
Rest of the country	13 120	0.0	0.0
Total/average	44 535	194.9	4.4

the other side, the lowest marginal costs are found outside the towns where the material density is low.

In Statistics Norway (1995a) the emissions in each area are distributed on six separate sources (i.e., 4 mobile sources, stationary emission and process emission). Thus, as we assumed that the marginal effects of emissions from various sources in a municipality are equal, the costs displayed in table 3.5 can also be distributed on the various emission sources. Then, by summing over the whole country, the total and average cost of emissions from one specific source on a national level can be easily calculated. This is shown in table 3.6, together with the national SO_2 emissions distributed by source. We emphasise that we ignore differences in average costs within a specific area. In e.g. Oslo the average cost of 70.9 Nkr/kg is assumed to apply to all emission sources.

The highest average cost is incurred by mobile road-traffic sources where a tonne of SO_2 inflicts damage worth about Nkr 11 700. The process industry contributes just under half of the total SO_2 emissions in Norway, but incurs less than a quarter of the costs. Thus, the average cost is relatively low, i.e. about Nkr 2 400. The reason is that this industry is often located in sparsely populated areas where the concentration of buildings is low. A large share of SO_2 emissions from ships comes in 'Rest of the country', where the costs are zero. However, due to large emissions in the harbour of Oslo and other big towns, the average cost of emissions from this source is not as small as for the process industry.

Table 3.6. **SO_2 emissions and material corrosion costs distributed by SO_2-source, 1994**

	Stationary	Mobile sources				Process	Total
		Road and constr.mach.	Rail	Aircraft	Ships		
SO_2 emission							
- Tonnes	7 932	3 745	76	132	13 418	19 232	44 535
- Per cent	17.8	8.4	0.2	0.3	30.1	43.2	100.0
Corrosion costs							
- Mill. 1995-Nkr	63.1	43.7	0.4	0.6	40.6	46.6	194.9
- Per cent	32.3	22.4	0.2	0.3	20.8	23.9	100.0
Average cost (Nkr/kg)	7.9	11.7	5.1	4.4	3.0	2.4	4.4

3.7. Macroeconomic effects of material corrosion

We have thus far seen how air pollution reduces the lifetime of various materials and how this increases the costs of building-stock maintenance. Buildings are an important economic factor, both as dwellings for households and as an input in the production of goods and services for enterprises. Although the estimated extra maintenance costs are not heavy in the national economic context, the indirect effect of changes in corrosion costs for economic adjustment may be substantial compared to the estimated extra maintenance costs.

Air pollution increases the cost of building maintenance and thus the user cost of capital rises. This implies that agents may reduce their use of buildings and increase their use of other factors to adjust to the price changes. Investment in new building capital is reduced and the total building stock scaled down in the long term. Since the economy's growth potential depends in part on the availability of capital, a reduction in the stock of building capital will lead to reduced economic growth compared with a situation with no air pollution. Thus, there are allocation effects in the economy which must be analysed in addition to the direct maintenance cost.

In the short term agents will not change their use of buildings even though it becomes more expensive to maintain them, since adjustment costs may be high. However, analysed within a long-term model, it is reasonable to assume that enterprises' and households' adjustment in terms of building choice is optimal and flexible.

These allocation effects cannot be observed. The only way to calculate them is to carry out controlled experiments in models that simulate economic activity. Several models exist that simulate the Norwegian economy, each being designed to study various problems. Hence the choice of model must be based on the problem to be elucidated. We have chosen the MSG-EE model (Alfsen et al. 1996) which is designed to analyse questions associated with energy, transport and the environment (emissions to air). The model is outlined in chapter 2 of this book. The user cost of capital depends inter alia on material corrosion, which in the model have been linked to the

Table 3.7. **Capital stock and material corrosion costs by sector group, end-1994. Mill. 1995-Nkr**

Sector group	Capital value	Share	Corrosion costs	Share
Building capital				
Primary industry	94 082	4.9	10.8	5.5
Manufacturing industry	331 365	17.2	4.9	2.5
Services	838 232	43.5	55.2	27.9
Dwellings	638 805	33.1	100.4	50.8
Infrastructure	26 265	1.4	26.6	13.4
Total	1 928 748	100.0	197.7	100.0

pollution level following the course documented in this chapter.[23] It is used a weighted average of the SO_2 concentrations in the various areas included in this work. The quantitative relationship between the user cost and SO_2 emissions will vary for the various economic sectors owing to differing material-mix and geographical distribution. We have divided the sectors into four; primary industry, manufacturing industry, services (incl. public services) and dwellings. Then the costs for different building types displayed in table 3.3 are distributed on these four groups of sectors, and further distributed on the individual sectors according to building capital values at end-1994. The costs for infrastructure are distributed on each sector according to car capital values.[24] The resulting figures are shown in table 3.7. We see that corrosion costs per building capital value are highest for dwellings, but the costs per capital value are even higher for infrastructure. The building capital of the manufacturing industry, on the other hand, is hit quite modestly compared to the value of their building capital.

The model estimates changes in gross domestic product and other variables prior to and after inclusion of material costs from air pollution. Since emissions to the atmosphere influence the user cost of buildings and infrastructure, and thus the level of investment, economic activity will be affected several years after the emissions take place. Future allocation effects resulting from an increase in current emissions are discounted by 7 per cent per year. The effects prove to be greatest in the years immediately following the emission increase; after about 10 years there is no longer any measurable effect as economic agents gradually move back to the reference path of the economy.

We will use the discounted changes in gross domestic product as an indicator of the allocation costs. Then we find that the marginal allocation cost at the national level caused by SO_2 emissions is 2.1 1995-Nkr per kg, which is about one-half of the marginal maintenance costs (see table 3.5). Assuming constant marginal costs, the total calculated allocation cost comes to Nkr 93 million. The total economic costs equal the sum of maintenance costs and the allocation costs.

[23] Because of the difficulties in distributing the corrosion costs on SO_2 and ozone concentrations, for simplicity all costs are attributed to SO_2 concentrations in this section. This will probably give a negligible bias on the allocation costs.

[24] As no final figures were available for the stock of building and transport capital as at end-1994, the figures presented are based on national accounts figures from 1991 adjusted for capital depreciation and gross investment (Statistics Norway 1993a, 1995b).

Table 3.8. **Changes in SO_2 emissions and maintenance costs 1985-1994**

	Stationary	Mobile sources				Process	Total
		Road and const.mach.	Rail	Aircraft	Ships		
1994							
SO_2 emissions (1 000 tonnes)	7.9	3.7	0.1	0.1	13.4	19.2	44.5
Costs (Mill. 1995-Nkr/yr)	63	44	0.4	0.6	41	47	195
Marginal costs (Nkr/kg)	7.9	11.7	5.1	4.4	3.0	2.4	4.4
1985							
SO_2 emissions (1 000 tonnes)[1]	31.4	6.1	0.2	0.4	26.4	47.2	111.6
Costs (Mill. 1995-Nkr/yr)[2]	250	71	1.1	1.6	80	114	489
1985-1994							
Emission reduction 1985-1994 (1 000 tonnes)	23.5	2.3	0.1	0.2	12.9	28.0	67.1
Cost reduction 1985-1994 (Mill. 1995-Nkr/yr)	187	27	0.7	1.0	39	68	294

[1] The distribution of mobile emissions in 1985 is not known and is calculated using the same weights as in 1994.
[2] Using present calculation method.

Allocation costs constitute about 50 per cent of maintenance costs. The model cannot compute regional effects but, based on a reasonable assumption of proportionality between allocation costs and maintenance costs, the total marginal costs in each region can be estimated by multiplying the figures in table 3.5 by 1.47. For Oslo the marginal cost including the allocation cost then comes to about Nkr 105 per kg of SO_2.

3.8. Change since 1985

A calculation of material costs resulting from SO_2 emission in Norway in 1985 found that maintenance costs totalled _220 million 1985-Nkr (Glomsrød and Rosland 1988). Applying the housing construction cost index this gives a figure of 348 million 1995-Nkr. When comparing this result with ours, two important changes must be taken into account. First, SO_2 emissions have been reduced by 60 per cent in the period 1985-1994. Second, we have included far more materials in the present work. These two effects pull in opposite directions in terms of costs, as our results show a slight reduction in maintenance costs.

If the level of pollution had remained unchanged since 1985 (111 600 tonnes of SO_2 emissions), maintenance costs due to SO_2 emissions calculated by today's methods would be 489 million 1995-Nkr per year, i.e., maintenance expenditure on buildings have been reduced by 294 million Nkr in the period 1985-1994 (see table 3.8).

Table 3.9 shows how the total costs, i.e., including the allocation costs, have changed since 1985. Here we have included costs from ozone concentrations, too, which we have assumed to be unchanged since 1985. The allocation costs are further assumed to be proportional to the direct maintenance costs. It should be noted that the saving is calculated for the year 1994 and not for the entire period 1985 to 1994. The annual saving prior to 1994 was smaller inasmuch as SO_2 emissions were larger than in 1994. Total costs in 1994 were

Table 3.9. **Comparison of yearly costs due to material corrosion. Mill. 1995-Nkr**

	Costs		Saving
	1985	1994	1985-1994
Maintenance costs	491	198	294
Allocation costs	230	93	137
Total costs	721	291	431

Nkr 431 million lower than in 1985 as a result of the reduction in SO_2 emissions.

3.9. Uncertain factors

Some uncertainties attach to the calculations presented, and we will briefly describe the most important ones. These are, however, difficult to quantify.

Regarding the chemical-physical relation between air pollution and material corrosion (dose-response functions), a main problem is that damage functions only exist for a small selection of materials. Recent decades have seen the development and use of a number of new building materials for which dose-response functions are not available. Developing such functions is very time-consuming since the materials must be exposed over a long period before the results can be processed. The ongoing test programme under the auspices of UN-ECE, e.g. Haagenrud and Henriksen (1995), extends over eight years. Good descriptions are unavailable particularly for cement, tiles, and concrete. These materials are employed so extensively that the economic implications may be substantial. Air pollution is known to have a harmful effect on other materials too, e.g. asphalt, rubber, textiles, sealing and electrical contact materials. The relations have so far not been quantified. The absence of dose-response functions for a number of materials suggests that our estimate for material costs is on the low side.

The key to calculating stocks of materials in the present report has been the MOBAK study from Sarpsborg which involved extensive fieldwork inspection of buildings. The results from that study have been used in the present report for material stock calculations for the entire country. The uncertainty lies in how far Sarpsborg is representative in terms of material-mix for the rest of the country. The MOBAK study shows close correspondence between material use in Sarpsborg and Stockholm. Hence we can be fairly confident that it provides a good description of material use throughout Scandinavia. Another uncertainty arises from the lack of material stock calculations for a number of building types. The sector 'Production and distribution of electrical power' accounts for a substantial stock of building capital (9 per cent of the total in value terms). Although large parts of this capital are located in areas of low pollution, the sector should have received special treatment in the study.

Costs of corrosion and weathering of buildings of historical value have not been calculated in the present study. A monetary value would have to be given to the individual cultural monument in order to take these costs into account.

Traditionally material corrosion has been associated exclusively with sulphur pollution because sulphur has been the predominant component of pollution in recent decades. Since recent years have seen a shift away from sulphur emissions towards ozone and nitrogen pollution, researchers have postulated that these pollution components also influence material corrosion, both alone and in synergy effect with each other. In the present report ozone has been incorporated as a variable for five materials.

Ozone also has (so far unquantified) effects on other materials. Other pollution parameters also influence the costs of maintaining building capital. A pertinent example in this context is soiling. Deposition of dust and soot particles diminishes buildings' economic lifetime and aesthetic quality. Today considerable resources are spent on exterior cleaning of buildings, especially window-cleaning.

The allocation cost is calculated using a model that simulates the entire national economy. Such models will of course simplify reality and therefore produce unceratin results. The allocation costs further depend on maintenance costs, and hence, the uncertainties attached to these calculations will influence the accuracy of the allocation cost.

The calculation of marginal costs is based on maintenance costs and a number of additional assumptions regarding emissions and dispersion of SO_2. These calculations are therefore more uncertain than the total maintenance costs calculations.

3.10. Conclusion

Even with the uncertainties discussed above, the analyses have been improved in several ways compared to a few years ago. Thanks to the ECE materials project, more realistic dose-response functions are available today for a greater number of important materials than previously. Moreover, it has now been documented that NO_2 and O_3 have a stronger corrosive effect on many materials than earlier thought. Substantial improvements in the calculation base have also been achieved by introducing a GIS-based modelling tool, which have been linked up to the Norwegian building register. For Oslo this linkage means that information is now available on building and pollution conditions in a grid of 500 x 500 m^2 covering the whole of Oslo. The calculations can be further improved by implementing these tools in other towns as well. The MOBAK study, giving information about the mean distribution of various materials on various building categories, has also improved the validity of the estimates. Finally, the economic modelling apparatus used to estimate allocation costs also represents an improvement on the one previously in use.

To sum up the results, maintenance costs ascribable to material corrosion of buildings and motor vehicles that is caused by air pollution have been calculated at about 198 million 1995-Nkr. In addition allocation costs in the economy were estimated at 93 million 1995-Nkr. We cannot assign the results a probability distribution because the basic data do not permit this. However, since many types of materials are not dealt with in the report the estimates will in all probability be on the low side in relation to the real costs inflicted by air pollutants on the building capital and the motor vehicle population of Norway.

The marginal-cost calculations reflect that the smaller the distance between an emission source and a building the greater damage the emission will inflict on the building. Hence emissions in areas where there is a high concentration of buildings will cause relatively heavy damage. This means that emissions from sources such as vehicle traffic will generally cause greater damage than industrial emissions, as industrial emissions in Norway are often located in smaller towns and villages. The highest marginal costs were found in Bergen and Oslo, where the emission of

1 kg of SO_2 inflicts damages valued at Nkr 85 and 71, respectively (excluding allocation costs).

Of the pollution parameters included in the calculation, SO_2 is by far the most dominant cost factor. Since only 15 per cent of costs are related to materials that are affected by O_3 and H^+, changes in these parameters will only produce small changes in costs. On the other hand, we found that the large reduction in Norwegian SO_2 emissions in the period 1985-1994 has resulted in an annual saving of 431 million 1995-Nkr. Thus, reducing emissions of air pollutants are clearly not merely associated with economic costs.

4. Social costs of crop damage from ground-level ozone[25]

Anett C. Hansen[26], Henning Høie[26], Leiv Mortensen[27], Knut Einar Rosendahl[26] and Kjetil Tørseth[28]

4.1. Introduction

Controlled experiments have found significant evidence of a negative relationship between exposure to ground-level ozone and the yields of several types of vegetation. On a European level consensus dose-response functions (or damage functions) have been formulated for wheat and potato (UN-ECE 1994), whereas several Norwegian studies have found significant dose-response functions for various grass species used on cultivated meadowland (see e.g. Mortensen (1995)).

The harmful effect of a given ozone dose is influenced by various climate factors such as temperature, daylight conditions and humidity as well as soil moisture. This, in addition to variation in ozone sensitivity between types within a species makes it difficult to establish reliable damage functions. As the number of experiments are limited so far, the parameters in the dose-response functions may therefore be considerably altered when more, and more sophisticated, studies are conducted. Despite the great uncertainty the recommended damage functions will nevertheless give some indications about the damages.

In this study we calculate the crop damages from ground-level ozone based on current knowledge. Moreover, the economic losses related to these damages are analysed. The calculations are conducted for the year 1992, when the ozone concentrations in Norway were particularly high. Thus, the results are probably not representative for an average year. On the other hand, potential damages to several agricultural plants are not considered because reliable, controlled experiments have not been conducted. Damages to tree species from ground-level ozone are neither included although some dose-response functions have been estimated (see, however, Tørseth et al. (1997)).

The contributors to ground-level ozone are emissions of NO_x and VOC. However, the formation of ozone is a complex

[25] This study was commissioned by the State Pollution Control Authority (SFT) and carried out jointly by the Norwegian Institute for Air Research (NILU), the Særheim Research Station and Statistics Norway. The article has earlier been published in Norwegian in Tørseth et al. (1997).

[26] Statistics Norway.

[27] Særheim Research Station.

[28] Norwegian Institute for Air Research.

process, and close to large emission sources of NO_x the concentration of ozone may actually be reduced (see chapter 3 in this book for a more thoroughly description). In Norway ozone concentrations are mainly determined by foreign emissions, which are transported in the air to Norway. Thus, the crop damages calculated in this study is to little extent affected by domestic actions.

So far the experiments indicate that AOT40, i.e. accumulated exposure to ozone concentrations exceeding 40 ppb (parts per billion) or 80 $\mu g/m^3$, may be a good indicator when it comes to crop damage from ground-level ozone. AOT40 is here measured during the growing season, which may differ between locations and type of vegetation. In Tørseth et al. (1996) levels of AOT40 for the period 1989 to 1993 have been calculated on the basis of measurement data recorded at Norwegian monitoring stations. These calculations were based on the recommendations from a UN-ECE meeting in Bern in 1993 (UN-ECE 1994), which inter alia recommended critical levels of AOT40. The question of how the recommendations can be adapted to Nordic conditions was considered by Tørseth et al. Levels of AOT40 for various types of vegetation in the Nordic region were mapped.

The results from Tørseth et al. (1996), which will be described below, are used directly in our calculations of crop damages. An important finding is that the decision of growing season, and particularly the start of the latter, is highly significant when calculating AOT40 levels and identifying areas where the critical levels are exceeded. This is because ozone concentrations are highest in spring and early summer. At a UN-ECE working meeting in Kuopio, Finland in 1996 (UN-ECE 1996), the recommendations at UN-ECE (1994) were revised based on new experience. These changes may substantially affect the calculations of crop damages in Norway. However, the present study does not take account of the new recommendations, but points out the most important changes.

Detailed information exists on areas, yields and position of various agricultural plants and meadow in Norway. Together with the calculated exposure doses and the available dose-response functions, this has provided a basis for rough estimates of crop yield losses that may result from exposure to ozone. Moreover, land area where critical ozone levels, as identified by UN-ECE (1994), are exceeded may be calculated, too. However, as indicated above, much uncertainty attaches to the resulting figures. Still, it is not possible to construct reasonable uncertainty intervals.

The social costs of a crop yield loss are difficult to analyse since the farming industry is heavily regulated and largely sheltered from imports. The costs will depend on, among other things, the authorities' policy aims for this sector. A policy aim to maintain a certain production potential in the interest of food security has different implications than an aim to maintain a certain level of activity (e.g. in terms of employment) in support of regional policy objectives. In the first case a crop yield loss and consequent production shortfall has to be compensated for by raising activity levels in the sector. The direct cost is then largely reflected in production costs in agriculture, and the domestic producer price plus any subsidies will be a good indicator. In the second case a crop yield loss can be compensated for by increasing imports,

and prices on the world market will be a good indicator.

The rest of the national economy is affected when crop yields per hectare are reduced as a result of ground-level ozone. Here too the effects are dependent on which scenario is most realistic. Inasmuch as far more resources are needed to achieve a particular production value in agriculture than in other industries, attracting resources to agriculture will entail substantial social costs over and above the direct value of the loss of yield.

The uncertainty of the economic calculations compounds the uncertainty involved in calculating the physical ozone damage. The economic uncertainties are related both to choice of shadow price for the individual type of crop (i.e. the direct social cost per unit volume), and to the analysis of the macroeconomic effects. The social costs must therefore be seen as rough estimates. However, the calculations do provide (based on current knowledge) some indications of what ground-level ozone can cost Norway in the form of crop yield reductions in years with very high ozone exposures.

In the next chapter the physical damage of ozone exposure is discussed. Then, in chapter 4.3 the economic analysis is presented.

4.2. Ozone exposure and crop damage

4.2.1. Methods for estimating crop yield losses

Damage functions for various types of vegetation
Ozone sensitivity varies widely among different types of vegetation. Of the approximately 50 species examined in Norway (Mortensen 1992, Mortensen and Nilsen 1992, Mortensen 1993, 1994a, 1994b), birch, timothy and clover can be characterised as highly ozone-sensitive. Dose-response curves for ozone effects on plant growth and yield are available for only a fairly small number of species at present. An overview is shown in table 4.1, where the fuctions are expressed in relative yield. By relative yield (y) is meant actual yield divided by yield without ozone exposure, and converted to percentage points. Wheat is the most thoroughly examined (UN-ECE 1994), and the critical ozone level for wheat has been recommended for other agricultural plants. The potato seems to have a sensitivity matching that of wheat. For cultivated meadowland where timothy and clover are important grass species, Norwegian tests have ascertained a somewhat lower sensitivity than for wheat (Mortensen 1995).

The damage functions are based on the assumption that there exists a linear relation between ozone exposure and yield loss. The relations have been derived empirically from a limited testing. However, at low exposures in controlled experiments, relations between dose and response have been difficult to discern, and no significant correlation have been established. For that reason UN-ECE (1994) recommended that the damage functions are only applied for AOT40 values that according to the function imply yield losses exceeding 10 per cent (see table 4.1). That is, 10 per cent is chosen as the smallest statistically reliable reduction, and the corresponding AOT40 value is identified as the critical level. This means that we actually use functions that are discontinuous around this critical level. The damage functions in table 4.1

Table 4.1. **Overview of damage functions for various types of vegetation, where *y* is relative yield and *x* is ozone exposure (AOT40 measured in ppm h). (1 ppm h = 1000 ppb h)**

Type of vegetation	Damage function	Area of application(critical level)
Wheat	*y = 99.6 - 1.804x* (UN-ECE, 1994)	x>5.3 (5 300 ppb h)
Potatoes	*y = 99.6 - 1.804x* (UN-ECE, 1994)	x>5.3 (5 300 ppb h)
Meadow	*y = 100 - 1.5x* (Mortensen, 1995)	x>6.7 (6 700 ppb h)

show that an AOT40 value of 5 300 ppb h for wheat and potatoes, and 6 700 ppb h for meadow causes a 10 per cent loss of yield, and these values are accordingly critical levels.

The timing of the start of the growth period for a given type of plant is crucially significant for whether the ozone dose will exceed the critical level. Since April, May and June are generally the months with the highest AOT40 values (normally accounting for more than 50 per cent of overall exposure in the growing season), an early growth start will result in higher exposure. The growing season starts later in Scandinavia than in the rest of Europe. Thus, the recommended choices of season from UN-ECE (1994) are ill suited in our case. Using growth periods more relevant to Nordic conditions reduces the exposure doses considerably. However, even when this is done, exposure doses may exceed the recommended critical levels for vegetation in southern Norway. Grass production in low-lying areas of southern Norway is the most heavily exposed (growth start 15-25 April). Cereals (germination 10-20 May) are less exposed to ozone, whereas potatoes, which can be extremely sensitive (wide differences between different types), do not start growing properly until about 1 June and are therefore exposed to a relatively low AOT40 dose through the growing season. Vegetation in the mountains of southern Norway will also in general be protected against high ozone exposure by the late growth start (June).

Accumulated exposure doses for ozone are determined as follows. For agricultural crops the growing season is defined as the period from germination of wheat to its maturation. Normal values for two weeks after sowing to two weeks before harvesting in the various regions are employed as a Nordic adjustment of the growing season. Only daylight hours are included in calculations of AOT40. For cultivated meadow we have, as a Nordic approximation, defined the growing season as those days whose normal mean temperature exceeds 5C. Only daylight hours are included in calculations of AOT40. The growing seasons employed are shown in table 4.2.

Measurements over a wheat field in southern Sweden have shown that ozone uptake leads to a considerable reduction in ozone concentrations closest to ground level (Pleijel and Grennfelt 1995). Hence calculating the exposure dose on the basis of measurements taken 2-3 metres above ground level could result in an over-estimation of 20 per cent in relation to the reference height used in dose-response experiments, which is 1.1 metres.

A recommendation from the UN-ECE working meeting in 1996 (UN-ECE 1996) was that present critical level criteria are not suited to estimating *regional* crop yield losses. This is primarily due to uncertainties related to the degree to which ozone is absorbed by vegetation and to other factors which may affect plants' sensitivity to ozone. There are indications that interacting effects may entail increased sensitivity to ozone exposure. On the other hand drought-induced stress could cause plants' leaf pores to become smaller, thereby reducing ozone uptake. Knowledge of these processes is limited, but could be incorporated in the guidelines when more information becomes available. Extending the critical level criterion to embrace such factors is referred to as level 2, while surveys in accordance with UN-ECE (1994) and the new recommendations from UN-ECE (1996) are designated level 1. The UN-ECE meeting in 1996 also agreed upon that the critical level for wheat should be reduced, so that 5 per cent yield loss becomes the smallest significant reduction. This would mean that the critical level of AOT40 were halved. This change would significantly increase the exceeded area, and thus the calculated crop damage. However, taking the other factors above into account, too, it is unclear whether the estimated crop damages are underestimated or not.

Use of measurements for calculating ozone exposure

Ground-level ozone is measured at about 15 monitoring sites in Norway with finance provided by the State Pollution Control Authority through a separate monitoring programme under the State Programme for Pollution Monitoring (SFT 1994). In addition 19 monitoring sites exist in the rest of Scandinavia. The locations of the sites are shown in figure 4.1, and the sites' coordinates, altitude and appropriate growing seasons are shown in table 4.2.

Calculating accumulated ozone-exposures over the whole country requires much in terms of the monitoring sites' representativeness, the monitoring network's density and data completeness. The representativeness of the individual sites varies somewhat and has not been quantified, above all in terms of AOT40 values. First, sites affected by local NO emissions will not be representative for larger regions. The monitoring sites at Nordmoen (outside Oslo), and to a lesser degree at Søgne (nearby Kristiansand) and Svanvik (close to the Russian border), are affected by local NO emissions, which entails lower observed ozone concentrations.

Second, as ozone is taken up by plants and is reacting with a large number of surfaces, ozone concentration at ground level is often considerably lower than at an altitude of 50 - 100 metres. Because of this, high valley sides and ridges are more vulnerable to high AOT40 values than are larger continuous forest areas and low-lying areas. Thus, regional exposure doses for various types of land cannot be determined precisely based on current ozone monitoring. The measurements do however give an indication of regional distribution and typical exposure doses and show geographical areas where various critical levels may be exceeded.

Measurements for the year 1992 from the 34 monitoring sites in Scandinavia are used (Lövblad et al. 1996). Most of these are EMEP (European Monitoring and Evaluation Programme) sites, but other measurements are also employed. Because

Figure 4.1. **Location of Scandinavian monitoring sites used to survey ozone exposure**

a number of sites provided low coverage or were not representative, the number of sites was reduced to 24. Exposure doses for varying length of growing season are calculated from the measurement results. Inclusion of the remaining Scandinavian sites substantially improves the database for regional surveys, particularly along the borders and in the north of the country.

Accumulated exposure doses (AOT40) are calculated as the sum of the differences between hourly mean concentrations and 40 ppb for each hour where ozone concentration exceeds 40 ppb. Exposure doses calculated for the various plant types and growth periods are shown in table 4.1. For agricultural crops only hours of global light influx in excess of 50 W/m^2 are included in the calculations. Global light influx is calculated as a function of the sun's meridian and no account is taken of cloud cover.

Since the AOT40 concept states a sum of measurement values in excess of 40 ppb, any absence of data will lead to great uncertainties in the calculated result. This is especially critical if no measurements are taken during episodes of high concentrations. Several considerations can be applied to reduce the uncertainties. Data coverage should be as good as possible for all parts of the growing season. Sites with poor data coverage should therefore be omitted from the calculations. However, setting an excessively high criterion for data coverage will reduce the number of sites, which creates problems for the overall survey. In 1992 data coverage exceeded 90 per cent at most monitoring sites. Sites offering data coverage between 80 and 90 per cent are, in the event, included after assessment and comparison with sites in the vicinity. Sites with less than 80 per cent data coverage are not included. To correct for absence of data, it is assumed that the absent data show the same distribution as the observed data and correction is obtained by dividing the calculated exposure dose (AOT40) by relative data coverage.

Calculated exposure doses at monitoring sites are interpolated to a grid by kriging interpolation, which is a statistical method for estimating unknown values from nearby observations. The method was originally developed for geostatic purposes (Matheron 1963, Journel and

Huijbregts 1981), but has in recent years also been used to describe regional distribution of, for instance, trans-boundary air pollution (Schaug et al. 1993). Kriging interpolation is based on the assumption that the variance between the observed values is a function of distance and direction, and weighting is determined by variogram analysis. Exposure doses of ground-level ozone based on the AOT40 principle are difficult to interpolate owing to wide variance in the calculated values. Moreover, because the number of sites is on the small side, the grid values calculated are rough approximations and relatively uncertain. A grid comprising 50 km squares is used based on division of the ordinary grid use by the EMEP (150x150 km^2).

A comparison between exposure doses calculated both on the basis of monitoring data and with the aid of EMEP's oxidant model (Lövblad et al. 1996) shows relatively good correspondence in southern Norway, southern Sweden and in southern Finland. In Denmark the model overestimated exposure by about 50 per cent compared with the exposure based on monitoring data. In the more northerly parts of Scandinavia the model showed substantially lower exposure than that based on measurements.

Areas and yields of ozone-sensitive vegetation

In order to link ozone exposure to damages on various types of vegetation, areas and yields of the following types of land were distributed on the EMEP grid (50x50km^2): spring-sown wheat, autumn-sown wheat, cultivated meadow, extensively cultivated meadow (i.e., surface-cultivated and fertilised pasture), and potatoes. Data on agricultural areas are taken from information provided by farm operators as a basis for annual governmental production subsidies (as at 31 July 1994). For the country as a whole applications for production subsidies covered an estimated 94.3 per cent of grain-growing land, 97.4 per cent of meadow and 98.7 per cent of land used for potato production. The data for farm areas are basically distributed by municipality. Yields are Normal Year Yields 1994 calculated by the Norwegian Agricultural Economics Research Institute. No distinction is drawn between yields of spring- and autumn-sown wheat. Information on yields on surface-cultivated and fertilised pasture is unavailable, and is assumed to be two-thirds of the yield from cultivated meadow.

Normal annual yields are calculated at county level and all municipalities in the county are each assigned an identical yield per hectare. Moreover, for the purpose of distribution on the EMEP grid, the municipalities' arable areas as well as production are distributed in proportion to the distribution of the entire area of the municipality. In reality variations will occur both among municipalities within a county, and within the municipality. These sources of error contributes somewhat to overstated agricultural production for higher-lying, and generally the northerly, EMEP grid squares, and correspondingly understated production levels for lower-lying and southerly squares.

Wheat is more dependent on warm temperatures than any other cereal plant grown in Norway, and all wheat production is concentrated in the areas around the Oslo Fjord. About 70 per cent of Norwegian wheat is grown in the counties of Akershus, Østfold and Vestfold. Potato cultivation is also concentrated in south-east Norway, with about

Table 4.2. **Growing seasons, data coverage and calculated exposure doses for ozone for agricultural plants and cultivated meadow in 1992**

		Agricultural plants (Nordic adjustment)			Cultivated meadow (Nordic adjustment)			Agricult. plants (UN-ECE)*	
Monitoring site	Alti-tude	Growing season	Data cover-age (%)	AOT40 (ppb h)	Growing season	Data cover-age (%)	AOT40 (ppb h)	Data cover-age (%)	AOT40 (ppb h)
Birkenes	190	15/5-15/8	91	10 102	15/4-1/11	93	11 990	92	11 052
Haukenes	20	15/5-15/8	–	–	15/4-1/11	–	–	–	–
Høylandet	60	1/6-1/9	–	–	1/5-1/10	–	–	–	–
Jeløya	3	15/5-15/8	82	6 567	15/4-1/11	87	7 215	96	5 991
Jergul	255	1/6-1/9	73	–	1/6-15/9	74	–	99	2 643
Klyve	60	15/5-15/8	94	5 872	15/4-1/11	97	7 606	95	6 620
Kårvatn	210	1/6-1/9	99	1 968	1/5-15/10	99	8 830	98	8 863
Langesund	5	15/5-15/8	96	5 080	15/4-1/11	95	6 041	96	5 352
Nordmoen	200	15/5-15/8	–	–	15/4-1/11	69	–	–	–
Osen	440	15/5-15/8	100	4 721	15/4-15/10	95	5 867	96	5 278
Prestebakke	160	15/5-15/8	–	–	15/4-1/11	–	–	–	–
Svanvik	30	1/6-1/9	–	–	1/6-1/9	–	–	–	–
Søgne	15	15/5-15/8	–	–	15/4-1/11	–	–	–	–
Tustervatn	439	1/6-1/9	100	387	15/5-15/9	100	1 101	100	1 225
Valle	250	15/5-15/8	91	6 127	1/5-15/10	–	–	91	6 423
Voss	500	15/5-15/8	100	5 490	1/5-15/10	100	5 663	100	5 630

* Growing season: 1 May - 1 August

45 per cent of the country's total production taking place in the neighbouring counties Hedmark and Oppland. Grass production is associated with husbandry of livestock feeding on rough grazings such as cattle, sheep and goats. These productions predominate in all farming areas apart from in the grain-growing areas around the Oslo Fjord.

4.2.2. Results and discussion

The study by Tørseth et al. (1996) for the years 1989-1993 found that in most years exposures range from 5 000 to 10 000 ppb h in southern Norway and from 2 000 to 5 000 ppb h in northern Norway in the period April - September. In the growing season the exposure doses are around the critical levels for cultivated meadow and grain. The highest exposure doses in the five years period were with a few exceptions observed in 1992, which is the year we focus on here. The high exposures in that year were due to meteorological conditions which resulted in effective transport of ozone and precursors from the Continent to Scandinavia during the spring and early summer. However, at the monitoring site Tustervatn in the northern part of Norway, AOT40 values were actually *lowest* in 1992. For a complete discussion of the results of the survey, see Tørseth et al. (1996).

Table 4.2 shows growing seasons, data coverage and calculated exposure doses for ozone in 1992 for agricultural plants and cultivated meadow at all monitoring sites. The determination of growing season was explained above, and this varies between locations and plants. For comparison, calculated AOT40 levels using the growing season generally recommended by UN-ECE (1994) for agricultural plants in Europe, are also shown in the table. However, these results are not used in the further calculations as this choice of growing season is thought to

Figure 4.2. **Distribution of AOT40 (ppb h) for growing seasons for wheat (Nordic adjustment), 1992**

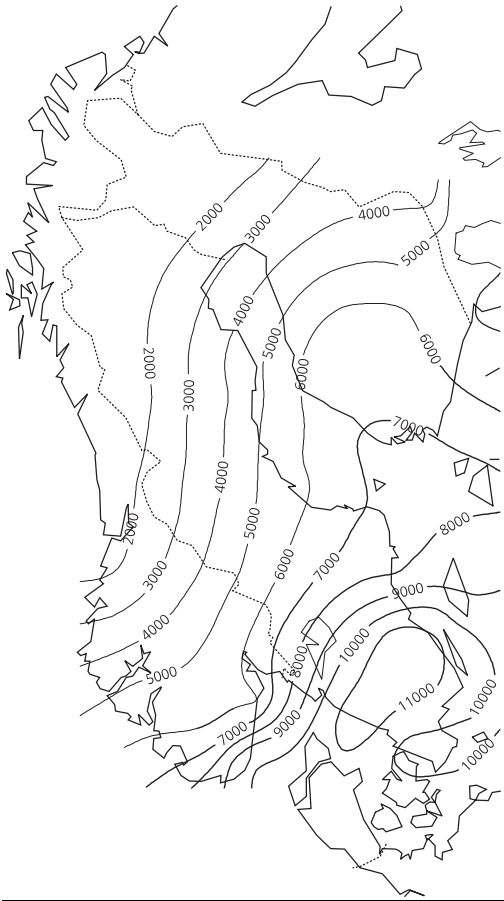
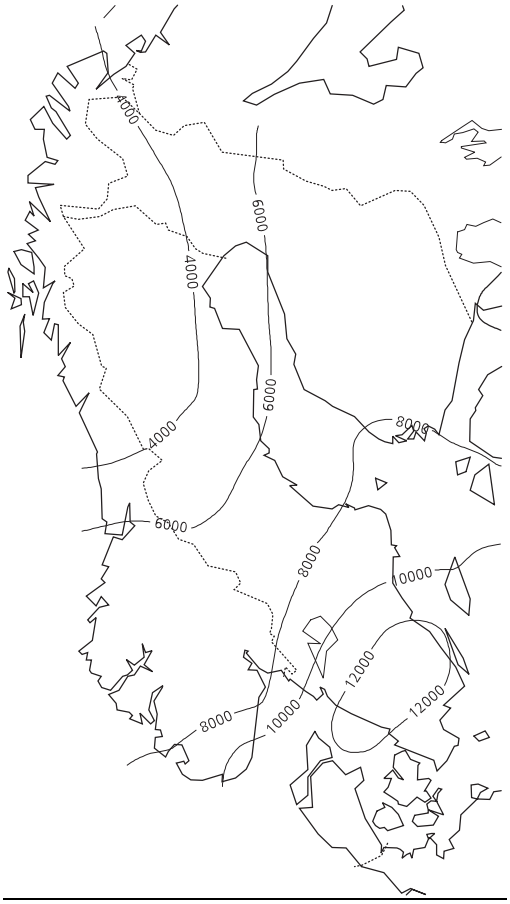


Figure 4.3. **Distribution of AOT40 (ppb h) for growing seasons for cultivated meadow (Nordic adjustment), 1992**



be inadequate for Nordic conditions (see above). Figure 4.2 and figure 4.3 show regional patterns in the distribution of AOT40 for growing seasons for wheat and meadow respectively.

Table 4.2 and figure 4.2 show that for agricultural plants the highest doses were observed in southern Scandinavia. At Birkenes at the south coast of Norway the AOT40 value was in excess of 10 000 ppb h in 1992, which according to the damage function in table 4.1 should indicate a yield reduction of almost 20 per cent. The critical level of 5 300 ppb h is exceeded south of 60°N (around Bergen). As shown in table 4.2, using the growing season recommended by UN-ECE (1994) increases the AOT40 levels significantly, and the critical level is exceeded in the whole of southern Norway (i.e. south of Trondheim). For cultivated meadow the results indicate that the critical level is exceeded in most of southern Norway. The highest AOT40 value was again measured at Birkenes, at about 12 000 ppb h.

Table 4.3. **Areas of exceedance and potential yield loss for various vegetation types in Norway, 1992**

Vegetation type	Area of exceedance		Yield loss	
	km^2	per cent	1 000 tonnes	per cent
Wheat*	597	88	30.7	11
Potatoes	97	59	32.4	7
Total meadow**	3 329	59	332	8

* includes spring-sown and autumn-sown wheat
** includes cultivated meadow, surface-cultivated meadow and fertilised pasture

Table 4.3 shows the areas of exceedance for the different types of vegetation in 1992. The areas are calculated by assuming that the entire area of a vegetation type in each EMEP square showed exceedance if the AOT40 value in the square was higher than the critical level. From the table we see that the critical levels were exceeded for a large share of agricultural land in 1992, particularly for wheat where the area of exceedance constituted 88 per cent of all wheat fields. If we had rather used the growing season recommended by UN-ECE (1994), the area of exceedance would have increased to a share of more than 99 per cent. In terms of absolute numbers the biggest exceedance was in the case of meadow, as meadow clearly is the most widespread type of land. For both meadow and potatoes the area of exceedance equalled about 59 per cent in 1992.

Based on calculated AOT40 values and the damage fuctions set out in table 4.1, yield losses may be determined. The calculations were based on the assumption that normal annual yield is affected by ozone exposure. Hence, we get the following expression for yield loss:

yield loss = (normal annual yield/relative yield) - normal annual yield

The biggest yield loss was found in grass production where the weight loss is about one order of magnitude higher than for wheat and potatoes respectively. However, this is due to the large production of grass compared to other plants. In terms of relative numbers the yield losses constituted around 10 per cent for all three crops. The UN-ECE (1994) recommendations would have increased the calculated yield loss in wheat production to 14 per cent.

4.3. Economic analyses of crop damage

We now go on to analyse the social costs associated with the yield losses resulting from ground-level ozone in Norway (see table 4.3). Reduced yield entails economic loss since a given set of resources produces less return than it would have done in the absence of ozone exposure. The pollution reduces the productivity of the resources, i.e. the relation between production level and quantity of inputs.

In this presentation we start out from a potential situation of no ozone exposure, and discuss the effects resulting from an occurrence of crop damage. Although the yield reductions are calculated for a year of particularly high ozone exposures (1992), we regard the problem of ground-level ozone as a long-term one. Crop damage resulting from excessive ozone

concentrations is not considered primarily as a short-lived shock to which agriculture is vulnerable, but as a lasting problem which the farmer takes into account in his adjustment on a par with climatic conditions. The economic analyses are therefore rooted in long-term perspective. However, since ozone exposure varies somewhat from year to year, and was particularly high in 1992, we also discuss how a short-term perspective affects the costs incurred.

Calculating real social costs of reduced productivity in agriculture is particularly difficult inasmuch as this sector is highly regulated and largely sheltered from imports. Hence it is important to ascertain a relevant price for various crop types. This price, often termed the shadow price, depends inter alia on the policy aims set by the authorities for the agricultural sector. This and other methodological questions are discussed in detail in the following subsection. Then, section 4.3.2. presents results for the direct costs of exposure to ground-level ozone, i.e. the shadow price multiplied by yield loss. In section 4.3.3 it is shown how the yield loss can affect the rest of the economy, either through reduced supply of domestically produced foodstuffs, or because the agricultural sector attracts more inputs in order to maintain production levels. These effects are analysed using a macro-economic model.

4.3.1. Methods for analysing crop damage

Aims and guidelines for Norwegian agricultural policy

There are several reasons why the agriculture sector in Norway is regulated and largely sheltered against imports. Proposition no. 8 to the Storting from the Ministry of Agriculture (1992) sets out guidelines for agriculture policy, and essentially expresses the authorities' current policy aims for agriculture.[29] Several factors are given weight, and in the context of the present study we focus on two of them. The first is long-term food security. The proposition states that "importance should be given to maintaining production potential on the limited geographical resources available", whereas a continual high level of self-sufficiency is regarded as less important (chap. 3.1.3). A palpable indication of this is the authorities' switch from production-dependent to area-dependent plant production subsidies. This switch of emphasis entails that lasting changes in extraneous factors affecting productivity in the sector may lead to policy shifts to preserve agricultural land. Such a factor could be changes in ozone exposure.

The second factor we focus on, and which is given weight by the authorities, is the agriculture sector's great significance for safeguarding population settlement and employment in outlying areas.[30] In this context activity levels in agriculture, in the first instance the number of persons employed in the sector, are important. However, the Ministry of Agriculture emphasises (1992) the need for a regional policy strategy focusing on a broader economic base, and the need to base activities in agriculture to a greater degree on market potential that offers a broader and more varied basis for employment.

[29] Although the Storting's treatment of the proposition resulted in minor changes in the adopted recommendation, wide disagreement exists between some parties in the Storting.

[30] The agricultural sector and its employment effects on other economic activity account for more than one half of jobs in about one fourth of the country's municipalities (Ministry of Agriculture 1992).

This may entail that reduced productivity as a result of extraneous conditions in some areas may contribute to a *reduction* in activity levels in agriculture at the expense of other economic activity. Putting the emphasis on regional policy conditions may thereby give rise to other and in some cases contrary conclusions to those resulting from emphasis on food security.

While production potential and activity levels are both presented as important, it is conceivable that one of these policy aims may already be amply fulfilled in the current situation. However, there is no basis for saying which of the two this might be. With this in mind, we examine two different scenarios.

In scenario 1, called "Maintenance of production potential", we assume that the authorities respond to reduced productivity in agriculture by adjusting agricultural land use to ensure that production potential remains unchanged. We also assume that the potential is exploited to the same degree as prior to the productivity fall, so that actual production also remains unchanged. This requires an increase in resource use, or activity levels, in the sector to offset reduced productivity.

In scenario 2, called "Maintenance of activity levels", we assume that the authorities opt for unchanged activity levels in agriculture if productivity is reduced. By "activity" we mean the overall use of inputs, including employment (number of person-hours worked) and real capital.[31] Hence in this scenario agricultural production is reduced in proportion to crop damage. It was suggested above that activity levels in agriculture could fall as a result of lower productivity, at the same time as other economic activity levels were stimulated. Since the scope of this mechanism is highly uncertain, and compensatory activity would probably require some government support in the same way as agriculture, we choose to disregard this factor.

The authorities seek to achieve the policy aims set for agriculture via various types of subsidies, and through prices set in the Main Agreement for Agriculture combined with high tariff rates. In plant production the subsidies are largely per unit of land, and not per unit of production as previously (Agricultural Budget Commission 1996). This is related to the goal of long-term food security and environment-friendly production. In the case of livestock products production subsidies persist, with an indirect bearing on grass production. Price-fixing through the Main Agreement for Agriculture also entails indirect support to the agriculture sector, inasmuch as prices in many cases are far higher than world market prices. This arrangement works in practice as production support. Part of the difference between the producer price and the world market price for grain is covered by the authorities in writing down the price of Norwegian grain supplied by the Norwegian National Grain Administration (Ministry of Agriculture 1994). The bulk of the difference however is borne by the consumer. The various support schemes in agriculture make it difficult to uncover the actual social cost of crop yield losses because the market price does not in general reflect the real shadow price of the crop. Hence we are compelled to take a basis in the policy goals for agriculture mentioned above in order to analyse the social cost of crop damage.

[31] In reality, the emphasis should be on employment. However, as the factor shares are fairly constant, we choose to focus on overall use of inputs for simplicity.

Valuing wheat and potatoes
The domestic price of wheat is generally far higher than the world market price. Whereas the Norwegian producer price has hovered around Nkr 2.50 per kg in the past five years (tending to edge down) (Agricultural Budget Commission 1995; 1996), the import price has averaged about Nkr 1 per kg. (Statistics Norway 1996a).[32] Although the price differences may conceal quality differences and different purchase times, this probably plays a small role for wheat. Hence the price difference, which is supported by high tariff rates and state price support is only assumed to reflect a value intrinsic to Norwegian production. The domestic price is fixed in the Main Agreement for Agriculture, and wheat production is consequently determined by the individual farm operator, based on profitability considerations (given other support schemes such as agricultural land area subsidies).[33] Imports of wheat and other types of grain cover just over half the total grain consumption in Norway (Statistics Norway 1994b).

Hence the shadow price of wheat is very dependent on the policy goal set for wheat production. In scenario 1, where production levels are in focus, the domestic price best reflects the direct cost of crop yield reductions, inasmuch as the authorities in this case wish to increase inputs in agriculture in response to crop damage. The shadow price for 1992 was Nkr 2.67 per kg (see table 4.4), and is taken from the Agricultural Budget Commission (1995). Since other support schemes, first and foremost agricultural land area subsidies, contribute to profitable operation of the individual holding, the real social cost is higher than the stated price. Land area support varies between regions and the size of the land area in question, and the rates for 1996/97 are estimated at Nkr 1 620-3 500 per hectare of grain (Norwegian National Grain Administration 1996). According to Statistics Norway (1996b), average yield of wheat per hectare in 1995 was 4 990 kg. In other words the implicit support per quantity of yield averages between Nkr 0.32 and 0.70 per kg. Inasmuch as the support is generally highest for farmland producing a small yield per hectare, support per kg of wheat varies widely. Compared with the producer price for 1992 referred to above, the land area subsidy increases the farmer's income from wheat production by an average of about one-fifth. This is captured by the macroeconomic analyses.

In scenario 2, where activity in the sector is in focus, production levels play a smaller role. In this case reduced production will be counterbalanced by increased imports of wheat, so that the world market price is the relevant shadow price. This applies even if the consumer has to pay a higher domestic price due to the customs tariff. According to external trade statistics (Statistics Norway 1996a), the average price for imported wheat (excl. tariff) was Nkr 0.83 per kg in 1992, i.e. less than one-third of the domestic price. In the years since, some increase in import prices and falling domestic prices have substantially narrowed the price difference.[34]

[32] The past year (1996), however, has seen a substantial increase in the world market price of wheat.

[33] In order to avoid overproduction, producers are charged if production exceeds a maximum level. The prices quoted are therefore the prices paid, and not pre-agreed prices (Agricultural Budget Commission 1996).

[34] In 1995 import prices were about half the domestic price (Agricultural Budget Commission 1996; Statistics Norway 1996a).

Table 4.4. **Shadow price for various types of crop in 1992 in various scenarios. 1992-Nkr per kg**

	Scenario 1 Maintenance of production potential	Scenario 2 Maintenance of activity level
Wheat	2.67	0.83
Potato	1.39	1.39
Grass	1.19	0.37

The producer price on potatoes is not regulated to the same degree as for types of grain. The price varies both over time and among regions, and with respect to quality. The average producer price can be calculated by dividing total sales income by total sales production (Agricultural Budget Commission 1995), giving Nkr 1.39 per kg of potatoes in 1992, which is employed as the shadow price in scenario 1 (i.e. unchanged production). The land area subsidy for potatoes is estimated at Nkr 1 860-1 970 per hectare for 1996/97 in southern Norway (Norwegian National Grain Administration 1996). Average yield per hectare in 1995 was 22 240 kg (Statistics Norway 1996b), such that implicit support per quantity of crop is Nkr 0.08-0.09 per kg. Thus for farmers in southern Norway the land area subsidy constitutes a far smaller component of income than the potato price.

Moreover, the domestic potato price is not very different from the world market price. According to Erdal and Nersten (1996) the price (cif) of Dutch and Danish potatoes averaged, respectively, 23 and 11 per cent less than the Norwegian producer price in the period 1990-1995. The price differences vary however from month to month, and in some cases the Norwegian price is lowest. Concurrently external trade statistics (Statistics Norway 1996a) show that the average price for imported potatoes (excl. tariff) was Nkr 1.74 per kg in 1992, i.e. 25 per cent *higher* than the domestic price. However, this is because imports, which in 1992 amounted to 10 per cent of Norwegian production (Statistics Norway (1996a); Agricultural Budget Commission (1995)), largely take place outside the high season for harvesting Norwegian potatoes. Thus, imports take place when prices are high both in Norway and abroad.[35] In 1995 60 per cent of imports were from Cyprus and Canada (Statistics Norway 1996c). It is therefore wrong to conclude that Nkr 1.74 per kg is the relevant shadow price for potatoes in scenario 2 (i.e. unchanged activity level). In the event of a shortfall in Norwegian production the import price at the time of the harvest must be used. Since no such explicit price exists, we use the same price as in scenario 1, i.e. the domestic price. This is probably on the high side.

Valuing grass

Grass is overall the most valuable plant product in Norway. It differs from wheat and potato in that it is not a final product, but is used as an input in livestock production. Furthermore, the farmer is usually self-sufficient in grass. This is because grass (both processed and unprocessed) has a high volume-to-weight ratio making it expensive to transport and store. It is therefore difficult to find a relevant market price for grass. Sales are minimal, and this does not reflect the value of grass. Since feed requirements are met by an appropriate mix of rough fodder (i.e. grass) and concentrates, unexpected production changes can be

[35] This is because potatoes are less durable than grain.

remedied by regulating the use of concentrates. Concentrates are produced from, among other items, barley and oats which are to some extent imported.

Livestock production is regulated to an even greater degree than plant production, and an explicit goal for Norway is self-sufficiency in livestock products. Moreover, for the farmer the returns on livestock husbandry are generally higher than on plant production. Hence a reduction in grass production as a result of ground-level ozone will not affect the volume of livestock production since the farmer has to cover the yield loss so as to meet his fodder requirements as previously. If a yield loss is anticipated, he can do this in several ways. In the first place the farmer can use more concentrates than previously, as is customary in the case of sudden yield losses. However, excessive use of concentrates is inadvisable, making this alternative problematic. The other possibility open to the farmer is to increase his pasture. This can be done either at the expense of other plant production, or by increasing total land use. In the first case it is natural to assume that land used for growing barley would be displaced. Alongside grass, barley is the most important plant product in Norway (in value terms)[36] and, as mentioned, is used in the production of concentrates. It is both propitious and relatively common to grow barley together with grass for rotation purposes since barley gives good protection to newly germinated grass-seed.

Against this background we calculate the shadow price in two different ways. In the first calculation we assume that the costs per hectare of producing barley and grass are virtually identical. This is because both types of plant are grown over much of the country, and because they are often grown in conjunction. Hence the wide regional cost differences do not give rise to large distortions. The second calculation assumes that the value per unit of fodder is identical for barley and grass, since both are mainly used to feed livestock (barley, however, has a lower protein content than grass and must first be processed into concentrates). In each case the shadow price of barley is used to estimate the shadow price of grass. The price of barley will however depend on which scenario we are studying.

Statistics Norway (1996b) provided data on yield per hectare of barley and cultivated meadow for the years 1988-1995. The ratio varies somewhat from year to year, but the average scaling factor in the period is 0.46. In other words the cost per kg of grass using the first method of calculation is assumed to be 0.46 times the cost per kg of barley. A unit of fodder for milk production is equivalent to 1.0 kg of barley and 1.9 kg of grass (Statistics Norway 1994b). Hence the value per kg of grass in the second calculation is assumed to be 0.53 times the value per kg of barley. We see that the two approaches give roughly the same ratio of cultivated grass to barley, and choose to employ the average of the two (= 0.495).

In scenario 1 the authorities wish to maintain domestic agricultural production. This entails increasing the total resource input in agriculture, including land use, as a result of crops being exposed to ground-level ozone. As in the case of wheat, the domestic producer price of barley is employed. According to Agricultural

[36] Incomes from barley production accounted for 45-52 per cent of total incomes from grain production in the years 1992-95 (Agricultural Budget Commission 1995, 1996).

Budget Commission (1995), the average price paid for barley in 1992 was Nkr 2.40 pr kg. This gives a shadow price for grass of Nkr 1.19 per kg. Here too, however, the social cost will be higher inasmuch as the land-area subsidy contributes to profitable production of both barley and grass for the individual farmer. According to the Norwegian National Grain Administration (1996), land-area support for rough fodder is estimated to range from Nkr 1 270 to Nkr 5 890 per hectare in 1996/97, whereas support for grain is put at Nkr 1 620 to Nkr 3 500 per hectare. Inasmuch as the yield per hectare is generally smaller than for wheat, at the same time as the price is lower, land-area subsidies constitute a larger share of the farmer's income in the case of barley production than wheat production. With our procedure this means that the real shadow price in table 4.4 is relatively speaking understated to an even greater extent for grass than for wheat.

In comparison, the average price of concentrates in 1992 was Nkr 3.36 per kg, according to the Agricultural Budget Commission (1995). If the unit value of rough fodder and concentrates is identical, this corresponds to a price of Nkr 1.89 per kg of grass, i.e. 60 per cent higher than the shadow price used by us. This suggests that it is more costly for the farmer to increase the share of concentrates than to cultivate more grassland at the expense of barley, which supports our assumption. However, in the event of a sudden occurrence of crop damage, the latter measure is infeasible.

In scenario 2 activity levels in agriculture are maintained at the same level as in the absence of crop damage, so that meadowland is increased at the expense of barley-growing land (see over). Reduced production of barley must then be compensated for by increased imports of barley (or other plants) for production of concentrates to maintain livestock production. Hence in this case the shadow price of barley is the import price. From Statistics Norway (1996a) we find that registered imports of barley in 1992 (and 1991) were so low that average import prices are uninformative. We therefore employ instead the average for corresponding prices in 1990 and 1993, which were respectively Nkr 0.80 and 0.70 per kg. The shadow price for grass in this scenario is then 0.37 per kg, i.e. about one-third of the price in scenario 1.

Macroeconomic analyses

When agricultural production is affected by ground-level ozone, the rest of the Norwegian economy is also affected. A correct estimate of the social costs of crop yield loss is only obtained when these effects are included. The effects are highly dependent on which scenario is in focus. If the authorities wish to maintain agricultural production at the same level as in the absence of ozone exposure (scenario 1), the agricultural sector must be supplied with labour and other resources from the rest of the economy. As a result production in other sectors will be reduced. Since substantially more resources are required to achieve a given production value in agriculture than in other sectors, this resource allocation will lead to an extra social cost over and above the direct value of the yield loss. Since the authorities wish to maintain the agricultural sector despite low productivity, there must be an intrinsic value, above that which shows in the accounts, linked either to production (scenario 1) or activity levels (scenario 2) in agriculture. Changed prices and income conditions will also affect the economy.

If the authorities choose to let activity levels (resource use) in agriculture remain unchanged (scenario 2), production in the sector will be reduced in the event of ozone exposure. Hence in this case there will be no transfer of resources from other sectors. However, since incomes in agriculture are reduced (because support remains unchanged) and the supply of domestic agricultural commodities diminishes, the rest of the economy is negatively affected in this scenario too.

In order to analyse these macroeconomic effects we apply the general equilibrium model MSG-EE (Alfsen et al. 1996), which is outlined in chapter 2 of this book. In this macro model resource allocation between various sectors may be studied. This is particularly important in our analysis because of the productivity differences mentioned above. However, modelling the agricultural sector within the macro model is straightforward. The sector by and large produces one type of commodity, called agricultural products, which can be perceived as a kind of average commodity in the sector. This commodity, together with imports of agricultural products, is demanded for various applications in the model. A large portion of agricultural products are further processed in a separate sector devoted to processing agricultural and fish products which also by and large produces one type of commodity. Another portion of agricultural products is used as inputs in the agricultural sector itself. This applies particularly to feed for livestock production. While some agricultural products go directly to food consumption, a larger share of consumption is via the sector that processes the products. A smaller share of agricultural products is exported. Production value in the agricultural sector is determined outside the model, since it is largely regulated by the authorities and is independent of economic factors. However, input use in the model changes somewhat with variations in factor prices. Imports of agricultural products are also endogenous, and depend on demand for agricultural products and on domestic production.

Because of the model's structure it is difficult to distinguish the individual crop yield losses from other types of production loss in the agricultural sector. Since both production structure and application of the products can vary somewhat, particularly where plant contra livestock production is concerned, the analysis may give a distorted picture of changes at the sector level. The model's most important contribution is however linked to the main economic aggregates such as GDP and private consumption. Here the model supplies valuable information.

Crop damage is modelled in such a way that productivity in agriculture is reduced; in other words the ratio between production value (at constant prices) and level of inputs diminishes. In scenario 1 the production value in agriculture is fixed such that it is at the same level both with and without crop damage. This entails that at given factor prices the use of all inputs rises proportionately if damage occurs. In scenario 2 the production value in agriculture is determined in such a way that the difference in level between a situation with crop damage and one without crop damage corresponds exactly to the value of the damage. This opens the way for higher imports in the case of ozone damage. Inasmuch as grass is not traded in the market, and hence does not show directly in the accounts, it may at first glance seem unreasonable to change the production value due to damage to

meadowland. However, we have assumed (see section 4.3.1) that the farmer desires an appropriate mix of rough fodder and concentrates, so that ozone damage leads to an increase of meadowland at the expense of areas used for barley production. As a result trading in barley is reduced, and production value declines. At given factor prices, the use of inputs in scenario 2 is identical regardless of whether the damage occurs or not.

State agricultural subsidies are assumed to be in proportion to activity levels in the sector. This is because a large share of the support is in the form of land-area subsidies, which are independent of production volume. However, the market regime for grain means that the support via the Government Grain Corporation depends on the volume of production. On the other hand it is conceivable that the authorities will wish to compensate for the farmers' income loss by raising the rates of support. Our supposition entails that scenario 1 increases the support in proportion with the increased resources used when ozone damage occurs. In scenario 2 the level of support remains unchanged because resource use is assumed to remain unchanged. The governmental overall net income is assumed to be unchanged, and the payroll tax rate is chosen as the endogenous variable.

4.3.2. Direct costs of crop damage

The direct costs of crop damage can be calculated by multiplying the yield losses set out in table 4.3 with the shadow prices in table 4.4 (adjusted to 1995-Nkr). As explained above, the costs depend on how the authorities respond to crop damage, and we therefore distinguish between scenario 1 and scenario 2.

It will be seen from the table that the direct costs are very dependent on the authorities' response, which in turn depends on what policy aims the authorities have for agriculture in Norway. If the authorities choose to allow activity levels in agriculture to remain unchanged (scenario 2), the direct costs are about Nkr 200 million. This is because the yield loss is compensated for by relatively cheap imports. In this case costs of reduced grass production make up about 64 per cent of total direct costs, while reduced wheat and potato production accounts for 13 and 22 per cent respectively. If the authorities choose instead to raise activity levels in order to maintain domestic production (scenario 1), costs come to about Nkr 550 million. This is what it costs to produce the concrete yield loss in Norway. In this case the costs of reduced grass production make up 76 per cent of total direct costs, while reduced wheat and potato production now account for 16 and 19 per cent respectively. As mentioned in section 4.3.1, no account is taken of land area support that comes in addition to the producer prices. The real costs are therefore somewhat higher than Nkr 550 million. However, this is captured in the macroeconomic analyses in the next section.

In 1992 the production value in agriculture was 27.2 billion 1995-Nkr (Statistics Norway 1995c). Since production is mainly measured by the same prices as stated for scenario 1, we find that the yield loss corresponded to 2.0 per cent of the potential agricultural production (i.e. in the absence of crop damage). This is attributable to yield loss of respectively 11, 8 and 7 per cent of the potential yield of wheat, grass and potatoes in 1992 (see table 4.3). Gross product, defined as production value less the value of purchased input products, is a better

Table 4.5. **Direct costs of crop damage resulting from ground-level ozone, 1992. Mill. 1995-Nkr**[1]

	Scenario 1 Maintenance of production potential		Scenario 2 Maintenance of activity level	
	Mill. 1995-Nkr	Share (per cent)	Mill. 1995-Nkr	Share (per cent)
Wheat	87	16	27	13
Potato	48	9	48	23
Grass	420	76	131	64
Overall total	555	100	206	100

[1] The consumer price index is used to index current prices to 1995-Nkr (Statistics Norway 1996b).

measure of value-added in the sector, as it shows the value increase produced by labour and real capital in the sector. In 1992 the agriculture sector's gross product came to 13 billion 1995-Nkr (Statistics Norway 1996b).

If yield loss is viewed in a short-term perspective, i.e. as a one-time occurrence, scenario 1 becomes less relevant. This can to a degree be defended since 1992 was a year of high ozone exposure. As a one-time occurrence the yield loss would probably be compensated for by higher imports so that the result in scenario 2 is representative for the costs. However, since the farmer will in this case increase the share of concentrates, which are dearer per unit of feed than rough fodder, the costs will probably be somewhat higher than illustrated by scenario 2. Further additional costs, such as adjustment costs, will also be associated with a sudden yield reduction of this kind. Moreover, income lost in agriculture will distort national income distribution.

4.3.3. Macroeconomic effects of crop damage

The macroeconomic effects depend as mentioned on how the authorities respond, which again depends on which policy aims are emphasised for agriculture. In the main we allow changes in gross domestic product (GDP) to be estimates for the total social costs of crop damage. GDP equals the sum of gross product in all sectors, and is therefore a measure of total value-added in the economy. However, as shown earlier, the GDP concept may give a distorted picture of value-added in agriculture, inasmuch as the product prices on which it is based may differ from the real shadow prices. Gross product in agriculture is therefore dealt with separately. The results are shown in table 4.6.

It is seen in scenario 1, "Maintenance of production potential", that GDP is reduced by the considerable margin of Nkr 1.2 billion, which is more than double the direct costs in table 4.5. Almost three quarters of the loss in GDP is incurred in sectors other than the agriculture sector, since resource use in agriculture has to be increased to compensate for the yield loss. As a result resources are withdrawn from other economic activity which (in the accounting context) are far more productive. Given the authorities' choice of response in this scenario, the results may be interpreted to mean that agricultural production has far greater real value than the GDP concept indicates. Even if agricultural production is maintained, gross product in the sector falls by 3-4 per cent. This is because farmers have to purchase more inputs in order to maintain the same production level, thereby reducing value-added in the agriculture sector.

Table 4.6. **Changes in macroeconomic aggregates in connection with crop damage resulting from ground-level ozone, 1992. Mill. 1995-Nkr**

	Scenario 1 Maintenance of production potential		Scenario 2 Maintenance of activity level	
	Mill. Nkr	Per cent	Mill. Nkr	Per cent
Gross domestic product (GDP)	-1 236	-0.2	-730	-0.1
Gross product in agriculture	-333	-3.6	-554	-5.8
GDP excl. agriculture	-903	-0.1	-176	0.0
Gross production value in agriculture[1]	0	0	-555	-2.3
Imports of agricultural commodities	122	1.2	486	4.8
Private consumption of foodstuffs	-41	-0.1	-1	0.0
Person-hours worked in agriculture (1 000 hrs)	3 369	2.4	223	0.2
Government transfers to agriculture	157	2.4	0	0

[1] The model's reference path gives a somewhat smaller production value (and gross product) in agriculture (to be exact 23.1 bn 1995-Nkr) than the amount mentioned in section 4.3.2 (i.e. 27.2 bn 1995-Nkr). Hence the yield loss measures 2.3 per cent of production rather than 2.0.

The resource allocation is illustrated by the fact that employment in agriculture increases by almost 3.5 million person-hours in this scenario. The relative increase roughly equals the relative decline in productivity. Increased resource use also means more inputs, consisting inter alia of agricultural commodities (e.g. concentrates). This explains why imports of agricultural commodities increase even when production is maintained. Such an outcome may seem unreasonable in this scenario, and a better approach could have been to allow these imports to be unchanged. Agricultural production would then have attracted even more domestic resources, and the fall in GDP would have been even greater.

Private consumption of foodstuffs is slightly reduced in this scenario, partly because agricultural production demands higher inputs of processed agricultural commodities. Prices of such commodities are consequently somewhat higher. Concurrently reduced GDP leads to lower incomes which is reflected in lower demand for all normal goods. Government subsidies to agriculture rise by about Nkr 150 million. This builds on our assumption of proportionality between activity levels and size of support.

In scenario 2, "Maintenance of activity levels", GDP is reduced by just over Nkr 700 million. The biggest reduction is in the agricultural sector, where gross production is reduced by 6 per cent. This is because production value is reduced by about 2.3 per cent, at the same time as use of inputs shows negligible change.[37] The relative reduction of value-added, or gross product, is therefore quite large. Compared with the direct costs of around Nkr 200 million in table 4.5, the decline of about Nkr 550 million in the agriculture sector's gross product seems large. This is because the shadow prices in scenario 2 are largely based on world market prices, while gross product is measured by domestic prices. We interpret this scenario such that in the event of marginal changes in activity and production, activity levels in agriculture have a greater intrinsic value

[37] Actually employment rises by 200 000 person-hours, whereas other inputs are somewhat reduced (e.g. real capital).

than production (beyond the world market price). In this case it may therefore be asserted that the reduction in gross product overstates the real cost of reduced value-added in the sector by about Nkr 350 million (i.e., the difference between reduced gross product in agriculture in table 4.6 and the direct cost in table 4.5). The real social costs in this scenario are accordingly about Nkr 380 million, i.e. *far lower than the reduction in GDP*.

Gross product in other sectors is reduced by Nkr 150-200 million, despite the fact that the agriculture sector does not lay claim to more resources. There are several reasons for this. First, the yield reduction leads to reduced incomes for farmers (government subsidies are unchanged in this scenario), so that total demand for goods in the economy is reduced. Second, imports of agricultural commodities rise by about 5 per cent due to yield loss. As a result the balance of trade is disrupted, and other imports have to be restricted to compensate (in the event, exports must be increased). This entails restrictions for the rest of the economy.

Private consumption of foodstuffs remains roughly unchanged despite the fact that incomes are reduced as a result of the drop in GDP. This is because food prices fall by 0.1 per cent since a larger share of agricultural products are imported.

A summary of the economic analyses suggests that the overall social costs are highly dependent on how the authorities respond to reduced plant production resulting from ozone exposure, see table 4.7. If the authorities choose to let activity levels in agriculture remain unchanged, and to compensate for the yield loss by higher imports, the costs come to about Nkr 400 million, according to our calculations. If,

Table 4.7. **Total social costs of crop damage resulting from ground-level ozone, 1992. Mill. 1995-Nkr**

	Scenario 1 Maintenance of production potential	Scenario 2 Maintenance of activity level
Direct costs	555	206
Indirect costs	681	176
Total costs	1 236	382

instead, the authorities choose to increase activity levels in agriculture so that production levels remain unchanged, we find that costs are of the order of Nkr 1.2 billion, i.e., three times as large. Moreover, note the importance of studying the indirect macroeconomic effects. In scenario 1 these effects actually constitute a larger share of total costs than do the direct costs. In scenario 2 the macroeconomic costs also constitute a considerable share.

4.4. Conclusion

In this study we have made an attempt on the basis of current knowledge to elucidate what consequences ground-level ozone may have for farm crops in years featuring very high ozone concentrations in Norway. Before summing up our conclusions, some of the main uncertainties in the calculations are set out. It is important to have these in mind when considering our results.

First, the assessments are based on the year 1992 when exposure doses of ground-level ozone in Norway were particularly high. It is not clear to what degree the results are relevant for more normal years. Second, the damage functions employed are in general highly provisional since documentation through crop experimentation is very limited with the possible exception of wheat. Wide variation in ozone-sensitivity between plant types within a species makes it difficult to arrive at correct damage functions. Moreover,

we know that various climatic factors impact on the ozone effect, but this has yet to be documented quantitatively. Third, the representativeness of monitoring sites for ground-level ozone has not been quantified, and the monitoring network is relatively sparse. Ozone gradients over vegetation that are affected by vegetation type, topography and meteorological conditions also make it complicated to calculate correct exposure doses to which vegetation is exposed. Fourth, the authorities have several policy aims for agriculture, and it is therefore unclear how they would respond to reduced yields over a long period. Moreover, as the agricultural sector is highly regulated, it is difficult to bring to light the real economic value of crop yields. Finally, macroeconomic models attempt to describe how players in the economy act, a process which is naturally extremely complex.

Thus, these factors indicate that as our knowledge increases the estimates for crop damage will probably change substantially. Our provisional results indicate that in 1992 ground-level ozone entailed a loss of about 30 thousand tonnes of wheat, and about the same volume of potatoes. As regards cultivated meadow, ozone exposure led to a loss of just over 300 thousand tonnes of grass, according to the results. These losses correspond to about 10 per cent of a normal annual yield. The size of the losses is related to the fact that almost 90 per cent of the total wheat-growing area experienced ozone exposures in excess of recommended critical levels, while the corresponding shares for potatoes and cultivated meadow were just under 60 per cent.

The further calculations indicate that the economic cost of yield loss caused by ground-level ozone in 1992 was in the region of Nkr 400 - 1 200 million. The wide variation is due to two alternative assumptions regarding the authorities' policy aims for the agricultural sector. The lowest cost emerges when the focus is on activity levels in the sector. The yield loss in this case entails reduced production, which is compensated for by increasing imports of agricultural products. The highest cost emerges when maintenance of production levels is regarded as especially important. In this case the ozone exposure entails withdrawal of resources from other sectors to the agriculture sector in order to compensate for the crop damage. Since far more resources are needed to attain a given production value in agriculture than in other sectors, the total costs of the yield loss are extra large in this case.

In both cases the direct costs make up only about half of the total costs. This demonstrates the importance of analysing macroeconomic impacts in such calculations in order to study spillover effects in the economy. In the case of unchanged production the total costs are as much as 130 per cent higher than the direct costs owing to productivity differences among the various sectors.

Because of the substantial uncertainty associated both with physical and economic calculations, the social costs must be viewed as rough estimates. However, they provide a good indication that long-range pollution in the form of ground-level ozone gives rise to major costs for Norway in years of high ozone concentrations. This underscores the authorities' view that it is “necessary to reduce the environmental burden inflicted on agricultural land by, for example, air pollution” (Ministry of Agriculture 1992, chapter 3.1.2).

5. Health effects of air pollution and impacts on economic activity[38]

Knut Einar Rosendahl

5.1. Introduction

5.1.1. Background

It has long been clear that exposure to various polluting gases and particulates may give rise to, or exacerbate, a variety of respiratory diseases. Greater uncertainty however has attached to the level of risk of incurring such ailments. Recent research have brought increased knowledge in this field. Numerous epidemiological studies have pinpointed so-called dose-response functions, i.e. statistical relationships between air pollution levels and morbidity and mortality. These functions can be used to compute associations between economic activity, pollution and health effects. Some health effects also have a feedback-effect on the economy in that increased morbidity leads to workforce reductions and lower worker productivity.

This study examines the international literature dealing with associations between air pollution and health effects, and shows how such associations can be utilised to estimate the feedback-effects of pollution on economic activity. Moreover, concrete calculations of health effects in Oslo resulting from the current pollution situation are presented together with associated costs. Alongside the dose-response functions figuring in the international literature, use is made of functions for the concentration of air pollutants (particulates and NO_2) in Oslo at various emission levels. These functions have been constructed by the Norwegian Institute for Air Research (NILU) at the request of Statistics Norway (Walker 1997).

Previous Norwegian studies of health costs of pollution were based largely on the Norwegian Pollution Control Authority's (SFT's) analyses of pollution abatement measures for the capital Oslo (SFT 1987) and the small neighbouring towns Sarpsborg and Fredrikstad (SFT 1988)[39]. These analyses were in turn based on epidemiological studies by Lave and Seskin (1970), and were indirectly linked to numbers of persons exposed to SO_2 concentrations in excess of the SFT's recommended threshold values. The cost estimates were based on contingent-valuation studies, and costs

[38] The article has earlier been published in Norwegian in Rosendahl (1996).

[39] See for instance Brendemoen et al. (1992).

associated with other pollutants were related to SO_2 costs by a panel of experts.

There are several reasons why it is now time to update health costs in Norway. First, the focus on SO_2 in the studies carried out in the seventies has been called into question; internationally attention is now primarily directed at various particulate categories and ozone since these components seem to correlate best with severe health effects. Based on this, Hall et al. (1992) have calculated the health benefits of cleaner air in the Los Angeles area. They found that the gain achieved by reducing pollution levels to national standards would amount to USD 6.4 billion and USD 2.7 billion for particulates and ozone respectively (best estimate). Other studies (e.g. EC 1995, ORNL/RFF 1994) also find health costs to be greatest for particulates and thereafter ozone. However, it should be realised that these components are indicators for a complex pollution situation and that direct causal relationships are difficult to prove. Hence the possibility that other components, such as SO_2 and NO_2, are just as harmful as particulates and ozone cannot be disregarded.

Second, there is little reason to focus particularly on specific threshold values for air pollution in a population; such values are specific to individuals, and so far it has not proved possible to document any lower level of concentration for health effects that is applicable to all individuals. This is especially relevant for particulates where the results from a number of epidemiological studies have prompted an expert group attached to the European Office of the World Health Organisation – in a new report on guidelines for air quality, WHO (1995) – to no longer recommend guidelines for particulates.

Third, recent literature (see for instance Ostro (1993)) has provided a broader basis for identifying dose-response functions that can be utilised directly in analyses of health effects. Unlike in earlier Norwegian studies it is now possible to calculate effects of various pollution levels on mortality, lost labour productivity and public health expenditure etc. Hence it is also possible to distinguish between purely economic costs and other costs which must be valued separately (e.g. the value of reduced mortality).

Recent years' increasing awareness of associations between air pollution and health effects has been accompanied by major international efforts to compute external costs of various types of electricity production. Two wide-ranging projects carried out by the US Department of Energy (ORNL/RFF 1994) and the EU Commission (EC 1995) are particularly significant. Dose-response functions were central to their analyses. These studies, together with Ostro (1993,1994), a study from the US Environmental Protection Agency (EPA 1995), and draft reports by the National Institute of Public Health in Norway (1995), form much of the background information for the present study.

5.1.2. Dose-response functions

There are two main methods for uncovering links between air pollution and health effects. One involves clinical studies where volunteers (or animals) are placed in a chamber containing a high concentration of a certain airborne pollutant and subsequently examined for various health effects, e.g. impaired pulmonary function. While such studies can be used to demonstrate biological effects, they throw no light on the frequency or risk of various health effects in a normal pollution situation. One reason

for this is that the situation in a chamber differs greatly from the real situation in an urban atmosphere. There are also constraints on the types and extent of health effects that lend themselves to experimentation in a chamber (c.f. sickness absence and mortality).

The second method is epidemiological studies where an area is examined for statistical relationships between air pollution levels and the incidence of certain health effects in a sample of the population. These studies can be used to formulate quantitative associations between air pollution and health effects, i.e. dose-response functions. All functions presented in this study stem from such studies, and they can in general be expressed in the following form:

(5.1) $\Delta H = F(\Delta C) * P$

where ΔH is the change in frequency or risk of a specific health effect (i.e. mortality, sickness absence), ΔC is change in concentration of a certain atmospheric pollutant (e.g. mean annual concentration of particulates), and function F denotes the estimated quantitative relation between these two. P is the number of persons living in the area concerned[40]. In most cases the estimated relation can be expressed as a linear function. The actual relation will of course be more complex.

The health damage that arises may have effects on economic activity, e.g. via increased sickness absence. This can in general be expressed as

(5.2) $\Delta E = G(\Delta H)$

where ΔE denotes change in the level of a specific economic variable, and function G denotes the quantitative relationship between this and the resultant health impairment. Whereas the functions in equation (5.1) stem from international studies, the functions in equation (5.2) are constructed on the basis of various Norwegian statistics and various choices of assumptions.

By combining these two functions we obtain the following general relationship between the concentration of air pollution and economic activity[41]:

(5.3) $\Delta E = G(F(\Delta C)*P) = J(\Delta C,P)$

We distinguish between acute and chronic dose-response functions, or effects. By acute effects we mean health effects that occur after short-term exposure to air pollution. In this case most epidemiological studies utilise average concentrations over one or more days as an indicator of the pollution level. Chronic effects refer to health effects that occur after long-term exposure, i.e., one or several years. These are only referred for particulate matter.

Pioneering studies of dose-response relationships between air pollution and health were carried out by Lave and Seskin (1970, 1972). Since then a variety of similar studies have been carried out which will be discussed more fully in the forthcoming sections. Since the end of the 1980s both the number and quality of such studies have made substantial advances. International confidence in their findings has grown, partly because in many cases they support each other to a surpris-

[40] In some functions the left-hand side in (5.1) is $\Delta H / H$, and P is removed from the right-hand side.

[41] In some functions the left-hand side in (5.3) is $\Delta E / E$, and P is removed from the right-hand side.

ingly large degree. It is also interesting to note that the studies have been performed in very different areas in terms of pollution levels, climate etc.

The quality of the studies has improved in several ways. First, more and more studies are using time series data, particularly to reveal acute effects that arise with short-term changes in pollution levels. The advantage of using time series data is that population-specific factors which also have a bearing on health are fairly constant: these include socioeconomic factors, smoking habits, state of health etc. The most important remaining factors are the environmental and meteorological ones.

Second: an important criticism that can be levelled at earlier studies is the varying degree to which they managed to take the various pollution components into account. The studies by Lave and Seskin have for example been interpreted to demonstrate the existence of a link between exposure to SO_2 and mortality. However, they used an aggregated pollution index, and subsequent studies indicate that the strongest association is between particulate concentration and mortality (and other health effects). EC (1995) comment that in several studies SO_2 effects apparently disappear when particulates are measured correctly. On the other hand, a number of recent studies exist where this is not the case (see section 5.4). Most epidemiological studies now estimate with respect to several pollutants simultaneously. Some studies find separate effects of particulates and NO_2 respectively (e.g. Braun-Fahrländer et al. 1992), implying that it ought to be possible to add them together. In other cases it shows difficult to find separate effects of different pollutants as these are often strongly correlated (Calthrop and Maddison 1996). One study may find significant relationships between particulates and a health impairment while another study finds significant relations between NO_2 and the same health impairment. Interpretation then becomes more problematic. The interaction between different components is altogether complex. According to EC (1995) this is primarily a problem for particulates, NO_2 and SO_2, whereas ozone effects can reasonably be considered as additive in relation to the other components. Thus, awareness of the problem of double-counting must be maintained. There is, however, little danger of overlapping between the associations we use in the calculation of social costs in this study.

The growing number of studies allows a more discriminating choice to be made. Ostro (1995) explains the criteria that had to be applied to make use of a study in connection with ORNL/RFF (1994): all dose-response relations available in the specialist literature were studied, including relations linked to mortality and morbidity. The relations were assessed on the basis of criteria such as generalisation (for application in other towns) and significance. The same procedure has been applied in other applications, such as EC (1995). Hence the dose-response functions taken from these studies have been thoroughly assessed in internationally recognised studies. Other functions utilised in our studies have also been thoroughly assessed by recognised specialist bodies, such as EPA (1995).

Although relatively few epidemiological studies have found significant associations between NO_2 and health effects, there are several reasons to warn against toning down the importance of NO_2. First, the mix of air pollutants in Norway differs in

some respects from the mix in the USA and on the continent where epidemiological studies have largely been carried out. Moreover, several Finnish studies indicate that NO_2 may have serious consequences in a climate resembling that in Norway (see section 5.3). Another important reason why NO_2 has been considered harmful is health effects observed in clinical studies and in studies of indoor pollution.

The difficulties associated with demonstrating quantitative relationships between air pollution and health effects (see for instance Ostro (1994)) are worth emphasising in this context. It is conceivable that measurement problems explain why effects of outdoor NO_2 are frequently unidentifiable. If effects primarily arise as a result of short-term exposure to particularly high concentrations (e.g. close to a heavily trafficked road), the effects will be highly dependent on individual exposure which may be difficult to measure. The same applies if the health effects arise as a result of several years' exposure.

A pertinent question is whether results from epidemiological studies carried out in one area can be applied in other areas. This is a relevant concern since the great majority of studies referred to in the literature have been carried out in the USA. Commenting on this, Ostro (1995) recommends that further studies of a similar nature be carried out in Europe. He also states that the perceived uncertainties should not prevent the results observed in the USA from being transferred to European cities with a view to estimating social costs of pollution in Europe. He bases this view on the fact that the studies utilised for instance in ORNL/RFF (1994) and EC (1995) have made allowance for disturbing factors in their estimations, and that they are mutually supportive (especially for mortality). Moreover, they have been carried out in widely differing climatic areas. A number of studies carried out in Europe and other parts of the world in recent years are also worth noting. We cite studies in for example Finland, Germany, Switzerland, Spain, the United Kingdom, Chile and China that essentially confirm the US results.

Pearce (1995) addresses the uncertainty aspect in his valuation analysis, and asserts that any uncertainty involved in the transfer of dose-response functions is of a minor nature. EC (1995) also touches on this uncertainty, but the authors are content to state that the functions describing a biological response, such as mortality, are to a higher degree transferrable to other towns than functions which also describe a social response, such as hospital admissions. However, this is not to say that EC (1995) omits the latter type of functions.

When applying dose-response functions in the present study the effects are accumulated over a year and expressed as the effect of changes to mean annual concentrations. This is common practice in similar applications (EC (1995); ORNL/RFF (1994)). It presupposes that associations between air pollution and health damage are approximately linear, and that they are relevant for the pollution level at which the functions are applied. The first condition is met in the sense that the original studies largely found linear or approximately linear functions that correspond significantly with the observations[42]. There are, however, grounds for

[42] A number of the functions are linear on a percentage basis; within the area of application these functions are approximately linear inasmuch as the pollution level only explains a small share of the particular health effect.

suspecting that the actual relationship may be more complex than this. A possible objection is that acute health damage may only arise with episodes of high pollution over several days, and that pollution levels are otherwise of little significance. Whether or not this is correct is somewhat unclear. However, since approximately linear functions seem to correspond well with the observations it is reasonable to use them. Moreover, there is reason to believe that episodes of heavy pollution are strongly correlated with the annual mean concentration. As for the second condition and the question of threshold values, these are discussed for each individual component in the following sections. It has already been pointed out that in the case of particulates the dose-response functions also seem to apply at very low concentrations.

We present dose-response functions for particulates, NO_2, ozone and SO_2. In the application for Oslo, however, we concentrate on the two first-mentioned. Episodes of high ozone concentrations in Norway are mainly due to foreign emissions, although Norwegian emissions of NO_x are of some significance (see Simpson et al. 1996). SO_2 concentrations are relatively low in Oslo, and it is unclear whether they have a separate health effect beyond that of particulates. Since the present study is especially concerned with effects on economic activity, the selection of dose-response functions presented will by no means be complete (see Aunan (1995) for a similar review of the literature from a somewhat different angle). However we also focus on serious health effects which are not necessarily significant for economic activity, such as higher mortality among the elderly and chronically ill. In that connection we discuss to some extent economic valuation of such effects.

Although a number of quantitative associations between pollution levels and health effects have been demonstrated, there is no reason to believe that all the ground has been covered (see e.g. Ostro (1994)). As mentioned above, associations may in many cases be difficult to find. The few studies that have so far found links between long-term exposure and various health effects suggest that a substantial volume of effects remains to be clarified. In other words, using quantitative relations to calculate costs of pollution will in all probability yield an underestimate.

In the next sections dose-response functions based on international literature is presented for the four pollutants. Moreover, we analyse the economic impacts following from health effects induced by air pollution. Threshold values are discussed in the end of each section. In section 5.6 the population exposure to air pollution in Oslo is discussed, and we present annual health effects and social costs in Oslo based on the dose-response functions.

5.2. Health effects of particulates

The term particulates includes a number of chemical compounds, among them sulphates and nitrates. However, size is what is considered most important in a normal pollution situation[43]. Particulates with a diameter less than 10μm (PM_{10}) are mainly considered harmful to the lower respiratory tract. In the upper respiratory tract larger particulates may also be harmful. PM_{10} further comprises coarse particulates and fine particulates, the latter with a diameter below 2.5μm ($PM_{2.5}$).

[43] If the particulates contain toxic heavy metals owing to emissions from a particular industrial source, the chemical composition may be just as hazardous as size.

These are thought to be of the greatest significance for health since they penetrate deeper into the respiratory tract. Recently discussion has turned on whether PM_1 or even $PM_{0.1}$ is most important. Particulates are carriers of other pollutants, among them SO_2, and such combinations are also considered important. The main sources of fine particulates are combustion processes, especially related to vehicle traffic, residential heating and, in some towns, port traffic. The sources of coarse particulates are largely mechanically generated, e.g. by use of studded tires. Studies of dose-response relations linked to particulates have been carried out using various choices of target variables such as $PM_{2.5}$, PM_{10}, TSP (total suspended particles) and sulphates. In most applications of dose-response functions the various variables are converted to PM_{10}, on the assumption of fixed relations between the various particulate sizes.[44] This is a simplification inasmuch as composition may vary from town to town.

In the following we distinguish between acute and chronic effects. Each health effect will be dealt with in a separate subsection. Appendix B contains a table summarising the dose-response functions taken from the literature and a table showing the impact of the health effects on economic activity, etc.

5.2.1. Acute effects

Premature mortality
Studies that have found a significantly higher risk of death on days (or in periods) of high particulate concentration (see for instance Plagiannakos and Parker (1988) and Schwartz (1993b)) are now in double figures. Moreover, the results from most of these studies are strikingly uniform, despite the fact that the underlying observations have been made at widely different levels of concentration, climatic conditions and mix of other pollutants. Some of the studies build on time series data, while others build on cross-sectional data.

Based on a number of these studies, Ostro (1993) concludes that the consensus dose-response relationship between mortality and short-term change in particulate concentration is:

$$(5.4) \quad \Delta D / D = 9.6*10^{-4} * \Delta PM_{10}$$

where D denotes the number of deaths in the period, while ΔPM_{10} denotes change in PM_{10} concentration (period mean, $\mu g/m^3$). In this context, period means one or more days, since the studies focused on short-term changes in concentration. The lower and upper limits for the coefficient are respectively $6.3*10^{-4}$ and $1.3*10^{-3}$. In other words *an increase of 10$\mu g/m^3$ in PM_{10} concentration is associated with an increase of about 1 per cent in the number of deaths.* Pearce (1995) states that a linear relation appears to be soundly documented. He bases this on the fairly high level of consistency shown by the studies despite their having been carried out at different concentration levels. A new study from Chile (Ostro et al. 1995) confirms the dose-response function mentioned, indicating that this function applies in large measure universally.

In some of the studies it is possible to isolate various causes of death, and it turns out that a link exists between deaths from respiratory ailments and increases in

[44] The relations between $PM_{2.5}$ and PM_{10}, and between PM_{10} and TSP, are assumed to be 0.65 and 0.55, respectively (ORNL/RFF 1994). These, according to the Norwegian Institute for Air Research, are also fair conversion rates for Oslo.

particulate concentration. This accords with the results for inter alia hospital admissions in section 5.2.1.

The degree to which increases in particulate concentration cause an increase in the mortality rate in that year is uncertain. The reason is uncertainty as to how long those affected would otherwise have remained alive. Rowe et al. (1995) refer to a study by Schwartz and Dockery (1992) which found that the risk of mortality at increased concentrations of PM_{10} is more than 70 times higher for individuals above 65 than for individuals below 65. However, this figure must be viewed in the light of the higher general mortality in the oldest age group. The National Institute of Public Health (1995) refers to a study in the Czech Republic indicating that deaths due to pulmonary disease among children aged one month to one year rise by 58 per cent with a 10 $\mu g/m^3$ increase in PM_{10} concentration. Clearly this particular group has a long residual lifetime. All in all the results indicate that it is primarily the chronically ill and the elderly who are affected, but that residual lifetime is not insignificant (Schwartz and Dockery 1992).

This must however be collated with recent results for the relationship between long-term particulate exposure and mortality which are discussed in section 5.2.2. Although the uncertainty here is larger, they at any rate indicate that particulate pollution (short-term or long-term) leads to several years' loss of lifetime. Pearce (1995) also refers to a study by Cropper and Simon (1994) which states that probable residual lifetime is 10 to 15 years in the case of deaths resulting from pollution.

The fact that particulates include components with various chemical and physical characteristics, and that particulates generally carry other pollutants, has prompted discussion about real causal relations. Perceptions variously focus on the importance of fine particulates ($PM_{2.5}$), acid aerosols (sulphates/nitrates) or SO_2. A study by Pope et al. (1992) is especially pertinent in this respect. This study was carried out in the winter half-year in Utah Valley where there was little SO_2 and ozone in the atmosphere, and where no measurable acidity was attached to particulates. The result for mortality from this study was somewhat higher than the interval for the dose-response function in equation (5.4).[45] This and similar studies (e.g. Fairley 1990) indicate that a causal relationship exists between (at any rate) particulates, measured in size, and mortality. Ostro (1995) also asserts that the numerous studies showing consistent relations between PM_{10} (and TSP) and mortality (and morbidity) across climate, season, other pollutants and population, strongly suggest that the causal component is closely correlated with PM_{10} (and TSP), although many of them do not include acid aerosols. However, a causal connection can never be proven by epidemiological studies.

Ostro (1993) states that most criteria for causality, as established by Hill (1965), are satisfied by the dose-response function in equation (5.4). This entails, briefly, that the relation between particulates and mortality is consistent, that it is disease-specific (linked to respiratory diseases among others), that the relation generates a dose-response curve, and that it accords with observed effects on other health parameters. The only criterion which

[45] The increased mortality is due to cardiovascular disease, pneumonia and chronic obstructive respiratory disease.

according to Ostro is not satisfied is the biological mechanism between pollution and mortality. However, theories exist (e.g., Seaton et al. (1995)). Ostro (1993) specifies that not all the criteria have to be met in order to establish a strong indication of a causal relation.

It is reasonable to believe that the effects on mortality discussed in this section have negligible impact on economic activity through a reduced work force. Persons who die due to a short-term increase in pollution levels are mainly elderly and chronically ill and are outside the workforce at the outset. The prime exception here is sick children who could have recovered if they had been able to grow up. However, this group represents a marginal share of deaths. Hence most studies which attempt to calculate the costs of increased mortality opt for an estimate for the value of a statistical life. Effects on economic productivity are not considered. However, in section 5.2.2 such effects are discussed for chronic effects on mortality risks.

Sickness absence and reduced labour productivity

Relatively few studies have estimated the relationship between air pollution and sickness absence or reduced labour productivity. Most relevant studies in this field are those carried out by Bart Ostro on an extensive annual US health survey (120 000 persons in 84 urban districts). The first study utilised data material from 1976 (Ostro 1983). Significant relations were observed between particulate concentration and the number of restricted activity days (RAD). In a later study a specification test was carried out on the 1976 result for the years 1976-1981 (Ostro 1987). This study confirmed the results from the first study in all years.

Based on these results, Ostro (1994) himself arrives at the following relationship between PM_{10} concentration and RAD (best estimate), which is the one used in all the cost studies referred to in the introduction.

$$(5.5) \quad \Delta RAD = 0.058 * \Delta PM_{10} * P$$

RAD denotes the number of restricted activity days per year, and *P* is the number of persons exposed. Here PM_{10} denotes mean annual concentration.

The relationship applies to all adults. The function states that a one-unit increase in PM_{10} concentration entails an increase in restricted activity days of 0.058 days per person per year.

Subsequent studies have tested for persons in employment (Ostro 1990), and for several pollutants simultaneously (Ostro and Rothschild 1989). Similar results were found. A recent study on sick-leaves in Oslo by Hansen and Selte (1997) also confirms the results by Ostro (1987). This is particularly interesting for the present study, as we calculate annual health effects in Oslo. Moreover, it should be added that the function is also supported by the numerous studies that find a relation between particulate concentration and various types of morbidity. Since it is based on the largest data material, the function referred to in equation (5.5) has been chosen in applied studies (see for instance EC (1995) and ORNL/RFF (1994)).

According to ORNL/RFF (1994), 62 per cent of all RAD are bed-disability days and work-loss days, while the remainder are described as minor restricted activity days (MRAD). This breakdown features in the health study on which Ostro's study is

based, and is used in the application of his results. ORNL/RFF have valued the cost of one MRAD at just over 1/4 of wages. This is based on a study of willingness to pay, implying that the valuation probably also covers some welfare loss. We therefore assume that one MRAD results in an estimated 10 per cent reduction in labour productivity. Other RAD of course reduce labour productivity by 100 per cent. Hence an average day of restricted activity corresponds to a 66 per cent reduction in productivity. We further assume that RAD are evenly spread across workdays and leisure days. In the latter case this has no effect on the supply of labour. If each person has 0.058 more RAD per year with a one-unit increase in PM_{10} concentration, the average daily increase in RAD is 0.058÷365=0.00016. Since an average RAD reduces productivity by 66 per cent, the average daily reduction in productivity will equal 0.00016*0.66=0.00011. This will apply regardless of whether a person is employed or not, so an average person-hour in the economy is correspondingly reduced by a factor of 0.00011 by a one-unit increase in concentration. This gives the following function for change in the supply of labour resulting from changed particulate concentration:

$$(5.6) \quad \Delta L / L = - 1.1*10^{-4} * \Delta PM_{10} * P/TP$$

L denotes the supply of labour in terms of person-hours worked throughout the economy, and *P/TP* is the share of the total population exposed to this change in concentration. The function states that if PM_{10} concentration increases by 10 $\mu g/m^3$, labour productivity diminishes by an average of 1‰ per person-hour.

A much utilised study of the occurrence of respiratory symptoms is Krupnick et al. (1990). The study focuses on symptoms in the upper and lower respiratory tract. According to Ostro (1994) and Rowe et al. (1995) the results lead to the following function:

$$(5.7) \quad \Delta RS = 0.18 * \Delta PM_{10} * P$$

where *RS* denotes the number of days with respiratory symptoms per year, and *P* signifies the number of persons exposed. PM_{10} signifies the mean annual concentration measured in $\mu g/m^3$.

The function states that an increase of 0.18 days in respiratory symptoms per person per year will accompany a one-unit increase in annual concentration of PM_{10}. It is however conceivable that a day with respiratory symptoms may partially overlap a day of restricted activity (RAD - see equation (5.5)). Hence we deduct the number of RAD from the function for the number of days with respiratory symptoms resulting from changed PM_{10} concentration. Eskeland (1995) also makes use of the dose-response functions for RAD and respiratory symptoms, but without deducting any overlapping. He further assumes that a day with respiratory symptoms entails a 6 per cent reduction in labour productivity. Since he does not believe in overlapping between respiratory symptoms and RAD, 6 per cent cannot accommodate more extensive symptoms that would lead to greater restrictions on activity. The basis for this supposition is unclear. Following the same procedure as for RAD, we obtain the following function:

$$(5.8) \quad \Delta L / L = - 2.1*10^{-5} * \Delta PM_{10} * P/TP$$

This function states that an increase of 10 $\mu g/m^3$ in PM_{10} concentration results in a reduction of labour productivity averaging 0.2‰ per person-hour in the economy.

Although Krupnick et al. (1990) is the only study to have focused on respiratory symptoms in general, many studies have for example focused on asthma attacks, the common cold etc. A Swiss study by Braun-Fahrländer et al. (1992) found inter alia significant links between PM_{10} concentration and the duration of various respiratory symptoms in children. We have not included these functions because they probably overlap the function from Krupnick et al. They do contribute however to support the function we utilise.

Hospital admissions
Many studies have focused on the relationship between particulate concentration and the number of respiratory hospital admissions. Some of the studies address specific respiratory diseases while others take in the entire group. To our knowledge the only two studies that focus on respiratory admissions in general are Plagiannakos and Parker (1988) which is applied by ORNL/RFF (1994), and Pope (1991) which is used by Rowe et al. (1995) and Ostro (1994). The two studies differ greatly: Plagiannakos and Parker suggest a relationship that is 8-9 times larger than that found by Pope. The reason for this is not clear. The study by Pope (1991) is particularly interesting for Norwegian conditions since it was carried out in Utah Valley in the winter half-year with low concentrations of SO_2 and ozone (cf. the discussion under mortality). On the other hand the the number of respiratory hospital admissions per capita is about three times greater in Norway (cf. Statistics Norway 1995d) than in the area studied by Pope. If this is because the threshold for admission is lower in Norway's welfare state, it suggests that Pope's results will underestimate the effect in Norway.

EC (1995) employ the number of hospital admissions related to respiratory infections and chronic obstructive pulmonary disease (COPD) respectively as their target variable. If we assume that the increase in respiratory hospital admissions breaks down to equal percentages on various respiratory diseases, we find by comparing with Norwegian statistics on patients (Statistics Norway 1995d) that these results fit in well with Pope's result (1991). A Spanish study by Sunyer et al. (1991) focused on hospital admissions related to COPD. By rough and ready conversion of the results from this study, we find a relationship that is about three times larger than Pope's result (1991). This result is confirmed by other studies.

For our purpose we opt for the result from Pope (1991), multiplied by a factor of 3. This appears to be an acceptable estimate based on the various results and assessments commented on above. We obtain the following relation between PM_{10} concentration and respiratory hospital admissions:

(5.9) $$\Delta RHA = 3.6*10^{-5} * \Delta PM_{10} * P$$

where RHA denotes the number of respiratory hospital admissions per year, and P is the number of persons exposed. PM_{10} signifies the mean annual concentration measured in $\mu g/m^3$. The function states that a one-unit increase in the mean annual concentration of PM_{10} results in an increase in hospital admissions of 3.6 per year in a population of 100 000. A natural choice of uncertainty interval here would be the results of Pope (1991), and Plagiannakos and Parker (1988), respectively.

We can now utilise the dose-response function to calculate the relationship

between change in PM_{10} concentration and change in public health expenditure. An implicit assumption is that the change in number of respiratory admissions resulting from a changed pollution situation changes the level of public health expenditure. The alternative would be to assume that it leads to changes in hospital queues. In the short term the latter alternative is probably most realistic, while in the long term there is reason to believe that the level of public health expenditure will be affected by the demand for hospital services.

Since the studies referred to above do not focus on the duration of hospital admissions, it is natural to assume that these do not differ from respiratory admissions in general. In 1993 bed-days per admission averaged 5.96 (8.78) for persons admitted with diseases of the respiratory organs in Norway (figures for Oslo in parentheses) (Statistics Norway 1995d). The figure is lower for respiratory infections, and higher for pulmonary diseases. We opt to keep to the average figure. Net operating expenditure per bed-day at Norwegian hospitals in 1994 was Nkr 3 187 (Statistics Norway 1993b).[46] In addition there is investment expenditure which we put at 5 per cent of operating expenditure. We obtain the following functions (figures for Oslo in parentheses):

(5.10) $\Delta BD = 2.1*10^{-4}\ (3.2*10^{-4}) * \Delta PM_{10} * P$

(5.11) $\Delta PHE = 0.76\ (1.12) * \Delta PM_{10} * P$

where BD is the number of bed-days and PHE is public health expenditure measured in 1994 Nkr.

[46] Wage costs account for 71 per cent of this.

5.2.2. Chronic effects

It is difficult to measure chronic health effects of pollution. However, in recent years studies have been carried out in this field indicating that costs associated with chronic effects are especially heavy. Even though the uncertainty is larger for these effects, it is important to throw light on their impacts, where they are available.

Mortality risk

A cohort study by Dockery et al. (1993) examined the effect on mortality of long-term exposure to various pollutants based on historical data for six towns. They monitored 8 000 adults, and controlled for individual risk factors such as smoking. They found a significantly higher risk of mortality at higher concentrations of particulates (PM_{10} and $PM_{2.5}$). For PM_{10} they found that an increase of 1 $\mu g/m^3$ in mean concentration over several years was associated with an increase in the mortality rate of about $8.5*10^{-3}$, i.e. 0.85 per cent (95 per cent coefficient interval: $2.7*10^{-3}$ - $1.4*10^{-2}$). A similar cohort study by Pope at al. (1995) found results of almost the same order of magnitude. The WHO (1995) refers to these two studies and writes that a combination of their results indicates a relative risk of 1.10 at an increase of 10 $\mu g/m^3$ in concentration of $PM_{2.5}$. Converting to PM_{10} gives the following dose-response function (see footnote 44):

(5.12) $\Delta D / D = 6.5*10^{-3} * \Delta PM_{10}$

Whether it is in fact the average concentration over several years or an accumulation of episodic effects over a long period that is the deciding factor so far remains unclear.

It is important to use these coefficients correctly. They cannot be used directly to

estimate the effect on the overall mortality rate in the population in the long term. The coefficients must be interprested in the light of the mortality rate in various age groups, i.e. the risk of dying within a certain period (e.g. a year) upon reaching a certain age. The function in equation (5.12) states that this risk increases by an average of 0.65 per cent for every unit increase in PM_{10} concentration. How this is reflected in the overall mortality rate depends inter alia on the distribution of deaths on age groups.[47] These result can at all events safely be stated to indicate a considerably larger effect of chronic exposure than that found with short-term variations in particulate concentration (see section 5.2.1). In the first place the coefficient is about 7 times larger. Second, there is no question here of small reductions in lifetime inasmuch as the mortality rate rises.

Dockery et al. (1993) point out that the results may be influenced by concentrations present in earlier periods (i.e. prior to 1974) which could not be taken into account. Since pollution levels at that time were higher (hence also the difference between concentrations in different towns), the effect may to some extent have been overstated. Against this background EC (1995) choose not to make direct use of this estimate in their calculations, but present it to indicate that mortality resulting from chronic exposure may be a substantial factor. The WHO (1995) also writes that further cohort studies of this type are needed before confident conclusions can be drawn about particulates' long-term effects on mortality.

Despite the uncertainty related to this effect on mortality risk, we want to illustrate how this effect may influence the work force of the economy. In the study by Dockery et al. (1993) persons were divided into 5-year groups (from 25 to 74 years), and distinctions were drawn for instance between those who were exposed to dust or gases at work and those who were not. The article does not make clear whether or not the risk factor varied among the age-groups. However, the results do show that increased risk was primarily associated with death from lung cancer (8.4 per cent) and heart and pulmonary diseases (53.1 per cent). For the remainder of the deaths as a whole risk was unaffected by particulate concentration. We therefore choose risk factors for these causes of death corresponding to coefficients of $1.1*10^{-2}$ per unit of PM_{10}, and assume that the relative risk increase is identical in all age-groups. In this way we avoid problems associated with causes of death that are typical for younger age-groups, such as road accidents. Using statistics on causes of death in 1993 (Statistics Norway 1995e), change in mortality at different ages can be calculated, both for Norway and Oslo. Moreover, the effect this has on the workforce can be calculated by assuming that affected persons in the age-group 25-64 years are average workers with a retirement age of 65 years. This calculation does not take account of the loss of labour in the period these persons are sick due to lung cancer or heart and pulmonary disease. Hence the total effect on employment will probably be substantially understated.

[47] In an extreme population where everyone died on reaching the age of 80, this type of increased risk would have virtually zero effect. Everyone would still reach 80 years of age.

In 1993, 464 (53) persons in the age group 25-64 years died as a result of "a malignant tumour in the respiratory organs and the organs in the thoracic cavity" in Norway (Oslo figure in parentheses) (Statistics Norway 1995e). All in all this category comprised 3.6 per cent of all deaths in 1993 (against 8.4 per cent in the study by Dockery et al.). If we assume that the average age in each 5-year group is halfway through the 5 years, we obtain an average age of 57.2 years for these 464 (53) persons, i.e. they had 7.8 years left to retirement. Similarly we find that 2 106 (223) persons in this age group died from "diseases of the circulatory organs or respiratory organs". This group accounted for a total of 56.4 per cent of all deaths in 1993 (53.1 per cent in Dockery et al.). The average age of these 2 106 (233) deceased persons was 56.3 years, i.e. 8.7 years prior to assumed retirement age. Combining these two groups gives 2 570 (276) deceased with an average of 8.5 years to assumed retirement age. By dividing the number of person-years in the economy in 1993 (2.87 billion - Statistics Norway (1994c)) by the population in the age range 20-70 years at year-end (2.70 million - Statistics Norway (1995f)), we obtain an average of 1 060 hours worked per person between 20 and 70 years in 1993. This is probably a good approximation for the age group upon which we are focusing (mainly 55-65 years).

We can now calculate by way of illustration the effect on the workforce of changes in long-term concentration of PM_{10}. The above statistics indicate an annual loss of 23.2 (2.5) million person-hours owing to deaths in these categories in Norway (Oslo). The above dose-response function suggested a 1.1 per cent increase in risk per unit increase in concentration of PM_{10}. This gives the following function for Norway (figures for Oslo still in parenthesis), which must be regarded as highly illustrative:

(5.13) $\Delta L = -2.6*10^5\ (2.8*10^4) * \Delta PM_{10} * P/TP$

where L is the number of person-hours worked in the entire economy per year and P/TP denotes the share of the population exposed to changed PM_{10} concentration (e.g., in Oslo P=TP when the figure in parenthesis is used). PM_{10} is a long-term mean. The next section shows how this function can be converted to a function of the mean annual concentration of PM_{10}.

The impact on the workforce of such an effect is naturally less serious than the direct health effect, namely a higher risk of death. It is difficult to quantify this in terms of increased mortality. However, using mortality tables (Statistics Norway 1995f) we can calculate the effect of such a risk increase on life expectancy in the population. If we apply the estimates presented in WHO (1995), i.e. a risk increase of 0.65 per cent per unit increase in PM_{10} concentration, to the age group 25 years and over, we find the following dose-response function which again must be regarded as illustrative (the relation we computed is approximately linear):

(5.14) $\Delta LE_W = -0.059 * \Delta PM_{10}$

(5.15) $\Delta LE_M = -0.064 * \Delta PM_{10}$

where LE is lifetime expectancy for women and men measured in number of years. Here too PM_{10} is a long-term mean. Thus, life expectancy for women falls by 0.6 years at an increase of 10 $\mu g/m^3$ in PM_{10} concentration, whereas the equivalent reduction for men is 0.65 years. This

fits in well with calculations done in the Netherlands, according to WHO (1995). The WHO refer to a study which, applying the same risk increase to Dutch men, found a reduction of 1.1 years in lifetime expectancy with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration. This corresponds to about 15 $\mu g/m^3$ of PM_{10} (see footnote 44). It should be stressed that this effect is highly uncertain, and should only be regarded as indicative.

Chronic pulmonary diseases
Four studies from different areas - Abbey et al. (1993), Schwartz (1993a), Xu and Wang (1993) and Portney and Mullahy (1990) - have analysed the relationship between particulate concentration and the occurrence of various types of chronic obstructive pulmonary disease (COPD - incl. inter alia chronic bronchitis, asthma and emphysema). What is surprising is that all four find that the risk increases by about 0.6 per cent with an increase of 1 $\mu g/m^3$ in long-term concentration of TSP. The relationships are significant. Converting to PM_{10} concentration (see footnote 44), we obtain the following association between the long-term concentration of PM_{10} and the relative change in occurrences of COPD:

(5.16) $$\Delta COPD / COPD = 1.1*10^{-2} * \Delta PM_{10}$$

That is, the frequency of COPD in the population increases by 1.1 per cent per unit increase in the long-term concentration of PM_{10}. Although there is some uncertainty attached to the conversion between TSP and PM_{10}, this is reduced by the availability of four different studies. PM_{10} is assumed to be a better health indicator than TSP.

The study by Abbey et al. (1993) is especially interesting since it is a time series study over 10 years in which a sample of 4 000 persons were monitored and examined for chronic pulmonary diseases. The authors state that there was a significant association between particulate concentration over the 10-year period and the risk of developing chronic pulmonary diseases (COPD) in general. The same applied to chronic bronchitis in particular, but not to asthma (both belong in the COPD group). In a follow-up to this study Abbey et al. (1995) find similar results and, in addition, a significant relationship between particulate concentration and the risk of developing asthma.

In Rowe et al. (1995) and Ostro (1994), the result from Abbey et al. (1995) is converted to a linear relationship applying on an annual basis. This is done in light of the fact that the study was carried out over a ten-year period. The effect of increased concentration in any year is assumed to be one tenth of the effect observed over the entire ten-year period. They arrive at the following relationship:

(5.17) $$\Delta COPD = 6.1*10^{-5} * \Delta PM_{10} * P$$

Here ΔCOPD denotes change in the number of persons in the population with a chronic pulmonary disease, while P is the size of the exposed population. As stated, PM_{10} is the annual mean in this function. It is natural to envisage some lag in this relationship inasmuch as the results were observed over a ten-year period. Hence a more correct function would be one where PM_{10} denotes mean concentration over a ten-year period at the same time as the function still indicates annual effects. It follows from the above calculation that the incidence of COPD in the population is at the outset stipulated at

5-6 per cent. This probably fits in well with Norwegian conditions.

It is difficult to use equation (5.17) to calculate effects on economic variables such as sickness absence and hospital admissions. It is simpler to apply equation (5.16), which moreover showed a high degree of consistency across the various studies. We therefore construct linear relations for various economic variables based on available statistics for Norwegian conditions. For hospital admissions we assume that the relative increase in the number of cases of COPD is reflected in a proportional increase in the number of bed-days. In Norway in 1993 there were 28.3 bed-days with the diagnosis COPD per 1 000 head of population (Statistics Norway 1995d). Assuming that a 1.1 per cent increase in risk accompanies a permanent increase of 1 $\mu g/m^3$ in the concentration of PM_{10}, we obtain (the factor for Oslo - where the average number of bed-days was 35 per cent higher - is in parentheses):

$$(5.18)\quad \Delta BD = 3.1*10^{-4}\ (4.2*10^{-4}) * \Delta PM_{10} * P$$

Here *BD* denotes the number of bed-days at Norwegian hospitals per year, while PM_{10} now denotes a long-term mean.

The number of bed-days in a given year thus depends on PM_{10} concentrations in previous years. In order to convert from a long-term mean (chronic exposure) to an annual mean, we assume that PM_{10} concentration in a given year has an effect on the number of bed-days in the 10 ensuing years and that this effect is identical for all 10 years. This is of course a simplification, but should be viewed in light of the lack of knowledge about how a chronic disease arises. Our choice reflects the study by Abbey at al. (1993) which covered a 10-year period. The change in the number of bed-days resulting from new cases of COPD now becomes a function of changed PM_{10} concentration in the preceding 10 years. By utilising the same cost data as in section 5.2.1, we obtain the following function for change in public health expenditure (the factor for Oslo is in parentheses):

$$(5.19)\quad \Delta PHE = 1.1(1.5)*\Delta[\sum_{i=0}^{9} (PM_{10})_{-i}/10] * P$$

As previously, PHE denotes public health expenditure measured in 1994 Nkr, while $(PM_{10})_{-i}$ signifies the mean annual concentration of PM_{10} *i* years ago. Hence the expression in square brackets is a 10-year mean.

We also assume that the relative increase in incidence of COPD results in a similar increase in sickness absence, rehabilitation and disability pensioners with this diagnosis. Here we have drawn on figures from the National Insurance Administration for disbursements and number of persons in these categories for the diagnosis COPD. The figures refer to 1993/1994. The effect of increased sickness absence and rehabilitation on the number of hours worked in the economy is calculated by dividing the total disbursement by the average hourly wage.[48] There is a risk of some understatement here since the data from the National Insurance Administration may be incom-

[48] Disbursements for sickness absence with the diagnosis COPD was Nkr 8.6 million in 1994, and for rehabilitation Nkr 47.6 million, according to data from the National Insurance Administration. However, uncertainty attaches to the data. The average hourly wage in 1994 was Nkr 135.1 per hour.

plete. Where sickness absence is concerned the statistics only cover absence beyond the first 14 days. For disability pensioners the disbursement is at the outset far lower than the average annual wage, and is also determined by factors such as responsibility for dependants etc. We therefore choose to multiply the number of disability pensioners by the average number of hours worked per person in the age range 20-70 years. This figure was 1 061 in 1993.[49] We now have the following relation between long-term concentration of PM_{10} and the workforce:

(5.20) $\Delta L = -6.9*10^{2} * \Delta[\sum_{i=0}^{9} (PM_{10})_{-i}/10] * P/TP$

(sickness absence)

(5.21) $\Delta L = -3.8*10^{3} * \Delta[\sum_{i=0}^{9} (PM_{10})_{-i}/10] * P/TP$

(rehabilitation)

(5.22) $\Delta L = -6.9*10^{4} * \Delta[\sum_{i=0}^{9} (PM_{10})_{-i}/10] * P/TP$

(disability)

L denotes the number of person-hours worked in the economy, while P/TP signifies the share of the Norwegian population that is exposed. $(PM_{10})_{-i}$ denotes the mean annual concentration of PM_{10} *i* years ago, so the expression in square brackets is a 10-year mean.

There is little risk of overlap between the functions in this section and the functions in sections 5.2.1. The effects on the labour force in this section do not include short-term absences (i.e., less than two weeks), whereas the functions in section 5.2.1 were based on short-term studies and therefore do not capture longer absences (i.e., more than two weeks). Moreover, the impacts on public health expenditures in this section regards the effects of more people being chronicly ill, whereas the interpretation of the effects in section 5.2.1 is presumably that chronicly ill persons are being sent to hospital more often than without pollution.

5.2.3. Threshold values for particulates

The question of threshold levels is an important one when discussing the relevance of transferring dose-response functions to Norway. Concentrations in Norwegian towns are somewhat low compared with American cities where most of the studies have been undertaken. Internationally however the trend is moving more and more away from specific threshold levels in a population. The reason is that threshold levels are at base specific to the individual, and so far it has not been possible to establish any minimum value that is applicable to all individuals. Moreover, significant relationships have been found between particulate concentration and mortality at concentrations well below previously established threshold levels. This has inter alia prompted an expert group attached to the European Office of the World Health Organisation (WHO 1995) to state in a new report on guidelines for air quality that it will no longer recommend guidelines for particulates. The WHO also write

[49] The total number of person-hours worked in the economy in 1993 was 2.87 billion (Statistics Norway 1994c), while the number of persons between 20 and 70 years totalled 2.70 million at year-end (Statistics Norway 1995f). The number of disability pensioners with the diagnosis COPD was 5 984.

that *long-term effects* appear to arise at very low concentrations, specifically 10 μg/m^3 of $PM_{2.5}$ (corresponding to about 15 μg/m^3 of PM_{10}, which is well below the level in Oslo - see section 5.6).

Another interesting point is that the slope coefficient in the dose-response functions does not seem to diminish with diminishing concentration. A simple plot of average concentration and coefficient estimates from the 9 mortality studies referred in Ostro (1993) (see section 5.2.1) actually indicates a negative slope. Average concentrations in these studies were in the area 37-80 μg/m^3. Moreover, a fairly recent study by Dockery et al. (1992) found coefficient estimates 1.5 times the consensus estimate in Ostro (1993). This was in areas featuring an average PM_{10} level of 28 and 30 μg/m^3 respectively. This picture is in contrast with the previous thought that the incidence of health damage is a convex function of concentration at low pollution levels (gradual threshold), and thereafter approximately linear. Moreover, EC (1995) also points to indications that the marginal effect is greatest at low concentrations, whereas WHO (1995) asserts that in the case of particulates the effect is approximately linear up to 200 μg/m^3, and only then does the marginal effect recede.

Dockery et al's (1993) study of mortality resulting from long-term exposure found mean annual concentrations of PM_{10} between 18 and 47 μg/m^3. Hence towns with the lowest particulate pollution showed lower PM_{10} concentrations than for example Oslo. This means that if the results of Dockery et al. can be generalised successfully, the dose-response function is clearly relevant for Norwegian towns.

In the study of restricted activity days (Ostro 1987) in section 5.2.1 the mean concentration of PM_{10} was about 43 μg/m^3. and this average was based on 84 urban areas with a wide range of concentration levels (probably also covering the level in Norwegian towns).

The mean concentration in Abbey et al's study of chronic pulmonary diseases (1993) is not specified. However, 13 per cent were stated to live in areas featuring concentrations below about 33 μg/m^3 of PM_{10}. This and other figures may suggest that the mean concentration was relatively high (probably about 50 μg/m^3). Collated with the WHO assessment above, namely that long-term effects also seem to arise at very low concentrations, we nonetheless find it relevant to utilise the results from Abbey at al. (and other studies) for Norwegian towns.

5.3. Health effects of nitrogen dioxide (NO_2)

Nitrogen dioxide (NO_2) is a gas formed by combustion of fossil fuels. The principal source in Norwegian towns is vehicle traffic. In some locations space heating, manufacturing or shipping may also be important emission sources.

Compared to particulates, relatively few epidemiological studies have found significant relationships between outdoor NO_2 concentrations and health effects. However, as mentioned in the introduction, there are several reasons why the importance of NO_2 should not be toned down. First, numerous clinical studies have demonstrated health effects. However, since such experiments differ in many ways from normal outdoor conditions it is difficult to draw quantitative conclusions. Similarly, many studies have demonstrated relationships between indoor NO_2

concentrations related to use of gas stoves, and various respiratory diseases. Here too, however, it is not clear to what extent these relations can be converted for use with outdoor concentrations. None of the applied studies we referred to earlier utilise dose-response functions from indoor NO_2 concentrations. Only two of the studies, ORNL/RFF (1994) and Ostro (1994), apply a function for outdoor concentrations, and this is related to respiratory symptoms. Certain studies, however, do find effects of NO_2, but not of particulates. In this study we describe and apply a study from Finland related to asthma attacks.

Ostro (1994) mentions three possible methodological reasons why little has been found in the way of effects of NO_2 concentrations outdoors. These are 1) problems of measuring NO_2 concentrations outdoors, 2) effects of NO_2 only arise at high concentrations, and 3) chronic effects of NO_2 are important, not acute. If point 2 is correct then it is primarily brief exposures to particularly high concentrations that have a bearing on acute health effects, for instance along a road carrying heavy traffic. In such a case individual exposure is particularly important, and this may be difficult to measure. Moreover, the resultant health effects will show a smaller degree of correlation with daily mean concentrations which on the whole are used in epidemiological studies.

Appendix B includes a table summarising the dose-response functions taken from the literature, and a table showing how these functions have been used in regard to economic activity etc.

5.3.1. Acute effects

Hospital admissions
A Finnish study by Pönkä (1991) found a positive association between hospital admissions due to asthma attacks and exposure to NO_2. This study is particularly interesting for Norwegian conditions because it was carried out in a neighbouring Nordic country in an area with a cold climate. An increase in NO_2 concentration from 28 to 45 $\mu g/m^3$ was accompanied by a 29 per cent increase in hospital admissions. We assume that the percentage increase in admissions is identical at all concentration levels (i.e. a 1.5 per cent increase per unit increase in NO_2 concentration), and that the relationship between admissions and number of bed-days is constant. We can then derive a linear function based on Norwegian patient statistics (Statistics Norway 1995d) which approximates to the underlying function. We find that for every 1 000 head of population in Norway (Oslo) in 1993 there were 8.7 (9) bed-days with the diagnosis bronchial asthma. This gives the following function for change in number of bed-days (the factor for Oslo is in parentheses):

$$(5.23) \quad \Delta BD = 1.3*10^{-4}\ (1.4*10^{-4}) * \Delta NO_2 * P$$

where BD denotes number of bed-days per year and P signifies the number of persons exposed. NO_2 denotes mean annual concentration measured in $\mu g/m^3$.

If we apply the cost per bed-day at Norwegian hospitals (see section 5.2.1) we obtain the following function for change in public health expenditure (the factor for Oslo in parentheses):

$$(5.24) \quad \Delta PHE = 0.47\ (0.48) * \Delta NO_2 * P$$

where PHE denotes health expenditure in 1994 Nkr.

Compared with equivalent results for particulates in section 5.2.1, which applied to all types of respiratory hospital admissions, this figure seems fairly high (NO_2 concentration in Oslo is about twice as high as PM_{10} concentration). A reason for this may be that the combination of NO_2 and low temperature has a large impact on health effects such as asthma attacks. Rossi et al. (1993), another Finnish study, found a significant relation between NO_2 concentration and asthma attacks, even though the mean value was as low as 13 $\mu g/m^3$.

A German study by Schwartz et al. (1991) found a significant relation between NO_2 concentration and the number of cases of croup. An increase from 10 to 70 $\mu g/m^3$ was associated with an increase of 27 per cent in incidence of this ailment, according to the National Institute of Public Health (1995). Applying the same assumptions as above, this corresponds to 0.4 per cent greater risk of croup per unit increase in NO_2 concentration. According to the background material from Statistics Norway (1995d) there were 5 323 bed-days with the diagnosis "acute laryngitis and tracheitis", including croup. This implies 1.23 bed-days per 1 000 head of population. In the same way and with the same assumptions as above, we can now calculate the effect of NO_2 pollution on the number of bed-days and public health expenditure:

(5.25) $\Delta BD = 4.9*10^{-6} * \Delta NO_2 * P$

(5.26) $\Delta PHE = 0.019 * \Delta NO_2 * P$

where *BD* is the number of bed-days per year, *P* is the number of persons exposed and PHE is public health expenditure in 1994 Nkr. NO_2 denotes mean annual concentration. We see that hospital costs attributable to increased incidence of croup are substantially lower than corresponding costs resulting from increased asthma attacks.

A number of studies have demonstrated an association between indoor NO_2 concentrations and respiratory diseases. Hasselblad et al. (1992) carried out a metaanalysis of existing studies . They found that an increase of 30 $\mu g/m^3$ was accompanied by a 20 per cent increase in diseases of the lower respiratory tract. Whether or not it is appropriate to convert these results to functions for outdoor concentrations is not clear. Until more is known about the relative importance of outdoor contra indoor concentrations, it is difficult to make use of these results.

Respiratory symptoms
ORNL/RFF (1994) and Ostro (1994) have used results from Schwartz and Zeger (1990) for the relation between NO_2 concentration and frequency of phlegm production. In Ostro the result is converted to the following function:

(5.27) $\Delta RS = 5.1*10^{-3} * \Delta NO_2 * P$

RS denotes the number of days with respiratory symptoms per year, while *P* is the number of persons exposed. In this function NO_2 signifies the average hourly maximum (daily), measured in $\mu g/m^3$. According to the Norwegian Institute for Air Research the relationship between the hourly maximum and daily mean varies from 1.2 to 2.8 in Oslo, with 1.8 as the 'best' estimate. Some uncertainty attaches to whether this relationship varies systematically with pollution levels. If not, there is reason to believe that the mean

daily concentration can be used as an indicator for the one-hour maximum. If we assume this to be the case, and apply the conversion factor 1.8, we obtain the following function:

(5.28) $\Delta RS = 9.2*10^{-3} * \Delta NO_2 * P$

where NO_2 now denotes the mean annual concentration (the transition from daily mean to annual mean is assumed to apply as previously).

A shortcoming of the study by Schwartz and Zeger (1990) is that particulates were not controlled for. Hence there is a risk that this result may overlap the result in section 5.2.1. However, other studies support the relation between outdoor NO_2 concentration and respiratory diseases. A Swiss study by Braun-Fahrländer et al. (1992) found for instance that the duration of respiratory symptoms in children rose with increasing exposure to NO_2, even when particulates were taken into account. As mentioned in the preceding section, a Finnish study by Rossi et al. (1993) found significant relations between NO_2 concentration and asthma attacks. Since it is currently difficult to convert these results to a dose-response function we let them support the above function instead.

What this entails for labour productivity is somewhat unclear. Ostro (1994) is not concerned with valuation in his analysis, while ORNL/RFF (1994) omit to value phlegm production for lack of relevant studies. We choose to follow the same procedure as in section 5.2.1, i.e. to estimate the productivity loss resulting from one day of respiratory symptoms at 6 per cent (based on Eskeland (1995)). The relative change in the supply of effective labour resulting from increased NO_2 concentration is then calculated to:

(5.29) $\Delta L / L = - 1.5*10^{-6} * \Delta NO_2 * P/TP$

L is the number of person-hours worked per year in the entire economy, and P/TP signifies the share of the Norwegian population that is exposed. Although the mean annual concentration of NO_2 is normally higher than for PM_{10}, the effect of NO_2 here is clearly lower than found for particulates in section 5.2.1.

5.3.2. Threshold levels for NO_2

The question of threshold levels is more problematic for NO_2 than for particulates. The comparative lack of studies to turn to makes it difficult to draw conclusions. WHO (1995) consequently continue to recommend air quality guidelines for NO_2. Their guideline annual mean is 40 to 50 $\mu g/m^3$. By way of comparison the Norwegian Pollution Control Authority (SFT 1992) employs a six-month mean of 50 $\mu g/m^3$. This is slightly above the mean annual concentration in larger Norwegian towns. The guidelines are not intended to be absolute threshold levels. A certain safety margin is included at the same time as no guarantee is given that particularly sensitive individuals will not experience health effects at concentrations below the threshold levels. It must also be kept in mind that mean annual concentrations vary within the individual town, and that the short-term guidelines may be substantially exceeded.

A pertinent question is whether the air quality criteria for NO_2 will in time be tightened in the same way as for particulates. The criteria for particulates have been adjusted downwards in the light of increased knowledge, and if methodological problems explain why there are

currently fewer studies to turn to for NO_2, it is not improbable that in time the criteria for NO_2 will also be tightened.

The results from two Finnish studies, Pönkä (1991) and Rossi et al. (1993) which were discussed above, are worth noting. In the first-mentioned study a significant relation was found between NO_2 concentration and hospital admissions due to asthma attacks at concentrations in the interval 28-45 $\mu g/m^3$. This is well below the guidelines from the WHO and the Norwegian Pollution Control Authority. Rossi et al. (1993) found a significant relation between NO_2 concentration and asthma attacks at a mean concentration as low as 13 $\mu g/m^3$. This is less than a third of the mean annual concentration in Oslo, and corresponds to the background level in south-east Norway. An interesting feature shared by these two studies is that they were undertaken in a cold climate (temperature was of course taken into account in the regressions). They ought therefore to be particularly relevant for Norwegian conditions, and indicate that the dose-response functions may apply well below the guidelines recommended by the WHO and the Norwegian Pollution Control Authority.

5.4. Health effects of sulphur dioxide (SO_2)

Sulphur dioxide (SO_2) is formed by combustion of oil and coal, and the emissions are due to the sulphur content of these fuels. SO_2 has historically been viewed as an important pollutant in the context of health effects. However, the significance of SO_2 has been toned down in the past decade. There are two important reasons for this. First, the importance of SO_2 is believed to have been somewhat overstated in relation to other pollutants, in the first instance particulates. Second, with the substantial reduction in SO_2 concentrations in most areas the health problem is now mainly confined to specific local areas with heavy sulphur emissions from manufacturing industry (SFT 1992). However, SO_2 may have a substantial health effect in its own right in such locations. SO_2 emissions also lead to the formation of sulphates belonging in the group of small particulates ($PM_{2.5}$).

Although the effect of SO_2 disappears in many studies when particulates are correctly measured (EC 1995), some studies continue to find health effects of SO_2 when particulates are included (ORNL/RFF 1994). Below we present some dose-response functions described in the literature. It is however unclear to what extent these effects can be included in addition to the effects of particulates.

5.4.1. Acute effects

Mortality

ORNL/RFF (1994) and Ostro (1994) describe several European studies that have found significant links between concentration of SO_2 and mortality. Common to all of them is that they were undertaken in areas with relatively high concentrations, all exceeding an average of 60 $\mu g/m^3$. This is considerably higher than the mean annual concentration in Oslo. Ostro constructs a dose-response function based on a Greek study by Hatzakis et al. (1986):

(5.30) $\Delta D / D = 4.8*10^{-4} * \Delta SO_2$

where D denotes number of deaths per year, while SO_2 denotes an annual mean measured in $\mu g/m^3$.

Several of the studies described by Ostro find no relation between particulates and mortality. This confirms the supposition in the introduction that particulates (or other pollutants) are primarily indicators of a complex pollution situation. Hence it would be advisable not to add together the mortality effects of particulates and SO_2.

Respiratory symptoms
ORNL/RFF (1994) and Ostro (1994) also give an account of studies of various types of respiratory symptoms. One of them (Schwartz et al. 1988) finds significant relations between concentration of SO_2 and frequency of chest pains. Ostro derives the following dose-response function:

(5.31) $\Delta RS = 1.0*10^{-2} * \Delta SO_2 * P$

where *RS* denotes the number of days with respiratory symptoms per year, while *P* is the number of persons exposed. SO_2 denotes mean annual concentration.

In the same way as in sections 5.2.1 we can derive the effect on the workforce:

(5.32) $\Delta L / L = - 1.64*10^{-6} * \Delta SO_2 * P/TP$

where *L* denotes number of person-hours worked and *P/TP* the exposed share of the population.

5.4.2. Threshold levels for SO_2

Studies that have found significant associations between SO_2 concentration and health effects have been carried out in areas with high concentrations. Against this background the WHO (1995) recommends a mean annual guideline of 50 $\mu g/m^3$, which is substantially higher than the level in Oslo. The Norwegian Pollution Control Authority (SFT 1992) employs a six-month mean of 40 $\mu g/m^3$. Against this background it is uncertain to what degree the dose-response functions for SO_2 are relevant in Norwegian towns (with the exception of particular areas). It is however conceivable that health damage may also be associated with SO_2 in episodes of high SO_2 concentration.

5.5. Health effects of ozone (O_3)

Ozone (O_3) is a so-called secondary air pollution component formed by reactions between NO_x, methane and VOCs (volatile organic compounds). As mentioned in section 5.1 ozone is the component receiving the greatest international attention alongside particulates. In Norway, however, ozone concentration is very largely determined by long-range pollution from elsewhere in Europe. Increased emissions of NO_x in Norwegian towns lead for the most part to somewhat lower local ozone concentrations, but to somewhat higher concentrations at regional level and in suburban areas.[50] However, the relationship between local and regional effects are not well understood yet. Furthermore, the overall indication is that economic activity in Norway is of little significance for health effects caused by ozone. Thus, discussion of ozone is limited.

According to EC (1995) the effects of ozone are regarded as additive in relation to particulates, NO_2 and SO_2. This is because correlations between ozone and the other components are generally weak.

[50] A report from The Norwegian Meteorological Institute, Simpson et al. (1996), calculates the regional effects on ozone concentrations of Norwegian emissions of NO_x and VOCs. It finds that while Norwegian NO_x emissions have a certain regional effect on ozone concentrations, Norwegian VOC emissions have little regional effect.

5.5.1. Acute effects

Mortality
EC (1995) refers to a study by Kinney et al. (1994) which found the following significant relation between ozone concentration and mortality:

(5.33) $\Delta D / D = 7.5*10^{-5} * \Delta O_3$

where *D* denotes the number of deaths per year, and O_3 is the average daily one-hour maximum measured in $\mu g/m^3$. Rowe et al. (1995) refer to two studies which find approximately the same risk increase. Rowe et al. choose however to halve the coefficient since several studies have found a weaker or no relationship between ozone concentrations and mortality. Moreover, a pertinent question is whether these studies, which were all carried out in the distinctive Los Angeles area, are relevant for Norwegian conditions.

Reduced labour productivity
Two studies by, respectively, Ostro and Rothschild (1989) and Portney and Mullahy (1986) have found relations between concentration of ozone and minor restricted activity days (MRAD). The latter study (described in Ostro (1994)) found an effect that was twice as large as that found in the first-mentioned study (recounted in EC (1995) and Rowe et al. (1995)). We present a dose-response function with a coefficient estimate equal to the average of the two studies:

(5.34) $\Delta MRAD = 0.012 * \Delta O_3 * P$

MRAD denotes the number of minor restricted activity days per year, and *P* the number of persons exposed. O_3 signifies the annual average of daily one-hour maxima, measured in $\mu g/m^3$.

Using the procedure described in section 5.2.1, we obtain the following function for the effect on the workforce:

(5.35) $\Delta L / L = - 3.4*10^{-6} * \Delta O_3 * P/TP$

L is the number of person-hours worked in the economy per year, while *P/TP* denotes the exposed portion of the population.

Respiratory symptoms
In section 5.2.1 reference was made to a study by Krupnick et al. (1990) which found a significant relation between concentration of particulates and respiratory symptoms. The same study found a similar relation for ozone concentration. EC (1995) sets out the following function:

(5.36) $\Delta RS = 0.026 * \Delta O_3 * P$

where RS denotes the number of days with respiratory symptoms per year, and *P* denotes the number of persons exposed. O_3 signifies the annual average of daily one-hour maxima, measured in $\mu g/m^3$. Several other studies have found effects of ozone on respiratory symptoms, especially asthma.

Following the same procedure as in section 5.2.1, including deduction of MRAD as in the previous section, we find the following effect on the workforce:

(5.37) $\Delta L / L = - 2.3*10^{-6} * \Delta O_3 * P/TP$

L is the number of person-hours worked in the economy per year, while *P/TP* denotes the exposed portion of the population.

Hospital admissions
Several studies have found significant relations between ozone concentration and hospital admissions. Ostro (1994) gives an account of a study by Thurston et

al. (1992) which gives the following dose-response function:

(5.38) $\Delta RHA = 3.9*10^{-6} * \Delta O_3 * P$

where RHA denotes the number of respiratory hospital admissions per year, *P* the number of exposed persons, while O_3 remains the annual average of daily one-hour maxima. EC (1995) describes several studies that have found significant relations between ozone concentration and hospital admissions resulting from various types of respiratory diseases. These studies indicate that the function in equation (5.38) may be an underestimate.

Based on the above function we construct the following function for costs in the public health system (same procedure as in section 5.2.1 with figures for Oslo in parenthesis):

(5.39) $\Delta PHE = 0.08\ (0.12) * \Delta O_3 * P$

where *PHE* denotes public health expenditure measured in 1994 Nkr.

5.5.2. Threshold levels for ozone

The WHO (1995) recommends an eight-hour guideline of 120 μg/m^3 for ozone, but writes that this guideline gives no guarantee against *all* acute health effects of ozone. In comparison the Norwegian Pollution Control Authority (SFT 1992) employs an equivalent guideline of 80 μg/m^3. According to the SFT, the air quality criteria for ozone (one-hour mean) are exceeded about 3 per cent of the time in southern Norway and 1 per cent of the time in northern Norway. Hence there is reason to believe that the dose-response functions found for ozone are also relevant for Norway.

5.6. Population exposure to air pollution in Oslo

The Norwegian Institute for Air Research (NILU) has elaborated a detailed dispersion model, called EPISODE, for air pollution in Oslo. Given variation in emissions from different sources, it determines population exposure to inter alia particulates and NO_2 in squares of 1 km^2. NILU was commissioned by Statistics Norway to use this model to calculate a function for the population-weighted mean annual concentration of PM_{10} and NO_2 in Oslo (Walker 1997). The function depends on changes in local emissions from vehicle traffic, other local emissions (largely space heating) and background concentration due to foreign and Norwegian emissions outside Oslo. Mean annual concentration is computed for each square and is then weighted with the population in the square. This provides a detailed description of population exposure in Oslo compared with similar international studies. By way of comparison EC (1995) employ squares of 100 km^2, while Ostro (1994) uses squares of 25 km^2 for Jakarta and calls it a substantial improvement on other studies.

In view of the fact that a large portion of Oslo's population works in the city centre (where the pollution is heaviest) and lives on the outskirts, a possible objection to the method is that it partially underestimates the actual population exposure. Moreover, dispersion of air pollution is a complicated process and considerable uncertainty attaches to the functions.

Walker (1997) presents the following function for population-weighted mean annual concentration of $PM_{2.5}$ in Oslo:

(5.40) $PM_{2.5} = 3.9 * I_T + 5.3 * I_O + 5.9 * I_B$

I_j denotes indexes (I_j=1 in 1992) for respectively vehicle traffic emissions (*T*), other emissions (*O*) and background concentration (*B*) of $PM_{2.5}$. From equation (5.40) we see that in 1992 mean concentration in Oslo was calculated at 15.1 µg/m^3, and 61 per cent of this concentration was due to local emissions within the municipality. Walker makes clear that since it is not possible in such calculations to take account of the high concentrations along roads carrying heavy traffic, the above function will underestimate the effect of vehicle traffic emissions.

Walker (1997) presents scaling factors between the PM_{10} and $PM_{2.5}$ concentrations for the summer and winter half-year respectively. These are based on observations at measuring stations in Oslo. For vehicle traffic emissions there is a considerable seasonal difference where this scaling factor is concerned, i.e. 3.0 and 1.2 respectively for the winter and summer half-year, according to Walker. The wide seasonal variation is due to use of studded tires which whirl up asphalt dust consisting partly of PM_{10}. Since this contribution primarily depends on the number of kilometres driven, and not on exhaust emissions, we opt for a scaling factor between PM_{10} and $PM_{2.5}$ of 1.2 for the whole year, and include an index for kilometres driven. This is particularly important if it is wished to split the effect of exhaust emissions from the effect of wear and tear by studded tires. For other emissions the factor is 1.1 for the whole year, according to Walker. The background concentration of PM_{10} is stated to be 8.7 µg/m^3. This gives the following function for PM_{10} concentration in Oslo where the indexes I_T and I_O refer to emissions of PM_{10}, while I_{KM} is the index for distance driven (1992 remains the basis year):[51]

(5.41) $PM_{10} = 4.7 * IT + 5.8 * IO + 4.0 * IKM + 8.7$

Hence in 1992 the mean annual concentration in Oslo was 23.2 µg/m^3, of which 14.5 µg/m^3 (63 per cent) was due to local contributions in Oslo. Of the local contribution vehicle traffic accounts for 60 per cent, while other emissions account for 40 per cent. The contribution of vehicle traffic can be further divided into 54 per cent from exhaust emissions and 46 per cent resulting from the use of studded tires. The latter distribution must however be viewed with caution inasmuch as it is estimated by the author and not by Walker (1997).

In contrast to particulates, NO_2 concentration is not a linear function of local emissions. The concentration is determined by atmospheric reactions between local emissions of NO_x and the regional contribution of NO_x and O_3 (background contribution). Walker (1997) has calculated the population-weighted mean annual concentration of NO_2 for variations in vehicle traffic emissions, other emissions and background contribution.[52] This gives a calculated average mean annual NO_2 concentration of 46.5 µg/m^3 in Oslo in 1992. A 40 per cent reduction in local emissions reduces concentration by 7.7 µg/m^3, i.e. 17 per cent, while a corresponding reduction of the regional contribution reduces concentration by all

[51] Total contribution from road traffic is stated by Walker to be 8.7 µg/m^3. Of this, 1.2*3.9=4.7 µg/m^3 is assumed to stem from exhaust emissions, while the remainder (4.0 µg/m^3) is assumed to stem from asphalt dust.

[52] This is done for indexes (equal to 1 in 1992) which vary stepwise from 0.6 to 1.4 with intervals of 0.1.

of 30 per cent. What the effect of bigger reductions might be is unclear. In regard to changes within the +/- 30 per cent range a linear function is found which is approximately identical to the discreet function calculated by Walker (1997):[53]

$$(5.42)\quad NO_2 = 14.6 * I_T + 2.1 * I_O + 32.3 * I_B - 3.1$$

The indexes here refer to NO_x emissions. The large difference in the first two weights is partly related to the fact that NO_x emissions from vehicle traffic in Oslo in 1992 were 3-4 times bigger than the sum total of other emissions. For NO_2 as well, Walker makes it clear that the calculations understate the relative effect of vehicle traffic emissions which are clearly the dominating local contributor to NO_2 pollution in Oslo (87 per cent of the local contribution on a marginal basis). It is uncertain how large a portion of the background contribution is due to Norwegian emissions from outside Oslo. Hence in this calculation we confine ourselves to local emissions in Oslo.

5.7. Public health effects and social costs of air pollution in Oslo

5.7.1. Annual health effects

In this section we present calculations of health effects of air pollution in Oslo, based on the functions described earlier in the study. As mentioned in section 5.1, there are problems involved in transferring such relations from one area to another where people and physical surroundings are different. However, this has become common practice in many studies, such as EC (1995) for Europe and ORNL/RFF (1994) and EPA (1995) for the USA. By virtue of their compass and the bodies that commissioned them, these studies carry much weight internationally, and for this reason the present study is to some extent based on their choice of functions. Moreover, two studies from the World Bank, Ostro (1994) and Eskeland (1995), have used dose-response functions to calculate health effects of air pollution in respectively Jakarta (Indonesia) and Santiago (Chile). They discuss the problem of transferring functions to developing countries and consider the procedure justifiable. Compared with these two studies the calculations presented in the present chapter must be said to possess greater credibility.

It is worth noting that the 'predecessor' to ORNL/RFF (1994) and EC (1995), viz. PACE (1990), did not make use of dose-response functions. Instead it used the result from a study by ECO (1987) which found a relation between increased emissions of particulates and higher risk of mortality in urban and sparsely populated areas respectively. Such an approach is naturally somewhat less precise than using the dose-response functions described above so long as one has serviceable models to calculate the relation between emissions and concentration levels. Hence the results in the new generation of applications must be said to be considerably more reliable than was previously the case.

In the previous section it emerged that local emissions in Oslo contributed 14.5 $\mu g/m^3$ (population weighted) to the concentration of PM_{10} in 1992. We now apply this contribution to the dose-response functions set out in section 5.2 and calculate the total annual health effects of local air pollution in Oslo. This entails that we are not looking at the effect of the regional contribution to the

[53] $R^2>0.99$

Table 5.1. **Total annual health effects and impacts on economic activity of local particulate pollution in Oslo, 1992**

	Annual effects
Acute effects:	
Number of premature deaths	90
Person-years lost	
- reduced productivity	70
- short-term sickness absence	260
Bed-days in hospital (respiratory ailments)	2 100
Public hospital expenditure (mill. 1994-Nkr)	7
*Chronic effects:**	
New cases of chronic pulmonary disease	400
Person-years lost	
- long-term sickness absence/rehabilitation/disability	70
Bed-days in hospital (respiratory ailments)	2 800
Public hospital expenditure (mill. 1994-Nkr)	11
Reduced life expectancy (years) - highly uncertain	0.9

* Calculations of chronic effects presuppose that local particulate pollution has been stable for several years

level of pollution in Oslo. The calculations refer to a typical meteorological year with emission data for 1992, but since pollution levels have been relatively stable in recent years, the results are also relevant for the situation today. We would point out that uncertainty attaches to the calculations and that the results must be viewed as indications (cf. the discussions in section 5.2). Similar calculations have been made for example by the WHO (1995) for a notional town. The results for Oslo are presented in table 5.1 (rounded off to the nearest one or two significant digits).[54]

The results indicate that in about 90 fatalities in Oslo, equivalent to 1.4 per cent of the total, death was hastened by episodes of high local air pollution. As mentioned earlier the size of the lifetime reduction is unclear. By way of comparison, Pearce and Crowards (1996) calculates that 7 000 persons die each year in England and Wales as a result of episodes of particulate concentration. This calculation was made for the urban population which in his calculation accounts for 44 per cent. Bown (1994) makes a similar calculation but employs a higher figure for urban population (68 per cent). Her result is 10 000 deaths each year.

The calculations also indicate that each year about 400 new persons are diagnosed as suffering from chronic pulmonary disease resulting from polluting emissions in Oslo. This is on the reasonable assumption that the level of concentration in 1992 is representative of previous years. In the longer term it also probably causes an increase of about 100 persons (not an additional 100 each year) in the number of recipients of disability benefit. This corresponds to an annual disappearance of about 70 person-years from the labour market. In addition, a further 330 person-years may disappear in Oslo as a result of reduced productivity

[54] Data on for example population, deaths and employment (Statistics Norway 1994c,1995f) are used in the calculations. Some of the data are given for 1993 rather than 1992. Further, a full person-year is assumed to contain about 1 700 person-hours.

and short-term sickness absence. The person-years lost owing to short-term sickness absence are equivalent to between 5 and 10 per cent of total sickness absence lasting less than 14 days in Oslo.

Moreover, particulate emissions in Oslo (including asphalt dust) cause about 5 000 hospital bed-days per year, i.e. about 12 per cent of all bed-days related to respiratory diseases. This corresponds to expenditure of about Nkr 18 million. Almost half the bed-days are due to acute pollution episodes.

If we calculate the effect on mortality of long-term particulate pollution, we find that life expectancy for women and men, respectively, in Oslo is about 0.86 and 0.93 years less than it would have been in the absence of local pollution. It is not clear whether this is a uniform effect across the population or whether it for example entails that 10 per cent of the population has its life expectancy reduced by about 9 years. Another consequence is the further loss of 200 person-years per year in Oslo for people who die before reaching the age of 65. We should however reiterate the clarification by the WHO (1995) that further cohort studies are needed in order to draw firm conclusions. Calculations of the reduction in life expectancy, and the effects of such reduction, must therefore be regarded as rough and ready indications.

Section 5.6 showed the difficulty of establishing the size of the increase in NO_2-concentration caused by total local NO_x emissions. We found that a 40 per cent reduction in emissions would entail a reduction of 7.7 $\mu g/m^3$ in NO_2 concentration. If, for simplicity's sake, we extrapolate this to a reduction of 100 per cent, we find that the local contribution to the concentration is 19.25 $\mu g/m^3$. This is probably an underestimate inasmuch as the function seems to be somewhat concave. We nonetheless apply this concentration contribution to the dose-response functions in section 5.3. The uncertainty here is just as great as for particulates. The results are shown in table 5.2.

Table 5.2. **Total annual health effects and impacts on economic activity of local NO_2 pollution in Oslo, 1992**

	Annual effects
Acute effects:	
Bed-days in hospital (respiratory ailments)	1 300
Public hospital expenditure (mill. 1994-Nkr)	4.5
Person-years lost - reduced productivity	5

We note that the effects of NO_2 pollution are apparently far smaller than corresponding effects of particulates. As already mentioned, it is not clear to what degree this is due to measuring problems. According to the results the greatest health effect of NO_2 pollution is hospital admissions as a result of asthma attacks. This leads to more than 1 000 bed-days at hospital in Oslo each year, and costs the government more than Nkr 4 million. In addition there are person-hours lost due to absence from work, either because of parents' or children's hospital admissions. These are not included in the calculations. NO_2 pollution also leads to reduced labour productivity, quantified to the equivalent of about 5 person-years.

5.7.2. Social costs of air pollution

What, then, are the social costs of the health effects discussed above? The costs may be divided into pure economic costs and costs due to reduced quality of life. The purely economic costs refer primarily to the direct value of lost person-years and

of public hospital expenditure. The value of lost person-years can either be calculated with the aid of average hourly wages or average hourly labour costs (including employers' social security contributions). Hourly labour costs are probably the most relevant since they reflect the value of person-years for enterprises. In 1994 this value was Nkr 178.5 per hour while the average hourly wage was Nkr 135.1.[55]

In addition to the direct costs there are indirect effects on the economy through the workforce becoming a scarcer resource. By employing the general equilibrium model MSG-EE for the Norwegian economy (see chapter 2), we can calculate the total costs of the health effects. These are defined as the reduction in GDP plus the increase in public health expenditure. The model has been expanded to include the relations presented in this study. Thus, increased pollution leads to reduced employment and higher public expenditure. The calculations capture secondary effects in the economy due to a scarcer supply of labour. This causes changes in the inputs composition throughout the economy. Hence industry structure also undergoes change. An important effect is that a reduced supply of labour reduces production of capital goods so that growth in real capital slows down. Furthermore, higher public expenditure entails some withdrawal of resources from the private sector, which is assumed to be more productive than the public sector. These allocation effects are in addition to the direct productive costs and are often omitted in similar cost calculations (e.g EC (1995) and ORNL/RFF (1994)).

However, the most important health cost of air pollution is associated with the reduction in the quality of life resulting from higher risk of mortality and increased incidence of disease. Hence the total cost largely depends on how quality-of-life reductions are valued. This is a difficult question to which no objective answer can be postulated. Hence the physical effects should be allowed to speak for themselves when such information is sufficient. Sometimes, however, it would be preferable to make calculations based on concrete valuations to exemplify how large the costs may be. In our study this is done for the two major health effects, i.e., increased mortality and chronic pulmonary disease. The valuations are based on various willingness to pay (WTP) studies.

For mortality we employ the estimate used by the Norwegian public administration to value a statistical life. This was devised by the Institute of Transport Economics (ITE) in the context of road accidents, and is equivalent to 10.5 million 1993-kroner (Elvik 1993). It is open to question whether this is relevant for deaths resulting from pollution inasmuch as the residual life expectancy may be lower than the residual life expectancy in the case of road accidents. The estimate from ITE only expresses the welfare effect and is therefore relatively low compared with corresponding international estimates. For instance Pearce (1995) employs an estimate of GBP 1.5 million (i.e. about Nkr 15 million) and calls it conservative, while EPA (1995) uses an average estimate of USD 3.5 million (i.e. about Nkr 25 million).[56] Both these studies focus on deaths hastened by episodes of

[55] Since we are interested in the health costs in a year with a typical pollution situation, and specify costs in 1994-kroner, we use figures for 1994 instead of 1992.

[56] This is based on estimates of $3.4 million for persons over 65 years and estimates of $4.5 million for persons below 65 years.

air pollution. Another pertinent question is whether the value of a statistical life is underestimated when certain groups of the population (the elderly and sick) are more at risk than others. We do not wish ourselves to decide the value of a statistical life in this study, and point out that our choice is merely intended to be illustrative.

The same reservation may be applied to our choice of valuation in regard to the development of chronic pulmonary disease. Here we opted for an estimate used in the same connection by Rowe et al. (1995), which builds on study of willingness to pay (WTP). Their value is USD 210 000 (i.e. about Nkr 1.5 million). In comparison, the EPA (1995) chooses a value of USD 240 000, which is also based on WTP. It may be objected that these estimates overlap with the pure economic costs related to more disabilities and increased public expenditures. This depends on to what degree the WTP figure includes the consideration of reduced income in addition to reduced quality of life. As the estimates are obtained from American studies, where compensation is lower than in Norway, there is reason to believe that the income situation is taken into account. However, the pure economic costs in our results only cover about 10 per cent of the WTP, so the overlap is relatively low.

Given these assumptions the total health cost of air pollution in Oslo can be estimated. We have summated costs linked to the effects set out in table 5.1 and table 5.2. The results are shown in table 5.3. As mentioned, costs linked to mortality and chronic pulmonary diseases are to be regarded as illustrative. Since the value of reduced quality of life due to increased morbidity is not fully captured, the results are not intended to cover all health costs, or indeed other environmental costs.

Calculations performed using the MSG-EE model indicate that the total pure economic costs are about Nkr 163 million. At the same time the direct economic costs may be calculated to about Nkr 147 million, so that the allocation costs are Nkr 16 million. This means that the indirect effect of a reduced supply of labour and increased public expenditure amounts to about 11 per cent of the direct costs.[57] In fact the immediate effect related to structural changes in the economy places a slight damper on the direct cost. This is due to shifts in the economy from sectors with low productivity to sectors with high productivity. The more long-term effect, on the other hand, which is related to reduced rates of investment, contributes to raising the total cost. These costs are discounted at a rate of 7 per cent.

As seen from table 5.3, the total social costs of Nkr 1.7 billion depend heavily on how increased mortality and increased incidence of chronic pulmonary diseases are valued. With the valuation estimates we have employed, the purely economic costs are only around 10 per cent of the total costs. If we had applied the highest valuation estimates referred to above, the total costs would have been more than Nkr 3 billion. Thus, there are major uncertainties both with regard to the annual health effects and the valuations of these effects. The most important pollutant in our calculation is clearly particulates, as

[57] Had we valued the direct cost of lost person-years by using average hourly wages, the direct costs would have been 115 million Nkr, so that the allocation cost would have been 48 million Nkr, i.e., 42 per cent of the direct costs.

Table 5.3. **Total annual social costs associated with health effects of local air pollution in Oslo, 1992. Mill. 1994-Nkr**

	Total costs	Share of total costs Per cent
Person-years lost	125	7.2
Public expenditure	22	1.3
Allocation costs	16	0.9
Total (pure) economic costs	**163**	**9.4**
Increased mortality	936	54.2
Extra chronic pulmonary diseases	629	36.4
Total costs of reduced quality of life	**1 565**	**90.6**
Total social costs	**1 728**	**100**

less than 0.5 per cent of the total costs stem from health effects of NO_2 pollution. This is mainly because the quality of life reductions are associated with health effects resulting from particulate pollution.

Based on the discussion in section 5.6 we know that 60 per cent of PM_{10} concentration stems from vehicle traffic, and that almost half the contribution by vehicle traffic is due to the use of studded tires. Hence these shares can be used to calculate directly the health effects and social costs attributable to various particulate sources. For example, annual social costs associated with use of studded tires amount to about Nkr 480 million. However, it is necessary to discuss whether PM_{10} really is the particulate to focus on. As mentioned in section 5.2, there is a possibility that $PM_{2.5}$ is a better indicator, and that the gravest health effects observed in connection with PM_{10} pollution may primarily be associated with $PM_{2.5}$. If this is the case, use of studded tires is less hazardous than suggested above, even though it probably also contribute somewhat to the $PM_{2,5}$ concentration. On the other hand, if PM_{10} concentration is less harmful than indicated here, $PM_{2.5}$ concentration is *more* harmful such that the total cost is about the same. This is due to the relationship between $PM_{2.5}$ and PM_{10} which is about the same in Oslo as in most towns where epidemiological studies have been carried out.

We have also calculated the marginal cost of increasing emissions of particulates (PM_{10}) by one tonne in Oslo in 1995.[58] This increases concentration in 1995 and entails a reduction in the supply of labour etc., in 1995 and to some extent in the years ahead (owing to the long-term effect of pollution). Here too the contraction of GDP for the years after 1995 is discounted at a rate of 7 per cent per year. The same discount rate applies to the valuation of new chronic pulmonary diseases arising after a period of several years. Table 5.4 shows the marginal cost. It also shows the marginal cost per litre of petrol and diesel based on figures for average emissions per litre for the entire vehicle population. We note that costs related to use of diesel are particularly high. This is because diesel engines emit on average more then 10

[58] Owing to the large uncertainty attached to health effects of NO_2 exposure, we have chosen not to calculate any marginal cost for NO_x emissions. Such a calculation would probably greatly underestimate the real marginal cost.

times as many particulates per litre of fuel as petrol engines. In addition we have calculated the social cost of using studded tires, due to asphalt dust. This is based on data on annual traffic volume in Oslo (Vegdirektoratet 1996) and the assumption that 75 per cent of vehicles in Oslo use studded tires in the winter half-year. The average cost of using studded tires is then about Nkr 0.40 per kilometre, while costs for light vehicles are somewhat lower and for heavy vehicles somewhat higher.

A reservation is made here about the importance of PM_{10} versus $PM_{2.5}$. If $PM_{2.5}$ is the pollutant to focus on, the cost per kilometre will be lower, while the cost per litre of petrol and diesel will be higher. It is also worth mentioning that if particulates are above all an indicator of a pollution situation where components such as NO_2, SO_2 etc., are more or less equally harmful to health (see discussions earlier in this chapter), the cost estimates for petrol and diesel will tend to converge. Moreover, the same applies if the PM_{10} concentration is partly caused by emissions of other components like NO_x or SO_2 (in the form of nitrate and sulphate particles). However, according to the Norwegian Institute for Air Research, this is not of importance in Norwegian towns.

Here too the marginal cost does not cover all social costs of health damages, nor indeed other environmental costs. If other valuation estimates are desired, e.g. for mortality, the shares shown in table 5.3 may be used to calculate marginal costs. We find for example that pure economic costs make up about 10 per cent of the total, i.e. about Nkr 190 per kilogram of PM_{10}.

Table 5.4. **Social health costs associated with particulate pollution in 1995. 1994-Nkr**

	Marginal cost
PM_{10}	2 020 per kg
Petrol	
- light vehicles	0.54 per litre
- medium vehicles	0.54 per litre
- heavy vehicles	0.15 per litre
Diesel	
- light vehicles	6.6 per litre
- medium vehicles	7.3 per litre
- heavy vehicles	4.0 per litre
Use of studded tires	0.41 per km

We have so far disregarded costs associated with increased mortality resulting from *long-term* particulate pollution. As mentioned this is due to the very great uncertainty attached to these estimates. Moreover, it is difficult, if not impossible, to value reductions in lifetime expectancy. We have however studied what effect this may have on the economy in terms of people in the workforce dying before retirement age. The marginal cost per kilogram of PM_{10} in terms of purely economic effects increases in this case by about Nkr 30, i.e. by 15 per cent. However, the sickness period prior to death, which may well be of even greater importance, is not included here.

5.8. Conclusion

This study has described a number of associations between air pollution and various health effects as demonstrated by international studies. We have also shown how these effects may impact on economic activity via changes in sickness absence and public health expenditure. We closed with calculations of health effects in Oslo and endeavoured to quantify social costs associated with such effects.

In recent years international literature has presented an increasing number of epidemiological studies of the relations between air pollution and various health effects. The relation between short-term changes in particulate concentration and the number of deaths in the population is especially well documented, and a surprisingly high degree of concordance is found between the results observed in various towns. Fewer studies are currently available for other health effects, but results so far give a good indication of what may be expected in the way of health damage at various pollution levels. An interesting feature of several recent studies is that associations between particulate pollution and health effects appear to arise at relatively low concentrations. This has prompted an expert group at the WHO (1995) to cease recommending guidelines for particulates. Hence the results observed in other towns are probably also relevant for Norwegian towns.

The calculations for Oslo illustrate that air pollution has a substantial effect on the state of health of the population, and that this may entail a major cost for society. This cost is partially linked to pure economic effects which are calculated at about Nkr 160 million per year, but the biggest costs are probably associated with reduced quality of life. We have inter alia found that around 90 persons may die prematurely each year in Oslo as a result of air pollution, and that about 400 persons may be diagnosed as suffering from chronic pulmonary disease each year. We have also seen that the long-term consequences of air pollution in terms of reduced life expectancy may be even more serious.

Although some uncertainty attaches to the functions used, it is important to specify that the greatest uncertainty refers to items for which functions have yet to be established. This applies above all to the long-term effects of air pollution, of which we still have inadequate knowledge. We have discussed certain relations of this type in the study, based on the few studies that have been carried out. Air pollution has also been demonstrated to affect the body's immune system, indicating that the risk of falling ill is increasing (National Institute of Public Health 1995). However, this effect is difficult to quantify inasmuch as the initial stages of a disease may extend over a long period if the immune system is impaired. Results indicating that increased risk of cancer accompanies increased air pollution are also a part of this picture (see for example Törnkvist and Ehrenberg (1992) on cancer risk associated with PAH). There is also a real danger that the health effects of NO_2 are substantially understated in this study.

These uncertainties entail that calculations of environmental costs based on available dose-response functions only capture parts of the cost. This is also a key point in EC (1995). Moreover, when calculating social costs of health damages in Oslo we have not valued all costs associated with reduced quality of life. However, some physical health effects are stated, enabling costs to be calculated based on various choices of 'price tickets'. This lends greater transparency to the result, and required variables can be extracted. The study shows that the approach chosen for valuing the reduction in quality of life that results from higher mortality risk and increased incidence of disease is crucial for the size of costs involved.

6. Modelling impacts of traffic injuries on labour supply and public health expenditures

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6.1. Introduction

A simple model framework for assessing environmental costs and other external costs in road traffic in Norway was presented in Alfsen et al. (1992) and Brendemoen et al. (1992). They calculated several important costs associated with fossil fuel consumption, in order to establish some systematic information about local benefits from carbon emission control. In Brendemoen et al. (1992) it came out that traffic related costs were dominant among external marginal costs of fossil fuel consumption in Norway. This has called for a further effort in investigating traffic related externalities, in the same manner as for the environmental externalities elaborated in chapter 3, 4 and 5 in this book. Recently, the data on particularly traffic *accidents* and related costs in Norway have been improved considerably. This study elaborates on the relations between fuel use by road vehicles, traffic accidents and the labour supply within a CGE[61] model framework. The approach is simultaneous, in the sense that economic growth increases road traffic volumes causing person injuries with feedbacks to labour supply and public health sector costs, which in turn affects the economic growth.

The welfare loss associated with individual suffering from reduced health standard is not included in this study. Instead, by intention, we focus on impacts which can be traced along flows of goods and services. The valuation of suffering and limited physical capabilities of the victims is not left out because this cost is assumed to be insignificant. Haukeland (1991) provides considerable information on how traffic injuries affect life quality of those who survive. This is further evaluated in economic terms by Elvik (1993). Our guideline here is, however, to describe the link between the scale of traffic injuries and the national economy as pictured by GDP, via the effect on the available labour resources and the drain on public health expenditures. Our approach is a somewhat narrow human capital approach aiming at catching some relevant impacts for general policy simulations.

For this study, the question whether costs are internal or external to the driver is not of primary concern. We only focus on how

[59] Statistics Norway.
[60] Fellesekspedisjonen for Medisinsk Informasjon (Statistics Norway at the time of the study).
[61] Computable General Equlibrium.

traffic injuries affect the available resources to the society in terms of labour supply and the subsequent impact on production and income generation. The question of how costs are allocated and which incentives they generate is not looked into. However, the extensive public health and social security system in Norway makes medical costs almost completely external to the car driver. The cost of traffic accidents identified in our study has the strength, compared to more partial studies, that it includes the indirect effects throughout the economy of setting, in the case of Norway, some 20 000 man-years out of function every year due to traffic injuries. Also, the public hospital costs are evaluated including the dead-weight loss due to distortive taxes, which is dealt with in the CGE-model. These are improvements compared to Alfsen et al. (1992) and Brendemoen et al. (1992), where the costs were calculated in a linear *sub-model* of the CGE-model (based on the CGE-forecasted consumption of gasoline and diesel for various economic policies).

We start by giving a description of the available sources of information on traffic accidents and person injuries. Then, in section 6.3 the model framework is set out. In section 6.4 we discuss the association between traffic accidents and gasoline/diesel consumption (and other variables). The reductions in the labour force, contemporary and permanent, are described in section 6.5. Section 6.6 contains information about public costs related to traffic injuries. Finally, we present scenario simulations illustrating the feedback effects of traffic accidents within a CGE modelling framework.

6.2. Data sources

A statistical study of the association between the incidence and severity of person injuries in road accidents, and potentially explanatory variables at an aggregate level has been carried out by Fridstrøm and Bjørnskau (1989). The combined cross-section and time-series study is based on monthly data for 18 counties in Norway over the years 1974-1986. Road casualties are explained by fuel consumption (instrument for driving distance), traffic density, road capital, precipitation, daylight, road maintenance and several variables related to drivers' behaviour and traffic control. Traffic accidents with material damage only are not included in the study.

Hagen (1993) establishes a social accounting system for the costs of traffic accidents, i.e. an accounting of various cost elements like hospital costs, treatment costs etc. The purpose of the accounting system is to improve estimates of total costs generated by traffic accidents and to facilitate annual up-dating. Among sources of input to this information and accounting system, we briefly mention below those which in particular have been relevant for our CGE study of costs due to traffic injuries.

Haukeland (1991) surveyed some welfare impacts of person injury in traffic accidents. This is a thorough study of the health and living conditions of the injured 1-5 years after the accident took place. The impact on relatives of the injured or other persons was not assessed, except for the value of some supporting services to the injured. For injured children, however, the impact on family members is included. In our study we have applied the information on typical reduced ability in working situations to assess the impact on labour productivity. Also, a survey by Elvik (1988) provides information on the reduction in the labour supply associated

with temporary absence from work. In Hagen (1993) there are data on sick leaves related to traffic accidents.

In our model, the cost of labour productivity loss is spread out among production sectors, via the reduced supply of labour and rising real wage.

6.3. The model framework

The interlinkage between general economic activity, traffic injuries and labour supply is modelled in the following way, see figure 6.1: Aggregated use of gasoline (B)[62] and autodiesel (D) in the economy is calculated by the computable general equilibrium model MSG-EE (Alfsen et al. 1996) for the Norwegian economy. A brief description of MSG-EE is given in chapter 2 of this book. Then, a traffic injuries module within the MSG-EE model framework, called FEEDBACK, relates the use of transport fuels to the traffic volume (KM):

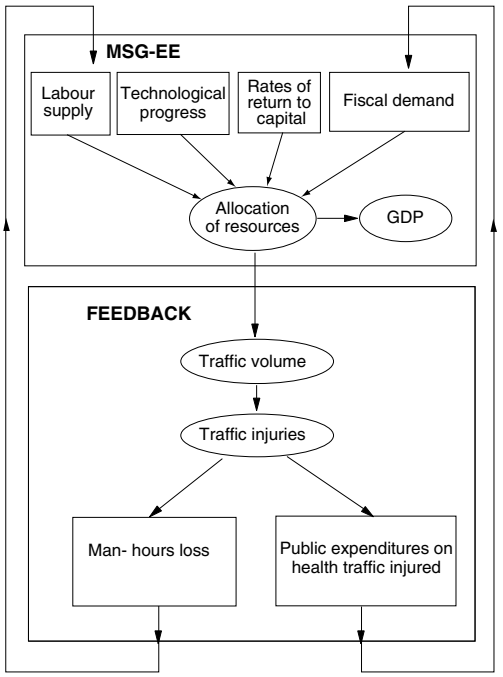
(6.1) $KM = KM(B, D, E)$

The rate of energy saving (E) in vehicles is included to take account of the fact that more kilometres will be driven per unit fuel in the future. Then, the number of traffic injuries (S) is modelled as a function of the traffic volume, traffic density (CON) and a trend variable (T):

(6.2) $S = S(KM, CON, T)$.

The traffic density is determined by traffic volume (vehicle kilometres) in relation to the extension of the road net. The road net measured in kilometres is assumed to follow the trend regressed on historical data from 1966-1993 and data from the Road Plan for Norway 1994-1997. In section 6.4 we elaborate further equations (6.1) and (6.2).

Figure 6.1. **Modelling impacts of traffic injuries in MSG-EE**



Rather than focusing on the total effects of traffic injuries, we are interested in changes due to an increase or decrease in the number of injuries compared to a base year. Thus, we will relate all changes below to the number of injuries diverging from the base year level (ΔS). In the FEEDBACK module we have focused on two main types of feedbacks from traffic injuries to the macro model:

- Changes in labour supply
- Changes in public expenditure

[62] All variables presented in this section relate to the current period t, thus the index t is dropped from the variable notations.

Changes in labour supply in the economy (ΔL) due to changes in traffic injuries consist of present changes (ΔL^C) due to short term sick leaves and less productivity for traffic injured back at work, and permanent changes (ΔL^P) due to fatalities and disabilities. Both are functions l of the change in traffic injuries (ΔS):

$$(6.3)\ \Delta L = \Delta L^C + \Delta L^P = l^c(\Delta S) + l^p(\Delta S)$$

The relations for present and permanent changes in labour supply are discussed in section 6.5.

In the macro model the labour supply is exogenous and based on population forecasts. In our study we assume that these population forecasts reflect death rates and employment rates in the base year. When the number of traffic injuries in our model deviate from the base year level, labour supply is adjusted accordingly. Thus, the socio-demographic forecasts of labour supply (L) is adjusted by:

$$(6.4)\quad L^* = L + \Delta L$$

which define the updated labour supply L^* in the macro model.

Public expenditures are generally exogenous, but we allow expenditures due to traffic injuries to be sensitive to changes in demand. The *public resources* allocated to capital (G_K^H), labour (G_L^H) and materials (G_M^H) used for treatment of traffic injuries in the health sector are assumed proportional to the number of injuries:

$$(6.5)\quad \Delta G_i^H = g_i^H(\Delta S)\ ,\ i = K,L,M$$

Overall public expenditures (G_i^H) are then adjusted for changes in public resources allocated to treatment of traffic injuries in the health sector:

$$(6.6)\quad G_i^{*H} = G_i^H + \Delta G_i^H\ ,\ i = K,L,M$$

Section 6.6 elaborates these functions further.

6.4. Traffic accidents as a function of fossil fuel consumption and other variables

The study by Fridstrøm and Bjørnskau (1989) provides information for relating the number of person injuries in traffic accidents to fuel consumption as an instrument for driving distance. Gasoline fuelled cars generate 85-90 per cent of the traffic volume measured in vehicle kilometres, although they represent only 60 per cent of the total road transport fuel use. It turned out in their estimates that a 10 per cent increase in driving distance of gasoline fuelled cars leads to a 8-9 per cent rise in the number of traffic injuries, other factors held constant. All types of person injuries, including fatal accidents, are equally affected.

Contrary to what is frequently argued, for instance by Barker et al. (1993), but consistent with other studies (see below), Fridstrøm and Bjørnskau found that more available roads and less crowded traffic increase the number of person injuries. A study of the impact on traffic accidents of building parallel roads in Denmark supports this result (Vejdirektoratet 1979), showing that when new roads take over traffic from the existing roads, the number of accidents on the old roads decreases less than proportional to traffic volume. Thus, high traffic density may seem to have a beneficial effect on the number of accidents with person injuries. The reason may be found in lower speed and possibly more alert drivers. However,

the results are ambiguous concerning the impact of traffic density on accident rates in general. Similar to Fridstrøm and Bjørnskau, Zlatoper (1987) found a significant negative effect of *rural* traffic density on motor vehicle deaths in a cross-sectional study for the US. *Urban* traffic density on the other hand, had a positive although statistically insignificant effect. Later, Zlatoper (1991) found a positive effect of traffic density on motor vehicle death rates. Vitaliano and Held (1991) found no significant evidence that traffic density affects accident rates in New York State. However, they included accidents with property damage in their sample, which makes comparison difficult, since it is possible that accidents with property damage respond differently to changes in traffic density than do accidents with person injuries.

The relation between traffic density and accident rates is crucial for the determination of *external* costs related to road traffic. Even privately financed insurance costs are exposed to externalities, *if traffic flow affects the rate of accidents* (Newbery 1988). In his study of road user charges, Newbury applies an assumption that the ratio between accident rates of marginal and average vehicle kilometres (VKT) is 1.25, which means that the risk of accident is higher the more cars are on the roaad. This estimate is a compromise between Vickery's (1969, cited by Newbury 1988) ratio of 1.5, with evidence from California freeway driving, and the practice applied by Department of Transport in Britain based on results showing no significant externality taking place, i.e. a ratio of 1. On the other hand, the statistical evidence from Norway in Fridstrøm, and Bjørnskau (1989) (see above) points to a *positive* externality, since increasing traffic volume reduces the average risk per kilometre of an accident with person injury. This effect applies particularly to deaths among pedestrians and bicyclists, but also on fatal accidents in general.

Below, we state formally the above mentioned effects for Norway as implemented in our model. Other variables than fuel use and traffic density are assumed to be constant (road capital, exposure of pedestrians, climatic variables, demographic variables). Equation (6.7) describes the number of injured (S_t) in year t as a function of the total driving distance in kilometres by gasoline cars (KM^B_t), diesel vehicles (KM^D_t), a traffic density (congestion) index (CON_t) and a trend term ($e^{\varepsilon t}$). The derivation of the functional form and the values of the elasticities are documented in appendix C. S_t includes all accidents with person injury where one or more vehicles are involved (also bicycles). Costs where only bicycles are involved are negligible, however. Relative changes in S_t are proportional to relative changes in explanatory variables.

(6.7)

$$S_t = K \cdot e^{\varepsilon \cdot t} \cdot (KM_t^B)^{\alpha} \cdot (KM_t^D)^{\sigma} \cdot \left(\frac{CON_t}{CON_0}\right)^{\beta}$$

The trend term ($e^{\varepsilon \cdot t}$) is included to take care of other variables than fuel consumption and congestion, which might affect the frequency of injuries, but which is not specified in our study. According to Fridstrøm and Bjørnskau (1989), there was a considerable downward shift in risk of person injury for a given traffic volume in the period 1974-1986. The probability of an accident with person injury would have been 50-60 per cent higher than observed in 1986 if it had not been affected by other variables than fuel consumption since 1974. To the extent that congestion

has been increasing over time, part of this effect is taken care of by CON_t in our model. However, substantial downward shift in the probability of person injury followed other particular events in the estimation period. The most important one was the introduction of seat belts which was estimated to explain 20 per cent of the observed risk decline.

In the future, several events might affect the risk of injury. Although the effect of introducing seat belts is no longer relevant (the frequency of seat belts reached 90 per cent in 1986), there is further technological potential for lowering the risk of injury (airbag etc.). In the opposite direction works an increasing share of elderly drivers in the future (possibly with a higher risk of accident) and the tendency towards an increase in exposure of bicyclists. A recent study (Borger and Frøysadal 1993) found that the transport work by bicyclists (15 years and older) in 1992 was 150 per cent above the 1987-level. Thus the exposure of unprotected participants in the traffic show a rising trend. The model user may test assumptions on shifting behaviour through the trend variable ε. It is, however, important to remember that the downward shift in risk may be attended with considerable costs. These costs may take different forms, like investment in infrastructure to separate cars from pedestrians and bicyclists, the time cost of pedestrians/ drivers, or the stress involved in taking care of children's or old people's security.

Equations (6.8) and (6.9) transform gasoline and diesel consumption to kilometres driven. M_O^B and M_O^D are base year mileage per tonnes of gasoline and diesel, respectively. B_t and D_t are consumption of gasoline and diesel, and θ and ω are fuel specific annual rates of energy efficiency increases in gasoline and diesel vehicles, respectively.

$$KM_t^B = M_0^B B_t e^{\theta t} \tag{6.8}$$

$$KM_t^D = M_0^D D_t e^{\omega t} \tag{6.9}$$

Equation (6.10) defines the traffic density variable CON_t as the relation between total traffic, measured as vehicle kilometres ($KM_t^B + KM_t^D$), and total length of the road system ($ROADS_t$).

$$CON_t = \frac{(KM_t^B + KM_t^D)}{ROADS_t} \tag{6.10}$$

In our calculations, $ROADS_t$ is exogenous and determined as an extrapolation based on historical undertakings in road construction measured in kilometre roads. An exponentially decreasing function is fitted to historical data from 1966 to 1993 and numbers for 1994-1997 from the Norwegian Road Plan, see appendix C. The decreasing growth rate of available road length may reflect that the rate of investment is falling and/or that investments per kilometre road are rising. Alternatively, we could have specified *ROADS* endogenously as a function of economic activity.

The possible effect on the number of traffic injuries of improved road standard, measured as increasing *investments* per kilometre road is not included in this model. The impact of increased road capital per kilometre came out ambiguous in the statistical study by Friedstrøm and Bjørnskau (1989). At the county level, higher road capital significantly reduced the number of injured persons, but on state roads, the effect was opposite (also significant). However, increasing road

maintenance had an unambiguous (negative) impact, reducing the number of injured. This effect is not included in our model.

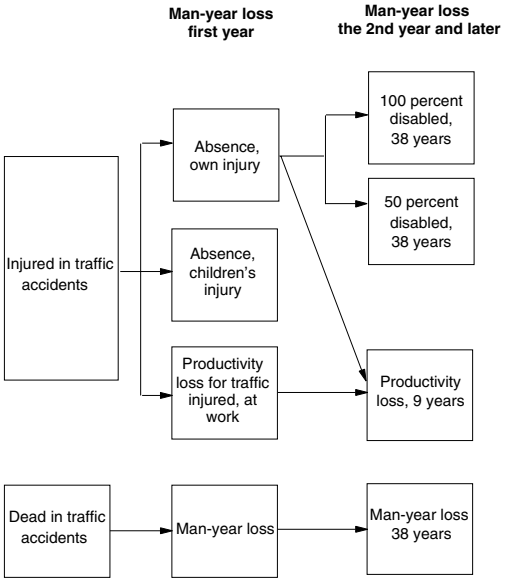
6.5. Labour supply reductions due to traffic accidents

Each year a number of persons are injured in car accidents. By expectation, a certain share (69 per cent) of traffic accident victims are members of the labour force and on average each working person represents 0.8 man-years due to some amount of part time working (Statistics Norway 1992c). Some of these are permanently excluded from work due to death or reduced capabilities from road accidents. Those cases are discussed in section 6.5.2 below. Others are temporary absent from work or less productive in a period after returning to their jobs. This is discussed in section 6.5.1.

In the FEEDBACK module, the labour supply variable is affected when the number of accidents deviates from the base year level. Thus we implicitly assume that labour supply along the reference baseline path in the CGE model does not reflect any foreseen changes in traffic accident rates.

Figure 6.2 sketches the various elements which are included in the injury/labour supply-module. Non-fatal injuries affect the labour supply in several ways. The first year after the accident, there is absence from work due to sick leaves for own injury or children's injuries. Also, injured returning to work have somewhat reduced productivity. After the first year, those injured who are not going to fully recover, are classified as being 100 per cent or 50 per cent disabled. The remaining persons are assumed to be less productive at work than before the injury.

Figure 6.2. **Impacts of traffic accidents on labour supply**



Productivity loss is assumed to take place until 10 years after the accident. Since most victims are young people, disability as well as fatalities on average lead to loss of 38 man-years. These elements are discussed further in the following sections.

6.5.1. Temporary reduction in labour supply

By temporary reduction in the labour force we mean sick leaves during the first year after the accident, and productivity losses up to the 10th year after the accident. After 10 years, the productivity impact is assumed to be negligible. Thus, the change in labour supply due to temporary reduction in year t (ΔL_t^T) is depending on the number of accidents (S_t) in the 10 previous years, and in the base year (S_0).

$$\Delta L_t^T = \sum_{\tau=0}^{9} \beta_\tau (S_{t-\tau} - S_0) + \sigma(S_t - S_0) + \lambda(S_t - S_0) \quad (6.11)$$

The first term on the right hand side of equation (6.11) sums up the change in productivity for employed workers who were injured τ years earlier, but still suffer in year *t*. All changes are measured in man-years. The next element reflects absence due to sick leaves the same year as the accident takes place. This is related to own injury. Moreover, in some cases members of the labour force stay away from work to care for other injured. Among these cases the available information only allows for including absence due to taking care of injured children in our study. This is reflected in the last element of equation (6.11). We assume all parameters to be constant in our model simulations. sections 6.5.1 provide the background information for the parameter estimates.

Productivity loss

When productivity of labour is lowered by a long term impact of traffic injury, the efficiency per working hour should be reduced, not the quantity. To simplify, however, we transform the efficiency loss to a reduced amount of man-years of the homogenous labour which is available in the model.

The parameters β_τ in equation (6.11) is the average loss of work effort (measured in man-years) per injured person τ years after the accident (τ= 0,...,9). β_τ are fixed coefficients estimated from data provided by Haukeland (1991). The study surveys the share of injured adults who suffer from the injury up to 5 years after the accident. Haukeland's sample contained injured with serious and heavy injuries, as well as minor head or neck injuries. Although his sample only included up to 5 year old injuries, there is reason to believe that even older injuries might cause productivity losses. Based on his observations, we have linearized the relation between the proportion of injured that are still suffering, and the number of years after the accident. In our model, we assume this relation to be valid up to 9 years after the accident.

To estimate β_τ we first identify the number of man-years (*MY*) performed by individuals who were injured τ years ago in a traffic accident, and stiller suffer:

$$MY = a \cdot AS_\tau - b \cdot SL_\tau - a \cdot INV_\tau \quad (6.12)$$

The injured adults still suffering (*AS*) are assumed to participate in the labour force with the same frequency (*a*) as the average of the working population. This working intensity is the product of two components. One component is the proportion of *persons* expected to be employed (i.e., 0.69 according to Statistics Norway (1992c)), and the other component is the average number of man-years per employed person (i.e., 0.80 according to Statistics Norway (1992c)), which is lower than 1 due to part time work. Thus, we find that a=0.55. To adjust for those who are suffering, but on sick leave, we subtract the number of persons on sick leave (*SL*), assigning a work participation rate of b= 0.80 since they by definition are employed. We also subtract the expected work amount of those who are disabled, who by assumption are assigned the average work participation rate *a*. For each cohort τ of injured adults who still suffer, a certain share (R_τ) experience reduced capacity, implying a productivity

loss of 10 per cent on average.[63] Hence the man-year loss per traffic injured due to reduced productivity can be written as:

(6.13)
$$\beta_\tau = \frac{R_\tau \cdot 0.1 \cdot (a \cdot AS_\tau - b \cdot SL_\tau - a \cdot INV_\tau)}{S_0} \qquad (\tau = 0)$$

(6.14)
$$\beta_\tau = \frac{R_\tau \cdot 0.1 \cdot (a \cdot AS_\tau - a \cdot INV_\tau)}{S_0} \qquad (\tau = 1, \ldots, 9)$$

Then β_τ can be estimated from Haukeland's (1991) data. Equation (6.13) represents productivity loss due to accidents in the same year, taking account of the injured still on sick leave. In the following years, all have either returned to the working force or become classified as disabled, and equation (6.14) is simplified accordingly. Probably some fraction of the injured (although not disabled) suffers more than 10 years after the accident. A possible effect on this group is not included in our study.

Absence from work due to own and children's injury
The average loss of man-years per injured associated with own and children's injury are σ and λ respectively (equation 6.11)). The coefficient for own injury is based on Hagen (1993), and found to be 0.04. Man-year loss associated with children's injury is estimated by Elvik (1988) for 1986, with information also from Lereim (1984) who studied the temporary absence from work related to traffic accidents in the city of Trondheim. The coefficient for children's injury is estimated to be 0.005.

6.5.2. Labour supply reduction due to fatalities and disability

Reduction due to fatalities
The effect of fatal traffic accidents in this model is limited to the impact of losing the labour potential of an average person killed in the accident. No value of life-considerations are made. The change in labour supply due to fatal traffic accidents $\left(\Delta L_t^F\right)$ is given by:

(6.15) $$\Delta L_t^F = -\eta(S_t - S_0)$$

where the coefficient η is determined by:

(6.16) $$\eta = \frac{a\, FA_0}{S_0}$$

FA_0 is the number of fatalities in the base year (1991), counting 332 persons (Bil og vei 1992), whereas the parameter a (=0.55) was discussed above. Thus, the labour supply reduction is calculated to be 186 man-years. The majority of those who are killed by traffic injuries are relatively young people. Elvik (1988) provides an age distribution for men and women killed in traffic accidents. Based on this age distribution and an upper age limit in the labour force of 74 years, it turns out that the members of the labour force killed by traffic injuries in average lose 38 active working years. The upper age limit of the labour force might seem high. However, the average number of man-years carried out by a person in the work force (a) is taking account of the low work participation rates of elderly people.

[63] R_τ corresponds to Haukeland's (1991) proportion of injured with reduced functional capacity.

Reduction due to disability
Disability also incur long term changes in labour supply (ΔL_t^D) and this is treated in a similar way to fatalities. However, disability occurs after an initial year on sick leave:

$$(6.17) \quad \Delta L_t^D = -\mu(S_{t-1} - S_0)$$

$$(6.18) \quad \mu = \frac{a\ INV_1}{S_0}$$

INV_1 is the number of persons who are declared disabled one year after an accident. We assume that those who have not returned to their work one year after the accident are transferred to job training or social pension. (The first year of sick leaves after the accident, including man-years of persons who later face disability, is dealt with in section 6.5.1)

According to Hagen (1993), 2.7 per cent of the traffic injured were on sick leave for more than a year. It is assumed that 75 per cent of these individuals later received pensions for their living (738 persons in 1991). Some of these have first been in jobtraining programs. We assume here, though, that job trainees have not contributed to productive activities. From Haukeland (1991) we know that 63 per cent of the disabled are classified as 100 per cent disabled, and the rest are classified as 50 per cent disabled. Moreover, we still assume that the injured persons on average worked 0.55 man-years before the accident. The average man-year loss per injury (μ) is then found to be 0.009 per year. It is assumed that disability lasts the whole lifetime. Thus, by assuming the same age distribution for disabled as for people killed in traffic accidents (Elvik 1988), we conclude that this yearly effect applies for 38 years on average.

Adjusting the labour supply
We assume the age distribution among injured and the share of deaths or disabled in traffic injuries to be constant. Hence the impact on the labour supply per injured will be constant throughout the simulation period. As the time horizon in our model simulations is shorter than 38 years, the man-year loss per killed and disabled is treated as a permanent shift in the labour stock. The labour supply is thus changed according to the following equation:

$$(6.19) \quad \Delta L_t^P = \Delta L_t^F + \Delta L_t^D + \Delta L_{t-1}^P$$

where ΔL_t^P is the permanent reduction in labour supply due to feedbacks from traffic accidents (in excess of the accident level in the base year).

6.5.3. Annual effects on labour supply of traffic accidents

Table 6.1 depicts some key variables of man-year loss related to traffic accidents based on the associations discussed in this section. In 1990, 33 900 persons were injured, of these 332 fatally. Man-year losses from the fatal accidents, including future losses, were about 7 000. Moreover, 13 000 man-years were lost due to disability. Productivity loss accounts for about the same reduction in labour supply as sick leaves (1 400 man-years). The resulting estimate of total man-year loss over the whole time horizon of injured is about 23 000.

6.6. Public health sector costs

Traffic accidents demand considerable resources through medical treatment and legal actions. When the number of accidents decline, these resources can be used for other purposes. In our model, we let the level of government expenditures in the

Table 6.1. **Key variables related to traffic accidents in 1990**

	Persons affected	Man-year loss
Deaths in traffic accidents	332	7 254
Traffic injured	33 900	
Sick leaves the first year after the accident (own injury)		1 350
Absence from work due to children's injury in traffic accidents		167
Productivity loss for former traffic injured (total for *ten years* after the accidents)		1 346
50 per cent disabled	272	2 888
100 per cent disabled	477	10 146
Total		**23 151**

health sector deviate from the exogenous reference path when the number of traffic accidents deviates from the base year level. The level of all other services from the public health sector is assumed to remain at the level of the reference path.
In addition to allow for more private consumption, the whole economy might gain some efficiency by transferring resources from public to private sector. As estimated by Brendemoen and Vennemo (1993), the marginal cost of public funds in Norway is about 1.6, i.e. 1 Nkr used in the public sector reduces the income in the private sector by 1.6 Nkr.

Elvik (1988) calculated the total medical treatment cost of traffic injuries to be 229 million Nkr in 1986, including 6 million in medical care in the case of fatalities. The complete surveys of the number of traffic accident do not go back to 1986. However, so does the police registration of traffic accidents. To find the level of health costs in our base year (1991), we assume that the cost developed proportionally to the police registered number of traffic accidents over the years from 1986 to 1991, adjusting for inflation which was 30 per cent from 1986 to 1991 (Statistics Norway 1996b). The number of police registered accidents fell from 12 458 in 1986 to 12 035 in 1991 (Bil og vei 1992). The health sector expenses thus amounted to 289 million Nkr in 1991. These costs are mainly hospital expenses. With 36 450 injured persons in 1991, the average medical cost per injured was 7 900 Nkr.

Table 6.2. **Public health sector expenses in 1991 due to traffic accidents[1]. Million 1991-Nkr**

Public sector:	
Hospital stay	334
Nursing homes	146
Policlinic consultations and treatment	42
Ambulance transport	3
Primary health sector consultations (and physiotherapy)	18
Total public sector costs	**543**
Private Sector:	
Nursing homes	36
Policlinic consultations and treatment	11
Primary health sector consultations	4
Medicines	203
Total private sector costs	**254**

[1] Hospital stay is 100 per cent publicly financed, while the private sector covers 20 per cent of costs in other health institutions.
Source: Hagen (1993).

Hagen (1993) provides a revised estimate for 1991. This survey includes more elements of medical care than Elvik (1988). Table 6.2 presents the main elements in the cost assessment. The estimated hospital costs are about the same level as estimated by Elvik (1988), but they make up only 60 per cent of total public health sector expenditures. For an

overview, table 6.2 also presents the private sector costs, although they are not included in the FEEDBACK module.

In this study we focus on the public sector costs. Table 6.3 presents the distribution of *variable* hospital costs on various input variables. In addition comes *capital* costs. Due to the nature of the public sector accounting systems, data on capital costs are not readily available. However, a low estimate of capital costs is included in order not to completely overlook these real costs. The capital costs are set to 5 per cent of variable costs, which is regarded as a low estimate by the State Agency for Buildings and Property (as referred by Hagen (1993)).

We assume that the variable medical costs related to road traffic accidents are allocated on input factors roughly proportional to the general allocation of input factors in the hospital sector (see table 6.3). Thus, we assume that 70 per cent of variable costs are labour costs; the other 30 per cent are intermediates.

The activity in the public health sector is basically exogenously determined within the model, in the sense that the various factor inputs are fixed. When traffic accidents deviate from the base year level, the use of each input factor in the public health sector is adjusted according to the distribution displayed above:

$$(6.20)\ \Delta G_{it}^{H} = \omega_i^{H} \cdot (S_t - S_0)\ ,\ i = K,L,M$$

G_i^H is the specific cost related to input of factor i in public hospitals, which is proportional to the number of injured persons in traffic accidents (see equation (6.5) in section 6.3).

Table 6.3. **Variable hospital costs 1991. Per cent**

Wages	71
Equipment	2
Maintenance	1
Other costs	24
Transfers	1

Source: Statistics Norway (1993b)

Table 6.4. **Scenarios**

	MSG-EE without feedbacks	MSG-EE with feedbacks
Reference - constant 1993-level CO_2 tax	REF1	REF2
Alternative - increasing CO_2 tax	TAX1	TAX2

6.7. Simulations

In this section we first describe how the inclusion of the traffic module into the CGE model MSG-EE changes the reference scenario. Then we report from simulations of a CO_2-tax scenario, with and without the feedback from traffic injuries on labour supply and public health sector costs.

6.7.1. The scenarios

Simulations with two model versions have been carried out. Model version 1 is MSG-EE itself. Model version 2 is MSG-EE with the traffic injury module, adjusting labour supply and public health expenditures as explained above. For each of the models we first simulate a reference path where exogenous variables including the real values of the CO_2 taxes are kept constant at their 1993 levels throughout the simulation period 1988-2020. Hence, the reference scenarios REF1 and REF2 only differs when it comes to modelling of the feedbacks from traffic injuries. We then

Table 6.5. **Main variables, reference paths**

	LEVEL 1988	REF1 Annual growth 1988-2020. Per cent	REF2 Dev. from REF1, level 2020. Per cent
GDP (billion 1988-Nkr)	583	1.7	-0.34
Labour supply (million man-hours)	3 019	0.3	-0.29
Fuel use in road transport (1000 metric tonnes)	2 867	1.0	-0.35
Persons injured in traffic accidents	31 464	1.3	-0.24

simulate within both model versions the effect of introducing an increasing CO_2 tax. These scenarios, TAX1 and TAX2, are discussed in the next section.

Table 6.5 shows the development in the two reference scenarios. In REF1, based on demographic forecasts, labour supply grows by 0.265 per cent per year over the simulation period (1988-2020). Annual GDP growth is 1.65 per cent in the same period. This growth is absorbed by increased private consumption and a rise in government expenditures, while net export and gross investments are unchanged. Factor neutral technological progress is assumed to be 0.5 per cent per year in all the production sectors except for road, air and domestic sea transport. Based on a study by the Norwegian Institute for Transport Economics (Thune-Larsen 1991) annual autonomous fuel efficiency improvement for road transport using gasoline and autodiesel is assumed to be 1.0 and 0.6 per cent respectively, while the rates of improved energy efficiency for air and domestic sea transport are 0.9 and 1.3 per cent. Assumptions for exogenous variables are further documented in Johnsen et al. (1996). The assumptions are roughly based on the assumptions in the "KLØKT" analysis of climate policy problems on a national scale (Moum 1992).

As production and consumption increase in the REF1 scenario, the total fuel use in road transport also increases. The annual growth of 1 per cent in transport fuels corresponds to an annual growth of 2 and 1.6 per cent in kilometres driven by gasoline and diesel vehicles respectively. The direct effect of the growth in road transport activity is an increase in the number of persons injured or killed in traffic accidents. However, the traffic growth also has an opposite smaller effect on the injuries through increased congestion. In total, the number of person injuries rises by 52 per cent over the simulation period in the REF1 scenario, to reach 47 831 injured per year in 2020.[64]

In the REF2 scenario the increase in injuries and fatal accidents leads to a fall in labour supply as a first order effect. The negative feedback on labour supply will slightly slow down the economic growth and, ceteris paribus, the increase in fuel use and accidents in road transportation. An opposite second order effect on accidents occurs when the decrease in labour supply increases the wage rate to clear the labour market. The substitution

[64] The number of persons injured or killed in 1988 (i.e., 31 464) is based on the estimated number of 33 900 persons injured or killed in 1990 (Haukeland 1991) and adjusted proportionally to the change in police registered traffic accidents from 1988 to 1990 (Bil og vei 1992).

Table 6.6. **Main variables, tax simulations**

	Deviation 2020. Per cent	
	TAX1 vs. REF1	TAX2 vs. REF2
CO_2 tax	198	198
GDP	-0.47	-0.44
Labour supply	0	0.02
Labour use, public health sector	0	-0.03
Materials/energy use, public health sector	0	-0.04
Capital use, public health sector	0	-0.02
Fuel use in road transport	-4.94	-4.92
Person injuries, road traffic	-3.37	-3.35

effect of this change in relative prices is an increased demand for other input factors, among them transport fuels, accompanied by an increase in the number of traffic accidents.

The increase in traffic accidents from the base year level also leads to a rise in public expenditures on labour, capital, energy and materials needed for treatment of traffic victims in the health sector. With a given amount of productive resources in the economy as a whole, these incremental needs in the health sector has to be covered by resources withdrawn from other sectors. Our results cover the efficiency loss when distorting taxes reallocate resources from private to public sectors. However, the effect turns out to be small.

When coming to terms with all of these effects, the number of persons killed or injured in traffic accidents in the REF2 scenario compared to the REF1 scenario turns out to be lowered by 114 in 2020 due to the feedback effects in the economy, whereas the effect on the labour supply is a 0.29 per cent decline in 2020. Although not insignificant, the decline taking place in the period 1988-2020 corresponds to only one year expected increase in the labour supply during the period. The reduced labour supply and increased public expenditures result in a GDP level in REF2 which is 0.34 per cent, or about 3.3 billion 1988-Nkr, lower than the REF1 level in 2020. The effect via reduced labour supply is far stronger than the effect via increased demand for resources in the public health sector. REF2 may be considered as a reference path which is an alternative to the "official" path from the simulation with MSG-EE without any feedbacks.

6.7.2. The impacts of a CO_2 tax

In the reference paths REF1 and REF2, the real value of the CO_2 tax is kept constant at the 1993 level throughout the simulation period, i.e. the nominal value increases by 3.5 per cent per year. Below we report some results from simulations with an increasing CO_2 tax designed to stabilise the carbon dioxide emissions in the REF1 scenario on a 1989 level around year 2020. See table 6.6.

The scenarios TAX1 and TAX2 show the impact of increasing the nominal value of the CO_2 tax by 10 per cent per year from 1994 to 2000, and then by 7 per cent per year from 2001 to 2020. The difference between the reference scenarios and the CO_2 tax scenarios for some main variables in 2020 are studied.

In model version 1, with no feedbacks from traffic accidents to the economy, the introduction of an increasing CO_2 tax reduces GDP by 0.47 per cent in 2020. The tax increases the cost of fuel use and thus implies a restriction on economic activity. For a further discussion of the impact of increased CO_2 taxes in MSG-EE, see Johnsen et al. (1996). It should be noted that the tax does not affect the labour supply in this model version. When simulating the impact of the CO_2 tax with model 2, the cost of carbon control comes out somewhat less expensive by 2020 than with the model 1 alternative. The tax reduces the fuel use and thus the traffic volume, leading to fewer accidents and increased labour supply. In total the tax reduces GDP by 0.44 per cent in 2020. Thus, the GDP reduction is about 0.3 billion 1988-Nkr smaller than in the Model 1 case.

6.8. Conclusions

It should be stressed that the simulations in this study are of illustrative nature. Although the scale of the effects on the labour force is fairly well documented, there are, of course, uncertainties associated with the data and the values used in the tentative calculations. However, the simulations indicate that climate policies in the long run might not be as costly – measured by GDP loss – as shown by model simulations without negative feedback effects to the economy from the use of fossil fuels. The above simulations only include two of these feedbacks. Other effects have been presented in other chapters of this book, and several others could be mentioned (e.g., congestion and noise). It should also be remembered that welfare gains from reduction in the number of person injuries is not included in this study.

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Appendix A

Appendix to chapter 3: Tables

Table A1. **Air quality and climate parameters by region**

Region	SO_2, µg/m^3	O_3, µg/m^3	H^+, mg/l	Regn m/yr	TOW
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	5-24	17-51	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
Kristiansand	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	55	0.031	1.03	0.42

Table A2. **Material-mix per type of building from the MOBAK study. Per cent**

Building type in GAB register	Mean area m^2/ building	Galvanised steel: Untreated: Sh. repl	Galvanised steel: Untreated: Sh. maint	Galvanised steel: Untreated: Wire	Galvanised steel: Untreated: Profile	Galvanised steel: Treated: Str.-lacq.	Galvanised steel: Treated: Painted	Aluminium: Untreat*	Aluminium: B.l.	Weath-ering steel*	Copper
Dwellings											
Small houses (incl. garages)	470	0.6	0.6	0.0	0.0	2.8	1.7	0.1	1.1	0.9	0.1
Block/condominium	2470	1.6	1.6	0.0		10.3	3.6	0.2	4.0	2.7	0.2
Mining/manufacturing	2450	2.0	2.0	0.0	0.0	21.4	2.7	0.4	6.2	4.5	0.4
Office/Comm./Transport											
Wholesale & retail, for-warding, warehouse, gar.	875	2.8	2.8	0.0	0.0	7.0	5.1	0.3	4.0	2.8	0.2
Office & admin.buiold., other build.	875	2.8	2.8	0.0	0.0	7.0	5.1	0.3	4.0	2.8	0.2
Hotels & restaurants	875	2.8	2.8	0.0	0.0	7.0	5.1	0.3	4.0	2.8	0.2
Public and private services	875	2.8	2.8	0.0	0.0	7.0	5.1	0.3	4.0	2.8	0.2
Agriculture, Forestry, Fishery etc.	785	1.6	1.6	0.0	0.0	8.7	3.6	0.1	3.7	2.6	0.0
Other buildings	470	2.0	2.0	0.0	0.0	5.0	3.6	0.2	2.8	2.0	0.2
Infrastructure (per capita)	9.9	0.0	0.0	12.1	7.1	0.0	80.8	0.0	0.0	0.0	0.0

	Wood stained/ painted	Plaster: Untr.	Plaster: Painted	Con-crete	Roofing felt	Tiles	Stone*	Glass*	Asbesto-cem.*	Other	Total
Dwellings											
Small houses (incl. garages)	42.5	2.5	5.7	13.2	5.1	11.6	1.5	3.9	5.2	0.9	99.9
Block/condominium	6.6	8.3	23.5	12.6	5.2	8.2	1.6	7.8	1.3	0.8	100.0
Mining/manufacturing	3.6	2.1	2.9	5.9	21.1	8.6	1.5	4.7	3.5	6.6	100.0
Office/Comm./Transport											
Wholesale & retail, for-warding, warehouse, gar.	18.9	1.3	7.5	8.0	19.0	12.3	1.5	5.3	0.4	0.8	100.0
Office & admin.buiold., other build.	18.9	1.3	7.5	8.0	19.0	12.3	1.5	5.3	0.4	0.8	100.0
Hotels & restaurants	18.9	1.3	7.5	8.0	19.0	12.3	1.5	5.3	0.4	0.8	100.0
Public and private services	18.9	1.3	7.5	8.0	19.0	12.3	1.5	5.3	0.4	0.8	100.0
Agriculture, Forestry, Fishery etc.	34.2	1.1	0.1	4.9	0.2	9.6	1.0	11.1	9.0	7.0	100.0
Other buildings	23.5	7.0	7.6	13.9	9.2	11.2	1.0	5.3	1.5	2.0	100.0
Infrastructure (per capita)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0

* These materials are not included in the cost calculations as damage functions are lacking.
Abbreviations: Sh.repl = sheeting, replacement, Sh. maint = sheeting, maintenance, str.-lacq. = strip-lacquered, untr. = untreated, Cem. = cement.

Table A3. **Geographical distribution of building categories. Per cent**

Region	Small houses/ other	Appartm. blocks	Manufac-turing	Office/ services	Hotel & rest.	Agri-culture	Infra-structure
Halden	0.7	0.5	1.2	0.5	0.5	0.6	0.6
Sarpsborg	0.3	0.5	1.6	0.2	0.2	0.0	1.1
Fredrikstad	0.6	0.8	1.5	0.5	0.5	0.1	0.6
Moss	0.6	0.7	2.2	0.5	0.6	0.1	0.6
Bærum	1.9	2.8	0.9	3.0	1.4	0.2	2.2
Asker	1.0	0.6	0.3	1.4	0.6	0.2	1.0
Oslo	5.3	46.6	10.6	13.7	11.3	0.1	11.0
Drammen	1.1	2.0	1.6	1.2	1.0	0.2	1.2
Porsgrunn	0.8	0.6	6.2	0.6	0.6	0.1	0.7
Skien	1.2	0.9	1.2	1.0	0.9	0.5	1.1
Bamble	0.4	0.1	3.9	0.2	0.4	0.1	0.3
Kristiansand	1.4	2.1	1.4	1.7	1.6	0.1	1.6
Stavanger	2.5	1.9	2.4	2.3	2.4	0.2	2.4
Bergen	4.1	9.8	2.6	5.9	5.5	0.3	5.1
Trondheim	2.7	5.9	2.3	3.9	3.7	0.7	3.3
Tromsø	1.3	0.8	0.7	1.5	1.7	0.2	1.2
Urban-south	25.1	7.9	20.1	20.9	22.6	32.5	22.3
Urban-north	5.5	1.7	4.4	4.5	4.9	7.1	4.9
Rest of the country	43.8	13.8	35.1	36.4	39.4	56.8	38.9
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Table A4. **Stocks of materials by area. 1 000m^2**

	Galvanied steel						Alumi-nium str.-lacq.	Copper
	Untreated				Treated			
Area	Sh.maint	Sh.repl	Wire	Profile	Str.-lacq	Painted		
Halden	89	89	31	18	410	397	175	8
Sarpsborg	42	42	56	33	240	460	91	6
Fredrikstad	81	81	32	19	373	382	156	9
Moss	80	80	30	18	396	362	159	9
Bærum	314	314	113	66	1 166	1 395	546	30
Asker	136	136	52	30	464	624	225	13
Oslo	2 059	2 059	575	335	9 693	8 127	4 150	220
Drammen	168	168	63	37	725	769	316	18
Porsgrunn	115	115	38	22	701	470	254	16
Skien	144	144	59	34	599	694	265	14
Bamble	56	56	17	10	374	215	130	8
Kristiansand	208	208	81	48	837	972	376	21
Stavanger	291	291	123	72	1 129	1 420	512	30
Bergen	740	740	265	154	2 986	3 296	1 352	75
Trondheim	493	493	171	100	2 006	2 157	905	48
Tromsø	172	172	65	38	604	783	288	16
Urban-south	3 404	3 404	1 163	678	14 238	15 006	6 394	260
Urban-north	742	742	254	148	3 106	3 273	1 395	57
Rest of the ountry	5 943	5 943	2 031	1 184	24 860	26 201	11 164	454
Total	15 278	15 278	5 219	3 044	64 908	67 002	28 853	1 311
Per cent	1.8	1.8	0.6	0.4	7.6	7.8	3.4	0.2

Table A4. Cont.

Area	Wood stained/ painted	Plaster		Con-crete	Roofing felt	Brick	Total	Per cent
		Untreat	Painted					
Halden	1 642	170	412	590	504	633	5 170	0.6
Sarpsborg	580	102	261	290	299	281	2 784	0.3
Fredrikstad	1 334	184	481	582	538	573	4 825	0.6
Moss	1 193	171	446	538	548	538	4 568	0.5
Bærum	4 591	626	1 788	2 005	1 957	2 042	16 953	2.0
Asker	2 248	231	650	874	877	925	7 483	0.9
Oslo	18 314	5 986	17 505	12 902	11 211	11 510	104 645	12.2
Drammen	2 481	392	1 074	1 148	1 037	1 119	9 515	1.1
Porsgrunn	1 649	220	519	734	889	769	6 510	0.8
Skien	2 705	293	743	1 023	866	1 051	8 634	1.0
Bamble	768	85	179	319	456	362	3 032	0.4
Kristiansand	3 157	448	1 242	1 398	1 311	1 393	11 701	1.4
Stavanger	5 101	587	1 569	2 067	1 919	2 084	17 195	2.0
Bergen	9 889	1 721	4 918	4 837	4 450	4 698	40 121	4.7
Trondheim	6 740	1 087	3 080	3 143	2 932	3 128	26 482	3.1
Tromsø	2 889	300	832	1 127	1 118	1 184	9 589	1.1
Urban-south	69 036	5 335	12 205	21 423	17 355	24 555	194 456	22.7
Urban-north	15 058	1 164	2 662	4 673	3 785	5 356	42 415	5.0
Rest of the ountry	120 540	9 316	21 311	37 405	30 302	42 874	339 526	39.7
Total	269 914	28 418	71 875	97 078	82 352	105 073	855 603	100.0
Per cent	31.5	3.3	8.4	11.3	9.6	12.3	100.0	

Table A5. **Stocks of materials by type of building for the whole country. 1 000 m²**

Building	Galvanised steel						Alumi-nium str.-lacq.	Copper
	Untreated				Treated			
	Sh.maint	Sh.repl	Wire	Profile	Str.-lacq.	Painted		
Small house	1 775	1 775	0	0	9 036	5 487	3 550	322
Apart.block	1 939	1 939	0	0	12 882	4 503	5 003	250
Manufact.	607	607	0	0	6 658	840	1 929	125
Office	6 021	6 021	0	0	15 054	10 968	8 602	431
Hotel	53	53	0	0	133	97	76	4
Service	871	871	0	0	2 177	1 586	1 244	63
Agriculture	2 860	2 860	0	0	16 085	6 654	6 838	0
Other	1 152	1 152	0	0	2 883	2 074	1 611	117
Infrastr.	0	0	5 219	3 044	0	34 793	0	0
Total	15 278	15 278	5 219	3 044	64 908	67 002	28 853	1 311

	Wood stained/ painted	Plaster		Con-crete	Roofing felt	Brick	Total	Per cent
		Untreat	Painted					
Small house	137 165	8 070	18 396	42 601	16 460	37 438	282 077	33.0
Apart.block	8 255	10 381	29 392	15 759	6 504	10 256	107 061	12.5
Manufact.	1 120	653	902	1 836	6 565	2 676	24 516	2.9
Office	40 644	2 796	16 129	17 204	40 859	26 451	191 178	22.3
Hotel	360	25	143	152	362	235	1 694	0.2
Service	5 877	404	2 333	2 487	5 908	3 825	27 645	3.2
Agriculture	63 249	2 057	204	9 034	398	17 743	127 981	15.0
Other	13 245	4 033	4 376	8 005	5 297	6 450	50 395	5.9
Infrastr.	0	0	0	0	0	0	43 056	5.0
Total	269 914	28 418	71 875	97 078	82 352	105 073	855 603	100.0

Table A6. **Maintenance prices**

			Price (Nkr/m^2)			
			Excl. VAT			Incl. VAT[1]
Type of material	Treatment	Assumption	Min	Max	Average	Average
Galvanised steel sheet	Maintenance	Cleaning + 2 coats of paint	100	200	150	183
Galvanised steel sheet	Replacement		250	300	275	336
Galvanised steel wire	Replacement		90	120	105	128
Galvanised steel profile	Maintenance	Cleaning + 2 coats of paint	250	350	300	366
Limestone/Cement plaster	Replacement	3-tiered new plaster incl. carve and scaffolding	300	400	350	427
Painted plaster	Maintenance	Repair + 2 coats of original paint	200	300	250	305
Copper roofing	Replacement	Incl. new felt	400	500	450	549
Strip-lacquered aluminium	Maintenance	Cleaning + 2 coats of paint	100	200	150	183
Strip-lacquered galvanised steel	Maintenance	Cleaning + 2 coats of paint	100	200	150	183
Painted galvanised steel	Maintenance	Sandblasting + 3 coats of paint	250	350	300	366
Roofing felt	Replacement	New covering, two layers	120	200	160	195
Painted/stained wood	Maintenance	Cleaning + 2 coats of paint	60	100	80	98
Brick	Maintenance	Repair, resealing incl. scaffolding	200	400	300	366
Concrete	Maintenance	Repair and painting incl. scaffolding	350	700	525	641

[1] 22 per cent in 1994. Our cost calculations are based on prices incl. VAT.

Table A7. **Material corrosion costs by region, 1994. 1 000 Nkr**

Region	Galvanised steel						Alumi-nium str.lacq.	Copper
	Untreated				Treated			
	Sh.maint	Sh.repl	Wire	Profile	Str.-lacq.	Painted		
Halden	109	134	18	15	47	467	14	3
Sarpsborg	172	210	107	89	102	2 029	27	7
Fredrikstad	131	161	24	20	63	673	18	5
Moss	103	126	18	15	45	425	12	4
Bærum	387	473	65	54	165	2 049	53	12
Asker	141	173	25	21	53	733	18	4
Oslo	2 421	2 959	313	261	2 277	19 243	671	100
Drammen	171	209	30	25	103	1 131	31	7
Porsgrunn	122	150	19	16	80	553	20	6
Skien	476	581	91	76	221	2 652	68	17
Bamble	30	37	4	4	21	126	5	2
Kristiansand	171	209	31	26	47	571	15	4
Stavanger	894	1 092	177	147	160	2 086	50	16
Bergen	2 120	2 591	354	295	508	5 813	159	50
Trondheim	652	797	105	88	228	2 535	71	19
Tromsø	40	48	7	6	17	230	6	2
Urban-south	2 424	2 962	386	322	808	8 820	250	55
Urban-north	171	209	27	23	88	962	27	7
Rest of the country	508	621	81	67	0	0	0	12
Total	11 243	13 742	1 883	1 569	5 033	51 100	1 515	332
Per cent	5.7	6.9	1.0	0.8	2.5	25.8	0.8	0.2

Table A7. Cont.

Region	Wood stained/ painted	Plaster		Con- crete	Roofing felt	Brick	Total	Per cent
		Untreat	Painted					
Halden	660	36	140	0	129	0	1 771	0.9
Sarpsborg	874	81	332	929	287	113	5 360	2.7
Fredrikstad	805	58	245	0	206	0	2 410	1.2
Moss	480	36	151	0	140	0	1 555	0.8
Bærum	2 307	166	758	0	625	0	7 114	3.6
Asker	904	49	220	0	224	0	2 565	1.3
Oslo	14 365	2 611	12 207	10 011	5 876	1 087	74 401	37.6
Drammen	1 247	104	455	0	331	0	3 843	1.9
Porsgrunn	663	46	176	0	227	0	2 077	1.1
Skien	3 535	202	819	3 276	718	423	13 153	6.7
Bamble	154	9	30	0	58	0	481	0.2
Kristiansand	635	47	211	0	167	0	2 135	1.1
Stavanger	2 564	156	665	0	612	0	8 620	4.4
Bergen	5 965	547	2 502	0	1 704	0	22 609	11.4
Trondheim	2 710	230	1 045	0	749	0	9 229	4.7
Tromsø	290	16	71	0	71	0	804	0.4
Urban-south	13 880	565	2 070	0	2 215	0	34 758	17.6
Urban-north	1 514	62	226	0	242	0	3 557	1.8
Rest of the ountry	0	0	0	0	0	0	1 288	0.7
Total	53 552	5 020	22 322	14 216	14 581	1 623	197 732	100.0
Per cent	27.1	2.5	11.3	7.2	7.4	0.8	100.0	

Table A8. **Material corrosion costs by type of building, 1994 (1 000 Nkr)**

	Galvanised steel						Alumi-	Copper
	Untreated				Treated		nium	
Building	Sh.maint	Sh. repl	Wire	Profile	Str.-lacq.	Painted	str.lacq.	
Small house	1 177	1 439	0	0	502	3 161	136	63
Apart.block	2 326	2 843	0	0	2 041	7 393	547	97
Manufact.	475	580	0	0	546	714	109	31
Office	4 779	5 841	0	0	1 175	8 867	463	103
Hotel	41	50	0	0	11	87	5	1
Services	656	801	0	0	167	1 263	66	14
Agriculture	1 020	1 247	0	0	420	1 799	123	0
Other	770	941	0	0	169	1 264	65	23
Infrastr.	0	0	1 883	1 569	0	26 552	0	0
Total	11 243	13 742	1 883	1 569	5 033	51 100	1 515	332
Per cent	5.7	6.9	1.0	0.8	2.5	25.8	0.8	0.2

	Wood stained/ painted	Plaster		Con- crete	Roofing felt	Brick	Total	Per cent
		Untreat.	Painted					
Small house	27 030	838	3 058	2 869	2 060	317	42 650	21.6
Apart.block	4 636	3 071	13 923	7 307	2 319	597	47 101	23.8
Manufact.	326	100	221	387	1 212	71	4 771	2.4
Office	11 240	407	3 762	2 148	7 175	415	46 375	23.5
Hotel	110	4	37	42	70	8	465	0.2
Services	1 600	58	536	474	1 021	91	6 748	3.4
Agriculture	5 850	100	16	163	23	40	10 803	5.5
Other	2 760	443	769	827	701	84	8 815	4.5
Infrastr.	0	0	0	0	0	0	30 005	15.2
Total	53 552	5 020	22 322	14 216	14 581	1 623	197 732	100.0
Per cent	27.1	2.5	11.3	7.2	7.4	0.8	100.0	

Table A9. **Material corrosion costs distributed by SO_2-source and region, 1994 (1 000 Nkr)**

	Stationary	Mobile sources				Process	Total
		Road and constr.mach.	Rail	Aircraft	Ships		
Halden	1 176	516	13	0	48	-	1 753
Sarpsborg	674	124	1	0	6	4 548	5 352
Fredrikstad	956	103	0	0	14	1 326	2 399
Moss	1 341	34	0	0	7	156	1 538
Bærum	2 113	4 333	2	274	371	-	7 092
Asker	827	1 675	15	0	38	-	2 555
Oslo	27 603	16 410	158	10	30 301	-	74 481
Drammen	1 256	1 905	12	0	664	-	3 837
Porsgrunn	414	93	0	0	41	1 509	2 057
Skien	2 042	1 458	7	16	103	9 501	13 128
Bamble	96	268	-	-	108	-	473
Kristiansand	99	72	0	2	60	1 851	2 084
Stavanger	1 815	1 674	9	52	4 925	-	8 475
Bergen	10 194	9 380	37	199	2 498	-	22 307
Trondheim	3 799	652	46	0	915	3 710	9 123
Tromsø	463	227	-	6	86	-	782
Urban-south	7 624	4 485	67	13	406	21 424	34 020
Urban-north	559	327	20	5	14	2 539	3 463
Rest of the country	-	-	-	-	-	-	-
Total	63 051	43 735	386	577	40 606	46 565	194 920

Appendix B

Appendix to chapter 5: Overview of dose-response functions for health effects

Table B1 and table B2 below contain an overview of the dose-response functions taken from international literature and applied in this study. The functions build on epidemiological studies in various parts of the world. Uncertainty intervals are stated where they exist. They are based either on comparison of several studies (e.g. for mortality), or on standard deviations in the study/studies undertaken. Hence they are not uncertainty intervals in the strict sense, and the uncertainty is compounded in the applications in Norway. Construction of such uncertainty intervals for Norway is not possible at present.

Table B1. **Dose-response functions for particulates**

Change in health effect	Coefficient estimates (per change in mean annual conc. of PM_{10} ($\mu g/m^3$))	Source (source of uncertainty intervals in parentheses)
Acute effects:		
P1. Premature mortality per 1 000 deaths	0.96 [0.63 ; 1.30]	Ostro (1993) - consensus estimate
P2. Restricted activity days per person per year	0.058 [0.036 ; 0.090]	Ostro (1987) (ORNL/FF (1994))
P3. No. of days with respiratory symptoms per person per year	0.18 [0.09 ; 0.27]	Krupnick et al. (1990) (Ostro (1994))
P4. Annual respiratory hospital admissions per 100 000 persons	3.6 [1.2 ; 10.2]	Based on various studies (Pope (1991); Plagiannakos and Parker (1988), Sunyer et al. (1991))
Chronic effects:		
P5. Mortality risk (associated with dying at a particular age)	0.65 per cent [0.46 ; 0.91]	WHO (1995) - combination of Pope et al. (1995) and Dockery et al. (1993)
P6. Risk of chronic obstructive pulmonary disease (COPD)	1.1 per cent [0.5 ; 1.7]	Abbey et al. (1993) (Rowe et al. (1995))

Table B2. **Dose-response functions for NO_2**

Change in health effect	Coefficient estimates (per change in mean annual conc. of NO_2 ($\mu g/m^3$))	Source (source of uncertainty intervals in parentheses)
Acute effects:		
N1. Risk of hospital admissions because of asthma	1.5 per cent	Pönkä (1991)
N2. Risk of hospital admissions due to croup	0.4 per cent	Schwartz et al. (1991)
N3. No. of days with respiratory symptoms per person per year	0.009 [0.0013 ; 0.005]	Schwartz and Zeger (1990) (ORNL/RFF (1994))

Table B3 and table B4 give an overview of functions we have arrived at on the basis of the dose-response functions in and, respectively. They are primarily functions associated with economic activity. The coefficient estimates apply to Norway in general, with estimates for Oslo in parentheses.

Table B3. **Functions for change in economic activity etc., associated with changed particulate concentration**

Change in activity etc.	Coefficient estimates (per change in mean annual conc. of PM_{10} ($\mu g/m^3$))	Source (refers to dose-response functions in table B1)
Acute effects:		
Changed supply of labour (short-term sickness absence and reduced productivity)	-0.011 per cent (of total supply of labour)	P2
Changed supply of labour (reduced productivity)	-0.0021 per cent (of total supply of labour)	P3
No. of bed-days in hospital per 100 000 population per year	21 (32)	P4
Public hospital expenditure per capita per year	Nkr 0.76 (Nkr 1.12)	P4
Chronic effects:		
Life expectancy (women)	-0.059	P5
Life expectancy (men)	-0.064	P5
No. of bed-days in hospital per 100 000 population per year	31 (42)	P6
Public hospital expenditure per capita per year	Nkr 1.1 (Nkr 1.5)	P6
Changed supply of labour; person-hours (long-term sickness absence)	$-6.9*10^2*\alpha$	P6
Changed supply of labour; person-hours (rehabilitation)	$-3.8*10^3*\alpha$	P6
Changed supply of labour; person-hours (disabled)	$-6.9*10^4*\alpha$	P6

α = share of Norwegian population that is exposed (α=0.11 for Oslo).

Table B4. **Functions for change in economic activity etc., associated with changed NO_2 concentration**

Change in activity etc.	Coefficient estimates (per change in mean annual conc. of NO_2 ($\mu g/m^3$))	Source (refers to dose-response functions in table B2)
Acute effects:		
Changed supply of labour (reduced productivity)	-0.0015 per cent (of total supply of labour)	N3
No. of bed-days in hospital per 100 000 population per year (asthma)	13 (14)	N1
Public hospital expenditure per capita per year (asthma)	Nkr 0.47 (Nkr 0.48)	N1
No. of bed-days in hospital per100 000 population per year (croup)	0.5	N2
Public hospital expenditure per capita per year (croup)	Nkr 0.02	N2

Appendix C

Appendix to chapter 6: The relation between traffic volume, traffic density and traffic injuries

Equation (6.7) in chapter 6 describes the impact of traffic volume and traffic density on the number of person injuries in road traffic, S_t. The parameters in this equation are based on information provided by Fridstrøm and Bjørnskau (1989). Below we show how parameters for our model are developed from their estimates.

Fridstrøm and Bjørnskau (1989) estimated the impact of different variables on the number of person injuries. The estimations were based on microeconomic data from different Norwegian counties and different months. As the relation estimated were on relative form with constant elasticities and there were only minor differences between counties, we use the relation on a national level by scaling up.

Fridstrøm and Bjørnskau estimated the following equation

(C1) $$ln\ S_t = k_t + \alpha\ ln\, B_t + \sigma\ ln\, BUSKM_t + \beta\,(ln\, CON_t - ln\, CON_0)$$

where

S_t = number of persons injured
B_t = consumption of gasoline - applied as proxy for total driving distance in kilometres by gasoline and automotive diesel fuelled vehicles (except buses)
$BUSKM_t$ = driving distance (kilometres) for buses
CON_t = traffic density (congestion) indicator.
k_t = vector of other variables (climatic and demographic variables, consumption of alcoholic and a scale constant).

α, σ and β are the parameters estimated by Fridstrøm and Bjørnskau (1989). The estimated values are:

$\alpha = 0.812$
$\sigma = 0.136$
$\beta = 0.284$

The indicator CON_t is expressed by

(C2) $$CON_t = \frac{B_t}{ROADS_t}$$

where

$ROADS_t$ = total kilometres of national and county roads.

The road length seems to increase over time, but at a decreasing rate. Thus we have fitted the following curve to historical data on road lengths and the expected increase in road length 1994-1997 according to the Norwegian Road Plan:

(C3) $ROADS_t = 58495 + 8769.4 \ ln(t - t_{1965})$

The relation (C1) can be written in the form

(C4) $$S_t = K_t \cdot B_t^{\alpha} \cdot BUSKM_t^{\sigma} \cdot \left(\frac{CON_t}{CON_0}\right)^{\beta}$$

where $ln\ K_t = k_t$

As gasoline consumption is used as proxy for the traffic activity in Fridstrøm and Bjørnskau's study, α is interpreted as the elasticity of number of person injuries with respect to total road traffic activity except bus traffic. We therefore assume that no other variables than gasoline describes the effect of traffic activity by diesel vehicles (other than buses). Thus, we rewrite relation (C4) as follows

(C5) $$S_t = K \cdot e^{\varepsilon \cdot t} \cdot \left(KM_t^B + KM_t^{DEB}\right)^{\alpha} \cdot BUSKM_t^{\sigma} \cdot \left(\frac{CON_t}{CON_0}\right)^{\beta}$$

where (c2) is changed to

(C6) $$CON_t = \frac{\left(KM_t^B + KM_t^D\right)}{ROADS_t}$$

KM^B_t = kilometres driven by gasoline vehicles
KM^{DEB}_t = kilometres driven by diesel vehicles except buses
KM^D_t = kilometres driven by all diesel vehicles

We have also split K_t from relation (C4) into $K \cdot e^{\varepsilon t}$, which represents the impact of climate and demography variables and consumption of alcoholic, as well as a scale constant. K is kept at its base year level during the simulations. ε is a parameter representing trends in the constant variables, preliminary set equal to zero in our simulations.

We want to relate the number of injuries to use of gasoline and diesel, variables which are determined in the CGE model (MSG-EE). We do this by explicitly taking into account the technological change affecting average mileage per fuel use over time.

Assuming annual rates of energy saving of θ and ω for gasoline and diesel, respectively, and a constant relation between KM_t^{DEB} and $BUSKM_t$ given by $\gamma/(1-\gamma)$, the relations between vehicle kilometres and fuel use can be written as:

(C7) $KM_t^B = M_0^B B_t e^{\theta t}$

(C8) $KM_t^{DEB} = \gamma \cdot KM_t^D = \gamma \cdot M_0^D D_t e^{\omega t}$

(C9) $BUSKM_t = (1-\gamma) \cdot KM_t^D = (1-\gamma) \cdot M_0^D D_t e^{\omega t}$

θ = rate of technological change, i.e. increase in fuel efficiency, for gasoline cars
ω = rate of technological change, i.e. increase in fuel efficiency, for diesel vehicles
M_0^B = kilometres per litre gasoline in the base year
M_0^D = kilometres per litre diesel in the base year
D_t = total diesel consumption

Thus equations (C5) - (C9) give the relations between total use of diesel and gasoline and the number of persons injured.

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