Health Impacts of Pollutants

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Ari Rabl, PhD
Ecole des Mines, 60 boul. St.-Michel, 75272 Paris CEDEX 06, FRANCE
ari.rabl@ensmp.fr
http://www-cenerg.ensmp.fr/english/themes/index.html
click on Impacts of Energy Production

Part I: General Considerations

Air Pollutants and their effects on health

Primary Pollutant	Secondary Pollutant	Impacts
particles (BS, PM_{10}, $PM_{2.5}$)		**mortality** **morbidity** respiratory and cardio-vascular, asthma, reduction of lung capacity *(hospitalization, consultation of doctor, sick leave, restricted activity)* cancers
SO_2		**mortality** **morbidity** respiratory and cardio-vascular, asthma, reduction of lung capacity *(hospitalization, consultation of doctor, sick leave, restricted activity)*
SO_2	**sulfates**	like particles?
NO_x		**morbidity** (but direct effects of NOx not important)

Air Pollutants and their effects on health, cont'd

NO_x	**nitrates**	like particles**???** (lack of epidemiological studies)
NO_x+VOC	**ozone**	**mortality** **morbidity** respiratory, eye irritation
VOC (volatile organic compounds)		little or no direct effects at typical ambient concentrations (except PAC)
PAC (polycyclic aromatic compounds)		**cancers**
CO		**mortality** **morbidity** cardio-vascular
dioxins		**cancers**
As, Cd, Cr, Ni		**Cancers, other morbidity**
Hg, Pb		**morbidity** (neurotoxic)

Sources of environmental cancers

(related to energy)

Carcinogen	Source
Some **organic compounds**, especially certain polycyclic aromatic hydrocarbons and chlorinated compounds, e.g.:	
benzene	gasoline, cigarettes
benzo-pyrenes	cars
soot (products of incomplete combustion)	combustion, e.g. particles from diesel motors
dioxins	trace contaminant released by certain industrial processes (pesticides) and by combustion in the presence of Cl
PCBs (polychlorinated biphenyls)	

Sources of environmental cancers, cont'd

Metals:	trace contaminants of **coal** and **oil**, also released by waste **incineration**
Arsenic (As)	
Cadmium (Cd)	Ni-Cd batteries, additives to paint
Chromium (Cr, in oxid. state VI)	tanning
Nickel (Ni)	Ni-Cd batteries
Radiation e.g.:	soil, buildings, cosmic, x-ray, nuclear weapons, etc.
radon (Rn)	buildings, uranium mines and mill tailings

note **difference between source and exposure**, e.g. for benzene

Source of atmospheric benzene	**Exposure**
cars 82%, industry 14% cigarettes 0.1%	cars 18%, industry 3% cigarettes, active 40% cigarettes, passive 5%

source of information on carcinogens:
the IRIS database of US EPA, http://www.epa.gov/ngispgm3/iris/

Approaches to measure health impacts

1) **Epidemiology**: comparing populations with different exposures.

2) **Laboratory experiments with humans**: exposure in test chambers with controlled concentration of air pollutants (but this approach is very limited because of ethical constraints).

3) **Toxicology**:

a) Expose animals (usually rats or mice) to a pollutant; sample sizes are usually very small compared to epidemiological studies, and the animals are selected to be as homogenous as possible (unlike real populations). *Extrapolation to humans???*

b) Expose tissue cultures to pollutants. *Extrapolation to real organism???*

Approaches to measure health impacts, cont'd

Epidemiology: can measure impacts on real human populations, by observing correlations ("associations") between exposure and impact. But in most cases the **uncertainties** are very **large**. *Is the impact due to the pollutant or due to other variables that have not been taken into account (the problem of "confounders", especially smoking)?*

Toxicology: can identify **mechanisms of action** of the pollutants. For many substance tests with animals are the only way to identify carcinogenic effects. Toxicology can also suggest new questions to be investigated by epidemiology.

The two approaches are complementary.

Types of epidemiological studies

1) **Time series** (only for air pollution):

Observe correlations, in a large city, between concentration and occurrence of health impacts during the following days (in practice at most during the following five days).

Advantage: inexpensive; most confounders (especially smoking) are eliminated.

Disadvantage: only acute effects can be observed.

2) **Cohort studies**:

Compare different populations, using detailed information on the individuals to minimize effect of confounders.

Advantage: can observe chronic effects.

Disadvantage: expensive; often requires observations over many years; confounders are difficult to eliminate.

There are other types, and several variants, e.g. observation of population during a large and permanent change of exposure (e.g. Dublin and Hong before and after new regulation on use of certain fuels).

Dose-response functions (DRFs)

(for air pollutants also known as exposure-response or ***concentration response functions****)*

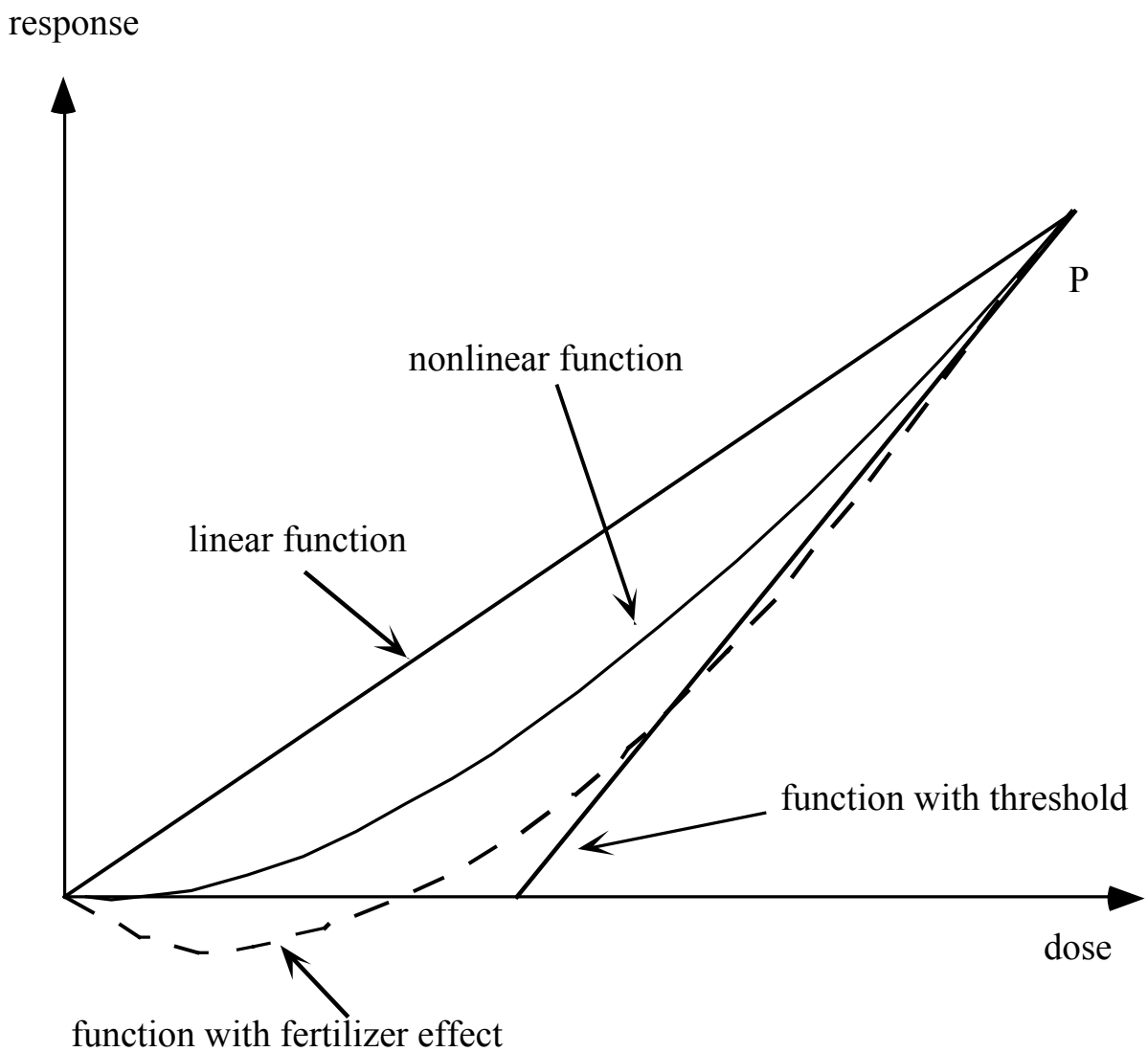
Crucial for calculating impacts of a pollutant.

Note:

a) most epidemiological studies do not report explicit DRFs but only a **relative risk** (= increase in occurrence of a health impact due to increase of exposure). To obtain DRF one also needs data on background rates of occurrence.

b) Watch out for **consistency** of DRF with the specification of exposure (calculated by dispersion models) and with monetary valuation. E.g. is exposure specified as hourly peak or as 24 hr average?

Functional form of dose-response functions at low doses



Functional form of dose-response functions at low doses, cont'd

The problem: in most regions the concentrations are so low that their impacts are difficult or impossible to measure. Suppose P is the lowest concentration where an impact could be measured with reasonable accuracy. **How should one extrapolate to lower doses?**

All of these functional forms can occur, for example

linear: radioactivity, particulates $\rightarrow$ health

threshold: ozone $\rightarrow$ crops

fertilizer: SO_2 for crops

but apparently nothing above linear in low dose limit.

Linearity without threshold seems to be the **most plausible for health** impacts of air pollutants and for substances that initiate cancers (also for radioactivity).

Is it Causal?

Hill's criteria for causality

of statistical correlations ("associations") found in epidemiological studies

Hill's criteria	**Situation for air pollution**
Strength of the association (is effect weak or strong?)	effects at typical exposures are **weak**, only observable in large populations (large uncertainty in individual studies)
Consistency of the association (is effect same at different times and places?)	Mostly **yes**, consistent results in North America, Europe, Asia, South America
Temporality (does effect occur after exposure?)	**Yes**
Dose-response (does effect increase with exposure?)	**Yes**
Biological plausibility (is effect plausible in terms of biological mechanisms?)	In recent years more and more studies have identified mechanisms, but are they sufficient**?**

Is it Causal?

Hill's criteria for causality, cont'd

Hill's criteria	**Situation for air pollution**
Specificity of the association (is exposure to specific pollutant associated with specific effects?)	**Respiratory** and **cardiovascular** illness Problem: composition of PM is not well defined; association with specific pollutants is not clear
Coherence (of whole body of data, includ. animal studies etc)	**more or less**
Experimentation (does removal of exposure remove effect?)	limited data, yes *(London episode 1952; shut down of steel mill Utah Valley 1986-87; fuel change in Dublin and Hong Kong)*
Analogy (is effect plausible on the basis of analogous situations?)	smoking, exposure to radiation

Slow convergence towards a consensus :
"air pollution appears to be harmful to your health"

but uncertainties about some specifics, in particular which pollutant causes which effects. The dominant opinion in the US has been that **PM** and **O_3** are the main culprits, but recent results suggest that direct effects of **SO_2** and **CO** may be important after all.

Major uncertainty: composition of PM

Quite variable, typically

soot and other direct combustion particles 10 to 30%

soil particles 10 to 50% (wind blown or stirred up by human activities)

sulfates 10 to 30%

nitrates 10 to 30%

Some nitrates and sulfates are of natural origin

What is relative toxicity of soil particles, nitrates and sulfates?

Role of other characteristics (acidity, solubility, surface area, number of particles, detailed composition)? Synergistic effects?

Part II: Specific CRFs

(concentration-response functions)

1) Mortality

Time series: determine only acute effects **("acute mortality")**

well over 100 studies in many different countries (North America, Europe, Asia, South America)

Cohort studies: determine **total** mortality**("chronic mortality")**

Authors	Sample	Duration	Result
Abbey et al. 1999	6000 non smoking sect, CA	10 yrs	Cardio-pulmonary mortality for men but not for women.
Dockery et al. 1993	8000 6 cities USA	14 - 16 yrs	Cardio-pulmonary mortality and lung cancers
Pope et al. 2002	550000 151 cities USA	16 yrs	Cardio-pulmonary mortality and lung cancers
Hoek et al 2002	5000 in Netherlands	8 yrs	Cardio-pulmonary mortality

What is observable?

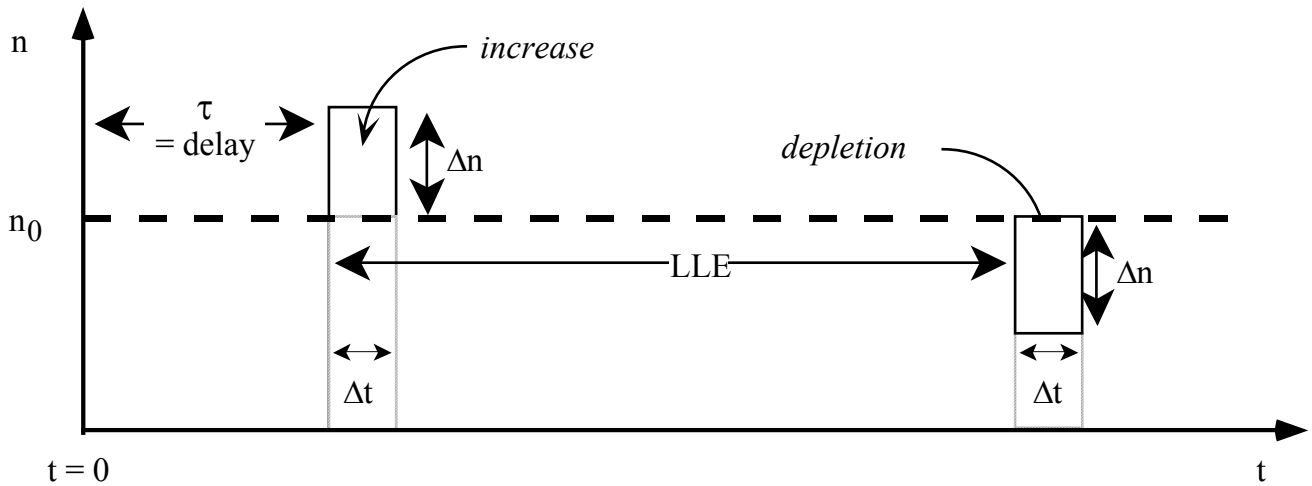
Air pollution is contributing cause (not a primary cause identified on death certificates) ⇒ Dose-response functions can only be inferred from variations in total number of deaths

Consider stationary population (birthrate = death rate = constant)

In absence of pollution daily death count = n_0

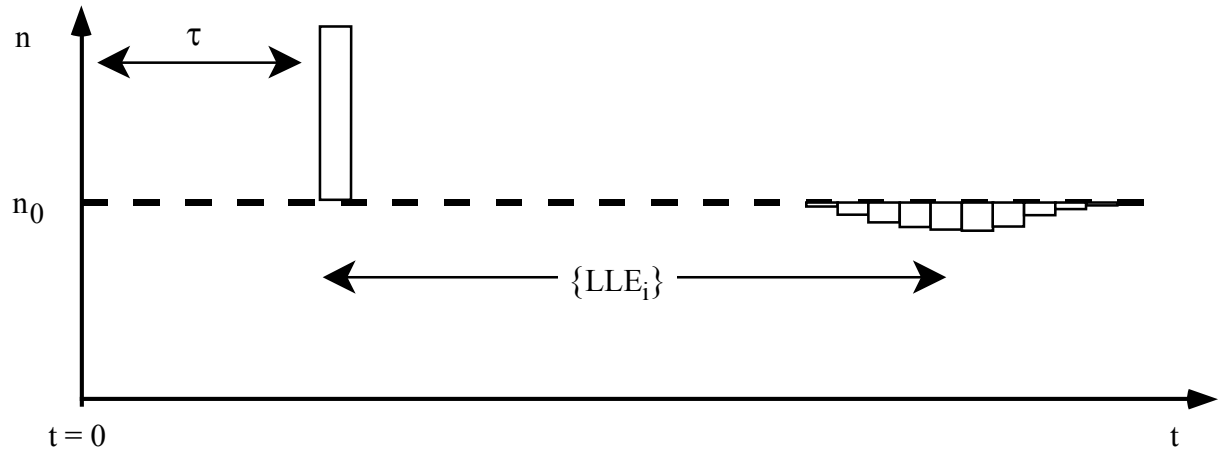
exposure pulse at t=0

1) simplest model: all individuals who are affected **die between τ (delay time) and τ+Δt** after pulse, and **lose LLE**

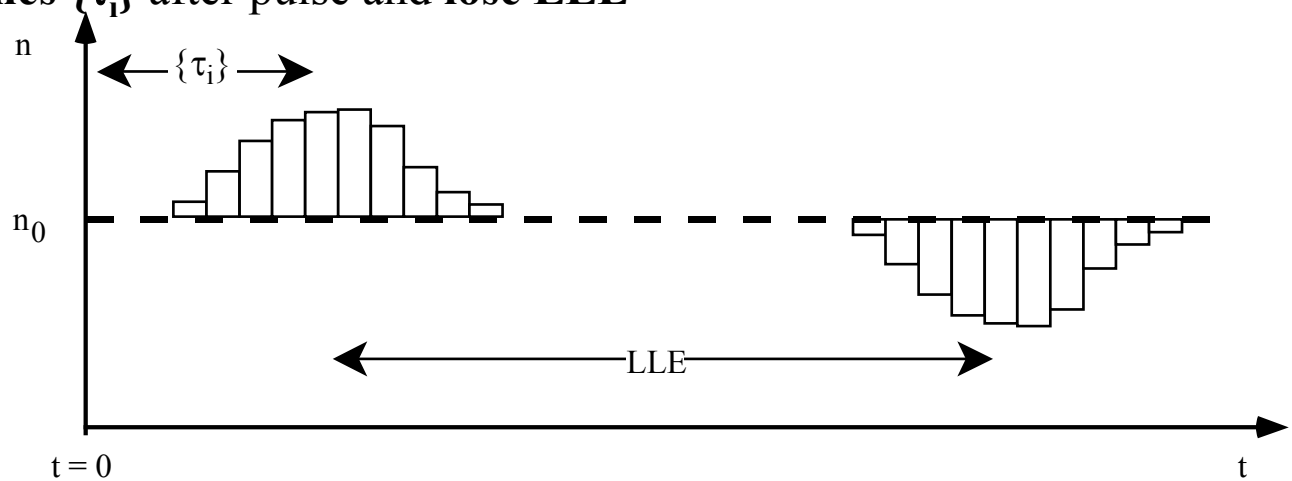


What is observable? Cont'd

2) generalization: all individuals who are affected **die between τ and $\tau+\Delta t$** after pulse and suffer a **range of losses $\{LLE_i\}$**



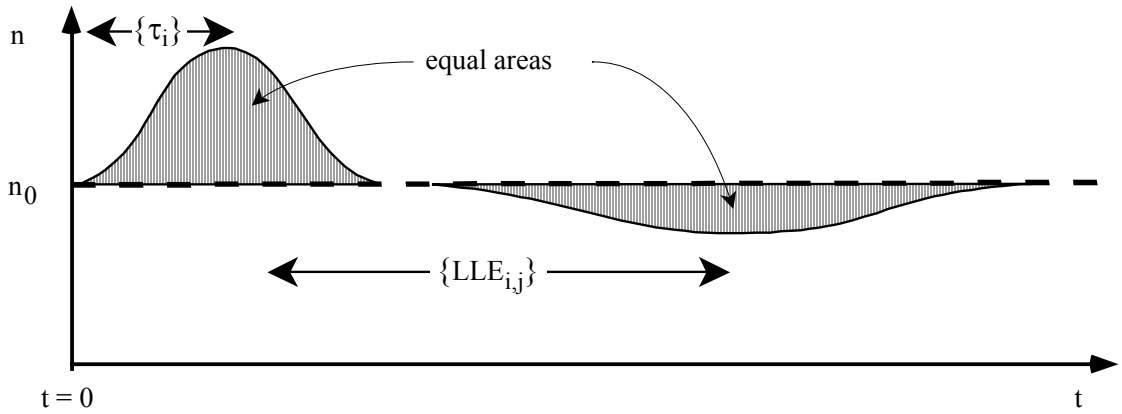
3) generalization: all individuals who are affected **die over a range of delay times $\{\tau_i\}$** after pulse and **lose LLE**



What is observable? Cont'd

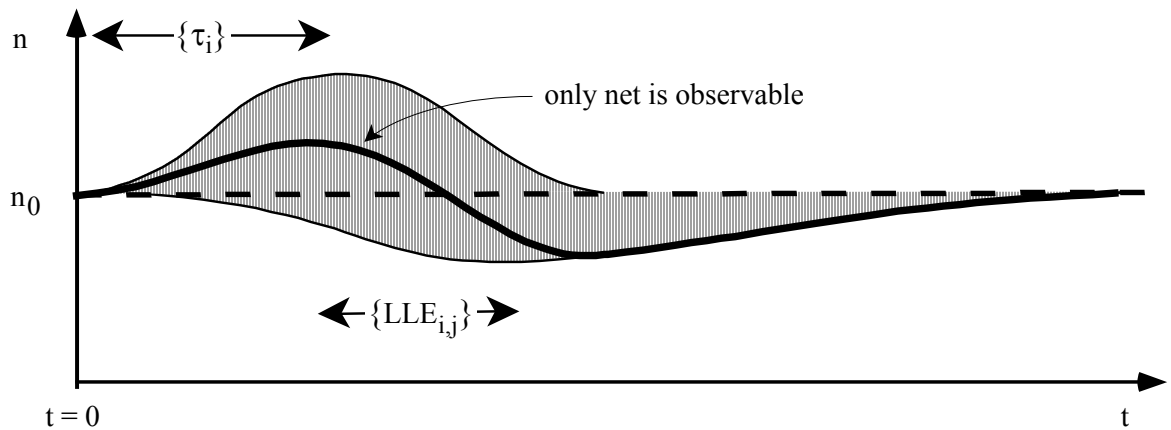
4) generalization: all individuals who are affected die over **a range of delay times $\{\tau_i\}$** after pulse and suffer a **range of losses $\{LLE_{i,j}\}$**

a) depletion is distinct ($Min[\Delta L_{i,j}] > Max[\tau_i] - Min[\tau_i]$)



⇒ total number of deaths is observable

b) depletion overlaps (case of **air pollution**)



⇒ total number of deaths is *not* observable even by long term studies (long observation window)

What is observable? Cont'd

Short term (time series)

Large variations of concentration c, for each city

Depletion becomes background (approx. uniform)

⇒ **Observe** (mostly) **initial deaths**

no information on loss of life expectancy LLE/death

Long term (cohort studies)

no variations of concentration c, for each city

(variations between cities)

⇒ **Observe net deaths = initial - depletion**

information on LLE,

but not "attributable" number of deaths

Because the studies yield the same result for a population where everybody suffers the same LLE and for a population where only some are affected and lose a large LLE.

Calculation of life expectancy

There are several different definitions of mortality rates:

(i) **Time series** determine the relative risk R for the **daily mortality m** [deaths/day] of the total population, or in some case for a subgroup e.g. people over 65; if the rate at a reference concentration c_0 is m_0, at concentration c it is $m = R\ m_0$.

Typical value **$R - 1 = 0.06\%$ per $\mu g/m^3$ of PM_{10}.**

(ii) **Cohort studies** determine the relative risk R for the **age-specific mortality $\mu(x)$** [probability/yr] which is defined as the probability for an average person of age x to die during the coming year; if the rate at a reference concentration c_0 is μ_0, at concentration c it is $\mu = R\ \mu_0$.

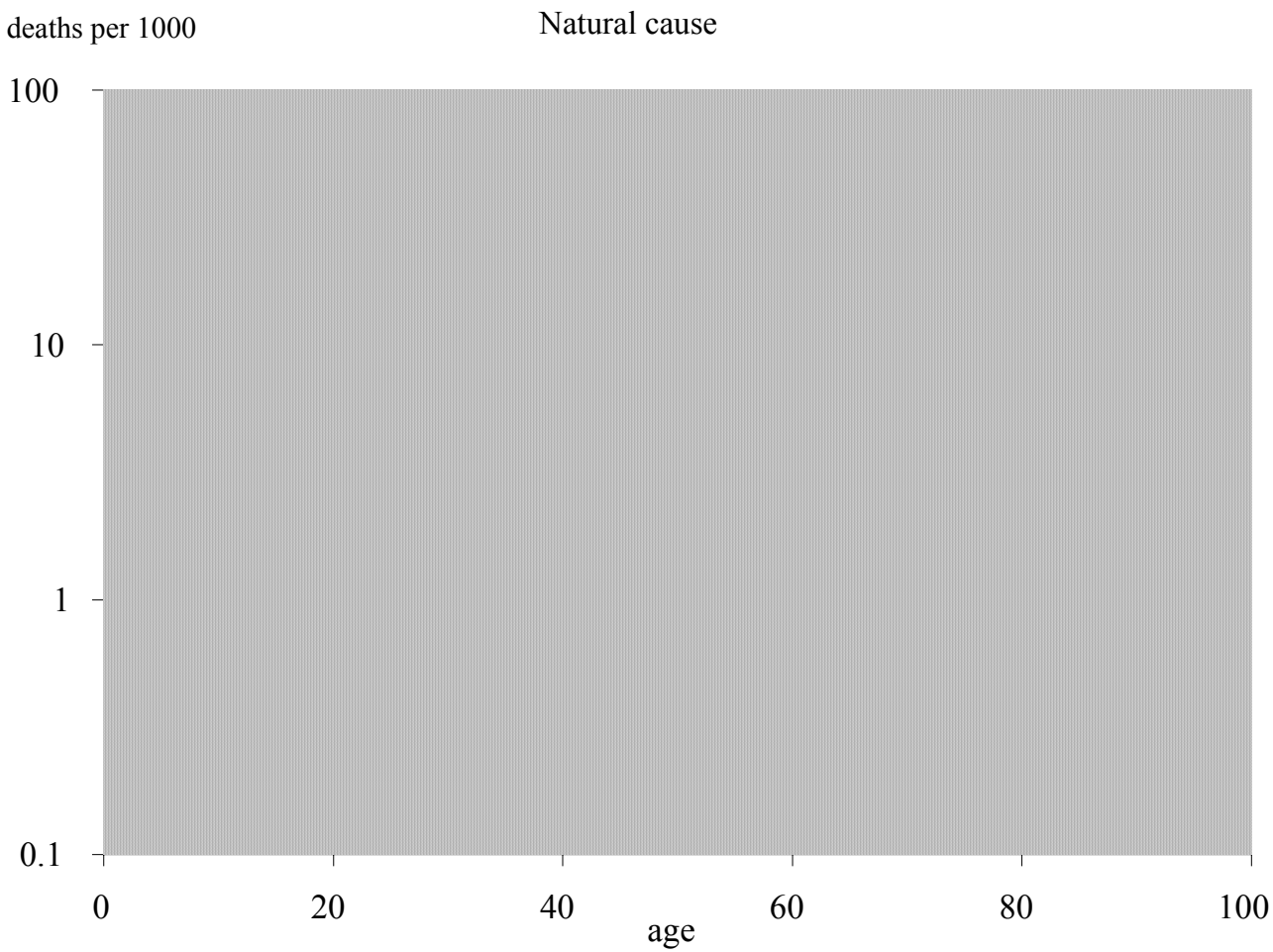
Typical value $R = 0.6\%$ per $\mu g/m^3$ of $PM_{2.5}$,

or **$R - 1 = 0.36\%$ per $\mu g/m^3$ of PM_{10}**, for typical ratio $PM_{2.5}/PM_{10} = 0.6$.

The values of R are very different because they measure different effects: acute only for time series, total for cohort studies.

Calculation of life expectancy, cont'd

Data for age-specific mortality, USA



Calculation of life expectancy, cont'd

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

$dS_\mu(x,x') = -S_\mu(x,x')\,\mu(x')\,dx'$ with boundary condition $S_\mu(x,x) = 1$. Solution

$$S_\mu(x,x') = \exp\left[-\int_x^{x'} \mu(x'')dx''\right]$$

The probability distribution for a member of the age x cohort to survive to and die at age x' is $p(x,x') = S_\mu(x,x')\,\mu(x')$,

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

The difference between the expected age of death and the starting age x is the **remaining life expectancy L(x)** of this cohort

$$L(x) = \int_x^\infty x' S_\mu(x,x')\mu(x')dx' - x$$

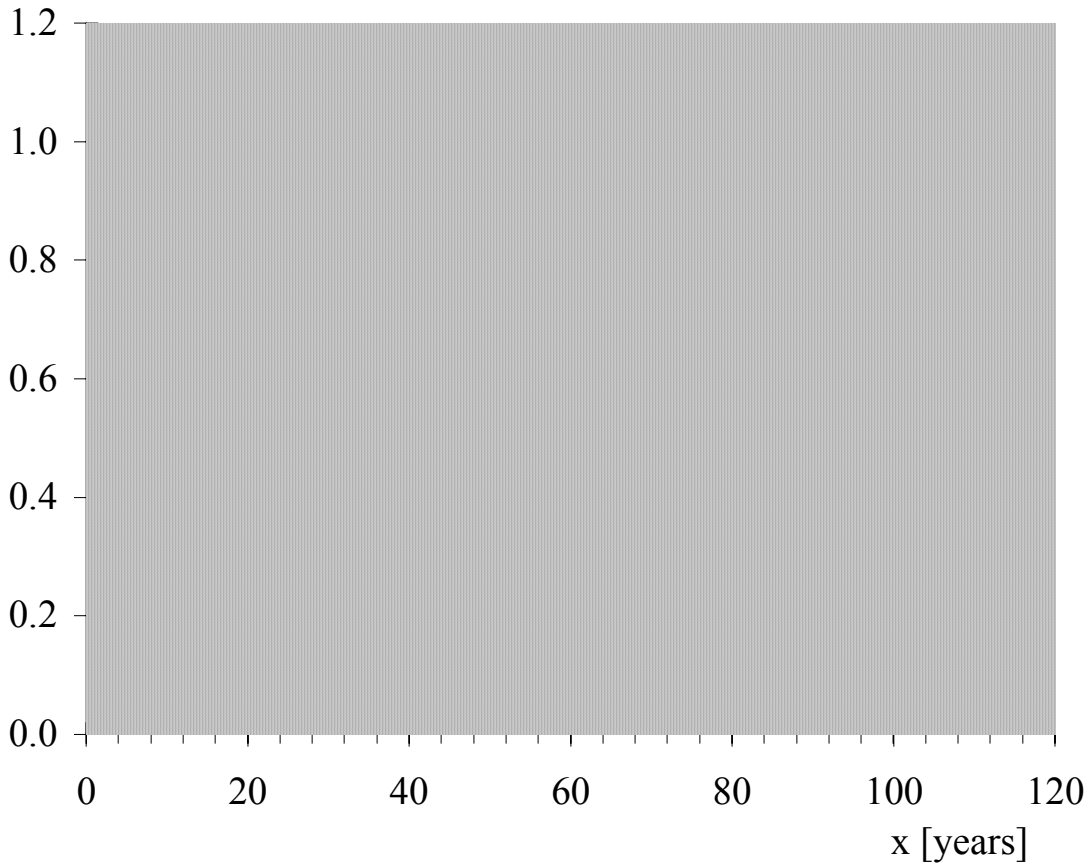
For practical calculations integrate by parts

$$L(x) = \int_x^\infty S_\mu(x,x')dx'$$

and approximate by annual values from **life tables**.

Calculation of life expectancy, cont'd

Survival function S(0,x) for surviving to age x.
Solid line = $S_{\mu 0}(0,x)$ for $\mu_0(t)$ with life expectancy T = 75 yr;
dotted line = $S_{\mu}(0,x)$ for $\mu_0(t) = 1.17\ \mu_0(t)$ with T = 73.4 yr.



Calculation of life expectancy, cont'd

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

$$LLE(x) = \int_x^\infty [S_{\mu 0}(x,x') - S_\mu(x,x')]dx'$$

where $S_{\mu 0}(x,x')$ is the survival curve for the baseline mortality $\mu_0(x)$.

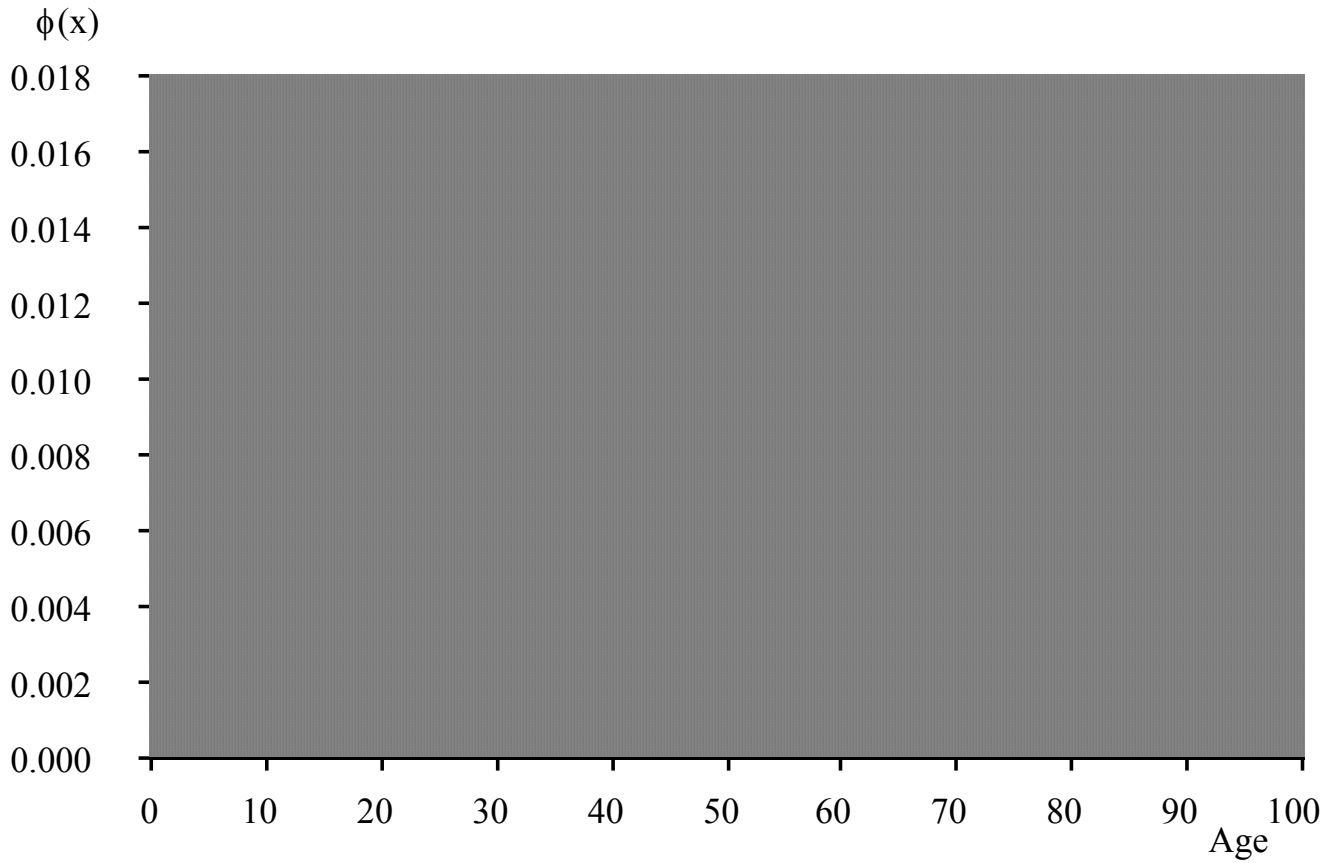
The impact on the entire population is obtained by summing $LLE(x)$ over all affected cohorts, weighted by the age distribution $\Phi(x)$

$$LLE_{tot} = \int_0^\infty LLE(x)\Phi(x)dx$$

In practice for adult mortality the lower limit is replaced by 30 because the cohort studies have considered only people over 30.

Calculation of life expectancy, cont'd

The age distribution $\phi(x)$ for EU15 for 1997, USA for 1996, and a stationary population with total mortality of EU15.



Results for change in life expectancy LE

Gain of life expectancy LE
(population average, per person)
for reduction of PM_{10} concentration by 15 $\mu g/m^3$

calculated by Rabl, *J Air&Waste Management Assoc*. Vol.53(1), 41-50 (2003), on the basis of the indicated references.

Type of study	**Gain of LE**	**Reference**
Total mortality, adults	140 days	Pope et al [2002]
Time series, adults	1.3 days *(if 6 months/death)*	Samet et al [2000a and b], Katsouyanni et al [1997], Levy et al [2000]
Total mortality, infants (<12 months)	≤8 days	Woodruff et al [1997], Bobak & Leon [1999]

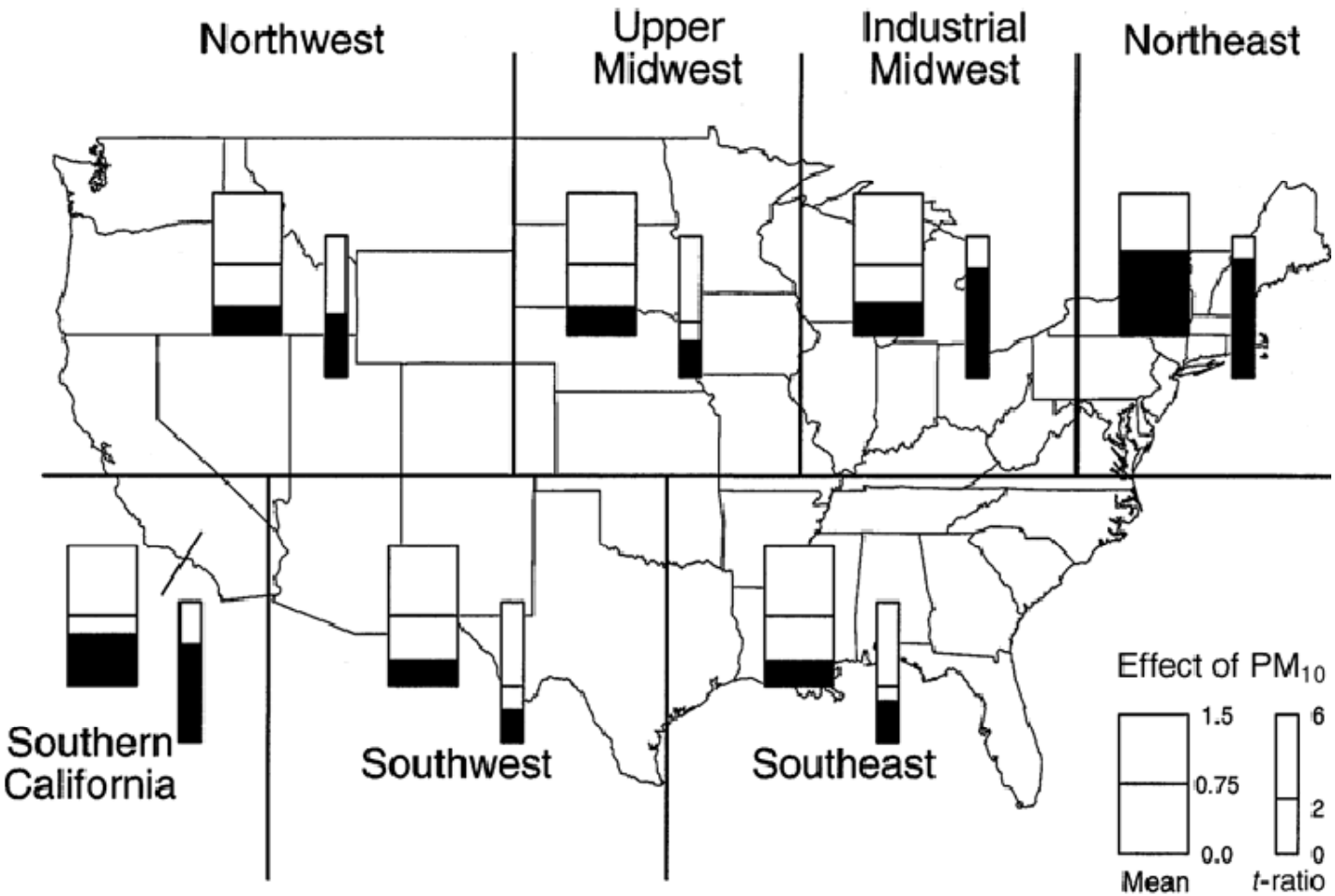
Results for other countries

Coefficients α and β of fit **$\mu(x) = \alpha + \beta x$**, and **LLE from chronic mortality** for several populations. LLE in years lost per person for an exposure to 1 μg/m^3 of PM_{10} during 1 year of exposure, calculated with the age distribution of each population.

Population	α	β	**LLE**
	[per yr^2]	[per yr]	**[yr_{lost}/(person·yr_{exp}·μg/m^3)]**
USA, natural causes, m+f	5.38E-05	8.78E-02	**2.69E-04**
USA, natural causes, m	7.76E-05	8.59E-02	**2.73E-04**
USA, natural causes, f	3.19E-05	9.20E-02	**2.56E-04**
EU15, all causes, m+f	3.70E-05	9.24E-02	**2.56E-04**
Sweden, natural causes, m+f	9.67E-06	1.10E-01	**2.25E-04**
France, all causes, m+f	6.66E-05	8.50E-02	**2.77E-04**
Russia, all causes, m+f	3.96E-04	6.78E-02	**3.59E-04**
China, all causes, m+f	5.89E-05	9.15E-02	**2.04E-04**

Questions about relative toxicity of PM components

Regional variation of acute mortality due to PM_{10} in the USA.



Possible variations due to other effects

Variation of acute mortality due to PM_{10} in Europe [Katsouyanni 2001]. (in parentheses 95% CI).

	% increase per 10 $\mu g/m^3$
Average, Europe	0.60% (0.40-0.80%)
City with low average NO_2	0.19% (0.00-0.41%)
City with high average NO_2	0.80% (0.67-0.93%)
Cold climate	0.29% (0.16-0.42%)
Warm climate	0.82% (0.69-0.96%)
City with low standardized mortality rate	0.80% (0.65-0.95%)
City with high standardized mortality rate	0.43% (0.24-0.62%)

Morbidity

Impacts ("end points") for which there are CRFs

(i) Chronic impacts

CB = chronic bronchitis

(another impact is **reduced lung function**, but there is no monetary valuation).

(ii) Acute impacts

HA = hospital admission

LRS = lower respiratory symptoms

mRAD = minor restricted activity day

RAD = restricted activity day

URS = upper respiratory symptoms

WDL = work days lost

Some of these impacts have been identified for asthmatics (about 4 to 6% of total population in industrialized countries, incidence has been increasing in recent years)

Mortality,morbidity, gaps and double counting

Air pollution mortality is closely linked to **morbidity** and **quality of life**. An individual who dies prematurely due to air pollution loses not just a few months of poor health at the end of life, but suffers an **impairment of general health during the entire period of exposure** and this impairment can considerably affect the quality of life, especially for people who are old or already in poor health. For example, a young person with 4 l lung capacity can survive very well with a 0.5 l decrease but if the capacity is already as low as 1.5 l such a decrease causes serious problems.

Even though air pollution can affect everybody's health, the **effects are observable only among** the most vulnerable, typically the **very young, the old and the sick**. And of course, even the young will be old someday.

The identified CRFs, and the corresponding monetary values, are only **proxies for the real impact** of air pollution; it is not entirely clear to what extent they cover all the important impacts or involve double counting. This type of **uncertainty** involves subjective judgment rather than formal analysis.

Results for morbidity

CRFs for PM_{10} [Rabl 2001].

End point for PM_{10}	**$\gamma = \Delta R/\Delta c$ [%/(μg/m³)]**	**Incidence rate I_{ref}**	**s_{CR}**	**Comments**
CB adults	2.14	0.0036 cases/(pers·yr) in USA	7.65E-5 f_{pop} cases/(pers·yr·μg/m³)	f_{pop} = fraction of population over age 18
WDL	0.95	1.057 WDL/(emp·yr)	1.0E-2 f_{pop} WDL/(pers·yr·μg/m³)	γ and WDL include only respiratory and cardiovascular causes. f_{pop} = fraction of population employed
RAD adults	0.26	19 RAD/(pers·yr) in USA	5.00E-2 f_{pop} RAD/(pers·yr·μg/m³)	f_{pop} = adult population. Overlap with WDL
HA, cardiovascular	0.1	0.06 HA/(pers·yr)	6.0E-5 f_{pop} HA/(pers·yr·μg/m³)	f_{pop} = fraction of population above age 65
HA, respiratory	0.04	0.0071 HA/(pers·yr)	2.56E-6 HA/(pers·yr·μg/m³)	Original based on BS, here converted to PM_{10} by multiplying by 0.6

Results for morbidity, cont'd

CRFs for PM_{10}, cont'd [Rabl 2001].

End point for PM_{10}	$\gamma = \Delta R/\Delta c$ [%/(μg/m³)]	**Incidence rate I_{ref}**	**s_{CR}**	**Comments**
Bronchodilator usage, asthmatic adults	0.22	28 cases/(pers·yr)	0.06 f_{pop} cases/(pers·yr·μg/m³)	f_{pop} = fraction of population that is adult asthmatic
Bronchodilator usage, asthmatic children	0.23	34 cases/(pers·yr)	0.078 f_{pop} cases/(pers·yr·μg/m³)	f_{pop} = fraction of population that is asthmatic children
LRS, asthmatic adults	0.18	91 cases/(pers·yr)	0.163 f_{pop} cases/(pers·yr·μg/m³)	f_{pop} = fraction of population that is adult asthmatic
LRS, asthmatic children	0.33	30 cases/(pers·yr)	0.10 f_{pop} cases/(pers·yr·μg/m³)	f_{pop} = fraction of population that is asthmatic children

Results for morbidity, cont'd

CRFs for SO_2 [Rabl 2001].

End point for SO_2	$\gamma = \Delta R/\Delta c$ [%/(μg/m³)]	**Incidence rate I_{ref}**	s_{CR} and fraction f_{pop} of total population	**Comments**
Acute Mortality	0.046	for I_{ref} = 0.01 deaths/(pers·yr)	2.3E-6 yr_{lost}/(pers·yr·μg/m³)	Assuming 0.5 yr lost per death, a very uncertain number
HA, Respiratory	0.04	0.0071 HA/(pers·yr)	2.84E-6 HA/(pers·yr·μg/m³)	

For sulfates assume that the CRF's are equal to those for $PM_{2.5}$, which are 1.67 times those for PM_{10}. This is very **uncertain**.

For nitrates assume that the CRF's are equal to those for PM_{10}. This is very **uncertain**.

Results for morbidity, cont'd

CRFs for SO_2 [Rabl 2001].

End point for O_3	$\gamma = \Delta R/\Delta c$ [%/(µg/m³)]	**Incidence rate I_{ref}**	s_{CR} and fraction f_{pop} of total population	**Comments**
Acute Mortality	0.058	For I_{ref} = 0.01 deaths/(pers·yr), see Section 3.1.3	2.9E-6 yr_{lost}/(pers·yr·µg/m³)	Assuming 0.5 yr_{lost} per death, a very uncertain number
HA, Respiratory	0.06	0.0071 HA/(pers·yr)	4.26E-6 HA/(pers·yr·µg/m³)	
WDL	0.11	1.057 WDL/(emp·yr)	0.11E-2 f_{pop} WDL/(pers·yr·µg/m³)	γ and WDL include only respiratory and cardiovascular causes. f_{pop} = fraction of population employed. Association was not significant at 95% level
mRAD	0.13	7.8 mRAD/(pers·yr) USA	0.98E-2 f_{pop} mRAD/(pers·yr·µg/m³)	f_{pop} = adult population. Overlap with WDL

Glossary and conversion factors

1 ppb O_3 = 1.997 μg/m^3 of O_3
1 ppb NO_2 = 1.913 μg/m^3 of NO_2
1 ppm CO = 1.165 mg/m^3 of CO
BS = black smoke
c = concentration
CB = chronic bronchitis
COPD = chronic obstructive pulmonary disease
CR function = concentration-response function (also known as exposure-response function)
EPA = Environmental Protection Agency of USA
f_{pop} = fraction of the population affected by the end point in question.
HA = hospital admission
I_{ref} = baseline or reference level of incidence of the end point in question.
LLE = loss of life expectancy
LRS = lower respiratory symptoms
mRAD = minor restricted activity day
NO_x = unspecified mixture of NO and NO_2
PM_d = particulate matter, with subscript d indicating that only particles with aerodynamic diameter below d, in μm, are included
R = relative risk
RAD = restricted activity day
s_{CR} = slope of CR function
URS = upper respiratory symptoms
VOC = volatile organic compounds
WDL = work day lost
YOLL = years of life lost

α = coefficient of Gompertz function for mortality
β = coefficient of Gompertz function for mortality
$\gamma = \ln(R)/\Delta c \approx \Delta R/\Delta c$
Δc = change in concentration.
ΔR = change in relative risk

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