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Mortality risks of air pollution: the role of exposure–response functions

A. Rabl *

Ecole des Mines, 60 boul. St.-Michel, F-75272 Paris Cedex 06, France

Abstract

A cost–benefit analysis of air pollution control presupposes a policy decision about what is to be valued: number of premature deaths or years of life lost (YOLL)? The difference is more than an order of magnitude. We argue for a YOLL valuation on the grounds of economic rationality. For the implementation there is a difficulty: the relation between E–R (exposure–response) functions for mortality and YOLL is not clear. Whereas, a YOLL calculation needs variations in life expectancy (inverse of population mortality rate), acute mortality E–R functions (based on time-series analysis) report variations in daily death counts, and chronic mortality E–R functions (based on cohort studies) report variations in age-specific mortality. Acute mortality E–R functions carry no information on YOLL, but we try to estimate a typical value based on plausible upper and lower bounds. Chronic mortality E–R functions, by contrast, allow a determination of YOLL at least in principle, although in practice there are large uncertainties in the extrapolation from the study population to the general population. We apply our YOLL estimates to a comparison of mortality costs with morbidity costs, for particles and for ozone. The costs of acute mortality turn out to be small compared to morbidity. For particles all is dominated by chronic mortality. © 1998 Elsevier Science B.V. All rights reserved.

Keywords: Air pollution health impacts; Exposure–response functions; Acute mortality; Chronic mortality; Time-series studies; Cohort studies; Cost–benefit analysis; Comparative risk analysis; Value of year-of-life-lost

1. Introduction

In recent years, several major studies have estimated the social costs of air pollution ([1–4], and others; for review, see Ref. [5]). For mortality impacts, all of these studies have followed the simple approach of using E–R functions (exposure–response) to

* Present address: Department of Civil, Environmental and Architectural Engineering, University of Colorado, Boulder, CO 80309-0428, USA.

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calculate the increase in the number of deaths and multiplying the result by the value of life V. The resulting numbers are very large, in some cases implying that the external costs of energy approach or even exceed the market price.

This approach has been questioned on the grounds that only a small portion of the population, mostly the old and the very sick, seem to be affected by air pollution to the point of dying; the life span reduction ΔT, also called years-of-life-lost (YOLL), is presumably small compared to the average human life span T or the typical loss T_l implicit in value of life studies which is on the order of 30 to 40 years. This highlights a fundamental question about the valuation of mortality: should we value the number of premature deaths N_{deaths}

$$\text{cost}_{N_{\text{deaths}}} = N_{\text{deaths}} \times V \tag{1}$$

or the number of YOLL

$$\text{cost}_{\Delta T} = N_{\text{deaths}} \times \Delta T \times v_{\text{YOLL}} \ ? \tag{2}$$

For the cost of acute mortality, the difference is at least an order of magnitude. These two valuations can be considered extremes, with a continuum of possibilities in between.

We argue that the YOLL valuation is more rational. For the practical implementation, there is, however, a difficulty: the E–R functions by themselves contain no information on ΔT. We examine therefore to what extent a YOLL valuation is possible. This requires attention to different definitions of mortality rates. E–R functions for acute mortality report variations in daily death counts, E–R functions for chronic mortality report variations in age-specific mortality, and a YOLL calculation needs variations in population mortality.

2. Number of deaths or YOLL?

The value v_{YOLL} of one YOLL is derived from V by imagining the latter as a discounted series

$$V = v_{\text{YOLL}} + \frac{v_{\text{YOLL}}}{l+r} + \frac{v_{\text{YOLL}}}{(l+r)^2} + \ldots + \frac{v_{\text{YOLL}}}{(l+r)^{T_l}} \tag{3}$$

where $r =$ discount rate and $T_l =$ number of years of life lost. For $r = 0$ one finds $v_{\text{YOLL}} = V/T_l$. In the $r \to \infty$ limit V approaches v_{YOLL} and the two valuation methods become equivalent. For example, suppose that a policy A can be implemented in two steps, the first, A′, which increases life expectancy by 1 year, and the second, A″, which adds 4 more years. For this latter, the value according to $\text{cost}_{N\text{deaths}}$ is zero. In effect, any years beyond the initial extension are discounted so heavily as to have no benefit. In other words, a policy that increases (decreases) life expectancy, without changing the number of premature deaths, has zero benefit (cost).

Analysts of public policy lament the shortsightedness of the unreasonably high discount rates implicit in so many public or private decisions. We believe it is more appropriate to offer rational guidelines for public policy rather than trying to reflect the irrationalities and inconsistencies of uninformed decisions.

Table 1
Comparison of incremental costs of morbidity and mortality for PM_{10}, according to the E–R functions and costs of ExternE Program [3,12]

Particles PM_{10}	Cases/year per person per $\mu g/m^3$	ECU/case	ECU/year per person per $\mu g/m^3$
HA for respiratory infections	1.87×10^{-6}	6015	0.011
HA for COPD	2.27×10^{-6}	6015	0.014
ERV for COPD	7.2×10^{-6}	170	0.001
ERV for asthma	12.9×10^{-6}	170	0.002
Croup	29.1×10^{-6}	9	0.000
Restricted activity days	$49\,900\times10^{-6}$	48	2.395
Asthma short breath	4200×10^{-6}	38	0.159
Respiratory symptoms	$465\,000\times10^{-6}$	6	2.620
Total morbidity			5.20
Acute mortality YOLL	4.68×10^{-6}	116 000[b]	0.54
Chronic mortality YOLL	$f\times8.7\times10^{-4}\times PM_{2.5}/PM_{10}$[a]	84 000[b]	21.9

COPD = chronic obstructive pulmonary disease, ERV = emergency room visit, HA = hospital admission.
[a] Eq. (12), multiplied by factor $f = 0.5$ for latency and by $PM_{2.5}/PM_{10} = 0.6$.
[b] $v_{YOLL} = 116$ KECU for acute and 84 KECU for chronic (different because of discounting, with rate 3%).
1 ECU = US$1.25.

For these reasons we use a YOLL valuation. Valuing a year of life is of course a delicate matter, even more problematic than an average value of life V as used in Eq. (2). Should the value of a YOLL be higher or lower for old age than for youth? There seem to be no firm data, and a priori one can think of arguments either way. For collective decision making it might be preferable to avoid such intergenerational questions altogether by using a single value per YOLL. That is also in the spirit of the general practice of using a single value of V for all individuals in a population, without distinction of wealth, health or will to live. For the present paper, we take v_{YOLL} as fundamental unit (see Table 1).

3. Acute mortality E–R functions

Most E–R functions for health impacts of air pollution are of the acute type, i.e. they have been determined by analyzing short-term (at most a few days) correlations between ambient concentrations and incidents of the health impact under consideration. This approach has the great advantage of being easy to implement and insensitive to the confounders (such as smoking) that plague the determination of chronic E–R functions, which requires long-term or cross-sectional studies. By analogy the terms acute and chronic are also applied to mortality even though the attributes appear strange in that context.

The E–R functions for acute mortality are obtained from correlations between ambient concentrations and daily death counts. If one examines the formal relation between fluctuations of concentration and of death counts, one finds that the resulting E–R function is independent of ΔT. One would find the same acute mortality E–R

function even if ΔT were negative, i.e. if the pollutant were an elixir that prolongs people's life by ΔT. It is difficult to obtain any data for ΔT, short of being either god (i.e. omniscient) or devil (i.e. willing to perform unlimited human experiments).

If, as seems plausible, the processes that determine ΔT are mostly multiplicative rather than additive, the distribution of an individual ΔT's is approximately lognormal. To estimate the corresponding geometric standard deviation σ_g, let us examine plausible upper and lower limits for ΔT. The lower limit is at least a few days, otherwise the effect could not be observed by time-series studies. The upper limit seems unlikely to be more than a few years. Taking 4 days and 1024 days as 95% confidence interval, one obtains a σ_g of 4 if the distribution is lognormal; the corresponding median ΔT is 64 days = geometric mean μ_g. For the calculation of the population total YOLL one needs the ordinary mean μ. The relation between μ and μ_g of a lognormal distribution is

$$\mu/\mu_g = \exp\left(\ln(\sigma_g)^2/2\right) \tag{4}$$

With a σ_g of 4 this ratio is 2.6. That would imply a mean ΔT on the order of 0.5 years.

4. Chronic mortality

It is unlikely that air pollution causes only short-term effects detectable by time-series studies. Imagine trying to measure mortality from cigarettes by restricting smoking to certain days and observing the variations in daily mortality during the following days. One would see the effect on a few hypersensitive individuals who are pushed over the edge by that extra puff. The loss of life expectancy would be negligible compared to long-term effects such as emphysema and lung cancer which cause very significant shortening of life without being detectable by short-term studies.

In recent years, several prospective cohort studies of chronic mortality from particulate air pollution have been published [6–8]. Abbey et al. [6] found no significant increase in general mortality, although female cancers appeared correlated with air pollution. By contrast, the other two studies found a statistically significant increase of mortality with particulate and/or sulfate concentration. Pope et al. [8] studied a very large population, about 550 000 adults (in 151 metropolitan areas, followed during 7 years), by contrast to about 6000 (followed during 10 years) in Abbey et al. and over 8000 (in six cities, followed during 14–16 years) in Dockery et al. [7]. The E–R function of Pope et al. has approximately half the slope of Dockery et al.; here we use the one of Pope et al. because it is based on the largest population by far.

In these studies, cohorts in regions with different pollution levels were selected and observed over many years. The observation time is long enough that differences in the number of deaths between different regions can be seen. The analysis is carried out as if conditions were in a steady state; thus, no information is provided on latency or the ability of the body to repair damage after exposure stops. In particular, there is no information on the effect of air pollution on age groups not included in the studies (younger than 25 years). Therefore, we also assume steady state in the present paper; at the end we apply a correction factor f for the fraction of the population that is affected.

The age-specific mortality $\mu(t)$ is defined as the number of deaths per year in a cohort of age t. In the range of 30 to 90 years this rate follows, to an excellent approximation, the Gompertz formula

$$\mu(t) = \alpha \exp(\beta t) \tag{5}$$

where α and β are constants. The precise values of the constants depend on the population, but the general form has been found among all human populations and even most other species [9]. For the US population the data on p. 5 of Ref. [9], together with the life expectancy at birth of $T = 75$ years [10] can be fit by

$$\alpha = 4.57\text{E} - 05 \text{ and } \beta = 0.0921 \tag{6}$$

Before 30 years, the pattern is different, with a high rate at birth, dropping to a minimum around 10 years, then rising slightly to a plateau around 20 years, and then increasing again. For the present purpose the precise form in this age range does not matter because $\mu(t)$ is so low that the contribution to the results is negligible. For this paper, we use a reference rate $\mu_0(t)$, obtained by taking a constant value for all $t < 30$ years, with continuous transition to the Gompertz formula at 30 years, with α and β of Eq. (6). Let $F(t)$ be the fraction of a population that survives to age t. It is related to the mortality $\mu(t)$ by

$$\mathrm{d}F = -F(t)\,\mu(t)\,\mathrm{d}t \tag{7}$$

With the initial condition $F(0) = 1$ one finds

$$\ln(F(t)) = -\int_0^t \mu(t')\,\mathrm{d}t' \tag{8}$$

The life expectancy is readily shown to be

$$T = -\int_0^\infty F(t)\,\mu(t)\,\mathrm{d}t. \tag{9}$$

The above prospective cohort studies employ the Cox proportional-hazards regression model [see e.g. Ref. [11]], and the survival data are fitted by assuming that the mortality rate for a cohort with pollution c is related to the rate without pollution according to

$$\mu(t) = \mu_0(t)\exp(\theta c + K). \tag{10}$$

Thus, the mortality is multiplied by the same factor for all ages, with the parameter θ to be determined. The factor $\exp(\theta c)$ is reported as mortality risk ratio for air pollution. The term K serves to account for nonpollution risk factors such as smoking. The reference rates used in the above studies are based on the specific populations, not the model in Eq. (5). This is of no concern since the relative risk is supposed to be transferable. The largest relative mortality risk reported by Pope et al. is

$$\exp(\theta \Delta c) = 1.17 \text{ for } \Delta c = 24.5\ \mu\mathrm{g/m^3} \text{ of } \mathrm{PM}_{2.5}. \tag{11}$$

The survival fractions are shown in Fig. 1, as solid line $F(t)$ for $\mu_0(t)$ and as dotted line $F'(t)$ for $1.17\ \mu_0(t)$. The corresponding life expectancies are $T = 75$ years and $T' = 73.4$ years. This is an increase of about 2% in population mortality $m = 1/T$, much smaller than the change in mortality risk ratio from 1.0 to 1.17. The relation between

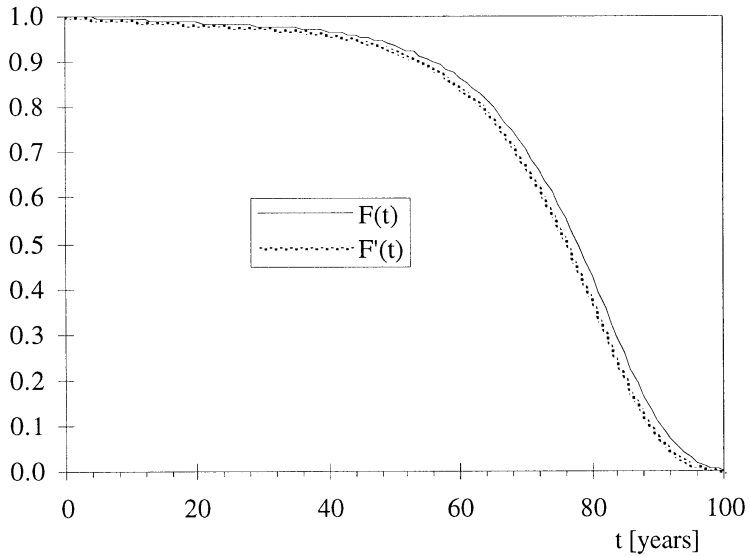

Fig. 1. Fraction $F(t)$ surviving to age t. Solid line = $F(t)$ for $\mu_0(t)$ of Eq. (5) with life expectancy $T = 75$ years; dotted line = $F'(t)$ for 1.17 $\mu_0(t)$ with $T = 73.4$ years, corresponding to $\Delta c = 24.5\ \mu g/m^3$ of $PM_{2.5}$.

$\Delta T/T$ and $\mu(t)/\mu_0(t)$ can be calculated numerically from Eq. (9), assuming $\mu_0(t)$ of Eq. (5). If one plots the result, one finds it almost indistinguishable from a logarithmic curve. The logarithm in this fit cancels the exponent in Eq. (11), leaving as net result a linear E–R function for $\Delta T/T$ vs. Δc. The lifetime YOLL per person is ΔT, the inverse of the life expectancy T is the steady state population mortality rate, and $\Delta T/T$ is the annualized YOLL per person per year of exposure. This allows us to state the slope of the E–R function of Pope et al. [8] as YOLL per person per year per $\mu g/m^3$ of $PM_{2.5}$ in the form

$$\text{slope} = f\frac{\Delta T/T}{\Delta c} = f \times \frac{1.6\,\text{years}/75\,\text{years}}{24.5\,\mu g/m^3}$$

$$= f \times 8.7 \times 10^{-4}\,\text{YOLL}/(\text{person}\cdot\text{year})\text{per}\,\mu g/m^3\,PM_{2.5} \qquad (12)$$

where we have added a factor f to account for latency and the fraction of the population that is affected. For the calculations below we will take $f = 0.5$, corresponding to the fraction of the total population included in the study of Pope et al. This factor is the major uncertainty.

5. Conclusions

It is interesting to apply these YOLL estimates to mortality costs of particulate and ozone air pollution, and to compare the results with morbidity. This is shown in Table 1, for a population exposed to an increment of 1 $\mu g/m^3$ of PM_{10}, assuming linear E–R

Table 2
Comparison of incremental costs of morbidity and mortality for O_3, based on E–R functions and costs of ExternE Program [3,12]

	Cases per person × ppb × year	ECU/ case	ECU/ (year × person × ppb)	%
Acute mortality YOLL	5.9E-06	116 000[a]	0.679	28%
Respiratory HA	1.42E-05	6600	0.093	4%
Restricted activity days	1.95E-02	62	1.209	50%
Symptoms days	6.60E-02	6.3	0.416	17%
Total ECU/(year × person × ppb)			2.362	100%

[a]Assuming average life span reduction of 6 months and $v_{YOLL} = 110$ KECU for acute mortality.
There are no data for chronic mortality of ozone. Same assumptions as Table 1.

functions (or equivalently, that the background exposure is above the threshold, if any). The E–R functions and cost data are taken from the ExternE Program [3,12]; they are comparable to data published in the USA [1,2,5]. Since chronic mortality of Pope et al. is based on $PM_{2.5}$, we have reduced cases/year and cost/year by a factor $PM_{2.5}/PM_{10} = 0.6$. Analogous results for ozone are shown in Table 2.

For particulate air pollution the E–R functions for chronic mortality of Dockery et al. and Pope et al. imply that the cost of chronic mortality is larger than the costs of morbidity. Morbidity, in turn, dominates acute mortality, both for particles and for ozone. The uncertainties of the YOLL estimation are large, corresponding to a σ_g of perhaps 2 for chronic and 4 for acute.

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