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Air pollution in perspective: Health risks of air pollution expressed in equivalent numbers of passively smoked cigarettes

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ABSTRACT

Background: Although the health effects of long term exposure to air pollution are well established, it is difficult to effectively communicate the health risks of this (largely invisible) risk factor to the public and policy makers. The purpose of this study is to develop a method that expresses the health effects of air pollution in an equivalent number of daily passively smoked cigarettes.

Methods: Defined changes in PM_{2.5}, nitrogen dioxide (NO₂) and Black Carbon (BC) concentration were expressed into number of passively smoked cigarettes, based on equivalent health risks for four outcome measures: Low Birth Weight (< 2500 g at term), decreased lung function (FEV₁), cardiovascular mortality and lung cancer. To describe the strength of the relationship with ETS and air pollutants, we summarized the epidemiological literature using published or new meta-analyses.

Results: Realistic increments of 10 µg/m³ in PM_{2.5} and NO₂ concentration and a 1 µg/m³ increment in BC concentration correspond to on average (standard error in parentheses) 5.5 (1.6), 2.5 (0.6) and 4.0 (1.2) passively smoked cigarettes per day across the four health endpoints, respectively. The uncertainty reflects differences in equivalence between the health endpoints and uncertainty in the concentration response functions. The health risk of living along a major freeway in Amsterdam is, compared to a counterfactual situation with 'clean' air, equivalent to 10 daily passively smoked cigarettes..

Conclusions: We developed a method that expresses the health risks of air pollution and the health benefits of better air quality in a simple, appealing manner. The method can be used both at the national/regional and the local level. Evaluation of the usefulness of the method as a communication tool is needed.

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1. Introduction

Numerous studies have documented the adverse health effects of air pollution, even at levels well below the EU limit values (HEI, 2010; WHO, 2013; Beelen et al., 2015). In order to meet the European Union limit values, national and local authorities have to take action and attempt to reduce the emissions from mobile, domestic, agricultural and industrial sources. Because health effects of air pollution

also occur below air quality limits, improvement of population health is an argument for further air pollution reduction policies. At the local level, policy measures are being implemented like congestion charges, low emission zones, increased parking rates. Policy makers need to “sell” the (often) expensive and restrictive measures to the public and create enough political support to implement them. Insight in the health impact of local air quality enhances acceptance and adoption of preventive measures (Briggs and Stern, 2007). This requires effective communication with the public about the health risks of local air pollution and the health benefits of improved air quality. However, it is difficult for local policy makers and health professionals to effectively communicate about the health risk of air pollution (Slovic, 1999; Weber, 2006; Bickerstaff and Walker, 2001).

One way to express health risks of local sources is by means of a risk quotient (relative risk or odds ratio) but this does not necessarily reflect perception of risks in a population, since perception is only partly based on scientific information (Slovic, 1999; Weber, 2006; Stewart et al., 2010). Alternatively, excess mortality

Abbreviations: BC, black carbon; CRF, concentration response function; ETS, environmental tobacco smoke; FEV₁, Forced Expiratory Volume in 1 s; LBW, Low Birth Weight (Birthweight < 2500 g after 37 weeks of gestation); IHD, ischaemic heart disease; NO₂, nitrogen dioxide; OR, odds ratio; PM_{2.5}, particles smaller than 2.5 µm; PM₁₀, particles smaller than 10 µm; RAP, risk advancement period; RR, relative risk; YLL, years of life lost

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risks resulting from exposure to local air pollution can be translated into years of life lost (Brunekreef et al., 2007) or risk-advancement period (Brenner et al., 1993), where years of life lost (YLL) are extrapolated into 'real age'. In a recent study, Geelen et al. (2013) reported that 'real age' of an individual increased with up to 36 days near the highway in the Moerdijk area in the Netherlands, compared to the background concentration. The impact of the implementation of a low emission zone in Rome was expressed as the gain in life expectancy: 921 years per 100.000 inhabitants, on average 3.4 days per person (Cesaroni et al., 2012). Modeled benefits of the London congestion charging zone was 183 years of life per 100.000 (0.7 days per person) inhabitants in the charging zone wards (Tonne et al., 2008). The effectiveness of communicating risk advancement periods, or years of life lost or gained due to air pollution to the public and policy is generally not evaluated. Yet, effective communication is important for the above mentioned reasons.

In a previous Dutch study, the impact of local traffic on Black Carbon (BC) concentration was translated into YLL based on the relation between BC exposure and life expectancy described by Janssen et al. (2011) and presented graphically at building-level for all major roads in the densely populated provinces North- and South-Holland (van der Sluis et al., 2012). The usefulness of this information for local policy makers and governors was evaluated in interviews. They were insufficiently able to interpret the effect on YLL and were unanimous in their wish for a simple, appealing comparison of the health risks of local air pollution with other, well-known risk factors. Risk factors that were mentioned were: passive or active smoking; obesity; unhealthy diet; traffic accidents (van der Sluis et al., 2012). Recently, Kelly and Fussell (2015) stressed that in order to increase public awareness, communication about the health risks of air pollution should be blatant and put in the context of other public health risks such as passive smoking. The principle of risk comparison for better communication of risk to the public was used earlier, for example in the Global Burden of Disease project, where the risks of a large number of risk factors including outdoor air pollution and passive smoking have been compared globally and for various regions of the world (Lim et al., 2012; Forouzanfar et al., 2015).

Pope et al. (2009) and Smith and Peel (2010) used the inhaled dose of PM_{2.5} from active smoking, household air pollution (indoor biomass and coal burning), passive smoking and outdoor air pollution to compare deaths due to cardiovascular disease. Due to the non-linear shape of the exposure-response relationship, much larger health benefits may occur at the lower end of the dose spectrum (Smith and Peel, 2010). The inhaled dose of PM_{2.5} is more than 300 fold higher for the average smoker than for the average passive smoker (Pope et al., 2009; Öberg et al., 2010). Inhaled PM_{2.5} doses for outdoor air pollution and passive smoking are comparable. As a consequence, the health effects of outdoor air pollution can more meaningfully be compared with those of passive smoking than of active smoking.

The aim of this study is to express the health effects of air pollution in equivalent amounts of passive smoking. We compared health risks of air pollution with passive smoking because both risk factors are comparable with respect to the exposure route (inhalation); have similar health effects both resulting from a complex mixture of particles and gases; and exposure to both air pollution and environmental tobacco smoke (ETS) is largely involuntary.

A simple tool is developed based on the relative risks describing the association between exposure to ETS and three key air pollutants: particulate matter with aerodynamic diameters < 2.5 µm (PM_{2.5}), nitrogen dioxide (NO₂) and Black Carbon (BC) and four health outcomes (Low Birth Weight, lung function decrements in children, cardiovascular mortality and lung cancer).

We illustrate the method by expressing the health effects of living near a freeway, the health gain of a local traffic measure and the health effects of the emissions of a steel factory into equivalent amounts of exposure to ETS.

2. Material and methods

2.1. Selection of health outcomes

First, we searched the air pollution epidemiology literature for health outcomes with the most secure evidence of an association with PM_{2.5}, NO₂ and Black Carbon (BC). PM_{2.5} is the preferred air pollution indicator for health impact assessment at the national or regional level. NO₂ and BC are the preferred air pollution indicators for health impact assessment at the local level, in situations where traffic is the primary source of air pollution. We made use of published (systematic) reviews, supplemented with more recent key studies.

Second, we searched the passive smoking epidemiology literature to select health outcomes with the most secure evidence of an association with ETS exposure. We made use of WHO and Surgeon General Reports and published (systematic) reviews.

Next, we selected health outcomes with the most secure evidence of an association for both the relation with ETS exposure and air pollution. We further aimed at including health outcomes reflecting adult and childhood health responses to evaluate differences in the ratio of air pollution and ETS health effects. We finally included four health outcomes:

1. Low Birth Weight (LBW) defined as a birth weight less than 2500 g after 37 weeks of gestation.
2. Lung function (FEV₁) in school aged children.
3. Lung cancer.
4. Cardiovascular mortality.

2.2. Exposure-response functions for the relation between ETS exposure and health outcomes

Continuous data on ETS exposure (number of cigarettes) is rarely available in epidemiological studies. Some epidemiological studies have a more quantified assessment of ETS exposure, often classified into "low or moderate ETS exposure" or "moderate to high exposure". However, the cut-off points for the different exposure categories differ between studies. Therefore, meta-analyses such as the WHO's Global Burden of disease related to ETS (Öberg et al., 2010) provide estimates based on dichotomous exposure classification (presence / absence of ETS in the home or at work). Table 1 provides an overview of the risk estimates for ETS exposure for the selected health outcomes.

2.3. Assessment of ETS exposure

The risk estimates in Table 1 are based on dichotomous exposure classification. However, an estimate of the average daily residential exposure to ETS is essential to express air pollution health effects in an equivalent amount of cigarettes smoked. Based on estimates from the WHO for smokers in the US and North-West Europe, we assume an average of 14 daily cigarettes (Öberg et al., 2010).

The average daily residential exposure was estimated following a (modified) approach by Nazaroff and Singer (2004). This is based on the assumption that the average smoker consumes half of their daily cigarettes indoors at home, which follows from an expectation that habitual smokers will consume cigarettes at a roughly uniform rate throughout the hours that they are awake. People in

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