

CURRENT POSITION ON HEALTH IMPACTS

Fintan Hurley and Ari Rabl

On behalf of the ExternE/ DIEM team

Brussels, 10 March 2004



Institute of Occupational Medicine

***INSTITUTE OF OCCUPATIONAL MEDICINE
EDINBURGH, EH8 9SU, UK***

Health Impact Assessment (HIA) in ExternE

ExternE: Health effects of energy production and transport. Main issues are:

- Outdoor (ambient) air pollution
- Noise (traffic)

Traffic and energy production (electricity; domestic fuel burning) are also two of the main sources of outdoor air pollution

HIA for is better developed for outdoor air pollution than for any other health application that we know

- Impact pathways are relatively simple
- There has been a huge amount of research on air pollution and health in last 15 years – ongoing
- HIA for air pollution has been a focus of methodological development

Impact pathways for outdoor air pollution and health



HIA and the specific objectives of ExternE

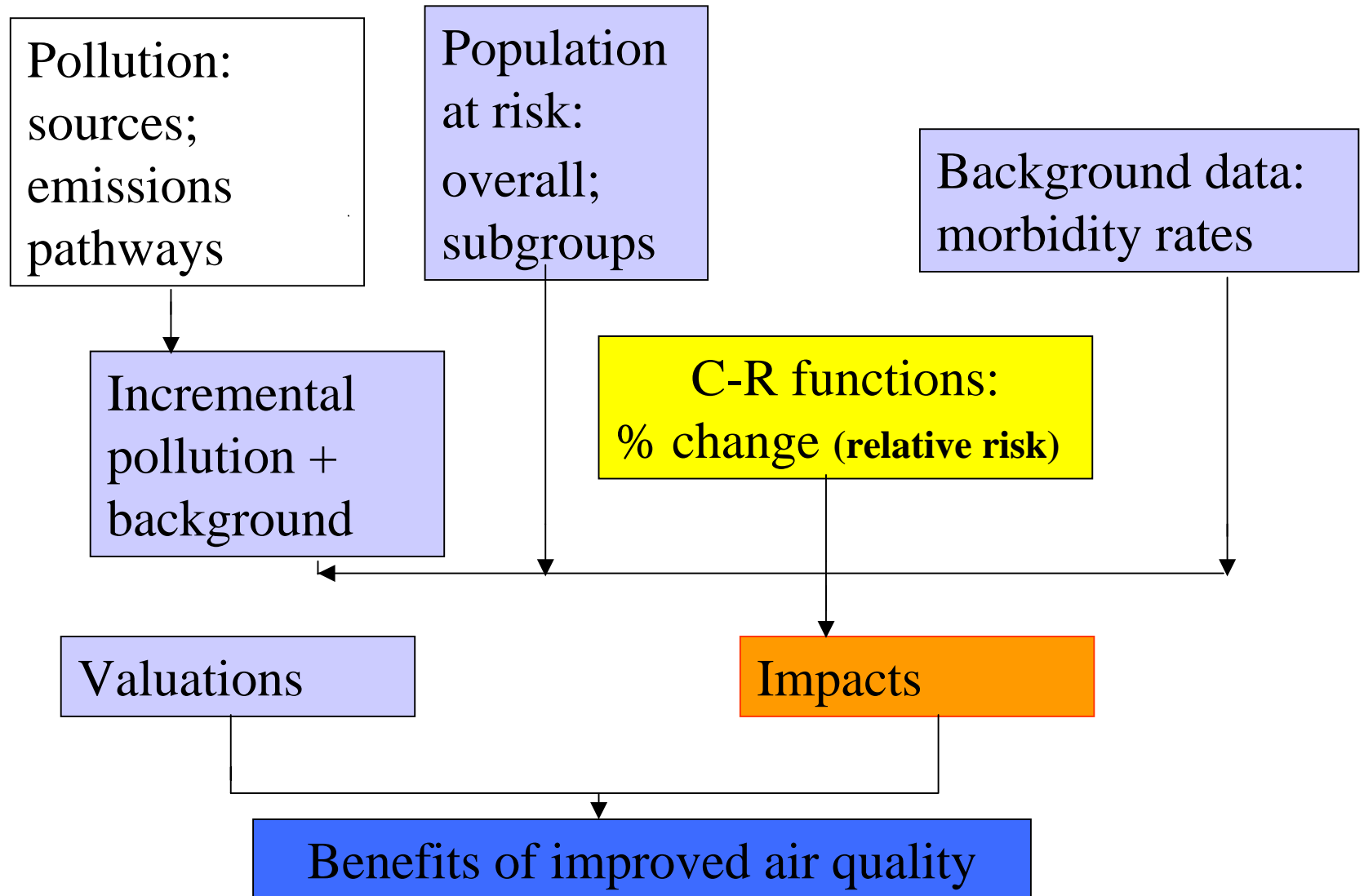
Many important examples of HIA estimate the effects of **general (urban) pollution mixtures, regardless of source**

- **Useful for policy makers on benefit of general regulations** (e.g. ambient air quality standards or National Emission Ceilings)
- Examples are Benefits of US Clean Air Act; UK National Air Quality Strategy; EU Greensense studies;
- Can directly use ambient concentration data and Concentration-Response Functions (C-R functions or CRFs) from epidemiological studies – these CRFs are based on current ambient (urban) mixtures and concentrations.

ExternE is different. For *optimal implementation of general regulations*, policy makers need to know how much damage is caused by **specific pollutants emitted by specific sources**.

- E.g. specific transport policies; what fuels to use for power generation etc.
- Needs CRFs for the specific emitted pollutants (+ secondaries) not just the ambient pollutant mix;
- Judgments about what pollutants are causal are more important; but impact pathways are the same

Components of HIA for air pollution



Major ExternE HIA evaluations

1995 : Vol. 2 Methodology: Comprehensive evaluation

1998: Vol. 7 Methodology update

- Another comprehensive evaluation

1998-03: Various further improvements

No substantial ExternE research on C-R functions

Progress on associated projects; e.g.

- ExternE (2000)
- IOM + AEA for UK Dept of Health (2000); Scotland (2003)
- Ari (2002) for IAEA
- Various ExternE *ad hoc* improvements – e.g. in DIEM, NewExt etc.

2004-05: Opportunity for further improvements

- HIA/ CBA for the EU CAFE programme – use of ExternE for CAFE CBA is a very major recognition of ExternE
- New FP6 projects – especially NEEDS – another comprehensive evaluation planned

ExternE is closely linked to international work on HIA and air pollution

ExternE judgments about causality and choice of CRFs uses reviews by other expert groups; we have close links with:

- World Health Organisation (WHO) and its reviews
- Other expert groups of air pollution researchers; e.g. COMEAP and EPAQS in the UK

ExternE also has close contacts with other international HIA teams

- FH, Ari Rabl, Rudi Torfs, Brian Miller, Mike Holland....
- Bart Ostro/ US EPA: Benefits of the US Clean Air Act
- WHO on Long-Range Trans Boundary Air Pollution
- Nino Kuenzli and colleagues in Europe; Sylvia Medina and APHEIS
- AIRNET, especially its HIA Working Group
- Special experts' workshop under DIEM – organised by Ari Rabl



Status of HIA work in ExternE

ExternE strategic decisions now strongly supported by the wider community. On some key issues, ExternE has led the way:

- Mortality from long-term exposure to air pollution
 - Early inclusion of this pathway
 - Using life table methods to implement it
 - Developing methods for this (Brian Miller; Ari Rabl)
- Use of no-threshold assumptions for ozone
- Attribution of different effects to different kinds of particles

Specific ExternE C-R functions are 'old' and not based on meta-analyses;

- **They don't 'look good'**
- **But mostly they still give good answers**
- **Most important ones have been updated**

General difficult with having reliable baseline rates for morbidity

ExternE attitude to HIA and quantification

Purpose of HIA: Make best estimate of impacts

- If you quantify only what is certain – answers too low
- If you always use ‘worst-case’ – answers too high

Requires judgments in the face of uncertainties

- If you don’t quantify, impact gets ignored
- Default quantification is zero – can we do better than zero? – usually, yes
- Our policy: Make best estimate (‘on the balance of probabilities’); assess and express uncertainty
- Epidemiology and toxicology are more cautious regarding conclusions: ‘Beyond reasonable doubt’

HIA needs to work ahead of consensus; anticipate trends



Mortality: Two kinds of studies give different kinds of information

Time series studies measure the more-or-less immediate effects of acute exposure (daily variations in pollution)

- Give results in terms of risk of ‘extra’ numbers of daily deaths – vulnerable people
- In HIA, this gives ‘extra’ daily or annual ‘earlier’ deaths
- No direct information on the loss of life expectancy (LE) per death
- LE believed to be short; ExternE has assumed **6 months/death** for valuation

Cohort studies measure effects of both acute and long-term exposure

- Maybe doesn’t capture

In energy and transport applications, mortality is approx. 90% of total damage cost of PM, SO₂ and NO_x. Mortality measured by cohort studies is by far the single most important impact of air pollution.

- Now widely accepted (WHO, US EPA) that HIA *must* include mortality from cohort studies
- ExternE (1995) had this as a sensitivity analysis; ExternE (1998) as a main pathway
- The main methodological development in using life tables was ExternE

Cohort studies ('chronic mortality')

Effects linked especially to PM, represented as PM_{2.5}

- PM_{2.5} may be carrying the impact of the mixture as a whole
- No strong evidence of coarse particles effect
- Pope et al. (2002) showed associations with SO₂ also; probably not causal; US EPA will not use these to quantify US cohort studies

Cohort results in terms of change in age-specific mortality rate;

Allows calculation of **population-average life expectancy (years-of-life-lost, or gained)**, but not of number of deaths due to pollution

- WHO, US EPA now support use of life-table methods
- Number of deaths due chronic mortality has been reported by various teams:
- Simple methods that use 'extra deaths' are not correct
- More work is needed to see if they are too wrong to use, or if 'extra deaths' can be derived in other ways
- The total number of air pollution deaths cannot be determined reliably at present.

Quantifying 'chronic' mortality in adults

What C-R functions (CRF) to use?

- Best to use Pope et al, 2002 (updated ACS study) – WHO, US EPA position also
- Sensitivity from original ACS and original 6-cities: use estimates from Krewski et al., 2000 – US EPA

Do time series may have some additional information?

- Yes, some; but do not add time series for particles to cohort results
- US EPA does not add time series for ozone or NO₂ or SO₂ either

What is time lag between change in exposure and changes in death rates?

- Cohort studies do not tell us but we can make informed judgment and can be modelled
- Will vary by cause of death
- Part of the effect is immediate

Links with valuation are very important

- Discount rates
- Value Statistical Life or Life-Year?



Some other key issues

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

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 generally supports no threshold, *including for ozone*

Ozone and ‘acute’ mortality

- This relationship is being questioned because of seasonal differences
- US EPA is reviewing the evidence; meantime, I (FH) would use

Effects of pollution mixtures: attributing to particular pollutants

1. Principal CRFs are for particles (PM), then for ozone (acute effects only) – this is US EPA position also
2. Impacts in PM and in O₃ are ‘independent’ – can be added – this is US EPA position also
3. Limited extra CRFs for SO₂, NO₂ and CO – US EPA agrees
 - NO₂ has some direct effects but C-R functions for NO₂ are probably a surrogate for mixture as a whole; for *particle number* especially
 - CO may also be a surrogate for traffic pollution
 - SO₂ unclear but not such a big problem (important for some policies)
4. Also, effects of particles vary by type of particle

Differential effects (per $\mu\text{g}/\text{m}^3$) of different kinds of particles – implications for HIA

Effects of particles vary by size, composition, source, surface properties:

- Main problems may lie with ‘small particles from combustion sources’
- Primary particles almost certainly worse than secondary (sulphates, nitrates)
- Coarse particles have some health effects – maybe ‘acute’ effects only

Approach of ExternE is to try to capture these differences. Others are slow to quantify by source – we think it is necessary. E.g. ExternE (1998):

- Assume that PM_{2.5} is about 1.7 times as toxic as PM₁₀ (per $\mu\text{g}/\text{m}^3$) (also used by Ostro in USA);
- Treat sulphates like PM_{2.5} (ExternE only)
- Treat nitrates like PM₁₀ (ExternE only)

ExternE is working with experts to distinguish better the effects of individual pollutants/ different kinds of particles

Generalize this approach?

Proposal (Ari): attribute total impact as a linear combination of various pollutants (gases and different kinds of particles)

Have coefficients chosen by experts – if possible, by consensus

DIEM project held two workshops in 2003

- 10 very well known epidemiologists + toxicologists (USA, EU)
 - Agreed with the general approach
 - Unwilling to specify coefficients

Next steps: Include Symposium on this issue at the next congress of the International Society of Environmental Epidemiology (ISEE) in Aug.2004.

Morbidity impacts (“end-points”) for effects of acute exposure (‘daily variations’)

Pathways for which there are C-R functions in PM or ozone

- HA = hospital admissions – Respiratory (RHA) or cardiovascular (CHA)
- ERVs = emergency room visits
- WDL = work days lost
- Visits to primary care doctors
- RAD = restricted activity day; mRAD = minor restricted activity day
- LRS = lower respiratory symptoms; URS = upper respiratory symptoms
- Temporary loss of lung function

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 – probably not pollution

Morbidity impacts (“end-points”) for effects of long-term exposure (‘chronic effects)

C-R functions apply to ambient PM only

- CB = chronic bronchitis
- [sustained reduced lung function \Rightarrow reduces life expectancy and quality of life, but there is no direct monetary valuation
- [lung cancer – no good CRF’s yet]
- impacts in infants – low birth-weight etc.

Morbidity: baseline rates

Necessary to have them, *or to estimate them*, for the population(s) exposed. Usually get much less attention than C-R functions; but *can* be v 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 C-R and baseline separately
- Issues in matching definitions, e.g. ‘bronchitis’
- Sometimes use baselines from where C-R 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

Health effects from noise exposure	Expectancy value ^{a)} (per 1000 adults exposed)
Myocard infarction (MI), fatal, Years of life lost (YOLL)	0.084 $L_{DEN} - 5.25$
Myocard infarction (non-fatal), days in hospital	0.504 $L_{DEN} - 31.5$
Myocard infarction (non-fatal), days absent from work	0.896 $L_{DEN} - 56$
Myocard infarction, expected cases of morbidity	0.028 $L_{DEN} - 1.75$
Angina pectoris, days in hospital	0.168 $L_{DEN} - 10.5$
Angina pectoris, days absent from work	0.684 $L_{DEN} - 42.75$
Angina pectoris, expected no. of morbidity days	0.240 $L_{DEN} - 15$
Hypertension, days in hospital	0.063 $L_{DEN} - 4.5$
Sleep disturbance, road traffic	0.62 ($L_{Aeq,23-07h} - 43.2$) ^{b)}
Sleep disturbance, rail traffic	0.32 ($L_{Aeq,23-07h} - 40.0$) ^{c)}
Sleep disturbance, aircraft traffic	0.48 ($L_{Aeq,23-07h} - 32.6$) ^{d)}

^{a)} Threshold is 70 dB(A) L_{DEN} except for ^{b)} 43.2 dB(A), ^{c)} 40 dB(A) and ^{d)} 32.6 dB(A); Other assumptions: MI, 7 years of life lost per fatal heart attack in average; base risk of MI: 0.005; survival probability of MI: 0.7; Angina pectoris, base risk: 0.0015; $L_{Aeq,23-07h}$ as assessed outside at the most exposed façade.

Representing uncertainty in estimates

Some approaches we have used

- Prose (non-mathematical) description at all stages
- 'Confidence Intervals' (sampling uncertainty) for C-R functions
- Sensitivity analyses of strategic options
- Stratified presentation of results, according to reliability
- (Quantitative compounding of uncertainties; based on subjective assessments of uncertainty)
- (Reality check against observed changes in life expectancy)

Even better assessment and representation of uncertainty is an important area for development

Final remarks/ Conclusions

Very substantial progress on quantified HIA in air pollution

There is a broad base of agreement on many issues:

- Over-riding importance of mortality from cohort studies;
- That mortality is best described in terms of life expectancy;
- The best explanation is in terms of PM_{2.5};
- There are acute exposure effects of PM and ozone;
- These are additive to one another;
- Relationships in NO₂ and CO may be surrogates of traffic mixtures; a causal role of SO₂ is unclear
- In general, a no-threshold implementation is 1st choice

Important topics and uncertainties are being investigated

- Quantifying effects of different kinds of particles
- Updating C-R functions
- Time-lags from long-term exposure to health effects
- Getting better baseline rates for morbidity
- Representing uncertainty even better