Risk Analysis, Vol. 21, No. 5, 2001

Air Pollution and Mortality: Quantification and Valuation of Years of Life Lost

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To analyze the loss of life expectancy (LLE) due to air pollution and the associated social cost, a dynamic model was developed that took into account the decrease of risk after the termination of an exposure to pollution. A key parameter was the time constant for the decrease of risk, for which estimates from studies of smoking were used. A sensitivity analysis showed that the precise value of the time constant(s) was not critical for the resulting LLE. An interesting aspect of the model was that the relation between population total LLE and $PM_{2.5}$ concentration was numerically almost indistinguishable from a straight line, even though the functional dependence was nonlinear. This essentially linear behavior implies that the detailed history of a change in concentration does not matter, except for the effects of discounting. This model was used to correct the data of the largest study of chronic mortality for variations in past exposure, performed by Pope *et al.* in 1995; the correction factor was shown to depend on assumptions about the relative toxicity of the components of $PM_{2.5}$. In the European Union, an increment of 1 $\mu g/m^3$ of $PM_{2.5}$ for 1 year implies an average LLE of 0.22 days per person. With regard to the social cost of an air pollution pulse, it was found that for typical discount rates (3% to 8% real) the cost was reduced by a factor of about 0.4 to 0.6 relative to the case with zero discount rate, if the value of a life year was taken as given; if the value of a life year was calculated from the "value of statistical life" by assuming the latter as a series of discounted annual values, the cost varied by at most $\pm 20\%$ relative to the case with zero discount rate. To assess the uncertainties, this study also examined how the LLE depended on the demographics (mortality and age pyramid) of a population, and how it would change if the relative risk varied with age, in the manner suggested by smoking studies. These points were found to have a relatively small effect (compared to the epidemiological uncertainties) on the calculated LLE.

KEY WORDS: Air pollution; mortality; monetary valuation; value of life; discounting; particulate matter; life expectancy; years of life lost

1. INTRODUCTION

To guide environmental policy, it is desirable to know how large the damages caused by air pollution are, both in physical and monetary terms. In recent years, several major studies in the United States (Oak Ridge National Laboratory and Resources for the Future, 1994; Rowe *et al.*, 1995) and in the European Union (ExternE, 1995; ExternE, 1998; for a short summary, see Rabl & Spadaro, 2000) have provided damage estimates; they all highlight the importance of mortality due to air pollution.

It has also been recognized that the full impact of air pollution on health is much larger than what can

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0272-4332/01/1000-0843$16.00/1

be measured by the easy and widely used time series epidemiology (which observes only acute impacts, i.e., impacts that are observable within a few days after exposure to air pollution). Unfortunately it is very difficult and costly to measure the total impacts (short plus long term), and there are only a few long-term studies available. However, in recent years, three important epidemiological studies examined the long-term impacts; two of these (Dockery *et al.*, 1993; Pope *et al.*, 1995) found positive correlations between exposure to particles and total mortality (also called chronic mortality in contrast to the acute mortality measured by time series studies); whereas the third (Abbey *et al.*, 1999) found a positive correlation with mortality for men, but not for women.

The results of such studies are expressed as a factor (the relative risk) that indicates how much the age-specific mortality rate increases. To quantify the corresponding loss of life expectancy (LLE) requires a fairly complicated analysis using life table data for the age distribution and mortality rates of a population (Brunekreef, 1997; ExternE, 1998; Rabl, 1998). The LLE calculations carried out so far have been limited to steady-state conditions and have not accounted for the time delay between exposure and death; thus each year of life lost (YOLL) was counted equally regardless of when or at what age it was incurred. The purpose of the present study was to extend the calculation of damage and cost to realistic nonsteady-state conditions, to include discounting and the variation of the value of a YOLL with age.

Because the results of the three chronic mortality studies assumed, implicitly, steady-state conditions, it is necessary to introduce a model for the variation of the relative risk with time after exposure to a pollution pulse. To do so, we invoked similarities between air pollution and smoking, both of which involve pollutants from the combustion of organic matter and are correlated with similar cardiovascular and pulmonary diseases, as well as cancers. Extensive studies have been carried out to measure the health benefits that smokers may expect after they stop smoking. It appears that after cessation of smoking, the body has the ability to repair much of the damage; and that the repair processes can be characterized in terms of several time constants ranging from hours to over 10 years. Only the longest time constants have an appreciable impact on the LLE due to air pollution. We, therefore, present a detailed determination of the time constant(s) that can be inferred from the literature. Whereas a meta-analysis by Lightwood and Glantz (1997) suggested a time constant in the range of 1 to 2 years for acute myocardial infarction and stroke, the long-term study of smoking by Doll, Peto, Wheatley, Gray, and Sutherland (1994) implied a time constant of about 13 years for total mortality. Because these studies involved different end points, we based our model on a weighted average of two time constants: 1.5 years with weight 0.3, and 13 year with weight 0.7.

Knowledge of the time constant(s) is also necessary to answer a question that has been overlooked by the LLE calculations thus far: how should the pollutant concentrations used by Pope *et al.* (1995; the study with the largest cohort by far, which used a time-independent analysis) be modified to account for the fact that the exposures were much higher in the past? Because exposure data do not extend sufficiently far into the past, we used U.S. average emissions data (available since 1900) and assumed proportionality between average emissions and concentrations to estimate a correction factor.

When we examined the relation between LLE and incremental $PM_{2.5}$ concentration implied by Pope *et al.*, we found that it was remarkably close to a straight line numerically, even though the functional dependence was nonlinear; the deviation from linearity was a small fraction of a percent for typical concentrations in the United States or the European Union. This essentially linear behavior implied that the detailed history of a change in concentration did not matter (except for the effects of discounting). Thus, one could express the resulting LLE in terms of a single number, the slope of the dose–response function for LLE as function of exposure. Of course, for concentrations of $PM_{2.5}$ lower than the lowest ones in Pope *et al.* (1995), the form of the dose–response function is uncertain—in particular, whether there is a no-effects threshold. However, that does not affect the LLE, due to an incremental exposure above the lowest concentrations in Pope *et al.* (1995).

This study also investigated several additional questions:

- What is the sensitivity of the results to the demographic characteristics of a population?
- How do the results change if the relative risk varies with age, in the manner suggested for smokers by the data of Doll *et al.* (1994)?
- How do the results change if one includes discounting and a possible variation of the value of a YOLL with age?
- What is the contribution of the various uncertainties of the calculation to the uncertainty of the result?

2. LIFE EXPECTANCY

A key element for the analysis is the age-specific mortality rate $\mu(x')$. It is defined such that someone who has reached age x' has a probability $\mu(x')\,\Delta x'$ of dying between x' and $x' + \Delta x'$, the limit of $\Delta x' \to 0$ being, of course, understood. The survival function $S(x,x')$ is the fraction of a cohort of age x that survives at least to age x'. Because the fraction that dies between x' and $x' + \Delta x'$ is $\Delta S(x,x') = S(x,x')\,\mu(x')\,\Delta x'$, one gets the differential equation

$$dS(x,x') = -S(x,x')\,\mu(x')\,dx. \tag{1}$$

The boundary condition is $S(x,x) = 1$. One readily finds the solution

$$S(x,x') = \exp\left[-\int_x^{x'} \mu(x'')dx''\right] \tag{2}$$

for $x' > x$. $S(x,x')\,\mu(x')$ is the probability distribution for a member of the age x cohort to survive to and die at age x'; it is normalized to unity over the interval from x to ∞. The expected age of death is, therefore, the integral of $x'\,S(x,x')\,\mu(x')$ from x to ∞. The difference between the expected age of death and the starting age x is the remaining life expectancy $L(x)$ of this cohort,

$$L(x) = \int_x^\infty x'\,S(x,x')\,\mu(x')\,dx' - x. \tag{3}$$

For practical calculations this formula is awkward, because it converges too slowly as the upper bound approaches infinity. It is preferable to first integrate by parts and obtain the simpler formula

$$L(x) = \int_x^\infty S(x,x')\,dx'. \tag{4}$$

Convergence is improved radically because the slowly converging part is contained in the term x' $S(x,x')$ evaluated between $x' = x$ and $x' = \infty$, which yields x, exactly canceling the first term in Equation 3.

For calculations with life tables, one approximates the integral by a sum over 1-year intervals,

$$L(x) = S(x,x+1) + S(x,x+2)\ldots. \tag{5}$$

In this article we instead used an analytical approach, based on the observation that with remarkable accuracy and for all populations, the mortality above age 30 can be represented by the Gompertz model,

$$\mu(x') = \alpha \exp(\beta x'), \tag{6}$$

where α and β are constants whose precise values depend on the population under study. Fits to data for several populations are given in Table I. A plot of $\ln[\mu(x')]$ versus x' is thus a straight line for $x' > 30$, as can be seen in Fig. 1 for the United States. For many countries (e.g., the United States), the total mortality between ages 30 and 45 is noticeably above the Gompertz line of the older ages, due to violent deaths and AIDS.

For Fig. 1 and Table I we removed deaths due to accidents, homicides, and suicides for the United States, according to data of Peters, Kochanek, and Murphy (1998), shown in Table II. A correction factor of $r_{nat} = 1/0.975 = 1.026$ (last line of Table II) is relevant for the interpretation of the results of Pope *et al.* (1995) because these authors stated the risk increase in terms of total mortality, rather than mortality from natural causes. Incidentally, inclusion of the 30- to 35-

Table I. Coefficients α and β of the Gompertz Model for Several Populations

Population	α	β	λ
US, natural causes, male + female[a]	5.38E-05	8.78E-02	0.62E-03[f]
US, natural causes, male[a]	7.76E-05	8.59E-02	0.63E-03[f]
US, natural causes, female[a]	3.19E-05	9.20E-02	0.59E-03[f]
EU15, all causes, male + female[b]	3.70E-05	9.24E-02	0.59E-03
Sweden, natural causes, male + female[c]	9.67E-06	1.10E-01	0.52E-03[f]
France, all causes, male + female[b]	6.66E-05	8.50E-02	0.64E-03
Russia, all causes, male + female[d]	3.96E-04	6.78E-02	0.83E-03
China, all causes, male + female[e]	5.89E-05	9.15E-02	0.47E-03

Note: The last column shows the coefficient λ for the loss of life expectancy of Equation (32), in years of life lost per person for an exposure to 1 $\mu g/m^3$ during 1 year, as calculated with the real age distribution of each population.

[a] Fit to data above age 30 in Fig. 1

[b] Fit to data above age 30 of Eurostat (1999), for 1995.

[c] Fit to data above age 38 for Sweden, for 1993–1997.

[d] Fit to data ages 37 to 84 for Russia, for 1996.

[e] Fit to data above age 35 for China, for 1998.

[f] Includes correction factor for natural mortality $r_{nat} = 1/0.975$ (see last line of Table II).

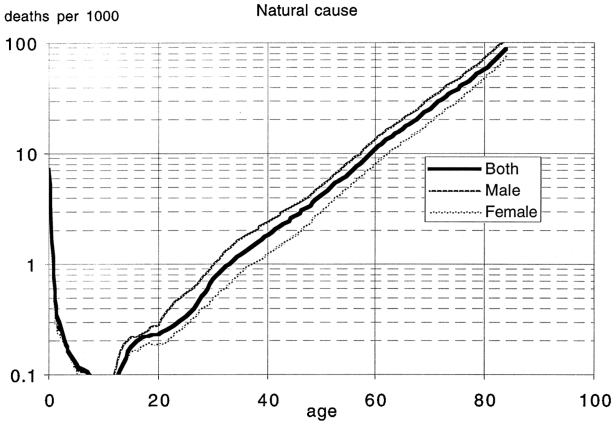

Fig. 1. Plot of mortality rate [deaths per 1,000 per year = 1,000 $\mu(x)$] versus age x for the United States, natural causes only. Based on life table data of the National Center for Health Statistics (1998), after subtracting nonnatural deaths according to Peters *et al.* (1998).

year age group in the average of Table II would not have changed the correction factor appreciably, because the total number of deaths for this age group was very small.

The Gompertz model can yield more accurate results than raw life table data because it is a smooth interpolation based on a large body of data and avoids discretization errors—especially at old age, where data are sparse. With the Gompertz model, the survival fraction for cohorts above 30 years is

$$S(x,x') = \exp\{[\exp(\beta x) - \exp(\beta x')]\,\alpha/\beta\} \qquad (7)$$

for 30 years $< x < x'$. For a stationary population, the death rate m is equal to the birth rate and given by the inverse of the life expectancy at birth,

Table II. Contribution of Natural Causes to Total Mortality above Age 35 in the United States, Rate per 1,000

Age group	All causes	Nonnatural[a]	% Natural
35–44	2.21	0.57	74.1
45–54	4.46	0.51	88.5
55–64	10.94	0.50	95.4
65–74	25.38	0.65	97.4
75–84	58.03	1.25	97.8
Over 85	153.27	2.99	98.0
Sum	254.30	6.48	97.5

Note: Data from Peters, Kochanek, and Murphy, 1998.
[a] Accidents, homicides, and suicides.

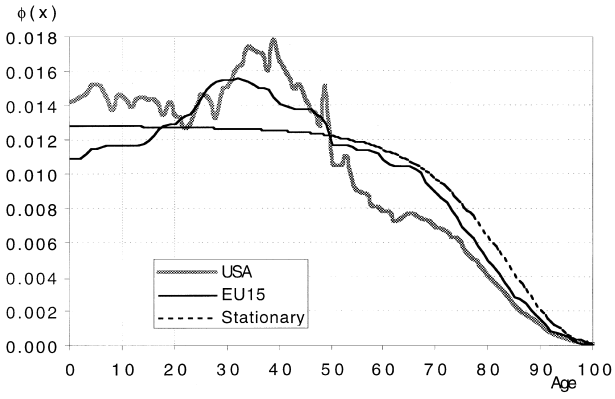

Fig. 2. The age distribution $\phi(x)$ for the European Union (EU15) for 1997 (Eurostat, 1999), the United States (USA) for 1996 (U.S. Department of Commerce, 1997), and a stationary population with total mortality of EU15.

$$m = 1/L(0), \qquad (8)$$

and the age distribution $\phi(x)$, defined such that $\phi(x)\,\Delta x$ is the fraction of the population between ages x and $x + \Delta x$, is

$$\phi_s(x) = S(0,x)/L(0). \qquad (9)$$

This is because a fraction m of the population is born each year and, of that cohort, a fraction $S(0,x)$ survives to age x. The age distributions for the United States, the European Union, and a stationary population are plotted in Fig. 2.

3. CONCENTRATION, EXPOSURE, AND RELATIVE RISK

In view of the repair processes of the body, it is appropriate to define the exposure $E(x)$ of a person at age x as the time integral of the concentrations $c(x')$ to which the person has been exposed since birth, weighted by a decay factor. Because exponential decay is the most natural choice, we define exposure as

$$E(x) = \int_0^x c(x') \exp\,[-(x - x'/\tau]\,dx', \qquad (10)$$

with time constant τ (Leksell, 2000).[3] This equation, and all that are derived from it, can readily be generalized to a set of repair processes with time constants $\{\tau_i\}$ and weights $\{w_i\}$, by making the replacement

$$\exp[-(x - x')/\tau)] \rightarrow \sum_i w_i \exp[-(x - x')/\tau_i)]. \qquad (11)$$

[3] Note that in this reference the roles of x and x' are interchanged compared with the present article.

Because this model, and the determination of the time constant(s) in Section 4, are somewhat speculative, we evaluate the sensitivity to this choice by varying the time constant(s). As it turns out, the uncertainty due to the choice of the time constant(s) is so small (see Fig. 6) as to be minor compared with the uncertainties of the epidemiological studies.

A case of special interest is the steady-state situation of constant concentration $c(x') = c_s$, for which one finds that

$$E_s(x) = c_s\,\tau[1 - \exp(-x/\tau)] \tag{12}$$

for a person of age x. For the applications in this article, τ is less than 15 years, whereas x is at least 30 years (and the excess mortality due to air pollution is small below 50 years). Therefore, we approximate the steady-state exposure by its $x/\tau \to \infty$ limit,

$$E_s = c_s\tau. \tag{13}$$

Another interesting special case is the exposure $E_p(t)$ due to a pulse of constant concentration c_p lasting from $t = 0$ to $t = t_1$, the concentration being zero before and after. Because the exposure due to a pulse is the same for all people who are alive during the pulse, we indicate only time t and not age x as a variable for $E_p(t)$. For such a pulse, the exposure first increases toward the asymptotic value $c_p\,\tau$, reaching a peak at t_1,

$$E_p(t) = c_p\,\tau[1 - \exp(-t/\tau)], \tag{14}$$

for $0 < t < t_1$, and then falls exponentially from its peak $E_p(t_1)$ back to zero,

$$E_p(t) = E_p(t_1)\exp[-(t - t_1)/\tau, \tag{15}$$

for $t > t_1$. If the pulse duration $t_1 = \Delta t$ is short compared with the time constant τ, this becomes

$$E_p(t) = c_p\,\Delta t\exp(-t/\tau) \tag{16}$$

for $t > 0$. Exposure to air pollution is assumed to increase the baseline age-specific mortality $\mu_0(x)$ by a factor R, called the relative risk:

$$\mu(x) = R\,\mu_0(x). \tag{17}$$

In most of this article, we followed the assumption, made in all the available long-term air pollution mortality studies, that R is independent of age x; however, we relaxed this assumption in Appendix C.

For the relation between exposure and relative risk, all the above-mentioned studies of long-term mortality due to air pollution have used the Cox proportional hazards model, which assumes

$$R(t) = \exp[k_{ER}\,E(t)], \tag{18}$$

with a constant of proportionality k_{ER} determined by maximum likelihood estimation. This model implies that each exposure increment increases the risk in a multiplicative fashion. According to Equation (4), the risk R shortens the life expectancy of a cohort of age x by

$$\Delta L(x) = \int_x^\infty [S_0(x,x') - S(x,x')\,dx', \tag{19}$$

where $S_0(x,x')$ and $S(x,x')$ are the survival functions for the baseline mortality $\mu_0(x)$ and for the increased mortality $\mu(x)$ of Equation (17), respectively. To find the LLE averaged over the entire population, one integrates $\Delta L(x)$ over the age distribution $\phi(x)$ and obtains

$$\Delta L_{av} = \int_{30}^\infty \Delta L(x)\,\phi(x)\,dx. \tag{20}$$

We took the lower limit as 30 years, because the studies of Dockery *et al.* (1993), Pope *et al.* (1995), and Abbey *et al.* (1999) did not consider people below this age. Also, from about 5 to 30 years, the mortality is so low that the effect of pollution appears entirely negligible. In passing, however, we should cite studies that found a significant effect of pollution on infant mortality (Bobak & Leon, 1992; Woodruff, Grillo, & Schoendorf, 1997) and note that the corresponding LLE could be appreciable, because a child who is saved from early death could go on to live a long life.

Previous calculations of LLE due to air pollution concentration used a simple steady-state model, comparing a reference population with a population exposed (Brunekreef, 1997; ExternE, RIA 1998; Rabl, 1998). For steady-state exposure to a constant concentration increment c_s, the risk is

$$R_s = \exp(k_{ER}\,c_s\,\tau). \tag{21}$$

In case of several time constants, this is replaced by

$$R_s = \exp\left(k_{ER}c_s\sum_i w_i\tau_i\right), \tag{22}$$

if the same coefficient k_{ER} applies to all time constants. For the steady-state calculation, the time constant does not matter because only the product,

$$\kappa = k_{ER}\,\tau, \tag{23}$$

is needed; one simply replaces $k_{ER}\,\tau$ with κ, which is determined directly from the relation between R_s and c_s. Inserting R_s into Equations (17) through (20), one obtains the LLE for a lifetime exposure to c_s. To estimate the effect of shorter exposures, Rabl (1998) allocated this loss uniformly over a life span of 75 years. In the following, we perform a more accurate and rigorous calculation by taking into account transient effects.

4. ESTIMATION OF THE TIME CONSTANT

No information on the time constant(s) τ can be gained from the available studies of long-term mortality due to air pollution, because they implicitly assumed steady-state conditions when calculating the relative risk. To obtain an estimate of τ, we therefore looked at another air pollutant from combustion, namely tobacco smoke. Urban air pollution and tobacco smoke both contain fine particles, volatile organic compounds, CO, NOx, and so forth, and both are correlated with cardiovascular and pulmonary diseases, as well as cancers. Of course, we cannot hope for more than a probable range for τ, because the composition of pollutants is different (especially with regard to secondary pollutants such as sulfates and nitrates) and the concentrations are different—although mean concentrations are comparable between air pollution and passive smoking (Dockery & Spengler, 1981). Therefore, the magnitude of this uncertainty was examined by performing a sensitivity analysis.

Lightwood and Glantz (1997) carried out a metastudy based on seven earlier studies of how the excess risks for some diseases decline after a smoker has quit smoking. They looked at two end points: namely, acute myocardial infarction and stroke. The data were fitted to an exponentially declining risk function with time constant τ,

$$R(x) = (R_0 - R_\infty) \exp\left[-(x - x_q)/\tau\right] + R_\infty. \quad (24)$$

Here $R(x)$ is the relative risk as a function of the time $(x - x_q)$ since the age of smoking cessation x_q. R_0 denotes the relative risk at age x_q, and R_∞ is the asymptotic limit of $R(x)$ as $x \to \infty$. $R_0 - R_\infty$ is the excess risk caused by the earlier years of smoking.

Lightwood and Glantz defined one risk function for men and one for women, but saw that the time constant was gender independent. Their results were $\tau = 1.6$ years for acute myocardial infarction and $\tau = 1.4$ years for stroke. In some, but not all, of the seven studies, a tendency toward a slower risk reduction for a higher number of cigarettes smoked per day or a longer duration of smoking before quitting was observed.

In an earlier study (US DHHS, 1990) it appeared that the time constant for recovery increased with age. There may be two explanations for this finding:

1. the self-healing capacity of the human body is reduced at advanced ages, and/or
2. the longer exposure to tobacco smoke for a smoking cessation later in life might slow down the recovery process.

The studies examined by Lightwood and Glantz concerned only end points with time constants shorter than a few years. Because the total risk of smoking includes other end points, we looked at Doll *et al.* (1994) who followed a cohort of 34,000 male medical doctors in the United Kingdom over a period of 40 years. Their study included data on individuals who had quit smoking. Visual inspection of their Fig. 4 suggests a time constant in the range of 10 to 20 years—much longer than those of Lightwood and Glantz. However, on the time scale of the data in Doll *et al.* (1994), only effects with a time constant longer than about 5 years were visible. Our best fit was 13 years, as described in Appendix A.

To combine this result with the information from Lightwood and Glantz (1997), we noted that the end points studied by the latter, namely acute myocardial infarction and stroke, accounted for approximately 0.3 of the total mortality observed by Doll *et al.* (1994; who provided a detailed breakdown of the contributions of each end point). Hence, we were inclined to favor a combination of $\tau_1 = 1.5$ years with weight $w_1 = 0.3$ and $\tau_2 = 13$ years with weight $w_2 = 0.7$. However, we also used the model with a single time constant.

5. CORRECTION FOR PAST EXPOSURE

Of the three long-term mortality studies, the one by Pope *et al.* (1995) had, by far, the largest sample, and thus, we focused on this study's results. In that study, a correlation of the relative risk R and the concentration of $PM_{2.5}$ and of sulfates was found. For the $PM_{2.5}$ correlation, Pope *et al.* used the average ambient concentrations during the period 1979 through 1983; for the sulfate correlation, they used the average ambient concentration during 1980. By using the concentration at one point in time, they assumed, in effect, steady-state conditions. In reality, however, typical exposures in the United States have been decreasing since their peak around 1970, and the data of or around 1980 underestimate the true exposure.

To obtain a better estimate of the factor k_{ER} in Equation (18) for the relation between risk and exposure, we took Equation (21) for the steady-state risk R_s from exposure E_s to a constant concentration c_s, but instead of inserting the concentrations of Pope *et al.* directly, we introduced a correction factor r_e for past exposure:

$$R_s = \exp(k_{ER}\, r_e c_{s79\text{–}83}\, \tau). \quad (25)$$

The correction factor r_e should be calculated as the ratio of the steady-state exposure at the average

value c_{s79-83} from 1979 to 1983, and the real exposure $E(x)$ of Equation (10) for each age x. To do so, one would need concentration data before 1980, which are generally not available. As an alternative, we assumed that concentrations were proportional to emissions, for which inventories were available back to 1900 (Nizich *et al.*, 1997). Whereas proportionality between emissions and concentrations is true in an average sense, it is uncertain in the present case because we have neither a list of the sites studied by Pope *et al.* nor a breakdown of emissions by site. Hence, we simply assumed that the sulfate concentrations were proportional to the total SO_2 emissions in the United States, and that the $PM_{2.5}$ concentrations were proportional to the total particulate emissions from combustion sources in the inventory of Nizich *et al.* (1997).

Thus, we calculated the correction factor r_e by replacing concentrations $c(t)$ by emissions $e(t)$ in Equations (10) and (13). For simplicity, we integrated over all past emissions, even those before the birth of some individuals in the cohorts [this approximation is justified because x/τ 2 for all ages in the cohort, as explained in the text above to Equation (13)]. After a change of variables in Equation (10), we thus obtained the ratio of steady-state exposure and real exposure,

$$r_e = \frac{1}{e_{79-83}\tau}\int_0^\infty e(1985-u)\exp(-u/\tau)du, \quad (26)$$

where e_{79-83} is the average of emissions from 1979 to 1983. We chose 1985 as the reference year for the real exposure in the integral because it was the midpoint of the period over which Pope *et al.* (1995) observed the mortality.

For $PM_{2.5}$ concentrations, further uncertainty arises from the lack of knowledge about the composition of particulate matter (PM). The inventories indicate primary particles, that is, PM as emitted, which is quite different from PM in ambient air. The latter contains sulfates (from SO_2), nitrates (from NO_x), and crustal particles, in addition to primary particles from combustion. Lack of knowledge about the composition of PM and the toxicity of individual components of PM is one of the main sources of uncertainty in any attempt to quantify the health impacts.

In fact, most air quality monitoring stations have measured only the mass, typically as total suspended particles (TSP), PM_{10}, and more recently $PM_{2.5}$, without any information on the composition of the particles. Among components of PM, only sulfates and/or acidity have been measured by a few stations (note that even the category of sulfates is an ambiguous mixture). A few studies with spot measurements are reported by Seinfeld and Pandis (1998), and only recently have more systematic data become available (USEPA, 1997). Of course, epidemiological studies are limited by the data that have been measured by monitoring stations. Nitrates, for example, have not been monitored until recently and, hence, there have been no epidemiological studies on the effects of nitrate aerosols.

In view of this uncertainty, the emissions history for each of the three principal precursor emissions of $PM_{2.5}$—primary combustion particles, SO_2, and NO_x—were plotted in Fig. 3. In addition, a weighted average of these three emissions was plotted, with weights 0.6 for primary combustion particles, 0.35 for SO_2, and 0.05 for NO_x. This choice of weights was based not only on the fact that in the United States the composition of ambient $PM_{2.5}$ is typically 30% to 60% primary combustion particles, 20% to 50% sulfates, and 2% to 20% nitrates (USEPA, 1997), but also on the lack of epidemiological evidence for health effects of nitrates. In any case, other weights were also considered (not shown here). Only the weighting of primary combustion particles had a major effect. From Fig. 3 it can be seen that for the period from 1950 to 1985, the weighted emission was reduced relatively less than the PM_{10} emission.

Results for r_e are shown in Table III, for a wide range of assumptions. r_e depends on the assumed time constant τ and, for $PM_{2.5}$, on the assumed weighting factors for the precursors. For the weighting of precursor emissions, the highest values of r_e correspond to counting only PM_{10}, and the lowest values of r_e correspond to counting only SO_2. The latter is relevant if one uses the correlation with sulfates of Pope *et al.* (1995).

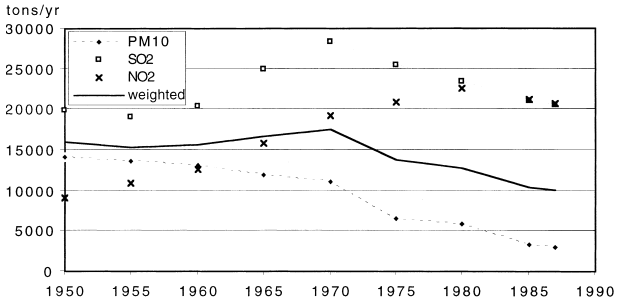

Fig. 3. Emissions, in metric tons, of primary combustion particles (here designated as PM_{10}), SO_2, and NO_x in the United States (data from Nizich *et al.*, 1997). No_x is expressed as NO_2 equivalent; "weighted" is a weighted average with weights 0.6 for PM_{10}, 0.35 for SO_2, and 0.05 for NO_2.

Table III. The Correction Factor r_e for Past Exposure of Equation (26) for Several Values of the Time Constant and for Several Weightings of the Precursor Emissions

τ	SO_2	PM	Weighted
20	1.02	1.80	1.20
15	1.04	1.64	1.18
13	1.06	1.56	1.17
10	1.07	1.41	1.15
5	1.10	1.11	1.10
1.5 (weight 0.3) and 13 (weight 0.7)	1.07	1.53	**1.17**

Note: "SO_2" counts only sulfur emissions; "PM" counts only particulate emissions from combustion; and "weighted" is a weighted average with weights 0.6 for PM from combustion, 0.35 for SO_2, and 0.05 for NO_x. Bold face indicates choice for this paper.

Because we found the "weighted" scenario, with time constants $\tau_1 = 1.5$ years and $\tau_2 = 13$ years, to be the most plausible, we conclude that

$$r_e = 1.17 \text{ (almost certainly between 1.1 and 1.8).} \quad (27)$$

Inserting the numbers for the highest concentration of Pope *et al.* (1995), $R_{smax} = 1.17$ (with 95% confidence interval 1.09 to 1.26) and $c_{smax} = 24.5\ \mu g/m^3$, into Equation (25) for a single time constant $\tau = 13$ years, one finds

$$k_{ER}\,\tau = \ln(R_{smax})/(r_e c_{smax}\,\tau) = 0.000421 \text{ per } (\text{year}\cdot\mu g\cdot m^3) \quad (SE = 0.00010), \quad (28a)$$

whereas the version with two time constants (1.5 years and 13 years) yields

$$k_{ER}, \tau_1\tau_2 = \ln(R_{smax})/[r_e c_{smax}\,(w_1\tau_1 + w_1\tau_2)] = 0.000574 \text{ per } (\text{year}\cdot\mu g\cdot m^3)\ (SE = 0.00014), \quad (28b)$$

all to be applied with data for mortality due to natural causes. The *SE*s were estimated as corresponding to 1/(2*1.96) times the difference between ln(1.26) and ln(1.09).

Because Pope *et al.* (1995) stated their results in terms of total mortality, whereas Table II indicates that the natural-cause mortality is 2.5% lower, we recommend multiplying the calculated LLE by a correction factor of 1/0.975:

$$r_{nat} = 1.026, \quad (29)$$

when used with data for natural cause mortality. When data for cardiopulmonary mortality are available, it seems more appropriate to base the calculations on the risk for cardiopulmonary mortality $R_{smax} = 1.31$ (with 95% confidence interval 1.17–1.46), also for $c_{smax} = 24.5\ \mu g/m^3$.

Of course, whatever time constant or combination of time constants one takes, the choice must be used consistently for the entire calculation of life years lost. Even though k_{ER} varies strongly with τ, only the final result for the years of life lost matters, and, as shown in Fig. 6, it is not very sensitive to the choice of τ.

It might be interesting to carry out the same analysis for the other long-term mortality studies and then to pool the results, with weighting factors inversely proportional to the *SE*s, in the manner of Bellander, Svartengren, Berglind, Staxler, and Järup (1999). We refrained because Abbey *et al.* (1999) involved uncertainties due to the extrapolation from TSP to $PM_{2.5}$ that are difficult to assess, and, in any case, the result would not have been very different, the coefficient of Pope *et al.* (1995) being intermediate between the other studies and its statistical weight much larger.

When applying k_{ER} to regions other than the United States, there is additional uncertainty when the composition of $PM_{2.5}$ is different. In Europe, for example, the ratio of NH_3 over SO_2 and NO_x emissions is about twice as high (Nizich *et al.* 1997), sufficient to neutralize much of the acidity of the $PM_{2.5}$ in ambient air. The uncertainty due to the difference in composition between particles emitted by a source and PM in ambient air hampers any attempt to calculate the damage due to emission of particles.

6. LLE DUE TO INCREMENTAL EXPOSURE

Let $\mu_0(x')$ be the age-specific mortality for the baseline exposure to pollution. When the exposure changes by an increment $E(t)$ for $t > 0$, the relative risk changes from unity to $R(t) = \exp(k_{ER}\,E(t))$ of Equation (18) for $t > 0$ and the mortality changes to

$$\mu_0(x') \rightarrow \mu(x',t) = R(t)\,\mu_0(x') \quad (30)$$

for $t > 0$; it becomes a function of time t and of age x'. When one evaluates the effect on a population by following a cohort that has starting age x at time $t = 0$, age of death x' and time t are related by

$$t = x' - x. \quad (31)$$

The LLE due to a short exposure pulse of concentration c_p and duration Δt is calculated in Appendix B, following the formalism of Sections 2 and 3. Even though the exact result is a nonlinear function of exposure, numerical evaluations show that deviation from linearity is totally negligible for typical ambient

concentrations. Thus, it is sufficient to keep only the first term of a series expansion, with the result

$$\Delta L_{av} = \lambda\, c_p\, \Delta t, \tag{32}$$

with

$$\lambda = k_{ER} \int_{30}^{\infty} dx\ \phi(x) \int_{x}^{\infty} dx, S_0(x,x') \left\{ \int_{x}^{x'} dx''\, \mu_0(x'')\, \exp[-(x'' - x)/\tau] \right\}. \tag{33}$$

Because of the negligible deviation from linearity, one obtains essentially the same loss of life, whether an exposure history is analyzed in terms of integrated exposure or as a series of concentration pulses. This justifies the starting point of our calculations, that is, taking the background mortality rate $\mu_0(x)$ as independent of time and calculating the change due to an incremental exposure $E(t)$ according to Equation (32).

With the two-time constant model and the mortality data in Table I for the EU we find

$$\lambda = 0.592 \times 10^{-3}\ \text{YOLL per (year}\cdot\mu\text{g}\cdot\text{m}^3\text{)} = 0.216\ \text{days of life lost per (year}\cdot\mu\text{g}\cdot\text{m}^3\text{)}. \tag{34}$$

This means, for example, that if a population of 1,000 is exposed to an increment of 1 μg/m^3 of $PM_{2.5}$ for 1 year, the loss is 0.592 YOLL [this is almost exactly the same as used by ExternE (1998) apart from the correction factor $r_e = 1.17$ of Equation (27) for past exposure that had not been included]. For environmental policy in the European Union, a reasonable goal might be to reduce the concentration of $PM_{2.5}$ by about 10 μg/m^3; the corresponding increase in LLE for a lifetime (75 year) exposure would be $0.592 \times 10^{-3} \times 75 \times 10 = 0.44$ YOLL per person on average, with similar numbers for the United States.

The last column of Table I shows the values of λ for the respective populations. To test the sensitivity of the result to details of the age distribution, we repeated the calculation of λ for the mortality of the European Union, but used the stationary age distribution instead of the real one; the result was $\lambda = 0.616 \times 10^{-3}$, about 4% larger.

In the steady-state calculation of Rabl (1998), allocating the steady-state loss uniformly over a 75-year lifetime to get the effect of a 1-year pulse, this loss was estimated as $f \times 0.87 \times 10^{-3}$ YOLL per person per year$\cdot\mu$g$\cdot$m^3 for the United States, where f was the fraction of the population at risk; Rabl estimated f to be approximately 0.5. The present result is about a third higher, an acceptable difference in view of the uncertainty of this factor f and of the approximations involved in a steady-state calculation.

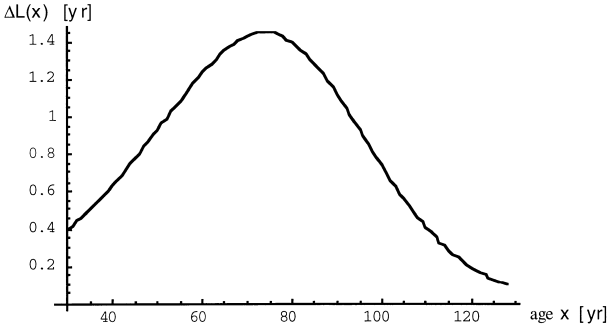

Fig. 4. The loss $\Delta L(x)$ versus age x of exposed cohort, in years of life lost per 1,000 persons for the average population of the EU15 for a 1-year pulse of 1 μg/m^3.

It is interesting to examine which age group loses how much. In Fig. 4 the loss $\Delta L(x)$ for a cohort of age x is plotted versus x. The distribution peaks around 70 years and has a long tail beyond 100 years: for the few individuals who survive to a very old age, the potential loss due to pollution is relatively high. However, when calculating the population average ΔL_{av} of Equation (32), $\Delta L(x)$ is integrated with the age distribution $\phi(x)$ as weighting factor, whereby the contribution of those few very old individuals becomes negligible. The distribution $\phi(x)$ is shown in Fig. 2 for the United States, for the European Union, and for a stationary population with the mortality of the European Union.

A related question is at what age the loss occurs averaged over the population. By changing the order of integration in Equation (33), one can write ΔL_{av} as

$$\Delta L_{av} = \int_{30}^{\infty} dx'\, \Delta L_d(x'), \tag{35}$$

where

$$\Delta L_d(x') = k_{ER} c_p \Delta t \int_{30}^{x'} dx\ \phi(x) S_0(x,x') \left\{ \int_{x}^{x'} dx''\, \mu_0(x'') \exp[-(x'' - x)/\tau] \right\} \tag{36}$$

is the contribution of age x' to the average loss of the population. This function is plotted versus age of death x' in Fig. 5; it peaks around an age of death of 80 to 85 years.

To evaluate the sensitivity to τ, ΔL was plotted versus τ in Fig. 6. Note that k_{ER} itself varies inversely with τ as per Equation (28), but only the effect on ΔL is of concern. This graph shows that the precise value of τ is not critical: between $\tau = 5$ and $\tau = 20$ years, ΔL varies only about 10%.

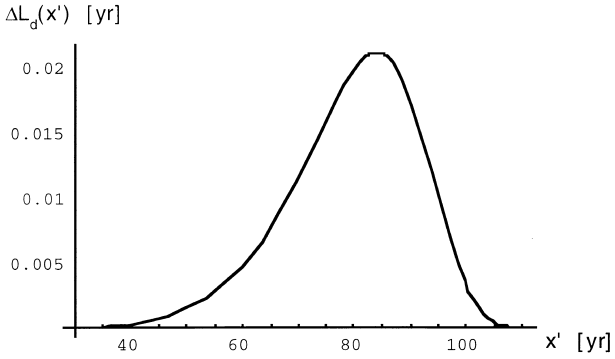

Fig. 5. Loss $\Delta L_d(x')$ versus age of death x' of Equation (36) for a 1-year pulse of 1 μg/m³, in years of life lost per 1,000 persons for the average population of the EU15.

Like all the available studies of long-term mortality due to air pollution, we assumed that the relative risk was independent of age. In Appendix C we relaxed this assumption but found that the effect on the result was not large.

7. MONETARY VALUATION

If the value v_{YOLL} of a YOLL is independent of age and if the discount rate δ is zero, the cost C is simply obtained by multiplying ΔL_{av} of Equation (32) by v_{YOLL}. With nonzero discounting the value of a loss occurring at time t from now has to be multiplied by the discounting factor $\exp(-\delta t)$ to obtain the present value. The time of death is, of course, related to x and x' by $t = x' - x$ as per Equation (31). The variation of the value of a YOLL with age is most conveniently accounted for by taking v_{YOLL} as the average over all ages and by including in the integral a factor $r_{\mathrm{YOLL}}(x')$ such that $v_{\mathrm{YOLL}}\, r_{\mathrm{YOLL}}(x')$ is the value of a YOLL at age x'. $v_{\mathrm{YOLL}}\, r_{\mathrm{YOLL}}(x')$ may be looked on as the value of a quality-adjusted life year at age x and may be assumed to decline at old age. With these modifications of Equations (32) and (33), one finds that the cost C associated with a pollution pulse $c_p\, \Delta t$ is

$$C = k_{ER} c_p \Delta t\, v_{\mathrm{YOLL}} \int_{30}^{\infty} dx' \phi(x) \int_{x}^{\infty} dx' r_{\mathrm{YOLL}}(x') \exp[-\delta(x' - x)] S_0(x, x') \left\{ \int_{x}^{x'} dx'' \mu_0(x'') \exp[-(x'' - x)/\tau] \right\} \quad (37)$$

The effect of the discount rate δ is shown in Figs. 7 and 8 where C, normalized by its value for $\delta = 0$, is plotted versus δ, for the case where $r_{\mathrm{YOLL}}(x') = 1$, that is, the value of a YOLL does not vary with age x'. For Fig. 7, we have taken v_{YOLL} as given and independent of δ. However, until recently v_{YOLL} had to be derived on theoretical grounds as the prorated annual equivalent of VSL, the so-called value of statistical life VSL (for which a better designation would be "willingness to pay for reducing the risk of premature death"), because there have been essentially no studies that determined v_{YOLL} directly (except for Johannesson & Johannesson, 1996); hence, the uncertainty of the monetary valuation is very large. In its simplest form, the postulated relation between VSL and v_{YOLL} is

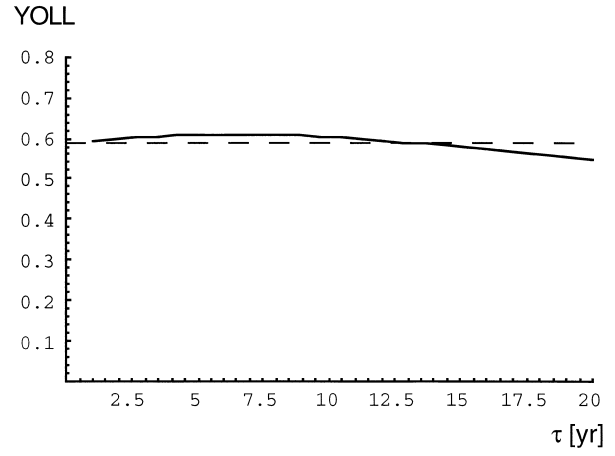

Fig. 6. Variation of ΔL with τ in a model with one-time constant, for a 1-year pulse of 1 μg/m³, in years of life lost (YOLL) per 1,000 persons for the average population of the EU15. Dashed line shows the result for the model with two-time constants (1.5 and 13 years).

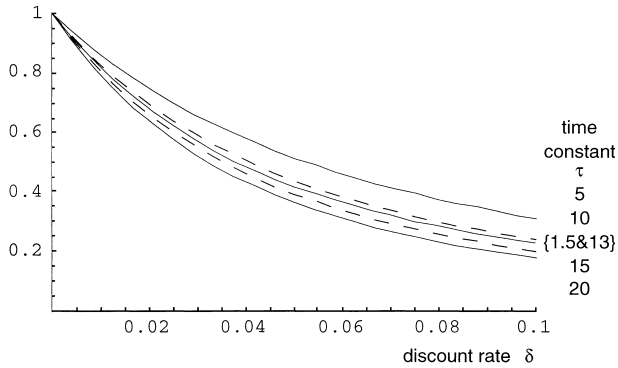

Fig. 7. The cost C of a concentration pulse, Equation (37), as a function of discount rate, for the case in which v_{YOLL} is given (independent of δ). Curves are shown for models with one- and with two-time constants, as indicated; each curve is normalized by its value at $\delta = 0$.

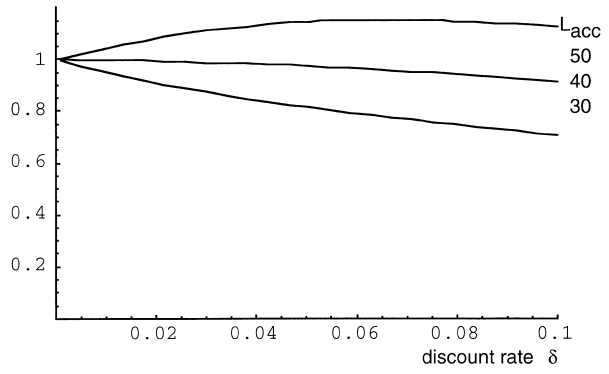

Fig. 8. The cost C of a concentration pulse, Equation (37), as a function of discount rate, for the case in which value of statistical life is given (independent of δ). Curves are shown for the model with τ_1 = 1.5 years and τ_2 = 13 years, and for three plausible values of L_{acc} in Equation (38); each curve is normalized by its value at δ = 0.

$$\text{VSL} = v_{\text{YOLL}}\left\{\frac{1}{(1+\delta)} + \frac{1}{(1+\delta)^2} + \ldots + \frac{1}{(1+\delta)^{L_{acc}}}\right\}, \quad (38)$$

where L_{acc} = average LLE due to accidents, because VSL studies are based on accidental deaths. A more rigorous version would include in the ith term the probability of surviving to age i, the sum going to infinity. If VSL, rather than v_{YOLL}, is taken as given and independent of δ, the variation of the cost C with δ is at most ±20% as can be seen in Fig. 8: the variation of v_{YOLL} with δ implied by Equation (38) roughly compensates the variation in Fig. 7.

As for the variation of the value of a YOLL with age, the available data suggest that $v(x)$ might peak around 30 years and then decrease with age. The effect on the cost C is similar to the effect of the factor $r_{\text{age}}(x)$ of Equation (C3) for age dependence of risk; it can increase or decrease C slightly, depending on the precise form of $r_{\text{YOLL}}(x)$.

8. CONCLUSIONS

To analyze the LLE due to air pollution, we developed a dynamic model, in contrast to the simple steady-state calculations of previous investigations. The dynamic model allowed us to include discounting in a calculation of social costs of air pollution. We found that for typical discount rates (3% to 8% real), the cost was reduced by a factor of about 0.4 to 0.6, relative to the case with zero discount rate, if the value of a life year was taken as given. However, if the value of a life year was calculated from the "value of statistical life" by assuming the latter as a series of discounted annual values, the cost varied by at most ±20%, relative to the case with zero discount rate.

The dynamic model was also needed to correct the data of the largest study of chronic mortality (Pope *et al.*, 1995) for variations in past exposure. We found that the dose–response function coefficient of Pope *et al.* should be divided by a correction factor r_e, the precise value of which depends on assumptions about the relative toxicity of the constituents of $PM_{2.5}$ in ambient air. If only the mass concentration matters, the correction factor is r_e = 1.17, but it could be as low as 1.1 if only sulfates are harmful, or as high as 1.8 if only primary combustion particles are harmful.

A key parameter is the time constant for the decrease of risk after the termination of an exposure to pollution. Because air pollution epidemiology does not provide any information on this point, we estimated the time constant from studies of smoking. Our model involves a combination of a time constant of 1.5 years (with weight 0.3) and one of 13 years (with weight 0.7). We evaluated the sensitivity of the results to the choice of the time constant(s) and found that the uncertainty due to the time constant determination amounts to only a few percent, as far as LLE is concerned; the uncertainty due to the time constant was somewhat larger for the social cost with typical discount rates, but still small compared with the uncertainties of the epidemiology.

To assess another source of uncertainty, we examined also how the results changed if the relative risk varied with age, in the manner suggested by the data of Doll *et al.* (1994). The effect on the LLE was on the order of 10%.

An important finding was that the relation between population total LLE and $PM_{2.5}$ concentration was numerically almost indistinguishable from a straight line, even though the functional dependence was nonlinear. This essentially linear behavior implies that the detailed history of a change in concentration does not matter (except for the effects of discounting). Thus, one can express the result for LLE as product of exposure (= time integral of concentration increment) and a coefficient whose precise value depends only on the demographic characteristic of the population in question; we provided results for the United States, the European Union, France, Sweden, Russia, and China. In the European Union, an increment of 1 μg/m^3 of $PM_{2.5}$ for 1 year implies an average LLE of 0.22 days per person; the number for

the United States is similar, but for Russia it is about 40% higher and for China it is about 25% lower.

ACKNOWLEDGMENTS

This work was supported, in part, by the ExternE Project of the European Commission DG Research. The authors are grateful to Dr. Olivier Chanel, Prof. V. Demin, Prof. Anil Markandya, and Prof. Pan Zi Qiang for providing population data.

APPENDIX A. DETERMINATION OF TIME CONSTANT FOR THE DATA OF DOLL *ET AL.* (1994)

To determine the time constant for the data of Doll *et al* (1994), we adapted the formalism of Section 3, in particular Equations (14) and (15) for a pulse of constant concentration c_p from $t = 0$ to $t = t_1$. With the multiplicative model of Equation (18), the corresponding risk R is given by

$$\ln[R(t)] = k_{ER}\, c_p\, \tau[1 - \exp(-t/\tau)] \tag{A1}$$

for $0 < t < t_1$. Defining $R(\infty)$ as the asymptotic limit for infinitely long exposure, this can be written as

$$\ln[R(t)] = \ln[R(\infty)]\,[1 - \exp(-t/\tau)] \tag{A2}$$

for $0 < t < t_1$ For a smoker who smokes at a constant rate, starting at age $x_{st} = 18$ and quitting at age x_q, this equation implies that the risk $R_{sm}(x)$ at age x is given by

$$\ln[R_{sm}(x)] = \ln[R_{sm}(\infty)]\,\{1 - \exp[x - x_{st})/\tau]\} \tag{A3}$$

for $x_{st} < x < x_q$. After quitting at age x_q, the risk becomes, by analogy with Equation (15),

$$\ln[R_q(x)] = [R_{sm}(\infty)]\,\{1 - \exp[-(x_q - x_{st})/\tau]\}\, \exp[-(x - x_q)/\tau \tag{A4}$$

for $x > x_q$. With these relative risks, the mortality is

$$\mu_{sm}(x) = R_{sm}(x)\, \mu_0(x) \tag{A5}$$

for $x_{st} < x < x_q$ for a smoker, and

$$\mu_q(x) = R_q(x)\, \mu_0(x) \tag{A6}$$

for $x > x_q$ for a quitter, μ_0 being the mortality for a nonsmoker. The corresponding survival functions are

$$S_{sm}(x_{st},x) = \exp\left[-\int_{x_{st}}^{x} \mu_{sm}(x')\, dx \right] \tag{A7}$$

for $x_{st} < x < x_q$, and

$$S_q(x_{st},x) = S_{sm}(x_{st},x_q)\, \exp\left[-\int_{x_q}^{x} R_q(x) \cdot \mu_0(x')\, dx' \right] \tag{A8}$$

for $x > x_q$. The survival function for nonsmokers is $S_0(x_{st},x)$

$$S_0(x_{st},x) = \exp\left[-\int_{x_{st}}^{x} \mu_0(x')\, dx' \right] \tag{A9}$$

for $x > x_{st}$. To find τ, we evaluate the quotient

$$q(x) = \frac{S_q(x_{st},x) - S_{sm}(x_{st},x)}{S_0(x_{st},x) - S_{sm}(x_{st},x)} \tag{A10}$$

as a function of τ and for several values of x (60, 70, and 85 years), using the Gompertz model with coefficients $\alpha = 0.73\text{E}10\text{-}5$ and $\beta = 0.114$ estimated from the survival curves in Doll *et al.* (1994) for the British male doctors. $q(x)$ is a measure of how much the survival probability of a quitter approaches that of a nonsmoker. Comparing the calculated q(x) with the data of Doll *et al.*, we obtain the best fit with $\tau = 13$ years.

APPENDIX B. CALCULATION OF LLE DUE TO AN EXPOSURE PULSE

The new age-specific mortality rate of Equation (30) implies a new survival fraction $S(x,x')$. By analogy with Equation (1) it is determined by the differential equation

$$dS(x,x') = -S(x,x')\, R(t)\, \mu_0(x')\, dx', \tag{B1}$$

with $t = x' - x$ for a cohort that has age x at the start $t = 0$ of the incremental exposure. With boundary condition $S(x,x) = 1$, the solution is

$$S(x,x') = \exp\left[-\int_{x}^{x'} R(x'' - x)\mu_0(x'')dx'' \right]. \tag{B2}$$

It will be convenient to replace R by $1 + (R - 1)$ and rewrite the survival function in terms of the survival function $S_0(x,x')$ for the baseline exposure

$$S(x,x') = S_0(x,x') \exp\left\{ \int_{x}^{x'} [R(x''-x) - 1]\mu_0(x'')dx'' \right\}. \tag{B3}$$

Recalling Equation (19) for the loss of life expectancy (LLE) of a cohort of age x exposed to the risk R

$$\Delta L(x) = \int_{x}^{\infty} [S_0(x,x') - S(x,x')]\, dx',$$

we obtain

$$\Delta L(x) = \int_x^\infty dx' S_0(x,x') \left(1 - \exp\left\{\int_x^{x'} Rx'' - x - 1\,\mu_0 x'' dx''\right\}\right). \quad \text{(B4)}$$

The average loss of the population is then obtained by integrating over the age distribution $\phi(x)$, as in Equation (20),

$$\Delta L_{av} = \int_x^\infty \Delta L(x)\, \phi(x)\, dx. \quad \text{(B5)}$$

Note that the units of Equations (20) and (B5) are years of life lost per person because we take the integral of $\phi(x)$ from 0 to ∞ as normalized to unity.

Let us now consider the short exposure pulse of Equation (16),

$$E_p(t) = c_p\, \Delta t \exp(-t/\tau)$$

for $t > 0$, and the corresponding risk of Equation (18)

$$R_p(t) = \exp[k_{ER} E_p(t)].$$

Even though Equation (B5) for the resulting loss of life expectancy ΔL_{av} is not a linear function of $k_{ER}\, c_p\, \Delta t$, the deviation from linearity is remarkably small, except at extremely high concentrations where ΔL_{av} levels off at the life expectancy. Even for c_p = 1,000 μg/m^3 lasting $\Delta t = 1$ year, the exact result of Equation (B5) is within 4% of the linear extrapolation of its $c_p \to 0$ limit. For typical concentrations in Europe and North America, the deviation from linearity is a small fraction of a percent—totally negligible. Therefore, we expand Equation (B5) in powers of $k_{ER}\, c_p$, beginning with the replacement of $R(x'' - x) - 1$ by

$$R_p(x'' - x) - 1 = k_{ER}\, c_p\, \Delta t \exp[-(x'' + x)/\tau] \quad \text{(B6)}$$

for a pulse with $k_{ER}\, c_p\, \Delta_p\, \Delta t << 1$, the pulse occurring at time $t = x'' - x = 0$. Continuing by expanding the exponential in Equation (B4) for $c_p\, \Delta t \to 0$, one obtains the LLE due to an exposure pulse $c_p\, \Delta t$,

$$\Delta L_{av} = \lambda\, c_p\, \Delta t, \quad \text{(B7)}$$

with

$$\lambda = k_{ER} \int_{30}^\infty dx\, \phi(x) \int_x^\infty dx' S_0(x,x') \left\{\int_x^{x'} dx'' \mu_0(x'') \exp[-(x'' - x)/\tau]\right\}. \quad \text{(B8)}$$

APPENDIX C. ADJUSTMENT FOR AGE DEPENDENCE OF RELATIVE RISK

All the studies of long-term mortality assumed that the relative risk is independent of age; in other words, that the mortality $\mu(x,c)$ in a region with concentration c is

$$\mu(x,c) = \mu_0(x) \exp(k_{ER}\, c\, \tau), \quad \text{(C1)}$$

where $\mu_0(x)$ = baseline mortality. However, Fig. 5 of Doll *et al.* (1994) for the age dependence of relative risk of smokers suggests that the mortality risk of air pollution may vary also with age. It is interesting to examine how such variation of risk with age would change our results. Obviously, we do not have the means to redo the analysis of the long-term mortality studies, but as a simple shortcut we can assume that the age dependence for air pollution is the same as for smoking. Thus, we replace Equation (C1) with

$$\mu^*(x,c) = \mu_0(x) = \exp[k_{ER}{}^* r_{\text{age}}(x)\, c\, \tau], \quad \text{(C2)}$$

where $r_{\text{age}}(x)$ is the function that describes the variation of risk with age and $k_{ER}{}^*$ is the new value of the coefficient. Averaging over the curve for 1951 to 1971 and the one for 1971 to 1991 in Fig. 5 of Doll *et al.*, the relative risk R is approximately constant at 2.4 below age 60 and decreases in approximately linear fashion, reaching 1.3 at 90; for the entire cohort, Table V of Doll *et al.* implies a relative risk of 1.78. Thus, we assume $r_{\text{age}}(x)$ to have the value 2.4/1.78 = 1.35 below age 60 and to decrease with slope $(1.3 - 2.4)/(1.78 \times 30) = 0.021$ above 60:

$$r_{age}(x) = \begin{cases} 1.35 \text{ (for } x < 60) \\ 1.35 - 0.021\ (x - 60) \text{ (for } x > 60). \end{cases} \quad \text{(C3)}$$

This function crosses unity at age 77. We also tested whether this variation of risk with age affects the estimation of the time constant by including it in Equation A3. The value of τ increases somewhat, but the fit becomes less good.

To determine $k_{ER}{}^*$, we demand that the distribution of deaths be as similar as possible between $\mu(x,c)$ and $\mu^*(x,c)$, in the sense of least squares. Specifically, we consider a dataset for the number of deaths at different ages x_i that would be observed in a cohort of a long-term mortality study without age dependence, and we compare it with an analogous set in which the relative risk varies according to Equation (C2). If the age distribution in the cohort is $\phi(x)$, we thus have the set $[\phi(x_i)\ \mu(x_i,c)]$ for Equation (C1) and the set $[\phi(x_i)\ \mu^*(x_i,c)]$ for Equation (C2) where x_i is uniformly spaced. Now we adjust $k_{ER}{}^*$ to minimize the sum of squared differences between these two sets; this determines $k_{ER}{}^*$ as the solution of

$$0 = \frac{d}{dk_{ER}^*} \sum_i \phi(x_i)\mu_0(x_i)(e^{k_{ER}^* c\tau} - e^{k_{ER}^* r_{age}(x_i) c\tau})^2. \quad \text{(C4)}$$

Because the arguments of the exponential functions are small, one can expand and keep just the lowest order

$$0 = \frac{d}{dk_{ER}^*} \sum_i \{\phi(x_i)\mu_0(x_i)[k_{ER} c\tau - k_{ER}^* r_{\text{age}}(x_i) c\tau]\}^2 \quad \text{(C5)}$$

The solution is

$$k_{ER}^* = k_{ER} \frac{\sum_i [\phi(x_i)\mu_0(x_i)]^2 r_{age}(x_i)}{\sum_i [\phi(x_i)\mu_0(x_i) r_{age}(x_i)]^2}. \quad \text{(C6)}$$

For the summation we take 1-year intervals in x_i. Because there are uncertainties concerning the age distribution $\phi(x)$ and the normalization of the function $r_{\text{age}}(x)$, we evaluated several plausible possibilities. We conclude that the correction factor k_{ER}^*/k_{ER} is in the range

$$k_{ER}^*/k_{ER} = 0.9 \text{ to } 1.1. \quad \text{(C7)}$$

When k_{ER} in the equations of this article is replaced by $k_{ER}^* r_{\text{age}}(x)$, the mortality $\mu_0(x'')$ in Equation (33) is multiplied by $r_{\text{age}}(x)$, and ΔL_{av} increases by about 10%. $r_{\text{age}}(x)$ gives more weight to ages below 77 and less to ages above; on balance there is a slight increase. To sum up this section, the variation of risk with age increases the uncertainty, but does not introduce a very large systematic error.

APPENDIX D: NOMENCLATURE

$c(t)$ = concentration at time t.
C = mortality cost due to air pollution pulse.
$E(t)$ = exposure at time t; Equation (10).
EU15 = European Union with 15 members.
k_{ER} = coefficient in relation between exposure and risk.
$L(x)$ = remaining life expectancy for someone who has already reached age x.
LLE = loss of life expectancy.
m = total death rate = $1/L(0)$.
PM_d = particulate matter with diameter less than d in μm.
r_e = correction factor for past exposure, Equations (25) through (29).
r_{age} = correction factor for variation of risk with age.
$r_{\text{YOLL}}(x)$ = variation of value of a years of life lost (YOLL) with age, relative to average value v_{YOLL}.
r_e = correction factor for past exposure.
$R(t)$ = relative risk at time t; Equations (17) and (18).
$S(x,x')$ = survival fraction (fraction of a cohort of age x that survives to age x').
t = time.
v_{YOLL} = average value of a YOLL.
VSL = value of statistical life.
w_i = weighting factor for time constant τ_i.
x = age.
x' = age of death.
α = coefficient in Gompertz equation.
β = coefficient in Gompertz equation.
δ = discount rate.
Δt = duration of exposure pulse.
ΔL = LLE due to an exposure.
$\phi(x)$ = age pyramid (fraction of population that has age x).
κ = coefficient in relation between exposure and risk.
λ = coefficient in Equation (32) for LLE = LLE for exposure to 1 μg/m³ for 1 year.
$\mu(x_d)$ = age-specific mortality.
τ = time constant.
$_p$ = exposure pulse.
$_s$ = steady-state conditions.

REFERENCES

Abbey, D. E., Nishino, N., McDonnell, W. F., Burchette, R. J., Knutsen, S. F., Beeson, W. L., & Yang, J. X. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *American Journal of Respiratory and Critical Care Medicine, 159*, 373–382.

Bellander, T., Svartengren, M., Berglind, N., Staxler, L., & Järup, L. (1999). *The Stockholm study on health effects of air pollution and their economic consequences. Part II. Particulate matter, nitrogen dioxide, and health effects. Dose-response relations and health consequences in Stockholm county* (publication 1999:160). Stockholm, Sweden: The National Road Administration.

Bobak, M., & Leon, D.A. (1992). Air pollution and infant mortality in the Czech Republic, 1986–88. *Lancet, 340*, 1010–1014.

Brunekreef, B. (1997). Air pollution and life expectancy: Is there a relation? *Occupational and Environmental Medicine, 54*, 781–784.

Dockery, D. W., Pope, C. A., III, Xiping, X., Spengler, J. D., Ware, J. H., Fay, M. E., Ferris, B. G. & Speizer, F. E. (1993). An association between air pollution and mortality in six US cities. *New England Journal of Medicine, 329*, 1753–1759.

Dockery, D. W. & Spengler, J. D. (1981). Personal exposure to respirable particulates and sulfates. *Journal of Air Pollution Control Association, 31*, 153–159.

Doll, R., Peto, R., Wheatley, K., Gray, R., & Sutherland, I. (1994). Mortality in relation to smoking: 40 years' observations on male British doctors. *British Medical Journal, 309*, 901–911.

Eurostat. (1999). *Demographic statistics—data 1960–1999.* Statistical Office of the European Communities. http://europa.eu.int/comm/eurostat/ (October 16, 2001).

ExternE. (1995). *ExternE: Externalities of Energy, JOULE Programme Reports. Vol. 1: Summary* (EUR 16520); *Vol. 2: Methodology* (EUR 16521); *Vol. 3: Coal and Lignite* (EUR 16522); *Vol. 4: Oil and Gas* (EUR 16523); *Vol. 5: Nuclear* (EUR 16524); *Vol. 6: Wind and Hydro Fuel Cycles* (EUR 16525). Office for Official Publications of the European Communities, European Commission, Directorate-General XII, Science, Research and Development. Luxembourg. http://ExternE.jrc.es/publica/html (October 16, 2001).

ExternE. (1998). *ExternE: Externalities of Energy, LOULE Programme Reports. Vol. 7: Methodology 1998 Update* (EUR 19083); *Vol. 8: Global Warming* (EUR 18836); *Vol. 9: Fuel Cycles for Emerging and End-Use Technologies, Transport and Waste* (EUR 18887); *Vol. 10: National Implementation* (EUR 18528). Luxembourg: Office for Official Publications of the European Communities. European Commission, Directorate-General XII, Science Research and Development. http://ExternE.jrc.es/publica.html (October 16, 2001).

Johannesson, M., & Johannesson, P. O. (1996). To be or not to be, that is the question: An empirical study of the WTP for an increased life expectancy at an advanced age. *Journal of Risk and Uncertainty, 13*, 163–174.

Leksell, I. (2000). *Health costs of particle emissions: Economic valuation of increased mortality due to exhaust emissions of fine particles.* Doctoral dissertation, Chalmers University of Technology, Göteborg, Sweden.

Lightwood, J. M., & Glantz, S. A. (1997). Short-term economic and health benefits of smoking cessation: Myocardial infarction and stroke. *Circulation, 96*, 1089–1096.

National Center for Health Statistics. (1998). *Vital statistics of the United States 1995* (Vol. II, Mortality, Part A, Section 6). Hyattsville, MD: Author.

Nizich, S. V., Pierce, T., Pope, A., Carlson, P., & Barnhard, B. (1997). *National air pollutant emission trends, 1900–1996* (Report EPA-454/R-97-011). Washington, DC: U.S. Environmental Protection Agency.

Oak Ridge National Laboratory and Resources for the Future. R. Lee (Ed.). (1994). *External costs and benefits of fuel cycles.* Oak Ridge, TN: Author.

Peters, K. D., Kochanek, K. D., & Murphy, S. L. (1998, November). Deaths: Final data for 1996. National Center for Health Statistics. *National Vital Statistics Reports, 47*(9), 1–99.

Pope, C. A., Thun, M. J., Namboodri, M. M., Dockery, D. W., Evans, J. S., Speizer, F. E., & Heath, C. W. (1995). Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Amererican Journal of Respiratory and Critical Care Medicine, 151*, 669–674.

Rabl, A. (1998). Mortality risks of air pollution: The role of exposure-response functions. *Journal of Hazardous Materials, 61*, 91–98.

Rabl, A., & Spadaro, J. V. (2000). Public health impact of air pollution and implications for the energy system. *Annual Review of Energy and the Environment, 25*, 601–627.

Rowe, R. D., Lang, C. M., Chestnut, L. G., Latimer, D., Rae, D., Bernow, S. M., & White, D. (1995). *The New York electricity externality study.* Dobbs Ferry, NY: Oceana Publications.

Seinfeld, J. H. & Pandis, S. N. (1998). *Atmospheric chemistry and physics: From air pollution to climate change.* New York: John Wiley.

U.S. Department of Commerce (USDOC). (1997). *Statistical abstract of the United States.* Washington, DC: Author.

U.S. Department of Health and Human Services (USDHHS). (1990). *The health benefits of smoking cessation: A report to the Surgeon General* (DHHS Publication No. (CDC) 90-8416). Atlanta, GA: Centers for Disease Control, USDHHS.

U.S. Environmental Protection Agency (USEPA). (1997). *National air quality and emissions trends report, 1997.* http://www.epa.gov/oar/aqtrnd97/chapter2.pdf (October 16, 2001).

Woodruff, T. J., Grillo, J., & Schoendorf, K. C. (1997). The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives, 105*, 608–613.