Impacts of Air Pollution:

General Relationships and Site Dependence

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(Fig.5 has been updated since the version in the journal)

Abstract

We analyze the structure of the impact pathway methodology and show that, for the important problem of calculating the expectation value of the marginal damage from a point pollution source, the equations can be simplified greatly. In particular, in a uniform world (uniform receptor density and uniform atmosphere) they can be integrated in closed form to yield a very simple formula for the total damage D_{uni} in a uniform world. The generalization to secondary pollutants is straightforward. We then test the relevance of this simple formula in the real world, by using curve fits to the results of detailed models for atmospheric dispersion and chemistry and by varying the receptor density according to typical patterns. We also compare D_{uni} with the output of a software package that carries out an accurate numerical integration of atmospheric dispersion results over geographic data for population and other receptors. Evaluating a set of five sites in France that appear generally representative, ranging from rural to extremely urban, we find that the true damage is within a factor of about three of D_{uni}. This remarkably small sensitivity to geographical detail can be explained by the conservation of matter and the large geographic range of the dispersion. We provide guidelines for several questions that are relevant for environmental policy:
What are the functional relations and key parameters, as input to an uncertainty analysis?
How does the damage vary with key parameters, e.g. stack height?
How much accuracy and resolution is needed, in the atmospheric modeling and in the data for receptor distribution?
How large is the error if one uses typical average values rather than detailed calculations?

Key words: pollution damage, atmospheric modeling, dose-response function, receptor data, impact pathway methodology, stack height, life cycle analysis

1. Introduction

Rational management of the environment requires an assessment of the damage caused by pollution. Pollutants can be emitted to air, water or soil. The majority of pollutants are first emitted into the air, even if they later pass into the water or the soil. A problem arises from the fact that air pollution damage depends on the sites of emission and receptors, whereas from the point of view of policy it is not practical to try and take into account each and every local detail. Rather one needs guidelines that are a compromise between precision and practicality, in the spirit of "Better approximately right than precisely wrong!"

To provide such guidelines for environmental policy, the present paper addresses the following questions:

- What are the functional relations and key parameters, as input to an uncertainty analysis?
- How does the damage vary with key parameters, e.g. stack height?
- How much accuracy and resolution is needed, in the atmospheric modeling and in the data for receptor distribution?
- How large is the error if one uses typical average values rather than detailed calculations?

This paper focuses on the impacts of air pollution; some of the key features of the functional relations are, however, applicable even for water and soil pollution. We address only expectation values of pollution damage, not damage from particular pollution episodes.

We analyze the structure of the impact pathway methodology and show that for the important problem of marginal damage from a point pollution source the equations can be simplified greatly. In particular, in a uniform world (uniform receptor density and uniform atmosphere) they can be integrated in closed form to yield a very simple formula for the total damage D_{uni} in a uniform world. The generalization to secondary pollutants is straightforward. We then test the relevance of this simple formula in the real world, by using curve fits to the results of detailed models for atmospheric dispersion and chemistry and by varying the receptor density according to typical patterns. We also compare D_{uni} with the output of PATHWAYS [Curtiss and Rabl 1995], a software package that carries out an accurate numerical integration of atmospheric dispersion results over real geographic data for population and other receptors.

We find that the simple formula is surprisingly accurate. For example, in France the damage ranges from about one third D_{uni} to three times D_{uni}, if a power plant is moved from a rural region on the Atlantic Coast to Paris (a metropolitan area with 11 million people, about 20% of the population of France). For another test of the D_{uni} formula we calculate how much the total damage changes relative to D_{uni} if the real receptor density has a concentric ring pattern with equal radial increments, being twice the average in the first, third, fifth, ... rings (e.g., from 0 to 50 km, 100 to 150 km, 200 to 250 km, ...) and zero in the second, forth, ... rings (50 to 100 km, 150 to 200 km, ...), or vice versa. The change is surprisingly small, typically less than about ten percent. This implies that the damage calculation is very tolerant to inaccuracies in geographical contours.

The explanation for this insensitivity to geographical detail lies in the law of conservation of matter. Loosely speaking, what goes up must come down. If more comes down in one region,

less comes down in another. In particular, in the limit of uniform receptor density, the total damage depends only on a single atmospheric characteristic: the removal rate of the pollutant.

2. Impact Pathway Analysis

The logically correct way to analyze environmental impacts is the impact pathway methodology whose principal steps are the following:

- characterization of the relevant technologies and the environmental burdens they impose (e. g. kg s^{-1} of particulates emitted by the plant);
- calculation of increased pollutant concentration in all affected regions (e. g. µg m^{-3} of particulates, using models of atmospheric dispersion and chemistry);
- calculation of physical impacts (e. g. number of cases of asthma due to these particulates, using a dose-response function);
- in some cases a fourth step may be desirable: the economic valuation of these impacts (e. g. multiplication by the cost of a case of asthma).

The numbers are summed over all receptors that are affected. Formally the procedure can be represented as an equation for the incremental damage D due to an incremental quantity Q of a pollutant emitted by the plant

$$D = \sum_i f_{dr,i}(f_{disp \rightarrow i}(Q)) \quad , \tag{1}$$

where
$f_{disp \rightarrow i}(Q) = c$ = increase in pollutant concentration for receptor i, and
$f_{dr,i}(c)$ = dose-response function for receptor i;
the summation index i runs over all receptors (population, crops, buildings, ...) that may be affected by this pollutant. The notation allows the possibility that the impact may be different for different individual receptors. This equation expresses the damage in functional form, hence this methodology is also known under the name damage function. Of course, while this methodology is logically correct, the practical implementation may not always be feasible for lack of appropriate data or models.

The dose-response function

$$Y = f_{dr}(X) \tag{2}$$

relates the quantity X of a pollutant that affects a receptor (e.g. population) to the physical impact Y on this receptor (e.g. incremental number of deaths). In the narrow sense of the term, X should be the dose actually absorbed by a receptor. But often one uses, as we do in the present paper, the term dose-response function in the sense of exposure-response function where X represents the concentration of a pollutant in the ambient air; in that case $f_{dr}(X)$ accounts implicitly for the absorption of the pollutant from the air into the body. Dose-response functions for the classical air pollutants (NO_x, SO_x, O_3, and particulates) are typically of that kind. One can even define aggregated dose-response functions that include more complicated pathways, for instance dioxins passing through the food chain, if one interprets the dose-response function

to include the aggregated effects of the pathways from a point at the earth's surface to all final receptors.

It will be convenient to write the damage as an integral over land area by introducing $\rho(\mathbf{x})$, the density of receptors at point $\mathbf{x} = (x,y)$,

$$D = \int dx \int dy \quad \rho(\mathbf{x})\, f_{dr}(\mathbf{x}, c(\mathbf{x})) \tag{3}$$

where $c(\mathbf{x}) = f_{disp \rightarrow \mathbf{x}}(Q)$ is the concentration increase at $\mathbf{x}$ due to emission Q. Eqs.1 and 3 describe the damage D due to a point source; they can of course be extended to a set of sources, e.g. line or area sources, by summation or integration over the sources.

In the above we have implicitly assumed a steady state situation where a pollutant is emitted at a constant rate Q, $c(\mathbf{x})$ is the steady state concentration increment and D is the resulting damage per time. Eq.3 can readily be extended to time varying emissions by indicating a time dependence as Q(t'), $c(\mathbf{x},t')$ and D(t'). Time delays of a damage can be indicated by writing the dose-response function as $f_{dr}(\mathbf{x}, c(\mathbf{x}), t-t')$.

3. Dose-response functions

3.1. The form of the dose-response function

By definition a dose-response function starts at the origin, and in most cases it increases monotonically with dose X, as sketched schematically in Fig.1. At very high doses the function may level off in S-shaped fashion, implying saturation. Dose-response functions are determined from epidemiological studies or from laboratory studies. Since the latter are mostly limited to animals, the extrapolation to humans introduces large uncertainties. Another major difficulty is that one needs relatively high doses in order to obtain observable nonzero responses in a sample of realistic size; such doses are usually far in excess of the levels one is concerned with in environmental impact studies. Thus there is a serious problem of how to extrapolate from the observed data towards low doses. Fig.1 indicates several possibilities. The simplest is the linear model, i.e. a straight line from the origin through the observed data point(s). Cancer from radioactivity is an example. Linearity also seems to be observed for mortality from fine particulates [Dockery et al. 1993, Dockery and Pope 1994, Lipfert 1994].

Another possibility is a straight line down to some threshold, and zero effect below that threshold. Thresholds occur when an organism has a natural repair mechanism that can prevent or counteract damage up to a certain limit. Many dose-response functions for non cancer toxicity are of this type.

There is even the possibility of a "fertilizer effect" at low doses, as indicated by the dashed line in Fig.1. This can be observed, for example, in the dose-response functions for the impact of NO_X and SO_X on crops: a low dose of these pollutants can increase the crop yield, in other words the damage is negative. Such a fertilizer effect can occur with pollutants that provide trace elements needed by an organism. It depends on local conditions, in particular the overall balance

of nutrients. The fertilizer effect illustrates the link between the understanding of the underlying processes and the choice of the appropriate form for the dose-response function: since N and S are known to be important nutrients for plants, a functional form like the dashed line in Fig.1 is the most plausible.

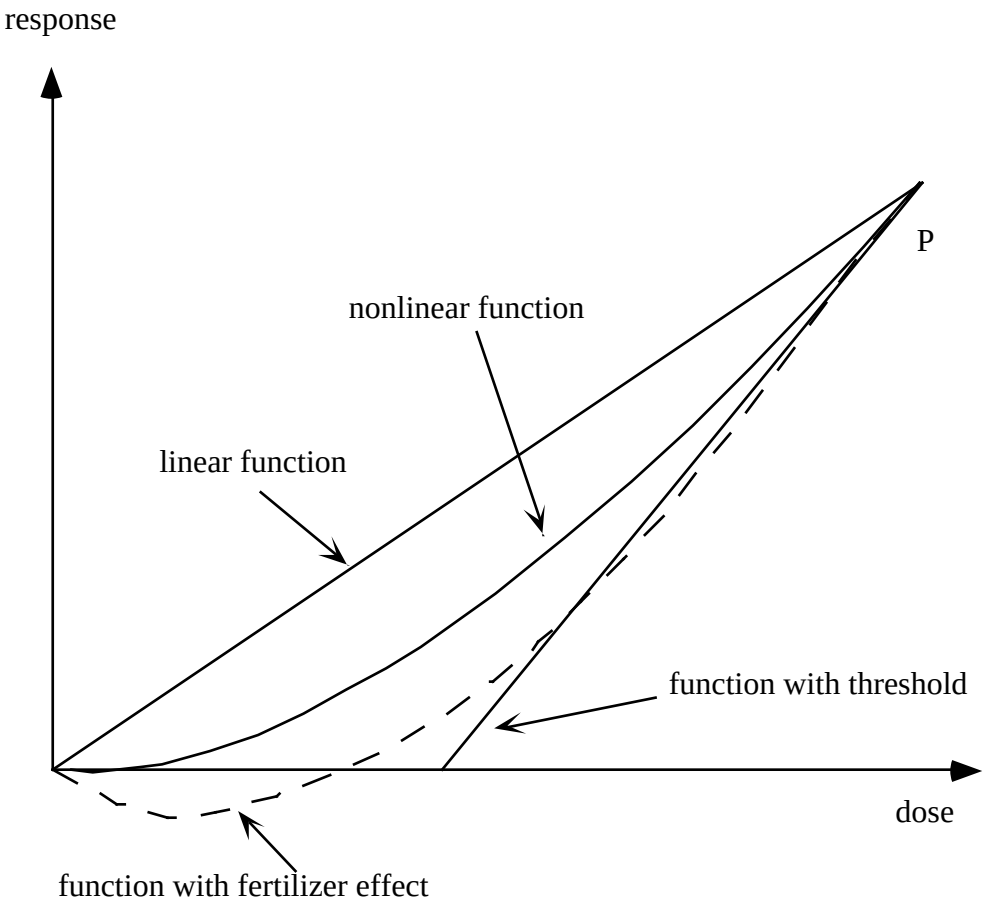


Fig.1. Possible behavior of dose-response functions at low doses:
the four functions shown have the same value at P. For the function with threshold the discontinuity in slope at the threshold is a simplification; in reality there is a smooth transition.

If nothing is known about a threshold, the dose-response function could be anywhere between zero and the straight line through the origin, for instance the curved line shown in Fig.1. A priori there is no general rule about the extrapolation to low doses, other than there being no known cases of a dose-response function above the straight line. There is even a case where the same substance causes different cancers according to different dose-response functions, one with and one without threshold. This was established in an experiment (sometimes referred to as the megamouse experiment) in which some 24000 mice were exposed to the carcinogen 2-acetyl-amino-fluorene at several different dose levels [Frith, Littlefield and Umholtz 1981]. The response for liver tumor is linear whereas the one for bladder tumor has a threshold.

3.2. Implications of the Threshold for the Analysis

The form of the dose-response function, Fig.1, has implications for the way an impact analysis is to be carried out. It is appropriate to distinguish two extreme cases. These two extreme cases are of great practical importance, and the corresponding analysis is relatively simple.

One extreme occurs when the dose-response function is a straight line through the origin (no threshold). In that case any incremental pollution causes an impact, and the range of the analysis needs to be extended over hundreds or thousands of km if most of the impact is to be included. This situation also pertains in the presence of a threshold, if the background concentration is everywhere above this threshold. For some air pollutants, e.g. particulates, the background in most industrialized countries is above the level where effects are known to occur [Dockery et al. 1993]. Thus the question of the precise form of the dose-response function at extremely low doses is irrelevant for these pollutants: whatever the threshold, if there is one, it is below the background concentrations of interest.

The other extreme occurs if the dose-response function has a threshold that is above the background concentration of the pollutant and if the pollution added by the source does not push the concentration above the threshold. In that case there is no impact. The analysis is simple: it suffices to verify that the resulting concentrations remain below the threshold. A short range (< 50 km) dispersion model is adequate for this purpose because the peak concentration increase certainly occurs within that region.

4. Marginal Damage

From here on we limit ourselves to the important case where the dose-response function $f_{dr}(\mathbf{x},c(\mathbf{x}))$ can be approximated by

$$f_{dr}(\mathbf{x},c(\mathbf{x})) = d(\mathbf{x})\, c(\mathbf{x}) \qquad \text{where} \qquad d(\mathbf{x}) = \frac{df_{dr}(\mathbf{x},c(\mathbf{x}))}{dc} \tag{4}$$

is the slope of the dose-response function. With that assumption one can write Eq.3 for the damage in the form

$$D = \int dx \int dy \quad \rho(\mathbf{x})\, d(\mathbf{x})\, c(\mathbf{x}) \qquad . \tag{5}$$

This is obviously exact for any pollutant whose dose-response function is linear, or a straight line with a threshold that is everywhere below the background. It is also valid, regardless of dose-response function, for the evaluation of any marginal impacts, i.e. impacts from small pollutant increments because in that case one can linearize the dose-response function. Since $c(\mathbf{x})$ is linear in the emission, it follows that Eq.5, and the remainder of this paper, are equally applicable to steady state situations and to emissions that vary with time.

It will be interesting to relate the concentration $c(\mathbf{x})$ to the removal rate of the pollutant. There are essentially three mechanisms by which an air pollutant can disappear from the atmosphere [Seinfeld 1986]:

1) dry deposition (uptake at the earth's surface by soil, water or vegetation)
2) wet deposition (absorption into droplets followed by droplet removal by precipitation),
3) decay or transformation (e.g. decay of radionuclides, or chemical transformation of SO_2 to $(NH_4)_2SO_4$).

When evaluating the damage of the original pollutant, this pollutant is no longer counted in the equation once it has been transformed; rather from that point on a different dose-response function comes into play for the secondary pollutant. That issue will be addressed in Section 5.2.

The dry deposition rate is proportional to the concentration c(**x**) at the earth's surface, and it is customarily written in the form

$$F_{dry}(\mathbf{x}) = v_{dry}\, c(\mathbf{x}) \tag{6}$$

where

$F_{dry}(\mathbf{x})$ = deposition flux [in kg m^{-2} s^{-1}], and

v_{dry} = dry deposition velocity [m s^{-1}].

Wet deposition and decay or transformation can likewise be characterized in terms of fluxes $F_{wet}(\mathbf{x})$ and $F_{trans}(\mathbf{x})$, defined as the rate at which the pollutant is removed by these mechanisms per m^2 and per s. Even though in general these fluxes are not proportional to the surface concentration but rather to the average concentration in the air column above x, we can write the total removal flux

$$F(\mathbf{x}) = F_{dry}(\mathbf{x}) + F_{wet}(\mathbf{x}) + F_{trans}(\mathbf{x}) \tag{7}$$

in terms of the surface concentration c(**x**) as

$$F(\mathbf{x}) = k(\mathbf{x})\ c(\mathbf{x}) \tag{8}$$

if we allow the proportionality constant k(**x**) to vary with **x**. The units of k are m s^{-1}, like a velocity. Using F(**x**) and k(**x**) we can write the damage in the form

$$D = \int dx \int dy \quad \rho(\mathbf{x})\, d(\mathbf{x})\, F(\mathbf{x})/k(\mathbf{x}) \tag{9}$$

This equation is exact if we interpret Eq.8 as the definition of k(**x**).

5. Uniform World Model

5.1. Primary Pollutants

If the world were homogeneous, with uniform receptor density $\rho(\mathbf{x}) = \rho_{uni}$, uniform dose-response function slope $d(\mathbf{x}) = d_{uni}$, and uniform atmosphere $k(\mathbf{x}) = k_{uni}$, the integral in Eq.9 would be simply

$$D = D_{uni} = d_{uni}\, \rho_{uni}\, Q/k_{uni} \tag{10}$$

because the surface integral of the removal flux equals the emission by conservation of matter

$$Q = \int dx \int dy \quad F(\mathbf{x}) \quad . \tag{11}$$

Even though the assumption $k(\mathbf{x}) = k_{uni}$ may not appear very realistic, especially near a point source, the sensitivity to deviations from uniformity turns out to be surprisingly small, as we will demonstrate in Section 7. The reason is that for typical values of atmospheric dispersion parameters the total impact is dominated by regions sufficiently far from the source that the pollutant can be considered to be vertically well mixed in the planetary boundary layer, at least as far as expectation values are concerned.

Thus the simple Eq.10 can be a useful first estimate, good to an order of magnitude or better. Details of atmospheric dispersion do not matter very much. It is intuitively plausible that the damage is proportional to the slope d of the dose-response function, to the density ρ of receptors and to the emission rate Q. Furthermore, it is inversely proportional to the removal velocity k. If there were no removal mechanism, the pollutant concentration in the air would increase without limit and the damage through this pathway would be infinite (for the extension to more complicated pathways see Section 5.2).

Eq.10 has been derived by others, for instance by Trukenmüller and Krewitt [1993]. The originality of the present paper lies in the generality of the derivation and in the evaluation of the relevance for the real world.

5.2. Secondary Pollutants

The same approach can be used for the damage due to a secondary pollutant. We simply add a subscript 2 in Eq.5 to indicate that concentration, dose-response function and damage refer to the secondary pollutant

$$D_2 = \int dx \int dy \quad \rho_2(\mathbf{x})\, d_2(\mathbf{x})\, c_2(\mathbf{x}) \quad . \tag{12}$$

Analogous to Eq.9 for the primary pollutant, the removal of the secondary pollutant can also be characterized in terms of a removal flux $F_2(\mathbf{x})$ which can be expressed in terms of the corresponding concentration $c_2(\mathbf{x})$ if we define a proportionality factor $k_2(\mathbf{x})$ by

$$F_2(\mathbf{x}) = k_2(\mathbf{x})\ c_2(\mathbf{x}) \quad . \tag{13}$$

In a uniform world with $k_2(\mathbf{x}) = k_{2,uni}$, $d_2(\mathbf{x}) = d_{2,uni}$ and $\rho_2(\mathbf{x}) = \rho_{2,uni}$ we find a damage analogous to Eq.10

$$D_2 = D_{2,uni} = d_{2,uni}\, \rho_{2,uni}\, Q_2/k_{2,uni} \tag{14}$$

because the surface integral of the removal flux $F_2(\mathbf{x})$ equals the total quantity

$$Q_2 = \int dx \int dy \quad F_2(\mathbf{x}) \tag{15}$$

of the secondary pollutant Q_2 that has been created. Let us relate Q_2 to the emission Q_1 of the primary pollutant, by expressing the rate at which the secondary pollutant is created from the primary in terms of a flux $F_{1\text{-}2}(x)$, defined as mass of secondary pollutant created per s and per m^2 of horizontal surface

$$F_{1\text{-}2}(\mathbf{x}) = k_{1\text{-}2}(\mathbf{x})\ c_1(\mathbf{x}) \tag{16}$$

where $k_{1\text{-}2}(\mathbf{x})$ is a factor defined as local ratio of $F_{1\text{-}2}(\mathbf{x})$ and $c_1(\mathbf{x})$. Of course, the integral over the creation flux $F_{1\text{-}2}(\mathbf{x})$ is also equal to the total quantity of the secondary pollutant

$$Q_2 = \int dx \int dy \quad F_{1\text{-}2}(\mathbf{x}) \qquad . \tag{17}$$

Assuming a uniform atmosphere with $k_{1\text{-}2}(\mathbf{x}) = k_{1\text{-}2,uni}$, we can combine Eqs.12 to 17 to write the damage $D_{2,uni}$ in the form

$$D_{2,uni} = \frac{d_{2,uni}\ \rho_{2,uni}\ k_{1\text{-}2,uni}\ Q}{k_{2,uni}\ k_1} \qquad . \tag{18}$$

Incidentally, the basic features of this Section apply even to dispersion in water or soil. Of course, Eqs.10 and 18 must be applied separately in each medium and for each new pollutant that is formed. Consider for example dioxins, typically emitted into the air. Only a small part of their damage to humans arises from inhalation; most human dioxin intake occurs through the food chain, after deposition on plants and soil. If the removal rate in air were zero, there would be no damage through the deposition pathway, but the damage through air would be infinite. With finite deposition rate in air, if the removal rate in soil were zero, the concentration in the soil would build up without limit.

6. Dispersion Modeling

6.1 Gaussian Plume

A simple model for atmospheric dispersion is the gaussian plume, usually considered adequate for the short range, up to tens of km from the source [Seinfeld 1986, Zannetti 1990]. According to this model the concentration is described by the product of two gaussian distributions, one for the spread in the vertical direction and one for the spread in the horizontal direction perpendicular to the prevailing wind direction. For a point source at (0, 0, h_e) emitting at a rate Q [in kg s^{-1}] the concentration c(x,y,z) of the pollutant at a point (x,y,z), in the absence of reflecting boundaries, is given by

$$c(x,y,z) = \frac{Q}{2\,\pi\sigma_y\,\sigma_z\,v} \quad \exp\left[-\frac{1}{2}\left(\frac{y}{\sigma_y}\right)^2\right] \quad \exp\left[-\frac{1}{2}\left(\frac{(z-h_e)}{\sigma_z}\right)^2\right] \tag{19}$$

where the wind velocity is v, in the x direction. Note that we take h_e to be the effective emission height, including the plume rise.

The plume width parameters σ_y and σ_z are based on empirical correlations and take into account the relevant meteorological conditions. There are many different models for the plume width parameters. A particular example is the Brookhaven model [as cited by Zannetti 1990] given, for both σ_y and σ_z, by equations of the form

$$\sigma = a\, x^b \tag{20}$$

with numerical coefficients a and b; the coefficients for the vertical (z) direction are listed in columns two and three of Table 1, for future reference. Also of interest will be the distance x at which the vertical spread becomes equal to height H of the planetary boundary layer. This distance, listed in the last three columns of Table 6.2 for several values of H, is an indication where one can assume the pollutant to be vertically mixed in the atmosphere.

Table 1. Coefficients a and b of Brookhaven model for vertical plume width $\sigma = \sigma_z$ of Eq.20, in m, (columns two and three). The last three columns indicate distance x where vertical plume width σ_z of Eq.20 equal to height H of planetary boundary layer, for several values of H.

	Coefficients		Distance x [in km] where σ = H		
Gustiness category	a	b	for H= 400 m	for H= 800 m	for H= 1600 m
B2 (very unstable)	0.41	0.91	1.9	4.1	8.8
B1 (unstable)	0.33	0.86	3.9	8.6	19.3
C (neutral)	0.22	0.78	15.1	36.7	89.3
D (stable)	0.06	0.71	243.1	645.3	1713.0

Beyond the distance where a significant portion of the plume of Eq.19 would extend beyond the planetary boundary layer or below the ground, the equation needs to be modified to account for absorption or reflection. With the usual assumption of total reflection, both above and below, the second exponential in Eq.19 is replaced according to

$$S(z) = \exp\left[-\frac{1}{2}\left(\frac{z - h_e}{\sigma_z}\right)^2\right] \tag{21}$$

$$\rightarrow \sum_j \left\{ \exp\left[-\frac{1}{2}\left(\frac{z + 2\,j\,H - h_e}{\sigma_z}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{z + 2\,j\,H + h_e}{\sigma_z}\right)^2\right] \right\}$$

the sum going over j = 0, ±1, ±2, ... We will need this equation only at ground level, i.e., at z = 0.

To avoid slow convergence of this sum and to speed up the calculation, Zannetti [1990] recommends the following scheme which approximates Eq. 21 within 1.3%:

- for $\sigma_z/H \le 0.63$ (22a)
  truncate Eq.21 at j = 0, ±1

- for $0.63 < \sigma_z/H \le 1.08$

  replace $S(z) \rightarrow \frac{\sqrt{2\pi}\sigma_z}{H}(1 - ß^2)\quad [1 + ß^2 + 2\,ß\cos(\pi z/H)\cos(\pi h_e/H)]$ (22b)

  where $ß = \exp\left(-\frac{1}{2}\left(\frac{\pi\sigma_z}{H}\right)^2\right)$

- for $1.08 < \sigma_z/H$ (22c)

  replace $S(z) \rightarrow \frac{\sqrt{2\pi}\sigma_z}{H}$.

It is easy to see that Eq.22c corresponds to uniform vertical mixing (see also Eq.25 below).

To account for the removal of the pollutant, the right hand side of the gaussian plume equation is further multiplied by a decay factor of the form

$$\text{decay factor} = \exp(-\beta x) \tag{23}$$

where β can be a function of x. As first approximation β may be taken as constant

$$\beta = \frac{k}{v\,H} \tag{24}$$

where k is defined by Eq.8 and assumed site-independent.

6.2. Dispersion Over Large Distances

For the dispersion over large distances we use results from a detailed computer simulation, the EMEP model (European Monitoring and Evaluation Programme) of the Norwegian Meteorological Service [Barrett 1992, Sandnes 1993, Iversen 1993]. This model is used for the official allocation of acid rain budgets among the countries of Europe. EMEP models the dispersion and transformation of NO_x and SO_x for a grid system with a resolution of 150 km × 150 km. We have obtained the concentration data resulting from emission from each of five grid cells, chosen to be representative of France. Uniform emission is assumed in a particular cell which has been chosen as source cell. The data are averages over 5 years.

An important question concerns the geographic range over which the analysis needs to be extended in order to capture most of the impacts. A look at the EMEP results for SO_2 and NO_2 shows that these pollutants are transported over hundreds, even thousands of km. This is illustrated in Fig.2 using the EMEP data for a source at Nantes, assuming uniform receptor density and a linear dose-response function. The range of the analysis must be extended to over one thousand km if one wants to capture 80 (for SO_2) to 90% (for NO_2) of the total impact due

to the primary pollutants. This order of magnitude for the geographic range is typical of other pollutants with comparable removal rates, for instance fine particulates.

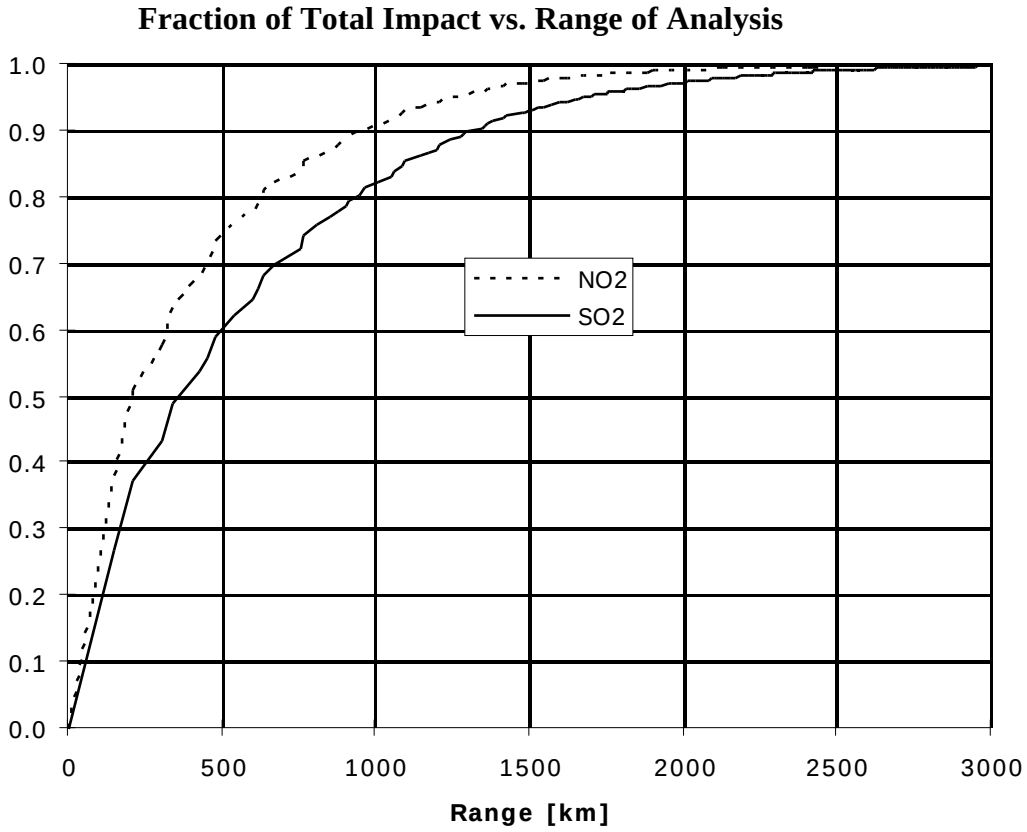


Fig.2. Fraction of total impact versus range of analysis, for uniform receptor density and linear dose-response function, based on EMEP data. Wiggles are due to discretization.

If the wind always had the same speed v, blowing in the same direction long enough that any air parcel follows a straight trajectory until the pollutant concentration becomes negligible, with equal probability for all directions, and if one assumes that the atmosphere is vertically mixed in a layer of height H, then it is easy to show from conservation of matter that the concentration at a distance r from the source is given by

$$c(r) = \frac{Q}{2\,\pi\, r\, v\, H}\ \exp\left(-\frac{k\, r}{v\, H}\right)\ , \qquad (25)$$

where k is the decay constant defined by Eq.8 and assumed site independent. This model also holds if the wind rose is not azimuthally symmetric, provided there is only one value of the wind speed in each direction. Note that Eq.25 is the $r = x \rightarrow \infty$ limit of the gaussian plume, according to Eq.22c, together with the decay factor Eq.23.

It is interesting to see how the EMEP data compare with this simple model. Performing a linear regression of data for the concentration c(r) in the form

$$\ln(r\, c(r)) = \alpha - \beta\, r \qquad , \qquad (26)$$

we have determined coefficients α and β to obtain a model according to Eq.25. The results are shown in Fig.3 for a fit along one particular direction (we do not fit the r = 0 point because for a point source there is a singularity, whereas the EMEP data assume uniform emission over an entire grid cell). The fit is remarkably good, considering the crude assumptions. The deviation of the data from the fit can be characterized as geometric standard deviation σ_G, defined in terms of the standard deviation of the logarithm of the concentration

$$\ln(\sigma_G) = \sqrt{\frac{1}{N}\sum_{n=1}^{N}[\ln(c_{data,n}) - \ln(c_{fit,n})]^2} \quad . \tag{27}$$

For a lognormal distribution of errors (usually a good assumption for such data) σ_G has a simple interpretation: there is a 67% probability of being within $1/\sigma_G$ and $1\sigma_G$ of the correct value, and a 95% probability of being within $1/\sigma_G^2$ and $1\sigma_G^2$ of the correct value. For a fit along any one direction we find geometric standard deviations around 1.2, although the correlation of the coefficients α and β with the wind rose of the emission site is relatively poor. If one tries to use Eq.25 with a single value of k/H in all directions and the average wind speed in each direction based on the wind rose, the fit becomes much worse, with σ_G around 2. However, for the purpose of the present paper it is sufficient to know the functional form along a radial direction; the numerical values of the coefficients do not matter.

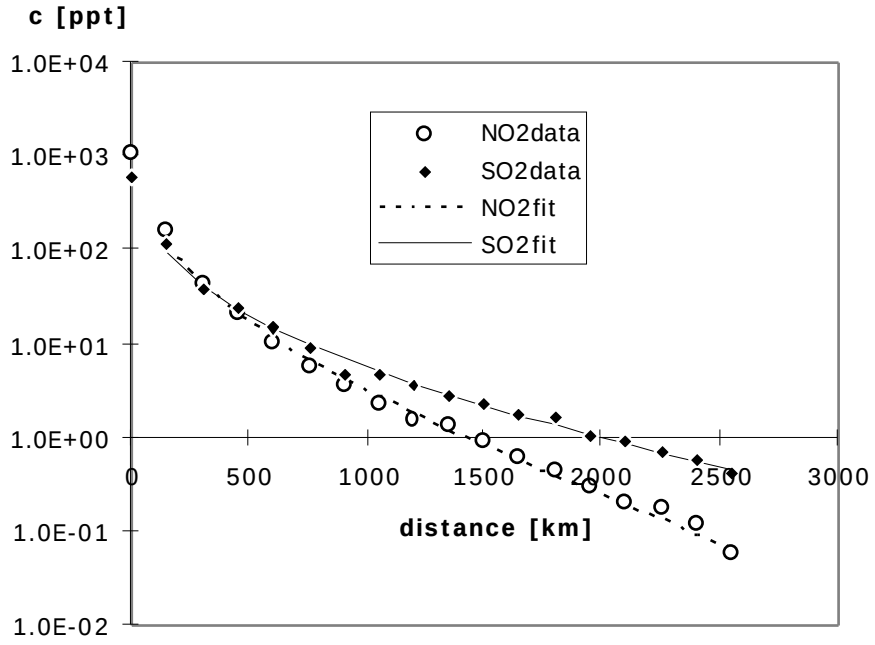


Fig.3. EMEP data for dispersion from a source at Nantes towards the south east: concentration in ppt for an emission rate of 100 000 t yr^{-1}, versus distance from source in km, fit (Eqs.25 and 26) and data.

7. Deviations from Uniform World

7.1. A Formula for the Damage

We would like to derive general conclusions about the validity of the uniform world model and about the sensitivity of damages to the distribution of receptors relative to the emission source. For this purpose we insert the atmospheric dispersion models of Eqs.19 to 23 (gaussian plume) and Eq.25 (fit to EMEP results) into Eq.5 for the damage. Thus we obtain an explicit model whose functional behavior can be explored.

We proceed in several steps to replace Eq.5, which is exact, by an approximation which is easy to evaluate, and by a number of numerical tests we demonstrate that this approximation is indeed acceptable. In particular we let $\rho(\mathbf{x})$ vary with $\mathbf{x}$ while keeping the removal rate $k(\mathbf{x})$ constant at $k(\mathbf{x}) = k_{uni}$. The symmetry between $\rho(\mathbf{x})$ and $1/k(\mathbf{x})$ in Eq.9 for the damage then implies that the approximation $k(\mathbf{x}) = k_{uni}$ is also justified.

First we show that one can evaluate Eq.5, without loss of generality, in an equivalent world with azimuthal symmetry about the source. We can consider Eq.5 as a sum over iso-contours of the concentration $c(\mathbf{x})$ in the limit where the spacing between contours becomes infinitesimal. The damage changes only if a receptor is moved across an iso-contour. It does not change if we move all receptors within an iso-contour, for example to the point corresponding to zero azimuth φ when we express $\mathbf{x}$ in (r, φ) coordinates. Therefore we can assume, for the purpose of the present paper, azimuthal symmetry for the receptor density by writing $\rho(\mathbf{x}) = \rho(r)$. By the same token the damage remains unchanged if we replace the concentration $c(\mathbf{x})$ by a new field which has the same value as the old one at $\varphi = 0$ but is azimuthally symmetric; thus we write $c(\mathbf{x}) = c(r)$. Since we are still assuming $k(\mathbf{x}) = k_{uni}$, we can restrict the gaussian plume of Eq.19 to a concentration field with azimuthal symmetry. This implies that we can integrate Eq.19 (evaluated at ground level z = 0) over y to work with the concentration c(r) as a function of the distance r from the source

$$c(r) = \frac{Q}{(2\pi)^{3/2}\, r\, \sigma\, v}\quad S(z=0)\, \exp\left(-\frac{k\, r}{v\, H}\right) \qquad , \tag{28}$$

where S(z) of Eq.21 describes the vertical concentration distribution and where

$$\sigma = \sigma_z = a\, r^b \tag{29}$$

is the vertical plume width, with the coefficients of Table 1, and we have included an exponential decay term according to Eq.25. As shown above, this form of the gaussian plume (in particular with Eq.22c) matches the EMEP data and can therefore be used everywhere.

In the following we will want to calculate the damage D_R in a region R with contours that are bounded by radial lines and concentric circles relative to the source. We insert the concentration c(r) of Eq.28 into Eq.5, with $k(\mathbf{x}) = k_{uni}$ and $d(\mathbf{x}) = d_{uni}$, and with receptor density ρ_R, assumed uniform within R. Integrating according to the specified boundaries we find

$$D_R = \frac{Q\, d_{uni}\, \rho_R\, \varnothing}{v\, (2\pi)^{3/2}} \int_{r_1}^{r_2} \frac{dr}{\sigma}\quad S(z=0)\ \exp\left(-\frac{k\, r}{v\, H}\right) \qquad , \tag{30}$$

where ø = angle between the radial lines and r_1 and r_2 = radii of the circular arcs. The total damage corresponds to the case where ø = 2π, and the integration extends from $r_1 = 0$ to $r_2 = \infty$; thus the total damage for a receptor density ρ_{uni} is

$$D_{tot} = \frac{Q\, d_{uni}\, \rho_{uni}}{v\,(2\pi)^{1/2}} \int_0^{\infty} \frac{dr}{\sigma}\; S(z=0)\; \exp\left(-\frac{k\,r}{v\,H}\right) \qquad . \tag{31}$$

Numerically D_{tot} is slightly different from D_{uni} of Eq.10 because near the source the gaussian plume is not well mixed in the vertical direction.

Eqs.30 and 31 are the key tools for our numerical work. They can be evaluated for annual averages or for specific meteorological conditions. Since we are looking for general relationships, we choose to evaluate these equations separately for each of the four atmospheric stability conditions in Table 1; annual averages can then be obtained by weighting these results according to the frequency of occurrence of the stability conditions. We have also evaluated the results for a fifth, synthetic, stability condition corresponding to the transition stable → neutral after 12 hr; this appears more realistic than the persistence of maximal stability day after night after day. However, we find that the numbers are within a few percent of those for stability condition D, and so we do not show them.

7.2. Variations in Receptor Density

To evaluate the sensitivity to variations in receptor density it is convenient to keep the density ρ(r) as an arbitrary function inside the integral of Eq.31, while normalizing D_{tot} by D_{uni} and defining a function f_{tot}

$$f_{tot} = \frac{D_{tot}}{D_{uni}} = \frac{1}{\sqrt{2\pi}}\,\frac{k}{v} \int_0^{\infty} \frac{dr}{\sigma}\; S(z=0)\; \exp\left(-\frac{k\,r}{v\,H}\right)\; \frac{\rho(r)}{\rho_{uni}} \qquad . \tag{32}$$

As a simple yet rather extreme kind of density variation we consider a concentric pattern of rings with ρ(r) alternating between two values:

$\rho(r) = 0$ from r = Δr to 2Δr, 3Δr to 4Δr, 5Δr to 6Δr, ...

and (33)

$\rho(r) = 2\,\rho_{uni}$ from r = 0 to Δr, 2Δr to 3Δr, 4Δr to 5Δr, ...

or vice versa. The result, shown in Table 2, indicates that this kind of density variation introduces errors on the order of ten percent at most. Within the policy orientation of the present paper this is entirely acceptable.

As pointed out above, the symmetry $\rho(\mathbf{x}) \leftrightarrow 1/k(\mathbf{x})$ implies the same conclusion for the validity of the constant k assumption. Table 2 is also a comforting about the risk of discretization errors:

when Eq.5 for the damage is evaluated numerically with real receptor data, the effect even of sizable errors in the receptor contours is negligible.

Table 2. Sensitivity to variations in receptor density: f_{tot} of Eq.32 with density of Eq.33, for several values of Δr = difference in radius between concentric rings, with k = 0.01 m s^{-1}, v = 7.5 m s^{-1}, H = 800 m, h_e = 100 m.

Δr / Gustiness category	25 km	50 km	100 km
B2 (very unstable)	1.026	1.046	1.088
B1 (unstable)	1.031	1.051	1.092
C (neutral)	1.057	1.079	1.12
D (stable)	0.991	1.049	1.17

7.3. Site Dependence

Now we have a simple and convenient tool for evaluating the effect of variations in receptor density. We can, for example, consider a city, on top of a background with uniform population density ρ_{uni}, and move the city relative to the source. France is a good model for this, being highly centralized, with about twenty percent of the total population living in the metropolitan area of Paris, as shown by the data in Table 3. To simplify the integration we take the city to be a region R with contours that are bounded by radial lines and concentric circles relative to the source. Thus the area of R is

$$A = \frac{ø}{2} (r_2^2 - r_1^2) \tag{34}$$

where ø = angle between the straight boundary lines of R.

Table 3. Area and population of France and of Paris.

	Area, in km^2	Radius of equivalent circle, in km	Population, in 1000	Density, per km^2
Paris, city	105	5.8	2153	20500
Paris, metropolitan area	12008	61.8	10830	902
France	551000	419	58000	105
France without met. Paris	539000	414	47170	88

To keep the boundary of the region R as "round" as possible as we move it relative to the source, we demand that the sum of the straight boundary segments be equal to the sum of the curved segments, thus changing the shape as we move it, by setting

$$ø = 2 \frac{(r_2 - r_1)}{(r_2 + r_1)} \quad . \tag{35}$$

Let r_0 = radius of a circle with the same area as R. To preserve equal areas as R is moved, we therefore take

$$r_2 = r_1 + r_0 \sqrt{\pi} \quad . \tag{36}$$

To obtain a dimensionless indicator for site dependence, it is convenient to define a function

$$f_R = \frac{D_R}{D_{tot}} = \frac{ø}{2\pi} \frac{\int_{r_1}^{r_2} \oint \frac{dr}{\sigma} S(z=0) \exp(-\frac{k\,r}{v\,H})}{\int_{0}^{\infty} \oint \frac{dr}{\sigma} S(z=0) \exp(-\frac{k\,r}{v\,H})} \quad . \tag{37}$$

In addition to ø, r_1 and r_2 it depends on k/v, H, h_e and stability condition (gustiness category).

Now let us evaluate the total damage $D_{back} + D_R$ as R is moved away from the source, D_{back} being the damage for a uniform background with receptor density ρ_{back} and D_R the damage in R evaluated with density ρ_R. D_{back} is, of course, the damage when R is infinitely far away. Thus the ratio

$$\frac{D_{back} + D_R}{D_{back}} = 1 + \frac{\rho_R}{\rho_{tot}} \quad f_R \tag{38}$$

indicates in dimensionless form how much the damage increases as R is moved closer to the source, i.e. as r_1 is varied.

The function f_R is plotted in Fig.4, for r_0 = 5 km in part a) and for r_0 = 50 km in part b). For example of r_0 = 5 km, the highest value of D_R/D_{uni} is about 0.012 for the least stable atmosphere. Taking a ratio of receptor densities between the city of Paris and the rest of France according to Table 2, we find that the damage in R can be as large as 0.012 × 20500/88 = 2.8 times the damage for the uniform background. Thus the total damage for a source adjacent to R is 3.8 times the damage of a source far from R (in a country with uniform receptor density). This corresponds the least stable atmosphere and is an upper bound. For average conditions Fig.4a suggests a ratio of D_R/D_{uni} in the vicinity of 0.006, implying that the total damage for a source adjacent to R is 2.4 times the damage of a source far from R.

For a second example let us take Fig.4b with r_0 = 50 km and density for the metropolitan area of Paris. Here the largest ratio of D_R/D_{uni} is around 0.06 for average atmospheric conditions, and with a receptor density ratio of 10 according to Table 3 we now find that the total damage for a source adjacent to R is 1 + 10 × 0.06 = 1.6 times the damage of a source far from R.

It is interesting to compare these findings with real site-dependent results, calculated with the PATHWAYS software package [Curtiss and Rabl 1995] which is based on Eq.5 and carries out

an accurate numerical integration of atmospheric dispersion results over geographic data for population and other receptors. To add substance to the results, we consider a specific impact: the increase in mortality due to particulate matter emitted by coal fired power plants. The dose-response function [based on Schwartz 1993, as cited in EC 1995] is linear and can be written in the form

$$\Delta\text{deaths yr}^{-1} = 10.4 \times \Delta\,\text{PM}_{10}\text{ concentration [in g m}^{-3}] \quad . \qquad (39)$$

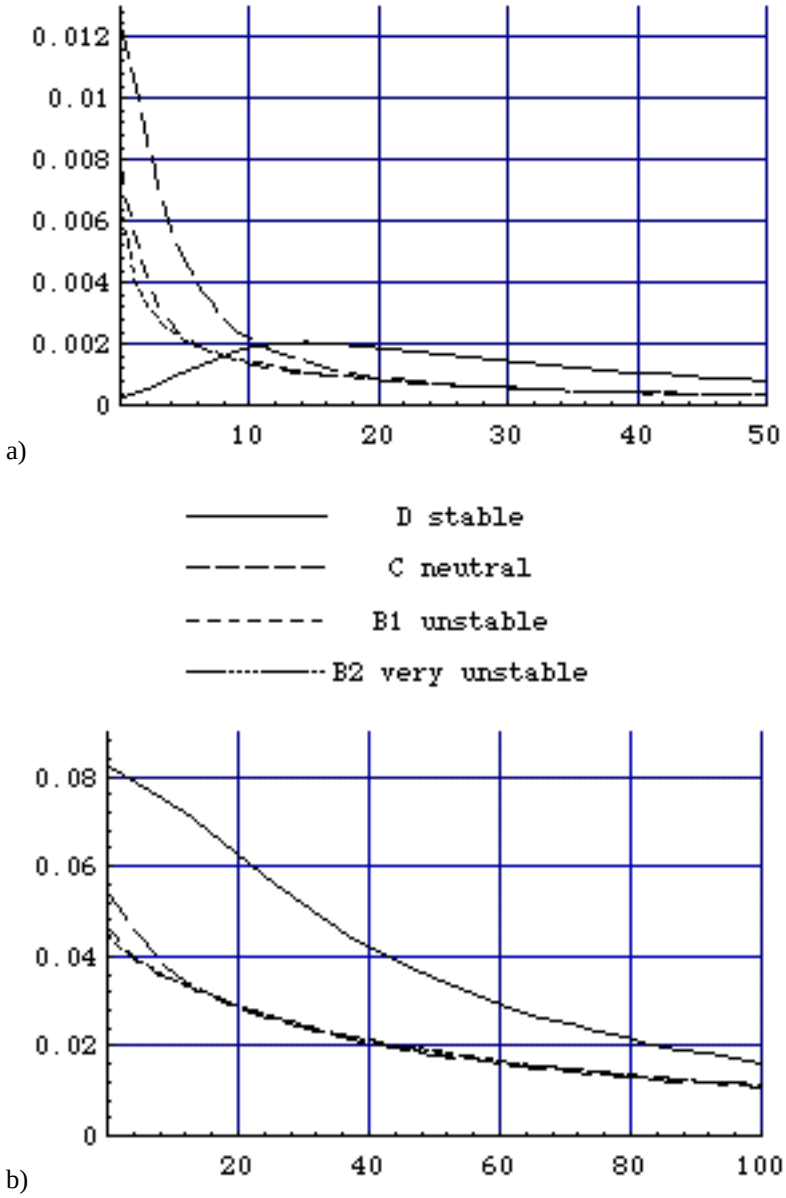


Fig.4. D_R/D_{tot} of Eq.37 versus r_1 [in km], for k = 0.01 m s^{-1}, wind = 7.5 m s^{-1}, H = 800 m, h_e = 100 m.
a) r_0 = 5 km, b) r_0 = 50 km.

We consider a coal fired power plant with an annual electricity production of 2.1×10^9 kWh yr^{-1} and a particulate emission rate of 0.17 g kWh^{-1}, hence an annual particulate output of Q = 357 ton yr^{-1} = 11.32 g s^{-1} [Rabl et al. 1996]. For the atmospheric dispersion we take the EMEP SO_2 data for a grid cell in the center of France, for which a curve fit yields a range $1/\beta$ = 540 km and a removal velocity k = v H β = 0.014 m s^{-1} (EMEP does not calculate

particulate dispersion, but the removal velocities are comparable, within the approximation of a single calculation for the entire spectrum of particle sizes). Inserting $Q = 11.32$ g s^{-1}, $k_{uni} = 0.0062$ m s^{-1}, $d_{uni} = 10.4$ (deaths yr^{-1})/(g m^{-3}) and $\rho_{uni} = 1.05 \times 10^{-4}$ m^{-2} (for France from Table 3) into Eq.10 we obtain

$$D_{uni} = \frac{10.4 \text{ (deaths yr}^{-1})/(\text{g m}^{-3}) \times 1.05 \times 10^{-4} \text{ m}^{-2} \times 11.32 \text{ g s}^{-1}}{0.014 \text{ m s}^{-1}} = 0.83 \text{ deaths yr}^{-1}.$$

This number is shown as horizontal solid line in Fig.5 where the number of deaths per year is plotted versus emission height. The points, connected by lines, show the impacts for five specific sites. We have chosen these sites because there are in fact fossil fuel power plants at these sites (the nearest big city, 25 to 50 km away, is indicated in parentheses). Although the real emissions at the different sites are different, here we have assumed the emissions of the Cordemais plant at all sites to bring out the point of the comparison. The impact is about three times larger than D_{uni} for the site near Paris and about three times smaller than D_{uni} for Cordemais, a rural site on the Atlantic Ocean. This confirms our generic results above: Paris is a metropolis containing about a fifth of the population of France, while for Cordemais the effective receptor density is much lower because it is a rural site with the Atlantic ocean on one side.

How representative are these results? Emission height dependence and deviations from uniformity are most pronounced when the receptors are concentrated near the source. A source near Paris seems like a fairly extreme example because France is highly centralized, with 19% of its population in Greater Paris. However, there are more extreme cases. In terms of geographic extent and population, France (58 million people, density 105 per km^2) and the North East census region (50 million people, density 118 per km^2) of the USA are more or less comparable. The New York metropolitan area comprises 36% of the population of the North East census region. Furthermore, the ratio local/regional impact is enhanced for a source in New York, close to the ocean (i.e., reduced impact outside local area), compared to one in Paris which is inland. A recent study by Rowe et al [1995] found variations with emission height and emission site that are consistent with Fig.5.

Furthermore, all our results have been derived in the flat terrain approximation. Not taken into account is the canyon effect: the increase in local concentration from sources in streets or valleys. That can be especially significant for the impacts of air pollution from cars. As an indication of the latter but without canyon effect, we have included in Fig.5 the impact from a source in Paris at ground level with zero vertical exhaust velocity.

Note that in Fig.5 the emission height is the real height of the stack itself because the local impact has been modeled with the ISC gaussian plume [Wackter and Foster 1987] which accounts for plume rise due to temperature and velocity of the exhaust; by contrast the other figures in this Section are based on Eqs.30 and 31 with h_e = effective emission height (including plume rise).

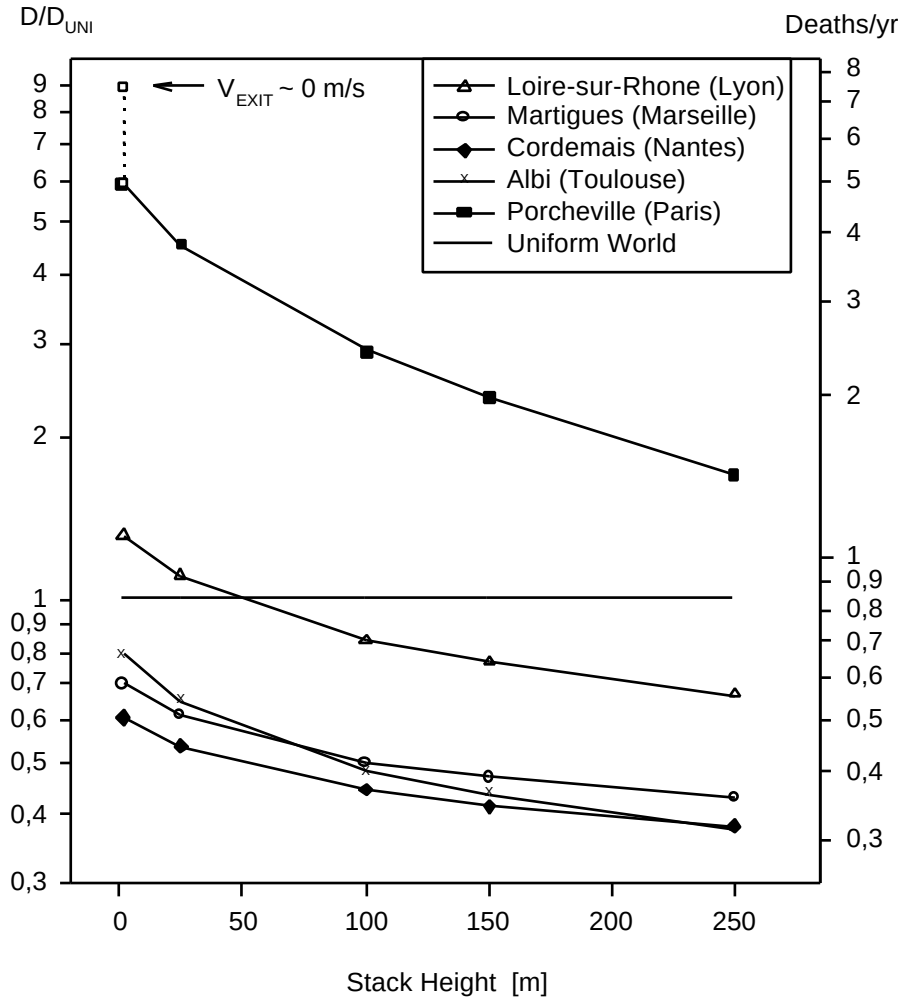


Fig.5. Example of dependence of damage D on site and on stack height,
for linear dose-response function: mortality due to particulate matter PM_{10} from a coal fired power plant,
for five sites in France and for uniform world model (D_{uni} of Eq.10).
Emission 357 ton yr^{-1} (for 2.1×10^9 kWh_e yr^{-1}). *

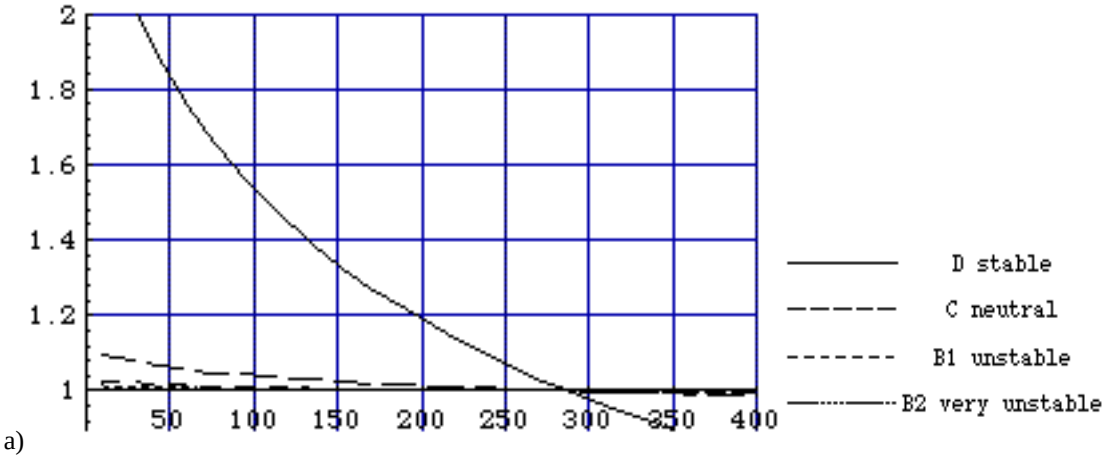
We have not yet performed an analogous test of the uniform world model for secondary pollutants, Eq.18, but it is plausible that site dependence is less pronounced for secondary pollutants than for primary pollutants. Site dependence arises from correlated variations of the factors in the integral of Eq.5, in particular receptor density and concentration. Secondary

* In the original version of this paper we had adapted the CALCONC gaussian plume model (developed by the Centre d'Etudes sur l'Evaluation de la Protection dans le Domaine Nucléaire for the ExternE program of the EC [1995]) that had been designed for sites without solar data and determined the stability classes from wind speed only. In the meantime we have obtained the ISC model [Wackter and Foster 1987] as well as weather data with hourly solar radiation. The emission height dependence is much stronger than in the old Fig.5. The reason is that ISC is dominated by neutral and stable conditions, which means that the plume travels much further before being vertically mixed than for CALCONC which is dominated by unstable conditions. We are very grateful to J. Spadaro for the ISC calculations.

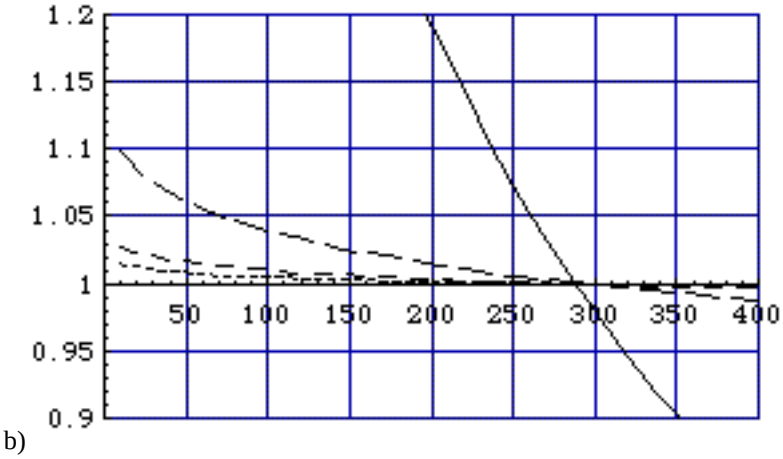
pollutants are created far from the source and in diffuse manner; thus the variation of their concentration is much more gradual than the variation of a primary pollutant near its source. Eq.18 should hold even for ozone whose nonlinear creation mechanism implies that the rate $k_{1\text{-}2}$ can have different signs at different places; such sign change does not invalidate the derivation of Eq.18. More problematic is the fact that ozone formation depends on local concentrations of volatile organics and the NO/NO_2 ratio, which in turn may be correlated with population density. One needs a test with real data.

7.4. Dependence on Emission Height

Next we evaluate how the total damage varies with emission height. For this purpose it is convenient to normalize D_{tot} of Eq.31 by D_{uni} of Eq.10, with $\rho(r) = \rho_{uni}$. The ratio $f_{tot} = D_{tot}/D_{uni}$ is plotted as function of emission height h_e in Fig.6, for each of the four stability conditions. The analogous variation for real sites and average meteorological conditions has been shown already in Fig.5.



a)



b)

Fig.6. Total damage D_{tot} as function of emission height h_e, in units of D_{uni}, for k = 0.01 m s^{-1}, wind = 7.5 m s^{-1}, and H = 800 m.
a) Complete plot, b) close-up.

From Figs.5 and 6 we see that the saying "the solution to pollution is dilution" is misleading. There is, of course, a very significant damage reduction for receptors near the source. But for pollutants with linear dose-response function, in particular the important case of fine particulates, the total damage is not reduced very much. The variation of damage with h_e is enhanced if the

source is near a large population center, but even for the extreme case of a source in Paris Fig.5 shows the damage to decrease only by about three if the stack height is increased from 0 to 200 m.

7.5. Dependence on Atmospheric Parameters

Finally we consider the variation of the ratio D_{tot}/D_{uni} with atmospheric parameters. In Fig.7 we plot this ratio as function of the planetary boundary layer thickness H, and in Fig.8 as function of removal velocity k. In either case the variation is quite small, with the exception of most stable condition, gustiness category D.

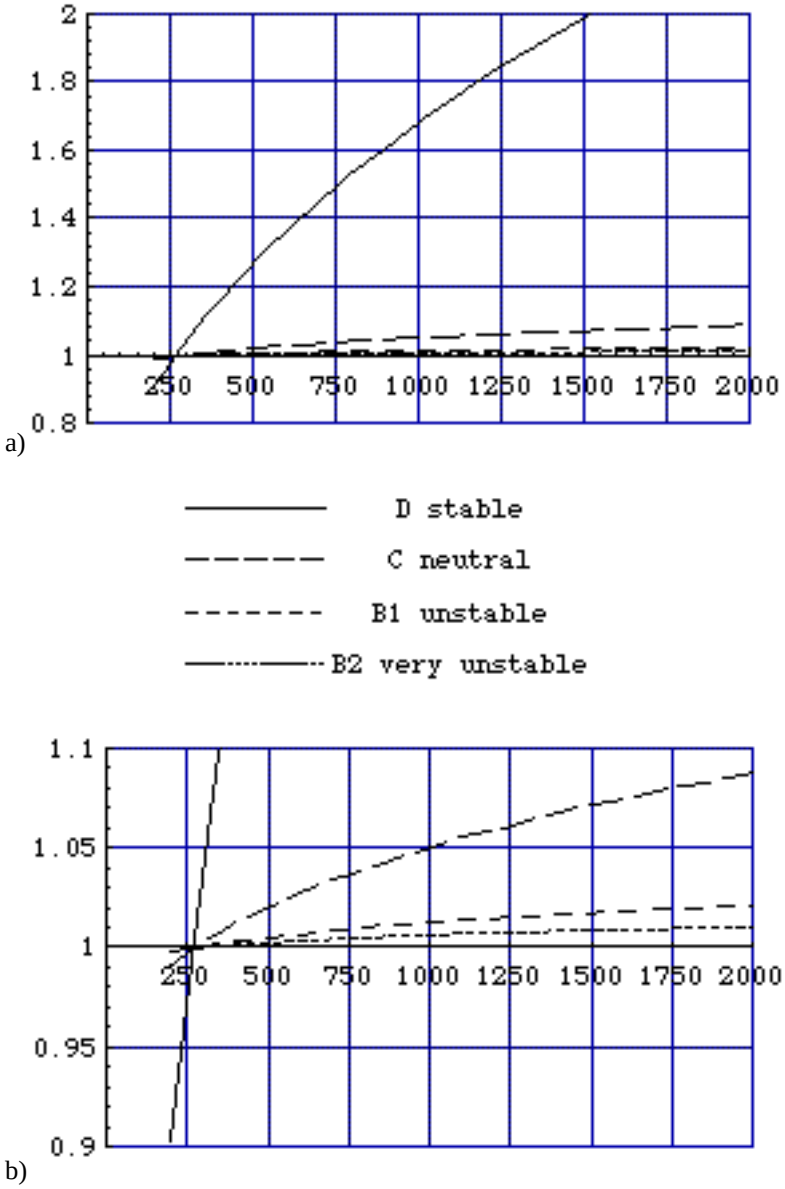


Fig.7. Total impact D_{tot} as function of height H of mixed boundary layer [in m], in units of D_{uni}, for k = 0.01 m s^{-1}, wind = 7.5 m s^{-1} and h_e = 100 m.

a) Complete plot, b) close-up.

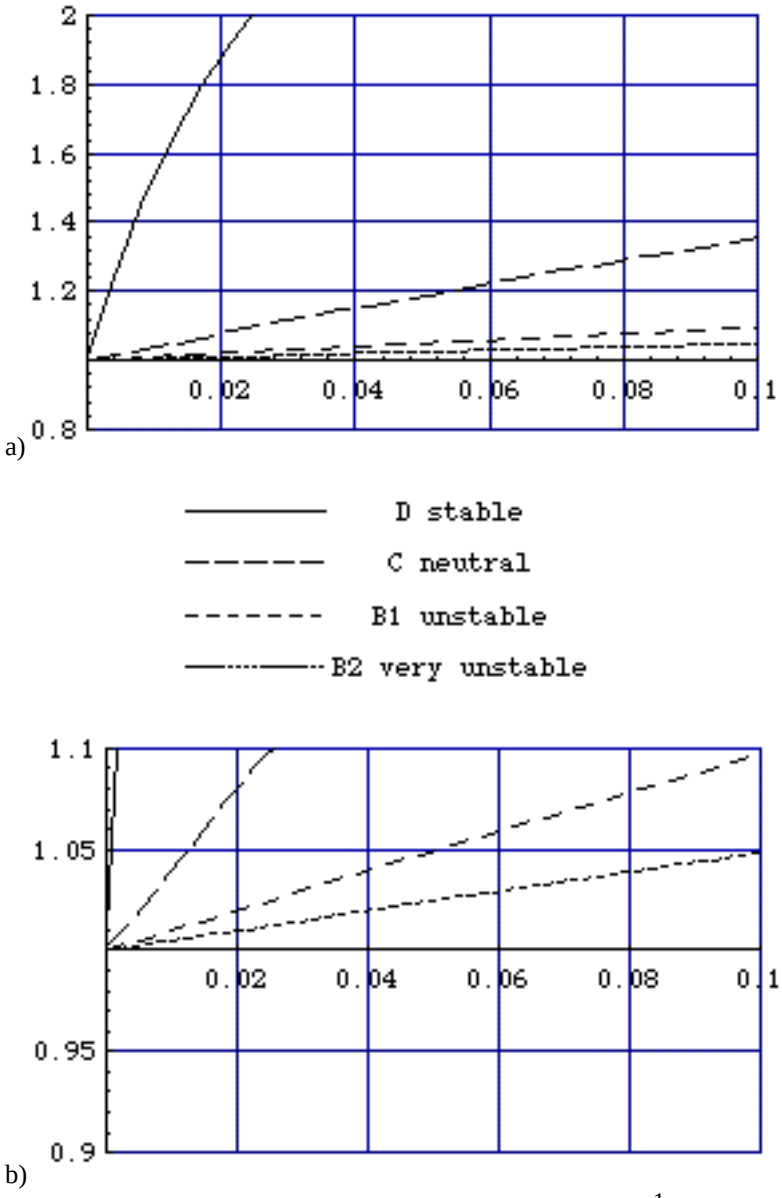


Fig.8. Total impact D_{tot} as function of removal velocity k [in m s^{-1}], in units of D_{uni}, for H = 800 m, wind = 7.5 m s^{-1} and h_e = 100 m.

a) Complete plot, b) close-up.

8. Conclusions

By combining atmospheric dispersion and dose-response functions we have developed a framework for analyzing general features of air pollution damage. We have provided generic relationships for site dependence and compared them with results calculated for specific sites, using a detailed computer program. We have shown that the site dependence is remarkably small. The simple formula for the total damage for linear dose-response functions,

$$D = D_{uni} = d_{uni}\, \rho_{uni}\, Q/k_{uni} \qquad (10)$$

derived for a uniform world, yields damage estimates that are correct within an order of magnitude. The generalization to secondary pollutants is straightforward (although we have not

yet carried out explicit tests with data). A related finding is that the tolerance to errors in receptor distribution is high.

We have also examined the total damage as a function of emission height and have found that the saying "the solution to pollution is dilution" is misleading. There is, of course, a very significant damage reduction for receptors near the source (and total avoidance of damage if the concentration can thus be kept below the threshold of the dose-response function). But for pollutants with linear dose-response function the total damage does not decrease very much with stack height. Emission height dependence and deviations from the uniform world model are most pronounced when the receptors are concentrated near the source. But note that all our results have been derived in the flat terrain approximation. Not taken into account is the canyon effect (increase in local concentration from sources in streets or valleys).

These results are directly relevant for the field of life cycle analysis (LCA), where a variety of indices have been proposed to estimate environmental impacts [SETAC 1992, Heijungs et al. 1992]. None of those indices have realistically accounted for the processes of atmospheric dispersion and removal, and even the proponents of these indices advise that they should only be considered as "potential impacts". By contrast, Eq.10 quantifies the real impact. It is exact for a uniform world, in the sense of Section 5, if dose-response function d_{uni}, receptor density ρ_{uni}, emission rate Q and removal rate k_{uni} are known.

Acknowledgment

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Nomenclature

A = area of region R

a = plume width parameter in equation $\sigma = a\, x^b$

b = plume width parameter in equation $\sigma = a\, x^b$

$c(\mathbf{x})$ = concentration at $\mathbf{x}$, with subscripts 1 and 2 for primary and secondary pollutant

D = damage

$d(\mathbf{x}) = \frac{df_{dr}(\mathbf{x},c(\mathbf{x}))}{dc}$ = slope of dose-response function

$f_{dr}(\mathbf{x},c(\mathbf{x}))$ = dose-response function

$f_R = \frac{D_R}{D_{tot}}$ = fraction of total damage which occurs in region R, according to Eq.37

$f_{tot} = \frac{D_{tot}}{D_{uni}}$ = total damage, normalized by damage in uniform world

$F_{dry}(\mathbf{x})$ = dry deposition flux, $[g/m^2 \cdot s]$

$F_{wet}(\mathbf{x})$ = wet deposition flux, [g/m^2·s]
$F_{trans}(\mathbf{x})$ = transformation or decay flux, per s and per m^2 of horizontal surface, [g/m^2·s]
$F(\mathbf{x}) = F_{dry}(\mathbf{x}) + F_{wet}(\mathbf{x}) + F_{trans}(\mathbf{x})$,[g/m^2·s]
$F_{1\text{-}2}(x)$ = mass of secondary pollutant created per s and per m^2 of horizontal surface
H = height of planetary boundary layer
h_e = effective emission height (including plume rise) of pollution source above ground
$k(\mathbf{x})$ = deposition or decay/transformation velocity, defined by $k(\mathbf{x}) = F(\mathbf{x})/c(\mathbf{x})$ and referred to in this paper as "removal velocity"
$k_{1\text{-}2}(\mathbf{x}) = F_{1\text{-}2}(\mathbf{x})/c_1(\mathbf{x})$
Q = emission [kg s^{-1}]
r = radial coordinate
S(z) = factor describing the concentration dependence in vertical direction
v = wind speed
v_{dry} = dry deposition velocity [m s^{-1}].
x = x coordinate
y = y coordinate

$\beta = \frac{k}{v\,H}$ = decay constant
$\rho(\mathbf{x})$ = receptor density
σ = plume width, with subscript y for horizontal and z for vertical direction
σ_G = geometric standard deviation
ø = polar coordinate, angle between the straight boundary lines of R.

EC = European Commission
EMEP = (European Monitoring and Evaluation Programme) acid rain modeling program of Norwegian Meteorological Institute
NO_X = unspecified mixture of nitrogen oxides
PM = particulate matter (with subscript to indicate upper limit of diameter in μm)
ppt = parts per trillion
SO_X = unspecified mixture of sulfur oxides
TSP = total suspended particles
VOC = volatile organic compounds.

References

Barrett , K. 1992. "Dispersion of nitrogen and sulphur across Europe from individual grid elements: marine and terrestrial deposition". EMEP/MSC-W Note 3/92. August 1992. Norwegian Meteorological Institute, P.O.Box 43, Blindern, N-0313 Oslo 3.

Cohrssen, J. J. and V. T. Covello. 1989. *Risk Analysis: a Guide to Principles and Methods for Analyzing Health and Environmental Risks.* United States Environmental Protection Agency. Report EPA PB89-137772. Washington, DC 20460.

Curtiss, P. S. and A. Rabl. 1995. *PATHWAYS : a software package for calculating impacts and costs of environmental burdens due to electricity production by nuclear or fossil fuels. Program Manual.* Ecole des Mines de Paris.

Dockery, D. W. and C. A. Pope III. 1994. "Acute respiratory effects of particulate air pollution". *Ann. Rev. Public Health*. vol.15, p.107-132.

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

EC 1995a. *ExternE: Externalities of Energy.* Vol.1 Summary. European Commission, Directorate-General XII, Science Research and Development. Office for Official Publications of the EC, 2 rue Mercier, L-2985 Luxembourg.

EC 1995b. *ExternE: Externalities of Energy.* Vol.2 Methodology. European Commission, Directorate-General XII, Science Research and Development. Office for Official Publications of the EC, 2 rue Mercier, L-2985 Luxembourg.

EPA. 1983. *Guidelines for Performing Regulatory Impact Analysis.* US Environmental Protection Agency, Office of Policy Analysis. Report EPA-230-01-84-003, reprinted March 1991. Washington, DC 20460.

Frith, C. H., N. A. Littlefield and R. Umholtz. 1981. "Incidence of pulmonary metastases for various neoplasms in BALB/cStCrlfC3H/Nctr female mice fed N-2-fluorenylacetamide". JNCI, vol.66, p.703-712.

Heijungs, R. et al. 1992. *Environmental Life Cycle Assessment of Products.* Part 1. Guide. Part 2. Backgrounds. Centre of Environmental Science, Garenmarkt 1, P.O.Box 9518, 2300 RA Leiden, Netherlands.

Iversen, T. 1993. "Modeled and measured transboundary acidifying pollution in Europe - verification and trends". *Atmospheric Environment*, 27A. 889-920.

Lipfert, F. W. 1994. *Air Pollution and Community Health: a Critical Review and Data Sourcebook.* Van Nostrand Reinhold. New York.

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.

Rabl, A., P. S. Curtiss, J. Spadaro, B. Hernandez, A. Pons, M. Dreicer, V. Tort, H. Margerie, G. Landrieu, B. Desaigues and D. Proult. 1995. "Environmental Impacts and Costs: the Nuclear and the Fossil Fuel Cycles". Version 2.1 June 1996. Coordinated by A. Rabl, Centre d'Energétique, Ecole des Mines, 60 boul. St.-Michel, 75272 Paris CEDEX 06.

Rowe, R.D., C.M. Lang, L.G. Chestnut, D. Latimer, D. Rae, S.M. Bernow, and D.White. 1995. *The New York Electricity Externality Study.* Oceana Publications, Dobbs Ferry, New York.

Rodricks, J. V. 1992. *Calculated Risks: the Toxicity and Human Health Risks of Chemicals in our Environment.* Cambridge University Press. Cambridge, UK.

Sandnes, H. 1993. "Calculated budgets for airborne acidifying components in Europe". EMEP/MSC-W Report 1/93. July 1993. Norwegian Meteorological Institute, P.O.Box 43, Blindern, N-0313 Oslo 3.

Schwartz J. 1993. "Air pollution and daily mortality in Birmingham, Alabama". *American Journal of Epidemiology*; 137: 1136-1147.

SETAC 1992. *Code Practice of Life Cycle Assessment.* SETAC (Society of Environmental Toxicology and Chemistry).

Seinfeld, J. H. 1986. *Atmospheric chemistry and physics of air pollution.* John Wiley and Sons.

Trukenmüller, A. and W. Krewitt. 1993. "*Some reflections on the problem of system boundaries for a regional-range analysis of human impacts.*" Discussion paper, 10.9.1993, IER, Universität Stuttgart.

Wackter, D. J. and J. A. Foster. 1987. *Industrial source complex (ISC) dispersion model user's guide.* 2nd edition. Vol.1, EPA 450/4-88-002a. US Environmental Protection Agnecy, Research Triangle Park, North Carolina.

Zannetti, P. 1990. *Air Pollution Modeling: Theories, computational methods and available software.* Van Nostrand Reinhold, NY.