Health impact assessment of exposure to transport emissions in Flanders: methodology study

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Abstract

Traffic is identified as one of the main contributors to ambient air pollution. New evolutions in exposure assessment provide both more accurate estimations of traffic exhaust concentrations and individual exposure concentrations. However current epidemiology and health impact assessment (HIA) methods are not yet capable of dealing with these new exposure evolutions. In this paper methods are explored to perform a HIA based on an activity-based traffic model. Based on the main traditions of a HIA a stepwise approach is presented, estimating the impact of particulate matter, benzene, ozone and nitrogen dioxide in two strategies. A first strategy serves as the core analysis while a second more expansive strategy, allows more uncertainty in assessing the impact of air pollution.
Keywords: air pollution, health impact assessment, traffic, methodology.

1 Introduction

Flanders, the Northern part of Belgium, is a highly dense region typified as one urban nebula, where individual car traffic dominates the coverage of regional movement requirements. Transport is one of the main contributors to the emissions of air pollutants, including particulate matter (PM) and nitrogen dioxide (NO_2). On average the contribution of transport to PM_{10} in Flanders is 28% and 35% for $PM_{2,5}$ [1]. For NO_X the share of traffic in 2002 was 44% of the total NO_X-emissions [2]. Recent research consistently indicates that outdoor air pollution harms health, and points to air pollution that stems from transport as an important contributor. Health impact assessments (HIAs) allow quantifying the effect of transport emissions on health [3-5]. However the number of HIAs on


WIT Transactions on Biomedicine and Health, Vol 14, © 2009 WIT Press
www.witpress.com, ISSN 1743-3525 (on-line)
doi:10.2495/EHR090041

transport-related air pollution is rather limited. Main reason is that epidemiological information is still too inconsistent to derive a well-based exposure–response function, which is needed to quantify the health effects [6].

The current HIA relies on an activity-based model, producing more accurate emission estimates and individual dynamic exposure concentrations. However, current health impact assessment methods and epidemiology are not capable of dealing with these recent evolutions in exposure assessment. Therefore to be able to perform a HIA, possible answers are explored based on current epidemiologic and toxicological research. Main attention is given to the absence of traffic exposure-response functions and how to handle individual exposure estimations. A methodology is proposed on how the health-impacts of air pollution in Flanders based on a traffic model can be assessed. The main steps of a HIA serve as a guideline in the motivation of the methodological choices being made.

2 Hazard identification

Traffic is one of the main contributors to emissions of PM and NO_X. Ozone as a secondary pollutant formed by precursors including nitrogen dioxide, is also highly related to traffic emissions. Toxicological research identifies these three pollutants as having adverse effects on health [7–11], through inflammatory reactions on the lung tissue, which in turn promote blood clot formation and the constriction of blood vessels [12]. Benzene with a permissible volume up to 5% in petrol, is a causative agent in the development of leukemia [13].

Notwithstanding the many uncertainties on the underlying processes that cause a health effect to occur, toxicology provides the biological plausibility of a causal relationship between traffic-related air pollution and health. Epidemiological research does not [6]. In HIAs pollutants, and more specific particulate matter, are therefore usually handled as a uniform pollutant, regardless the contribution from different sources, such as traffic, and thus assuming the same effect on health. This likely leads to an underestimation of the effects of local traffic emissions, since most epidemiological studies do not fully reflect the effects of exposure in hot spots near traffic [5] and the supposed higher toxicity of traffic related particle matter [14, 15]. Some studies [16] established a direct association between traffic and health, through looking at the distance between place of residence and nearby major roads. However the use of this risk estimate is not applicable into the current traffic model.

3 Exposure assessment

To obtain exposure concentrations an activity-based model (AB-model) is used. AB-models provide detailed Origin-Destination (O/D) matrices, by estimating activity-travel schedules for every individual or household. The model can predict which activities are carried out, where, with whom, when, how long, with which transportation mode and finally which route is followed [17]. By changing some parameters in the model, this information can be calculated for different circumstances.

When this extensive information from is applied to a transportation network, detailed information about activity-related traffic flows on the different roads (e.g. average speed and amount of traffic) and total distances for each trip can be calculated. Based on pollutant-specific emission factors, these activity-related traffic flows on the different traffic links can then be converted into vehicle exhaust emissions. Combined with dispersion models this provides more accurate estimates on hourly ambient (traffic) pollutant concentrations [18]. Combining these pollutant concentrations with the simulated activity patterns from the AB-model, revealing each individual's location on a specific time, dynamic exposure estimates can be calculated for each individual. Preliminary results reveal large differences between the static and the dynamic approach, mainly pointing to an underestimation of exposure levels of the first [19].

However, both traffic pollution concentrations and dynamic exposure values offer challenges to current epidemiology. To counter the problem of traffic pollution concentrations, the estimated emissions from traffic are completed with emissions from other sources, such as industry and agriculture, taken from the national emission inventory [20]. By estimating the all-source exposure it is possible to use the existing ambient PM-estimates. The possibility to derive a traffic-attributable fraction of PM (both PM_{10} and $PM_{2,5}$) from the emission-total after dispersion has yet to be researched. The calculation of such a fraction would however allow one to estimate the traffic attributable health effects [3].

For the problem of individual exposure estimates there is no straightforward answer to be found in current epidemiology. Epidemiology uses estimates of population exposure based on fixed monitoring sites and does not reflect the spatial and temporal variability of personal exposure [6]. Therefore this type of variability cannot be taken into account and only the static exposure estimates will be used. These exposure estimates are obtained from the immission data combined with demographic data in a geographic information system (GIS). GIS is used for the (spatial) integration of the different data and can be used to produce both maps of exposure and disease. Concentration data are plotted on a map into grid cells of 1x1km, whilst demographic data are provided for every census tract, a geographical classification allowing more detail than the municipal level. There are 9.906 census tracts in Flanders and Brussels, compared to 327 municipalities. By combining both high spatial resolution concentration and demographic data, detailed concentration distribution maps and tables of Flanders and Brussels are obtained. By applying the age-distribution of the population within every census tract on the concentration data, special attention can be given to vulnerable groups such as children and elderly.

4 Dose-response evaluation

When assessing the effects of air pollution on health the WHO [6, 21] recommends assessing both cause- and age-specific mortality and morbidity. In this study both morbidity and mortality for respiratory and cardiovascular diseases will be calculated. For mortality, cancer is included as well: lung cancer due to (fine) particulate matter and leukaemia due to benzene. For morbidity,

hospital admissions are the prior outcome, with specific attention to asthma and chronic bronchitis or chronic obstructive pulmonary disease (COPD).

When assessing age-specific health outcomes, there is still a great deal of uncertainty between a particular subgroup and the specific health effects: who is susceptible is dependent on the specific health end point being evaluated and the level and length of exposure [7]. Infants and elderly are identified to be particularly at risk.

Risk estimates or concentration-response functions are the main key to perform a HIA, as they link exposure estimates with health data to calculate the health outcomes. Due to their important role in a HIA there are some considerations to bear in mind when selecting the functions to be used, such as the use of estimates from meta-analyses. Especially within Europe such meta-analyses are being carried out [22]. In line with the APHEIS-project (Air Pollution and Health: a European Information System) [23] estimates were chosen from multi-centre studies, such as APHEA (Air Pollution and Health: a European Approach). This is in line with epidemiologic reasoning, giving more weight to the overall results of all adequately conducted studies rather than one single result [24]. Table 1 gives an overview of the different risk estimates selected. The estimates used in APHEIS are as much as possible adopted

It is important to bear in mind the distinction between the health effects of long term and short-term exposure. Hospital admissions are more than often related to short-term exposure, whilst mortality is both related to short- and long-term exposure, so the effects on mortality from both exposures will be calculated. For mortality however there has been a growing recognition that the effects of long-term exposure (expressed as years of life lost, YLL) are more meaningful [3, 25] and should be seen as the main effects [26]. The short-term effects (expressed in number of attributable cases) are only calculated to give a complete and more detailed view on the mortality effects [25, 27]. Because where short-term time series studies clearly show a relationship between ambient PM and mortality from respiratory causes, the long-term cohort studies do not [28]. The mortality effects of long-term and short-term exposure should however not be summed up.

4.1 Short-term exposure estimates

For the short-term effects of PM, the time series studies estimates for cardiovascular and respiratory mortality [29, 37] and hospital admissions [31, 32] within APHEA are adopted. They provide the most recent multi-centre meta-analyses and most robust estimates in Europe. For the elderly both cardiovascular mortality and hospital admissions for cardiovascular and respiratory diseases, such as chronic bronchitis, will be assessed. The estimates are adopted from the APHEA-project [30, 37] and the WHO meta-analysis [22]. For children only hospital admissions for respiratory diseases, including asthma, will be assessed based the short-term exposure estimates from the WHO meta-analysis [22].

Table 1: Overview of selected risk estimates (given as relative risks).

	Health indicator	Age group	ICD 10	RR for a 10 µg/m3 increase	(95% CI)	
Attributable cases						
PM10 very short term	Cardiovascular mortality	all ages	I00-99	1,0076	1,0047-1,0105	[29]
		> 65 years	I00-50	1,0070	1,004 - 1,010	[30]
	Respiratory mortality	all ages	J00-99	1,0058	1,0021-1,0095	[29]
	Cardiovascular hospital admissions	all ages	I00-50	1,0050	1,002 - 1,008	[30]
		> 65 years	I00-50	1,0070	1,004 - 1,01	[30]
	Respiratory hospital admissions	all ages	J00-99	1,0114	1,0062 - 1,0167	[31]
		< 15 years	J00-99	1,0100	0,998 - 1,021	[22]
		> 65 years	J00-99	1,0070	1,002 - 1,013	[22]
	Asthma hospital admissions	15 - 64 years	J45-46	1,0110	1,003 - 1,018	[32]
		< 15 years	J45-46	1,0120	1,002 - 1,023	[32]
	Asthma and COPD hosp admissions	> 65 years	J40-47	1,0100	1,004 - 1,015	[32]
PM2,5 short term	Cardiovascular mortality		I00-99	1,0130	1,005 - 1,021	[22]
	Respiratory mortality		J00-99	1,0110	1,002 -1,020	[22]
O3 short term	All cause mortality (excl ext causes)	all ages	A00-R99	1,0046	1,0022 - 1,0073	[33]
	Cardiovascular mortality	all ages	I00-99	1,0040	1,0022 - 1,0073	[33]
	Respiratory mortality	all ages	J00-99	1,0113	1,0074 - 1,0151	[33]
	Respiratory hospital admissions	15 - 64 years	J00-99	1,0010	0,991 - 1,012	[22]
		15 - 64 years	J00-99	1,0310	1,013 - 1,049	[34]
		> 65 years	J00-99	1,0050	0,998 - 1,012	[22]
		> 64 years	J00-99	1,0380	1,018 - 1,058	[34]
	Asthma hospital admissions	15 - 64 years	J45-46	1,0150	0,955 - 1,078	[35]
		< 15 years	J45-46	1,0060	0,976 - 1,037	[35]
	COPD hospital admissions	all ages	J40-44, J47	1,0430	1,022 - 1,065	[36]
NO2 short term	Cardiovascular mortality	all ages	I00-99	1,0040	1,0029 - 1,0052	[11]
	Respiratory mortality	all ages	J00-99	1,0038	1,0017 - 1,0058	[11]
	Asthma hospital admissions	15 - 64 years	J45-46	1,0290	1,003 - 1,055	[35]
		< 15 years	J45-46	1,0260	1,006 - 1,049	[35]
	COPD hospital admissions	all ages	J40-44, J47	1,0190	1,002 - 1,047	[36]
PM10 accumulative (40 days)	Cardiovascular mortality	all ages	I00-99	1,0197	1,0139 - 1,0255	[37]
		65-74 years	I00-99	1,0206	1,0105 - 1,0309	[37]
		>74 years	I00-99	1,0235	1,0142 - 1,0329	[37]
	Respiratory mortality	all ages	J00-99	1,0421	1,0109 - 1,0742	[37]
		>74 years	J00-99	1,0457	1,0125 - 1,0799	[37]
PM10 long term	Respiratory mortality	1 month - 1 year	J00-99	1,2160	1,102 - 1,342	[38]
	Infant mortality	1 month - 1 year	A00-Y98	1,0480	1,022 - 1,075	[38]
NO2 long term	Respiratory mortality	55 - 69 years	J00-99	1,3700	1,00 - 1,87	[39]
Loss in life expectancy						
PM2,5	All cause mortality	> 29 years	A00-Y98	1,0600	1,02 - 1,11	[40]
	Cardiopulmonary mortality	> 29 years	I10-70, J00-99	1,0930	1,033 - 1,16	[40]
	Lung cancer mortality	> 29 years	C33-34	1,1350	1,044 - 1,23	[40]
Benzene	Leukaemia mortality	all ages	C91-95	6 x 10-6	4,4 - 7,5 x 10-6	[41]

The effects of ozone will be assessed for short-term exposure only. As Flanders (including Brussels) has a moderate climate, high ozone concentrations only occur during summer. So contrary to the recommended estimates of the WHO [22, 42] which are all-year estimates, the estimates from Gryparis *et al.* [33] are adopted, which assess both all-cause mortality (including asthma and

COPD) and cause specific mortality during summer only. The recommended estimates by the WHO [22, 42] for hospital admissions for respiratory causes are slightly insignificant, therefore two studies from APHEA1 will be adopted as well [34, 36], however with greater uncertainties than the proposed meta-analysis, due to the limited cities where the studies are based on.

Epidemiological risk estimates of nitrogen dioxide are uncertain and are therefore treated last. This is mainly due to the inability to derive effects independent from other pollutants in the air pollution mix. However within APHEA2 a relationship between cause-specific mortality could be found [11]. For hospital admissions for asthma and COPD the studies from APHEA1 were adopted [35, 36].

4.2 Long term exposure estimates

For long-term exposure effects of PM, the estimates from cohort studies are used [39, 40, 43]. However these and the more recent and extended studies [28, 44] do not find an association respiratory mortality. Nonetheless the estimates of the original cohort study from Pope *et al.* [40] will be used as they represent more or less the standard for HIAs in air pollution [20, 26]. Next to the all-cause estimate, recommended by the WHO [21], the cause-specific estimate for mortality from cardiopulmonary diseases will be used. This allows being coherent with the estimates for short-term exposure that are also cause-specific. For lung cancer the estimates from Pope *et al.* [40] will be used for $PM_{2,5}$. For leukaemia the risk estimate of the WHO Air Quality Guidelines for benzene [41] is used.

Recently a cohort study from a neighbouring country researched the effects of $PM_{2,5}$ on the same health outcomes as Pope et al. [40]. This Dutch study is not adopted, but is worth mentioning because no association could be found between $PM_{2,5}$ and the three outcomes assessed here [45].

Where the Dutch cohort study could not find an association with $PM_{2,5}$, this was not the case for NO_2. This allows an assessment of the effects of long-term exposure to NO_2 on respiratory mortality.

Special attention is given to infant (respiratory) mortality. For infant mortality, rather than loss in life-years the attributable cases will be calculated, following Kaiser *et al.* [46]. All risk estimates are taken from Lacasana *et al.* [38].

5 Health impact characterization and integration of data

5.1 Consequences of epidemiologic restrictions

The last step in a HIA is to integrate all data in order to quantify the health burden. The many uncertainties related to the risk estimates and the restrictions of epidemiology forces to adopt a twofold strategy in which the health effects are assessed. These strategies are based on the main traditions within HIAs. Depending on the aim of the HIA there's a distinction between a 'conservative'

and an 'expansive' approach [26]. The 'conservative' approach puts emphasis on ensuring that every impact pathway that is included has been quantified in a reliable way. Estimates are based strongly in the available evidence and it makes no claim to capture the full effects of air pollution. The main purpose is to compare and evaluate the costs of different strategies to reduce pollution. The 'expansive' approach on the other hand, puts priority on the reliability of the quantification as a whole as an estimate of the overall benefits of reducing air pollution. It aims to capture all the effects or air pollution, with a wider coverage of the assumptions and less reliable estimates.

Combining both approaches in one assessment seems – in view of the many health effects for PM, benzene, ozone and NO_2 – preferable. In a first phase we will adopt the first 'conservative' tradition, as our aims resemble those of comparing different policy scenarios to reduce air pollution levels and their health impacts. This to establish a solid base for the second strategy in which we will adopt the more 'expansive' approach. In the latter concentration-response functions will be used for which there is good evidence of effect, but a weakness at some point in the impact pathway, e.g. where either toxicological or epidemiologic research fails to deliver certainty on how an impact is to be understood, while the impact itself is undisputed.

Related to the pollutants assessed, this means that a step-wise approach has to be applied (Figure 1), where all pollutants are assessed in descending order of certainty (PM > Benzene > Ozone > Nitrogen dioxide). This hierarchy has to be seen in light of the absence of risk estimates for traffic air pollution and the question which air pollutant is the best indicator for traffic air pollution. Recently NO_2 is seen as a better surrogate for the health effects of transport than PM [21, 45, 48]. However uncertainty remains as it is not clear whether NO_2 is really a better indicator for traffic pollution or whether there is a confounding effect [29]. The results of the individual pollutants however cannot be added together as this would lead to overestimation [24].

5.1.1 The conservative strategy

As the main goal of this HIA resembles most the 'conservative' approach, this was adopted as the first strategy. The conservative tradition is characterized by using the 'at least' approach [23], which consistently selects methodological assumptions in a way to get an impact 'at least' attributable to air pollution, such as calculating the health effects from a baseline frequency –a derived baseline incidence from a situation without pollution- and using an additive instead of multiplicative risk function [3, 24]. These methods are adopted here as well.

Assuming that the relation between particles and benzene on health is causal, major uncertainties arise from the selection of the risk estimate and the methods to measure the concentrations. However by adopting the 'at least' approach, the influence of these are minimized as much as possible [24, 47], by selecting only concentration-response functions that are well established and limiting the health effects to those from PM and benzene, a known carcinogen.

5.1.2 The 'expansive' strategy

The second strategy allows a more comprehensive view on the health effects of air pollution. In this strategy the effects of ozone and NO_2 will be assessed, together with all age-specific health effects. Ozone is not adopted in the first strategy as the impact on health is not fully established yet, especially regarding the moderate climate in Flanders. For NO_2 it is clear that the health effects of NO_2 are independent or either correlates with the complex mixture of traffic exhaust. And the age-specific effects are not adopted in the first strategy, because of the many uncertainties that remain in many associations between a particular age and a specific health effect [7]. A sensitivity analysis will be part of the second strategy as well. These alternative analyses provide an insight on how the different decisions and assumptions affect the eventual impact and quantify the many uncertainties associated with HIAs.

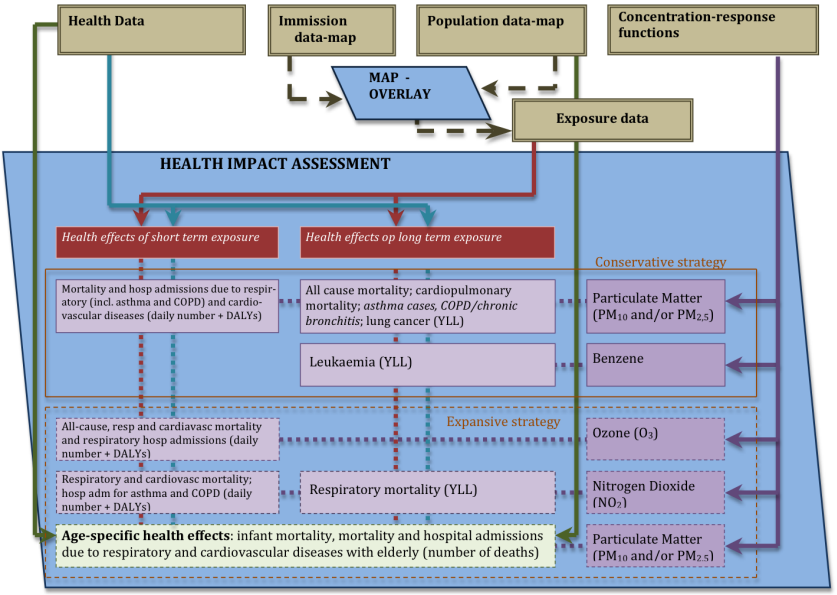

Figure 1: Data sources, processing and expected outcomes.

5.2 Integration of data into GIS

To evaluate the health impact of the exposure to air pollution, the exposure estimates have to be linked to data on the current health burden and demographic data. Using a GIS it is possible to integrate these data, to export them to calculation sheets and to visualize them geographically [49]. Maps are valuable tools in risk assessment, because of their ability to explore (spatial) changes in disease patterns associated with air pollution exposure [50].

However there is a difference in the level of detail between concentration data and health data. Exposure data are provided for every census tract, but health

data are not. Mortality data are available for each municipality and hospital admission data are only available at the district level. Therefore depending on the health outcome results will be presented on either commune or district level.

6 Discussion

Current methods of HIA are not yet capable of dealing with recent evolutions in exposure assessment. In this paper preliminary answers are formulated to these evolutions. As it is not yet possible to take advantage from the newly developed individual exposure concentrations, this HIA will still use ambient concentrations, but on a detailed geographical level. To counter the problem of traffic-related pollution a stepwise approach is adopted in which every pollutant, which is related to traffic, is assessed. Particulate matter and benzene are taken as the main pollutants, based on their robust associations with health. Ozone and nitrogen dioxide are assessed through their relationship with traffic exhaust, but as their associations with health are still troubled with uncertainties, they are not added in the core analysis.

However, real answers should come from new epidemiologic research in finding risk estimates for traffic exhaust and individual exposure concentrations. HIAs allow governments to gain insight on the health impact of their mobility patterns. However if governments want to invest in performing HIAs, they should also invest in epidemiology. Up to this date to many barriers exist in gaining detailed data on health, because of confidentiality issues, which hinders a fast development of epidemiology, especially when calculating risk estimates for individual exposure.

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