CURRENT POSITION ON HEALTH IMPACTS

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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 current ambient concentrations.

But for optimal implementation of general regulations policy makers need to know **how much damage is 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

Epidemiology and HIA (Health Impact Assessment)

HIA uses epidemiology (and also toxicology) but has a **different** emphasis (attitude) **from epidemiology**

Epidemiology: Cautious regarding conclusions

- 'beyond reasonable doubt'

HIA: Need to make judgements more decisively in the face of uncertainties

- If you don't quantify, impact gets ignored
- Default quantification is zero
- Can we do better than zero? – often, yes
- Caution under-estimates – and so is anti-precautionary; but
- Need to assess and express uncertainty

ExternE Health evaluations: Changes over time

1995 : Vol 2 Methodology: detailed evaluation

1998: Vol 7 Methodology update: another detailed evaluation. Still generally gives good answers but

- •Concentration-response functions (CRFs) are old – bad for credibility with stakeholders
- •Weak on background rates (e.g. for hospital admissions) which vary across Europe

1998-03: Various small improvements (no ExternE research on CRFs)

- •to life table methods for assessing mortality from cohort studies
- •to some specific functions
- •not strongly co-ordinated

Other parallel initiatives

WHO (World Health Organization)

- Fintan + Rudi Torfs (VITO) met with Michal Krzyzanowski of WHO in Summer 2000
- November 2000: **WHO** workshop on Health Impact Assessment (HIA) for air pollution
- **Broadly supported ExternE work**, including life table work

AIRNET: a network of air pollution scientists and stakeholders in Europe

- Several working groups
- Fintan very active on HIA WG
- Ari + Rudi on Science-Policy Interface (SPI)

US EPA evaluations of benefits of US Clean Air Act

- Overview Science Advisory Board; Fintan on sub-group dealing with estimating benefits to health – Chair Bart Ostro

Update of CRFs for IAEA by Ari (2002)

The problem of pollution mixtures

1. Principal CRFs are for particles (PM), then for ozone (acute effects only) – US EPA agrees
2. These are additive to one another – US EPA agrees
3. Limited extra CRFs for SO2, NO2 and CO – US EPA agrees
   - Functions for **NO2** are **probably not causal in NO2 –** WHO agrees. NO2 probably a surrogate for mixture as a whole. May be a surrogate for *particle number* especially.
   - New view for ExternE: functions in CO may also be a surrogate for traffic pollution
4. For PM ExternE differentiates primary and secondary (nitrates & sulfates) particles
   - Growing evidence in favour of doing this
   - But US EPA thinks evidence not strong enough to use in quantifying
   - In general, great reluctance to apportion effects to individual pollutants or to particular aspects of particles

Pollution mixtures, causality, thresholds

Widespread acceptance that air pollution is **causally associated** with:

- Some aspects of PM
- Ozone (can be additive to PM effects; fewer endpoints)
- Other gases: ??? In terms of causality?? (There are CRFs).
  - SO2? Industrial pollution?
  - NO2 and CO as tracers of (all?) traffic pollution? As surrogates for particle number?

Thresholds:

- ExternE has had a '**No threshold'** position **for all pollutants**, including ozone
- Impossible to prove or disprove
- WHO expert groups have reviewed the evidence, as part of work for the Clean Air for Europe (CAFE) programme
- **Evidence supports no threshold**, ***including for ozone***

Mortality

Approx. 90% of total damage cost of PM, SO2 and NOx

Two types of epidemiological studies:

1) **Time series** = correlations between pollution and number of deaths during the following days (<6 days)

⇒ only acute impacts ("**acute mortality**")

2) **Cohort studies** = comparison of populations over long term (> 5 years) in different cities

⇒total impacts due to chronic exposure ("**chronic mortality**")

The most important chronic mortality study: Pope et al [2000], based on approx. half a million individuals in the USA during 18 years.

Effects associated with PM (PM2.5 for Pope et al)

Results very widely (not universally) accepted as causal

Mortality, cont'd

1) **Time series**

Measure increase in number of deaths, without any information on the loss of life expectancy (LE) per death (**ExternE** has assumed **6 months/death** for valuation)

2) **Cohort studies**

Results include both acute and delayed deaths ⇒ **total mortality**

Reported as increase in age-specific mortality rate, which allows calculation of **population-average LE**, but not of number of deaths due to pollution

Linear without threshold

Number of deaths due chronic mortality has been reported by various teams, especially in the US, but it is not meaningful:

The total number of air pollution deaths cannot be determined at present.

Mortality: infants, O3

Infant mortality

- New information over past 5 years
- Increasing evidence that air pollution does affect mortality of the very young – studies in Europe and USA
- Can be quantified [Rabl 2003]

Ozone and 'acute' mortality

- This relationship is being questioned because of seasonal differences
- US EPA is reviewing the evidence

Mortality: time-lag between exposure and impact; mechanisms

- Cohort studies compare cities ('metropolitan areas' in Pope et al)

- Uninformative about time lag between exposure and increased risk of mortality *within* populations (cities)

- Possible to think mechanistically, via cause of death

    - Some more-or-less immediate changes – the 'acute' exposure mortality that the time series capture
    - Some may be a contribution to initiation – e.g. chronic bronchitis; lung cancer
    - Some (?much) may be promotion/ acceleration of multi-causal cardio-respiratory disease – impact of exposure in 'recent' years

Morbidity

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

(i) Chronic impacts

CB = chronic bronchitis

another impact is **reduced lung function** $\Rightarrow$ reduces life expectancy and quality of life, but there is no direct 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 separately for asthmatics (about 4 to 6% of total population in industrialized countries, incidence has been increasing in recent years)

Morbidity: baseline rates

Necessary to have them, *or to estimate them*, for the population(s) exposed. Usually get much less attention than CRFs; but *can* be very influential on final answers

- 'Routine' data vary in relevance/ availability/ reliability; from a modelling perspective: they are one kind of estimate
- Other sources are specific studies – not necessarily air pollution studies – we can get CRF and baseline separately
- Issues in matching definitions, e.g. 'bronchitis'
- Sometimes use baselines from where CRF studies were done – definitions match, but results transferable??
- Some exploration of the issues already – APHEIS, UK, other
- Pilot study systematic work @ RIVM (IVM); WHO interested
- Need to find out/ review…
- We will make progress but will not 'solve' this in timescale of CAFE CBA

Workshop with Epidemiologists and Toxicologists to Evaluate ExternE Methodology

One of the work packages of the DIEM project, Nov.2002-Apr.2004
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)

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 like PM2..5 and nitrates like PM10 (together with the assumption that PM2.5 are about 1.7 times as toxic as PM10)
Only particulate nitrates are taken into account, not HNO3.
*In addition a few CRFs for **SO2** and **CO** are also used, but their contribution to the total cost is negligible.*

New Approach?

Incremental impact ΔI, for a particular end point, is a linear combination of the contributions of the individual pollutants, each with CRF slope s_i and concentration increment Δc_i

$$\Delta I = \sum s_i \, \Delta c_i$$

ExternE until now:

$$\Delta I = s_{PM} \, \Delta c_{PMp} + s_{PM2.5} \, \Delta c_{sulf} + s_{PM10} \, \Delta c_{nitr} + s_{O3} \, \Delta c_{O3} + s_{SO2} \, \Delta c_{SO2} + s_{CO} \, \Delta c_{CO}$$

with $s_{PM10}/s_{PM2.5} = 0.6$

Proposed generalization

$$\Delta I = s_{PM} \, f_{PM} \, \Delta c_{PMp} + s_{PM10} \, (f_{sulf} \, \Delta c_{sulf} + f_{nitr,P} \, \Delta c_{nitr} + f_{HNO3.P} \, \Delta c_{HNO3}) + s_{O3} \, (f_{nitr,O} \, \Delta c_{nitr} + f_{HNO3,O} \, \Delta c_{HNO3} + f_{O3} \, \Delta c_{O3}) + s_{SO2} \, f_{SO2} \, \Delta c_{SO2} + s_{NO2} \, f_{NO2} \, \Delta c_{NO2} + s_{CO} \, f_{CO} \, \Delta c_{CO}$$

with coefficients f chosen by consensus of experts.

(the subscripts O and P for nitrates distinguish different effects on the body: “like oxidants” and “like particles”)

*The experts of the DIEM workshop **agreed with the general approach** but refused to specify coefficients. They will hold a symposium on this issue at the next congress of the International Soc. of Environmental Epidemiologists, Aug.2004.*

Next steps/ further updates

Two major initiatives will allow further updating:

1. **CAFE Cost-Benefit Analysis**
   - High profile, wide stakeholder involvement
   - Methodology to be finished by September 2004
2. **EU 6th Framework NEEDS project**
   - Longer time-frame but more resources
   - Should enable a comprehensive review of C-R functions