

Uncertainty about components of PM₁₀ and their toxicities and its effect on estimation of health damage

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Presented at *AIR POLLUTION 99*, Palo Alto, 27 - 29 July 1999.
Proceedings: *Adv. Air Pollut.*, 7, 467-474 (1999)

Abstract

To guide public policy on regulation of emissions, it is desirable to know how much damage is caused by each source of pollution and by each pollutant. Concentration-response (C-R) functions, established by epidemiological studies, play a crucial role in the estimation of the damage per kg of emitted pollutant. Of particular importance are those based on particulate matter (PM_{2.5} and PM₁₀). Unfortunately there is considerable uncertainty in the application of the C-R functions for particulate matter, due to a lack of detailed information about the relative toxicity of the different components of PM in ambient air. The purpose of the present paper is to examine the effect of this uncertainty on the estimation of health damage. As starting point we assume linearity of incremental health impact with incremental concentration, in view of the lack of epidemiological evidence for no-effect thresholds or significant deviations from linearity. We write the observed C-R function as a linear combination of (unknown) C-R functions for the main components of PM. We consider four components: primary combustion particles, nitrates, sulfates, and other (mostly of soil or marine origin). We make assumptions about plausible ranges for the slopes of the component C-R functions and about the composition of ambient air in the cities where the C-R functions were obtained. Varying the unknown parameters we evaluate how much the health impacts could change with different plausible assumptions. As an example we consider the damage cost per kWh for a modern coal fired power plant.

1. Introduction

For environmental policy it is desirable to know how much damage is caused by each source of pollution and by each pollutant. To evaluate the impact and damage cost of a pollutant, one needs to carry out an impact pathway analysis

which involves the calculation of the pollutant concentration increment in all affected regions due to an incremental emission (e.g. $\mu\text{g}/\text{m}^3$ of particles, using models of atmospheric dispersion and chemistry), followed by the calculation of physical impacts (e.g. number of cases of asthma due to these particles, using a concentration-response function). The numbers are summed over all receptors (population, crops, buildings, ...) of concern.

In recent years there has been much progress in the analysis of environmental damage costs, thanks to several major projects to evaluate the externalities of energy in the USA [ORNL/RFF 1, Rowe et al 2] and in Europe [ExternE 3, ExternE 4, Rabl and Spadaro 5]. A key finding of these studies is the predominant contribution of health costs to the total damage.

Among epidemiological studies the air pollutant most frequently implicated is particulate matter. Unfortunately this is not a very definite quantity. Most air quality monitoring stations have measured only the mass, typically as TSP (total suspended particles), PM_{10} and more recently $\text{PM}_{2.5}$, without any information on the composition of the particles. Some stations have measured black smoke - which is not very specific either. Among components of PM only sulfates and/or acidity have been measured by a few stations (note that even the category of sulfates is an ambiguous mixture). Of course, epidemiological studies are limited by the data that have been measured by monitoring stations. Nitrates, for example, have not been monitored until recently and so there are no epidemiological studies on the effects of nitrate aerosols.

This leads to considerable uncertainty about the damage caused by particulate emissions because one does not know which characteristics of particles are important: chemical composition, acidity, oxidizing potential, solubility, size, mass, number of particles, ... ? Since epidemiological studies are based on whatever mixture of primary and secondary particles happens to be in the air in the cities under consideration, it is not even clear to what extent the results can be transferred to other places. In view of this situation the best one can do is to evaluate and compare the consequences of different assumptions about causal relationships. In this paper we focus on the CR (concentration-response) functions for particulate matter.

2. The Composition of PM in Ambient Air

Detailed data on composition or size of ambient particles are only known from a few spot measurements. As main components one can consider:

- primary combustion particles (soot, fly ash, ...)
- sulfates (H_2SO_4 , $(\text{NH}_4)\text{HSO}_4$, $(\text{NH}_4)_2\text{SO}_4$, ...)
- nitrates (HNO_3 , NH_4NO_3 , ...)
- other (soil, road dust, ...).

Table 1 indicates some typical results that have been reported in the literature, as cited by Seinfeld and Pandis [6]. In urban areas carbon and sulfates are especially important.

Table 1. Typical results for composition of ambient particulate matter.

Region	$\mu\text{g}/\text{m}^3$	C(elem)	C(org)	NH_4^+	NO_3^-	SO_4^{--}	Other
Remote ^a	4.8	0.3%	11%	7%	3%	22%	57%
Nonurban continental ^a	15	5%	24%	11%	4%	37%	19%
Urban ^a	32	9%	31%	8%	6%	28%	18%
Rubidoux, CA ^b	87.4	3%	18%	6%	20%	6%	47%

^a Heintzenberg [7]

^b Solomon et al [8]

3. A Framework for Assessing the Uncertainties

Our key assumption is linearity of all CR functions for particulate matter in the air, at least for incremental impacts above current ambient concentrations (note that a threshold has no effect if it is below ambient concentrations, and to date there is no evidence for a no-effect threshold). Not only does linearity appear to be consistent with the data within the confidence intervals, but there is not sufficient evidence to prefer any other form [Dockery et al. 9, Dockery and Pope 10, Lipfert 11, Pope et al 12, Wilson and Spengler 13].

Let us consider four main components of particulate matter in the air:

- Primary combustion particles;
- Nitrates;
- Sulfates; and
- Other,

designated by the subscripts P, N, S and O, respectively. For each of these components we assume a linear CR function with slope $f_{\text{CR},i}$ where $i = \text{P, N, S}$ and O, the effects being independent of each other (i.e. without positive or negative interference). Then the slope of the observed CR function for PM_{10} is

$$f_{\text{CR},\text{PM}_{10}} = p_{\text{P}} f_{\text{CR},\text{P}} + p_{\text{S}} f_{\text{CR},\text{S}} + p_{\text{N}} f_{\text{CR},\text{N}} + p_{\text{O}} f_{\text{CR},\text{O}} \quad (1)$$

where p_i designates the percentage of the respective component in the ambient air at the sites where the epidemiological study was carried out. Of course the p_i must satisfy the constraint

$$p_{\text{P}} + p_{\text{S}} + p_{\text{N}} + p_{\text{O}} = 1 \quad (2)$$

It is convenient to rewrite eqn.1 as

$$p_{\text{P}} t_{\text{P}} + p_{\text{S}} t_{\text{S}} + p_{\text{N}} t_{\text{N}} + p_{\text{O}} t_{\text{O}} = 1 \quad (3)$$

by defining the relative toxicity t_i of component i relative to PM_{10} as

$$t_i = f_{\text{CR},i}/f_{\text{CR},\text{PM}_{10}} \quad (4)$$

Now we can evaluate alternative hypotheses by varying p_i and t_i , within reasonable limits subject to the constraints of Eqs.2 and 3. Here we take

$$p_P = 33\% \quad p_S = 33\% \quad p_N = 7\% \quad p_O = 27\% \quad (5)$$

for the compositions, in view of the data in Table 1.

4. Suggestive Parallels with Smoking

Before applying this framework, it is interesting to look at another source of primary combustion particles: tobacco smoke. Parallels and differences between exposure to tobacco smoke and air pollution are listed in Table 2. Many of the primary air pollutants are significant in both, although the proportions may be different. Probably the most important differences are nicotine on one side, and sulfates, nitrates and crustal particles on the other.

Table 2. Parallels and differences between exposure to tobacco smoke and air pollution.

Common in both	soot, tar, volatile organic compounds, CO, NO _x , fly ash
Only in tobacco smoke	nicotine
Only in ambient air	sulfates, nitrates, crustal particles

The parallels go even further for passive smoking because the concentrations are comparable. According to measurements by Dockery and Spengler [14] the presence of a smoker increases the concentration of PM_{2.5} in a home by about 20 $\mu\text{g}/\text{m}^3$, on average. Among the cities of the air pollution study of Pope et al [12], the difference between highest and lowest PM_{2.5} levels is 24.5 $\mu\text{g}/\text{m}^3$. Since direct effects of NO_x and CO (at low concentrations) appear to be less important than those of particles, it seems plausible that much of the damage from tobacco smoke can be attributed to primary combustion particles.

It is therefore instructive to compare, in Table 3, the relative risks between smoking and air pollution. Unfortunately the different studies do not all cover exactly the same end points. Nonetheless it seems that the risks for passive smoking are, at least very roughly, of the same order of magnitude as for air pollution, suggesting that exposure to low concentration of combustion particles can indeed have the sort of impacts found in epidemiological studies.

Table 3. Comparison of relative risks between smoking and air pollution.

Cause of death	Smoking, active	Smoking, passive	Sulfates (19.9 $\mu\text{g}/\text{m}^3$)	PM _{2.5} (24.5 $\mu\text{g}/\text{m}^3$)
All	2.1 ^a		1.15 ^a	1.17 ^a
Lung cancer	9.7 ^a	1.35 ^b	1.36 ^a	1.03 ^a

Cardiopulmonary	2.3 ^a		1.26 ^a	1.31 ^a
Ischemic heart disease	1.7 ^c	1.2 ^c		

^a Pope et al [12]

^b Académie Nationale de Médecine [15]

^c Wells [16].

5. An Example

As an example, let us consider the total damage per kWh for a coal fired power plant with emissions per kWh in Table 4. The table also shows the damage costs per kg of pollutant, as calculated by Rabl and Spadaro [5] for typical conditions in Europe, following the assumptions of ExternE [4].

Table 4. Total damage per kWh for a new coal fired power plant, for the damage cost per ton of pollutant as calculated by ExternE [4] for typical conditions in Europe [Rabl and Spadaro 5].

	Euro/kg	g/kWh	mEuro/kWh
Primary particles	13.53	0.2	2.7
NO₂	16.92	1.0	16.9
SO₂	11.87	2.0	23.7

ExternE calculates the concentrations of primary particles (i.e. emitted by the source of pollution), and of nitrates and of sulfates, resulting from the respective emissions of NO_x and of SO₂. Then the damages are calculated by making the following assumptions:

- the CR functions are linear;
 - the damage is proportional to the mass of particulate matter in the air, as measured by PM₁₀ or PM_{2.5};
- in addition, for a given end point (asthma attack, hospital admission, premature death, ...)
- the CR function for primary particles from power plants is equal to that for PM₁₀;
 - the CR function for primary particles from cars is equal to that for PM_{2.5};
 - the CR function for sulfates is 1.67 times that for PM₁₀;
 - the CR function for nitrates is equal to that for PM₁₀.

It is interesting to note that the ExternE assumptions imply that the health damage cost per kg of pollutant is not very different between NO₂, SO₂ and PM₁₀.

The assumptions of ExternE [4] correspond to

$$t_p = 1.0, t_s = 1.7, t_N = 1.0, \quad t_o = 0.2 \quad (6)$$

the latter being implied by eqn.3. For the corresponding damage cost per kg of pollutant we take the values calculated by the “uniform world model” of Curtiss and Rabl [17] because they have been found to represent typical conditions for power plants in Europe [Spadaro and Rabl 18]; this model has the simple form

$$D_i = P \frac{f_{CR,i} \rho Q_i}{k_i} = P t_i \frac{f_{CR,PM10} \rho Q_i}{k_i} \quad (7)$$

where

Q_i = emission [kg/s] of ith pollutant

k_i = depletion velocity [m/s] of ith pollutant
= receptor density

f_{CR} = slope of CR function

P = unit cost of end point of CR function [e.g. Euro/asthma attack].

The generalization to secondary pollutants is straightforward.

The total damage per kWh is

$$D_{tot} = \sum_i D_i \quad (8)$$

where D_i is the damage per kg for each of the three pollutants. The result is shown in Fig.1 for a wide range of possible hypotheses about the relative toxicities. For the alternatives shown in this figure, the choice of ExternE is an upper bound. If nitrate and sulfate aerosols are less toxic than assumed by ExternE, the damage per kWh could be a factor of two to three smaller. But note that this variation of total cost depends very much on individual emissions. Of course, Fig.1 is indicative only for pollution sources with emissions ratios that are not very different from the ones in Table 4.

6. Conclusion

Due to the differences between the PM data of the epidemiological studies and the particle concentrations resulting from specific pollution sources, there are major uncertainties in damage estimate, unavoidable at the present state of knowledge. The best one can do is a sensitivity study to evaluate how the results can change with different assumptions that can plausibly be made about the toxicity of the components of particles in ambient air. We have illustrated this with the example of the damage cost per kWh for a coal fired power plant. The resulting uncertainty is large. It comes on top of the already large uncertainties estimated by Rabl and Spadaro [19].

Unfortunately there is no good answer to an important question of environmental policy: what is the benefit of reducing the emissions of each pollutant separately? That question is particularly troubling in situations where one has to make tradeoffs between reductions of different pollutants. For instance, certain control technologies for diesel engines reduce the emissions of particles while increasing those of NO_x .

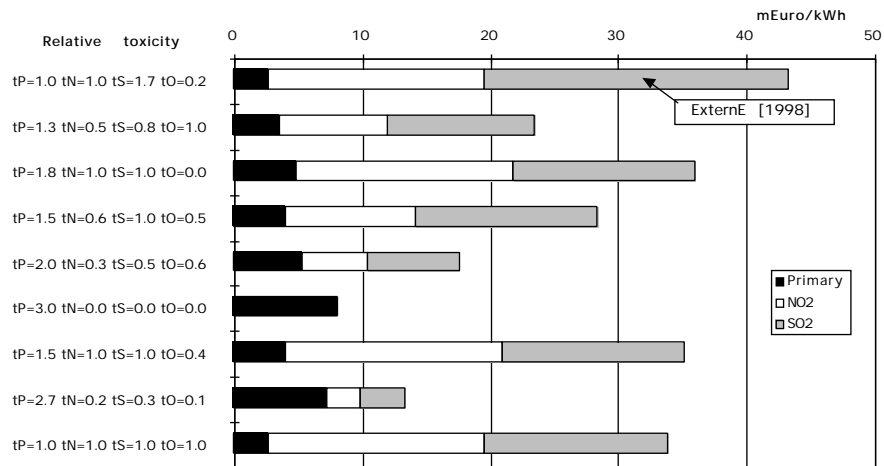


Fig.1. Total damage per kWh for a new coal fired power plant, for different hypotheses about relative toxicity of components of particulate matter.

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