Health Risks of Air Pollution from Incinerators: a Perspective

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Abstract

This paper offers a perspective on the health impacts of air pollution from incinerators, by making a number of comparisons, in terms of emissions, concentrations, damages and damage costs. The latter are estimated by an impact pathway analysis, tracing the fate of a pollutant from source to receptors. Linearity is assumed for the increase of damages with incremental exposure. The monetary valuation is based on a valuation of mortality in terms of years of life lost, rather than simply the number of premature deaths. The method is applied to the incineration of municipal solid waste (MSW) with emissions equal to the regulations proposed by the European Commission in 1994, for typical per capita MSW production. Even if all MSW is incinerated (in accordance with these regulations), the total health damage costs are relatively minor. Also, the impacts of dioxins and carcinogenic metals are small compared to those of particles, NO_x and SO_x.

Key words: health impacts, air pollution, incinerators, municipal solid waste, dioxins, toxic metals, dose-response function, impact pathway analysis, risk comparisons, mortality cost

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Health Risks of Air Pollution from Incinerators: a Perspective

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1. Introduction

Thermal treatment (incineration, pyrolysis, or gasification) is a major option for the disposal of waste, full of promise because it greatly reduces the space requirement of landfills (typically by a factor of 4 to 10) yet highly controversial because of perceived health risks from air pollution, especially dioxins. Whereas such fears are natural after the bad practices of the past, the decisions to be considered now concern new clean technologies, subject to stringent regulations such as the one proposed by the EC [1994]. With clean technologies the emission of pollutants is orders of magnitude lower than with the old unregulated incinerators that had little or no flue gas clean up.

In the present paper we offer a perspective on the health impacts of air pollution from incinerators, by making a number of comparisons, in terms of emissions, concentrations, damages and damage costs. We do not attempt a full analysis of the problem of waste treatment which should involve a comparison with alternative treatment options, but we provide elements for such an analysis. Also we do not address liquid or solid residues, other than to emphasize the need for good management. Compared to land filling, incineration can greatly reduce the quantity of organic pollutants (except for the creation of trace quantities of certain organic pollutants, in particular dioxins and furans). As for heavy metals, while their quantity does not change, their form and toxicity does; however, since the residues from incineration are much smaller in volume and more homogeneous than the original waste, it is much easier to ensure proper disposal in well-managed special landfills.

We try to quantify, as far as we have data and models, the damage due to air pollution by carrying out an impact pathway analysis (environmental fate) whose principal steps are the following:

- specification of the emissions (e.g. kg/s of particles emitted by stack);
- calculation of increased pollutant concentrations in all affected regions (e.g. $\mu g/m^3$ of particles, using models of atmospheric dispersion);
- calculation of damages (e.g. number of cases of asthma due to these particles, using a dose-response function);
- monetary valuation of this damage (e.g. multiplication by the cost of a case of asthma).

The resulting values are summed over all receptors, choosing the temporal and spatial boundaries of the analysis such as to ensure that essentially all the damage is taken into account. We draw on the major studies of external costs of pollution in the USA [ORNL/RFF 1994, Rowe et al 1995] and Europe [EC 1995]; the latter, known as the ExternE Program of the European Commission, is continuing and two of the authors are active participants [Rabl et al 1996] (for definition of external cost, see Glossary at end of paper). All of these studies have found the damage costs of air pollution to be dominated by health impacts, justifying the focus on health in the present paper.

To begin we comment on several key issues. The first is site dependence: a ton of SO_2 emitted in a big city causes severe health damage, but emitted in the middle of the ocean its health damage is negligible. Site dependence is explicitly taken into account in the impact pathway analysis. This realism is an awkward complication for the presentation of the results and their use for public policy. How could one obtain results that are "typical" and draw general conclusions?

To set the stage for general conclusions, we show results calculated by detailed site-specific modeling and compare them with a simple formula (the "uniform world" model) that follows from conservation laws in the limit of uniform receptor density. We find the simple formula instructive because it gives an order of magnitude estimate; the true damage can be about three times smaller or larger depending an stack height and type of site (rural vs. suburban).

The second key issue concerns epidemiology: the crucial assumption for our calculations of health impacts is linearity of incremental damage with incremental exposure. In reality the dose-response functions, or concentration-response (C-R) functions, may well have thresholds and curvature, but there are insufficient data to justify nonlinear models for our calculations. Linearity is commonly assumed for cancers (and justified by the data for some although not all cancers [Frith et al 1981]). Linearity is also suggested for population level mortality impacts of the classical air pollutants by Fig.3 of Dockery et al [1993] and Figs.1 and 2 of Pope et al [1995]. Note that for the calculation of incremental damage a threshold has no effect if it is below background exposure: a "hockey stick" gives the same results as a line through the origin with the same slope.

Linearity has important implications for public policy. Firstly, it directs the analysis toward the total population dose rather than peak individual doses, at least as far as new clean technologies are concerned because their emissions are so low that even the highest individual dose does not entail any significant augmentation of individual risk; also the analysis must extend over large regions to capture most of the damage. Secondly, linearity implies that there is no safe level for incremental emissions. Since the cost of pollution control is high and increases strongly as emissions are reduced further, rationality calls for risk comparisons and cost-benefit analysis.

The third key issue concerns monetary valuation. Monetization greatly simplifies the presentation of the results because it converts a large number of incommensurate impacts to a common unit. However, in view of the controversies surrounding the economic valuation of mortality, some people prefer to refrain from an economic valuation and use instead a multicriteria analysis. In any case, the information in this paper is sufficiently disaggregated to serve as input to a multicriteria analysis.

Going down the steps of an impact pathway analysis, the results come progressively closer to criteria of direct concern, but involve progressively more assumptions and uncertainties. It is therefore advisable to consider a gamut of different comparisons. As illustration we consider the incineration of municipal solid waste (MSW) with emissions equal to the regulations proposed by the EC [1994], for typical per capita MSW production (for the emissions we also highlight the possibility that energy recovery can greatly reduce the net emissions attributable to incineration). We offer the following comparisons, to the extent that we have been able to find suitable data:

- incremental emission compared to other emissions (natural and anthropogenic, e.g. cars);
- incremental concentration (or dose) compared to background concentration (or dose);
- incremental concentration (or dose) compared to health guidelines (EC or WHO);
- health risks from different pollutants compared to each other;
- incremental years of life lost compared to other risks of everyday life;
- incremental external cost compared to the cost of incineration itself;

The comparison of emissions and concentrations is instructive because it shows how significant MSW is relative to other pollution sources that we may or may not be able or willing to reduce. It also has the great advantage of being unaffected by the dominant source of uncertainty, namely epidemiology.

2. Emissions

2.1. Pollutants

In terms of total environmental costs the greenhouse emissions, especially carbon dioxide (CO_2) and methane (CH_4), are likely to dominate. However, most of their impacts are in categories other than direct health effects. Furthermore, an inventory of relative greenhouse gas emissions for alternative MSW treatment options would require fairly detailed specification of the landfill or other alternatives, in particular whether the CH_4 from the decay of organic matter is captured or not. That is beyond the scope of the present paper, and so we limit our discussion to the other air pollutants.

We consider the "classical air pollutants": the primary pollutants particulate matter (PM), nitrogen oxides (NO_x), sulfur oxides (SO_x), volatile organic compounds (VOC) and carbon monoxide (CO), as well as the secondary air pollutants nitrates (xNO_3), sulfates (xSO_4) and ozone (O_3). In addition we discuss heavy metals and dioxins (the "micropollutants"). We pay special attention to the latter because fear of dioxins is one of the main reasons for vehement opposition to most waste incineration projects.

Dioxin is a name for a family of 75 chlorinated aromatic compounds, to which one might add 135 closely related compounds, the polychlorinated dibenzofurans. Several of these are highly toxic, the most toxic being 2,3,7,8-tetrachlorodibenzo-p-dioxin, usually abbreviated TCDD; they may also be carcinogenic. Dioxins and furans can be produced in trace quantities during the combustion of chlorinated organic compounds. They are destroyed by exposure to light within hours, but in the soil they may persist for more than ten years [Tschirley 1986]. Implicated in the Seveso accident 1976 and blamed for health effects from the defoliation with Agent Orange in Vietnam, dioxins have acquired a frightful reputation.

Dioxins are not produced intentionally; rather, they are an undesirable byproduct of certain industrial processes. An important source is incineration of chlorinated plastics - which is one of the reasons why there has been so much opposition to waste incineration. But sources of dioxins are ubiquitous, and even the burning of ordinary wood produces some. Thus it comes as no surprise to find evidence for dioxins in preindustrial times [EPA 1994a, vol.II, p.3.146]. It is convenient to state all dioxin data as TEQ = toxic equivalent 2,3,7,8-TCDD, and we will do so throughout this paper. For MSW incineration we assume a mass ratio

$$\frac{\text{total dioxins}}{\text{dioxins TEQ}} = 60 \qquad (1)$$

as typical value in the USA [EPA 1994a, vol.II, p.3.104]. We note that the EPA's Scientific Reassessment of Dioxin, initiated in 1991 was published in preliminary draft form in August of 1994, in three volumes. The preliminary draft is labeled "review draft (do not cite or quote)" but as yet, a final draft or final health assessment document has not been produced.

2.2. Data and Regulations

Emissions depend on the composition of the waste and on the equipment used to treat the waste, especially the flue gas clean up; for a given installation the emission can vary with varying operating conditions. In Table 1 shows the concentration of pollutants in the flue gas, as

measured for older plants, and compares them with regulations in the US and Europe. The data for older plants are mostly based on data from the UK as reported by Williams [1994]. The regulations in the USA have been in effect for new incinerators since 1991. In the European Union the EC regulations of 1989 have been incorporated in the legislation of the member countries and apply to new incinerators; a further tightening as shown by the last column in Table 1 was proposed in 1994 (and will become law in due course).

Table 1. Emissions from MSW incinerators.
Measured data for older incinerators and regulatory limits for new incinerators. Blank = no data or regulations for the respective pollutant or group of pollutants.

	Older plant [a]	**US regulations** [b] for plants > 225 t/day	**EC regulations 1989**	**EC regulations proposed 1994**
Pollutant	**mg/Nm^3**		**mg/Nm^3**	**mg/Nm^3**
Particles	16 - 2800	34 mg/Nm^3	30	10
NO_2	180 - 360	180 ppm (340 mg/Nm^3)		200
SO_2	180 - 670	30 ppm (80 mg/Nm^3)	300	50
HCl	350 - 950	25 ppm (38 mg/Nm^3)	50	10
HF	4 - 9		2	1
CO	6 - 640	50 - 150 ppm (58 - 175 mg/Nm^3)	100	50
VOC				10 (organic C)
Pb	0.1 - 50			
Pb+Cr+Cu+Mn			5	
Ni+As			1	
Cd	<0.1 - 3.5			
Hg	0.21 - 0.39			0.05
Cd+Hg			0.2	
Cd+Tl				0.05
As+...+V [c]				0.5
Organic carbon			20	10
Dioxins, TEQ	1 - 40×10^{-6} [d,e,f]	0.5×10^{-6} mg/Nm^3 [g]		0.1 ×10^{-6}

[a] older plants, mostly for UK data, as cited in Table 1 of Williams [1994].

[b] New Source Performance Standards, as cited in Steverson [1994].

[c] As+Co+Cr+Cu+Mn+Ni+Pb+Sn+Sb+V.

[d] data for UK converted from Table 1 of Williams [1994], using TEQ = total dioxin/60 of Eq.1.

[e] EPA [1994a vol.I, p.23] indicates average dioxin TEQ for MSW incinerators in USA as 0.0000001 g/kg$_{waste}$, converted to 20×10^{-6} mg/Nm^3 by assuming 5150 Nm^3/t$_{waste}$ [ETSU 1996].

[f] for industrial waste incinerators built before 1980 Table 4 of Jones et al [1993] shows 1-7×10^{-6} mg/Nm^3 TEQ

[g] converted, using TEQ = total dioxin/60 of Eq.1.

There are technologies that easily meet even the proposed new EC regulations, as can be seen from the examples in Table 2. One is the MSW incinerator in Vienna which has been operating in its present version since 1992 [Wien 1995]. It is a showcase for clean technology, from the sophisticated clean up equipment (emissions monitored and publicly displayed in real time) to the exterior (designed by the painter and architect Hundertwasser). The other is a version of pyrolysis developed by Siemens and called thermolysis, with several features that minimize emission of pollutants and production of residues [Mortgat 1996]. For instance, the fly ash trapped by the electrofilter is sent back into the combustion chamber where it becomes part of

the slag. The gas leaving the pyrolysis unit is burned at 1300 ˚C to minimize the formation of dioxins. Liquid emissions are zero because waste water is evaporated. The process has reached technical maturity with a unit in Fürth, Germany, which has begun operations with a capacity of 100 000 t/yr. Clearly there has been impressive progress in pollution control.

Table 2. Measured emissions, in mg/Nm^3, for state-of-the-art thermal treatment of MSW: the incinerator of Vienna [Wien 1995] and the thermolysis process of Siemens [Mortgat 1996].

	Vienna (measured annual average)	Thermolysis (measured for pilot plant)
Particles	0.20	0.05
HCl	<0.80	< 0.2
HF	0.04	< 0.02
SO_2	7.90	0.8
CO	45.0	5.0
NO_2	17.0	< 70
Cr+Pb+Zn	0.06	
As+Ni+Co	<0.012	
Cd	7×10^{-4}	$< 5\times10^{-4}$
Hg	3×10^{-3}	$< 1\times10^{-4}$
other heavy metals		$< 1.2\times10^{-3}$
NH_3	3.5	
VOC	<1.0	0.28
Dioxins, TEQ	3×10^{-8}	4×10^{-9}

2.3. Emissions per Capita

For a first risk comparison of incinerators we calculate per capita emissions if all MSW were incinerated in plants that satisfy the proposed EC regulations, and we compare the results with emissions from other sources. For the quantities of solid waste produced, the data of Denison and Ruston [1990] indicate 250 to 750 kg/yr per capita in industrialized countries during the mid eighties. There is a tendency for waste to increase with general wealth, but this is compensated by source reduction and efforts to recycle. On balance we assume

$$\text{MSW production} = 500\ kg_{waste}/yr \text{ per capita} \tag{2}$$

as typical round number. Multiplying by a typical value of flue gas volume [ETSU 1996]

$$\text{flue gas volume} = 5150\ Nm^3/t_{waste} \tag{3}$$

and by the emissions in the last column of Table 1 we find the per capita emissions shown in the last column of Table 3. To find the emission in g/t_{waste}, multiply the last column of Table 3 by 2.

Comparison with other sources is less straightforward because most data are incomplete and/or uncertain. For total world emissions of heavy metals, both natural and anthropogenic, we have found data published by OECD [1995b] and by WRI [1994]; the latter numbers are up to several times higher. OECD [1995b] also lists metal emissions in 1982 for several European countries but not for the USA. For dioxins we have found total emission estimates only for the USA. Data for classical pollutants in 1990 can be found in OECD [1995a]. For Table 3 we have

selected two industrialized countries, France and the USA. We have converted the emissions data to g per capita by taking the populations as

World 5460 million,
France 57 million,
USA 255 million.

Table 3. Comparison of per capita emissions with MSW incinerators that respect proposed EC regulations (last column of Table 1), assuming 5150 Nm^3/t_{waste} and **0.5 t_{waste}/yr per capita**.

Blanks = we have not found any data.

	World, natural	**World, anthropogenic**	**USA, anthropogenic**	**France, anthropogenic**	**MSW incin. if EC reg. 1994**
Pollutant	**g/cap**	**g/cap**	**g/cap**	**g/cap**	**g/cap** [e]
Particles			27.8×10^3 [c]	4.0×10^3 [c]	26
NO_2			82.6×10^3 [c]	26.5×10^3 [c]	515
SO_2			76.5×10^3 [c]	21.3×10^3 [c]	129
HCl					26
HF					3
CO			329×10^3 [c]	170×10^3 [c]	129
As	1.4 [a]	3.4 [a]		0.00 [a]	≈0.04 (<1.29) e
Cd	0.2 [a]	1.4 [a]		0.70 [a]	≈0.10 (<0.13) e
Cr	10.6 [b]	17.2 [b]		2.26 [b]	≈0.08 (<1.29) e
Hg	1.1 [a]	0.7 [a]		0.19 [a]	0.13
Ni	5.1 [b]	18.0 [b]		6.10 [b]	≈0.44 (<1.29) e
Pb	3.5 [a]	60.8 [a]		108.0 [a]	≈0.28 (<1.29) e
Cd+Tl	>0.2	>1.4		0.70	0.13
As+...+V [f]	>20.7	>99.4		116.38	1.29
Dioxins,TEQ			3.65E-05 [d]		2.58E-07

[a] OECD [1995b], data for 1982.

[b] WRI [1994].

[c] OECD [1995a], data for 1990.

[d] EPA [1994b] vol.III, p.9.11.

[e] typical breakdown of metal emissions as estimated by ETSU [1996]: Cd =81% of Cd+Tl; As=3%, Cr=6%, Ni=34% and Pb=22% of As+...+V.

[f] As+Co+Cr+Cu+Mn+Ni+Pb+Sn+Sb+V.

The Pb levels of 1982 were much higher in Europe than in the USA because the general requirement of unleaded gasoline for new cars has been introduced only at the beginning of the nineties. For the classical air pollutants the per capita emissions in France are significantly lower than in the USA because of heavy reliance on nuclear power. There are also appreciable quantities of pollutants emitted by natural sources such as volcanoes (particles and SO_2) and lightning (NO_2); there is also H_2S emitted by biological sources and rapidly converted to SO_2. [Manahan 1994].

A first and very firm conclusion from Table 3 is that even if the entire MSW were incinerated (subject to the proposed EC regulations) the burden would be small (in most cases by orders of magnitude) compared to current per capita emissions in industrialized countries. A second conclusion is that the burden would not be large compared to world natural emissions (and for most pollutants it would be smaller), although that is less firm because of uncertainties of the latter.

These conclusions apply also to dioxins. The per capita burden would be two orders of magnitude less than current emissions in the USA. While we have no estimates for natural dioxin emissions, we can compare the emissions from simple burning of wood (forest fires, fire places, ...) per ton of wood with the emissions per ton of waste: dioxin emissions per ton of wood are about 1.0×10^{-6} g [EPA 1994a] whereas a ton of waste incinerated in accordance with EC [1994] generates only half as much (twice the last entry in Table 3).

Even though a comparison of emissions does not account for atmospheric dispersion and distribution of receptors, for most anthropogenic sources the situation is fairly comparable, most anthropogenic sources being very roughly proportional to population density. In any case, it is difficult to avoid the conclusion that incineration of MSW, if carried out according to the proposed EC regulations, is only a minor source of air pollution compared to other sources.

To underline this point we compare in Fig.1 the emissions of MSW incineration with emissions from private cars, both per capita. Three types of car are shown, representative of the current fleet in France [Joumard et al 1995]. The annual distance driven is 5850 vehicle km per person per year, based on data in OECD [1997] multiplied by a factor 0.75 to account for the fraction of the OECD data that corresponds to private cars. For MSW we show the regulation currently in force [EC 1989] and the proposed new regulation [EC 1994]; in addition we show the latter if there is heat recovery with the conditions of Table 4, averaged over gas and oil, as explained in Section 2.4. We also note that this comparison of emissions does not take into account the fact that particles from cars have higher damage per ton, being smaller (and more toxic) and emitted at street level rather than from tall stacks.

2.4. Energy Recovery and Avoided Emissions

There is another consideration: avoided emissions by virtue of energy recovery which can render the net emissions attributable to waste incineration much lower than the above values. Of course, net emissions depend on the energy source that is avoided. Obviously, the dirtier the displaced source, the lower the net emissions. Even though MSW is a dirty fuel, its incineration with clean technologies emits less than cleaner fuels with old technologies.

This is illustrated by the examples in Table 4, showing net emissions for three fairly typical conditions:

a) no energy recovery (2nd column);
b) the heat from the incinerator displaces a gas fired boiler (5th column);
c) the heat from the incinerator displaces an oil fired boiler (8th column).

For heat recovery we assume 2000 kWh_t/t_{waste} as typical value for incinerators coupled to a district heating system, based on data for MSW incinerators in Paris and in Vienna. Data for avoided emissions are adapted from Tabet [1994]. The SO_2 emissions are proportional to the sulfur content of the fuel since boilers and furnaces for space heating do not have flue gas desulfurization. Here we assume oil with a sulfur content of 1%. To explain the calculations for

Table 4, take particulate matter for which the emission is 52 g per t of waste. If heat recovery displaces an oil fired boiler that emits 0.11 g per kWh of thermal energy (designated by kWh_t), the credit per t of waste is 0.11 g/kWh_t × 2000 kWh_t/t_{waste} = 211 g/t_{waste}; the net emission is thus 52 - 211 = -160 g/t_{waste} (note that numbers have been rounded from original spreadsheet).

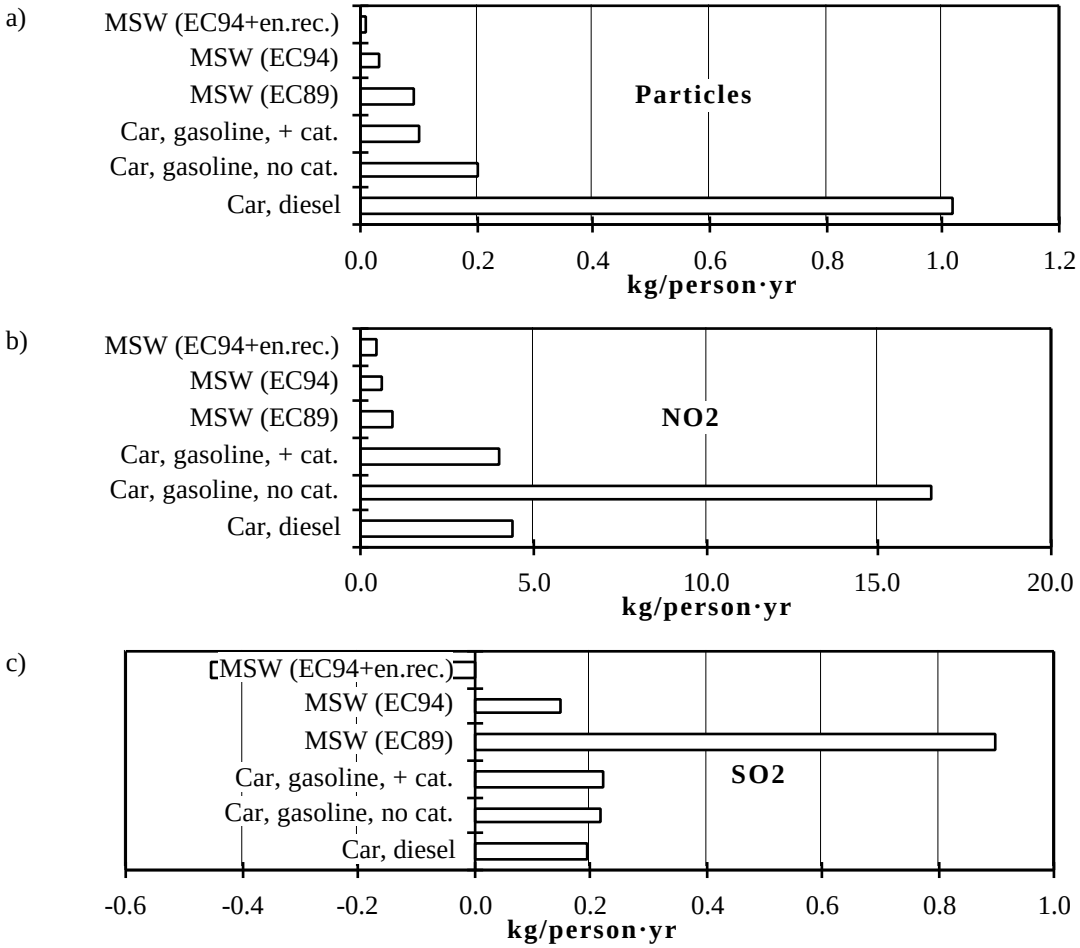

Fig.1. Emissions of MSW incineration compared with private cars, both per capita.
Cars representative of France, 5850 vehicle km/yr per person.
en.rec. = energy recovery, cat. = catalytic converter.

Table 4. Net emission of pollutants from MSW incineration (for EC 1994 regulation with flue gas volume 5150 Nm^3/t_{waste}), assuming 2000 kWh_t/t_{waste} heat recovery. Data for avoided emissions from Tabet [1994].

	Incinerator Emission	If incinerator displaces **gas** fired boiler or furnace			If incinerator displaces **oil** fired boiler or furnace @1% S		
		Avoided emission	Credit per t_{waste}	Net emission	Avoided emission	Credit per t_{waste}	Net emission
	g/t_{waste}	g/kWh_t	g/t_{waste}	g/t_{waste}	g/kWh_t	g/t_{waste}	g/t_{waste}
PM_{10}	52	0.00	0	52	0.11	211	-160
NO_2	1030	0.24	485	545	0.70	1393	-363
SO_2	258	0.00	0	258	2.12	4235	-3978
CO_2	861835	219	438406	423429	324	647059	214776

3. Dispersion and Peak Concentration

3.1. Effect Stack Height

Peak concentrations occur within a few km of the source. For modeling dispersion over the short range, up to tens of km from the source, the gaussian plume is considered adequate, and we have used the ISC model [Wackter and Foster 1987], a gaussian plume model approved by the USEPA. Over such short distances the removal rates of the pollutants under consideration are negligible; hence the same calculation gives the peak concentration for all pollutants.

Fig.2 show the variation of the peak concentration c_{max} (annual average), calculated by ISC for urban conditions with meteorological data for Paris. c_{max} decreases strongly as the stack height is increased. By contrast to the value of c_{max} its location does not vary much with stack height; this comes about because of a complicated interplay between stack height, stability classes and height of boundary layer. For this calculation Paris appears to be quite a representative choice: the wind speeds are average, and the wind directions are relatively uniform.

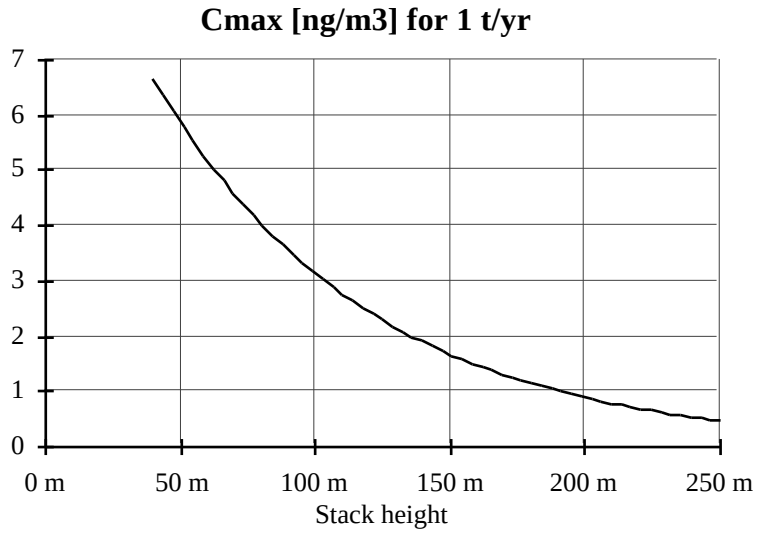

Fig.2. Maximum incremental concentration c_{max} due to incinerator, in ng/m^3 for an emission rate of 1 t/yr, as function of stack height, calculated with ISC [Wackter and Foster 1987] for urban conditions.

3.2. Comparison with Typical Ambient Concentrations

An interesting comparison emerges when one combines Fig.2 with the emissions data in Table 3. Let us consider an MSW incinerator with annual throughput of 250,000 t as appropriate for a city of half a million. If this incinerator has a stack height of 100 m and emits according to the proposed EC regulations, last column of Table 3, one readily finds the maximum incremental concentration values c_{max} in Table 5. Since the EC regulations for heavy metals are stated as total for a group, we estimate the emission of individual metals according to the breakdown given by ETSU [1996].

For comparison with c_{max} we show typical concentrations that have been measured in urban and rural environments. The concentrations for heavy metals are for Europe; note that Pb in Europe is high because unleaded gasoline has become compulsory for new cars in recent years only.

Table 5. Annual output of pollutant and resulting maximum incremental concentration c_{max} due to MSW incinerator that meets proposed EC regulations, with throughput of 250000 t_{waste}/yr and stack height 100 m. For comparison columns 4 -6 show typical ambient concentrations, and column 7 shows ratio of c_{max} and geometric mean of urban values. Last two columns show WHO guidelines and their ratio with c_{max}. Blanks = we have not found any data.

Pollutant [a]	Due to MSW incinerator t/yr	Due to MSW incinerator c_{max} ng/m^3	Typical ambient concentrations: Remote ng/m^3	Urban ng/m^3	Industrial ng/m^3	Urban/c_{max}	Health guidelines: EPA [c] ng/m^3	WHO [d] ng/m^3	WHO/c_{max}
As [b]	0.02	0.05	0-2	5-50	8-200	295			
Cd [b]	0.05	0.16	0.1-1	1-50	1-100	45			
Cr [b]	0.04	0.13	0-3	4-70	5-200	134			
Hg [b]	0.06	0.19	0.001-6	0.1-5	0.5-20	4		300	1580
Ni [b]	0.22	0.65	0.1-0.7	3-100	8-20	27			
Pb [b]	0.14	0.42	0.2-60	80-4000	50-450	1331	1.5×10^3	1×10^3	2350
Cd+Tl	0.06	0.19							
As+..+V	0.64	1.93		>126-5625		>436			
Dioxins	1.29×10^{-7}	3.86×10^{-7}		1.0×10^{-4}		260		4.4×10^{-4}	1300
PM	12.9	39	21×10^3	34×10^3	58×10^3	871	5×10^4	5×10^4	1280
NO_2	257.5	773	7×10^3	46×10^3	86×10^3	59	1.0×10^5	1.5×10^5	190
SO_2	64.4	193	4×10^3	25×10^3	52×10^3	129	8×10^4	5×10^4	260
CO	64.4	193		$1\text{-}5\times10^6$		11.5×10^3	1.0×10^7	1.0×10^7	51800
HCl	12.9	39							
HF	1.3	4							

Notes:

[a] As+..+V=As+Co+Cr+Cu+Mn+Ni+Pb+Sn+Sb+V; Dioxins as TEQ.

[b] typical breakdown of metal emissions as estimated by ETSU [1996], see Table 3.

[c] Guidelines by EPA [1991] for maximum permissible concentration (annual average, except quarterly for Pb and 8hr for CO).

[d] Guidelines by WHO [1987] for maximum permissible concentration; WHO limit for dioxins = 10 pg/kg·day TEQ, converted to 10 pg/kg·day×(2.2/119)× 0.42m^3/(kgbody.day)= 4.4×10^{-4} ng/m^3.

Sources for concentration data:

for PM, NO_2, SO_2 data for USA 1985 as cited in Chapter 2 of OECD [1995a];

for CO data for France 1993 [Stroebel R, V Berthelot and B Charré 1995];

for heavy metals: "Heavy metals: identification of air quality and environmental problems in the European Community", report EUR 10678 EN/I, Brussels-Luxemburg, as cited in T.5 of OECD [1995b]; includes only As+Co+Cr+Cu+Mn+Ni+Pb+Sb+V (no Sn);

typical dioxin TEQ concentration is 9.95×10^{-5} ng/m^3 in North America and 1.08×10^{-4} ng/m^3 in Europe [EPA 1994a, vol.2, Table 4.11].

Column seven, Urban/c_{max}, shows that the highest increment due to the assumed incinerator is small compared to existing backgrounds in urban or industrial environments; this is the case even for Hg which has the lowest ratio Urban/c_{max} = 4. For cases where a range is given under "Urban" we have taken the geometric mean because ambient pollutant concentrations tend to be lognormal [Ott 1995].

Finally we compare c_{max} with guidelines for public health; the ratio in the last column of Table 3.2 can be considered a "safety factor". Note that the comparison of c_{max} with EPA and WHO guidelines is independent of the calculation of damages, in the following Sections. If EPA

and WHO guidelines correspond to a threshold below which there is no impact, then none of the pollutants covered by the guidelines causes any damage, unless the existing background concentrations are already near or above the guideline: the last column in Table 5 (and analogous ratios for EPA) indicates that the safety factors are quite large, at least two orders of magnitude.

The calculation of damages and costs, by contrast, assumes linearity of incremental damage at current ambient concentrations, in clear contradiction with the interpretation of the guidelines as "zero risk" values. In view of the uncertainties surrounding C-R functions, we propose Table 5 and the calculations in the following sections as complementary perspectives.

4. Dispersion and Damage

4.1. Models

For most air pollutants from incinerators atmospheric dispersion is significant over hundreds to thousands of km [Seinfeld 1986, Zannetti 1990, Curtiss and Rabl 1966a]. Both local and regional effects are important. We have therefore used a combination of local and regional dispersion models to account for all significant damages. For modeling dispersion over the short range we have used the ISC model [Wackter and Foster 1987].

At the regional scale we have used two different models, the Harwell Trajectory model as employed by the ExternE Program [EC 1995], and the EMEP model of the Norwegian Meteorological Service [Barrett 1992, Sandnes 1993], the model chosen for the official allocation of acid rain budgets among the countries of Europe. The results presented here are a synthesis of calculations carried out in the ExternE Project of the European Commission [EC 1995, Rabl et al. 1996, ETSU 1996].

We have coupled these dispersion calculations with an integration over population data, using two software packages that have been developed independently for this purpose: ECOSENSE [Krewitt et al 1995] and PATHWAYS2.0 [Curtiss and Rabl 1996b]. ECOSENSE includes the Harwell Trajectory Model; for the PATHWAYS2.0 calculations we have used EMEP results for atmospheric dispersion. Both sets of calculations use ISC for the local dispersion. We have compared (not reported here) the results for total damage per ton of pollutant between these two sets of calculations and found agreement within approximately twenty percent. Results for the geographical range of the damage are shown in Fig.3 for a linear concentration-response (C-R) function, for two population distributions, one real (source in suburb of Paris), the other uniform. This graph illustrates the point made at the beginning of the paper about the need to extend the spatial boundaries over thousands of km.

For ozone damage due to the precursor NO_x we use a recent estimate by Rabl and Eyre [1997], based on EMEP results for the atmospheric dispersion and chemistry [Simpson 1993]. Only a single European average was derived, and we do not know how much the results would change if local ozone modeling were included. Taking only the morbidity and mortality costs of this paper, one has approximately 1150 ECU/t_{NO2} and 700 ECU/t_{VOC}. For VOC emissions the EC regulation states only a limit on organic carbon, so the exact mass of VOC emissions may be somewhat larger than the 10 mg/Nm^3 in Table 1. But in view of the NO_2 emission limit of 200 mg/Nm^3 it is clear that with these cost figures the damage of VOC is small compared to NO_2 and can be neglected.

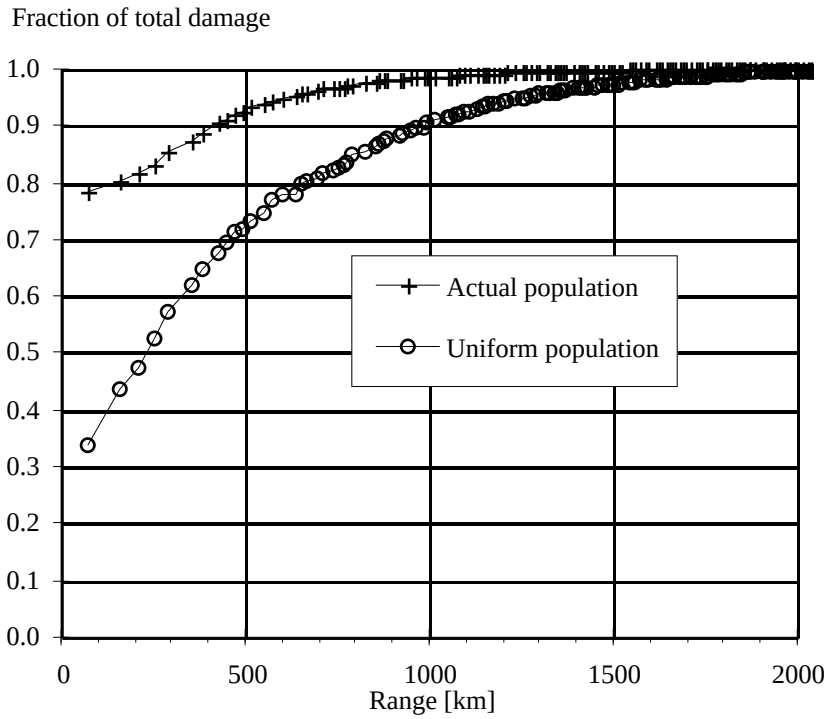

Fig.3. Calculated damage as fraction of total damage vs geographic range where calculation is cut off, with linear C-R function, for two distributions of population: uniform (with average population density of France) and real (for source in suburb of Paris, with stack height 25 m).

4.2 Site Dependence of Damage

Site dependence is particularly strong for primary pollutants, as shown by the examples in Fig.4 [Curtiss and Rabl 1966a]. For secondary pollutants, such as sulfates, nitrates and ozone, the sensitivity to local detail is lower because these pollutants are not created until some distance from the source. For nitrates and sulfates this occurs at tens to hundreds of km from the source, and so the site dependence is relatively weak; based on ECOSENSE results we estimate that variations of sulfate or nitrate damage, per ton of SO_2 or NO_2, with site are around 50%. The creation of ozone is more rapid, within several km to tens of km from the source; based on EMEP data we estimate site dependence of total ozone damage to be about a factor of four or perhaps more in Europe [Rabl and Eyre 1997].

Fig.4 shows in fact two variations at once: with stack height, and with source site for five specific sites in France (the nearest big city, 25 to 50 km away, is indicated in parentheses). Plume rise is included for typical incinerator conditions. As an example we consider a specific impact: the increase in acute mortality due to an emission of Q = 1 t/yr = 3.09E04 μg/s of SO_2 with the concentration-response (C-R) function of Sunyer et al. [1996] in Table 9 whose slope can be written as f_{CR} = 5.34E-06 YOLL/(pers.yr.μg/m^3). The damage is shown on two scales, as number of YOLL per year on the right hand scale, and in units of D_{uni} (to be explained below) on the left. At a stack height of 100 m the damage is about 3 times larger than D_{uni} for the site near Paris and about 0.4 times D_{uni} for Cordemais, a relatively rural site on the Atlantic Ocean. The damage for Martigues is rather small, despite the proximity of a large city, because the prevailing wind carries the pollutants out to sea.

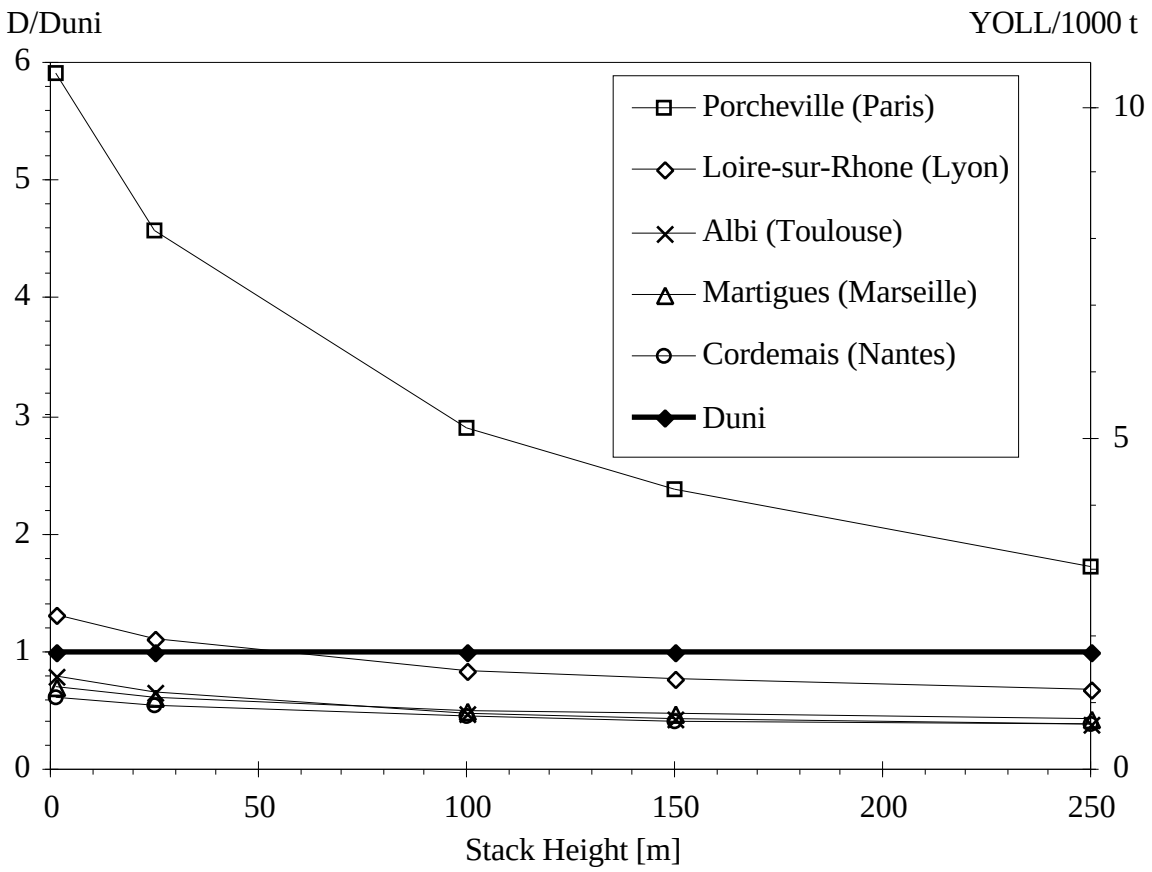

Fig.4. An example of dependence on site and on height of source for a primary pollutant: damage D from SO_2 emissions with linear C-R function, for five sites in France, in units of D_{uni} for uniform world model Eq.9 (the nearest big city, 25 to 50 km away, is indicated in parentheses). The scale on the right indicates YOLL/yr (acute mortality) from a plant with emission 1000 ton/yr. Plume rise for typical incinerator conditions is accounted for.

To explain D_{uni} let us write the damage D due to an emission Q as an integral over land area

$$D = f_{CR} \int dx \int dy \quad \rho(\mathbf{x})\, c(\mathbf{x}) \qquad . \tag{4}$$

where

$\rho(\mathbf{x})$ = density of receptors at point $\mathbf{x} = (x,y)$,

$c(\mathbf{x})$ = concentration increase at $\mathbf{x}$ due to Q, and

f_{CR} = slope of C-R function (concentration-response or dose-response).

The slope of the C-R function states the incremental number of cases (e.g hospitalizations) per concentration increment.

It is instructive 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]:

a) dry deposition (uptake at the earth's surface by soil, water or vegetation)

b) wet deposition (absorption into droplets, removed by precipitation),

c) decay or transformation (e.g. 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.

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

$$F_{dry}(\mathbf{x}) = v_{dry}\, c(\mathbf{x}) \tag{5}$$

where

$F_{dry}(\mathbf{x})$ = deposition flux [in kg/m^2·s], and
v_{dry} = dry deposition velocity [m/s].

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. Now let us define a "removal velocity" k(**x**) as ratio of the total removal flux

$$F(\mathbf{x}) = F_{dry}(\mathbf{x}) + F_{wet}(\mathbf{x}) + F_{trans}(\mathbf{x}) \tag{6}$$

and the surface concentration c(**x**) as

$$k(\mathbf{x}) = F(\mathbf{x})/c(\mathbf{x}) \;; \tag{7}$$

its units of k are m/s. Using F(**x**) and k(**x**) we can write the damage in the form

$$D = f_{CR} \int dx \int dy \quad \rho(\mathbf{x})\, F(\mathbf{x})/k(\mathbf{x}) \tag{8}$$

Let us now consider a situation where k(**x**) are ρ(**x**) are independent of **x**, with uniform receptor density ρ(**x**) = ρ_{uni} and uniform removal velocity k(**x**) = k_{uni}. In that case the integral in Eq.8 is simply

$$D = D_{uni} = \frac{f_{CR}\, \rho_{uni}}{k_{uni}}\; Q \tag{9}$$

because, averaged over time, the surface integral of the removal flux equals the emission

$$Q = \int dx \int dy \quad F(\mathbf{x}) \tag{10}$$

by conservation of matter (note that only time averages are of interest here). We shall refer to Eq.9 (and its generalization for secondary pollutants, Eq.14) as "uniform world model".

As an illustration we calculate D_{uni} for acute mortality due to SO_2, with f_{CR} = 5.34E-06 YOLL/(pers·yr·µg/m3) [Sunyer et al 1996]. By fitting ECOSENSE and EMEP dispersion data we have found a removal velocity k = 0.01 m/s for SO_2. Inserting these numbers into Eq.9 with the average population density ρ = 1.05E-4 person/m^2 as average for France we obtain for Q = 1 t/yr = 3.09E04 µg/s

$$D_{uni} = \frac{5.34\text{E-}06\ \text{YOLL/(pers·yr·µg/m3)} \times 1.05\text{E-}4\ \text{person/m}^2}{0.01\ \text{m/s}} \times 3.09\text{E}04\ \mu\text{g/s} \tag{11}$$
$$= 1.78\text{E-}03\ \text{YOLL/yr}$$

This is shown as the horizontal line in Fig.4. It lies right in the middle of the curves for the five sites.

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 is surprisingly small. The reason is that the total damage is dominated by regions sufficiently far from the source where the pollutant is fairly well mixed vertically in the planetary boundary layer, and variations of $k(\mathbf{x})$ are not too large. 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. Cordemais, in a semi-rural zone on the Atlantic cost, is an opposite extreme.

Thus it is plausible that these results are fairly representative and that the simple "uniform world model" of Eq.9 can be a useful first estimate, good to an order of magnitude. If one wants typical results for public policy, without being able to evaluate each and every site, D_{uni} seems as good a choice as any for incinerators in suburban settings with stack heights of 40 to 100 m. It also has the advantage of being simple and transparent.

Examining the results of detailed site specific calculations for more than fifty installations in the EU15 countries [ExternE 1997], we have found that D_{uni} of Eq.9, with k_{uni} of Table 6, represents the results for typical incinerator conditions if one chooses a population density ρ = 105 persons/km^2. This density is two thirds the average EU15 population density of 158 persons/km^2 per land area; this makes sense, however, since much of the EU15 is close to the sea, and averaged over land and nearby sea the effective density is lower. In the following we therefore calculate typical values of damages and damage costs, using k_{uni} of Table 6 and 105 persons/km^2.

A word about the k_{uni} of Table 6. For PM_{10} and SO_2 they have been determined by fits to concentrations calculated by the Harwell Trajectory Model (as integrated into ECOSENSE) and by EMEP. Since heavy metals and dioxins tend to be attached to particles, we take the same value as for PM_{10}. For CO we assume 0.001 m/s based on life time estimates of 36 to 110 days given by Manahan [1994], about a factor ten longer than for PM_{10} and SO_2.

For the damage D_2 due to a secondary pollutant we note that Eq.9 can be generalized to

$$D_{2uni} = \frac{f_{CR2}\ \rho_{uni}\ k_{1\text{-}2uni}}{k_{2uni}\ k_{1uni}}\ Q_1 \qquad (12)$$

where

Q_1 = emission rate of the primary pollutant,
f_{CR2} is the C-R function slope of the secondary pollutant,
k_{2uni} = removal velocity of the secondary pollutant, and
$k_{1\text{-}2uni}$ = average of transformation velocity, defined as

$$k_{1\text{-}2}(\mathbf{x}) = F_{1\text{-}2}(\mathbf{x})/c_1(\mathbf{x}). \qquad (13)$$

We can write this in the same form as Eq.9

$$D_{2uni} = \frac{f_{CR2}\ \rho_{uni}}{k_{2uni,eff}}\ Q_1 \qquad (14)$$

if we define an "effective removal velocity"

$$k_{2uni,eff} = \frac{k_{2uni}\, k_{1uni}}{k_{1\text{-}2uni}} \quad . \tag{15}$$

By numerical values by fits to detailed calculations with the Harwell Trajectory Model (as implemented in ECOSENSE) we have found the values in Table 6b.

Table 6. Values of k_{uni} for different pollutants, used in this paper for "uniform world model".

a) Primary pollutants, D_{uni} of Eq.9.

Pollutant	**k_{uni} [m/s]**
PM_{10}	0.01
SO_2	0.01
CO	0.001
heavy metals	0.01
dioxins	0.01

b) Secondary pollutants, D_{2uni} of Eq.14.

Pollutant	**$k_{2uni,eff}$ [m/s]**
NO_2 → nitrates	0.008
SO_2 → sulfates	0.019

5. Epidemiology and Economic Valuation

In this Section we discuss some general issues in the evaluation of health damages and costs. Specific results, based on the assumptions of the ExternE Project, will be presented in Section 6.

5.1. Acute and Chronic Effects

For the classical air pollutants the dose-response functions are usually based directly on air concentrations, hence the names C-R function (concentration-response) or exposure-response function seem more logical. There are two types, acute and chronic, reflecting different epidemiological approaches. The most common approach, and the easiest to implement, is to carry out a time series study of a population by identifying short term correlations (over a few days) between air pollution and a health end-point. One chooses a functional form (typically linear, logarithmic or exponential) with one adjustable parameter (more cannot be identified in practice), and determines the parameter by regression against the pollution data. All the functions used in this paper have been linearized. Time series studies identify only short term effects and yield acute C-R functions. This approach has the great advantage of being easy to implement and insensitive to the confounders (such as smoking) that plague the determination of chronic C-R functions.

End-points that show up only after a longer period require observations of several populations that are exposed to different average levels of pollution. Dose-response functions for chronic effects are notoriously difficult to establish with confidence, and there are only few studies that have determined chronic C-R functions. The difference between chronic and acute C-R functions is not so much in the exposure (most people are chronically exposed) as in the effects that are measured: do they show up within a few days after a pollution peak or only after a

longer period? By analogy the terms acute and chronic are also applied to C-R functions for mortality, even though the attributes appear strange in that context.

5.2. Relation between Concentration and Dose

For carcinogens the customary format is in terms of dose per body weight, and a conversion is required to link it to concentration. If one makes the conservative assumption that all of the inhaled mass of a pollutant is absorbed as dose, the relation between concentration c and daily inhalation dose I is simply

$$I = c \frac{V_{inhal}}{M_{body}} \tag{16}$$

where

M_{body} = body mass (for consistency with the unit of kg we use the term mass rather than weight) and

V_{inhal} = volume of air inhaled per day.

Based on the numbers in Table 7, we take 0.42 $m^3/(day \cdot kg_{body})$ as the population average of the ratio inhalation volume per body mass, and relate inhalation dose to concentration according to

$$I = c \times 0.42\ m^3/(day \cdot kg_{body}) \tag{17}$$

Table 7. Assumptions for the calculation of the dose [EPA 1989].

	man	**woman**	**child**	**average**
% of population	40%	40%	20%	
body mass, kg_{body}	70	60	20	
volume inhaled m^3/day	23	21	15	
$m^3/day \cdot kg_{body}$	0.33	0.35	0.75	0.42

With the assumption of linearity the dose-response functions for cancer are customarily stated as slope factor (SF). Cancer slope factors are usually estimated from human epidemiological or animal studies using mathematical models, most commonly the linearized multistage model, for estimating the largest possible linear slope (within the 95% confidence limit) at extrapolated low doses that is consistent with the data. The SF is expressed in units of mg intake per kg body mass per day. It represents the 95% upper confidence limit of the probability of a carcinogenic response per daily unit intake of a chemical over 70 years. Thus the slope factor is an upper-bound estimate, and so are the risks and costs of cancer calculated in this paper. The true risk to humans, while not known, is not likely to exceed the upper bound estimate. Data on SF's can be found in EPA's HEAST or IRIS database.

We will be stating the collective excess cancer risks per year of operation of the pollution source. Thus we divide the collective annual dose I by 70 yr to convert it to a continuous lifetime dose rate, for consistency with the definition of the slope factor. For the calculation we assume a continuous exposure over 70 years, even though few people will stay put in the same location over their entire life. This is appropriate for the collective risk with linear dose-response function, because only total dose matters, not which individual gets how much of this dose.

For example, the slope factor for inhalation of As is given by HEAST [1995] as 5.0E-2 cancers/(μg/kg_{body}·day), and we express it as population averaged C-R function slope f_{CR} as shown in Table 11

$$f_{CR} = \frac{\text{5.0E-2 cancers}}{\mu g/(kg_{body}\cdot day)} \; \frac{0.42\ m^3/(kg_{body}\cdot day)}{70\ yr} \tag{18}$$

$$= 3.01\text{E-4 cancers}/(yr\cdot\mu g/m^3) \text{ per person exposed .}$$

For carcinogenic heavy metals all the slope factors of HEAST are based on inhalation doses alone; hence a conversion according to Eq.18 is all that is needed. For dioxins, by contrast, noninhalation pathways are very important; we include them by means of a multiplicative factor as explained in Section 6.8.

5.3. Costs

Our economic valuation follows the ExternE program [EC 1995], including recent updates [ExternE 1997]. The unit costs will be presented in Section 6. Here we comment on the valuation of mortality because it is by far the most important component of the damage costs and also the most controversial. The key parameter is the reference value of life (also called statistical value of life) for which the ExternE Program is using

$$V = 3.1 \text{ MECU} \approx \$\ 3.5 \text{ million .} \tag{19}$$

A crucial question for the monetization of mortality is whether one should multiply the number of premature deaths N_{death} by the reference value of life V

$$cost_{Ndeaths} = N_{deaths} \times V \tag{20}$$

or whether one should base the valuation on the years of life lost (YOLL) = lifespan reduction ΔT

$$cost_{\Delta T} = N_{deaths} \times \Delta T \times v_{YOLL} \qquad ? \tag{21}$$

These two valuations can be considered extremes, with a continuum of possibilities in between.

This issue is particularly important for acute mortality, due to PM, SO_2 or O_3, which shortens life by a brief period. While there are no precise data, it is plausible that the life span reduction is perhaps a few weeks or months. In any case such a loss is certainly very much shorter than the typical loss implicit in value of life studies which is on the order of 30 to 40 years. Thus for the cost of acute mortality the difference between the two valuations is at least an order of magnitude.

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

$$V = v_{YOLL} + \frac{v_{YOLL}}{1+r} + \frac{v_{YOLL}}{(1+r)^2} + \ldots + \frac{v_{YOLL}}{(1+r)^{T_l}} \tag{22}$$

where r = discount rate and T_l = number of years of life lost. For r = 0 one finds $v_{YOLL} = V/T_l$. In the limit of very large discount rates V approaches v_{YOLL} and the two valuation methods

become equivalent. For example, suppose that a policy A can be implemented in two steps, the first, A', which increases life expectancy by 1 year, and the second, A", which adds 4 more years. For this latter the value according to $cost_{Ndeaths}$ is zero. In effect any years beyond the initial extension are discounted so heavily as to have no benefit. In other words, a policy that increases (decreases) life expectancy, without changing the number of premature deaths, has zero benefit (cost).

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

For these reasons we use a YOLL valuation. Valuing a year of life is of course a delicate matter, even more problematic than an average value of life V as used in Eq.19. Should the value of a YOLL be higher or lower for old age than for youth? There seem to be no firm data, and a priori one can think of arguments either way. For collective decision making it might be preferable to avoid such intergenerational questions altogether by using a single value per YOLL. That is also in the spirit of the general practice of using a single value of V for all individuals in a population, without distinction of wealth, health or will to live.

For the present paper we take v_{YOLL} as fundamental unit. Its value has been derived from the value of life V as a function of discount rate (3%) and life span reduction in value of life studies (35 years). Unfortunately the estimation of the life span reduction introduces major uncertainties, especially for acute mortality for which the ExternE program assumes 9 months per premature death. For chronic mortality the relative risk given by Pope et al [1995] can be converted to life span reduction per exposed individual [ExternE 1997, Rabl 1997], and this conversion has been assumed for the f_{CR} values used here.

The value of a YOLL also depends on when the loss occurs; thus the values in Tables 8 to 11 range from 0.083 to 0.155 MECU per YOLL, being different for different types of mortality because of different assumptions about the respective latency as explained in ExternE [1997]. For the cost of a cancer we assume 1.5 MECU, averaged over fatal and nonfatal cancers ExternE [1997].

6. Damages and Costs per Ton of Pollutant

6.1. Format of the Presentation

Most C-R functions in the literature have been reported as % increase in the base rate of occurrence of an end point, and multiplication by the respective base rate is required to yield the number of occurrences ("cases") per person per year per pollution increment. Only the product matters. To simplify the presentation in the present paper, we adapt the C-R functions to a uniform format by expressing their slope f_{CR} as number of cases per person·yr·μg/m^3, in Tables 8 to 11.

In addition to f_{CR} Tables 8 to 11 show the number of cases per ton of pollutant for typical incinerator conditions, calculated with the uniform world model D_{uni} of Eq.9, using k_{uni} of Table 6 and population density ρ = 105 persons/km^2 (as explained in Section 4.2). This is followed by

- the unit cost (=cost per case),
- the cost per person·yr·µg/m^3 (= cases per person·yr·µg/m^3 × cost per case), and
- the cost per ton of pollutant (= cases per ton of pollutant × cost per case).

We recall that an example for the calculation of the number of cases per ton of pollutant has been provided above in Eq.11: since the result 1.78E-03 YOLL/yr is for an SO_2 emission of 1 t/yr, there are 1.78E-03 YOLL/t, the "SO_2 acute mortality" entry in Table 9.

6.2. Particles

There are now numerous well-conducted studies linking particulate air pollution with a wide range of health effects, and there is no convincing evidence of a no-effects threshold [Dockery and Pope III 1994, Lipfert 1994, Wilson and Spengler 1996]. Particles have been associated with increased risk of cancer and a variety of noncarcinogenic effects on the respiratory system.

There is a growing tendency to treat the associations as causal, though the mechanism of action is unknown. It is not known for sure what size range or what components of respirable particulate air pollution are responsible. It seems however that particles emitted from combustion sources are more dangerous to health than wind-blown natural (crustal) particles.

Particles of more than 10 µm diameter are stopped in the upper respiratory ducts and appear less harmful. Between 2.5 and 10 µm, the particles penetrate more deeply into bronchi and bronchioles; particles smaller than 2.5 µm reach the alveoli of the lungs. Therefore most C-R functions have been based on the PM_{10} measure; in recent years some relations have also been based on $PM_{2.5}$. We assume a ratio of $PM_{2.5}/PM_{10}$ = 0.60 based on typical ambient concentration data in the USA.

In an impact pathway analysis the particulate emission data should of course be consistent in size and composition with the particulate measure in the C-R function. Unfortunately one is faced with a dearth of information. Most emissions data report TSP (total suspended particles) without any detail on size or composition. However, since most flue gas treatment is quite effective in removing larger particles and least effective for particles around a few microns, it is plausible that incinerator emissions are almost entirely PM_{10}, and this is indeed strongly suggested by measurements of coal fired boilers [Kim et al 1989]. On the other hand, application of $PM_{2.5}$ C-R functions is more problematic because we do not know what fraction is below 2.5 µm.

The dose-response function for acute mortality is based on short term correlations (time scales on the order of a day) of mortality data and ambient air pollution concentrations. The certainty is relatively high (95% confidence intervals around ±50%), but only part of the mortality impact is observed. The true mortality can be significantly higher due to chronic effects that do not show up in short term correlations. Recently two important studies have been published on chronic mortality from air pollution [Dockery et al. 1993, Pope et al. 1995]. These two cohort studies find clear associations of mortality with fine particles ($PM_{2.5}$) and with sulfates. Since these chronic mortality studies determine a change in age-specific mortality, one can derive implicit estimates of the YOLL (number of years of life lost). Thus in Table 8 the C-R for mortality function is stated in terms of YOLL, after converting from $PM_{2.5}$ to PM_{10} [ExternE 1997].

Table 8. C-R functions and costs for PM_{10}, as adapted and recommended by ExternE Program [EC 1995]. The exposure response slope, f_{CR}, has been expressed in units of cases/(person·yr·µg/m^3); cost is in ECU per case. Cases/t_{poll} and Cost/t_{poll} according to D_{uni} of Eq.9 with k of Table 6, for ρ=105 persons/km^2.

End point for PM_{10} and reference	**f_{CR}** cases per (pers·yr·µg/m^3)	**Cases** per t_{poll}	**Cost** ECU/case	ECU per (pers·yr·µg/m^3)	ECU per t_{poll}	**%** of PM_{10} cost
Chronic Mortality YOLL [Pope et al. 95]	4.10E-04	1.37E-01	84330	3.46E+01	1.15E+04	85.0%
CB, Adults [Abbey et al. 95]	3.92E-05	1.31E-02	105000	4.12E+00	1.37E+03	10.1%
RAD, Adults [Ostro 87] [a]	2.00E-02	6.65E+00	75	1.50E+00	4.98E+02	3.7%
Bronchodilator usage, Asthmatic adults [Dusseldorp et al. 95]	4.56E-03	1.52E+00	37	1.69E-01	5.62E+01	0.4%
Chronic cough, children [Dockery et al. 89]	4.14E-04	1.38E-01	225	9.32E-02	3.10E+01	0.2%
CB, children [Dockery et al. 89]	3.22E-04	1.07E-01	225	7.25E-02	2.41E+01	0.2%
HA, Cerebrovascular [Wordley et al. 97]	5.04E-06	1.68E-03	7870	3.97E-02	1.32E+01	0.1%
Cough, Asthmatic adults [Dusseldorp et al. 95]	4.69E-03	1.56E+00	7	3.28E-02	1.09E+01	0.1%
Congestive heart failure, Asthmatic 65+ [Schwartz&Morris 95]	2.59E-06	8.62E-04	7870	2.04E-02	6.79E+00	0.1%
Bronchodilator usage, Asthmatic children [Roemer et al. 93]	5.43E-04	1.81E-01	37	2.01E-02	6.68E+00	0.0%
HA, Respiratory [Dab et al. 96]	2.07E-06	6.89E-04	7870	1.63E-02	5.42E+00	0.0%
LRS, Asthmatic adults [Dusseldorp et al. 95]	1.70E-03	5.65E-01	7.5	1.27E-02	4.24E+00	0.0%
Cough, Asthmatic children [Pope&Dockery 92] [a]	9.34E-04	3.11E-01	7	6.54E-03	2.18E+00	0.0%
LRS, Asthmatic children [Roemer et al. 93]	7.20E-04	2.40E-01	7.5	5.40E-03	1.80E+00	0.0%
Total PM_{10}				**4.07E+01**	**1.36E+04**	100.0%

HA = hospital admission; CB = chronic bronchitis; LRS = lower respiratory symptoms;
RAD = restricted activity day; YOLL = years of life lost.
To derive f_{CR} from the data in the references (given e.g. as % increase per receptor), we have assumed:
3.5% of population is asthmatic, children are 20% of population, 14% of population is over 65.
For chronic mortality f_{CR} has been obtained by integration over life tables [ExternE 1997] or the Gompertz function for age-specific mortality [Rabl 1997], assuming that it applies only to the population over 30 years (= cohorts in Pope et al [1995]).
[a] ExternE [1997] decided to reduce these f_{CR} by a factor of 2 for the transfer from the USA to Europe in view of recent epidemiological studies in Europe that find lower PM_{10} impacts.

6.3. Sulfur Dioxide

The range and diversity of positive studies linking SO_2 with acute health effects is substantially greater than for NO_x, and human experimental studies are more suggestive of a real link. The situation is not entirely clear and may be confounded by simultaneous presence of particles. In several studies, apparent SO_2 effects disappear when particles are measured appropriately. In

view of recent studies in Europe that have found an SO_2 effect, we do include C-R functions for acute mortality and for respiratory hospital admissions. In any case the resulting costs, with a Y_{OLL} valuation, are relatively small. Results for the ExternE assumptions are shown in Table 9.

Table 9. C-R function slope f_{CR} and costs for SO_2 (direct and via sulfates), NO_2 (via nitrates and via O_3), and CO. Same format and assumptions as Table 8.

End point and ref.	**f_{CR}** cases per (pers·yr·µg/m^3)	**Cases** per t_{poll}	**Cost** ECU/case	ECU per (pers·yr·µg/m^3)	ECU per t_{poll}	**%** of SO_2 cost
SO_2 Acute Mortality YOLL [Sunyer et al. 96]	5.34E-06	1.78E-03	155000	8.27E-01	2.76E+02	2.3%
SO_2 Respiratory HA [Ponce de Leon 96]	2.04E-06	6.79E-04	7870	1.61E-02	5.35E+00	0.0%
SO_2 via xSO4 [a]					1.19E+04	97.7%
Total SO_2					**1.22E+04**	100.0%
NO_2, via xNO3 [b]					**1.69E+04**	
NO_2, via O_3					**1.15E+03**	
Total NO_2					**1.81E+04**	
CO Congestive heart failure asthmatics 65+, [Schwartz&Morris 95]	7.90E-08	2.63E-04	7870	**6.21E-04**	**2.07E+00**	

HA = hospital admission; YOLL = years of life lost.

[a] obtained by multiplying total in Table 8 by 1.67 of Eq.24 and by 0.01/0.019 for k's of Table 6

[b] obtained by multiplying total in Table 8 by 0.01/0.008 for k's of Table 6

6.4. Oxides of Nitrogen

Numerous laboratory animal studies on the health effects of NO_2 have been reported. Animal studies suggest that NO_2 may have effects on host defense systems such as pulmonary clearance, immunological function and susceptibility to respiratory infection. On the other hand, the epidemiological studies are not sufficiently convincing [Bascom et al 1996]. Even though some studies link NO_x or NO_2 with acute effects, the ExternE Program has not used these studies, because the apparent NO_x effect is arguably in reality not an effect of NO_x as such. Rather, NO_x may be a surrogate for a mixture of pollutants not otherwise well measured, including particles from combustion sources, especially traffic. NO_x is however implicated indirectly via nitrates and ozone.

6.5. Carbon Monoxide

Carbon monoxide (CO) is certainly toxic at concentrations much higher than found in typical urban environments; to protect human health, the ambient air quality standards of EPA and of WHO impose an upper limit of 10 mg/m^3, averaged over 8 hours. However, it seems that there are harmful effects at lower concentrations, and several recent studies have proposed linear C-R functions for CO. The evidence for a correlation with hospital admissions is quite strong and we have included it in Table 9. There may also be mortality impacts due to CO but the case is less clear.

6.6. Ozone

As mentioned in Section 3, we use an aggregated damage cost of 1150 ECU/t_{NO2} for average European conditions; it is the morbidity and mortality component of the cost estimate by Rabl and Eyre [1997]. The underlying assumptions for C-R functions and cost per case are listed in Table 10. The step from cost per ppb ozone to cost per ton of precursor involves results of the EMEP model for ozone formation [Simpson 1993], as explained in Rabl and Eyre [1997].

Table 10. Concentration-response function slope f_{CR} for O_3 health impacts and the corresponding costs. f_{CR} is based on 6hr concentrations (a factor 1.3 has been used to convert from 1 hr peak to 6 hr peak).

	f_{CR} (cases per person·ppb·yr) [a]	ECU per case [b]	ECU/(person·ppb·yr)	%
Acute mortality YOLL [Sunyer et al 1996]	1.17E-05	82500 [c]	0.965	36%
HA Respiratory [Ponce de Leon 1996]	1.42E-05	6600	0.093	3%
MRADs [Ostro et al 89]	1.95E-02	62	1.209	45%
Symptoms Days [Krupnick et al 90]	6.60E-02	6.3	0.416	15%
Total ECU/(yr·person·ppb)			**2.68**	100%

MRAD = Minor Restricted Activity Days; HA = Hospital Admission; YOLL = years of life lost.

[a] coefficients of Table 4.4 of EC [1995, vol.2] and updates [ExternE 1997], multiplied by 1.3

[b] from Table 16.3 of EC [1995, vol.2], except for acute mortality, see text below

[c] assuming average life span reduction of 9 months and 110 KECU per YOLL due to O_3.

6.7. Sulfate and Nitrate Aerosols

SO_2 and NO_x are transformed in the atmosphere to sulfates and nitrates, thus becoming a component of suspended particulate matter (we use the notation xSO4 and xNO3 for unspecified sulfates and nitrates). The detailed composition of these secondary aerosols depends on the local concentration of NH_3 which neutralizes sulfuric and nitric acid to form NH_4HSO_4, $(NH_4)_2SO_4$ and NH_4NO_3. The health impacts of these secondary pollutants are uncertain and their importance seems to have been recognized only in recent years, as a result of damage calculations performed in the ExternE Program [EC 1995] which found that the damage per ton of SO_2 and per ton of NO_2 was dominated by the assumption on the toxicity of these aerosols.

In the ExternE program the assumption has been made to apply the C-R functions for particles to these aerosols per concentration of pollutant mass. In particular, nitrate aerosols are treated like PM_{10}

$$f_{CR} \text{ for xNO3} = f_{CR} \text{ for } PM_{10} \qquad \text{per } \mu g/m^3 \tag{23}$$

and sulfate aerosols are like $PM_{2.5}$

$$f_{CR} \text{ for xSO4} = f_{CR} \text{ for } PM_{2.5} \; (= 1.67\, f_{CR} \text{ for } PM_{10}) \qquad \text{per } \mu g/m^3 \tag{24}$$

the conversion factor 1.67 being used for the usual case where C-R functions have been determined only with respect to PM_{10}; this conversion is based on ratios ($PM_{2.5}/PM_{10} \approx 0.6$)

that have been observed in ambient concentration data. The resulting damage estimates are very large, especially per ton of NO_2.

In the present paper we follow these assumptions, although with a strong warning about the uncertainties. There is insufficient epidemiological evidence for or against harmful effects of individual characteristics or components of particulate air pollution. Which characteristics determine the toxicity of particles: mass, particle number, composition, acidity, solubility, ...? While the evidence from smoking suggests that primary combustion particles are harmful, the evidence about secondary aerosols is not clear.

Although there are studies that report correlations of mortality and other end points with sulfate concentrations, it is not clear to what extent the true correlation is with some other component(s) of particles. There are no C-R functions for nitrates because in the past nitrates have not even been monitored as separate component of air pollution. Furthermore, nitrates and sulfates are mixtures of components with different properties, especially different acidities. Acidity has sometimes been implicated in epidemiological studies. NH_4NO_3, in particular, is a neutral and highly soluble substance and therefore no physical irritant, while HNO_3 is a strong acid.

6.8. Dioxins

Dioxin is one of the most thoroughly studied of all of the pollutants. Several human epidemiological studies and numerous studies in experimental animals have been carried out. There can be acute as well as chronic effects. Dioxins cause changes in laboratory animals that may be associated with developmental and hormonal effects; however, the mechanism of carcinogenicity is unclear. Whether the biochemical changes may result in adverse health effects in people and at what concentrations is not very well known.

In laboratory experiments with animals TCDD has been found to be one of the most potent toxins known, with LD_{50} ranging from 0.6 to 3000 µg per kg of body weight for different mammals (LD_{50} is the dose that kills half of a test group) [Tschirley 1986]. This wide range of values suggests that extrapolation from one animal species to another is quite uncertain.

However, Tschirley [1986] cites other bits of evidence that are directly relevant to humans. In particular, there is an experiment on prisoners, performed in days past when such experiments were not yet considered immoral. In one such experiment 60 prisoners were exposed, twice within 2 weeks, to a TCDD dose of 3 to 114 ng per kg_{body}. No symptoms were observed. An interesting additional data point, in the same reference, comes from another experiment with 10 volunteer prisoners who were exposed to a much larger dose of 107 µg per kg_{body}. Eight of them developed chloracne, but no other symptoms were noted.

These numbers indicate that man is certainly not among the most sensitive species as far as acute dioxin toxicity is concerned. They also suggest that the threshold for noncancer toxicity is at least 114 $ng/kg_{body}/(70 \times 365$ days) = 8.92E-06 $\mu g/kg_{body}\cdot$day, if one spreads the acute dose in the above experiment over a lifetime of 70 years. That should be a lower bound of the threshold because toxicity tends to be reduced if a dose is administered at a lower rate (e.g. fifty sleeping pills can kill if taken all at once). This number is close to the threshold 10 $pg/kg_{body}\cdot$day specified by the WHO [1987] as tolerable daily intake.

As for cancers, dioxins (2,3,7,8-TCDD and HxCDD) were said by EPA to be "the most potent carcinogen(s) evaluated by the EPA's Carcinogen Assessment Group". In this paper the only impacts of dioxins we quantify are cancers and we use the slope factor shown in Table 11.

Table 11. Cancer risks per t of pollutant, calculated with D_{uni} for emission rate of 1 t/yr, for population density of 105 persons/km^2.

	SF	**f_{CR}**	**Cases**	**Cost**		
	cancers per (µg/kgbody·day)	cancers per (pers.yr.µg/m^3)	per t_{poll}	ECU/case	ECU per (pers.yr.µg/m^3)	ECU per t_{poll}
As	5.00E-02	3.01E-04	1.00E-01	1500000	452	1.50E+05
Cd	6.10E-03	3.67E-05	1.22E-02	1500000	55	1.83E+04
Cr (VI)	4.10E-02	2.47E-04	8.22E-02	1500000	370	1.23E+05
Ni	8.40E-04	5.06E-06	1.68E-03	1500000	8	2.53E+03
Dioxin TEQ	1.00E+02	6.02E-01	1.08E+04	1500000	4.88E+07	1.63E+10

SF = slope factor

References:

for As, Cd, Cr, Ni: HEAST [1992]

for dioxin: "plausible upper bound" for slope factor vol.III p.9.85 [EPA 1994b].

In calculating the dose for dioxins, one needs to take noninhalation pathways into account, because dioxins are persistent and bioaccumulate, becoming concentrated in milk, meat and fish. Fig.II-5, p.37 of the report "Estimating exposure to dioxin-like compounds" vol.I Executive Summary [EPA 1994] indicates a typical value of

$$\frac{\text{total dose}}{\text{inhalation dose}} = \frac{119}{2.2} = 54.1 \tag{25}$$

for the ratio of total dose to inhalation dose for dioxins. The precise value can vary strongly with diet and local conditions.

In any case the noninhalation dose involves dispersion over large distances (e.g. by transport of cattle feed, followed by transport of meat). This has the effect of making the receptor distribution more uniform. Therefore we recommend estimating the total collective dioxin dose by calculating the damage due to inhalation in site specific manner and adding it to the "uniform world model" dose of Eq.9, the latter multiplied by the ratio of Eq.25 (after subtracting 1 to count only the non-inhalation dose),

$$D_{total} = D_{inhalation} + (54.1 - 1)\, f_{CR} \frac{\rho_{uni}\, Q}{k_{uni}} \tag{26}$$

where

D_{total} = total damage,

$D_{inhalation}$ = damage due to inhalation,

ρ_{uni} = average population density of the region,

Q = emission rate, in g/s, and

k_{uni} = mean removal velocity (= 0.01 m/s for particles).

The term $\rho_{uni}\, Q/k_{uni}$ is the collective inhalation dose for a uniform population density. By separating the inhalation dose as in Eq.26 one can take into account higher population densities for sources near cities. However, if the ratio of total and inhalation dose is indeed as large as indicated by Eq.25, the contribution of the first term in Eq.26 is so small that site dependence

can be neglected in most cases. In the present paper we assume therefore uniform population density for the inhalation dose as well. We note that the slope factor and the ratio of total and inhalation dose are uncertain and controversial.

6.9. Other Pollutants

Among the heavy metals the following are considered carcinogenic: arsenic (As), cadmium (Cd), chrome (Cr-VI) and nickel (Ni). The corresponding toxicity data are summarized in Table 11. Only inhalation dose has been taken into account because the available slope factors are for inhalation.

By contrast to the classical air pollutants and carcinogens, for noncancer impacts of heavy metals we only have data for thresholds below which no adverse effects have been observed. These threshold values, e.g. for mercury (Hg) and lead (Pb), have been taken into account in the ambient air quality guidelines such as WHO [1987]. As we have seen in Section 3, the incremental concentration due to the emissions from a modern incinerator are far below such guidelines. Thus there are no known observable health impacts for the typical situation where the background is already below the ambient air quality guideline, at least as far as inhalation doses are concerned. However, analogy with recent epidemiology of the classical air pollutants suggests that there might nevertheless be nonzero damages at the level of a large population, especially for substances such as Hg that can bioaccumulate.

Countless other pollutants may also be present, although we have not found any relevant data. Their risks are likely to be less significant; after all, if a pollutant has a strong effect, it tends to be noticed and epidemiological studies are carried out in response.

7. Summary of Health Costs

In this Section we summarize the results of the last Section and apply them to the emissions from an incinerator that satisfies the EC 1994 regulations. The health costs per t of pollutant are shown in Fig.5 and Table 12. The latter also gives an indication how much the damages may vary for different sites and stack conditions.

Table 12. Health damage costs per t of pollutant emitted in Europe, uniform world model D_{uni} of Eq.9 with population density 105 persons/km^2. (1 ECU = $ 1.00 to 1.25). Multipliers indicate how much the ECU/t_{poll} numbers can change with site and stack conditions (stack height h, temperature T, exhaust velocity v).

	Cost ECU/t_{poll}	**Multiplier for site** (rural ↔ urban)	**Multiplier for stack conditions** (height 250 ↔ 0m, T, v)
PM_{10}	1.36E+04	≈ 0.3 ↔ 3	≈ 0.6 ↔ 2.0
SO_2	1.22E+04	≈ 0.7 ↔ 1.5	≈ 1.0
NO_2 (via xNO3)	1.69E+04	≈ 0.7 ↔ 1.5	≈ 1.0
NO_2 (via O_3)	1.15E+03	?	?
VOC (via O_3)	7.0E+02	?	?
CO	2.07E+00	?	?
As	1.50E+05	≈ 0.3 ↔ 3	≈ 0.6 ↔ 2.0

Cd	1.83E+04	≈ 0.3 ↔ 3	≈ 0.6 ↔ 2.0
Cr	1.23E+05	≈ 0.3 ↔ 3	≈ 0.6 ↔ 2.0
Ni	2.53E+03	≈ 0.3 ↔ 3	≈ 0.6 ↔ 2.0
Dioxins, TEQ	1.63E+10	≈ 0.5 ↔ 2	≈ 0.6 ↔ 2.0

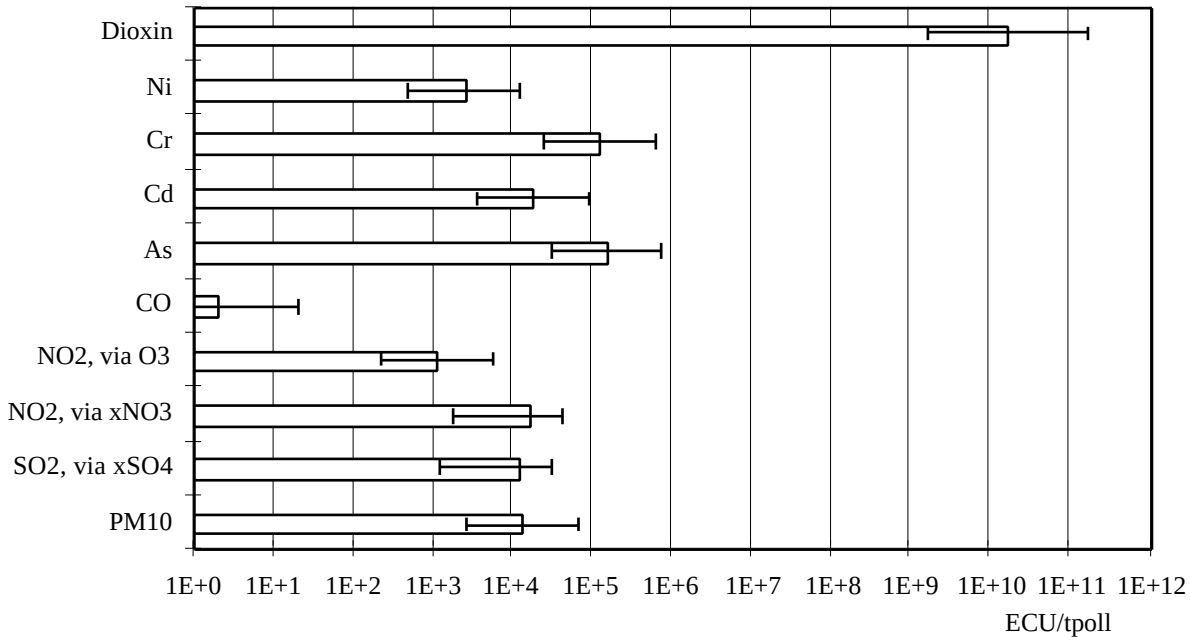

Fig.5. Mean damages per t of pollutant emitted in Europe, of Table 12.
Error bars indicate uncertainties as 1 σ_g intervals.

The error bars in Figs.5 and 6 indicate uncertainties as estimated by Rabl and Spadaro [1997] in consultation with the epidemiologists of the ExternE Program [Hurley 1997]. They are derived as geometric standard deviation σ_g and can be interpreted in terms of multiplicative confidence intervals of the lognormal distribution: if a cost has been estimated to be μ_g (geometric mean ≈ median) with geometric standard deviation σ_g, the probability is approximately 68% that the true value is in the interval $[\mu_g/\sigma_g, \mu_g.\sigma_g]$ and 95% that it is in $[\mu_g/\sigma_g^2, \mu_g.\sigma_g^2]$. Following the practice of the physical sciences we show error bars corresponding to 1 standard deviation, unlike epidemiology where 95% confidence intervals (≈ 2 standard deviations) are usually reported.

We show symmetric error bars, except for nitrate and sulfate aerosols the damages of which we believe that may well be lower than our central estimates. The reason for this belief is that chronic mortality (the dominant contribution) is plausible for primary combustion particles because of consistency with mortality from second hand smoke [Steenland et al 1996], whereas there is less evidence for chronic mortality due to nitrates and sulfates.

Fig.6 shows the implications for the damage per ton of waste, obtained by multiplying the numbers in Table 12 by the emissions per ton of waste. Despite their high toxicity, the micropollutants do not dominate the total damage. This is especially striking for dioxins whose toxicity is extreme but whose emission rate is so low that the damage per ton of waste is small compared to the classical air pollutants. Damage of NO_x via nitrates appears to dominate, but we recall the comment in Section 6.5 about the uncertainty of this pathway. Note that the damage estimates for the classical air pollutants are expectation values, whereas the ones for

micropollutants are upper bounds (95% confidence) because of the definition of the slope factor. Incidentally, for the carcinogens the costs can be converted to cancer cases by dividing by 1.5 MECU/cancer.

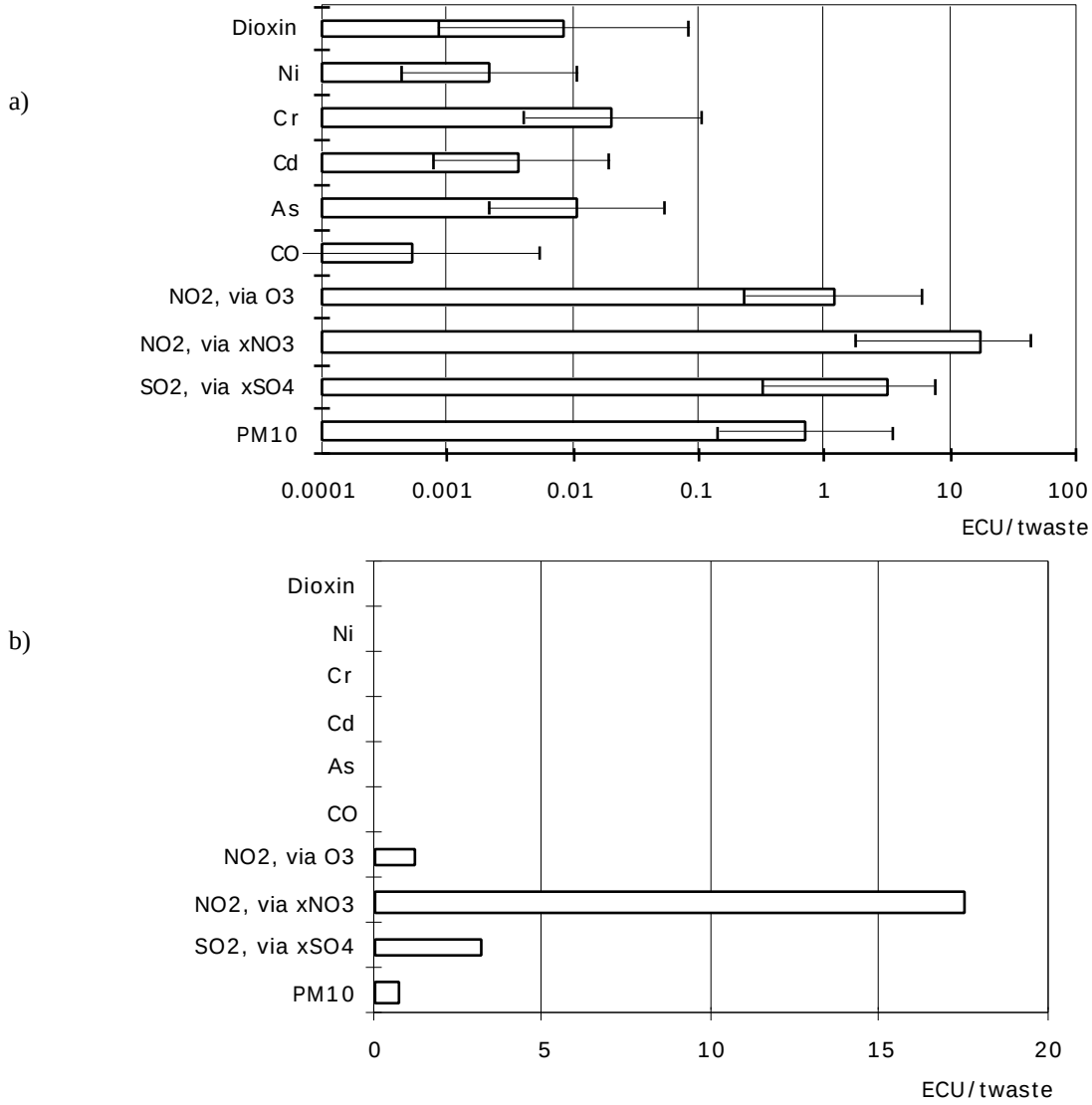

Fig.6. Damage per ton of waste, for the damage per ton of pollutant values in Fig.5 and emission rates of proposed EC regulation, Table 1. a) logarithmic scale, b) linear scale.

The sum of the health damage costs per ton waste is 12.3 ECU/t with and 3.8 ECU/t without the nitrate contribution. It is interesting to note that the market cost of MSW incineration in France is around 70 ECU/t with current (pre EC 1994) regulations and expected to increase about 25% with the new regulations [Peyrelongue 1997]. Therefore any remaining external costs after imposition of the EC 1994 Directive are small compared to the market cost of MSW incineration.

8. Risk Comparisons

The most relevant comparison would be with health damages from landfills and other waste management options, but unfortunately we have no suitable data. In fact, the difficulties are more general since most risks are not directly comparable. Mortality risks can differ in the nature of

the death (e.g. by accident, by cancer, or by other illness) as well as in attributes that affect the perception of a risk, such as:

- is the risk voluntary or involuntary?
- is the risk natural or manmade?
- to what extent is it associated with an activity that is considered socially desirable?
- how much control does an individual have over the exposure or consequences?

The classification of risks according to these attributes is not always clear. For instance, while radon is a natural substance, the exposure to radon depends on the design and construction of the residence where an individual lives; the individual can control the exposure to radon by making appropriate repairs.

Risks from exposure to air pollutants are involuntary and an individual has little or no control over the exposure (short of moving far away or filtering the air). Although society may benefit from waste reduction, there is little perceived benefit to an individual, unlike driving a car or smoking, which are voluntary risks. The risk is also clearly manmade.

The best format for the presentation of a risk comparison is not obvious. Risks could be expressed as life time total or as risks during an activity, e.g. actual time of driving a car [Johnson and Kearfott 1997]. Here we prefer to show years of life lost as average annual rate YOLL per person per year. Furthermore, a simple comparison such as in Table 13 does not distinguish individual differences, for instance between reckless drivers and people who rarely venture into the street. All the risks in Table 13 are averaged over the entire population, and the calculations are greatly simplified because in such a comparison only orders of magnitude matter, not precise numbers. After all these precautionary remarks, the risk estimates in Table 13 are presented to help put different risks in perspective. They indicate that health risks from MSW incineration are relatively unimportant.

9. Conclusions

We have assembled data on emissions, ambient concentrations and concentration-response functions for air pollutants from thermal treatment of waste, and we have used this information to estimate health damages and costs (and health effects dominate the total damage costs [EC 1995]). Issues of site dependence and geographic extent of the damage have also been addressed. For the quantification of damages and costs we have assumed linear dose-response functions, to the extent to which they are known.

On the basis of our data and calculations we have presented several comparisons to put the risks of incinerators in perspective. Assuming 500 kg/yr as typical per capita production of MSW, incinerated with emissions equal to the new regulations proposed by the EC [EC 1994] we have made several comparisons, to the extent that we have been able to find suitable data:

- incremental emission compared to other emissions (Section 2.3);
- incremental concentration compared to background concentration (Section 3.2);
- incremental concentration (or dose) compared to health guidelines (Section 3.2);
- health risks of different pollutants compared to each other (Section 7);
- incremental years of life lost compared to other risks of everyday life (Section 8);
- incremental external cost compared to the cost of incineration itself (Section 7).

By any of these comparisons the health impacts of MSW incineration appear insignificant, if the emissions respect the proposed EC 1994 regulations.

Table 13. Comparisons of some risks, expressed as years of life lost YOLL per person per year. Risks from air pollution due to MSW incineration assume 0.5 t of waste per yr per person for average conditions of Europe (EU15) subject to regulations of EC [1994], as in the rest of this paper. The risks are averaged over the entire population, except for radon for which we consider continuous exposure at the level of 150 Bq/m^3 = 4 pCi/l beyond which repairs are recommended in the USA.

	Deaths/yr α	Population β	Deaths/pers.yr γ = α/β	YOLL/death δ	**YOLL/pers.yr** = γ δ
Car accidents, France	9000	5.7E+07	1.58E-04	40	**6.3E-3**
Car accidents, USA	45000 [a]	2.5E+08	1.76E-04	40	**7.0E-3**
Lightning, France	30 [b]	5.7E+07	5.26E-07	40	**2.1E-5**
Lightning, USA	1760 [c]	2.5E+08	7.04E-06	40	**2.8E-4**
Tornados, USA	1760 [c]	2.5E+08	7.04E-06	40	**2.8E-4**
	Cancers/pers.life α	Relative Risk β	Cancers/pers.yr γ = α (β - 1)/70	YOLL/cancer δ	YOLL/pers.yr = γ δ
Lung cancer from **radon** in home with 150Bq/m^3	0.07	1.14 [d]	1.40E-04	10	**1.4E-3**
Air pollution from **MSW incineration**	t_{poll}/pers.yr α	ECU/t_{poll} β	ECU/YOLL γ	YOLL/t_{poll} δ = β/γ	YOLL/pers.yr = α δ
PM10	2.60E-05 (Table 3, last column)	11523 (Table 8, chron. mortal. only)	84330 (Table 8, chron. mortal. ECU/case)	0.137	**3.6E-6**
Dioxins	2.58E-13 (Table 3, last column)	1.63E+10 (Table 11, last column)	1.50E+05 (Table 11, if 10 YOLL/cancer)	1.08E+05	**2.8E-8**

notes:

the greek letters explain the calculations

[a] USDOC [1993]

[b] Frémy and Frémy [1996]

[c] Crouch and Wilson [1982]

[d] Lubin and Boyce [1997]

In particular, the incremental concentration of pollutants from such incinerators is far below the ambient air quality guidelines of the EPA or WHO. Since current background concentrations are usually also well below such guidelines, so is the total. Thus there would be no damage if these guidelines were no-effect thresholds. That does not guarantee the complete absence of harmful effects, but whatever they may be, air quality guidelines do not suffice to quantify them.

The cancer impacts of micropollutants, in particular of dioxins, are small compared to the mortality due to ordinary particulate matter from MSW incinerators which in turn is insignificant compared to the contribution of other sources of particulate matter or compared to other risks of everyday life. Similar conclusions about dioxins have also been reached by Eduljee and Gair [1997].

An assessment of the impacts of MSW incineration must not overlook the benefits of avoided emissions from energy recovery, for district heating, industrial process heat or power generation: the net emissions can be quite small or even negative. Even though MSW is a dirty fuel, the clean technologies required by EC 1994 regulations or equivalent ensure that the emissions will be lower than most conventional combustion technologies with much cleaner fuels.

Finally we note that regulations alone do not guarantee good environmental performance. Verification is crucial. As a good example we might mention the incinerator of the City of Vienna which publicly displays measured emissions data in real time [Wien 1995].

Acknowledgments

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Glossary

As = arsenic
c = concentration
CO = carbon monoxide
Co = cobalt
COPD = Chronic Obstructive Pulmonary Disease
Cr = chromium
C-R function = concentration-response function
Cu = copper
CVM = contingent valuation method
D = damage
Discount rate = rate r that allows comparison of monetary values incurred at different times, defined such that an amount P_n in year n has the same utility as an amount $P_0 = P_n\ (1+r)^{-n}$ in year 0.
EC = European Commission
ECU = European currency unit (1 ECU = 6.60 FF ≈ $ 1.00 to 1.25)
EMEP = European Monitoring and Evaluation Programme
EPA = Environmental Protection Agency of USA
ERV = Emergency Room Visit
EU = European Union (EU15 = the current 15 member countries)
External costs = costs that arise when the social or economic activities of one group of people have an impact on another for which the first group does not fully account, e.g when a polluter does not compensate others for the damage imposed on them.
F = removal flux [$kg/m^2 \cdot s$]
FGD = flue gas desulfurization
f_{CR} = slope of concentration-response function [$cases/(person \cdot yr \cdot \mu g/m^3)$]
I = inhalation dose [$\mu g/(kg_{body} \cdot day)$]
ISC = Industrial Source Complex gaussian plume dispersion model
Hg = mercury
k = removal velocity [m/s]
kWh = kilowatthour (with subscript $_t$ if thermal and $_e$ if electric)
KECU = 1000 ECU
mECU = milli ECU
MECU = million ECU
Mn = manganese

MSW = municipal solid waste
N = nitrogen
Ni = nickel
Nm^3 = m^3 under normal conditions of temperature and pressure
NO_x = unspecified mixture of nitrogen oxides, especially NO and NO_2
O_3 = ozone
ppb = parts per billion
Pb = lead
PM_d = particulate matter with aerodynamic diameter smaller than d μm.
Q = emission rate of a pollutant [kg/s or t/yr]
r = discount rate
RAD = Restricted Activity Days
S = sulfur
Sb = antimony
SF = slope factor (see Section 5.2)
Sn = tin
SO_x = unspecified mixture of sulfur oxides, especially SO_2 and SO_3
t = ton = 1000 kg (with subscripts $_{poll}$ for ton of pollutant and $_{waste}$ for ton of waste)
TEQ = toxic equivalence 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
Tl = thallium
TSP = total suspended particles
V = vanadium
V = reference value of life (also called value of statistical life VSL) [ECU or $]
VOC = volatile organic compounds.
VOL = reference value of life (also called value of statistical life VSL)
v_{YOLL} = value of a YOLL [ECU or $]
WHO = World Health Organization
WTP = willingness to pay
xNO3 = unspecified nitrate aerosols
xSO4 = unspecified sulfate aerosols
YOLL = years of life lost (reduction of life expectancy)

ρ = population density
ΔT = years of life lost (reduction of life expectancy)

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