Health Costs due to Road Traffic-related Air Pollution

An impact assessment project of Austria, France and Switzerland

Prepared for the Third Ministerial Conference for Environment and Health
London 1999

Air Pollution Attributable Cases

Technical Report on Epidemiology

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Table of Content

1. Executive Summary of the Epidemiology Report

1.1. Preamble

In preparation of the Third WHO Ministerial Conference of Environment & Health, , to be held in London in June 1999, a trilateral project was carried out by Austria, France and Switzerland. This project assessed the health costs of traffic related air pollution in the three countries using a common methodological framework.

From the French side, this tri-lateral research has been selected as part of the French co-ordinated research program on transport (PREDIT [1]) by both steering groups of PREDIT in charge of research co-ordination in the field of health effects of transport related pollution and externalities' monetarisation. This underlines the commitment of the PREDIT towards international co-operation.

1.2. Context of the Project

In addition to its positive impact on the growth and prosperity of the national economy and its importance for satisfying our individual needs for mobility, road transport also has adverse effects: accidents, noise, air pollution, harm to health, crop damage, traffic jams, etc.

In the last 10 to 20 years an increasing awareness may be observed for these negative effects of transport. Congestion, air pollution and noise affect more and more people. Their impact on health and welfare, the damage to buildings and the natural environment are considerable, just like the material and intangible costs caused by them.

These costs are mainly external costs which means that they are not covered by the polluters (the motorists) but that they are imposed on everybody. External costs cause a problem to the economy, as they are not included in the market price which leads to wrong decisions and to a wasting of scarce and vital resources (clean air, silence, clean water, etc.). Motorists behave as if those costs do not exist, since they have not to pay for them. By including the external costs, such trips may have produced higher total costs than the total benefit. As a consequence, many trips would have been avoided if all the external costs had to be considered by the driver.

In order to stop the wasting of scarce resources, the government has to take action and put a price on clean air and other environmental "products". As a result, negative impacts of road transport have to be paid for by the polluter. The usual terminology for this process is "internalisation of externalities".

A condition for such an environmental and transport policy is a knowledge about the negative impacts of road traffic and their monetary quantification.

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[1] The PREDIT is a joint research program developed and supported by following institutions:

Ministry of Transport, Ministry of Research, Ministry of Industry, Ministry of Environment, ADEME French Agency for Environment and Energy Management, ANVAR French Agency for Research Valorisation.

With the present study, an important part of the external traffic-related costs, namely the negative impacts of road traffic-related air pollution on human health, is evaluated and quantified in monetary terms.

1.3. Objective

In order to **quantify the road traffic-related health costs due to air pollution**, Austria, France and Switzerland have co-operated in a tri-lateral research project.

One objective was the choice of a common methodological framework and the evaluation of results that are comparable for the three countries. Of course, within the common methodological framework, some specific features of each country (data availability, health system, etc.) had to be considered.

The results of this co-operation provided an input for the WHO Ministerial Conference in June 1999[2]

The research project was based on an interdisciplinary co-operation in the fields of air pollution, epidemiology and economy. The tasks of the three domains may be summarised as follows:

1. **Air pollution: Evaluation of the (road traffic-related) exposure**
   For the three countries Austria, France and Switzerland, the exposure of the residential population has to be assessed. The result has to present a detailed register describing the number of persons living in each category of air pollution concentration. It must be considered that the emissions' source is not only transport but other sources as well, such as industry and households.

2. **Epidemiology: Evaluation of the exposure-response relationship between air pollution and health impacts**
   The relationship between air pollution and health had to be assessed. This step provides for each level of exposure the number of air pollution attributable cases of morbidity and mortality. This evaluation had to be based on the current epidemiologic evidence.

3. **Economy: Evaluation of the road traffic-related health impacts and their monetarisation**
   By combining the dose-response relationship with the exposure to PM10 in each country, the impacts of traffic-related air pollution on human health is quantified (number and type of additional cases of morbidity, number of additional cases of premature death). With adequate methods, these health effects finally have to be valued in monetary terms.

[2] Third WHO Ministerial Conference of Environment & Health, London, 16-18 June 1999.

The present project was building on the previous research in Switzerland.[3] In the framework of this trilateral co-operation, several methodological questions were further discussed within the international and interdisciplinary group and have partly resulted in new approaches. Furthermore, the most recent scientific results have been adopted and in addition, several methodological calculation steps have been modified in order to make the common methodological framework applicable for the evaluation of health costs in other countries also.

1.4. Working Process

Working on the basis of a common methodological approach, the input data, the methodological choice for the single working steps and the findings were discussed, adjusted and adopted by the entire tri-lateral team (see annex 1).

The single working steps of each scientific domain (air pollution, epidemiology, economy) were prepared by three separate technical sub-groups consisting of the three countries' respective experts. For each scientific domain, the methodological procedure and findings are presented in separate technical reports[4].

In all three domains, co-referees from the international scientific community were invited in order to critically advise and comment on the ongoing work (see annex 1).

1.5. Content of the Technical Report on Epidemiology

Aims

The aims of the Epidemiology Report were 1) to calculate the air pollution attributable number of cases of adverse health effects, and 2) to contribute to the development of a common science based methodological framework of international health impact assessment.

Methods

The trilateral project relied on experiences of a prior Swiss study, but further discussed and developed methodological issues. Modifications were undertaken to allow an international application.

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[3] ECOPLAN (1996), Monetarization of the external health costs attributalbe to transport; Künzli N. et al., Teilbericht Epidemiologie: Synthesebericht Monetarisierung der verkehrsbedingten Gesundheitskosten; Künzli N. et al (1997), Luftverschmutzung in der Schweiz - Quantifizierung gesundheitlicher Effekte unter Verwendung epidemiologischer Daten.

[4] **Filliger P**., Puybonnieux.Texier V., Schneider J., et al., PM10 Population Exposure.; **Künzli N**., Medina S., Studnicka M., Oberfeld G., Horak F., Air Pollution Attributable Cases.; **Sommer H**., Chanel O., Vergnaud J.-Ch., Herry M., Sedlak N., Seethaler R., Monetary Valuation of Road Traffic related Air Pollution.

Effect estimates from epidemiologic studies are a key component for the assessment of air pollution impacts on health. If applicable, it was decided to consider both short- and long-term effects for the assessment. The selection of methodological assumptions was guided by a principle of 'at least', presumably resulting in an impact which is 'at least' attributable to air pollution. As one single indicator of urban air pollution, the assessment was limited to particulate matter equivalent or less than 10 μm in diameter (PM10). A 'lowest assessed level' of 7.5 $\mu g/m^3$ was applied to take into account that currently available epidemiologic studies have not included populations exposed to levels below 5-10 $\mu g/m^3$ (mean 7.5 $\mu g/m^3$). For the same reasons, the impact assessment project provides estimates for the total impact of air pollution above the 'lowest assessed level' only. As there are no studies giving direct estimates for the health impact of ***traffic related*** PM10, the respective fraction has been estimated based on the data regarding the traffic-related proportion of PM10 (see Air Pollution Report).

The following health outcomes were selected: total mortality based on cohort studies (long-term), respiratory hospital admissions, cardiovascular hospital admissions, incidence of chronic bronchitis in adults, bronchitis in the last 12 months in children, restricted activity days in adults, asthma attacks in children, and asthma attacks in adults. Other health effects have not been included in the assessment as they may not easily be expressed in monetary values (e.g., lung function decrement, school absentees, physical performance, change in bronchial reactivity), or may be partially included in the above mentioned outcome measures, thus leading to partial double counting of the impact (e.g., premature death due to acute exposure, emergency room visits, respiratory symptoms in adults etc.). Infant and intrauterine mortality have not been included as by the time of this project only one study was available for each of these important outcomes.

The derivation of air pollution attributable cases was based on the attributable risk concept. For the selected health endpoints, epidemiologic exposure-response curves were derived from the available literature, using a meta-analytic approach to calculate variance weighted mean relative risks (RR). When significant heterogeneity was present, random effect estimates were calculated. The 95% percent confidence intervals of the exposure-response functions indicated the epidemiology-based range of uncertainty.

National epidemiologic baseline data of incidence/prevalence were derived from available population based data. The number of cases and/or person days of outcome attributable to a 10 $\mu g/m^3$ increment in PM10 exposure were calculated (D10, $D10_{low}$, $D10_{upp}$.), applying the exposure-response functions and the 95% confidence intervals to the respective population baseline frequency data.

Results

The joint effect estimates (relative risks) per 10 $\mu g/m^3$ ambient PM10 mean exposure, and the fixed baseline increments per 10 $\mu g/m^3$ PM10 and 1 Million inhabitants (D10, $D10_{low}$, $D10_{upp}$.) are given in Table S1. The national increments are in the same range of magnitude. Differences in the national increments (D10) mostly stem from differences in the age structure and the health risk profile of the countries.

Table S1: Joint effect estimates (relative risks) and fixed baseline increments per 10 μg/m^3 PM_{10} and 1 Mio. inhabitants (D10, $D10_{low}$, $D10_{upp}$.)

Health Outcome	Effect estimate Relative Risk (±95% Confidence Interval (CI))	Fixed baseline increment (D10) per 10 μg/m^3 PM10 and 1 Mio. Inhabitants Cases D_{10} (D_{10low}, - D_{10upp} based on ±95% CI estimates)		
		A	F	CH
Total mortality (adults ≥ 30 years)	1.043 (1.026-1.061)	374 (226-524)	340 (206-476)	337 (204-473)
Respiratory Hospital Admissions (all ages)	1.0131 (1.001-1.025)	228 (24-433)	148 (16-282)	133 (14-253)
Cardiovascular Hospital Admissions (all ages)	1.0125 (1.007-1.019)	449 (234-668)	212 (112-315)	303 (157-450)
Chronic Bronchitis Incidence (≥ 25 years)	1.098 (1.009-1.194)	413 (37-821)	394 (35-784)	431 (38-858)
Bronchitis (children < 15 years)	1.306 (1.135-1.502)	3'196 (1'409-5'774)	4'830 (2'129-8'728)	4'622 (2'037-8'352)
Restricted Activity Days (adults ≥ 20 years) [1)]	1.094 (1.079-1.102)	208'355 (175'399-241'754)	263'696 (221'987-305'966)	280'976 (236'533-326'016)
Asthmatics: Asthma attacks (children < 15 years [2)]	1.044 (1.027-1.062)	2'325 (1'430-3'231)	2'603 (1'600-3'617)	2'404 (1'478-3'341)
Asthmatics: Asthma attacks (adults ≥ 15 years) [2)]	1.039 (1.019-1.059)	6'279 (3'058-9'564)	6'192 (3'016-9'431)	6'366 (3'101-9'697)

1 Restricted activity days: total person-days per year
2 Asthma attacks: total person-days with asthma attacks

Discussion

Uncertainties stem from the exposure assessment in the epidemiologic studies (e.g., data from different regions, fixed site monitors instead of personal exposure), health outcome frequency estimates (e.g., differences in health care systems, lack of standardized health monitoring systems for morbidity), exposure-response estimates (e.g., comparability of international studies), and the impact assessment method. However, given the application of the 'at least' approach, the results are most likely an underestimate of the impact of air pollution. Compared to the previous Swiss and a current French project, the approach.was modified to move toward a more internationally applicable impact assessment method. Differences to other ongoing national projects are explained by the different underlying assumptions and methods used. E.g., other assessors derive the impact for a set of pollutants rather than only one indicator, leading to substantial over-estimations, given that pollutants such as PM10, NO2, or SO2 are strongly correlated and the effects may not be independently considered.Moreover, projects providing only the number of subjects affected by some advancement of death due to the level of air pollution on the days before death (short-term effect) ignore the impact of cumulative exposure on life expectancy.

Recommendations

- Health has to be acknowledged as an integral part of environmental impact assessment. The access to and quality of data used for health impact assessment in Europe has to be improved. The existing European structures and databases should be better coordinated and integrated, to make best use of them for health impact assessment.

- Further standardization is needed for all parts of air pollution health impact assessment: air quality monitoring, exposure assessment, health outcome monitoring, and epidemiologic studies. The development of a common methodological framework will allow to compare the results of different projects, and to better determine inherent uncertainties. *A priori* collaboration of interdisciplinary research groups is strongly recommended as many uncertainties in the assessment stem from the fact that the required background research has not been conducted under the common perspective of impact assessment, yielding to methodological discrepancies which can not simply be resolved *a posteriori*.

- Future exposure assessment for epidemiologic studies should broaden the assessment of indicators of air pollution, including focus on small particle size (PM $\leq$2.5µm), the content and heterogeneity of fine particles, and particle numbers and surface area in association to particle mass. Collaborative research regarding the mechanisms of particulate related health effects is needed. Availability of personal exposure-outcome functions will allow to apply personal exposure distribution data to the impact assessment.

- Further health outcome measures are needed regarding short- and long-term effects of air pollution, with a particular emphasis on chronic morbidity as a consequence of cumulative exposure. The assessment of the mortality displacement in different population subgroups (i.e., the life time lost due to premature death caused by air pollution) will help to better determine the relative importance of short-term effects of air pollution on health compared to the number of years lost, to be derived from cohort studies. Such cohort studies should also be conducted in Europe and will have to directly assess the years of life lost and distribution of age at death. Furthermore, the quality of life years gained has to be addressed. There is need for studies regarding air pollution and both, intrauterine and infant mortality, particularly in countries with low to moderate pollution. If the few currently available studies will be confirmed, these health outcomes will strongly influence the air pollution attributable impact.

- In many countries, ozone may be a very important additional air pollution related health problem. Thus, the ozone exposure distribution will be needed to derive the health impact of oxidant pollution in regions with high primary air pollution, and long and sunny summer periods. Accordingly, standardized ozone monitoring networks are needed and a review of the current epidemiologic literature regarding the exposure-response functions of ozone exposure has to be conducted.

2. Short summary of the Air Pollution Report [5]

The three countries of Austria, France and Switzerland have cooperated in carrying out an impact assessment project on road-traffic related health costs. In this context, the main task of the air pollution part was to estimate the exposure of the population to the ambient concentration of particulate matter. The selected indicator was the annual mean value of PM10. Population exposure was calculated both for total PM10 and for road-traffic related PM10.

A general methodological framework was defined. This involved four main steps: (a) acquisition and analysis of data; (b) PM10 mapping; (c) estimation of the road-traffic related fraction of PM10; and (d) calculation of population exposure. The differences between the countries in the procedures for measuring airborne particles and in the availability of emission data made it impossible to define a single uniform method for calculating population exposure. There was an obvious need for adaptation of the general methodological framework to the individual case. The three countries were obliged to select different procedures depending on the available data.

The modelled PM10 concentration values derived for the three countries are generally in good agreement with measured values. For PM10 mapping, the results show a tendency towards underestimation. This is in line with the selected 'at-least' approach of the overall project.

As a main result, population weighted PM10 averages are summarised in the following table:

Exposure domain	PM10 concentration in $\mu g/m^3$		
	Austria	France	Switzerland
Total PM10	26.0	23.5	21.4
PM10 without fraction attributable to road traffic	18.0	14.6	14.0
PM10 due to road traffic	8.0	8.9	7.4

The interpretation of the table has to take into account that PM10 due to road traffic varies considerably in space. In city centres, the relative contribution of road traffic to total PM10 is higher than in rural areas. Typical values are: 40 - 60 % in cities and < 30 % in rural areas, respectively.

Despite the different used methods, the results of the three countries are relatively similar, especially concerning PM10 levels caused by road traffic. The differences between the countries may be caused by: (a) different background concentration, (b) different sulphate fraction in background PM10, and (c) different size of areas in higher altitude. Anyhow, further investigations are needed to explore in detail the significance of the differences found.

Several difficulties had to be overcome during the work on this study. The most serious was a lack of comparable PM10 data. National PM10 networks must now be established. In building up these networks, it is crucial that the PM10 samplers used are compatible with the new European reference method, thus ensuring the full comparability with other countries. Beside PM10, also other indicators of particulate matter such as PM2.5 and even smaller fractions should be measured.

The establishment of reliable PM10 emission inventories is also crucial. In addition, receptor studies which try to make the source apportionment by using measured PM10 components should be started in different regions of Europe.

[5] *Filliger Paul, Puybonnieux-Texier Valérie, Schneider Jürgen. Health Costs due to Road Traffic-related Air Pollution. An impact assessment project of Austria, France and Switzerland. Prepared for the Third WHO Ministerial Conference of Environment & Health, London, 16-18 June 1999. PM10 Population Exposure Technical Report on Air Pollution. Bern/ Paris, Wien, June 1999*

3. Short summary of the Economy Report [6]

The three countries Austria, France and Switzerland realised together an impact assessment project on road traffic-related health costs.

The **economic approach** combines the exposure-response relationship with the number of people exposed at different levels for the computing of additional air pollution related number of mortality and morbidity. Based on adequate valuation methods, these additional health outcomes are expressed in monetary values.

For the monetary valuation of the air pollution related health effects, **the willingness-to-pay** is used as **main approach**. This method assesses the health costs based on the willingness-to-pay for a decrease in mortality and morbidity related risk. Thereby, the material costs (loss of production/consumption and treatment costs) as well as the intangible costs (pain, suffering, fear of disease and death, grief, etc.) are considered.

An alternative **partial assessment approach** is further conducted with limitation to the material costs only.

Based on the **willingness-to-pay approach**, in 1996 the total air pollution in Austria, France and Switzerland causes a high level of health costs. In **total**, the air pollution related costs of all three countries amount to some **49'400 million EUR**. The **road traffic** is responsible for some **26'400 million EUR**.

In Austria (6'670 million EUR) and Switzerland (4'170 million EUR) the air pollution related costs reach a similar level. Due to the much bigger population, the French costs amount to some 38'600 million EUR. According to the country, 72% to 75% of the health costs are related to mortality.

The **total air pollution related per capita costs** differed across countries, but the estimated range largely overlapped. The highest per capita costs arise in Austria (424 - 1'246 EUR), exceeding the Swiss costs (297 - 892 EUR per capita) of some 40%. In France the annual per capita costs amount to 344 - 1'007 EUR.

For the **road traffic-related health costs** the per capita results differ much less between the three countries: The highest value is obtained in France with about 370 EUR per capita, followed by Austria with about 360 EUR per capita and Switzerland about 310 EUR per capita.

The differences are mainly based on air pollution (average level of PM_{10} exposure weighted by the population, traffic-related share) and the epidemiology (different mortality and morbidity rates in general). On the other hand values stay within the same range. Therefore, the differences mentioned above should not be overinterpreted.

Regarding the **partial assessment approach** the results are essentially lower than the willingness-to-pay approach: especially in France they are 9 times lower, in Austria about 6 times, and in Switzerland about 4 times. The reasons of such differences are are due to the method of assessment: production loss method in Austria and Switzerland, with Switzerland including, in addition, part of the intangible costs, whereas results for France are based on the approach of loss of net consumption with conceptionally different evaluations of mortality risk.

[6] *Sommer H., Chanel O., Vergnaud J.-Ch., Herry M., Sedlak N., Seethaler R. , Health Costs due to Road Traffic-related Air Pollution. An impact assessment project of Austria, France and Switzerland. Prepared for the Third WHO Ministerial Conference of Environment & Health, London, 16-18 June 1999. Monetary Valuation of Road Traffic related Air Pollution. Bern/ Paris, Wien, June 1999*

Main recommendations:

- The magnitude of assessed health costs indicates need for action: Not only make statements about the need of reducing air pollution but define objectives and a concrete time schedule to realise them.
- The polluter pays principle has to be applied to the road traffic.
- Health costs due to air pollution have to be included in cost-benefit analysis of road projects and measures / action against further increase in traffic flow.

Further steps in the economic domain should be developed:

- empirical survey of the willingness-to-pay for reduction in air pollution related mortality and morbidity
- get some better estimates for long-term morbidity
- empirical studies of the age structure of the victims due to air pollution
- evaluation of absence from work related to different health outcomes
- find corresponding measures to be implemented and evaluate the implemented measures periodically as a tool of readjustment.
- investigate the time lag between exposure and health outcome on the one hand and the corresponding decrease in mortality and morbidity due to a exposure reduction, on the other hand

4. Technical Report on Epidemiology: Introduction

4.1. Preamble

In preparation of the Third WHO Ministerial Conference of Environment & Health, , to be held in London in June 1999, a trilateral project was carried out by Austria, France and Switzerland. This project assessed the health costs of traffic related air pollution in the three countries using a common methodological framework.

4.2. Context

In addition to its positive impact on the growth and prosperity of the national economy and its importance for satisfying our individual needs for mobility, road transport also has adverse effects: accidents, noise, air pollution, harm to health, crop failure, etc.

In the last 10 to 20 years an increasing awareness may be observed for these negative effects of transport. Congestion, air pollution and noise affect more and more people. Their impact on health and welfare, the damage to buildings and the natural environment are considerable, just like the material and intangible costs caused by them.

These costs are mainly external costs which means that they are not covered by the polluters (the motorists) but that they are imposed on everybody. External costs cause a problem to the economy, as they are not included in the market price which leads to wrong decisions and to a wasting of scarce and vital resources (clean air, silence, clean water, etc.). Motorists behave as if those costs did not exist because they do not have to pay them. Including the external costs, their trips would have produced higher total costs than the total benefit. As a consequence, many trips would be avoided if the driver had to take all the external costs into consideration.

In order to stop to the wasting of scarce resources, governments have to take action and put a price on clean air and other environmental ”products”. As a result, negative impacts of road transport have to be paid for by the polluter. The usual terminology for this process is ”internalisation of externalities”.

A condition for such an environmental and transport policy is a knowledge about the negative impacts of road traffic and their monetary quantification.

With the present study, an important part of the external traffic-related costs, namely the **negative impacts of road traffic-related air pollution on human health**, is evaluated and quantified in monetary terms.

4.3. Objective

In order to **quantify the road traffic-related health costs due to air pollution**, Austria, France and Switzerland have co-operated in a tri-lateral research project.

One objective is the choice of a common methodological framework and the evaluation of results that are comparable for the three countries. Of course, within the common methodological framework, some specific features of each country (data availability, health system, etc.) must be considered.

The results of this co-operation provide an input for the WHO Ministerial Conference in June 1999[7]

The research project is based on an interdisciplinary co-operation in the fields of air pollution, epidemiology and economy. The tasks of the three domains may be summarised as follows:

1. **Air pollution: Evaluation of the (road traffic-related) exposure**
   For the three countries Austria, France and Switzerland, the exposure of the residential population has to be assessed. The result has to present a detailed register describing the number of persons living in each category of air pollution concentration. It must be considered that the emissions' source is not only transport but other sources as well, such as industry and households.

2. **Epidemiology: Evaluation of the exposure-response relationship between air pollution and health impacts**
   The relationship between air pollution and health has to be assessed. This step provides for each level of exposure the number of air pollution attributable cases of morbidity and mortality. This evaluation has to be based on the current epidemiologic evidence.

3. **Economy: Evaluation of the road traffic-related health impacts and their monetarisation**
   By combining the dose-response relationship with the exposure to PM10 in each country, the impacts of traffic-related air pollution on human health is quantified (number and type of additional cases of morbidity, number of additional cases of premature death). With adequate methods, these health effects finally have to be valued in monetary terms.

The present project is building on the previous research in Switzerland.[8] In the framework of this trilateral co-operation, several methodological questions were further discussed within the international and interdisciplinary group and have partly resulted in new approaches. Furthermore, the most recent scientific results have been adopted and in addition, several methodological calculation steps have been modified in order to make the common methodological framework applicable for the evaluation of health costs in other countries also.

4.4. Working Process

Working on the basis of a common methodological approach, the input data, the methodological choice for the single working steps and the findings were discussed, adjusted and adopted by the entire tri-lateral team (see annex 1).

The single working steps of each scientific domain (air pollution, epidemiology, economy) were prepared by three separate technical sub-groups consisting of the three countries' respective experts. For each scientific domain, the methodological procedure and

[7] Third WHO Ministerial Conference of Environment & Health, London, 16-18 June 1999.

[8] ECOPLAN (1996), Monetarization of the external health costs attributalbe to transport; Künzli N. et al., Teilbericht Epidemiologie: Synthesebericht Monetarisierung der verkehrsbedingten Gesundheitskosten; Künzli N. et al (1997), Luftverschmutzung in der Schweiz - Quantifizierung gesundheitlicher Effekte unter Verwendung epidemiologischer Daten.

findings are presented in separate technical reports[9].

In all three domains, co-referees from the international scientific community were invited in order to critically advise and comment on the ongoing work (see annex 1).

4.5. Tasks of the Epidemiology Report

This report concentrates on the health aspects of the project. The importance of epidemiology, the requirements, and the methodological assumptions to conduct impact assessment are explained. The derivation of the number of air pollution attributable cases stays at the center: The total number of cases (morbidity and mortality) that may be attributed to every increase of 10 $\mu g/m^3$ in the ambient particulate concentration (PM10), taken as indicator of air pollution, will be provided.

Details regarding the estimation of the exposure distribution and the traffic related apportionment of air pollution are provided in the Technical Report on Air Pollution.

The last step consists of a monetary valuation of these air pollution attributed health impacts. For every health measure the specific costs per case are determined. This is the subject of the Economy Report.

4.6. Structure of the Epidemiology Report

The Epidemiology Report is structured as follows:

- Chapter 3 provides background information regarding epidemiology, it's application to the impact assessment, and the general framework.
- Chapter 4 introduces the specific methodological steps to derive the air pollution attributable cases. The selection of health outcome measures is explained. The effect estimates and the current background frequency in each country are provided for each health measure.
- The Discussion section (Chapter 5) highlights limitations and uncertainties of these calculations, including aspects of the exposure assessment in epidemiologic studies as well as issues related to the health outcome measures.
- Chapter 6 formulates open questions and needs for further impact assessments, with particular focus on epidemiology related issues.
- Chapter 7 summarizes the differences between this tri-national project and the previous Swiss and a current French impact assessment. A short list of other similar projects is added without detailed discussion.

[9] Filliger P., Puybonnieux.Texier V., Schneider J., et al., PM10 Population Exposure.
Künzli N., Medina S., Studnicka M., Oberfeld G., Horak F., Air Pollution Attributable Cases.
Sommer H., Chanel O., Vergnaud J.-Ch., Herry M., Sedlak N., Seethaler R., Monetary Valuation of Road Traffic related Air Pollution.

5. The Epidemiology Report: Background and concepts

5.1. Introductary Statement

This report describes the impact assessment of air pollution with regard to human health. The introductory chapters discuss the underlying paradigms and methods applied to the impact assessment project, which had to assess the extent of the *annual impact of traffic related air pollution in Austria, France, and Switzerland*.

We are well aware of the fact that there is a gap of knowledge to abridge research findings with the requirements of this project, which is guided by a policy makers perspective. This problem is inherent to risk assessment, as recently described by Samet (Samet J et al, 1998). Several open questions and uncertainties are attached to the many assumptions and methodological decisions which are required to estimate the air pollution impact on public health. For the following reasons we nevertheless agreed, as scientists, to participate and contribute required data to this project:

1. There is abundant evidence that current levels of air pollution have adverse health effects. From a public health perspective it is therefor an ethical consequence to estimate and communicate the impact to the public. This will allow societies to make decisions which also include and apply research findings, usually established through public funding.
2. Policy makers and societies have to make important decisions all the time. To abstain from impact assessment, given the many uncertainties, would promote decision making based exclusively on individual interests without considering general aspects of public health. This is particularly true for environmentally sensitive decisions. We consider the participation of epidemiologists in this interdisciplinary process as crucial (Samet et al, 1998).
3. In general, scientific uncertainty should not be taken as leading argument to entirely ignore current knowledge.
4. We are well aware that similar impact assessments are conducted by numerous groups. However, no common method has been developed so far, thus the results may grossly vary across different assessments. We would like to contribute and encourage the discussion to develop a common methodology.

It can be anticipated that the public will focus their interest on the Results section of this report. However, from a scientific perspective, the Discussion and the section on Open Questions should be considered a central piece of work. It is also to emphasize that the impact assessment may appear to focus on mortality. This does not reflect our priorities which we would set on morbidity, at least from a public health view in aging developed countries which are faced with an increasing longevity (i.e., a higher proportion of elderly in the population). However, uncertainties and gaps of knowledge are particularly broad to estimate the air pollution impact on morbidity.

The project strongly relied both, on the experience of a prior work conducted in Switzerland (Sommer H et al, 1996; Künzli N et al, 1996; Künzli N et al, 1997) and the important work done by Ostro (Ostro B, 1996; Ostro B and Chestnut L, 1998), who already was a consultant of the previous Swiss project. However, under the lead of the Swiss project partners, the international and interdisciplinary group further discussed and developed methodological issues. Some decisions were adapted in accordance with current knowledge. Modifications were done to develop a method which may be applicable to impact

assessment in other countries.

5.2. Crucial role of Epidemiology

To be able to estimate the public health impact of air pollution, it is essential to quantitatively know the association between the ambient air pollution exposure of populations and the respective health. To assess this relationship, the availability of epidemiologic studies is crucial. Epidemiology is the science of studying associations between potential risk factors, such as air pollution exposure, and health status in populations.

Due to the epidemiologic approach of studying populations, there are ethical and logistic limits to do controlled experiments. Accordingly, epidemiology is considered an observational science, i.e., people are studied in 'real life conditions'. The 'observational' assessment include broad sets of methods such as objective measures of health and exposure and the collection of subjective data (questionnaires). Carefully designed epidemiologic studies try to mimic experiments as well as possible. Experimental sciences can fully control and exclude factors which otherwise may influence the experiment. Epidemiology, however, needs 'to control' factors which may otherwise deviate the true association between exposure and health outcome. There are a variety of methods to control factors in epidemiologic studies, including careful selection of subjects and measuring rather than excluding a variety of confounding factors. Through bio-statistical methods, the impact of cofactors on the association of interest can thus be controlled and adjusted for statistically. Nevertheless, results of epidemiologic (and experimental) studies never can perfectly determine an exposure-response relationship. Therefore, results are reported with both the best estimate and a range of uncertainty around this estimate. Given the inherent limitation in measurement precision, replication of the 'observational experiment' is crucial in the process of scientific proof of epidemiologic exposure-response relationships. If several carefully conducted studies are available, they should be taken in consideration all together to make qualitative and quantitative conclusions about risk factors. In many cases, the average result of all available studies on the same topic rather than from one single study gives the more accurate estimate of the (unknown) true relationship between exposure and health. Therefore, this project considered a large set of epidemiologic studies to derive the associations and the respective range of uncertainty around these meta-analytic estimates.

Epidemiology is a key science to provide data for impact assessment. In fact, public health risks may be assessed and action may be successfully taken without detailed knowledge about the underlying patho-physiologic mechanisms. For example, the strong association of smoking and a variety of health effects has been clearly established with epidemiologic studies long before the detection of the patho-physiologic mechanisms which lead to the health effects (Doll R and Hill AB, 1952). In a famous historic example of applied epidemiology in the last century, John Snow suggested dirty drinking water to be the cause of cholera in London; consequently, closure of a pump successfully prevented cases of cholera years before the detection of the causing bacteria (Snow J, 1855). Similarly to the smoking or cholera example, there is strong epidemiologic evidence that air pollution causes mortality and morbidity in our populations.

Although the underlying patho-physiologic mechanisms are not fully explained yet, the research team of this project, in agreement with many scientists, considers air pollution a cause of health damage. Without this basic assumption, the project has no logic ground.

In order to measure the effects of existing exposure on health consequences which already have occurred in the population of concern, the term impact assessment may be

preferred to risk assessment (Krzyzanowski M, 1997). Based on the strong evidence for a causal relation between air pollution and adverse health effects from recent epidemiological research, the term impact assessment in this report indicates the increasing certainty about health consequences of traffic related air pollution.

5.3. The 'at least' approach in impact assessment

The Air Pollution Impact Assessment project requires data on the exposure-response relationship of several health outcomes, population frequency measures of these outcomes, definition of 'air pollution exposure', population exposure distributions, data about the traffic related fraction of total air pollution and economic valuation of the air pollution attributable cases. Therefore, many assumptions and methodological decisions have to be taken along the calculations. On each level, the mode to deal with the uncertainty had to be defined. It has been decided that the main calculation ought to apply an 'at least' approach, thus consistently selecting methodological assumptions in a way to get an impact which may be expected to be 'at least' attributable to air pollution. Accordingly, the overall impact of air pollution is expected to be greater than the final estimates. To unambiguously communicate the uncertainty in the approach, the final results will be reported as a range of impact rather than an exact point estimate.

5.4. Short-term and long-term effects

Air pollution may have short-term effects on health, i.e., health status of susceptible subjects may change within hours or days after exposure. Furthermore, over the long run, air pollution may repeatedly or continuously compromise health, leading to chronic diseases or limiting defense mechanisms. Given the question to monetarize the annual impact of air pollution on health, the adequate approach would be to assess both, the total short-term effects across one year and the long-term effect over a lifetime. The latter quantity needs to be expressed as the impact per one 'average year', taking into account the duration of the air pollution related chronic health condition. This project assesses parts of both short-term and long-term effects. In case of death (mortality), a once only outcome, the impact of air pollution on long-term mortality rates will be used; this will be discussed in more detail below.

5.5. The definition of 'Air pollution'

Air pollution is a mixture of many known and unknown substances. In air pollution epidemiology, the exposure has to be clearly defined. The usual approach consists in the measurement of at least one specific pollutant, considered to be an indicator of the complex mixture. Important examples are particulate matter (PM, e.g. defined by the upper size of the cut off diameter (μm) such as PM2.5 or PM10) , nitrogen oxides (NOx), carbon monoxide (CO) or ozone (O_3). The closer the indicator of the pollutant correlates with the true health relevant aspect(s) of air pollution, the better the indicator will be associated with the health effect. Furthermore, for some pollutants such as ozone or sulfur oxides, it has been clearly shown to be a direct cause of health effects at concentrations observed in real ambient air. Therefore, the total impact of air pollution on health may be considered the sum of 1) all

independent effects of specific pollutants, 2) the effects of mixtures and 3) the additional effects due to interactions between pollutants. Epidemiologic studies mostly report the associations of some pollutant indicator with health, whereas the independent effects are not published. Furthermore, many air pollutants are highly correlated in the ambient air due to their common sources. As a consequence, impact assessment has to select one or few indicators of air pollution. To simply sum up the pollutant specific impact would be a misconception, leading to overestimation of the overall impact. From a health effects point of view, 'general ambient air pollution' (characterized by pollutants such as PM2.5, PM10, TSP, NO2, SO2 and others) may be distinguished from the additional air quality problem observed in summer only, i.e., oxidant pollution. We decided to estimate the impact for one single indicator of 'urban air pollutant'. The impact of oxidant pollution - likely to cause at least in part additional and independent health effects - will not be quantified (see 'at least' approach).

The impact assessment ought to rely on indicators of air pollution for which epidemiological evidence is strong and for which effect estimates are available. For particulate matter up to 10 μm in diameter (PM10), there exists a broad and sound epidemiological literature to extract effect estimates from. Contrary to larger particles, PM10 is able to enter the pulmonary tract, where it interferes with the defense system of the bronchial tree, and can cause chronic inflammation.

Therefore, PM10 was used as an indicator of air pollution for this study. Although we mostly use studies reporting effects for PM10, some results relate to other measures of particulate matter. According to Dockery and Pope (Dockery DW and Pope CA, 1994), the following conversions were applied for different particulate matter indicators in the respective epidemiologic studies[10]:

Total Suspended Matter (TSP):	PM10 =TSP * 0.55
PM15:	PM10 = PM15
PM13:	PM10 = PM13
PM2.5:	PM10 = PM2.5 / 0.6
Black Smoke:	PM10 = Black Smoke

For both, long-term and short-term effects, the population exposure distribution of the *annual mean* level of ambient PM10 will be used rather than the exposure distribution for each and every day. Therefore, it is assumed that the average PM10 level on any day, throughout the year, corresponds to the annual mean PM10. Thus, the annual impact corresponds to the sum of all daily effects across one year.

The impact assessment requires knowledge about the distribution of the PM10 exposure in the population, to be estimated as the long-term average exposure of population subgroups, i.e., as the distribution of annual mean ambient exposure to PM10 for a recent year. It is to emphasize that for methodological consistency ambient concentration distributions rather than personal exposure distributions are required. As mentioned in the above section, epidemiology based exposure-response functions will be used. These studies, however, report the association of health outcomes with the ambient average level of air pollution rather than the association with personal exposure. So far, the impact of personal exposure on health outcome has rarely been assessed; thus, exposure-response functions based on personal exposure are not available. Although ambient average concentrations are not a perfect estimate of the true individual exposure, the semi-individual

[10] the conversion factor may considerably vary across regions, thus cannot be generalized

approach which assigns ambient rather than personal average concentrations to individuals is very valuable (Künzli N and Tager I, 1997). The discrepancies between personal and semi-individual dose-response estimates depends on the inherent error structure in the assigned exposure. Under the most likely assumptions, the semi-individual estimate may be close to or an underestimate of the average personal dose-response function (Wacholder S et al, 1995).

The project requires estimates of the population exposure distribution. We have agreed upon classifying the population into exposure levels of 5 $\mu g/m^3$ PM10.

5.6. Defining a 'Lowest Assessed Level'

In the calculations of the public health impact of ambient air pollution it is crucial to decide what level of exposure may be considered as the 'reference exposure'. We set the lowest assessed level at 7.5 $\mu g/m^3$ PM10 annual mean. Therefore, health effects of air pollution are only considered from the exposure level of 7.5 $\mu g/m^3$ upwards. The selection of this level strongly influences the calculation of the additional cases due to the 'total PM10' air pollution. As an example, the number of premature deaths would be considerably higher if the lowest assessed level would not be considered.

In contrast, the calculation of the *absolute* number of additional cases attributable to road traffic is only marginally influenced by the selection of the lowest assessed level. The number of the traffic-related cases is derived by subtracting the number of non-traffic-related cases from the total number of cases. Since the lowest assessed level affects the total number of cases and the non-traffic-related cases by the same multiplicative factor, it will not change the difference, i.e., the absolute number of traffic-related cases. The lowest assessed level, however, modifies:

- the *population baseline frequency*, which would be lower without the lowest assessed level due to a higher attributable proportion, resulting also in a lower fixed baseline increment (D10).

- the *proportion* of the traffic-related cases compared to the share that may be calculated using the PM10 concentration partition found in the air pollution report. Therefore, in this study absolute instead of relative numbers are used, whenever possible.

It is to emphasize that the 'lowes assessed level' can not be considered as a level of no effect. So far, epidemiologic studies give no indication for a 'no-effect threshold' for PM10, although some studies like those conducted in Switzerland include regions with rathr low annual mean PM10 levels (Ackermann-Liebrich, 1997; Braun-Fahrländer, 1997). However, studies did not include population living in regions with PM10 levels below 5-10 $\mu g/m^3$. Therefore, we did not extrapolate the risk function down to zero. This is again in line with the 'at least' approach.

5.7. Traffic related impact

Although the project has to assess the impact of *traffic related* air pollution, the epidemiologic project will give estimates for the *total* impact of air pollution above 5-10 μg/m^3 PM10 (average: 7.5 μg/m^3) as there are no studies giving direct estimates for the health impact of traffic related PM10. Depending on estimates of the traffic related PM10, which may vary across regions and countries, the traffic related impact will be derived as a proportion of the total impact. This will be discussed in more details in the Air Pollution Report of this interdisciplinary project (Technical Report on Air Pollution).

6. Methods

6.1. General approach

The general method of the epidemiologic impact assessment of this project is in accordance with the *population attributable risk* concept (Rothman KJ and Greenland S, 1998). Based on the observed frequency of health outcomes (incidence, prevalence) and the observed actual level of ambient particulate pollution (PM10), the expected number of cases will be calculated for an assumed baseline (or 'reference') level of air pollution.

In theory, the *population attributable proportion (PAP)* is the fraction of all cases that would not have occurred if exposure had not occurred.

Equation 1

$$PAP = \frac{[p * (RR - 1)]}{[1 + p * (RR - 1)]}$$

where RR = relative risk for the health outcome, and

p = the proportion exposed in the population.

Because the whole target population is exposed to air pollution (PM10), p equals 1 and the population attributable fraction is equivalent to the *attributable fraction among the exposed (PA_e)* (Last JM and Abramson JH, 1995). For air pollution impact assessment, this attributable proportion (AP) can be calculated with the formula (Krzyzanowski M, 1997)

Equation 2

$$AP = \frac{\sum \{[RR(c) - 1] * p(c)\}}{\sum [RR(c) * p(c)]}$$

where RR (c) = relative risk for the health outcome in category c of exposure, and

p (c) = proportion of the target population in category c of exposure.

The relative risk, RR, is one of the most common measure of effect used to report results in epidemiologic studies. The RR is the ratio of risk 1 devised by risk 0; risk 1 is the risk to experience some health outcome among an exposed population, and risk 0 is the risk for the outcome among unexposed. If the exposure is measured on a continuous scale (e.g., air pollution) rather then as a yes/no condition, the RR are reported as the risk ratio for a defined increment in exposure, e.g. the RR for a 10 μg/m^3 increase in the annual mean

PM10 level. The RR is not an absolute measure for the risk. If exposed and unexposed have the same risk (i.e., the exposure has no health impact), the RR equals one (RR = 1).

The calculation applies the exposure-response function derived from epidemiologic studies. Given the fact that ambient PM10 levels vary across regions, populations are partitioned in exposure groups of 5 $\mu g/m^3$ increments in mean PM10. Therefore, the estimation of cases of a specific outcome attributable to PM10 required detailed decisions on the following aspects:

1. Health outcome selection
2. Derivation of epidemiologic exposure-response curve for each health outcome for a 10 $\mu g/m^3$ increment in the selected indicator of pollution (PM10)
3. Search for national baseline data of incidence/prevalence for each outcome
4. Calculation of number of cases and/or person days of outcome attributable to a 10 $\mu g/m^3$ increment PM10 pollution.

For each study region (country), these equations lead to the respective impact for an increase of 10 $\mu g/m^3$ in the long-term average PM10 pollution. In the last step, the population exposure distribution of traffic related PM10 was applied to calculate the overall impact in the particular population. The following sections will explain each of these aspects.

6.2. Health outcome selection

'Health' is not one entity which can be measured with one parameter. The impact assessment has to sum up air pollution effects on a variety of health outcomes. We considered the following criteria to select health outcomes for impact assessment:

1. The exposure-response relationship between ambient PM10 and health outcome has to be published quantitatively (slopes or relative risks). The mere publication of correlation coefficients is not sufficient for impact assessment. Furthermore cross-sectional or cohort studies relying on only two or three levels of exposure, e.g. comparison of two cities with regard to air pollution and health outcome, were also omitted. Such two point comparisons are limited because confounding can not be adequately addressed, thus exposure-response slopes may not be valid (Künzli N and Tager I, 1997).
2. For health effects which clearly are overlapping entities, only one outcome will be included to avoid multiple counting of the same impact (and therefore costs). One example are emergency room visits and hospital admissions, both shown to be related to air pollution. It is known that emergency room visits, at least in part, may lead to admission.
3. Only health outcomes which allow monetary valuation are included. E.g., the reduction of lung function will be difficult to translate into monetary values.

The available and selected health outcomes are given in Table 1.

Morbidity

For hospital admissions, restricted activity days, bronchitis in children, and asthma attacks, short-term effect estimates were used and applied to the annual observed frequency of outcome in the population.

For the long-term impact of air pollution on chronic bronchitis, the impact assessment used air pollution effects on *incidence* of the disease. This allows the economy group of this project to attach monetary estimates for a new case of chronic bronchitis. We furthermore provided estimates of air pollution on *prevalence* of this disease, which had been the method used in the previous Swiss project. Thus, in the Swiss 1996 impact assessment study, the long-term impact of air pollution on the prevalence (i.e., the pool of existing cases) was measured rather than an annual effect on the number of new cases (incidence). The prevalence approach, for a chronic outcome, may be easier translated into monetary values if we were to know the life-time (diseased life-time) costs of chronic bronchitis and the average duration of the disease. Therefore, using incidence and prevalence measures, we also estimated the average duration of chronic bronchitis.

Short-term effects such as school absentees were excluded because they did not allow monetary valuation. The impact of air pollution on lung function (both, short-term and long-term effects are established) has not been included in the project. It may not be financially expressed, since the health outcome is related to other measures of morbidity and mortality already included in the assessment.

Cancer morbidity has also not been an explicit part of the assessment. The risk function for cancer incidence in association with ambient PM10 has not yet been clearly established. Death due to cancer, however, was indirectly included in the impact assessment of air pollution effects as we estimated the impact on *total* mortality (see below).

Mortality

The impact assessment is based on the long-term effect on mortality rates in adult populations.

Conceptually, the impact of air pollution on mortality may include several processes and the distinction between short-term advancing in death in very frail people and long term shortening of life time related to air pollution is difficult to establish. The impact of air pollution on mortality is a combination of acute as well as cumulative chronic effects and the assessment of each require different epidemiological study designs.

In case of short-term effects, air pollution levels of a given day or short period of days may trigger an increase in death within days or weeks. Current air pollution levels do not lead anymore to an excess mortality comparable to that observed in London in the 1950s but nevertheless bring forward deaths among susceptible people. Most of the literature on the short-term effects of air pollution on mortality is based on time-series studies. Time-series studies such as the APHEA project in Europe (Short-term effects of Air Pollution on Health, a European Approach) (Katsouyanni K et al, 1996.) and others accurately measure the short-term effects of air pollution as time-series studies, by design, assess the temporal association between a rather short exposure period and the number of consecutive deaths. Examining longer term averaging periods in time-series approaches would increase the risk of confounding by inadequately controlling for season and this represents an inherent limitation of time-series studies (Schwartz J, in press 1999). An important question regarding the observed short-term effects of air pollution on mortality relates to the time of advancement of these death, which has to be evaluated (Quénel P et al, in press; Spix C et al, 1993). E.g., if air pollution episodes only ‘harvest’ death among an extremly frail population, this effects may be of little public health relevance. Time-series allow to assess

the extent of harvesting. In some risk groups, e.g. persons with chronic obstructive pulmonary diseases, most of the mortality is probably displaced by only a short time ('harvesting'), for cardiovascular and other causes of deaths the effect size may increase at longer time scales, indicating a greater effect as time goes on (Schwartz J, in press 1999). As shown by Zeger et al., short-term effects of air pollution on mortality are not entirely nor predominantly explained by harvesting (Zeger SL et al, 1999 in press).

In terms of long-term effects of air pollution on mortality, the shortening of life-expectancy may be considered a consequence of cumulative long-term exposure, leading to both short-term and long-term morbidity. Life-time air pollution exposure may lead to recurrent injury and, in the long-term, cause chronic morbidity and, as a consequence, reduce life expectancy. In these cases, occurrence of death, i.e., the event of dying, may or may not be associated with the short-term air pollution exposure pattern. From a study design perspective, cohort studies address the association of exposure with time to death. Accordingly, for the purpose of impact assessment, it was decided not to use daily time-series mortality studies to estimate the excess annual mortality but the change in long-term mortality rates in association with ambient air pollution.

Two US cohort studies published the exposure-response function of ambient particulate pollution and long-term mortality (Dockery D et al, 1993; Pope CA et al, 1995). These cohort studies give the additional number of deaths per person-year which may be directly applied to the per year impact assessment. There is only one other cohort study published so far (Abbey DE et al, 1995)[11]. The project, however, reported the impact of the number of hours above cut off levels of exposure which can not be translated into the exposure measures used in this project. The impact assessment is, therefore, based on the long-term effect on mortality rates in adult populations as reported in the two US studies. These prospective cohort studies report substantially larger effect slopes for long-term exposure than are indicated by daily time-series studies (Pope CA et al, 1995).

From an economical assessment perspective, the ideal measure to estimate the impact would be *years of life lost* due to air pollution. Unfortunately, the two cohort studies did not provide results in this format. Therefore, we propose quantitative steps and assumptions to transform number of attributable cases into years of life lost. This is presented in the Discussion chapter on Health Outcomes.

To be in line with the methods and populations of the two selected US studies, the baseline frequency of deaths per year was restricted to the number of natural deaths occurring among those aged 30 and older. As discussed in more detail (see Discussion on Health Outcomes), we used the effect estimate for total mortality in this age group rather than cause specific mortality. Therefore, air pollution related cases of cancer are an inherent part of the assessment.

It was further decided not to include infant or intrauterine mortality into the estimate of traffic related health costs. This will be discussed in more detail (see Discussion on Health Outcomes).

As summarized in Table 1, the assessment will consider the following health outcome

[11] shortly after the final calculations of this project, the 15-year follow-up report of the same US cohort has beend published, increasing the evidence for an impact of air pollution on longevity (see Ref. Abbey DE et al, 1999).

measures:

- Total Mortality (adults ≥30 years)
- Respiratory Hospital Admissions (all ages)
- Cardiovascular Hospital Admissions (all ages)
- Chronic bronchitis (adults ≥25 years)
- Acute Bronchitis (children <15 years)
- Restricted Activity Days (adults ≥20 years)
- Asthmatics: Asthma attacks (children <15 years, adults ≥15 years)

6.3. Epidemiologic exposure-response function

Epidemiologic studies provide effect estimates as a key information in this impact assessment. These estimates are derived from statistical models which relate specified health outcomes to a measure of exposure. For each outcome, available studies were selected from the international peer-reviewed scientific literature to derive the dose-response function and the respective measure of precision (95^{th} percent confidence interval, standard error). An adequate study design, and published air pollution levels were also required as a quality standard. The overall effect estimate was calculated as the variance weighted average across the results of all studies with available quantitative effect estimates (beta coefficients or relative risks) (Petitti D, 1994). Studies with low standard errors had more weight in the resulting joint estimate. For each health endpoint, the pooled relative risk ($\pm$ 95% confidence interval) per 10 $\mu g/m^3$ PM10 was given. Heterogeneity of the pooled studies was evaluated using the Q-test (Petitti D, 1994). When significant heterogeneity was found (e.g., the probability of Chi^2 was equivalent to or lower than a sensibility limit of $\alpha = 0.20$), random effect estimates were used based on the DerSimonian and Laird method (DerSimonian R and Laird N, 1986). In this model, the variance between the study areas is added to the within-study variance, reducing the difference in weights between the studies, and widening the confidence interval. The joint effect estimate itself may change in both directions depending on the relative risk value of the studies with lower standard errors relative to those with higher standard errors.

To acknowledge uncertainty in the impact assessment, the final results of the assessment will include the range of expected impact, based on the impact estimates derived from the upper and lower 95% confidence interval (also see Discussion section on uncertainties).

Although there are some national studies which published 'national' dose-responses, we used meta-analytic summary estimates rather than national estimates. This is in line with epidemiologic reasoning, giving more weight to the overall results of all adequately conducted studies rather than one single result, in light of the uncertainties and variability inherent to each study. Therefore, the periods of data acquisition varied between the selected studies. This is not of major relevance for the health impact assessment since the derived joint estimates are 'summary measures'. The population baseline frequencies of outcome, in contrast, were obtained from the newest available data. Uncertainties in these estimates have not been further included in the calculation process.

For hospital admissions and asthma attacks, several European studies were available for each indicator. It was thus decided to use the joint estimate from Europe for the calculation of cases and costs, but also to report the non-European and overall joint

estimates.

6.4. Additive versus multiplicative risk function

For many reasons including statistical methods and convenience, most of the air pollution epidemiologic studies report results as *relative risks* rather than *absolute risks* or *risk difference*, i.e., the risk function is by default multiplicative. For small relative risks as usually observed for air pollution related outcomes, the difference of the impact assessment between relative and additive risk functions is very small across the range of observed exposure. E.g., given a population with 20'000 cases of chronic bronchitis at an average baseline level of 10 $\mu g/m^3$ PM10. For a risk difference of 5'000 cases (25% of 20'000) per 10 $\mu g/m^3$ ambient PM10, one would expect a total of 30'000 cases at 30 $\mu g/m^3$ PM10. An the other hand, for a multiplicative risk function of 1.25 per 10 $\mu g/m^3$, the total number of cases at 30 $\mu g/m^3$ would be 20'000 x 1.25 x 1.25 = 31'250. However, if risk functions were to be extrapolated to much higher levels of exposure, the multiplicative model yields an increasingly unrealistic number of attributable cases. To prevent unrealistic results in projects which may apply our method to their own situation - where PM10 levels may be much higher - we will apply an additive risk function, i.e., we will derive the number of additional cases expected for an 10 $\mu g/m^3$ increment in PM10 rather than a percent increment in the health outcome. This is in accordance with the 'at least' approach.

6.5. Baseline frequency at the 'Lowest Assessed Level' of PM10

The results of the calculation of the absolute number of cases per 10 $\mu g/m^3$, i.e., the transition from the published multiplicative function to the additive function, depends on the assumed frequency of the outcome. We first calculate the expected number of cases at the 'Lowest Assessed Level' of PM10, i.e., at 7.5 $\mu g/m^3$ annual mean (see 1.5) . Next, the number of additional cases is calculated, applying the RR (per 10 $\mu g/m^3$ PM10) to this expected 'reference frequency' (equation 3, see below). This number of attributable cases will be smaller than the direct application of the RR function to the observed frequency of cases. E.g., for a RR of 1.043, an annual mortality of 8'000 deaths per 1 Million population aged $\geq$ 30 years, and a PM10 level of 27.5 $\mu g/m^3$, our approach will calculate 7'366 at 7.5 $\mu g/m^3$ PM10 (see example below). Thus, 317 additional (7'366 x 0.043) cases will be attributed to a 10 $\mu g/m^3$ increase in PM10. The direct application of RR to the observed mortality would result in 344 attributable cases per 10 $\mu g/m^3$ (8'000 x 0.043).

6.6. National data of outcome frequency

The outcome frequency (prevalence, incidence) has been derived from national statistics or from published studies or scientific reports conducted in the respective country, if such data were available. To acknowledge national differences in health and health care systems, we consider this a preferable approach rather than using some external estimate of outcome frequency. Although outcome frequencies are subject to uncertainties in the estimates, we will not take into account measures of variability of health outcomes but rather use one point estimate. First, this is in line with the approach chosen by the World Bank and the work of the World Health Organization (WHO) (Ostro B and Chestnut L, 1998). Second,

we generally follow the 'at least approach'; thus we chose assumptions which yield low point estimates.

6.7. The quantification of attributable cases

The quantification procedure consists of 4 steps.

In **Step 1** (see Equation 3), the baseline population frequency was calculated from the observed frequency of the outcome in the population.

The baseline population frequency (P_0) was defined as the proportion of the relevant population that would experience the outcome (e.g., prevalence) assuming a baseline air pollution level B (in accordance with Krzyzanowski M, 1997):

Equation 3

$$Po = \frac{Pe}{1 + \left[(RR - 1)(E - B)/10\right]}$$

where

- P_e = the observed prevalence/incidence from studies/statistics
- P_0 = baseline population frequency
- E = the observed population average exposure level
- B = the baseline exposure level of 7.5 µg/m^3 PM10 (i.e., the average level of the exposure class below the Lowest Assessed Level (5-10 µg/m^3 PM10)).
- RR = the epidemiologically derived combined relative risk for a 10 µg/m^3 increment in PM10. Therefore division by 10 was done.

In **Step 2** (see Equation 4), fixed baseline increments, D_{10}, of the outcome per 1 million population were calculated, assuming a linear additive effect of air pollution above the lowest effect level.

The main values at this stage are the meta-analytic effect estimates and the population baseline frequencies derived from Step 1.

The fixed baseline increment D_{10} was defined as the delta for a 10 µg/m^3 increment in PM10, per 1 million population.

Equation 4

$$D_{10} = 1'000'000 * F_p * P_0 * (RR-1)$$

Where

- D_{10} = the number of additional cases (per million) for a 10µg/m^3 increment of PM10 annual mean concentration.
- F_p = the fraction of the total population which is relevant to the defined outcome (e.g., children only)
- P_0 = the baseline population frequency

- RR = the epidemiologically derived average relative risk for a 10 µg/m^3 increment in PM10.

To estimate a range of impact rather then a point estimate, the upper and lower 95% confidence interval values of the RR were used in Equation 4. Therefore, we derive a lower (D_{10low}) and upper (D_{10upp}) value of the fixed increment D_{10}.

The next two steps require knowledge of the population distribution of exposure. Therefore, they will be part of the integrated interdisciplinary study report. To give the reader of this report a complete overview of all related steps, we shortly include Step 3 (Equation 5) and 4.

In **Step 3** (see Equation 5) we use D_{10} to calculate the number of cases, N_c, attributable to air pollution for a given population group P_c :

Equation 5 $$N_c = D_{10} * P_c / 1'000'000 * [(x_c - B) / 10] * F_a$$

where

- N_c = the number of cases attributable to air pollution for a given population in category c of exposure
- Pc = the population in category c of exposure
- Xc = the average exposure in category c
- B = the baseline exposure level of 7.5 µg/m^3 PM10 (i.e., the average level of the exposure class below the lowest effect value of 10 µg/m^3 PM10)
- F_a is the fraction of air pollution related to traffic.

To reflect the 95% confidence interval of the effect estimates, we also used Equation 5 to derive an upper and lower level of N_c, replacing D_{10} with D_{10upp} or D_{10low}, respectively.

Finally, in **Step 4**, the overall number of cases per year, N, for a health outcome attributable to traffic related air pollution has been derived as the sum of all N_c.

Accordingly, based on the calculation of D_{10low} and D_{10upp}, the overall number of cases attributable to traffic related air pollution was provided not only as a point estimate (N) but also as upper and lower range of confidence , N_{low} and N_{upp}.

Figure 2 shows an example of the calculation process to derive P_0 (baseline population frequency) and *D10* (the number of additional cases per Million for a 10µg/m^3 increment of PM10 annual mean concentration).

6.8. Effect estimates and population frequencies of each health outcome

For each country and selected health outcome the joint effect estimates, population baseline frequencies of outcome (P_0), and fixed baseline increments (D_{10}, $D_{10low,}$ D_{10upp}) are summarized in Table 2. All data of the assessment process are given in the annex, for each country separately. Table 3 summarizes the underlying definitions of each health outcome as

this may be of relevance for the monetarization process. Details regarding the derivation and definitions of effect estimates and the corresponding population frequency measure are given in the following sections.

6.8.1. Total Mortality (adults ≥30 years)

Effect estimates

A variance weighted effect estimate of 1.043 (95% confidence interval (CI) 1.026-1.061) per 10 μg/m^3 PM10 for total long-term mortality was calculated from the results of two US cohort studies (Pope CA et al, 1995; Dockery DW et al, 1993). Both studies controlled for confounding factors such as social status and smoking. The joint estimate was dominated by the large cohort of the American Cancer Society (ACS) (Pope CA et al, 1995) which investigated the survival of 300'000 persons aged ≥ 30 years in relation to the air pollution exposure in 150 different areas (effect estimate 3.9% per 10 μg/m^3 PM10). The effect estimate of the smaller Harvard 6 Cities Cohort Study (n=8'111, age 25 to 74 years) (Dockery DW et al, 1993) was considerably higher (8.5% per 10 μg/m^3 PM10). The follow up periods were 1982 to 1989 (Pope CA et al, 1995) and 1974 to 1991 (Dockery DW et al, 1993). The particulate matter indicators were PM2.5 (Pope CA et al, 1995) and PM10 (Dockery DW et al, 1993). The effect estimates and the joint estimate are shown in Figure 3.

Population frequencies of outcome

The data for total mortality for adults aged ≥30 years were derived from the national death certificate statistics for 1996 (Austria and France) or 1995 (Switzerland). Deaths related to violence and accidents were excluded.

6.8.2. Respiratory Hospital Admissions (all ages)

Effect estimates

A joint estimate of 1.008 (95%CI 1.004-1.012) per 10 μg/m^3 PM10 was calculated from three European studies (Spix C et al, 1998: from the APHEA project combining the results from London (UK), Amsterdam (NL), Rotterdam (NL) and Paris (FR); Wordley J et al, 1997: Birmingham (UK); Prescott GJ et al, 1998: Edinburgh (UK)). The Q test revealed evidence of heterogeneity in the study results, possibly reflecting variation in admission practices in the different study areas. Therefore, the random effects estimate of 1.013 (95%CI 1.001-1.025) per 10 μg/m^3 PM10 was used to calculate D_{10} (D_{10low}, D_{10upp}). The data collection in the European studies covered different time periods between as early as 1977, when study periods started in part of the APHEA cities, and 1995. Only the APHEA project used hospital data for all respiratory diseases; the other two studies focused on specific outcomes. The particulate matter indicators were black smoke for the APHEA project, and PM10 for the others. The effect estimates and the joint estimate used to calculate D_{10} (D_{10low}, D_{10upp}) are shown in Figure 4.

A joint estimate of 1.017 (95%CI 1.013-1.020) per 10 μg/m^3 PM10 was derived from eight US and Canadian studies (Thurston GD et al, 1994: Toronto, Canada; Schwartz J, 1994: Detroit (MI), USA ; Schwartz J, 1994: Birmingham (AL), USA ; Schwartz J, 1994: Minneapolis-St.Paul (MN), USA; Schwartz J et al, 1995: New Haven (CN) and Tacoma (WA), USA; Schwartz J, 1996: Spokane (WA), USA; Schwartz J et al, 1996: Cleveland (OH), USA; Burnett RT et al, 1997: Toronto, Canada). Contrary to the European estimate, heterogeneity

was not present. The studies covered time periods between 1986 and 1994. The US studies used hospital data for all respiratory diseases; the Canadian studies focused on specific outcomes. The populations studied by Schwartz in the US were restricted to persons aged 65 and more years. PM10 was used in all studies.

For all European, US and Canadian studies, a fixed effects estimate of 1.013 (95%CI 1.010-1.015) per 10 $\mu g/m^3$ PM10, and a random effects estimate of 1.016 (95%CI 1.013-1.020) per 10 $\mu g/m^3$ PM10 was calculated.

Population frequencies of outcome

The data for acute respiratory hospital admissions (ICD9 460-519) were derived from national hospital statistics for 1996.

In Austria admission rates in 1996 were available for all hospitals and the complete observational period (BMAGS, 1996).

In Switzerland and France, the admission rates had to be extrapolated. In France, data were only available for the public hospitals, which represented 59.2% of all hospital admissions for respiratory patients (PMSI, 1998). In Switzerland, only 35% of the hospitals had been covered in 1996 (H+, 1997).

6.8.3. Cardiovascular Hospital Admissions (all ages)

Effect estimates

Four effect estimates from Europe (Medina S et al, 1997: Paris (FR); Poloniecki JD et al, 1997: London (UK); Wordley J et al, 1997: Birmingham (UK); Prescott GJ et al, 1998: Edinburgh (UK)) were selected to calculate a joint estimate of 1.013 (95%CI 1.007-1.019) per 10 $\mu g/m^3$ PM10 used for D_{10} (D_{10low}, D_{10upp}). The data collection in the European studies covered different time periods between 1987 and 1995. They focused on specific cardiovascular outcomes, but also looked at cerebro-vascular outcomes (ICD9 430-436; Wordley J et al, 1997), or gave an overall estimate for all circulatory diseases (ICD9 390-459; Poloniecki JD et al, 1997). All age groups were included. The particulate matter indicators were black smoke for the studies conducted by Poloniecki in London and Medina in Paris, and PM10 for the two others. The effect estimates and the joint estimate used to calculate D_{10} (D_{10low}, D_{10upp}) are shown in Figure 5.

Three studies from the north American continent (Schwartz J and Morris R, 1995: Detroit (MI), USA; Schwartz J, 1997: Tucson (AZ), USA; Burnett RT et al, 1997: Toronto, Canada) were selected. The joint effect estimate of 1.008 (95%CI 1.004-1.011) per 10 $\mu g/m^3$ PM10 was lower than that for Europe. The studies from the north American continent covered different periods of time between 1986 and 1994. They used hospital data for all (Schwartz J, 1997) or part (Schwartz J and Morris R, 1995, Burnett RT et al, 1997) of the cardiovascular diseases (ICD9 390-429). The populations studied by Schwartz in the US were restricted to persons aged 65 and more years. All studies used PM10 as particulate matter indicator.

For all European, US and Canadian studies, a fixed effects estimate of 1.009 (95%CI 1.006-1.013) per 10 $\mu g/m^3$ PM10.

Population frequencies of outcome

The data for acute cardiovascular hospital admissions were derived from national

hospital statistics for 1996. Because the biological mechanism of the effect of air pollution on cardiovascular diseases remains poorly understood, all circulatory diseases were used that were included in any of the selected epidemiologic studies (ICD9 390-459).

In Austria, admission rates in 1996 were available for all hospitals and the complete observational period (BMAGS, 1996).

In Switzerland and France, the admission rates had to be extrapolated. In France, data were only available for the public hospitals, which represented 64.0% of all hospital admissions for cardiovascular patients (PMSI, 1998). In Switzerland, only 35% of the hospitals had been covered in 1996 (H+, 1997). Assuming to be representative, the available data were generalized to the total population.

6.8.4. Chronic bronchitis (adults ≥25 years)

Effect estimates

On request of the economy group of this project, and in accordance with the approach used by Ostro and Chestnut (Ostro B and Chestnut L, 1998) we used the effect estimate of particulate pollution on the new occurrence of cases of chronic bronchitis (incidence). There is only one cohort study reporting the respective data (Abbey DE et al, 1993). Within the framework of the Adventist Health and Smog Study (ASHMOG), a cohort of 3'310 seventh-day Adventist (aged 25 years and more) was interviewed with the National Heart and Lung Institute (NHLI, now NHLBI) respiratory symptoms questionnaire in 1977 and 1987. One of the major objectives was to investigate associations between long-term cumulative ambient concentrations of total suspended particulates (TSP), SO2, and Ozone, and the incidence of airway obstructive disease, chronic bronchitis, and asthma. Chronic bronchitis was defined as having symptoms of cough and/or sputum production on most days, for at least three months per year, and for 2 years or more. Multivariate logistic regression models were used for the analysis, controlling for personal risk factors such as past or passive smoking, and occupational exposure. Current smoking was not an issue, because the use of tobacco products is proscribed by the church. After 10 years, 234 new cases of chronic bronchitis were found. Abbey reported a relative risk of 1.36 for the incidence of chronic bronchitis for a 60 $\mu g/m^3$ increment in the 10-year average exposure of TSP. Ostro (WHO, 1996) has used these results in the methodology paper for estimating air pollution health effects. The standard error was calculated from the beta and the additional information that the t-statistic of the beta was 2.16 (Ostro B, 1998). Thus, we derived a relative risk of 1.098 (95%CI 1.009-1.194) per 10 $\mu g/m^3$ PM10 for the annual incidence of chronic bronchitis.

The disadvantage to rely on one single study may be of some concern. However, there are other studies which indirectly confirm the impact of air pollution on the occurrence of this chronic disease, showing the relationship of ambient pollution with the prevalence of key symptoms of chronic bronchitis (Zemp E et al, in press; Schwartz J, 1993). The previous Swiss project was based on these prevalence studies. The effect estimate was 1.237 (95%CI 1.107-1.384) per 10 $\mu g/m^3$ PM10 for chronic bronchitis. Effect estimates were specifically analyzed for the never-smoking population aged 18 to 60, and 30 years and more, respectively. Chronic bronchitis prevalence in the Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) (Zemp E et al, in press) was defined as at least one affirmative answer to the questions for cough (1) and phlegm (2):

'Do you usually cough during the day, or at night, on most days for as much as 3 months each year ?, and an answer of ≥ 2 to the question 'For how many years do you cough like

this ?'

'Do you usually bring up any phlegm from your chest during the day, or at night, on most days for as much as 3 months each year ?, and an answer of ≥2 to the question 'For how many years do you bring up phlegm like this ?'

This corresponds to the definition used by Abbey (Abbey DE et al, 1993) for chronic bronchitis incidence. Schwartz (Schwartz J, 1993) defined chronic bronchitis as an affirmative answer to both parts of the question "Has a doctor ever told you had chronic bronchitis?" and "Do you still have the condition?", from the first National Health and Nutrition Examination Survey (NHANES I) in 1971 to 1975. The SAPALDIA data were collected in 1992/1993. The particulate matter indicators were TSP (Schwartz J, 1993) and PM10 (Zemp E et al, in press), respectively.

Given the similar definitions in both, the ASHMOG study and the prevalence studies, impact assessment may, from an epidemiology perspective, rely either on incidence or prevalence data. Incidence and prevalence are related measures which are linked to each other by the duration of the disease. Under the assumption of a stationary population, i.e., no one enters or leaves the prevalence pool except by disease onset, death, or recovery (which is unlikely after two or more years with chronic bronchitis symptoms), prevalence, incidence and mean duration are linked by the equation (Rothman KJ and Greenland S, 1998):

$$\frac{P}{(N - P)} = I * D$$

where

- P is the size of the prevalence pool (prevalence rate or prevalence proportion, it has no unit, and ranges from 0 to 1)
- N is the size of the population that 'feeds' the prevalence pool
- and P / (N-P) is the ratio of diseased to non-diseased people in the population, also called the prevalence odds; it is the odds of having the disease relative to not having the disease
- I is the incidence rate (dimension is reciprocal of time)
- D is the mean time until death (or recovery) (dimension is time)

Population frequencies of outcome

Incidence data require cohort studies which are, however, not available in the participating countries. It is reasonable, however, to directly use the incidence estimate from the cohort which was followed up from 1977 to 1987 in the ASHMOG study (Abbey DE et al, 1993). The obtained estimate of 0.71% annual incidence will be conservative as the Seventh Day Adventists Study participants are non-smokers. The impact D_{10} (D_{10low}, D_{10upp}), however, was applied to the total (smoking and non-smoking) population ≥25 years as there is no evidence that smokers are not, in addition, affected by air pollution (Ackermann-Liebrich U et al, 1997).

One baseline prevalence of 7.0% for the population ≥20 years was available for the three countries from the Swiss non-smoking population (Zemp E et al, in press). The data were collected in the cross-sectional part of SAPALDIA in 1991. Taking the prevalence from the non-smoking population is consistent with the underlying population of the incidence, and with the 'at least' approach, because a major part of the chronic bronchitis prevalence in

smokers may be exclusively related to tobacco consumption.

6.8.5. Acute Bronchitis (children <15 years)

Effect estimates

Three studies (Dockery DW et al, 1989; Dockery DW et al, 1996; Braun-Fahrländer C et al 1997) were available to calculate a joint effect estimate of 1.306 (95%CI 1.135-1.502) per 10 $\mu g/m^3$ PM10. The time periods of data collection varied from 1980/1981 (Dockery DW et al, 1989) to 1988 to 1991 (Dockery DW et al, 1996), and 1992/1993 (Braun-Fahrländer C et al, 1997). The age ranges were 10-12, 8-12 and 6-15 years, respectively. The definition for bronchitis in the 6-cities study was a parents answer to the question "Did your child have a doctor's diagnosed bronchitis within the last 12 months?". In the Harvard 24-cities study parents or a guardian were asked whether the child had bronchitis during the last 12 months. In the SCARPOL study parents were asked, whether their child had had an airway disease within the last 12 months, and if so whether this was bronchitis. The particulate matter indicator in the 6-cities study was PM15, PM10 in the 24-cities study and in the SCARPOL study. The effect estimates and the joint estimate used to calculate D_{10} (D_{10low}, D_{10upp}) are shown in Figure 6.

Population frequencies of outcome

Dockery et al. reported for data collected in 1980-81 (the Harvard 6-Cities study), in 5'422 children aged 10 to 12, a prevalence of last 12 months doctor's diagnosed bronchitis was 6.3%. For the extended Harvard 24-Cities study, the prevalence of last 12 months diagnosed bronchitis ('chest illness diagnosed as bronchitis') in 13'369 children aged 8 to 12 was reported between 3% and 10%.

For eight non-urban communities in Lower Austria (eastern part of Austria) frequency of doctor's diagnosed bronchitis ("Has a doctor in the past diagnosed asthma-like bronchitis or wheezy bronchitis in your child?") in 843 children aged 6 to 8 years and seen in 1994, prevalence was in 7.6% (Studnicka M et al, 1997). For studies in Salzburg (western part of Austria) in 1991, prevalence of doctor's diagnosed bronchitis was 5.9% for children aged 8-10 (n= 579) (Oberfeld G et al, 1996), while in 1995, among 2'852 adolescents aged 12-15, self-reported last 12 months bronchitis prevalence was 14.8% (Oberfeld G et al, 1997). By simply averaging these Austrian estimates, the observed joint prevalence of bronchitis in Austrian children aged 6-15 years was 9.4%. This prevalence was used to calculate D_{10} (D_{10low}, D_{10upp}).

The Swiss Study on Childhood Allergy and Respiratory Symptoms with Respect to Air Pollution, Climate and Pollen (SCARPOL) observed in 1992/93 a 12.2% prevalence of last 12 months bronchitis in 4'470 children, 6 to 15 years of age.

In France, a population frequency for bronchitis in children was not available. Therefore, the Swiss prevalence from SCARPOL was used to calculate D_{10} (D_{10low}, D_{10upp}).

6.8.6. Restricted Activity Days (adults ≥20 years)

Effect estimates

To quantify the economic impact of restricted activity days (RAD), it is essential to know how RAD's are defined in the epidemiologic studies. One common definition for RAD in epidemiology, however, does not exist. More recently, Ostro has used respiratory-related restricted activity days as a general indicator for respiratory morbidity (Ostro B, 1989; Ostro B and Rothschild S, 1989; Ostro B, 1990). Health outcome data were derived from the annual Health Interview Survey in the US, an individual-level survey of 50'000 households conducted by the Census Bureau for the National Center for Health Statistics. Based on a two weeks recall period (including weekends), days of restricted activity were defined as 'any days where a respondent was forced to alter his or her normal activity'. This included days of work loss or bed disability as well as minor restrictions. Respiratory morbidity was defined as common colds or other acute upper respiratory infections, acute bronchitis, influenza, pneumonia, or other acute respiratory conditions (ICD9 codes 460-466, 470-474, 480-486, 510-516, 519, and 783).

Because of the limited availability of gravimetric measurements of particulate matter, only one of the studies was selected to calculate D_{10} (D_{10low}, D_{10upp}). In this study, Ostro (Ostro B, 1990) used data of the 1979 to 1981 Health Interview Survey including 7'348 currently working adults aged 18 to 65 years from 25 metropolitan areas throughout the US. A multivariate regression model was used in the analysis, controlling for individual risk factors including several socio-economic variables, and the existence of a chronic health condition. Smoking was not controlled for, under the assumption that bias was unlikely due to a marginal correlation between smoking status and any of the pollutants. As in most of Ostro's previous studies on RAD, a lag of two weeks was found for the association between exposure to TSP, PM15, PM2.5 and sulfates, and RAD. Using PM15 as the particulate matter indicator, an effect estimate of 1.094 (95%CI 1.079-1.109) per 10 $\mu g/m^3$ PM10 was calculated.

Population frequencies of outcome

In the selected Ostro study (Ostro B, 1990), the observed frequency of respiratory-related restricted activity days per person and year was 3.23. Although the RAD definitions are not identical with 'absence from work', it may be assumed that a major part of the RAD lead to absence from work. Therefore, national data on work absenteeism may be an adequate source to derive the population frequency of RAD.

In Austria, the impact D_{10} (D_{10low}, D_{10upp}) was calculated using 3.38 days per person and year of absence from work due to respiratory disease in 1996 (Statistics Report of the Austrian Social Insurance, 1997). This information from the Austrian Association of Social Security Agencies (Hauptverband der Sozialversicherungsträger) was based on calendar days off work notified in 2.6 million employees (age range 16 to 65 years) by local general practitioners. Disease categories were upper airway disease and other respiratory disease. Further inclusion of days off work due to bacterial or viral infection would result into 3.47 days per person and year. When considering all 60 disease categories together, 14 days per person and year were reported absent from work.In Switzerland, mean absence from work was 12 to 16 days in 1995 (data from Postal Service and from the Federal Railway Statistics), but no information on the relative importance of respiratory disease was available. Therefore, SAPALDIA data were used to calculate the impact D_{10} (D_{10low}, D_{10upp}). In the diary study of SAPALDIA 1992/93, the average number of restricted activity days due to respiratory symptoms was 4.4 days per person and year, including weekends.

In France, the average number of restricted activity days was not available at the time of the study. Therefore, the Swiss estimate was used.

Although the study by Ostro used quite a different method to obtain restricted activity days due to respiratory disease, the observed population frequency was of similar magnitude.

6.8.7. Asthmatics: Asthma attacks (children <15 years)

Effect estimates

Three European children panel studies (Roemer W et al, 1993: Wageningen (NL); Gielen MH et al, 1997: Amsterdam (NL); Ségala C et al, 1998: Paris (FR)) were selected to derive a joint estimate of 1.044 (95%CI 1.027-1.062) per 10 μg/m^3 PM10 for the calculation of D_{10} (D_{10low}, D_{10upp}). The panels of asthmatics were studied for different periods of time between 1990 and 1995. The French panel consisted of two groups of mild and moderate asthmatics, which directly reported asthma attacks in the diaries. The indicator for asthma attacks in the Dutch panels was lower respiratory symptoms. PM10 was the air pollution indicator for particulate matter in the Dutch studies; Ségala in Paris used PM13. The effect estimates and the joint estimate used to calculate D_{10} (D_{10low}, D_{10upp}) are shown in Figure 7.

The joint estimate from two US studies (Pope CA et al, 1991: Utah Valley (UT); Ostro B et al, 1995: Los Angeles (CA)) was 1.051 (95%CI 1.047-1.056) per 10 μg/m^3 PM10. The studies have been conducted between 1989 and 1992; the indicators were lower respiratory symptoms and shortness of breath for asthma attacks, and PM10 for particulate matter. More recent studies on children panels (Delfino RJ et al, 1996; Delfino RJ et al, 1997; Vedal S et al, 1998) did not show consistent associations of asthma symptoms with PM10 or PM2.5, and effect estimates were not provided.

For all European and US studies, the joint estimate was 1.051 (95%CI 1.047-1.055) per 10 μg/m^3 PM10.

Population frequencies of outcome

Quantitative questions about the number of asthma attacks were used from the International Study of Asthma and Allergies in Childhood (ISAAC) done in Austria (Eder W et al, 1998; Haidinger et al, 1998 a, b) and the Swiss Study on Childhood Allergy and Respiratory Symptoms with Respect to Air Pollution, Climate and Pollen (SCARPOL) (Final Report, unpublished data). The participants were asked, how many asthma attacks they had during the last 12 months. Since the results were similar across the countries, a common population baseline frequency of 0.33 asthma attacks per child was used. The data were collected in 1995/96 (ISAAC) and 1992/93 (SCARPOL). This population average of asthma attacks corresponded to an average of 3 asthma attacks among children with doctors diagnosed asthma. Among subjects with current, more severe asthma in a French asthma panel (Segala C, 1999), a higher average of 3 to 4 asthma attacks per year was reported. This is again in line with the above mentioned surveys, which showed similar number of attacks among those children which reported at least one attack during the last 12 month.

6.8.8. Asthmatics: Asthma attacks (adults ≥15 years)

Effect estimates

Three European adult panel studies (Dusseldorp A et al, 1995: Wijk an Zee (NL); Hiltermann TJN et al, 1998: Leiden (NL); Neukirch F et al, 1998: Paris (FR)) were selected to derive a joint estimate of 1.039 (95%CI 1.019-1.059) per 10 μg/m^3 PM10 for the calculation of D_{10} (D_{10low}, D_{10upp}). The panels of asthmatics were studied for different periods of time between 1992 and 1995. Asthma attacks were defined as wheeze or shortness of breath. PM10 was the indicator for particulate matter in the Dutch studies; Neukirch in Paris used PM13. The effect estimates and the joint estimate used to calculate D_{10} (D_{10low}, D_{10upp}) are shown in Figure 8.

The joint estimate from two US panels on adult asthmatics (Pope CA et al, 1991: Utah Valley (UT); Ostro B et al, 1991: Denver (CO)) was 1.002 (95%CI 0.998-1.006) per 10 μg/m^3 PM10. Ostro studied the effect of PM10 on 'asthma' as the outcome. The study periods were between 1987 and 1990.

Combined for all European and US studies on adult asthma panels, the joint estimate was 1.004 (95%CI 1.000-1.008). Although significant heterogeneity was found, the random effect estimate was unchanged at the chosen level of rounding.

Population frequencies of outcome

As in children, quantitative questions about the number of asthma attacks were used from the European Community Respiratory Health Survey (ECRHS) (unpublished crude data) and the Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA) (unpublished crude data) which indicated very similar distributions in all three countries. The participants were asked, how many asthma attacks they had during the last 12 months. A common population baseline frequency of 0.21 asthma attacks per adult was used. In both studies, the data were collected in 1991/1992. This population average of asthma attacks corresponded with an average of 3 to 4 asthma attacks among persons with doctors diagnosed asthma (questionnaire based) and an average of 9 to 11 attacks among those reporting at least one attack within the last 12 month. A recent panel study among subjects with current, more severe asthma in a French panel (Soussan D, 1999) showed an average of 8 to 9 asthma attacks per year, thus clearly support the validity of the survey based data.

7. Discussion

7.1. General remarks

As already emphasized in the introductory paragraph, these impact estimates can not be taken at face value, as the steps to estimate these numbers include a broad set of uncertainties and assumptions. In contrast to directly countable events which can be listed in national health statistics, e.g., death or injuries due to traffic accidents, it is not possible to directly enumerate the victims of complex substances and mixtures with cumulative toxicity, such as smoking or air pollutants. Neither are the health relevant characteristics of the exposure unanimously defined nor are the health outcomes specific causes of air pollution but rather multi-causal end points. Therefore, uncertainty remains an inherent characteristic of any attempt to derive cases (mortality and morbidity) attributable to such environmental causes.

In contrast to toxicology based risk assessment, where large margins of safety are usually applied to risk functions, we chose an other strategy, based on epidemiology

findings. In order to underestimate rather than overestimate the health impact attributable to air pollution, a general 'at least' approach was chosen for all steps of the project. On each level of the assessment process, the estimate was based on decisions which resulted in an 'at least' impact. As a consequence, the final estimates for attributable number of cases have to be interpreted as the impact which can be expected 'at least'. From a public health perspective, the impact shown in Table 2 ought to be considered relevant.

The purpose of the following discussion is to address major limitations of this assessment. Firstly, we consider issues which relate to exposure within the epidemiologic framework. Secondly, problems of the health outcomes will be addressed. In the third section, uncertainties and limitations which relate to the joint effect estimates and the health impact assessment process will be discussed.

Given that the many limitations are due to issues not yet well addressed by the research community, we add a chapter which formulates open issues, again referring to the three topics of the discussion.

7.2. Exposure assessment

This impact assessment relies on epidemiologic knowledge. Epidemiology is the science that attempts to assess the association of *exposure* with health outcomes in populations under real life conditions, while taking other co-factors into account. Therefore, the impact assessment has to acknowledge issues of exposure assignment inherent to the exposure-response relationships derived from epidemiologic studies. As a further dimension of exposure assessment, the impact assessment relies on the population exposure distribution (based on current air pollution monitoring data) to calculate the attributable number of cases. This Epidemiology Report does not address the strengths and limitations of the methods used to derive the population exposure distributions in Austria, France, and Switzerland. The reader is referred to the respective report (Technical Report on Air Pollution).

a) We only considered ambient air pollution effects which are captured with the particulate matter exposure. Although PM10 may be a very good indicator of air pollution from a variety of sources, particularly in the participating countries, there is clear evidence that other pollutants which poorly correlate with PM10 may be of independent health relevance. One such example is ozone as an indicator of oxidant pollution. Although short-term effects of ambient ozone have been well described and quantified for a variety of outcomes, including mortality, the health impact of oxidant pollution has not been included for two main reasons. Firstly, ozone population exposure distribution would be required on a daily level as only short-term 'acute' effects are clearly established Secondly, due to the fact that high ozone concentrations occur on a rather limited number of days in most of the participating areas, the additional impact of ozone may be only a fraction of the herein assessed impact. However, in regions with both, high primary air pollution and long and sunny summer periods, the additional impact of oxidant pollution may be substantial.

b) A major reason for heterogeneity of the epidemiologic exposure-response estimate between different studies is the divergence of exposure assessment (WHO, in press). In the European project APHEA, the use of total suspended matter or black smoke as exposure indicators was discussed as a possible reason for weaker particle effects on hospital admissions for chronic obstructive pulmonary disease (Anderson HR et al, 1997),

asthma (Sunyer J et al, 1997) and all respiratory diseases (Spix C et al, 1998) in Europe as compared to US study results. Moreover, the effect sizes for short-term mortality in APHEA were compatible with those reported in the United States, but also at the lower end of the range of relative risks (Katsouyanni K et al, 1997). These findings may be explained by differences in the particle mixture. The average relationship between the different methods is not necessarily the same everywhere and would be affected by the local size distribution, the chemical nature of the particles, and perhaps by season (Spix C et al, 1998). There is an increasing interest in the role of the fine (<2.5 µm) and ultrafine (<0.1 µm) fractions, of which a substantial part is composed of chemical particles (e.g., sulfates, nitrates and aerosols). These are inadequately indicated by suspended matter and black smoke (Anderson HR et al, 1997).

c) There is an increasing discussion whether particle numbers or particle surface area correlate better to the broad range of adverse health outcomes than particle mass (Ayres JG, 1998). This hypothesis needs to be tested in future epidemiologic studies.

d) The underlying epidemiologic studies of this project provided estimates of the association of the *ambient* average exposure rather than the *personal* exposure with the health outcomes. Although ambient average concentrations are not a perfect estimate of the true individual exposure, the semi-individual studies which assign ambient rather than personal average concentrations to individuals is a very valuable and efficient design (Künzli N and Tager I, 1997). However, using such data for the impact assessment requires an internally consistent assignment of exposure in the impact assessment process. Accordingly, the herein provided estimates D_{10} (D_{10low}, D_{10upp}) are applied to the distribution of *ambient* rather than personal exposure concentrations in the population. Correspondingly, in the future, the use of personal exposure distribution data, soon available, e.g., for European cities (Jantunen M et al, 1998), need to be applied to the *personal* exposure-outcome function, if once available, rather than the semi-individual study coefficients. It would, however, be inadequate to apply *personal* exposure distribution data to the exposure-response functions derived from epidemiologic studies which relied on *ambient outdoor* air pollution data (i.e., semi-individual studies).

7.3. Health outcomes

The impact assessment required exposure-response functions from epidemiologic studies for several health outcomes. Furthermore, for the selected outcomes, population baseline frequency data were needed. In general, it is to emphasize that epidemiologic research has not been planned so far within an impact assessment framework, therefore, the methodological overlap between these approaches is limited. On both levels, methodological limitations with regard to health outcomes have to be acknowledged:

a) Several health outcomes had to be excluded because they were overlapping entities, or they could not be translated into monetary values (see Table 1). The lack of long-term effect estimates for most outcomes is a major limitation. Thus, the health outcomes included in the project do not include all outcomes for which their is evidence for an association with current or long-term ambient air pollution exposure.

b) Varying definitions and measurement methods of health end points are a major source for heterogeneity. This is of considerable relevance even if the meta-analysis is planned

beforehand and data acquisition is guided by a standardized study protocol, as practiced in APHEA (Katsouyanni K et al, 1996). One problem with data comparability in APHEA was that in some cities the data on hospital admissions did not separate planned and emergency admissions (Spix C et al, 1998). In general, morbidity and health care system data have inherent inconsistencies across countries, regions, and even hospitals. Thus, comparability has some inherent limitations. Spatial and temporal differences in, e.g., diagnostic labeling, awareness in the medical community, treatment rules and data collection may also result in limited comparability of measures like additional asthma attacks or 'restricted activity days'.

c) Health outcome frequencies strongly influence the impact assessment. Whereas for mortality the national sources may be considered accurate (for reliability of death certificates, see below) and not subject to sampling error, frequency measures of morbidity and health care system data have to be considered *estimates* with some inherent uncertainties. First, there are different ways to define these outcomes. European studies using standardized questionnaires can help to assure comparability of data sources. For example, population frequencies for asthma attacks have been taken from the International Study of Asthma and Allergies in Childhood (ISAAC), and both, the European Community Respiratory Health Survey (ECRHS) and SAPALDIA. Secondly, standardized questionnaires are essential to reduce misclassification bias, since epidemiologists often have to rely on the memory of patients, or parental perception and information. Thirdly, the data sources on morbidity and health care utilization data are usually less complete. In France and Switzerland, the hospital admission data had to be extrapolated, since only a part of the hospitals are covered by current national statistics. These uncertainties have not been quantified in the literature.

d) A further level of uncertainty relates to those outcomes where frequency measures had to be generalized from Swiss estimates as some French data (e.g., bronchitis prevalence in children) were not available. However, one may assume that differences across these neighboring countries may be within the range of other inherent uncertainties.

e) ***Total mortality****:* We used total rather than cause specific mortality. One may argue that this decision may inflate the attributed impact as it is well known that air pollution mainly affects the cardiopulmonary system. It is to emphasize that restriction of the impact assessment on cardiopulmonary death would require to use both, the cause specific effect estimates and the respective population frequency. Although the latter would be much smaller than the total number of death, the cause specific effect estimates are larger. Overall, the derived impact may be very similar. This has been empirically tested in an exercise during the previous Swiss project (Künzli N et al, 1996), using short-term impact assessment calculations on mortality for Switzerland. The total impact has been calculated both, for total mortality, using the respective time-series 'total mortality' effect estimate, and separately for the two main causes of death, i.e., cardiovascular and respiratory, applying the respective cause-specific time-series slopes. It has been shown that the cause specific equation yields very similar results with cardiopulmonary cases of death reaching about 90% of the attributable cases of total death (Künzli N et al, 1996). Reliance on cause specific mortality is compromised by the level of reliability of causes of death in death certificates. This may be particularly variable across different age groups, regions, cultures and countries. An investigation by the Statistical Office of the European Community (EUROSTAT) among all European Union countries in 1997 showed general discrepancies in the design of death certificates, nature and amount of information entered, way to establish the diagnosis, degree of consistency of the certification process,

autopsy practices, implementation of ICD10 and implementation of automated coding systems (Jougla E et al, 1998). Therefore, the most consistent approach may be based on *total mortality (except violent death)*, applying the effect estimates reported for *total mortality*.

f) ***Mortality and life expectancy*:** Our Epidemiology Report calculated the attributable number of death. However, from an economist perspective, this information ought to be translated into years of life lost. The available cohort studies did not directly report this information. According to the cohort studies, the number of deaths during follow-up (or some fraction of it, e.g. one year) increases with the ambient level of air pollution (our 'effect estimate'). To calculate the years of life lost we would need the age distribution at the time of death. The age at death may be compared with the population based life expectancy to calculate potential years of life lost. The epidemiologic studies, however, did not publish the age structure at the time of death which occurred during follow-up. Therefore, assumptions had to be made regarding the age structure of air pollution related death. The first Swiss project (Künzli N et al, 1996) used the age distribution of all deaths, excluding violent deaths. However, both from morbidity and mortality studies it is known, that air pollution may be mostly related to cardiopulmonary diseases, and lung cancer. Therefore, a more specific approach may assume that air pollution related deaths have the same age distribution as all cardiopulmonary and lung cancer death in the population. This subgroup tends to die, on average, at older ages than all other non-violent causes of death. For example, in 1995, the following age structure of cause specific deaths could be derived (Annual Statistics Report for Switzerland, 1998):

Age distribution of age at death for different causes of death (Switzerland, 1998)

Causes	**Sex**	**% of non-violent death**	**Age (mean & percentiles)** mean	5th	50th	95th
Cardiovascular	M	39.8%	77.1	54.1	79.3	92.5
Cardiovascular	F	44.9%	84.2	67.2	85.8	96.1
Cardiovascular	*all*	*42.4%*	*80.9*	*59.2*	*83.2*	*88.7*
Respiratory	M	7.2%	79.1	60.8	80.9	93.3
Respiratory	F	5.2%	83.2	62.6	85.2	96.6
Respiratory	*all*	*6.2%*	*80.8*	*61.4*	*82.9*	*95.1*
Lung Cancer	M	6.6%	69.1	49.7	70.2	84.9
Lung Cancer	F	2.0%	68.2	46.1	69.5	87.3
Lung Cancer	*all*	*4.2%*	*68.9*	*48.8*	*70.1*	*85.4*
Cardiorespir.+Ca	M	53.6%	76.4	53.8	78.3	92.2
Cardiorespir.+Ca	F	52.1%	83.5	63.9	85.4	96.1
Cardiorespir.+Ca	***all***	***52.8%***	***80.0***	***57.3***	***82.4***	***94.7***
other (non-violent)	M	46.4%	68.8	31.7	72.9	90.5
other (non-violent)	F	47.9%	75.1	41.0	80.0	93.3
other (non-violent)	*all*	*47.2%*	*71.9*	*34.5*	*76.1*	*92.7*

As a consequence of our more specific assumptions about the age distribution of 'victims', the impact of air pollution on the years of life lost will be smaller in this international project as compared to the previous Swiss project. The mean age at death of cardio-respiratory and lung cancer death is about 8 years older than the mean age of

all other non-violent death. This methodological adaptation is in line with the 'at least' approach. It is to emphasize that there is no epidemiologic evidence against our assumption. For example, there are no data to support the believe that air pollution related premature death may be much older than other deaths due to the pertinent causes (DOH,1999).

g) ***Intrauterine and infant mortality:*** The relationship between air pollution exposure and infant mortality or intrauterine mortality has not much been examined in the past. There is currently one semi-individual retrospective cohort study on *infant mortality*, based on about 4 million infants born 1989-1991 in 86 US metropolitan areas (Woodruff TJ et al 1997). The authors controlled for individual risk factors for infant mortality (i.e., maternal education, maternal ethnicity, parental marital status, maternal smoking during pregnancy) and other potential confounders. An infants exposure was defined as the mean PM10 levels for the first two months of life. As shown by the authors, using the exposure over a child's entire lifetime would have led to an overestimation of effects because of declines in PM10 levels over time. The study design allowed to estimate the effect of PM10 exposure on infant mortality during a significant period of time within the first year of life. The obtained effect estimate of 1.04 per 10µg/m^3 PM10 is very similar to the joint estimate of the US cohort studies on adults. A recent time series study reported an increase in *intrauterine death* in association with levels of air pollution in São Paulo, Brazil (Pereira LAA et al, 1998). Daily counts of intrauterine death from January 1991 to December 1992 were related to daily levels of NO2, SO2, CO, O3 and PM10. To be documented in the Brazilian death certificate, intrauterine death is defined as a late fetal loss requiring either an age of pregnancy over 28 weeks, fetal weight over 1'000 g, or fetal length over 35 cm. Exposure levels were obtained from fixed monitoring sites. The concentrations of the pollutants were highly correlated. For the analysis, the authors used a Poisson regression model, controlling for season and weather (temperature and humidity). A strong positive association between daily fluctuations in air pollution and daily fluctuations in intrauterine death was found for NO2. The associations for SO2 and CO were weaker and less robust to model modifications. No positive associations were found for O3 and PM10. The authors discussed the difficulty to ascribe the observed adverse health effect to a single pollutant. As one possible explanation for the results, they suggested an independent effect of NO2. Atmospheric nitrogen oxides increase methemoglobin levels, which interferes with the oxygen-carrying capacity of hemoglobin in children. Infants are particularly susceptible to such events because fetal hemoglobin is more prone to be oxidized to methemoglobin. Additional evidence of fetal exposure to air pollution was obtained by disclosing a significant association between the levels of carboxyhemoglobin of blood sampled from the umbilical cord and ambient CO levels in 47 children delivered by nonsmoking pregnant women in the period from May to July 1995.
It was, however, decided not to include infant or intrauterine mortality into the estimate of traffic related health costs. The decision is again in line with the 'at least approach'. In fact, considering years of live lost, accounting for infant death would have a relatively large impact. We had the following reasons to ignore these health outcome measures:

- the evidence from only one study for each outcome is limited. With regard to infant mortality, more studies are needed which control for individual risk factors. No positive association was found between PM10 and intrauterine mortality. Future studies may provide more insight into the relative importance of different air pollutants and/or the mixture of complex automotive emission products for fetal death
- the population frequency of the outcome is of limited quantitative relevance (i.e., for infant mortality, the number of cases is very low), or may not be available (i.e., for

intrauterine mortality), and

- methodological problems inherent to the monetary valuation of the cases.

h) ***Cancer:*** Although there is some evidence for air pollution causing cancer, particularly lung cancer, we did not separately assess this impact. It should be noted, however, that it is at least partially covered in the attributed cases of premature mortality as we used the estimates for total mortality, including cancer. However, the expected additional cancer related impact with regard to morbidity and quality of life has not been assessed.

7.4. Effect estimates and impact assessment

Specified uncertainties

a) Given the observational properties of epidemiologic studies, the exposure-response functions have inherent limitations in *precision*. Epidemiological studies usually give exposure-response slopes or relative risk estimates with 95% confidence intervals (CI). The underlying statistical concept means that there is a 95% certainty that the true (unknown) effect lies in fact within this range. Therefore, 95% confidence intervals are an important way to quantify uncertainties inherent to the pooled health outcome measures. The meta-analytic variance weighted calculation of point estimates and confidence intervals has been widely used in epidemiology. However, assumptions and judgments are applied when joint estimates across epidemiologic studies are derived and uncertainties are quantified. There is a growing literature available to guide the meta-analytic approach (Rothman KJ and Greenland S, 1998). Due to inherent measurement errors, mis-specifications of models, variations in population risk profiles and health care systems, differences in the effect estimates from studies looking at the same health effects are likely. Even if the studies used for the meta-analysis are selected using defined quality criteria, it is necessary to look for systematic variations in estimates across studies. If the Q test (Petitti D, 1994) reveals considerable heterogeneity of the effect estimates, a random effects model may be chosen, in which a term for between-study variation is added to the meta-analytic process.

b) The *use of confidence intervals* to express uncertainty in precision is another point of discussion. Impact assessments often prefer the analyst judgment approach, selecting low, central and high estimates for each exposure-response relationship (and each monetary value estimate) quantified in the assessment (Ostro B and Chestnut L,1998). In the aggregation step of the assessment, each low, central and high estimate is assigned a probability weight. The weights are summing to 100% for each quantified health effect and for each monetary value. The resulting probability distribution of the total health benefits estimate is assumed to reflect the uncertainty in precision in a more realistic way than summing up all the low and all the high estimates to obtain total low and high estimates. There is, however, no standardized procedure for this approach, and the selection of low and high parameter estimates for each health endpoint is arbitrary and guided by different conceptual bases.

c) For each country project, we applied identical exposure-response functions. However, the baseline frequencies rely on national conditions. Therefore, the additional number of cases attributed to a 10 $\mu g/m^3$ increase in PM10 may differ across countries, reflecting the variation in health conditions in different regions. If data are available, this is a more specific approach than assuming a fixed number of attributable cases. The latter may be

suitable as a crude estimate where health frequency data are not available (e.g., as applied in an unpublished, more detailed draft version for WHO, 1998).

d) Further quantified uncertainties such as the fraction of cases attributed to air pollution from traffic (Fa), and the air pollution baseline level (B) are subject to the overall sensitivity analysis at the interdisciplinary level (not part of this report). These calculations result in a N_{min} and N_{max}, where N_{min} is based on $D10_{low}$ and the lower bound estimates of all other parameters, and N_{max} on $D10_{upp}$ and the upper bound estimates of all other parameters (see Economy Report, Sommer et al, 1999).

Unspecified uncertainties

e) The use of the meta-analytic point estimates rather than estimates from one single study are a reasonable compromise to enhance the value of the available information (Blair A et al 1995), and to deal with heterogeneity across different studies. The approach, however, assumes, that the true exposure-response function for a given particle mass concentration is identical across countries. Thus, differences across studies stem from inherent methodological variations and random variability rather than from true differences in the effects of PM10. Although there is no proof of this assumption, e.g., the numerous mortality time-series studies conducted all over the world are of strong consistency in the effect estimates, thereby supporting this approach. With increasing measurements of size fractionated particle mass in Europe and the North American continent, it will be possible to base future impact assessments solely on effects of particles equivalent to or smaller than PM10.

f) The impact assessment entirely relied on epidemiologic studies. Although there is uncertainty about extrapolating epidemiologic findings from study samples to the entire population, living in similar or different context, risk assessment by default requires extrapolation. We agree with Ostro (Ostro B and Chestnut L, 1998) that the magnitude of uncertainty using epidemiologic studies is smaller than the extrapolation from toxicological or clinical studies. This is due to the fact, that toxicological or clinical studies of human subjects rely on circumstances grossly different from the target population (e.g. time of observation, selection of subjects etc.).

g) The extrapolation of *effect estimates* from studies conducted elsewhere is considered to be a non-quantifiable uncertainty which may lead to under- or overestimation of the results. Under ideal conditions, the estimated impact attributable to traffic related air pollution for each country were at least partly based on epidemiological studies originating from that country. The effect estimates for hospital admissions and asthma attacks were exclusively based on European studies. For long-term mortality, chronic bronchitis incidence, and restricted activity days, however, only studies from the US were available. The effect estimate for bronchitis in children was based on a Swiss and two US studies. Although meeting the same pre-defined criteria for high quality standards, a mixture of studies from different regions and populations may increase the heterogeneity of effect estimates.

h) The use of PM10 conversion factors to adjust effect estimates based on other measures of particulate matter bears an uncertainty which can not be quantified.

i) More elaborate approaches to quantify uncertainties include modeling of the *distribution* of impact probability, e.g., with Monte Carlo simulation techniques (EPA 1996, Krzyzanowski

M, 1997). Input data for such procedures may include information on the range of probabilities of the estimates of exposure, their consequences, and the baseline frequency measures in the populations. We did not apply any of these more sophisticated approaches. In total, the uncertainty in the monetary estimates are large and go well beyond the mere epidemiology based uncertainties. Thus, further sophistication may be of little scientific value in this project and rather suggest a degree of precision, which is not rectified by the data.

j) We do not disaggregate the impact assessment air pollution by age groups apart from our restriction to age groups used in the epidemiologic studies. This may be warranted if the effect estimates of air pollutants were to be clearly different by age, as might be suggested by some results (Spix C et al, 1998; Verhoeff AP et al, 1996). Disaggregation may have little influence on the overall impact estimates as the reduction due to using lower exposure-response slopes in young age groups, with lower baseline frequencies of the outcome, may well be balanced by the application of larger effect slopes among the elderly, with at the same time higher frequency of outcome.

k) ***Mortality:*** The joint estimate for long-term mortality was derived from two large US cohort studies. In order to support the evidence from these studies, additional research on the impact of long-term exposure to air pollution on mortality from chronic diseases is needed (McMichael AJ et al, 1998). As in most epidemiologic studies used for the impact assessment, the population exposure in the cohort studies was based on average values calculated from data measured at the stations operating in the region where the population lives. Although this design is very valuable and should not be considered an 'ecologic study' (Künzli N and Tager I, 1997), an inherent *caveat* in long-term cohort studies has to be taken into account. If air pollution levels in the past have been much higher than those measured during the follow up period, this may influence the effect estimates. Assuming, e.g., that the air quality improvement was much stronger in the most polluted areas compared to the less polluted study sites, the observed effects may be an overestimation. This uncertainty has not been quantified in this project. It is to emphasize, however, that the average effect estimate used in our equations are very close to the result of the ACS study (Pope CA et al, 1995). The ACS study, however, included more than 150 metropolitan areas, thus, the impact of differential air quality improvement may be smaller than in the Harvard Six City Study which reported higher effect estimates. Further unspecified uncertainties may be related to the size of the study areas. The metropolitan areas in the ACS study were larger than the areas in the Harvard Six City Study. This potentially increased the random exposure measurement error.

l) ***Hospital admissions:*** There is an increasing trend that studies on air pollution related hospital admissions use all respiratory (ICD9 460-519), and all cardiovascular (ICD9 390-429) or even circulatory (ICD9 390-459) categories, respectively. Thus, there is less uncertainty due to a combination of different diagnostic sub-groups. Contrary to the previous Swiss project (Künzli N et al, 1996), the effect estimates were derived only from European studies, which include all age groups, therefore also reducing uncertainty. As a consequence, and due to the use of a random effects model for respiratory hospital admissions, the confidence intervals were wider than in the previous Swiss project. The growing body of studies from Europe and the North American continent revealed joint estimates of 1-2% per increment of 10 $\mu g/m^3$ PM10, thus providing further evidence for a causal effect of this magnitude. Together with mortality and morbidity endpoints, hospital utilization data strongly support the coherence of a broad range of health consequences associated with air pollution (Bates D, 1992).

m) ***Chronic bronchitis in adults:*** In the previous Swiss project (Künzli N et al, 1996), prevalence of chronic bronchitis (ICD9 code 491) has been used as there are European effect estimates available for the increase in chronic bronchitis prevalence due to air pollution (Zemp E et. al, in press). To facilitate the monetarization process, we now provide the impact of air pollution on the *incidence* of chronic bronchitis, i.e., the increased occurrence of new cases as a consequence of long-term cumulative air pollution experience. Incidence data, however, require the longitudinal observation of populations over a number of years. Therefore, both effect estimates and baseline frequency had to be derived from one single U.S. study (Abbey DE et al, 1993). This approach has been recently used by Ostro and Chestnut (Ostro B and Chestnut L, 1998). The incidence was derived from a Seventh Day Adventist population of current non-smokers, who were potentially exposed only to past and passive smoking (Abbey DE et al, 1993). The prevalence and the effect estimate for prevalence from the Swiss SAPALDIA study (Zemp E et al, in press) were taken from a (theoretical) never-smoking population. Moreover, the second effect estimate for prevalence from the study by Schwartz (Schwartz J, 1993) was also based on never-smokers. Therefore, for incidence and prevalence, the impact is most likely underestimated ('at least' approach). The different age ranges used in the studies bear a non-quantified uncertainty about the relevant underlying age group. Incidence and prevalence were partly based on the same definition of having symptoms of cough and/or sputum production on most days, for at least three months per year, and for 2 years or more. Schwartz, however, asked for a doctors diagnosis, and participants had to confirm that the disease was still present. These differences in defining the outcome are a further uncertainty that can not be quantified. From an epidemiologic point of view, there is no relevant difference between the incidence and prevalence approach in the application to impact assessment. Prevalence is dependent on incidence and duration of disease. The average duration of chronic bronchitis may be of additional interest for the economist if the life-time (diseased life-time) costs would be known. There is, however, little information about the duration of chronic bronchitis. Among clinicians, it is considered to be a disease of the fifth decade. Assuming an average age at death of about 75-80 years (see table above), the average duration of chronic bronchitis is likely to be at least 20 years.

n) ***Bronchitis in children****:* The previous Swiss project (Kuenzli N et al, 1996) used bronchitis and repeated cough in the last 12 months, respectively, as endpoints. In order to avoid misclassifications due to overlapping between both entities, it was decided to keep only one of them. Bronchitis was chosen because more data were available. All three epidemiologic studies (Dockery DW et al 1989; Dockery DW et al 1996; Braun-Fahrländer et al 1997) used the definition 'bronchitis in the last 12 months', but applied it in different age groups (10 to 12 years, 8 to 12 years, and 6-15 years, respectively). Especially in children, it may be difficult to separate bronchitis from asthma symptoms. The prevalence in Austria was based on parents' answers about a doctors diagnosis of asthma-like bronchitis or wheezy bronchitis in their child, a doctors diagnosis of bronchitis in their child, and self-reported last 12 months bronchitis for adolescents, respectively. In the Swiss SCARPOL study, parents were asked whether their child had had an airway disease within the last 12 months, and separate answers could be given for bronchitis, asthma, pneumonia, croup, influenza, or other diseases. These definitions used for the classification of bronchitis cases in the different samples of children and adolescents bear some non-quantified uncertainty in the applied prevalence. A further uncertainty inherent to the Austrian prevalence is the use of different age groups and samples which are not representative for the whole country. A limitation for the monetarization is the lacking information on the number of bronchitis episodes during the 12 months.

o) ***Restricted Activity Days:*** The only exposure-response estimate with regard to particulate air pollution and RAD is presented in the paper by Ostro (Ostro B, 1990). This includes days off work as well as minor restrictions of daily life due to respiratory disease. The estimate for RADs is not partitioned into these different aspects, thus an estimate for 'days off work' in relation to particulate pollution is not explicitly reported. However, the annual mean RAD estimate reported by Ostro, which was based on personal interview of study participants, is close to the Austrian estimate for 'days off work' due to respiratory disease, derived from a general practitioner-based notification system. The diary estimate from the SAPALDIA study, although again based on another epidemiologic method for health assessment, comes close to those of the other two sources. Standards of living determining the population burden of respiratory disease, working conditions and medical standards can be considered comparable for the three countries. This comparison indicates that nearly all of the RAD burden reported by Ostro should relate to days off work. This assumption appears plausible, because the data were collected in a working population, and any restriction of daily activity due to respiratory disease should most likely also have caused a person not to go to work. Although the pressure not to take a sick-leave has likely changed over the last years, this only caused small changes in reported population means for days off work. E.g., in Austria the Association of Social Security Agencies reported all annual estimates for days off work from 1987 to 1996 in between 14.6 and 12.9 days. Although RAD estimates have been obtained in different ways, we are nevertheless confident, based on the consistency of these estimates, to use the Ostro estimate for deriving the pollution impact on RAD for the three case countries (Austria, Switzerland, and France).

p) ***Asthma attacks*****:** On request of the economy group of this project, we used additional asthma attacks as an outcome. Asthma attacks are more suitable for the Willingness To Pay approach. The previous Swiss project (Kuenzli N et al, 1996) had found the same effect size for asthma attacks and use of medication in asthmatics. The consistence of effects supports not only the evidence of air pollution consequences on the severity of asthma but also relates to definitions of asthma attacks, which may be operationalized through the information on asthma medication. The use of different definitions for asthma (i.e., wheeze, shortness of breath, lower respiratory symptoms), is a non-quantified uncertainty. Only European studies were used for the joint effect estimates on asthma attacks for children and adults. The effect sizes had the same magnitude of about 4-5% per 10 $\mu g/m^3$ PM10 as applied in the first Swiss project. Due to inherent problems, the results of the Pollution Effects on Asthmatic Children in Europe (PEACE) study were not included. The results of this study have not shown any strong and consistent relationship between air pollution and peak expiratory flow (PEF), respiratory symptoms or asthma medication use. The authors have discussed the factors which may have influenced the results. A considerable number of children may have been mis-classified as asthmatics due to a single affirmative answer to the question about nightly cough in the last 12 months, apart from cough with a cold or chest infection. Furthermore, the diary data did not allow to sufficiently control for infections. In a review of the PEACE study, the authors concluded that in previous studies, variations in respiratory health endpoints caused by factors other than air pollution may have been of insufficient magnitude to obscure relationships between air pollution and respiratory health (PEACE study, 1998). The number of asthma attacks in the population were based on questions from the International Study of Asthma and Allergies in Childhood (ISAAC), and the European Community Respiratory Health Survey (ECRHS). The results were very similar in the three countries, so that one common estimate was applied for children and adults, respectively. Data from French asthma panels supported the validity of the survey based data.

8. Open questions and needs for future health impact assessments

To improve the air pollution related impact assessment, a variety of research needs can be suggested. Some require further epidemiologic studies, whereas others may need development in statistical approaches or further literature review. The quality of and access to data has to be further improved. We highlight open issues which relate to exposure, health outcomes, and to general problems of the impact assessment. Most paragraphs can be attributed to issues of methodological research, data collection, or both (at the end of each paragraph, in parentheses). As mentioned, there is no common method for impact assessment. We are well aware that others are engaged in similar projects applying, however, different assumptions and methods (also see Annex). *For several steps, there may indeed be different options. We strongly favor convergence of methods and assumptions to reach comparability across projects. This could be done under the initiative of WHO.*

8.1. Open questions regarding exposure assessment

a) It is not clear how comparable effect estimates from different regions, based on different size fractions, composition and content of particulate matter pollution are. The observed heterogeneity may either reflect differences in the effects or random variation. Further studies regarding mechanisms of effects of particulate pollution and studies about the content and heterogeneity of fine particles will be helpful. There might be different air pollution indicators and effect estimates for specific endpoints in the future (e.g., surface area of diesel derived particles for the endpoints lung cancer and allergic rhinitis, surface area of fine particles for the endpoint cardiovascular diseases). Additional emphasis may be also put on particle numbers and its association to particle mass (methodological research).

b) For future health impact assessments, *personal* exposure distribution data may be applied to the *personal* exposure-outcome function, if available. It will then be the question how much this affects the results in comparison with the so far used semi-individual coefficients in combination with the ambient exposure distributions (methodological research).

c) Standardized air pollutant monitoring is required to derive the population exposure distribution, applicable both for epidemiologic studies and for impact assessment. Mainly fine particulate (PM2.5) and carcinogens are not yet routinely measured, and measurements of PM10 with different devices are not always comparable, therefore limiting to abridge health effect study results with impact assessment. The ozone exposure distribution will be needed to derive the health impact of oxidant pollution in regions with high primary air pollution, and long and sunny summer periods (data collection).

8.2. Open questions regarding health outcomes

a) Future epidemiologic studies should provide estimates of long-term cumulative air pollution effects on health outcome measures such as the impact on:

- mortality in Europe
- life expectancy and years of life lost
- severity and course of chronic morbidity
- cancer
- quality of life

These outcome measures require cohorts which are needed to be followed up, particularly in Europe where no such studies have been conducted yet. The most efficient way may be to follow up recently established respiratory health cohorts. European cohorts are those of the Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA), the French study Pollution Atmospherique et Affections Respiratoires Chroniques (PAARC) (Baldi I, 1998), both set up explicitly to assess the impact of air pollution, and the European Community Respiratory Health Survey (ECRHS) (Burney P, 1994). Although ECRHS has not been primarily set up to address air pollution effects, the cohort is perfectly suitable to address these issues as the exposure profile of the cohorts may be assessed *a posteriori*. Long-term effect estimates need to be established for all outcomes for which there is evidence for an association with current or long-term ambient air pollution exposure. Major efforts should be undertaken to standardize the outcome definitions and main characteristics of the populations under study (e.g., age groups, smoking status), both for the effect estimates and the population frequencies derived from epidemiologic studies (methodological research, data collection).

b) Further epidemiology studies addressing the short-term effect of air pollution on health outcomes such as intrauterine mortality, infant mortality, and cardiopulmonary morbidity (e.g., doctors and emergency room visits, absence from work, school absence in children, cardiac demand pacemakers, medication use observed in pharmacists sentinel networks, etc.) are needed (methodological research).

c) Epidemiologic studies which identify risk groups of air pollution effects (susceptibility), addressing age, sex, preexisting diseases, and other potential markers of susceptibility (e.g., low socio-economic class, unemployment, poverty, genetic setup) are also needed. The studies should also report the prevalence of the respective markers of susceptibility. The question will then be to what extent more detailed knowledge on subgroup effect estimates and frequencies may influence the overall public health impact of air pollution (methodological research).

d) Reasons for heterogeneity between effect estimates due to differences in health care systems and routine outcome data sources have to be further investigated. International projects, which follow a standardized protocol, allow to study variations between participating centers. This is one of the main objectives in the APHEA2 project (Short-term Effects of Air Pollution on Health: a European Approach) from 1998 to 2001, in co-operation with the U.S. National Morbidity, Mortality and Air Pollution Study (NMMAPS). Efforts have then to be undertaken to harmonize the ways routine data bases are

designed and maintained. Standardization of data collection, cleaning and storage in data bases is of major interest. Differences in health care systems in Europe may diminish in the long run (methodological research, data collection).

e) Research is needed to better describe the characteristics of those dying from air pollution. Time-series studies will be useful to improve knowledge regarding the short-term impact of air pollution on mortality. The impact of short-term effects of air pollution on public health will be further assessed by APHEA2. The impact may be a combination of acute and chronic cumulative effects, affecting severely diseased persons as well as rather healthy middle aged persons who loose a considerable part of their life expectancy (especially for cardiovascular mortality). The assessment of the mortality displacement in different population subgroups (i.e., the life time lost due to premature death caused by air pollution) will help to better determine the public health importance of short-term effects of air pollution on health. As part of a sensitivity analysis, the number of life years lost for society estimated from time series studies may then be compared with the loss of life expectancy derived from the cohort studies on long-term effects. In the meantime, the appropriate approach to estimate health costs related to air pollution is to use for the main analysis only the available cohort studies on long-term mortality (methodological research).

f) Further research is needed to specify the issue of years of life lost. Of particular interest is the quality adjusted years of life lost. From a policy and public health perspective it is important to weigh potential gain in years of life with the quality of life. This is particularly true for an aging population with its enormous impact on the society (methodological research).

g) We used the cohort based estimates of the impact of air pollution on mortality. Although one may expect that these estimates include the entire burden of air pollution which may lead to death, it is not yet established, to what extent short-term effects of air pollution episodes may be fully included in the cohort based estimates (methodological research).

8.3. Open questions regarding effect estimates and impact assessment

a) Air pollution is a complex mixture, monitored and assessed by just a few pollutant indicators. This is a potential limitation, which may not be entirely preventable but reduced and modified in quality. Therefore, in light of the different options of environmental development, the impact assessment should be further developed to compare the impact of different scenarios of future quality and mixtures of air pollution. One future strategy for air quality management consists in the application of health impact assessment as an integral part of environmental impact assessment for all projects, policies, programs and strategies with implications for air quality and public health (methodological research).

b) To be able to apply health impact assessment of air pollution on health, data are required on exposure, health outcomes and exposure-response relationships. The data used for epidemiologic studies can, in different units of time (e.g., annual averages), be also used for the impact assessment. The existing structures and databases of national air monitoring networks and European Air Quality databases (e.g., AIRBASE of the European

Environment Agency (EEA)), impact assessment centers (i.e. of the WHO European Centre for Environment and Health (WHO-ECEH) and the air pollution epidemiology community (e.g., national research and public health centers, European projects such as ECRHS, APHEA2) should be better coordinated and integrated, to make best use of them for impact assessment.

c) Applied in a *systematic* way, health impact assessment can be used for epidemiologic surveillance of air pollution health effects. On the European level, regularly updated information on health impacts is essential for the periodic reviews of the WHO air quality guidelines and the European Union air pollution limit values. European countries can use systematic health impact assessment to make air quality management more effective, and to improve the knowledge in the population on air pollution risks.

d) Epidemiologic studies should collaborate with economists to ensure the use of health outcome measures that may be monetarized. Otherwise, it will remain difficult to internalize indirect costs of environmental pollution. As seen in this project, a large body of epidemiologic data is not applicable to economic methods of monetarization.

e) The issue of competing risks should be addressed in the environmental health field. It is commonly believed that the 'attributable cases' are, at the same time, the 'preventable fraction' of cases if we were to remove the exposure. However, this may not be entirely the case due to competing risk factors which may affect the 'prevented cases' (Brunekreef B, 1997) (methodological research).

f) Further work is needed on methods to deal with uncertainty in the impact assessment (i.e., meta-analytic approach; simulations etc.). It will be most fruitfully done in close collaboration of air hygienist, economist, and epidemiologist. (methodological research).

9. Annex

9.1. Differences to the previous Swiss project

The international project of ambient air pollution impact assessment has some methodological differences with the previous Swiss project (Künzli N et al, 1996). As a consequence, the results will not be identical.

a) The effect estimates of some health outcome are slightly different due to the inclusion of recently published data in the meta-analysis. We particularly focused on new European data.

b) To quantify uncertainties inherent to the combined relative risks, 95% confidence intervals were used instead of $\pm$ 1 standard error, resulting in wider ranges.

c) All data regarding population frequencies and exposure relate to 1995 to 1997 rather than earlier years.

d) The population average exposure to PM10 has again been estimated, applying new emission-based methods which slightly changed the mean. Thus, the back-extrapolation procedure to the baseline exposure is a source of change.

e) For the conversion of particulate matter indicators in the respective epidemiologic studies, PM10 was now assumed to be equivalent to PM15 (before: PM10 = PM15 * 0.9).

f) We 'flattened' the exposure-response curve, applying an additive rather than multiplicative model. Although this has very little impact in the range of exposure observed in Switzerland, the new approach may prevent overestimation of impact, if applied to countries with high levels of PM10.

g) When significant heterogeneity between the studies was present, random effects estimates were used to calculate D_{10} (D_{10low}, D_{10upp}).

h) We now propose to assume that air pollution related death have the same age distribution as all other cardiopulmonary death (including lung cancer) in the population rather than all other non-violent death. The assumption is reasonable and will lead to a reduction in the attributed cases of death.

i) Given the recently published new evidence from Europe regarding the impact air pollution on hospital admissions and asthma attacks, we now restricted the project on European studies to derive the effect estimates. The joint estimates are in the same range than the U.S. based estimates.

j) In case of chronic bronchitis in adults, incidence rather than prevalence was used, in

accordance with Ostro and Chestnut (Ostro B and Chestnut L, 1998), and the needs of the economists, which, in contrast to the previous Swiss project, selected the Willingness To Pay method.

k) Bronchitis in children: In order to avoid misclassifications due to overlapping between repeated cough and bronchitis, it was decided to keep only one of them (bronchitis).

l) On request of the economy group of this project, acute effects of air pollution on asthmatics have now been assessed by attributable asthma attacks rather than medication.

9.2. Differences to the current French project

The French project (Institut de Veille Sanitaire (InVS), in preparation) is performed in the framework of the Regional Air Quality Plans implemented by the recent Air Quality Law to prevent or reduce the effects of air pollution. These plans include a local evaluation of the health effects of air pollution.

a) In the French project, the health impact assessment is not focused on costs estimates but on a more general and broader public heath perspective.

b) The assessment is not restricted to PM10, it also includes SO2, NO2, and ozone.

c) In the first step of the project, the health impact assessment is proposed only for urban areas.

d) Daily air pollution levels are measured by local air quality networks and follow a standardized protocol regarding the selection of stations, correlation between stations, and missing values.

e) Only short-term effects of air pollution on mortality and hospital admissions are selected in this first step of the project based on the consistent evidence of these effects worldwide and the availability of these results for the larger French cities and most of the European countries. As there is not such an experience on long term effects for Europe and only two U.S. studies are available, their inclusion will be discussed once the results of the trilateral study have been published.

f) In this step, the health indicators included in the assessment are restricted to total anticipated mortality and daily hospital admissions for respiratory and cardiovascular conditions.

g) The health impact assessment is presented on a daily basis and on an annual basis for different increases in daily levels of air pollution (from percentile p5 to p50, or from p25 to p 75) or for the daily mean levels of air pollution, or to show the respective contribution of low, medium and high levels of air pollution in one year.

h) For mortality, the effects of the air pollution indicators cannot be added to each other, so the hypothesis was made that the highest impact of only one of the air pollution indicators is lower than the minimum impact of all air pollution indicators combined. The same hypothesis was applied for respiratory and cardiovascular hospital admissions.

9.3. Examples of further impact assessment studies

Many impact assessment studies of air pollution on public health have recently been conducted or under way. This report can not fully cover all of these projects. Many are not yet published, not easily available (gray literature) or published in other languages. However, we want to give short references of recent projects and report some important results.

Case estimation

a) WHO estimated that worldwide about 3 million premature deaths have to be attributed to exposure to air pollution (http://www.who.int/ peh/resources/supercourse/ test4.1/13.htm).

b) In an impact assessment for the Ministerial Conference for Environment and Health, London 1999 (http://www.who.dk/London99), the WHO European Centre for Environment and Health (WHO-ECEH) has estimated 100'000-400'000 extra deaths per year due to suspended particulate matter for 314 million people in Europe of the mid nineties.

c) The Committee on the Medical Effects of Air Pollutants (COMEAP, 1997) has estimated 8'100 additional deaths per year and 10'500 additional hospital admissions per year due to exposure to PM10 in the urban areas of the UK. The estimated death rely exclusively on short-term effects of air pollution. Thus, the reported number can not be considered the annual attributable number of additional cases but rather the number of deaths whose occurrence has been affected by the level of air pollution shortly before death. Based on time-series studies, the time lost can not be assessed, thus, we can not estimate how many of these premature deaths may have occurred in the same year any way. (Also see i) in the next section)

d) Other recent case estimations have been undertaken by Natural Resources Defense Council for 239 U.S. metropolitan areas (NRDC, 1996), and the international Working Group on Public Health and Fossil-Fuel Combustion for the world (WGPHFFC, 1997).

Cost estimation

e) In total, about one billion EUR were attributed to traffic related health costs due to air pollution in Switzerland in 1993 (Sommer H et al 1996; Künzli N et al, 1996; Künzli N et al, 1997).

f) Based on health impact assessments, the U.S. Healthy People 2000 report estimates for each year in the United States health costs of human exposure to outdoor air pollutants in a range of 34 billion EUR to 43 billion EUR, and 50'000 to 120'000 premature deaths associated with exposure to air pollutants (http://www.cdc.gov/nceh/pubcatns/1994cdc/brosures/airpollu.htm)

g) A recent cost-benefit analysis estimated the health and economic benefits of reducing air

pollution to the new U.S. standards for PM2.5 (particulate matter 2.5 µm or less in diameter) of 15µg/m3 in urban areas covering 57% of the U.S. population: the main results were 7'200 annual deaths prevented (central estimate) and overall 27 billion EUR benefit (of which 82% are due to deaths) (Ostro B and Chestnut L, 1998).

h) Other recent cost estimations have been undertaken by the European Commission for European cities (EC, 1997), the Umwelt und Diagnose-Institut Heidelberg for Germany (UPI, 1997), the U.S. Environmental Protection Agency for the USA (EPA, 1996), the American Lung Association for the USA (ALA, 1995), and the Canadian Council of Ministers of the Environment for Canada (CCME, 1995).

i) The recent economic appraisal of the Department of Health (DOE, 1999) monetarized hospital admissions and death related to short-term pollution (PM10, SO2, and Ozone). They selected several steps of down-sizing the economic values. Their assumptions regarding the age structure and quality of life of those dying prematurely due to air pollution are based on clinical judgment. There is, however, no direct evidence to support the respective assumptions. In fact, more recent studies (Schwartz, 1999; Zeger et al. 1999) demonstrate that even in the case of short-term effects, death may be shifted by much longer periods than the time window assessable by means of time-series analyses. The assessment does not consider any long-term effects of air pollution.

A major limitation of these impact assessments is that the projects are based on different contents, methods and assumptions. The results can neither easily be compared nor transferred to other countries. Therefore, a broad application for regulative and preventive measures of air pollution health risks is questionable. WHO-ECEH is constantly working on the development of the methodological framework for health impact assessment, and its database HEGIS (Health and Environment Geographic Information System) (WHO 1995a; WHO 1995b; WHO, in preparation). A working group has been set up by WHO-ECEH in 1998 to improve air pollution impact assessment and to increase the effectiveness of the WHO-ECEH air quality program.

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11. Tables

Table 1: Health outcomes included and not included in the assessment

Included in the Impact Assessment	Not included
• Total mortality (adults ≥30 years; long-term effect) • Respiratory hospital admissions (all ages) • Cardiovascular hospital admissions (all ages) • Chronic bronchitis (adults ≥25 years) • Acute bronchitis (children <15 years) • Restricted activity days (adults ≥20 years) (RAD) • Asthmatics: asthma attacks in children <15 years asthma attacks in adults ≥15 years	• Acute (short-term) effects on mortality • Infant mortality • Intrauterine mortality • Emergency room visits • Respiratory symptoms in adults • Respiratory symptoms in children • Lung function • School absentees • Cancer §) • Physical performance • Eye irritations • Increase in bronchial reactivity

§) in terms of mortality: implicitly included; not included in terms of morbidity

Table 2: Additional cases per million inhabitants and per 10 μg/m^3 of pollutant concentration (of PM_{10})

Health outcome	Effect estimate **Relative Risk** (±95% Confidence Interval (CI))	Observed population frequency, P_e {and expected baseline frequency, P_0 at 7.5 μg/m^3 PM10 annual mean} Per 1 Mio. inhabitants			Fixed baseline increment (D10) per 10 μg/m^3 PM10 and 1 Mio. Inhabitants Cases D_{10} (D_{10low}, - D_{10upp} based on ±95% CI estimates)		
		A	F	CH	A	F	CH
Total mortality (adults ≥ 30 years)	1.043 (1.026-1.061)	9'326 {8'634}	8'391 {7'848}	8'263 {7'794}	374 (226-524)	340 (206-476)	337 (204-473)
Respiratory Hospital Admissions (all ages)	1.0131 (1.001-1.025)	17'826 {17'405}	11'550 {11'313}	10'300 {10'155}	228 (24-433)	148 (16-282)	133 (14-253)
Cardiovascular Hospital Admissions (all ages)	1.0125 (1.007-1.019)	36'790 {35'958}	17'270 {16'931}	24'640 {24'219}	449 (234-668)	212 (112-315)	303 (157-450)
Chronic Bronchitis Incidence (Adults ≥ 25 years)	1.098 (1.009-1.194)	4'986 {4'223}	4'661 {4'031}	5'013 {4'414}	413 (37-821)	394 (35-784)	431 (38-858)
Bronchitis (children < 15 years)	1.306 (1.135-1.502)	16'369 {10'457}	23'534 {15'806}	21'545 {15'125}	3'196 (1'409-5'774)	4'830 (2'129-8'728)	4'622 (2'037-8'352)
Restricted Activity Days (adults ≥ 20 years) [1)]	1.094 (1.079-1.102)	2'597'294 {2'211'837}	3'221'240 {2'799'326}	3'373'040 {2'982'515}	208'355 (175'399-241'754)	263'696 (221'987-305'966)	280'976 (236'533-326'016)
Asthmatics: Asthma attacks (children < 15 years [2)]	1.044 (1.027-1.062)	56'670 {52'368}	62'789 {58'624}	57'483 {54'142}	2'325 (1'430-3'231)	2'603 (1'600-3'617)	2'404 (1'478-3'341)
Asthmatics: Asthma attacks (adults ≥ 15 years) [2)]	1.039 (1.019-1.059)	173'439 {161'823}	169'491 {159'584}	172'914 {164'066}	6'279 (3'058-9'564)	6'192 (3'016-9'431)	6'366 (3'101-9'697)

1 Restricted activity days: total person-days per year 2 Asthma attacks: total person-days with asthma attacks

Table 3: Underlying definitions of health outcomes in the epidemiologic studies used for the air pollution impact assessment

Health outcome	Definition	Sources (References)
Long-term mortality (adults ≥30 years)	death rate, excluding violent death / accidents,	Dockery DW et al, 1993
	ages 25-75 and >30 years, respectively	Pope CA et al, 1995
Respiratory Hospital Admissions	hospital admissions for respiratory disease:	
(all ages)	ICD9 460-519, all ages	Spix C et al, 1998
	ICD9 466, 480-487, 493, 490-492, 494-496, all ages	Wordley J et al, 1997
	ICD9 480-487, 490-496, all ages	Prescott GJ et al, 1998
	(not all papers state that only unscheduled admissions have been included)	
Cardiovascular Hospital Admissions (all ages)	hospital admissions for cardiovascular/circulatory disease:	
	ICD9 410-436, all ages	Wordley J et al, 1997
	ICD9 390-459, all ages	Poloniecki JD et al, 1997
	ICD9 390-459, all ages	Medina S et al, 1997
	ICD9 410-414, 426-429, 434-440, all ages	Prescott GJ et al, 1998
	(not all papers state that only unscheduled admissions have been included)	
Chronic Bronchitis Incidence (adults ≥25 years)	symptoms of cough and/or sputum production on most days, for at least three months per year, and for 2 years or more, age >=25 years	Abbey DE et al, 1993
Bronchitis (children <15 years)	bronchitis in the last 12 months (parents or guardian's answer), ages 10-12, 8-12 and 6-15 years, respectively	Dockery DW et al, 1989 Dockery DW et al, 1996 Braun-Fahrländer C et al, 1997
Restricted Activity Days (adults ≥20 years)	any days where a respondent was forced to alter normal activity, due to respiratory disease ICD9 460-466, 470-474, 480-486, 510-516, 519, and 783, age 18-65 years	Ostro B et al, 1990
Asthmatics: Asthma attacks (children <15 years)	lower respiratory symptoms, age 6-12 years	Roemer W et al, 1993
	asthma, age 7-15 years	Segala C et al, 1998
	lower respiratory symptoms, age 7-13 years	Gielen MH et al, 1997
Asthmatics: Asthma attacks (adults ≥15 years)	wheeze, age 18-80 years	Dusseldorp A et al, 1995
	shortness of breath, age 18-55 years	Hiltermann TJN et al, 1998
	wheeze, age 16-70 years	Neukirch F et al,1998

12. Figures

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14. List of abbreviations and Glossary