Public-health impact of outdoor and traffic-related air pollution: a European assessment

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Summary

Background Air pollution contributes to mortality and morbidity. We estimated the impact of outdoor (total) and traffic-related air pollution on public health in Austria, France, and Switzerland. Attributable cases of morbidity and mortality were estimated.

Methods Epidemiology-based exposure-response functions for a 10 μg/m^3 increase in particulate matter (PM_{10}) were used to quantify the effects of air pollution. Cases attributable to air pollution were estimated for mortality (adults ⩾30 years), respiratory and cardiovascular hospital admissions (all ages), incidence of chronic bronchitis (adults ⩾25 years), bronchitis episodes in children (<15 years), restricted activity days (adults ⩾20 years), and asthma attacks in adults and children. Population exposure (PM_{10}) was modelled for each km^2. The traffic-related fraction was estimated based on PM_{10} emission inventories.

Findings Air pollution caused 6% of total mortality or more than 40 000 attributable cases per year. About half of all mortality caused by air pollution was attributed to motorised traffic, accounting also for: more than 25 000 new cases of chronic bronchitis (adults); more than 290 000 episodes of bronchitis (children); more than 0·5 million asthma attacks; and more than 16 million person-days of restricted activities.

Interpretation This assessment estimates the public-health impacts of current patterns of air pollution. Although individual health risks of air pollution are relatively small, the public-health consequences are considerable. Traffic-related air pollution remains a key target for public-health action in Europe. Our results, which have also been used for economic valuation, should guide decisions on the assessment of environmental health-policy options.

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Introduction

Research during the past 10–20 years confirms that outdoor air pollution contributes to morbidity and mortality.[1,2] Whereas some effects may be related to short-term exposure,[3] others have to be considered contributions of long-term exposure.[4,5] Although the mechanisms are not fully explained, epidemiological evidence suggests that outdoor air pollution is a contributing cause of morbidity and mortality.[6] State-of-the-art epidemiological research has found consistent and coherent associations between air pollution and various outcomes (eg, respiratory symptoms, reduced lung function, chronic bronchitis, and mortality).[6]

Relative risks related to air pollution, however, are rather small. For example, for an average adult, the risk of dying may increase on any given day by less than 1% if the concentration of inhalable (<10 μm diameter) particulate matter (PM_{10}) increases by 10 μg/m^3.[3] Given the finite resources available to protect health, there is a need to weight different risks and to allocate preventive resources to get the maximum benefit. We present a three-country interdisciplinary assessment of the impact related to air polution on morbidity and mortality. The project has been initiated by WHO Europe as a case study in the framework of the transport environment and health session of the WHO Ministerial Conference on Environment and Health, held in London, UK, in 1999.[7]

National agencies from Austria, France, and Switzerland assessed the external public-health costs of total air pollution and of traffic-related air pollution. The focus on traffic-related air pollution and on economic valuation is based on the argument that traffic creates costs which are not covered by the polluters (the motorists). Such costs cause economic problems, because they are not included in the market price, which leads to a wasting of scarce and important resources (eg, clean air, silence, and clean water). To stop this wastage, the real price should be put on clean air. With the present study, an important part of the external traffic-related costs, namely the negative impacts of traffic-related air pollution on human health were assessed in terms of attributable number of cases. The quantification of the related external costs are summarised in an Organisation for Economic Co-operation and Development (OECD) report. The full project reports are available from WHO (www.who.dk/london99/transport04.htm, accessed Aug 17, 2000).[8–10]

Methods

Design and participants

The impact assessment relies on calculating the attributable number of cases.[11] We extended the methods of Ostro and colleagues to further specify and standardise influential assumptions and decisions.

Cases of morbidity or mortality attributable to air pollution were derived for the health outcomes listed in table 1. Outcomes were ignored if quantitative data were not available, if costing was impossible (eg, valuing decrement in pulmonary function), and to prevent overlapping health measures from causing multiple counting of the same costs (eg, emergency visits were not considered because they were partly included in the hospital admissions).

To assess the effects of air pollution—a complex mixture of pollutants—epidemiological studies use several indicators of exposure, (eg, NO_2, CO, PM_{10}, total suspended particles, SO_2). These pollutants, however, are correlated. Hence, epidemiological studies cannot strictly allocate observed effects to single pollutants. A pollutant-by-pollutant assessment would grossly overestimate the impact. Therefore, we selected only one pollutant to derive the attributable cases. In this context PM_{10} is a useful indicator of several sources of outdoor air pollution such as fossil-fuel combustion.[1]

The model for our calculation, applied to each health outcome, is shown in figure 1.[9] Three data components are required to estimate the number of cases attributed to outdoor air pollution in a given population: the exposure-response function; the frequency of the health outcome (eg, the incidence or the prevalence) and the level of exposure.

The association between outdoor air pollution and health-outcome frequency is usually described with an exposure-response function (or effect estimate) that expresses the relative increase in adverse health for a given increment in air pollution. For each outcome we selected studies from the peer-reviewed literature to derive the exposure-response function and the 95% CI. For inclusion, an adequate study design and published PM_{10} levels were required. Cross-sectional or cohort studies relying on two or three levels of exposure were omitted, as were ecological studies, given their inherent limitations.[12]

The meta-analytical effect estimate was calculated as the variance weighted average across the results of all studies. Studies with low standard errors had, therefore, more weight in the joint estimate. For each health endpoint, the pooled relative risk (upper and lower 95% CI) per 10 $\mu g/m^3$ PM_{10} was given, and adjusted for heterogeneity where needed.

The health-outcome frequencies (mortality, prevalence, incidence, or person-days) may differ across countries; thus, national or European data were used when possible (table 2). For some population frequencies, rates had to be estimated because of limited national coverage (hospital admission rates in France and Switzerland); for others, epidemiological studies were the only source (bronchitis incidence from the Adventist Health and Smog Study,[13] which has also been used by Ostro and colleagues[14]). In these cases, common baseline frequencies were used for all three countries.

Annual mean outdoor PM_{10} had to be determined on a continuous scale. Therefore, the attributable number of cases depend on: the assumed exposure reference level and the population distribution—ie, the number of people living at the respective level of exposure. We divided exposure into categories of 5 $\mu g/m^3$, (>5–10 $\mu g/m^3$, >10–15 $\mu g/m^3$, and so on). 7·5 $\mu g/m^3$ (the mean of the category 5–10 $\mu g/m^3$) was used as the lowest assessed value. Thus, the health impact of air-pollution exposure below 7·5 $\mu g/m^3$ was ignored. Although there is no evidence for any threshold, there are also no studies available where participants were exposed to PM_{10} below 5–10 $\mu g/m^3$ (annual mean). Furthermore, this reference level[10] also includes the natural background PM_{10}. To derive the population exposure distribution, annual mean concentrations of PM_{10} were modelled for each country at a spatial resolution of 1 km^2 (in France it was 4 km^2).[10]

In each country, the best available data from 1996 were used. The Australia PM_{10} models mostly used total suspended particulate and NOx concentration data with source-specific factors to estimate the exposure.[10] The French PM_{10} exposure models were based on the data from a few sites for PM_{10} and an extensive monitoring network for black smoke, a measurement method for fine particulate matter from combustion sources. PM_{10} emission registries were available only for Switzerland, enabling the adoption of emission-based dispersion models which considered primary particulates, secondary particles formed in the atmosphere from precursor emissions, and transboundary large-scale PM. With this emission-based model, we calculated PM_{10} distributions for total PM_{10} and traffic-related PM_{10} directly.[10] Models were validated by measurements of PM_{10} and the chemical composition of particulates at several locations. For all three countries, results of the emission-based Swiss study were used to derive the traffic-related fraction of PM_{10} for Austria with measurement data on the chemical composition of PM and international modelling.[10]

Combining PM_{10} maps for 1996 (annual mean) with information on the place of residence of the population on a km^2 grid, annual mean exposure distributions for the population were derived. For short-term health effects, the use of annual mean implies that the short-term (daily) PM_{10} corresponds, on average, to the annual mean.

Assumptions and estimations

To take account of inherent uncertainty in the impact assessment, two principles have been adopted. First, for the main assumptions an "at least" approach was applied on each step—ie, methods were selected to obtain an impact which may be expected to be "at least" attributable to air pollution. Second, the uncertainties in the effect estimates were quantified and the results were given as a range (95% CI of the exposure-response function). The sensitivity of the impact estimates will be discussed both qualitatively and quantitatively for the main assumptions.

Derivation of attributable number of cases

Using the exposure-response functions, expressed as relative risk (RR) per 10 $\mu g/m^3$, and the health frequency per 1000 000 inhabitants, we calculated for each health outcome the attributable number of cases (D_{10}) for an increase of 10 $\mu g/m^3$ PM_{10} (figure 1), as: $D_{10}=(RR-1)*P_0$ where P_0 is the health frequency, given an exposure E_0 and RR is the mean exposure-response function across

Health outcome	Definition	Source of exposure-response function	Source of population frequency
Long-term mortality (adults ⩾30 years)	Death rate, excluding violent death or accidents, >30 years	Dockery DW et al 1993[4] Pope CA et al 1995[5]	National death certificate statistics for 1996 (Switzerland 1995)
Respiratory hospital admissions (all ages)	ICD9 460–519 ICD9 466, 480–487, 493, 490–492, 494–496 ICD9 480–487, 490–496	Spix C et al 1998[30] Wordley J et al 1997[31] Prescott GJ et al 1998[32]	National hospital statistics for 1996
Cardiovascular hospital admissions (all ages)	ICD9 410–436 ICD9 390–459 ICD9 390–459 ICD9 410–414, 426–429, 434–440	Wordley J et al 1997[31] Poloniecki JD et al 1997[33] Medina S et al 1997[34] Prescott GJ et al 1998[32]	National hospital statistics for 1996
Chronic-bronchitis incidence (adults ⩾25 years)	Symptoms of cough and/or sputum production on most days, for at least 3 months per year, and for 2 years or more, age ⩾25 years	Abbey DE et al 1993[35]	Abbey DE et al 1993[35]
Bronchitis episodes (children <15 years)	Bronchitis in past 12 months (parents or guardian's answer), ages 10–12, 8–12, and 6–15 years, respectively	Dockery DW et al 1989[36] Dockery DW et al 1996[37] Braun-Fahrländer C et al 1997[38]	Studnicka M et al 1997[39] Oberfeld G et al 1996 (Austria)[40] SCARPOL* (unpublished data, Switzerland, France)
Restricted activity days (adults ⩾20 years)	Any days where respondent was forced to alter normal activity, due to respiratory disease ICD9 460–466, 470–474, 480–486, 510–516, 519, and 783, age 20–65 years	Ostro B et al 1990[41]	Social Insurance Statistics Report 1997 (Austria) SAPALDIA† (unpublished data, Switzerland, France)
Asthma attacks (children <15 years)	Lower respiratory symptoms, age 6–12 years Asthma, age 7–15 years Lower respiratory symptoms, age 7–13 years	Roemer W et al 1993[42] Segala C et al 1998[43] Gielen MH et al 1997[44]	Eder W et al 1998[45]; Haidinger et al 1998[46]; SCARPOL* (unpublished data, joint estimate)
Asthma attacks (adults ⩾15 years)	Wheeze, age 18–80 years Shortness of breath, age 18–55 years Wheeze, age 16–70 years	Dusseldorp A et al 1996[47] Hiltermann TJN et al 1998[48] Neukirch F et al 1998[49]	ECRHS‡ (unpublished data); SAPALDIA† (unpublished data, joint estimate)

*Swiss study on childhood allergy and repiratory symptoms to air pollution. †Swiss study on air pollution and lung disease in adults. ‡European Community respiratory health survey. ICD9=International Classification of Diseases, ninth revision.

Table 1: **Health outcome definition and sources of data**

the studies used (table 1). The exposure-response functions are usually log-linear. For small risks and across limited ranges of exposure log-linear and linear functions would provide very similar results. However, if one may apply the method to populations with very large exposure ranges, the impact may be seriously overestimated on the log-linear scale. Thus, we derived the attributable number of cases (D in figure 1) on an additive scale. The calculation of D_{10} requires, however, prior definition of P_0. We defined P_0 as the health outcome frequency that one may expect, given the reference level of exposure, E_0 (7·5 μg/m³ annual mean PM_{10}). We derived P_0 from P_E, applying the risk function backwards from P_E (the current population mean exposure), to P_0 (chosen to be 7·5 μg/m³). This procedure is in line with the prudent "at least" approach. With D_{10} and the number of people living in each category of exposure, the total number of cases attributable to air pollution can be calculated.

Results

Table 2 summarises for each health outcome the effect estimates, the country-specific health-outcome frequencies at E_0, and the respective number of cases attributed to a 10 μg/m³ increase in PM_{10} (D_{10}). The population exposure distribution is summarised in table 3.

The mean exposure for the population was similar in all three countries with somewhat higher exposures in Austria. Using the Swiss emission-dispersion models for PM, we estimated that the traffic share of the total PM_{10} exposure depended on the mean concentration, ranging from 28% at an annual mean PM_{10} of 10–15 μg/m³, and increasing up to 58% in areas where the total annual mean PM_{10} concentrations were above 40 μg/m³.[10] For Austria, somewhat lower relative contributions of traffic to PM_{10} were obtained.[10]

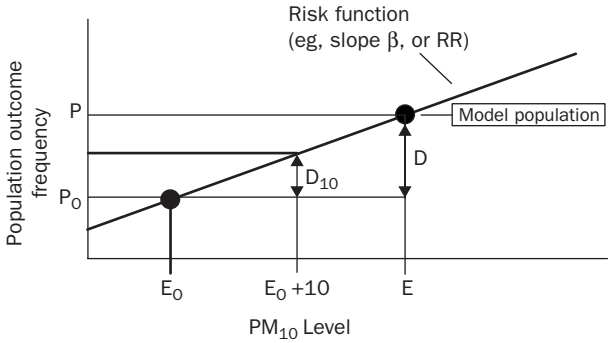
The traffic-related proportion of the total cases attributable to air pollution corresponded to the traffic-related fraction of PM_{10}, amounting to 43% in Austria, 56% in France, and 53% in Switzerland (table 4).

Discussion

By contrast with other projects,[14–17] which each used different assumptions, our results are comparable across the participating countries. Because the whole population is exposed the attributable proportion is substantial, even though epidemiological studies indicate that air pollution imposes on the individual a small risk for morbidity and mortality. The public-health impact, however, depends not only on the relative risk but also on the exposure distribution in the population. Our assessment assigned about 6% of annual deaths to outdoor air pollution.

As a major source of both primary PM emissions and precursors of secondary particulate matter, traffic substantially contributes to the overall impact of outdoor air pollution (table 3). This high contribution is also because the emission densities of traffic are, on average, highest in highly populated areas.

By contrast with directly countable events, which can be listed in national health statistics (eg, deaths or injuries due to traffic accidents), it is not possible to



Model to derive number of cases attributable to air pollution
Based on exposure-response function (slope or relative risks, RR, from epidemiological studies), population frequency of the outcome, P (ie, prevalence, incidence, or number of days), and respective level of exposure, E. We assume reference exposure level (E_0). Health effects below this level are ignored. P_0 is expected outcome frequency, given exposure level E_0. D_{10} is number of cases attributed to increase in exposure by 10 units (10 μg/m³ PM_{10}). D is total number of cases attributed to air pollution for model population.

Health outcome	Effect estimate relative risk (95% CI)	Health outome frequency per million inhabitants per year			Attrbutable number of cases per 10 μg/m³ PM_{10} (D_{10}) and 1 000 000 inhabitants ($D_{10lower}$-$D_{10upper}$ based on 95% CI estimates)		
		Austria	France	Switzerland	Austria	France	Switzerland
Total mortality (adults >30 years, excluding violent death)	1·043 (1·026–1·061)	9330	8390	8260	370 (230–520)	340 (210–480)	340 (200–470)
Respiratory hospital admission (all ages)	1·013 (1·001–1·025)	17 830	11 550	10 300	230 (20–430)	150 (20–280)	130 (10–250)
Cardiovascular hospital admission (all ages)	1·013 (1·007–1·019)	36 790	17 270	24 640	450 (230–670)	210 (110–320)	300 (160–450)
Chronic-bronchitis incidence (adults ≥25 years)	1·098 (1·009–1·194)	4990	4660	5010	410 (40–820)	390 (40–780)	430 (40–860)
Bronchitis episodes (children <15 years)	1·306 (1·135–1·502)	16 370	23 530	21 550	3200 (1410–5770)	4830 (2130–8730)	4620 (2040–8350)
Restricted activity days (adults ≥20 years)*	1·094 (1·079–1·502)	2 597 300	3 221 200	3 373 000	208 400 (175 400–241 800)	263 700 (222 000–306 000)	281 000 (236 500–326 000)
Asthma attacks (children <15 years)†	1·044 (1·027–1·062)	56 700	62 800	57 500	2330 (1430–3230)	2600 (1600–3620)	2400 (1480–3340)
Asthma attacks (adults ≥15 years)†	1·039 (1·019–1·059)	173 400	169 500	172 900	6280 (3060–9560)	6190 (3020–9430)	6370 (3100–9700)

*Total person-days per year. †Total person-days per year with asthma attacks.

Table 2: **Health outcomes with relative risks, outcome frequency, and attributable cases (per 10 μg/m³ and 1 million population)**

directly identify 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 specifically linked to air pollution only. Therefore, uncertainty remains an inherent characteristic of any attempt to derive attributable cases. We prudently dealt with uncertainty, deriving the number of cases "at least" attributable to air pollution. We did not include all health outcomes associated with ambient air. For mortality, we ignored potential effects on newborn babies or infants.[18] Although infant mortality is low in the countries considered, and thus the number of attributable cases is small, the impact on years of life lost, and therefore the economic valuation, could be considerable.

Restriction of the analysis to PM_{10} underestimates independent effects of air pollution not explained by or correlated with the PM fractions. One example may be ozone, most likely leading to further, as yet unquantified, effects.[16]

The allocation of the impact to specific sources adds to the uncertainty. In fact, source apportionment of outdoor air pollution is rarely done and objective source-specific measures of PM have never been used in the epidemiological studies. Thus, although PM_{10} correlates with various health outcomes, it is not clear whether the exposure-response function from outdoor air pollution related to traffic may differ from the non-traffic effects. However, given that traffic contributes a lot to outdoor air pollution, the observed epidemiological effects may be driven substantially by this source. In fact, combustion-related PMs may contribute to the smaller fraction, such as $PM_{2·5}$, for which some studies show even stronger associations with adverse health.[4] Thus, we consider it unlikely that we overestimated the relative impact of traffic.

Health-outcome frequencies may strongly influence the impact assessment. Whereas for mortality the national sources may be considered accurate, frequency measures of morbidity and data on health-care systems have to be considered estimates with some inherent uncertainties. Our decision to choose national health frequency data, if available, may have reduced the impact of these limitations.

For several reasons, mortality is the most controversial of our outcome measures. We used the cohort-based long-term effect estimates of outdoor air pollution on survival time, whereas short-term time-series-based effects were not included separately.[11] The number of deaths attributed to air pollution would be about 4–5 times smaller if the short-term effect estimates had been applied. Our decision, however, was based on methodological grounds. We consider it inappropriate to use short-term studies for the impact assessment of annual mortality.[11] The short-term studies capture only part of the air-pollution-related cases, namely those where exposure and event (death) are closely connected

PM_{10} concentration exposure class (μg/m³)	Population exposure distribution for total PM_{10}			Population exposure distribution for PM_{10} without traffic-attributable fraction		
	Austria	France	Switzerland	Austria	France	Switzerland
0–5	0	0·2%	0	0	0·4%	0·1%
>5–10	0	0·5%	1·2%	9·5%	3·3%	5·1%
>10–15	11·4%	5·2%	5·7%	21·9%	52·0%	59·6%
>15–20	14·2%	31·5%	31·8%	32·7%	41·8%	35·0%
>20–25	22·8%	33·3%	42·5%	23·5%	1·9%	0·2%
>25–30	27·7%	12·8%	14·6%	5·2%	0·5%	0
>30–35	8·5%	7·8%	3·0%	3·3%	0·1%	0
>35–40	4·7%	4·1%	0·9%	2·1%	0	0
>40	10·7%	4·6%	0·3%	1·9%	0	0
Mean*	26·0	23·5	21·4	18·0	14·6	14·0

*Population weighted mean. In considered range of exposure (ie, above 7·5 μg/m³) traffic-related PM_{10} was: Austria 43·2%, France 55·6%, Switzerland 53·2%.

Table 3: **Population exposure distribution of PM_{10}**

Health outcomes	Estimated attributable number of cases or days (95% CI)					
	Total outdoor air pollution (PM_{10})			Traffic-related air pollution (PM_{10})		
	Austria	France	Switzerland	Austria	France	Switzerland
Long-term mortality (adults ⩾30 years)	5600 (3400–7800)	31 700 (19 200–44 400)	3300 (2000–4700)	2400 (1500–3400)	17 600 (10 700–24 700)	1800 (1100–2500)
Respiratory hospital admissions (all ages)	3400 (400–6500)	13 800 (1400–26 300)	1308 (140–2500)	1500 (160–2800)	7700 (800–14 600)	700 (70–1300)
Cardiovascular hospital admissions (all ages)	6700 (3500–10 000)	19 800 (10 400–29 400)	3000 (1500–4400)	2900 (1500–4300)	11 000 (5800–16 300)	1600 (800–2400)
Chronic-bronchitis incidence (adults >25 years)	6200 (600–12 000)	36 700 (3300–73 100)	4200 (370–8400)	2700 (240–5300)	20 400 (1800–40 700)	2300 (200–4500)
Bronchitis (children < 15 years)	48 000 (21 000–86 000)	450 000 (198 500–813 600)	45 000 (20 000–82 000)	21 000 (9000–37 000)	250 000 (110 000–453 000)	24 000 (11 000–44 000)
Restricted activity days in adults ⩾20 years (in millions)	3·1 (2·6–3·6)	24·6 (20·7–28·5)	2·8 (2·4–3·2)	1·3 (1·1–1·6)	13·7 (11·5–15·9)	1·5 (1·2–1·7)
Asthmatics: asthma attacks (children <15 years)	35 000 (21 000–48 000)	243 000 (149 000–337 000)	24 000 (15 000–33 000)	15 000 (9000–21 000)	135 000 (83 000–188 000)	13 000 (8000–17 000)
Asthmatics: asthma attacks (adults ⩾15 years, person days)	94 000 (46 000–143 000)	577 000 (281 000–879 000)	63 000 (30 000–95 000)	40 000 (20 000–62 000)	321 000 (155 000–489 000)	33 000 (16 000–51 000)

Table 4: **Number of attributable cases to air pollution, and cases attributed to traffic pollution**

in time. Most importantly, however, reduced life-expectancy, due to long-term morbidity enhanced by air pollution, may not be captured in the time series; thus, the time-series-based impact assessment would be incomplete.[9] Cohort studies, by design, compare person-time, thus, time to death, across different levels of exposure. Therefore, our calculation based on cohort studies captures both the short-term effects and the long-term effects. Unfortunately, we had to rely on only two US studies, studies which were partly confirmed by a third US study.[20] Preliminary results of the French mortality follow-up of the PAARC study[21] in general support the USA findings.

Because the cohort studies published the numbers of deaths rather than lifetime lost, we primarily calculated the attributable number of deaths. In the economic valuation of death, however, assumptions about age structure of those affected, may be influential.[22] Both from morbidity and short-term mortality studies air pollution may be mostly related to cardiopulmonary disease, including lung cancer. This subgroup tends to die, on average, at older ages than all other non-violent causes of death. For example, in 1995, the mean age at death due to cardiopulmonary causes in Switzerland was 80 years, whereas the mean age of all other non-violent causes of death was 72 years.[23] As a consequence, life expectancy is shortened by about 6 months per 10 μg/m³ increment in PM_{10}.[8] With this assumption, the lifetime lost among those affected by air pollution is shorter than for other non-violent causes—for example, the average victim of traffic accidents is much younger (<40 years) than those who die as a result of air pollution. Therefore, the lifetime lost because of traffic accidents is longer, although our study attributes about twice as many deaths to air pollution than to accidents.[7] On the other hand, there is increasing evidence that air pollution may also influence mortality rates of newborn babies or infants. As we did not quantify attributable number of deaths below age 30 years, we might have underestimated life time lost.

For chronic bronchitis, our assessment relies on one study.[13] The advantage of the study is the reporting of effects of PM on the incidence of chronic bronchitis among a population with very low rates of smoking. This measure was particularly useful for the economic valuation and had been used before.[14] It is of note, however, that the Swiss SAPALDIA study[24] corroborates the US findings, showing increased prevalence of chronic bronchitis symptoms also in Europe, as shown before in the USA.

The selection of a common exposure-response function assumes that the effects of outdoor air pollution, characterised by PM_{10}, are quantitatively identical across countries. We base this assumption on the consistency of epidemiological results observed

Sensitivity Criteria and assumptions	Air pollution attributable number of cases		Sensitivity (ie, alternative result in % of the main point estimates)	
	Total	Due to traffic	Total	Due to traffic
Point estimate of this study	3300	1800	100%	100%
Quantified uncertainty (meta-analytic exposure-response estimate)				
Lower 95% CI value	2000	1100	60%	60%
Upper 95% CI value	4700	2500	140%	140%
PM_{10} population exposure distribution from 1993 model[10]	3200	2000	96%	114%
Higher traffic share: assume PM_{10} without traffic to be 10·7 μg/m³ insteaad of 14·0 μg/m³	3300	2600	100%	145%
Exposure reference value (E_0): 0 instead of 7·5 μg/m³*	5100	1800	154%	100%
Only impact of exposure >15 μg/m³ annual mean (instead of 7 μg/m³)†	1500	1500	46%	86%
Only impact of exposure >20 μg/m³ annual mean (instead of 7 μg/m³)†	400	400	10%	19%

*To simplify the example, we assume no traffic share below 7·5 μg/m³. †In this example, exposure>15 μg/m³ and >20 μg/m³, respectively, are assumed to originate from traffic only. Health impact due to exposure between 0–15 μg/m³ and 0–20 μg/m³, respectively, are omitted.

Table 5: **Sensitivity of the estimation of air pollution attributable number of cases of death (mortality >30 years of age) compared with the quantified uncertainty (95% CI of epidemiological risk estimates), based on Swiss data only**

across many countries—eg, for conditions such as bronchitis among children or hospital admissions. Therefore, we included some health-outcome measures which have not been specifically investigated in Austria, France, or Switzerland, such as long-term mortality and restricted-activity days, where we relied on US studies. The short-term association of PM and daily mortality are consistent between the USA and Europe. We have assumed consistency in the long-term effects as well.

Apart from the variability of epidemiological exposure-response estimates (95% CI), we did not quantify other sources of uncertainty such as, errors in the population exposure distribution, in the derivation of traffic-related fractions, or in the estimation of health-outcome frequencies. Simulations of multiple probability distributions may, however, erroneously suggest a level of precision in assessing uncertainty that cannot be achieved. It may also distract from the fact that basic assumptions such as our underlying "at least" approach are most influential. Furthermore, no data were available on the precision of population exposure estimates as this was the first time that tri-national exposure distributions have been derived for PM_{10}. However, in the framework of a previous Swiss impact assessment project PM_{10} exposure distributions have also been estimated, using different methods and input data (ie, ambient concentrations rather than emission data).[10] Given the availability of these Swiss data, we made some estimates of the sensitivity of the Swiss results for changes in the main assumptions. As shown in table 5 for "death", the population exposure distribution model from 1996, based on 1993 PM_{10} measurements, is of little influence on the total impact estimates. The alternative results fall well within the ±40% range of the indicated uncertainty, based on the 95% CI of the relative risk estimate. However, the assumed exposure reference value of 7·5 $\mu g/m^3$ is influential: the impact estimates would be some 54% higher if the exposure impact were to be quantified from zero. The importance of the reference value is further demonstrated in the last two rows of table 5. In Switzerland, the public-health impact of exposure levels >15 $\mu g/m^3$, corresponding to the reference value used in a 1996 US assessment,[25] would make up only 46% of our results. Only 10% of our impact estimates can be attributed to exposures above the Swiss target clean air value (20 $\mu g/m^3$ annual mean). This rather crude sensitivity analysis shows the importance of underlying methodological assumptions. Last but not least, in case of morbidity outcomes, estimates of the health outcome frequency have direct effects on the impact estimates. For example, we used the incidence of chronic bronchitis from a population where smoking is rare, assuming a 50% higher outcome frequency would result in a 50% larger estimate of the attributable cases.

In light of the uncertainties, one could argue that we should abstain from such impact assessment. We disagree. First, there is abundant evidence that current levels of air pollution have adverse health effects, thus the impact cannot be zero.[26] From a public-health perspective it is therefore an ethical consequence to estimate and communicate the impact to the public. Second, societies have to make important decisions at the time. To abstain from impact assessment, given the many uncertainties, would promote decisions without consideration of aspects of public health. This is particularly true for environmentally sensitive decisions. Third, we consider the participation of epidemiologists and other sciences in this interdisciplinary process of impact assessment as crucial.[27]

Attributable cases are commonly interpreted as the preventable fraction, which is meant to be prevented had exposure been removed. Caution, however, is warranted with such an interpretation. First, for long-term effects—the benefit of lower air-pollution levels—would take years to be fully realised.[8] Second, the attributable risk estimate does not take competing risks into account. Removing one risk factor—eg, air pollution—will increase the relative importance and contribution of other risks and causes of morbidity and mortality. Accordingly, it is well known in multicausal diseases that the sum of attributable cases across several risk factors will not add up to 100% but may be larger.[28] Impact measures that take competing risks into account need to be developed.[29] It is to be emphasised that the economic loss of the health impact of air pollution goes beyond the direct costs of medical treatment. Loss of production and consumption as well as intangible costs (pain, suffering) of disease and death have to be taken into account. Several economic valuation methods are available. Details of the economic valuation of this trinational project are presented elsewhere.[8,22]

Even after taking the overall uncertainty of this estimation into account, the project emphasises the need to consider air pollution and traffic-related air pollution as a widespread cause of impaired health. In a century moving toward sustainable development and health, closer collaboration of public health and environmental policies will enhance preventive success. Further development of standardised impact assessment methods is needed to achieve comparability of results, both across projects or countries, and over time, to assess the benefits from clean air strategies.

Contributors

Nino Künzil wrote the paper, and contributed to the methodological development of the project. Reinhard Kaiser wrote parts of the epidemiology report, and did the metatanalytic calculations. Sylvia Medina collected the French epidemiological data and contributed to the development of a common methodology. Michael Studnicka coordinated the epidemiological assessment for Austria and contributed to the development of the common interdisciplinary methodology. Fritz Horak Jr established the contact to national epidemiology and registries and developed a trinational common methodology. Olivier Chanel and Jean-Christophe Vergnaud contributed to the development of the economic methods and provided the French economy calculations. They also contributed to the tri-national synthesis. Paul Filliger led the air pollution exposure modelling for Switzerland. Max Herry devised the common methods of the economic valuation and provided data and results for the Austrian economic assessment and the tri-national synthesis. Valérie Puybonnieux-Texier helped calculate the French exposure distribution estimates. Philippe Quénel made major contributions to the development of the epidemiological methods. Juergen Schneider did the modelling and estimation of the population exposure distribution of the Austrian population. Rita Seethaler was the project coordinator, and participated in the economic research on external cost modelling. Heini Sommer did the final economic calculations and was the project leader.

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References

1 Wilson R, Spengler J. Particles in our air: concentrations and health effects. Boston: Harvard University Press, 1996.
2 Holgate S, Samet J, Koren H, Maynard R. Air pollution and health. San Diego/London: Academic Press, 1999.
3 Katsouyanni K, Touloumi G, Spix C. Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from times series data from the APHEA project. *BMJ* 1997; **314:** 1658–63.
4 Dockery D, Pope A, Xu X, et al. An association between air pollution and mortality in six US cities. *N Engl J Med* 1993; **329:** 1753–59.
5 Pope A, Thun M, Namboodiri M, et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Am J Respir Crit Care Med* 1995; **151:** 669–74.
6 Bates D. Health indices of the adverse effects of air pollution: the question of coherence. *Environ Res* 1992; **59:** 336–49.
7 Dora C. A different route to health: implications of transport policies. *BMJ* 1999; **318:** 1686–89.
8 Sommer H, Chanel O, Vergnaud JCh, Herry M, Sedlak N, Seethaler R. Monetary valuation of road traffic related air pollution: health costs due to road traffic-related air pollution: an impact assessment project of Austria, France and Switzerland third WHO Ministerial Conference of Environment & Health. London: WHO, 1999.
9 Künzli N, Kaiser R, Medina S, Studnicka M, Oberfeld G, Horak F Jr. Health costs due to road traffic-related air pollution: an impact assessment project of Austria, France and Switzerland. (Air pollution attributable cases. Technical report on epidemiology.) Switzerland: Federal Department for Environment, Energy and Communications Bureau for Transport Studies, 1999.
10 Filliger P, Puybonnieux-Texier V, Schneider J. PM_{10} population exposure technical report on air pollution: health costs due to road traffic-related air pollution: an impact assessment project of Austria, France and Switzerland. London: WHO, 1999.
11 Ostro B, Sanchez J, Aranda C, Eskeland G. Air pollution and mortality: results from a study of Santiago, Chile. *J Exp Anal Environ Epidemiol* 1996; **6:** 97–114.
12 Künzli N, Tager I. The semi-individual study in air pollution epidemiology: a valid design as compared to ecologic studies. *Environ Health Perspect* 1997; **105:** 1078–83.
13 Abbey D, Hwang B, Burchette R. Estimated long-term ambient concentrations of PM_{10} and development of respiratory symptoms in a nonsmoking population. *Arch Environ Health* 1995; **50:** 139–52.
14 Ostro B, Chesnut L. Assessing the health benefits of reducing particulate matter air pollution in the United States. *Environ Res* 1998; **76:** 94–106.
15 National Research Council. Research priorities for airborne particulate matter II: evaluating research progress and updating the portfolio. Washington DC: National Academy Press, 1999.
16 Department of Health Ad-hoc group. Economic appraisal of the health effects of air pollution. London: HM Stationery Office, 1999.
17 American Lung Association. Dollars and cents: the economic and health benefits of potential particulate reductions in the United States. New York: American Lung Association, 1998.
18 Brunekreef B. Air pollution kills babies. *Epidemiology* 1999; **10:** 661–62.
19 McMichael A, Anderson H, Brunekreef B, Cohen A. Inappropriate use of daily mortality analyses to estimate longer-term mortality effects of air pollution. *Int J Epidemiol* 1998; **27:** 450–53.
20 Abbey D, Nishino N, McDonnel W, et al. Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *Am J Respir Crit Care Med* 1999; **159:** 373–82.
21 Baldi I, Roussillon C, Filleul L, et al. Effect of air pollution on long-term mortality: description of mortality rates in relation to pollutants levels in the French PAARC Study. *Eur Respir J* 1999; **24** (suppl 30): S392.
22 Sommer H, Künzli, N, Seethaler R, et al. Health costs due to Road Traffic-related Air Pollution: an impact assessment project of Austria, France and Switzerland. New York: OECD-Report (in press).
23 Annual Statistics Report for Switzerland (Statistisches Jahrbuch). Zürich: Verlag Nene Zürcher (NZZ) 1998.
24 Zemp E, Elsasser S, Schindler C, et al. Long-term ambient air pollution and chronic respiratory symptoms (SAPALDIA). *Am J Respir Crit Care Med* 1999; **159:** 1257–66.
25 National Resources Defense Council, (NRDC). Breath taking: premature mortality due to particulate air pollution in 239 American cities. San Francisco: NRDC, 1996.
26 Bates DV. Lines that connect: assessing the causality inference in the case of particulate pollution. *Env Health Perspect* 2000; **108:** 91–92.
27 Samet J, Schnatter R, Gibb H. Invited commentary: epidemiology and risk assessment. *Am J Epidemiol* 1998; **148:** 929–36.
28 Smith K, Corvalan C, Kjellström T. How much global ill health is attributable to environmental facators? *Epidemiology* 1999; **10:** 573–84.
29 La D, Hardy R. Potential gains in life expectancy or years of potential life lost: impact of competing risks of death. *Int J Epidemiol* 1999; **28:** 894–98.
30 Zmirou D, Schawartz J, Saez M, et al. Time-series analysis of air pollution and cause-specific mortality. *Epidemiology* 1998; **9:** 495–503.
31 Wordley J, Walters S, Ayres J. Short term variations in hospital admissions and mortality and particulate air pollution. *Occup Environ Health* 1997; **54:** 108–16.
32 Prescott GJ, Cohen GR, Elton RA, Fowkes FG, Agium RM. Urban air pollution and cardiopulmonary ill health: a 14·5 year time series study. *Occup Environ Med* 1998; **55:** 697–704.
33 Poloniecki J, Atkinson R, Ponce de Leon A, Anderson H. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Health* 1997; **54:** 535–40.
34 Medina S, Le Terte A, Dusseux E. Evaluation des Risques de la Pollution Urbaine sur lar Santé (ERPURS). Analyse des liens à court terme entre pollution atmoshérique et santé: resultats 1991–95. Paris: Conseil Regional d'lle de France, 1997.
35 Abbey D, Petersen F, Mills P, Beeson W. Long-term ambient concentrations of total suspended particulates, ozone, and sulfur dioxide and respiratory symptoms in a nonsmoking population. *Arch Environ Health* 1993; **48:** 33–46.
36 Dockery D, Speizer F, Stram D, Ware J, Spengler J, Ferris BJ. Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis* 1989; **139:** 587–94.
37 Dockery D, Cunningham J, Damokosh A, et al. Health Effects of acid aerosols on north American Children: respiratory symptoms. *Env Health Perspect* 1996; **104:** 500–05.
38 Braun-Fahrländer C, Vuille J, Sennhauser F, et al. Respiratory health and long-term exposure to air pollutants in Swiss Schoolchildren. *Am J Respir Crit Care Med* 1997; **155:** 1042–49.
39 Studnicka M, Hackl E, Pischinger J. Traffic-related NO_2 and the prevalence of asthma and respiratory symptoms in seven year olds. *Eur Respir J* 1997; **10:** 2257–58.
40 Oberfeld G, König Ch. Erste Salzburger Kindergesund-heiltsuntersuchung, Hrsg 96. Salzburg, Amt der Salzburger Landesregierung, 1996.
41 Ostro B. Associations between morbidity and alternative measures of particulate matter. *Risk Analysis* 1990; **10:** 421–27.
42 Roemer W, Hoek G, Brunekreef B. Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. *Am Rev Respir Dis* 1993; **147:** 118–24.
43 Ségala C, Fauroux B, Just J. Short-term effect of winter air pollution on respiratory health of asthmatic children in Paris. *Eur Respir J* 1998; **11:** 677–85.
44 Gielen M, van der Zee S, van Wijnen J. Acute effects of summer air pollution on respiratory health of asthmatic children. *Am J Respir Crit Care Med* 1997; **155:** 2105–08.
45 Eder W, Gamper A, Oberfeld G, Riedler J. Prävalenz und Schweregrad von Asthma bronchiale, allergischer Rhinitis und atopischer Dermatitis bei Salzburger Schulkindern. *Wien Klin Wochenschr* 1998; **110:** 669–77.
46 Haidinger G, Waldhör T, Süss G, Vutuc Ch. Schluβbericht der ISAAC-Studie Oberösterreich 1995–97. Wien: Linz, 1998.
47 Dusseldorp A, Kruize H, Brunekreef B. Associations of PM_{10} and airborne iron with respiratory health of adults living near a steel factory. *Am J Respir Crit Care Med* 1995; **152:** 1032–39.
48 Hiltermann T, Stolk J, van der Zee S. Asthma severity and susceptibility to air pollution. *Eur Respir J* 1998; **11:** 686–93.
49 Neukirch F, Ségala C, Le Moullec Y. Short-term effects of low-level winter pollution on respiratory health of asthmatic adults. *Arch Environ Health* 1998; **53:** 320–28.