Health Impacts of Pollutants

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Traditional Health Impact Assessments vs. the Objectives of ExternE

Traditional health impact assessments have estimated the **total damage due to exposures to current ambient concentrations**

⇒ Guidelines for policy makers on benefit of **general regulations** (e.g. ambient air quality standards)

For that purpose one can directly use ambient concentration data and CRFs (concentration-response functions) as determined by epidemiological studies, because these CRFs are based on the current ambient mix of pollutants.

But for optimal implementation of general regulations one needs to know **damage caused by specific pollutant emitted by specific source** ***(most probable estimate, not "conservative" bound!!!)***.

That is the **objective of ExternE**.

⇒ *One needs CRFs for the emitted pollutants (and their secondaries)* ≠ *ambient pollutant mix*

Air Pollutants and their effects on health

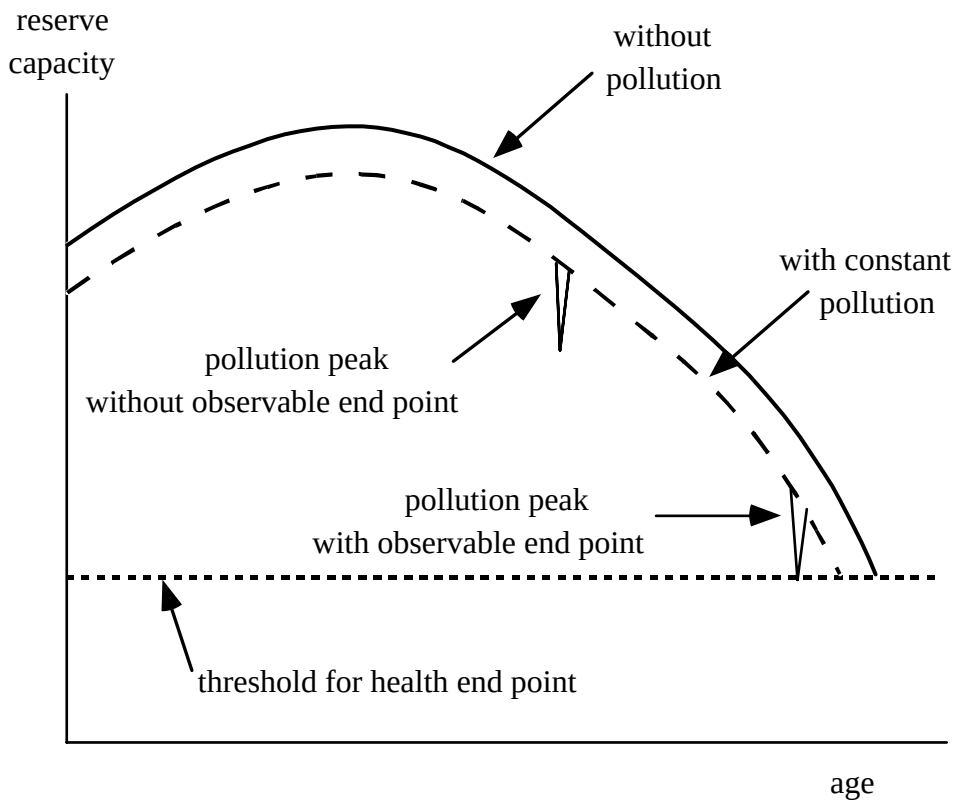
Primary Pollutant	Secondary Pollutant	Impacts
particles (BS, PM_{10}, $PM_{2.5}$)		**mortality** **morbidity** respiratory and cardio-vascular, asthma, reduction of lung capacity *(hospitalization, consultation of doctor, sick leave, restricted activity)* cancers
SO_2		**mortality** **morbidity** respiratory 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)

Cardio-pulmonary effects of air pollution

Healthy individuals have sufficient reserve capacity not to notice effects of pollution, but the **effects become observable at times of low reserve** (during extreme physical stress, severe illness, or near end of life)



Pollution reduces reserve capacity

⇒ Mortality impact is not the loss of a few months of misery at the end but the shrinking of the entire quality of life curve ("**accelerated aging**")

In large population there are always some individuals with very low reserve ⇒ impacts observable

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 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****)*

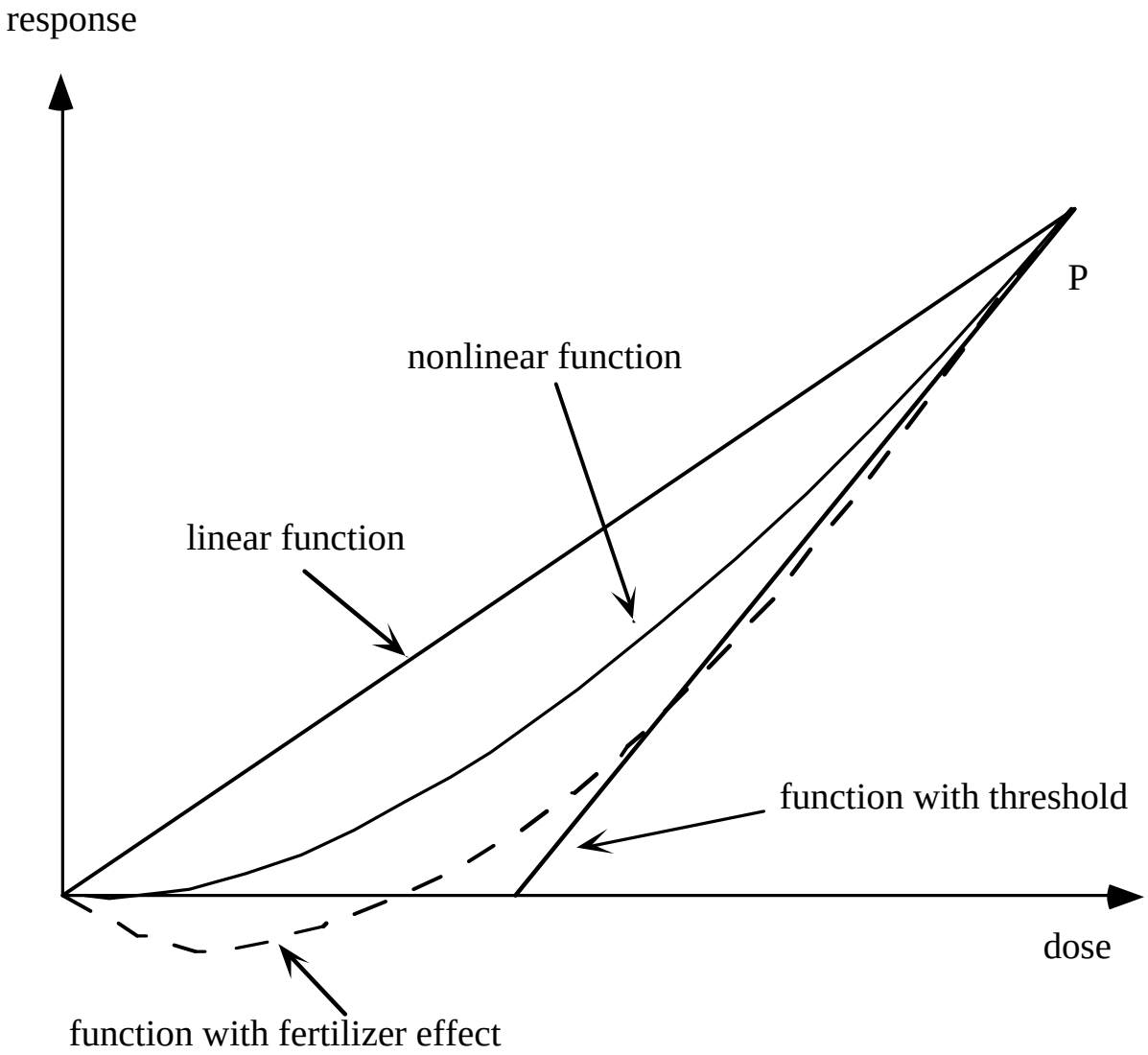
Crucial for calculating impacts of a pollutant.

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) Difference between country of epidemiological study and country where impacts are calculated? **ExternE assumes same DRFs for health in all countries** (no better information available)

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 → health
threshold: ozone → 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, for IQ decrement due to Pb, and for substances that initiate cancers (also for radioactivity).

Slow convergence towards a consensus :
"air pollution is 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 50%

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?

Which Pollutants, Which Impacts?

The current approach of ExternE:

Consider **PM and O3 as independent pollutants** (i.e. their impacts are additive), and **apply the CRFs of PM also to secondary particles**, assuming that sulfates are as toxic as PM10, and nitrates half as toxic as PM10

*In addition a few CRFs for **SO2** and **CO** are also used, but their contribution to the total cost is negligible.*

Validation of the approach of ExternE:

Workshops held 28 May and 1 Sept. 2003

with 10 internationally renowned **epidemiologists and toxicologists** (USA and EU)

They were asked to provide comments and recommendations

a) On the general approach (which pollutants?)

b) Specific CRFs (concentration-response functions)

Basic conclusion: the approach of ExternE is OK

Results for Mortality

Mortality ~ 2/3 of the total damage cost
(apart from global warming)

Gain of life expectancy LE
(population average, per person)
for reduction of PM_{10} concentration by 15 μg/m³
(= reasonable policy goal for coming decades)
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 *(cohort studies)*, adults	140 days	Pope et al [2002]
Acute mortality *(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]

Morbidity

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

(i) Chronic impacts

CB = chronic bronchitis **(approx. 23%** of total damage cost**)**

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

(ii) Acute impacts (approx. 10% of total damage cost)

HA = hospital admission

LRS = lower respiratory symptoms

mRAD = minor restricted activity day

RAD = restricted activity day **(approx. 7%** of total damage cost**)**

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)

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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