Interpretation of Air Pollution Mortality: Number of Deaths or Years of Life Lost?

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Journal of the Air & Waste Management Association
Vol.53(1), 41-50 (2003)

Abstract

This paper examines the relation between the results of epidemiological studies of air pollution mortality and impact indicators that can be informative for environmental policy decisions. Using models that are simple and transparent, yet contain the essential features, it is shown that (1) number of deaths is not meaningful for air pollution, whereas loss of life expectancy (LLE) is an appropriate impact indicator; (2) the usual short term (time series) studies yield a change in daily number of deaths attributable to acute effects of pollution, without any information on the associated LLE (although some information on this has recently become available by extending the observation window of time series); (3) long term studies yield a change in age-specific mortality which makes it possible to calculate the total population averaged LLE (acute and chronic effects) but not the total number of premature deaths attributable to air pollution. The latter is unobservable because one cannot distinguish whether few individuals suffer a large or many a small LLE. The paper calculates the LLE from exposure to PM_{10}, as implied by the long term mortality studies of adults and infants; population LLE for infants turns out to be an order of magnitude smaller than for adults. The LLE implied by short term studies is a small fraction of the total loss implied by long term studies, even if one assumes a very high loss per death. Applied to environmental policy, taking a permanent 50% to 70% reduction of PM_{10} as a reasonable goal, one finds a corresponding increase of average life expectancy in urban areas of EU and USA by roughly four months.

Key words: air pollution, mortality, infant mortality, causes of death, particulate matter, life expectancy, years of life lost, number of premature deaths

1. Introduction

In recent years, many studies have attempted to quantify the impacts of mortality due to air pollution [ORNL/RFF 1994, Rowe et al. 1995, ExternE 1998, ExternE 2000, Levy et al 1999, Abt 2000, Kuenzli et al 2000, and others]. Whereas all studies before 1996 calculated a number of premature deaths, there has been a growing recognition in recent years that it is more meaningful to look at loss of life expectancy (LLE) [see e.g. McMichael et al 1999, Wilson & Crouch 2001]. In particular, it is not reasonable to attribute the same benefit to the avoidance of an air pollution death, with an LLE in the order of months, as to a car accident which claims on average 30 to 40 years. Therefore the ExternE project series ("External Costs of Energy") has, since 1998, based the monetary valuation of air pollution mortality on LLE, and economists have begun to evaluate the willingness-to-pay to increase life expectancy [Johannesson & Johansson 1996, Krupnick et al 2000] (by contrast to previous "value of life" studies that are based on accidental deaths).

It has also been recognized that the full impact of air pollution on health is much larger than what can be measured by the easy and widely used time series epidemiology (which identifies only acute impacts, 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 few long-term studies available. However, in recent years three important epidemiological studies, of the prospective cohort type, have examined the long-term impacts. Two of these [Dockery et al. 1993, Pope et al. 1995] have found positive correlations between exposure to particles and long term mortality (also called chronic mortality in contrast to the acute, or short term, mortality measured by time series studies), while the third [Abbey et al. 1999] found a significant positive correlation with long term mortality for men but not for women. The methodology and analysis of Dockery et al. [1993] and of Pope et al. [1995] have been confirmed by the reanalysis project of the Health Effects Institute [Krewski et al 2001]. The analysis period of the cohorts studied by Pope et al [1995] has recently been more than doubled to 16 years, with much more thorough analysis and results that are numerically comparable [Pope et al 2002].

However, there continues to be confusion about the interpretation of epidemiological studies of air pollution mortality and their use for deriving impact indicators for environmental policy decisions. In particular some authors try to interpret the studies of chronic mortality by estimating a number of premature deaths. The term "excess deaths" has also been used but does not seem apt since the number of deaths is uniquely determined by the number of births; a risk factor can only cause a death to occur earlier. [1]

For air pollution a number of premature deaths is not meaningful for several reasons, the first two being obvious, the third being explained in Sections 2 and 6:

i) it makes no sense to add the number of deaths due to different contributing causes (such as air pollution, smoking or lack of exercise) because one would end up with numbers far in excess of total mortality;
ii) number of deaths fails to take into account the magnitude of the loss of life per death, very different between for example air pollution deaths and typical traffic accidents;

[1] "Excess death rate" would be meaningful because the number of deaths per unit time is increased by a risk factor, but it is equivalent to stating the LLE because the relative increase in long term average death rate is the ratio of LLE and life expectancy.

iii) by contrast to primary causes of death (such as accidents or cancers), the total number of premature deaths attributable to air pollution is not observable.

LLE avoids these problems; it is a meaningful and appropriate impact indicator for all risk factors, even those that are not observable as the cause of an individual death. In particular, it can be added across different risk factors (at least in the limit of small risks).

By means of models that are simple yet contain the essential features, this paper examines what kind of impact indicators can be extracted from epidemiological studies of air pollution mortality. Whereas this question has been addressed for cancers in previous studies, e.g. by Robins & Greenland [1991] and Thomas et al [1992], the issues are different in the present case since air pollution cannot be identified as primary cause of a particular death and most air pollution mortality is cardio-vascular, very different from cancers. Furthermore, the important methodology of time series is not appropriate for cancers. Epidemiological studies of the usual time series type measure a change in the number of deaths per day without any information on the LLE per death; only acute effects (i.e. due to short term exposure) are taken into account (although, two recent studies [Zeger et al 2000 and Schwartz 2000] have obtained some information on the LLE per death by extending the observation window of time series studies). Long term epidemiological studies can measure the total impact of air pollution or other contributing causes on mortality; their results are expressed as in increase in age-specific mortality from which LLE can be calculated.

As an illustration, numerical results are presented for the LLE implied by air pollution, based on the most recent references. The numbers for chronic mortality of adults have been published before, but here they are updated and adapted to the more relevant mean concentrations according to Pope et al [2002]. The numbers for acute mortality are based on Samet et al [2000a and b], Katsouyanni et al [1997] and Levy et al [2000]. The numbers for infant mortality, not published before, are calculated using Woodruff et al [1997] and Bobak &Leon [1999]. Finally the numbers are placed in perspective by showing how much the life expectancy could be increased by reducing the ambient concentration of particulate matter.

2. Observability

To see what can be observed under optimal conditions by an epidemiological study, let us take a stationary population (birthrate = death rate = constant) whose baseline daily death count is n_0 in the absence of pollution. Consider the effect of a brief exposure pulse at t=0, for a series of models, illustrated in Fig.1, that become progressively more realistic. In the simplest model, Fig.1a, all individuals who are affected die between Δ (delay time) and $\Delta+\delta t$ after the pulse, and their life expectancy L is shortened by LLE; δt is the period over which the data are aggregated, i.e. 1 day for daily death counts. The increase δn in daily death count is followed by an equal decrease after LLE. This phenomenon, sometimes called "mortality displacement" [2], is an inevitable consequence of the fact that sooner or later everybody dies. With the generalization in Fig.1b all individuals who are affected die over a range of delay times $\{\Delta\}$ after the pulse and lose LLE. Fig.1c supposes that all individuals who are affected die between Δ and $\Delta+\delta t$ after the pulse and suffer a range of losses $\{LLE_i\}$.

[2] The term “harvesting” has also been used, with the implication that deaths are advanced by only a very short period; here nothing is implied about the loss per death.

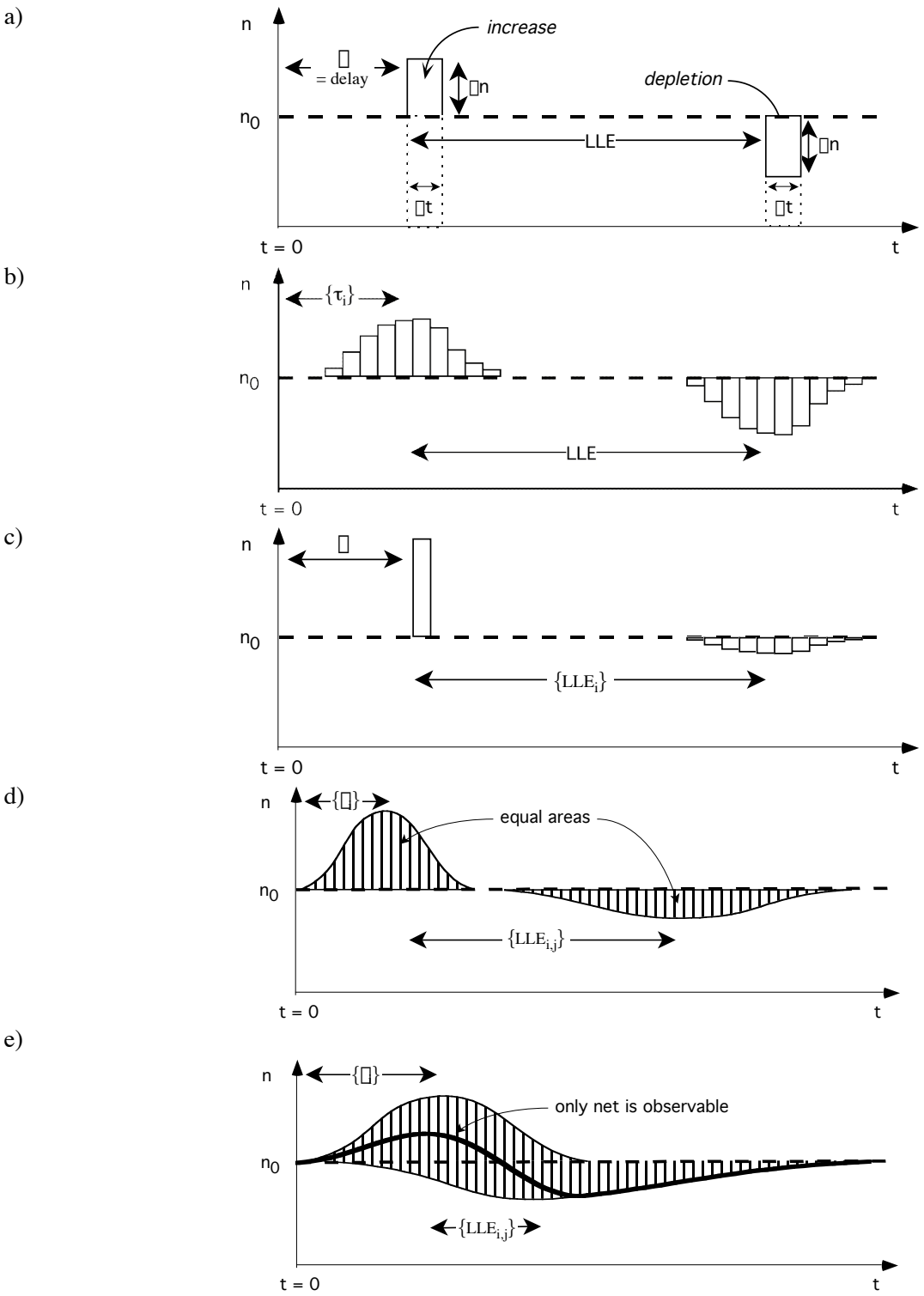

Fig.1. Several models for effect of air pollution pulse at t=0 on daily death count n. $\Delta t = 1$ day.
a) all affected individuals die between Δ and $\Delta+\Delta t$, and their life span is shortened by LLE.
b) all affected individuals die over a range of delay times $\{\Delta\}$ and lose LLE.
c) all affected individuals die between Δ and $\Delta+\Delta t$ and suffer a range of losses $\{LLE_i\}$.
d) affected individuals die over a range $\{\Delta\}$ and suffer a range of losses $\{LLE_i\}$: depletion is distinct.
e) affected individuals die over a range $\{\Delta\}$ and suffer a range of losses $\{LLE_i\}$: depletion overlaps and only the net (heavy line) is observable, not the total attributable to pollution.

Finally, in parts d) and e) of Fig.1, there is both a range of delay times $\{\Delta\}$ and a range of losses $\{LLE_i\}$. Since Δ and LLE_i are likely to be long compared to a day, smooth curves are shown instead of the steps in parts a)-c). In part d) the depletion is distinct ($Min[LLE_{i,j}] > Max[\Delta]$) and the total number of deaths is observable: it is the integral under the peak. In part e) the depletion overlaps and the observed death count, indicated by the heavy line, is less than the total number of deaths attributable to the pulse (= area between n_0 and upper envelope). To reconstruct the true mortality peak from the observed data, one would need to know the distributions of $\{\Delta\}$ and $\{LLE_i\}$. But they cannot be determined because only the thick line in Fig.1e) is observable. Therefore in the case with overlap the total number of deaths due to the pulse is larger than what is observable. Another way of putting it is that the total number of attributable deaths is observable only if they are all sufficiently immediate, in the sense of not being obscured by the subsequent depletion of the population.

Even though the discussion of Fig.1 started with the assumption of a single brief exposure pulse and the observation of a stationary population, the conclusions are general. In practice there will be a superposition of days with different exposures, some higher, some lower than the average (=baseline), confounding the overlap between positive and negative contributions (relative to the baseline mortality) even more.

Because of the large spread of the $\{LLE_i\}$ distribution and the fluctuations in concentrations, the depletion tends to become a uniform background, not visible in TS studies. By looking only at the death rate within the first few days after exposure, TS see mostly the initial deaths rather than the net death rate. This explains why TS are such a sensitive tool. The price is the loss of any information on the LLE per death.

But the longer the window over which the number of deaths is averaged, the less the initial change in deaths after a change in concentration stands out above the depletion background. Long term (cohort) studies see the net death rate, i.e. the initial deaths minus the depletion; the effect of pollution is identified by comparing populations that are exposed to different concentrations. As shown by Rabl [2002], long term studies allow the calculation of population total LLE, but not the number of deaths attributable to air pollution: the change in net death rate due to pollution is the same whether a few individuals lose much or many a little.

3. Short Term Studies

DR functions for acute mortality are obtained from correlations between variations in ambient concentration Δc and daily death count Δn; in practice the observation window t_{obs} (=maximum delay between exposure and impacts taken into account for the correlation) is at most 5 days. The result carries no information on LLE. This is easy to see for the model of Fig.1a where a short pollution pulse of concentration increase Δc causes Δn individuals to die exactly LLE sooner than in the absence of the pulse. In the situation of Fig.1a a time series analysis finds that Δn is proportional to Δc, independent of LLE. This feature remains unchanged with more realistic models (Fig.1d or e), sequences of days, or complications such as LLE per death varying with Δc. Therefore the usual short term mortality studies observe changes in the daily death count, without providing any information on the loss of life expectancy LLE per death. This fact has also been

noted by others, in particular Kuenzli et al [2001] who discuss the relation between acute and chronic mortality.

However, this situation changes if the observation window t_{obs} is enlarged to cover a significant portion of the {□} distribution and the concentration is correlated with the sum of the daily □n within this window, rather than just the □n for a particular day. The sum of the observed daily □n increases with t_{obs} until the heavy line in Fig.1e drops below the baseline n_0. Two recent studies [Schwartz 2000 and Zeger et al 2000] have succeeded in extending t_{obs}. Without going into detail one can say that they find the correlation coefficient between concentration and sum of the □n within t_{obs} to increase with t_{obs} until at least 60 days, implying that the average loss of life per death is at least in the order of 60 days. Unfortunately it does not seem possible with this approach to extend the window further because for longer windows the fluctuations of confounding factors overshadow the effect of concentration fluctuations.

The time series approach has the great advantage of being easy to implement and unaffected by the confounders, such as smoking that plague the determination of chronic DR functions. It is a very sensitive tool, crucial for identifying possible links between air pollution and health. But it can only measure short term impacts. To measure the total (short and long term) impacts of air pollution one needs long term studies.

4. Long Term Studies

In the long term studies cited above, cohorts in regions with different pollution levels were selected and observed over many years. A key element for the analysis is the age-specific mortality □(x), defined such that someone who has reached age x has a probability □(x) □x of dying between x and x + □x (data are usually stated in terms of □x = 1 year). Without going into the details of their statistical analysis one can say that such studies measure a change in □(x). The observation window must be long; it has been at least seven years in the cohort studies of Abbey et al, Dockery et al and Pope et al [1995], recently extended to 16 years for the latter [Pope et al 2002].

Using the Cox proportional-hazards regression model, the survival data are fitted by assuming that exposure to a concentration increment □c increases the mortality relative to the baseline $□_0(x)$ by

$$□(x) = □_0(x) \exp(k\, □c) \quad , \qquad (1)$$

with the parameter k to be determined. The factor exp(k □c) is reported as mortality risk ratio R, or relative risk, for air pollution. Pope et al found

$R = \exp(k\, □c) = 1.17$ for $□c = 24.5$ □g/m^3 of <u>median</u> $PM_{2.5}$ concentrations, (2a)

implying

k = 0.0064 per □g/m^3 of <u>median</u> $PM_{2.5}$ concentrations, (2b)

and until now the calculations of LLE [including Leksell & Rabl 2001] have been based on this value of R.

While the reanalysis of Krewski et al [2000] for median $PM_{2.5}$ concentrations confirms Pope et al [1995], the present paper uses the more recent result of Pope et al [2002], which is based on mean concentrations $PM_{2.5}$, because mean concentrations are more commonly reported than medians. The relative all-cause mortality risk reported by Pope et al [2002] for the average concentration 1979-2000, in their Table 2, is (in parentheses 95% confidence interval)

$R = 1.06$ (1.02 to 1.11) for $\Delta c = 10$ $\mu g/m^3$ of mean $PM_{2.5}$ concentrations, (3a)

implying

$k = 0.0058$ per $\mu g/m^3$ of mean $PM_{2.5}$ concentrations. (3b)

We choose the number corresponding to the average concentration 1979-2000, rather than the one for 1979-1983 which the authors cite in the abstract, because the average over the entire study period seems more relevant for policy purposes.

5. Loss of Life Expectancy due to Air Pollution

5.1. Mortality and Life Expectancy

Relatively simple steady state analyses have been published by Brunekreef [1997], ExternE [1998] and Rabl [1998], recently confirmed by a more thorough and detailed dynamic analysis to take into account variable pollution exposures [Leksell & Rabl 2001]. The present paper adds the calculation of LLE for infant mortality, not previously published. This calculation requires an assumption on how long the infants who die would have lived in the absence of air pollution, which introduces a large uncertainty. To explain why this uncertainty is larger for infants than for adults, it is instructive to recall the method for calculating LLE.

The survival function $S(x,x')$ is the fraction of a cohort of age x that survives at least to age x'. Since the fraction that dies between x' and $x' + \Delta x'$ is $\Delta S_\Box(x,x') = S_\Box(x,x')\ \Box(x)\ \Delta x$, one gets the differential equation

$$dS_\Box(x,x') = -\ S_\Box(x,x')\ \Box(x')\ dx' \quad ; \tag{4}$$

the boundary condition is $S_\Box(x,x) = 1$. One readily finds the solution

$$S_\Box(x,x') = \exp\left[-\int_x^{x'} \Box(x'')\ dx''\right] \quad . \tag{5}$$

The probability distribution for a member of the age x cohort to survive to and die at age x' is

$$p(x,x') = S_\Box(x,x')\ \Box(x') \quad . \tag{6}$$

it is normalized to unity over the interval from x to . The expected age of death is therefore the integral of x' p(x,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

$$L(x) = \int_x x' \, S_{\mu}(x,x') \, \mu(x') \, dx' - x. \qquad (7)$$

For practical calculations the formula

$$L(x) = \int_x S_{\mu}(x,x') \, dx' \qquad (8)$$

is more convenient and converges faster as the upper limit of the integral goes to . For calculations with life tables one approximates the integral by a sum over one-year intervals.

If $\mu(x)$ changes, for example due to air pollution, $S_{\mu}(x,x')$ and L(x) change accordingly. The resulting change LLE(x) for a cohort of age x is the difference between L(x) calculated without and with this increase

$$LLE(x) = \int_x [S_{\mu 0}(x,x') - S_{\mu}(x,x')] \, dx' \quad , \qquad (9)$$

where $S_{\mu 0}(x,x')$ is the survival curve for the baseline mortality $\mu_0(x)$. The impact on the entire population is obtained by summing LLE(x) over all affected cohorts, weighted by the age distribution.

5.2. LLE for Adults

The above calculation is directly applicable to steady state exposures, and Eq.9 yields the LLE due to constant exposure during an entire lifetime. The results of Pope et al also correspond to steady state exposure because time variation of the concentration data was not considered. But it is also of interest to consider time varying exposures, especially for applications to environmental policy. Furthermore, the real exposure of the cohorts studied by Pope et al declined during the study period, and it is not obvious how this may have affected the results.

For these reasons Leksell & Rabl extended the above framework to account for time varying concentrations and impacts. They do this by introducing the time constant(s) for the decrease of risk after exposure, based on estimates from studies of smoking (such decrease of risk occurs if the body repairs some of the damage). Even though the uncertainty of time constant(s) inferred from smoking studies is large, it turns out to have almost no effect on the resulting LLE, as Leksell & Rabl confirm by a sensitivity analysis.

Leksell & Rabl show that the LLE is almost exactly proportional to exposure, defined as time integral of the concentration increase under consideration; the time distribution itself does not matter. For comparison purposes it is therefore convenient to state the LLE for exposure during one year to a concentration increment of 1 $\mu g/m^3$ of PM_{10}; the result can readily be scaled to other exposures. Since the effect of a one-year pulse on infant mortality does not extend beyond the

first year of the cohort (by definition of infant mortality), the simple steady state analysis of Eq.9 is sufficient for infants. For adults, a good approximation can be obtained using the steady state analysis, if one divides the loss due to a constant life time exposure by the life expectancy at birth.

For adult chronic mortality due to a pollution pulse of 1 $\mu g/m^3$ $PM_{2.5}$ lasting 1 year, Leksell & Rabl find approximately 600 years of life lost per million persons as typical result in the EU and in the USA (see the Δ column in their Table 1), based on Pope et al, Eq.2, with a correction factor for past exposure. Variations due to differences in life table data between the USA and the EU change this number only by a few percent. Using instead the more recent Pope et al [2002], Eq.3, this is reduced to 550 years of life per million persons. Since PM_{10} data are more commonly available, it seems appropriate to multiply this number by the typical $PM_{2.5}/PM_{10}$ ratio of 0.6 to obtain

$$LLE = 330\ (110 \text{ to } 590)\ yr_{life}/(yr_{exp} \cdot \mu g_{PM10}/m^3) \text{ per million persons} \quad . \qquad (10)$$

The range in parentheses corresponds to the 95% confidence interval of R in Eq.3, and the subscripts of yr distinguish years of life lost and years of exposure. This is a global number, averaged over the entire population. Individual losses can be higher or lower but the distribution is not known.

5.3. LLE for Infants

Woodruff et al [1997] analyzed cohorts consisting of about 4 million infants born in the USA between 1989 and 1991. They found statistically significant associations between PM_{10} concentrations and several causes of postneonatal mortality: SIDS (sudden infant death syndrome) and respiratory causes for normal birth weight, as well as respiratory causes for low birth weight. The corresponding relative risks are shown in Table 1, together with the corresponding Δc. Woodruff et al looked only at the postneonatal period (between the ages of 27 days and 1 year), because postneonatal deaths are thought to be more influenced by the infant's external environment than deaths during the neonatal period.

Bobak & Leon [1999] carried out a case-control study of infant mortality in the Czech Republic from 1989 to 1991. Among the associations shown in their paper, the most relevant for the present purpose appear to be those for respiratory and for all cause postneonatal mortality due to TSP (total suspended particles), shown here in Table 1 together with the corresponding Δc. The latter has been converted to PM_{10}, assuming a PM_{10}/TSP ratio of 0.77, the center of the range 0.57 a 0.96 observed in the Czech Republic [M. Bobak, personal communication 14 Feb. 2002]. Contrary to Woodruff et al, Bobak & Leon did not find a significant association for SIDS.

Table 1. Calculation of LLE for postneonatal mortality (between 1 and 12 months of age). The LLE is upper bound because infants who die because of pollution may have had less than normal life expectancy (here assumed as 76 yr).

	Woodruff et al [1997]				Bobak&Leon[1999]	
	SIDS, NBW	Resp, NBW	Resp, LBW	**All causes**	Resp	**All causes**
Rel. risk R	1.12 [a]	1.20 [a]	1.05 [a]	1.04 [a]	1.95 [b]	1.19 [b]

Δc [$\mu g/m^3$ PM_{10}]	10	10	10	10	38.25 [c]	38.25 [c]
k [per ($\mu g/m^3$)]	0.0113	0.0182	0.0049	0.0039	0.0175	0.0045
mortality, per 1000 births				3.89 [d]		3.89 [d]
LLE [$yr_{life}/(\mu g/m^3)$, per birth]				-0.00115		-0.00133
LLE [$yr_{life}/(yr_{exp} \cdot \mu g/m^3)$ per million]				**-16.6** [e]		**-19.3** [e]

NBW = normal birth weight; LBW = low birth weight; SIDS = sudden infant death syndrome; Resp = respiratory causes.

[a] Table 3 of Woodruff et al [1997].

[b] Table 4 of Bobak & Leon [1999].

[c] Assuming a PM_{10}/TSP ratio of 0.77.

[d] Table 30 of NCHS [1999], subtracting perinatal from total infant mortality.

[e] For birth rate 14.5 per yr per 1000 population [NCHS 1999].

The authors note that "… the associations with mortality were approximately linear". Applying a dose-response relation of Bobak & Leon to other countries involves uncertainties, because of different levels of air pollution and infant mortality. However, the concentrations during this study were only about a factor of two higher than those found in urban environments of EU and USA, not a large difference in view of the approximate linearity observed by Bobak & Leon (the mean exposure in the study was 55 $\mu g/m^3$ of PM_{10}, with a range from 9 to 139 if one converts with a PM_{10}/TSP ratio of 0.77). The infant mortality rates are not very different: 9.6 per 1000 for the data set of Bobak & Leon and 7.2 per 1000 in the USA [NCHS 1999].

The bottom line of Table 1 shows the LLE implied by the associations for all cause mortality. The numbers, for Woodruff et al 16.6 $yr_{life}/(yr_{exp} \cdot \mu g_{PM10}/m^3)$ per million and for Bobak & Leon 19.3 $yr_{life}/(yr_{exp} \cdot \mu g_{PM10}/m^3)$ per million, are remarkably close (their average is approx. 18). However, the results for individual causes (SIDS and respiratory mortality) do not agree and/or are difficult to compare because of a lack of consistent data for the baseline rates. The details of the calculation are explained in the following paragraphs.

Assuming the usual exponential relation of Eq.2a between relative risk and concentration increment Δc, one finds the values of the constant k shown in Table 1. Since $k \cdot 1\ \mu g/m^3$ is very small compared to unity, k is essentially the fraction by which the respiratory infant mortality increases per $\mu g/m^3$. By definition, only the cohort having less than 1 yr of age is affected. According to the discrete version of Eq.9 the LLE(0) of this cohort is related to the difference $\Delta S = S_{\Delta 0} - S_{\Delta}$ in survival curves by

$$LLE(0) = \Delta S(0,1) + \Delta S(0,2) + \ldots \qquad (11)$$

Assuming that the mortality after the first year is the same with and without pollution, this can be written as

$$LLE(0) = \Delta S(0,1) + \Delta S(0,1)\,[S(1,2) + S(1,3) + \ldots\,] \qquad (12)$$

The sum in [ ] equals L(1), the remaining life expectancy at age 1, approximately 75 years, and $\Delta S(0,1)$ equals $(R - 1)\ \lambda_0(0)$, hence

$$LLE(0) = (R - 1)\,\ell_0(0)\,[1 + L(1)] \qquad (13)$$

This is equivalent to assuming that in the absence of air pollution a baby would have lived a normal life span, about [1 + L(1)] and here assumed equal to 76 years. This is an upper bound, but it is perhaps not too unrealistic since many babies end up living a normal healthy life despite a fragile infancy.

The result for LLE, per million persons, is shown in the last line of Table 1. Compared with adult mortality, the LLE due to infant mortality is an order of magnitude smaller. Even though there are large uncertainties in the epidemiological studies, further compounded for the comparison by uncertainties of the PM_{10}/TSP and $PM_{2.5}$/PM_{10} ratios, it seems unlikely that infant mortality would make a large contribution to the total air pollution mortality of the population. To explain the smallness of infant mortality, despite the maximal assumption of 76 years of life lost per infant death, note that the age-specific mortality around 50 is comparable to the rate for infants and increases exponentially with age; for infant mortality only a one-year cohort is affected by the 1-yr pulse, whereas the result for adults includes all cohorts above 30.

5.4. Groups with Different Sensitivities to Pollution

It is likely that different individuals and groups have different sensitivities to pollution. If the infants who are affected by pollution have a lower life expectancy to begin with, then the loss per infant death is less than assumed above. Since LLE is uniquely determined by the functions $\ell_0(x)$ and $\mu(x)$, this uncertainty would not be there if one had complete and accurate $\mu(x)$ data for all ages x, for an exposed and an unexposed cohort, but in practice that is not the case.

It is interesting to ask how this question of different sensitivities affects the LLE estimates for adults. So far all calculations of LLE for adults have assumed that R is independent of age x, since Pope et al provide no information on a possible variation of R with age. But suppose the population consists of two groups, one sensitive, the other insensitive to pollution. With pollution the sensitive group has a higher mortality and hence a lower survival curve; its share of the total population shrinks with age and so the average sensitivity of the remaining population decreases with age. If the sensitive group has a lower life expectancy to begin with, pollution will increase the share of the long lived group at high age. Thus an epidemiological study could even find the seemingly paradoxical result that a harmful substance reduces the mortality at high age.

The age dependence of relative risk carries information on this issue. However, unless one can identify the groups, one does not know how much of the age dependence is due to the variation of risk in the sensitive group and how much is due to the change in the composition of the total population.

Krewski et al looked at the variation of relative risk with age; see the results reproduced here in Table 2. There does not appear to be a significant variation, although age resolution and confidence intervals are not sufficiently fine to settle the issue. For a hint at what might be found with better data, one can look at smoking, in the study of Doll et al. [1994] who followed a cohort of 34,000 male medical doctors in the UK over a period of 40 years. Taking the average of the cohorts from 1951 to 1991 in Fig.5 of this reference, the relative risk R for smokers is approximately constant at 2.4 below age 60 and decreases in approximately linear fashion, reaching 1.3 at 90; the average risk of the entire cohort is 1.78. There is a marked decrease of R

with age above 60. This does not mean that older people are less affected by smoking; rather the share of surviving smokers in the population decreases with age.

Table 2. Age dependence of relative risk R of all-cause mortality associated with an increase in median $PM_{2.5}$ by 24.5 $\mu g/m^3$. From Table 21 of Krewski et al [2000].

Age at Enrollment	Percent of Cohort	Relative risk R (95% confid. interval)
<50	29.3	1.19 (0.91–1.56)
50–60	36.4	1.13 (0.97–1.30)
>60	34.3	1.19 (1.09–1.29)

Leksell & Rabl tried to estimate how much such a variation with age might change the LLE; they found only a relatively small effect (perhaps ten percent in either direction) for the LLE due to adult mortality, although it is difficult to be precise in view of the lack of data.

5.5. LLE From Short Term Studies

It is interesting to make a comparison with the LLE from short term studies. Even though there is little information about LLE per death, one can get a rough idea by assuming, for the sake of argument, 6 months per death as population average. For acute mortality due to PM, the most comprehensive source is HEI [2001] because it lists the results of the three most important studies:

- 0.05% per $\mu g/m^3$, by Samet et al [2000a and b], based on the 90 largest US cities;
- 0.06% per $\mu g/m^3$, by Katsouyanni et al [1997], based on APHEA results for 12 European cities; and
- 0.07% per $\mu g/m^3$ by Levy et al [2000], based on a meta-analysis of 29 studies in 23 locations in Europe and North and South America;

all three referring to PM_{10}. Taking the central value of

$$\beta = 0.06\% \text{ per } \mu g/m^3 \text{ of } PM_{10}. \tag{14}$$

and assuming 10000 deaths/yr per million as typical value of the reference mortality rate, this implies an average LLE of

$$10000 \text{ deaths/yr per million} * 0.0006 \text{ per } (\mu g_{PM10}/m^3) * 0.5 \text{ yr}_{life}/\text{death} \tag{15}$$
$$= 3 \text{ yr}_{life}/(\text{yr}_{exp} \cdot \mu g_{PM10}/m^3) \text{ per million persons, if 6 months per death.}$$

This is only about 1% of the total found by long term studies; even with LLE/death = 5 yr it would be only 10%. With any reasonable assumption for the LLE per acute death one finds that the mortality observed by short term studies is a small contribution to the total impact (and in any case it is included in the results of the long term studies by their very design). This is entirely plausible when one considers what time series studies would be able to observe about mortality from smoking if applied in a hypothetical country where cigarette smoking is forbidden on Sundays.

6. Number of Deaths

It is natural to ask whether information on the number of air pollution deaths can be extracted from cohort studies. Let $\beta(x)$ = age distribution, defined such that $\beta(x)\,\Delta x$ is the fraction of the population between ages x and $x + \Delta x$. In a population of N_{pop} individuals, with age distribution $\beta(x)$ and age-specific mortality $\mu(x)$, the number of deaths per time interval Δt (the interval used for specifying μ and n_{deaths}) is

$$n_{deaths,0} = N_{pop} \int_0 \beta(x)\,\mu_0(x)\,dx \qquad . \tag{16}$$

If pollution increases $\mu(x)$ by $\Delta\mu(x)=(R-1)\,\mu_0(x)$, it is therefore tempting to interpret the quantity

$$\Delta n_{deaths} = N_{pop} \int_0 \beta(x)\,\Delta\mu(x)\,dx = (R-1)\,n_{deaths,0} \tag{17}$$

as deaths due to this pollution, and several authors have done so. However, whereas Eq.17 would indeed be correct for immediate deaths such as traffic accidents, it is wrong for air pollution because it fails to take into account delayed deaths.

That something is wrong with the calculation of Δn_{deaths} according to Eq.17 is suggested by the following argument. The extra number of deaths of Eq.17 is proportional to the pollution increment, and so is the LLE as shown by Leksell & Rabl [2000]. This implies that the LLE per air pollution death (the number turns out to be about 10 years) is independent of the pollution exposure, a result that would be reasonable for cancers [Thomas et al 1992] but not for cardio-pulmonary deaths associated with PM. In fact, for chronic air pollution mortality a mechanism of the kind proposed by Evans and Wolff [1996] seem more plausible: fine particles reduce cardio-pulmonary function in proportion to exposure. Cardio-pulmonary function decreases naturally with age, and people die if it falls below a critical threshold. With such a mechanism the moment of failure is advanced in proportion to exposure and so is the LLE per death.

There is probably a difference in the mode of action between air pollution and carcinogens. Carcinogens act like bullets fired into a crowd: the number of cancers is proportional to the exposure and the LLE per cancer death is about ten to fifteen years, independent of exposure. Air pollution, by contrast, is more likely to be analogous to tear gas dispersed above a crowd: everybody is affected (or everybody among sensitive individuals) and the effect on each individual is proportional to the dose.

To see whether Eq.17 could be modified to account for delayed effects, let us simplify the situation by considering just a single cohort rather than an entire population. Consider two otherwise identical cohorts of age x at time t = 0, one exposed to pollution, the other not. Fig.2 shows schematically how the death rate of these cohorts might evolve with t, the death rate being

$$\frac{dn_{death}}{dt} = N_{cohort}\, S_\mu(x,x+t)\, \mu(x+t) \qquad , \tag{18}$$

where N_{cohort} = number of individuals in the cohort at t=0, and $S_{\lambda}(x,x+t)$ = fraction of cohort that survives until age x+t if the age-specific mortality is λ. The integral of this rate from t=0 to t_{max}, the time when the last cohort member dies, is equal to N_{cohort}. For the exposed cohort λ in this equation, including its appearance in $S_{\lambda}(x,x+t)$, is increased by the relative risk R. Of course the number of individuals in the cohort, and thus the total number of deaths, is not changed by exposure. One could in principle try to observe the instantaneous change in the death rate, i.e. the difference between the solid and the dashed thick lines in Fig.2, but even that does not tell us how many deaths are attributable to pollution: the difference could be due to many individuals suffering a small loss per person or a few individuals losing much.

Such a constraint does not apply to the effect of exposure on the deaths due to a specific cause, for instance lung cancer (thin lines in Fig.2): they can increase, with concomitant decrease in deaths due to other causes. Since much of the impact of air pollution is cardio-pulmonary and the cohort study of Pope et al [2002] provides results for the increase in cardio-pulmonary mortality, it might seem as if one could calculate at least the number of cardio-pulmonary deaths due to pollution, analogous to what is commonly done for cancers [e.g. Thomas et al 1992]. However, such a calculation would count only the extra cardio-pulmonary deaths in the exposed cohort; it would not count cardio-pulmonary deaths that have occurred earlier because of pollution but would have occurred anyway, and thus it would not correctly express the full meaning of "cardio-pulmonary deaths due to pollution". By contrast, it would be meaningful for lung cancers due to air pollution to the extent that air pollution does not advance the time of a cancer death.

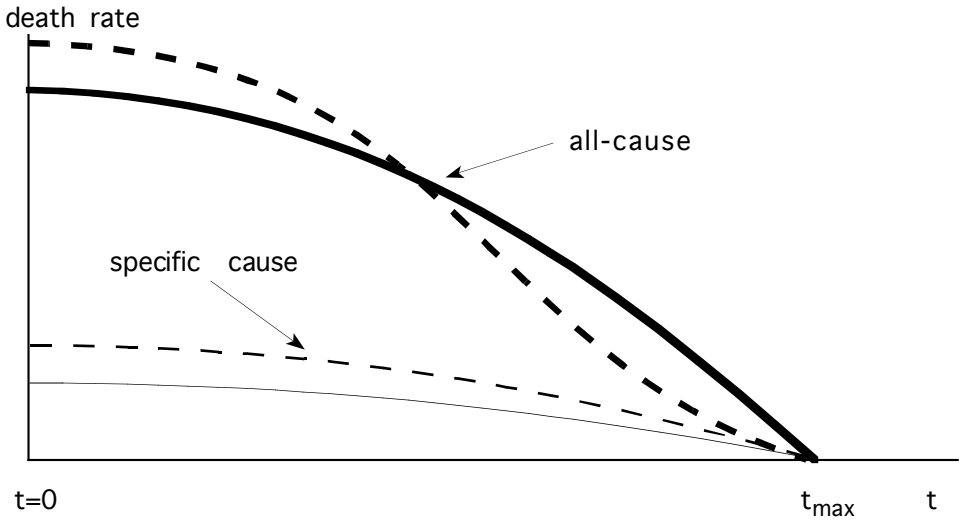

Fig.2. Possible evolution of the death rate, Eq.18, of a cohort that has age x at t=0. Thick lines = total (all-cause) rate, thin lines = rate due to a specific cause; solid lines = without, dashed lines = with exposure to pollution. The areas under the thick lines (all-cause) are equal. For a specific cause (thin lines) the area can increase, compensated by decrease from other causes.

It is instructive to give another argument why the total number of air pollution deaths is not observable by long term studies. Consider two cohorts of age x that are identical, except that cohort 2 is exposed to a constant level of air pollution, while cohort 1 is not. Suppose these cohorts consist of a mixture of two types of individuals: a fraction f_s of the cohort is sensitive to pollution and a fraction 1-f_s is not; the respective age-specific mortality rates are λ_s and λ_i. A priori f_s and the individual rates are unknown. Pollution increases λ_s by a factor R_s. The number

of deaths attributable to air pollution is times the number of individuals in the cohort. However, only the rates λ_1 and λ_2 can be observed, the relative risk being $R = \lambda_2/\lambda_1$. Thus we have the following two equations

$$\lambda_1 = f_s \lambda_s + (1 - f_s) \lambda_i \quad , \tag{19}$$

and

$$\lambda_2 = R_s f_s \lambda_s + (1 - f_s) \lambda_i \quad , \tag{20}$$

with 4 unkowns R_s, f_s, λ_s and λ_i. This is not sufficient to determine the fraction f_s. The argument carries over to more complicated examples, e.g. exposures that vary in time: one could find an extra time dependence, but the number of equations remains insufficient to determine f_s.

7. Conclusions

This paper has examined what impact indicators can be inferred from epidemiological studies of air pollution mortality. Ordinary short term studies (time series) observe a change in daily death rate attributable to acute effects of pollution, but without any information on the LLE per death. However, with any reasonable assumption about the LLE per death, the contribution to the total LLE due to air pollution is very small. The latter has been measured by long term studies (cohort studies); their results allow the calculation of population total LLE, but not the number of premature deaths.

Several arguments have been presented why number of premature deaths is not appropriate whereas loss of life expectancy (LLE) is. One of these arguments involved an examination of what can be observed about the mortality attributable to a contributing cause of death (e.g. air pollution, smoking, or lack of exercise). The total number of attributable deaths is observable only for

(i) primary causes (identified on death certificates, e.g. accidents or cancers), and
(ii) contributing causes if their action is sufficiently instantaneous that the initial increase in death rate is not obscured by the subsequent depletion of the population.

For air pollution most of the impact is not instantaneous but the cumulative result after years of exposure, therefore the total number of attributable deaths is not observable.

To illustrate these ideas, the LLE has been calculated based on the best currently available epidemiological studies. The results are summarized in Table 3. Whereas acute mortality is automatically included in the chronic mortality studies, infant mortality is not and could be added, although we hesitate to do so because we have only an upper limit; in any case it would not change the result significantly compared to the uncertainties. Taking the limits of the 95% confidence intervals of the chronic mortality study, the numbers could be about 50% smaller or larger.

To put these numbers in perspective, note that typical concentrations of PM_{10} in urban areas of the USA and the EU are around 20 to 30 $\mu g/m^3$ [EPA 1997]. Let us suppose a permanent reduction by 15 $\mu g/m^3$ as a reasonable policy goal. Multiplying 330 $yr_{life}/(yr_{exp} \cdot \mu g/m^3)$ per million persons by 15 $\mu g/m^3$ * 76 yr for the corresponding difference in life time exposure, one finds,

$$330\ yr_{life}/(yr_{exp} \cdot \mu g_{PM10}/m^3) \text{ per million persons} * 15\ \mu g_{PM10}/m^3 * 76\ yr_{exp} \tag{21}$$

$= 0.38\ yr_{life}$ per person for 15 $\mu g/m^3$ of PM_{10} over lifetime.

This reduction by 15 $\mu g/m^3$ increases average life expectancy by about 4.5 months.

Table 3. LLE attributable to an increase in mean PM_{10} concentration during 1 year, in $yr_{life}/(yr_{exp}\cdot\mu g/m^3)$ per million persons.

Type of study	**LLE** $yr_{life}/(yr_{exp}\cdot\mu g/m^3)$ per million	**Reference**
Chronic mortality, adults over 30	330	Pope et al [2002]
Acute mortality, total population	3 (if 6 months/death)	Samet et al [2000a and b], Katsouyanni et al [1997], Levy et al [2000]
Chronic mortality, Infants (<12 months)	18 [a]	Woodruff et al [1997], Bobak & Leon [1999]

[a] upper limit corresponding to assumption that infants who died because of pollution could have lived a normal life span of 76 yr.

Acknowledgments

This work has been supported in part by the ExternE Project of the European Commission DG Research. I am grateful to Vladimir Demin, Ingemar Leksell and Richard Wilson for discussions, to Martin Bobak for information on the data used for the study in the Czech Republic, and to Rick Burnett, Michael Jerrett, George Thurston and Warren White for helpful explanations of their papers. The exceptionally detailed and constructive comments by reviewers of the previous versions are very much appreciated.

References

Abbey DE, N Nishino, WF McDonnell, RJ Burchette, SF Knutsen, WL Beeson & JX Yang 1999. "Long-term inhalable particles and other air pollutants related to mortality in nonsmokers". *Am. J. Respir. Crit. Care Med.,* vol. 159, 373-382.

Abt 2000. "The Particulate-Related Health Benefits of Reducing Power Plant Emissions." October 2000. Prepared for EPA by Abt Associates Inc., 4800 Montgomery Lane, Bethesda,MD 20814-5341.

Bobak M and DA Leon 1999. "The effect of air pollution on infant mortality appears specific for respiratory causes in the postneonatal period". *Epidemiology* 10(6), 666-670.

Brunekreef B 1997. "Air pollution and life expectancy: is there a relation?" *Occupational and Environmental Medicine*, vol.54, pp.781-784.

Dockery DW, CA Pope III, X Xu, JD Spengler, JH Ware, ME Fay, BG Ferris and FE Speizer 1993. "An association between air pollution and mortality in six US cities". *New England J of Medicine*, vol.329, p.1753-1759.

Doll R, R Peto, K Wheatley, R Gray and I Sutherland 1994. "Mortality in relation to smoking: 40 years' observations on male British doctors". *British Medical Journal*, 309, pp.901-911.

EPA 1997. "National Air Quality and Emissions Trends Report, 1997". USEPA http://www.epa.gov/oar/aqtrnd97/chapter2.pdf

Evans J & S Wolff 1996. "Modeling of air pollution impacts: one possible explanation of the observed chronic mortality". Chapter 9, pp189-204 of Wilson R & JD Spengler, editors 1996. *Particles in Our Air: Concentrations and Health Effects*. Harvard University Press, Cambridge, MA.

ExternE 1998. ExternE: Externalities of Energy. 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). Published by European Commission, Directorate-General XII, Science Research and Development. Office for Official Publications of the European Communities, L-2920 Luxembourg. Results are also available at http://ExternE.jrc.es/publica.html.

ExternE 2000. "External Costs of Energy Conversion – Improvement of the Externe Methodology And Assessment Of Energy-Related Transport Externalities". Final Report for Contract JOS3-CT97-0015, published as *Environmental External Costs of Transport*. R. Friedrich & P. Bickel, editors. Springer Verlag Heidelberg 2001.

HEI 2001. "Airborne particles and health: HEI epidemiologic evidence". HEI Perspectives, June 2001. Health Effects Institute, Charlestown Navy Yard, 120 Second Avenue, Boston, MA 02129-4533. Available at http://www.healtheffects.org/

Johannesson M & P-O Johansson 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". *J Risk and Uncertainty*, 13, 163-174.

Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossi G, Wojtyniak B, Sunyer J, Bacharova L, Schouten JP, Ponka A, Anderson HR. 1997. "Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: Results from time series data from the APHEA project." *British Med. J* 314:1658–1663.

Krewski D, Burnett R T, Goldberg M S, Hoover K, Siemiatycki J, Abrahamowicz M, White W H, et al. 2000. "Particle Epidemiology Reanalysis Project". Health Effects Institute, Cambridge MA. Available at http://www.healtheffects.org/pubs-recent.htm.

Krupnick A, A. Alberini, M. Cropper, N. Simon, B. O'Brien, R. Goeree & M. Heintzelman 2000. "What are Older People Willing to Pay to Reduce Their Risk of Dying?" Resources for the Future, 1616 P Street, NW, Washington, DC 20036. Paper presented to the AERE Sessions, ASSA Meetings, Boston, Mass., January 7, 2000.

Kuenzli N, R Kaiser, S Medina, M Studnicka, O Chanel, M Herry, F Horak, V Puybonnieux-Texier, P Quénel, J Schneider, R Seetaler J-C Vergnaud & H Sommer 2000. "Public health impact of outdoor and traffic-related air pollution: a European assessment". *Lancet*, 356 (Sept.), 795-801.

Kuenzli N, Medina S, Kaiser R, Quénel P, Horak F, & Studnika M 2001. "Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies?" *Amercian J Epidemiology*, 153 (11), 1050-1055.

Leksell L & A Rabl 2001. "Air Pollution and Mortality: Quantification and Valuation of Years of Life Lost". *Risk Analysis*, vol.21(5), 843-857.

Levy JI, Hammitt, JK Yanagisawa Y & Spengler JD 1999. "Development of a new damage function model for power plants: methodology and applications". *Environmental Science & Technology,* vol.33(24), 4364-4372.

Levy JI, Hammitt, JK, Spengler JD. 2000. "Estimating the mortality impacts of particulate matter: What can be learned from between-study variability?" *Environ Health Perspect* 108(2):109–117.

McMichael, A J; Anderson, H R; Brunekreef, B; Cohen, A J 1998. "Inappropriate use of daily mortality analyses to estimate longer-term mortality effects of air pollution." *International Journal of Epidemiology*, Vol. 27 (3), 450-453.

NCHS 1999. National Center for Health Statistics, Hyattsville, Maryland. *National Vital Statistics Reports,* Vol. 47, No. 19, June 30, 1999. Available at http://www.cdc.gov/nchs/fastats/infmort.htm.

ORNL/RFF 1994. *External Costs and Benefits of Fuel Cycles*. Prepared by Oak Ridge National Laboratory and Resources for the Future. Edited by Russell Lee, Oak Ridge National Laboratory, Oak Ridge, TN 37831.

Pope CA, MJ Thun, MM Namboodri, DW Dockery, JS Evans, FE Speizer, & CW Heath 1995. "Particulate air pollution as a predictor of mortality in a prospective study of US adults". *Amer. J. of Resp. Critical Care Med.,* vol.151, 669-674.

Pope CA, RT Burnett, MJ Thun, EE Calle, D Krewski, K Ito, & GD Thurston 2002. "Lung cancer, cardiopulmonary mortality, and long term exposure to fine particulate air pollution ". *J. Amer. Med. Assoc.,* vol.287(9), 1132-1141.

Rabl A 1998. "Mortality risks of air pollution: the role of exposure-response functions". *Journal of Hazardous Materials*, vol.61, pp.91-98.

Robins JM & S Greenland 1991. "Estimability and estimation of expected years of life lost due to a hazardous exposure". *Statistics in Medicine*, vol.10, 79-83.

Rowe RD, CM Lang, LG Chestnut, D Latimer, D Rae, SM Bernow & DWhite. 1995. *The New York Electricity Externality Study*. Oceana Publications, Dobbs Ferry, New York.

Samet JM, Dominici F, Zeger SL, Schwartz J, Dockery DW. 2000a. "The National Morbidity, Mortality and Air Pollution Study, Part I: Methods and Methodologic Issues." Research Report 94, Part I. Health Effects Institute, Cambridge MA. Available at http://www.healtheffects.org/

Samet JM, F Dominici, FC Curriero, I Coursac & SL Zeger. 2000b. "Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987–1994". *N Engl J Medicine*, vol.343(24), 1742-1749.

Schwartz J. 2000. "Mortality Displacement and Long-term Exposure Effects Related to Air Pollution and Mortality". Section 4 of Samet JM, F Dominici, SL Zeger, J Schwartz, W Dockery et al. 2000a. "National Morbidity, Mortality, and Air Pollution Study". Part II: National Morbidity, Mortality, and Air Pollution in the United States. Research report 94, The Health Effects Institute, Cambridge MA.

Thomas T, S Darby, F Fagnani, P Hubert, M Vaeth & K Weiss. 1992. "Definition and estimation of lifetime detriment from radiation exposures: principles and methods". Health Physics, vol. 63(3), 259-272.

Wilson R & EAC Crouch 2001. *Risk-Benefit Analysis*. Harvard University Press, Cambridge, MA.

Woodruff TJ, Grillo J, Schoendorf KC 1997. "The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States". *Environ Health Perspect*, vol.105(6), 608-612.

Zeger SL, Dominici F, & Samet JM. 2000. "Mortality Displacement–Resistant Estimates of Air Pollution Effects on Mortality". Section 3 of Samet JM, F Dominici, SL Zeger, J Schwartz, W Dockery et al. 2000a. "National Morbidity, Mortality, and Air Pollution Study". Part II: National Morbidity, Mortality, and Air Pollution in the United States. Research report 94, The Health Effects Institute, Cambridge MA. See also *Epidemiology*, vol.10, 171-175 (1999).