ERJ Express. Published on March 21, 2013 as doi: 10.1183/09031936.00031112

Chronic burden of near-roadway traffic pollution in 10 European cities (APHEKOM network)

Laura Perez(1) (2), Christophe Declercq (3), Carmen Iñiguez (4) (5), Inmaculada Aguilera (5) (6), Chiara Badaloni (8), Ferran Ballester (4) (5), Catherine Bouland (7), Olivier Chanel (9), FB Cirarda (10), Francesco Forastiere (8), Bertil Forsberg (11), Daniela Haluza (12), Britta Hedlund (13), Koldo Cambra (14), Marina Lacasaña (5) (15), Hanns Moshammer (12), Peter Otorepec (16), Miguel Rodríguez-Barranco (15), Sylvia Medina (3), Nino Künzli (1) (2)

Work conducted at:

Swiss Tropical and Public Health Institute, Socinstrasse 59, 4051 Basel, Switzerland and Centre for Research in Environmental Epidemiology, Barcelona, Spain

Author's Affiliations:

Name	email	Affiliation
Laura Perez	l.perez@unibas.ch	(1) Swiss Tropical and Public Health Institute, Basel, Switzerland. (2).University of Basel, Basel, Switzerland
Christophe Declercq	c.declercq@invs.sante.fr	(3) Institut de Veille Sanitaire, France
Carmen Iñiguez	inyiguez_car@gva.es	(4) University of Valencia. Center for Public Health Research (CSISP), Valencia. (5) Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP).
Inmaculada Aguilera	iaguilera@creal.cat	(6) Centre for Research in Environmental Epidemiology (CREAL), Hospital del Mar Research Institute (IMIM), Barcelona (5) Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP).
Chiara Badaloni	badaloni@asplazio.it	(8) Department of Epidemiology, Lazio Regional Health Service, Via S. Costanza 53, 00198 Rome, Italy
Ferran Ballester	ballester_fer@gva.es	(4) University of Valencia. 2.Center for Public Health Research (CSISP), Valencia. (5) Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP).
Catherine Bouland	cbo@ibgebim.be; catherine.bouland@ulb.ac.be	(7) Brussels Institute for the Management of the Environment, Brussels-Capital Region, Belgium; School of Public Health, Université Libre de Bruxelles – ULB, Belgium
Olivier Chanel	Olivier.Chanel@univmed.fr	(9) -Aix Marseille University (Aix-Marseille School of Economics), CNRS&EHESS, France
Cirarda, FB	fbcirarda@ej-gv.es	(10) Dirección Territorial de Sanidad de Bizkaia. Bilbao. Basque Country. Spain.
Francesco Forestiere	forastiere@asplazio.it	(8) Department of Epidemiology, Lazio Regional Health Service, Via S. Costanza 53, 00198 Rome, Italy
Bertil Forsberg	Bertil.forsberg@envmed.umu.se	(11) Occupational and Environmental Medicine. Umeå University, Umeå, Sweden
Daniela Haluza	daniela.haluza@meduniwien.ac.at	(12) Medical University of Vienna, Austria, Institute of Environmental Health, Center for Public HealthVienna. Austria
Britta Hedlund,	britta.hedlund@naturvardsverket.se	(13) Swedish Protection Agency, Stockholm, Sweden

Cambra, Koldo	ki.cambra.contin@navarra.es	(14) Centro de Investigación Biomédica. Pamplona. Spain.
Marina Lacasaña	marina.lacasana.easp@juntadeandalucia.es	(15) Escuela Andaluza de Salud Pública (EASP); (5) Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP);
Hanns Moshammer	hanns.moshammer@meduniwien.ac.at	(12) Medical University of Vienna, Austria, Institute of Environmental Health, Center for Public HealthVienna. Austria
Peter Otorepec	peter.otorepec@ivz-rs.si	(16) National Institute of Public Health, Ljubljana, Slovenia
Miguel Rodríguez-Barranco	miguel.rodriguez.barranco.easp@juntadeandalucia.es	(15) Escuela Andaluza de Salud Pública (EASP);
Sylvia Medina	s.medina@invs.sante.fr	(3) Institut de Veille Sanitaire, France
Nino Kuenzli	Nino.kuenzli@unibas.ch	(1) Swiss Tropical and Public Health Institute, Basel, Switzerland. (2). University of Basel, Basel, Switzerland

Corresponding author:

Laura Perez, PhD
Swiss Tropical and Public Health Institute
Socinstrasse 57, P.O. Box, 4002 Basel-CH
www.Swisstph.ch
Tel: ++41(0)61284 8395
Fax: ++41(0)61284 8106
e-mail: l.perez@unibas.ch

Running title: Near-road traffic-related pollution and chronic burden

Key words:

Air pollution, childhood asthma, coronary heart disease, exacerbations, health impact assessment, traffic proximity

Abstract 210

Study

Recent epidemiological research suggests that near road traffic-related pollution may cause chronic disease, as well as exacerbate related pathologies, implying that the entire 'chronic disease progression' should be attributed to air pollution, no matter what the proximate cause

was. We estimated the burden of childhood asthma attributable to air pollution in 10 European cities by calculating the number of cases of 1) asthma caused by near road traffic-related pollution, and 2) acute asthma events related to urban air pollution levels. We then expanded our approach to include coronary heart diseases in adults.

Methods

Derivation of attributable cases required combining concentration-response function (CRF) between exposures and the respective health outcome of interest (obtained from published literature), an estimate of the distribution of selected exposures in the target population, and information about the frequency of the assessed morbidities.

Results

Exposure to roads with high vehicle traffic, a proxy for near road traffic-related pollution, accounted for 14% of all asthma cases. When a causal relationship between near road traffic-related pollution and asthma is assumed, 15% of all episodes of asthma symptoms were attributable to air pollution. Without this assumption, only 2% of asthma symptoms were attributable to air pollution. Similar patterns were found for coronary heart diseases in older adults.

Answer to question

Pollutants along busy roads are responsible for a large and preventable share of chronic disease and related acute exacerbation in European urban areas.

Introduction

Health impact assessments of air pollution usually rely on the availability of relevant epidemiological studies[1, 2]. In recent years, substantial progress has been made in unraveling the effects of air pollution on health. Two findings in particular have lead to a revision of approaches used to derive attributable burden.

First, numerous studies indicate that air pollution contributes to the development of chronic pathologies, and thus affects the incidence and prevalence of chronic diseases[3-5]. In the past, the chronic effects of air pollution have been taken into account by estimating the number of life years lost due to long-term exposure to air pollution. The morbidity burden is considered only for acute effects of air pollution (e.g. cardiorespiratory hospitalizations, bronchitis symptoms, myocardial infarctions (MI) or restricted activity days) [1, 6, 7]. However, chronic morbidity due to air pollution also heavily impacts health and the health care system in addition to acute effects. Thus, the burden of chronic disease due to air pollution has been grossly underestimated.

Second, recent evidence shows that living in close proximity to busy roads has a negative impact on health. [5]. Near-road concentrations of traffic-related gases and particles – in particular the ultrafine fraction of particles - are many times higher than urban background levels along busy roads.[5]. This spatial pattern is not observed for the pollutants most often investigated in impact assessments, such as PM_{10}, $PM_{2.5}$ or ozone. Furthermore, experimental studies show that the health effects of ultra-fine near-road particles are not necessarily the same as the effects of larger particles [8]. Therefore,

common measurement strategies and health assessments may not adequately characterize near-road pollution and associated health impacts.

Few Epidemiological studies use the near-road pollutants for exposure assessment. The studies which do examine near-road pollutants – including several European studies, suggest that the spatial distribution of near-road traffic-related pollutants corresponds to the distribution of a variety of health effects. [9-16]. Of special concern is the role that near-road exposure may play in the development of chronic pathologies, such as asthma and cardiovascular diseases, including coronary heart disease (CHD) [3-5]. Evidence linking near-road pollution exposure to childhood asthma - the most prevalent chronic disease in children – is particularly strong [3, 4]. Children living close to busy roads are more likely to develop asthma [3]. In contrast, markers of secondary particulate pollution, such as $PM_{2.5}$ or $PM_{10,}$ are not so clearly associated with childhood asthma incidence or prevalence[3]. This indicates that a more refined technique is needed to evaluate exposure and related health effects, and/or there are different mechanisms involved in the toxicity of near-road pollutants compared to urban background particulate matter.

If a risk factor – e.g. air pollution – affects both the development of chronic pathologies and the exacerbation of acute events, usual methods to derive the burden of events attributable to the risk factor become inappropriate [17]. For chronic diseases caused by a particular risk factor, the entire 'disease progression', including acute events, should be attributed to the risk factor, regardless of the proximate cause.[18]. Traditional air pollution impact assessments quantified only the burden of acute events directly associated with exposure. A recent case study of two communities in Southern California (USA) revealed

that traditional methods greatly under-estimated the impact of air pollution on childhood asthma[17, 19]. This effect may be particularly strong in Europe, where there are high urban densities, increased traffic loads, and limited policies which address the concentrations of near-road pollutants, such as ultrafine particles. Therefore, there is strong need to update methods, and expand health impact assessments in Europe.

Taking into account recent findings, the objective of this study was to determine the burden of acute events related to the chronic 'morbidity progression' attributable to near-road traffic-related pollution, compared to the burden of acute events attributable to urban background levels of air pollution in 10 European cities. While our assessment focused on childhood asthma, we also expanded our analysis to include chronic conditions in adults, in particular CHD.

Material and Methods

We used population-attributable fractions (PAF) to estimate the health impact of air pollution in 10 European cities, all of which are partners in the Aphekom project, *Improving Knowledge and Communication for Decision Making on Air Pollution and Health in Europe*. PAF is the standard methodology used to assess the contribution of a factor to disease. It uses excess risks obtained from epidemiological studies and applies them to target populations. This assessment is based on methods developed previously[17]. Derivation of attributable cases required knowledge about the concentration-response function (CRF) between exposures and the respective health outcome of interest

(obtained from published literature), an estimate of the distribution of selected exposures in the target population, and information about the frequency of the assessed morbidities. Specific assumptions and the data used are presented below. We identified CRFs for the association between near-road traffic-related pollution and chronic outcomes based on a recent review[5]. Residential proximity to busy roads, defined as >10,000 vehicles / day, was selected as a marker of long-term exposure to near-road traffic-related pollution (Table 1 and 2). Consistent with our recent studies [19], the Californian study was used for childhood asthma data.

For the acute outcomes, we obtained CRFs from hospitalization and symptom studies (Table 1 and 2). The acute effect evaluation was limited to the impact of ambient concentrations of PM_{10} or NO_2. Meta-analytic estimates were derived using inverse-variance-weighting when several studies were available. We corrected the odds ratio, calculated from odds ratios of published studies, to avoid overestimating the true relative risks in our final estimations [20]. Our target population was restricted to children aged 0-17 years for asthma-related outcomes and to adults older than 65 years for CHD-related outcomes. Hospital data were obtained from local registries. Other morbidity prevalence data that could not be drawn from local surveys were extrapolated from studies used to derive the CRFs (Table 3).

A near-road buffer to define ‘exposed populations to near-road traffic-related pollution‘ was determined based on the studies used to derive the CRFs: 75m for asthma and 150m for CHD. In the absence of routinely available distributions of populations along roads, methods developed previously were applied in all cities[17]. In brief, data from population

distribution maps given at centroids of census blocks were distributed within a hypothetical grid across residential areas to represent the number of inhabitants per household. Traffic density maps, showing roads with more than 10,000 vehicles/day (busy roads), were then overlaid and the distance from each grid point to the nearest pre-identified busy road was calculated to derive the population living within the distance-based near-road buffers. The level of detail available to develop these distributions varied across cities. As a rule, the most detailed information was always used. For example, population data given at the door address of buildings was preferred over assuming a distribution of population over larger geographical units, (i.e. when data was available at block level only). For cities for which complete traffic counts or traffic models were not available, local expertise and/or urban planning information was used to select major traffic arteries. In the case of exposure to urban background pollution, used for the acute exacerbation estimates, annual average concentrations derived from urban background fixed site monitors of PM_{10} and NO_2 were assumed to apply to the entire population. Daily levels measured at the fixed site monitors were used to calculate annual average concentrations using standard city protocols.

The disease burden attributable to air-pollution strongly depends on the points of reference, which in turn may depend on policy questions. In the case of the disease burden from near-road traffic-related pollution, we provided only one scenario: the total burden of these chronic conditions attributable to living within the determined buffers. In other words, the implicit assumption is that if no one lived within these buffers, the burden would be prevented. Alternatively, this translates into a scenario in which those

living along busy roads were no more exposed to traffic-related pollutants than those living outside of the buffer zone. If all near-road effects were due to tail pipe emissions, the latter scenario would reflect cities with only zero-emission vehicles on the roads.

In the case of urban background pollution, we used an annual mean of 20 $\mu g/m^3$ as the scenario reference point. For PM_{10}, this reflects the World Health Organization (WHO) guideline value [21]. For both PM_{10} and NO_2, the scenario reference point reflects an annual mean not yet reached in any of the participating cities except Stockholm, thus, we indicate the impact of further air quality improvement to levels that are thought to be realistic, as exemplified in case of Stockholm. Previous assessments used reference values as low as 7.5 $\mu g/m^3$ [1]. To derive acute effects, we expressed a change in scenario in terms of the change in the annual mean concentration. Summing up daily effects due to daily changes in concentrations would have given the same results, but changes in annual means are more meaningful, especially from a policy perspective.

To reflect statistical uncertainty, we provided a point estimate and an upper and lower bound for the 95% confidence interval of the chronic and acute CRFs combined, and 20% variability for chronic health outcomes. We used Monte Carlo simulations to propagate uncertainty distributions and to evaluate the sensitivity of our results. All calculations of disease burden were conducted at both the city level and aggregated across the ten cities.

Results

Table 4 summarizes exposure data obtained from the 10 cities and used to evaluate the burden of childhood asthma and CHD. On average, for the 10 cities considered, 31% of the combined population was estimated to live within 75m of a busy road and 53% within 150m of such roads. Across the 10 cities, the annual weighted mean for PM_{10} was 30 $\mu g/m^3$ and 39 $\mu g/m^3$ for NO_2.

Table 5 presents the number and proportion of chronic disease cases attributable to near-road traffic-related pollution. We estimated that an average of 33,200 asthma cases (14% of all asthmatic children, with a 95% confidence interval [95%CI] estimated between 3% and 25%) were attributable to near-road traffic-related pollutants. In other words these cases would not have occurred if no one lived within the buffer zone or if those pollutants did not exist. For CHD estimates, city-specific data was not available, thus using published prevalence we estimated an average of 37,200 CHD cases attributable to near-roadway traffic-related pollution (28% of all older adults with CHD, with a 95%CI estimated between 9% and 44%).

Table 6 presents the total number and percentage of selected exacerbation outcomes for childhood asthma attributable to air pollution on a yearly bases. Without considering the disease progressions induced by near-road traffic-related pollution, we estimated that reducing air pollution levels to the scenario reference values would prevent 1,585 episodes of asthma symptoms (1.7%) and 20 asthma hospitalizations (1.1%) (table 6, column 4). The estimated preventable burden substantially increases when taking into account the notion that living near busy roads — used as a marker for near-road traffic-related pollutants — caused the development of chronic pathologies. Considering disease

progressions, we estimated a total of 14,400 episodes of asthma symptoms and 260 asthma hospitalizations (table 6, last column) attributable to air pollution, accounting for 15% of acute events .

We found similar patterns for acute cardiovascular outcomes (Table 7). For example, aggregated over the 10 cities, we estimated that 27% of non-fatal MIs, 28% of MI hospitalizations, and 26% of stroke hospitalizations were attributable to causes other than air pollution among those who suffer CHD due to near-road traffic-related pollution (Table 6, column [5]). These cases had been previously unaccounted for.

Discussion

Our health risk assessment explored the morbidity attributable to near road traffic-related air pollution in 10 European cities, taking into account air pollution's dual role as a cause of chronic diseases, and a trigger for associated acute events. Results indicate that near road traffic related pollution may be responsible for a significant portion of the burden of asthma in children, as well as of CHD in older adults.

Spatial analysis showed that approximately 31% of the population live within 75m of busy roads,and 53% within 150m. This distribution is consistent with results from other epidemiological studies in Europe. [11, 22] Notably, however, this distribution is very different from urban areas in the United States, such as Long Beach and Riverside, where a lower percentage of the population lives in close proximity to busy roads. [23 24] This suggests that, in Europe, exposure to and impacts of near-road traffic-related pollution

may be substantial. Indeed, in cities where background pollution levels are low, and further mitigation strategies may appear to be unnecessary, near-road traffic-related pollution may in fact contribute greatly to the local disease burden. For example, in Stockholm, PM_{10} and NO_2 levels were below our reference values and the WHO recommended standards, yet approximately 7% of asthma cases and associated acute events were attributable to near road traffic-related pollution.

For the acute events selected, we found that the burden attributable to near road traffic related pollution was on average 30 times larger than estimates which did not consider exposure both a cause of underlying chronic disease as well as a trigger for acute events. These findings indicate that reducing near road traffic related pollution would be an effective way to reduce the health burden and economic cost of both chronic disease and acute events. [25].

Several studies in high income countries estimate that as much as 50% of the reduction in deaths from coronary diseases over the last 20 years can be attributed to preventive measures, such as lowering cholesterol levels, smoking prevalence, blood pressure, and physical inactivity. [26, 27] Our estimates for chronic diseases attributable to living near busy roads are quite large. Thus – assuming a causal relationship between near road traffic related pollution and CHD- results suggest that decreasing pollution near roads could be an effective additional preventive measure in the long-term.

Our approach is based on two major assumptions and related uncertainties: 1) that near-road traffic-related pollution plays a part in the development of chronic diseases, and 2)

that urban background air pollution exacerbates these diseases. The degree and type of uncertainty related to these assumptions differ, depending on the outcome studied.

In the case of asthma, the role that air pollution plays in exacerbating symptoms has been established for a long time, while the role that air pollution plays in asthma onset in children has only emerged in recent years. [3, 4, 28] The Health Effect Institute (HEI) classified the evidence that long-term exposure to near-road traffic-related pollutants may lead to asthma onset as "sufficient evidence" or "suggestive but not sufficient" [5]. A highly relevant analysis of the Children's Health Study, a cohort study of school age children, identified an interaction between genetic factors and the risk of asthma incidence among those living close to highways – a finding very hard to explain with uncontrolled biases or confounding factors. [29] However, there are additional uncertainties related to this outcome. The question remains whether removal of near-road pollution would in fact prevent or only delay the onset of the chronic underlying pathologies. [3] In the latter case, the burden will be overestimated, however, delaying the onset of asthma symptoms even for a few years may represent a large improvement in quality of life and savings for families.[25] Furthermore, the Dutch birth cohort study followed children up until 8 years old, and found no evidence supporting postponed onset, validating our assumption. [30]

In the case of MI, there is also a large amount of evidence that acute events are related to high daily levels of air pollution.[5, 31, 32] The evidence for a link between pollution and underlying chronic conditions is less established, as there are few population studies of near-road pollutants or proximity for these outcomes. On the other hand, the evidence

that atherosclerosis is related to particulate matter levels is very strong in animal studies, and at least suggestive in some epidemiological studies in humans.[18] There is also considerable biological evidence of mechanistic pathways linking air pollution to MI, such as oxidative stress and inflammation. [31] Considering these factors, the HEI classified the evidence for the association between traffic pollution and cardiovascular disease as "suggestive but not sufficient". Given current knowledge, the extent to which near-roadway traffic pollution contributes to the progression of atherosclerosis to the point of increasing the individual's risk of MI is unknown, and selecting a pool of individuals at risk of developing acute symptoms remains a challenge.

The interpretation of PAFs has several limitations. For most complex diseases, morbidity develops because of the interaction of several risk factors. This multicausal model implies that it is sufficient to eliminate or remove any one of the factors contributing to the disease in order to prevent it [33]. PAF are generally developed one risk factor at a time, ignoring the potential effects of interactions between contributing factors. If one sums the PAFs derived separately for each potential risk factor for a specific disease, the total PAF for the disease may add up to more than 100% (greater than the total number of cases) since attributable cases for individuals with more than one contributing factor may have been counted more than once. [34, 35] We present our results assuming that one risk factor is eliminated, with all other risk factors remaining unchanged. The large PAFs of disease prevalence due to near road traffic exposure is comparable to what has been found for other preventable risk factors, such as smoking. This is driven primarily by the very large fraction of the population living under conditions of high exposure, and underscores the

substantial relevance of this exposure to public health. Near-road traffic-related pollution attributable fraction does represent a preventable risk factor.

Nonetheless, because of the complex chain of biological and behavioral interactive pathways at play, and the uncertainty in the lag time of health changes that may result from small stepwise reductions in near road traffic-related pollutants, we cannot predict the true benefits of reducing near-road traffic-related exposure. If causality is true, our results indicate that decreasing exposure to near-road traffic-related pollution could effectively contribute to prevention of some chronic diseases. The extent to which reduced exposure will decrease the disease burden, however, remains unknown, and depends on changes in other risk factors. For example, in a real world scenario, the adverse effects of an increasing obesity epidemic may outweigh the benefits of reducing near-road traffic-related pollution, thus, disease rates may increase despite improvements in air quality.

For the two chronic outcomes evaluated in our study, we used the CRF of a single rather than a pooled estimate. This is a major limitation of the assessment, as we do not know how representative the CRFs are for the European cities of our study. We could not identify a sufficient number of studies that would allow for the derivation of a meta-analytic estimate, because the available studies differ largely in their approach to defining exposure metrics (e.g. chosen cut-off of distance; type of roads selected, etc.). While living close to busy roads was positively associated with asthma prevalence in children from both American [36-38] and European studies. [12, 16, 39-41] the local urban layout, meteorology, fuel type, vehicle fleet, or population susceptibility may differ, and

potentially influence the exposure-response relationship. Thus, extrapolation across regions for near-roadway traffic-related exposure is particularly delicate. Considering the lack of data, and to remain consistent with our previous methodological papers, we chose to keep the McConnell et al. 2006 risk estimate in our analysis, derived for Southern Californian children. [23] In the U.S. – in particular the Californian communities studied previously – street canyons are far less common, and the distance from traffic lanes to homes is generally much larger, than in typical European cities. Near-road traffic-related pollution levels are likely higher in Europe relative to the US, and our use of methodology developed in the U.S. may underestimate the health impact in Europe.

Moreover, we did not consider the impact of other chronic and acute outcomes also potentially associated with near-road traffic-related pollution and/or regional urban pollution, such as asthma in adults, chronic pulmonary obstructive disease, cognitive development in children, or reproductive outcomes. [5, 42] Asthma is most likely under-diagnosed, thus the prevalence used to derive attributable cases may be too low.[43] Also, we may also have ignored population exposure to traffic pollutants along smaller streets which might also have high levels of traffic-related pollutants, particularly diesel emissions, in European cities where street canyons prevail, such as Barcelona. Therefore, we believe that our approach underestimates the health impact of near-road traffic related pollution in Europe.

Another limitation of our study is related to using traffic proximity as a proxy for exposure to traffic-related pollution. One perspective suggests that such an approach lacks proper adjustments for socio-economic factors or other indirect confounders

associated with proximity to busy roads, resulting in biased estimates. A contrasting perspective holds that distance-outcome risk functions are less biased than others, because they better adjust for non-measurable confounders within the exposure term. [5]

We used residential proximity to traffic to represent long-term exposure to near-road traffic-related pollution because this metric is applicable to impact analyses across several cities. Studies do show association between chronic diseases and small-scale apportioned traffic-related pollutants (such as NO_2, $PM_{2.5,}$ and soot) [5, 44], however, in order to incorporate this data into impact assessments, more detailed information is needed concerning the spatial distribution of pollutants. Such data is not yet available for a large number of cities. In contrast, the spatial distribution of people and traffic can be studied relatively easily and cheaply. Therefore, using residential proximity to traffic to represent long-term exposure to near-road traffic-related pollution represents an effective practical approach.

Nonetheless, our choice of proxy does not reflect the variability of the concentration and toxicity of pollution which may exist within and across cities, due to differences in meteorology, type of fleet, street canyon effects, and predominant fuel use.[5] For example, we emphasize that traffic emissions are a major source of the pollutants used in our model for urban background pollution, however, the fraction attributable to traffic varies across cities. More relevant scenarios could also be explored if more data related to CRFs and spatial models of source specific pollutants was available.

In our analysis, the level of detail available to develop exposure distributions and other input data varied across cities. Our sensitivity analysis showed that the large confidence

interval around the chronic CRFs drives most of the uncertainty around the point estimates (results not shown), nonetheless, our experience calls for better integration of future environmental and health monitoring systems to obtain health assessments with fewer uncertainties.

In conclusion, we believe that it is important to move beyond traditional health impact assessments, largely focused on mortality, and expand our approach to include the potential health impacts of near road traffic-related pollution on morbidity. Despite uncertainty and limitations, our results indicate that near-road traffic related pollution may be responsible for a large but preventable burden of chronic diseases and related acute morbidities in urban areas.

Acknowledgements

APHEKOM project (Improving Knowledge and Communication for Decision Making on Air Pollution and Health in Europe) was funded jointly by the European Commission's Programme on Community Action in the Field of Public Health (2003-2008) under Grant Agreement No. 2007105. The huge amount of work behind the APHEKOM project is the fruit of the generous and constructive input from all the members of the APHEKOM network and in particular to all our local city partners. For this study, we would like to additionally acknowledge Marisa Estarlich, from Center for Public Health Research (CSISP), Valencia and Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP), Teresa Martín from Dirección Territorial de Sanidad de Bizkaia.

Bilbao. Basque Country, Spain.and Natalia Valero from the Agencia de Salut Publica de Barcelona. We thank Amena Briét from Swiss Tropical and Public Health Institute and Bruno Schull for revising the English syntax and editing the manuscript.

REFERENCES

1. Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, et al. Public-health impact of outdoor and traffic-related air pollution: A european assessment. *Lancet*. 2000;356:795-801
2. Ballester F, Medina S, Boldo E, Goodman P, Neuberger M, Iniguez C, et al. Reducing ambient levels of fine particulates could substantially improve health: A mortality impact assessment for 26 european cities. *J Epidemiol Community Health*. 2008;62:98-105
3. Anderson H, Favarato G, Atkinson R. Long-term exposure to air pollution and the incidence of asthma: Meta-analysis of cohort studies. *Air Quality, Atmosphere & Health*. 2010:1-10
4. Anderson H, Favarato G, Atkinson R. Long-term exposure to outdoor air pollution and the prevalence of asthma: Meta-analysis of multi-community prevalence studies. *Air Quality, Atmosphere & Health*. 2010:1-12
5. HEI. Health effect institute (hei) panel on the health effects of traffic-related air pollution. May 2009. Traffic-related air pollution: A critical review of the literature on emissions, exposure, and health effects. Hei special report 17. Health effects institute. Boston, mass. 2009
6. CAFE. Clean air for europe (cafe). Commission staff working paper. The communication on thematic on air pollution and the directive on "ambient ait quality and cleaner air for europe. Impact assessment. Com(2005)446 final. Available at: Http://ec.Europa.Eu/environment/air/cafe/index.Htm. 2005
7. EPA. United states environmental protection agency. Regulatory impact analysis. 2006 national ambient air quality standards for particle pollution. Available at [http://www.Epa.Gov/ttn/ecas/ria.Html], last accessed july 20, 2009. 2006
8. Cho SH, Tong H, McGee JK, Baldauf RW, Krantz QT, Gilmour MI. Comparative toxicity of size-fractionated airborne particulate matter collected at different distances from an urban highway. *Environ Health Perspect*. 2009;117:1682-1689
9. Hazenkamp-von Arx ME, Schindler C, Ragettli MS, Kunzli N, Braun-Fahrlander C, Liu LJ. Impacts of highway traffic exhaust in alpine valleys on the respiratory health in adults: A cross-sectional study. *Environ Health*. 2011;10:13
10. Hoffmann B, Moebus S, Mohlenkamp S, Stang A, Lehmann N, Dragano N, et al. Residential exposure to traffic is associated with coronary atherosclerosis. *Circulation*. 2007;116:489-496
11. Hoffmann B, Moebus S, Stang A, Beck EM, Dragano N, Mohlenkamp S, et al. Residence close to high traffic and prevalence of coronary heart disease. *Eur Heart J*. 2006;27:2696-2702
12. Janssen NA, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, et al. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in dutch schoolchildren. *Environ Health Perspect*. 2003;111:1512-1518

13. Modig L, Jarvholm B, Ronnmark E, Nystrom L, Lundback B, Andersson C, et al. Vehicle exhaust exposure in an incident case-control study of adult asthma. *Eur Respir J*. 2006;28:75-81
14. Morgenstern V, Zutavern A, Cyrys J, Brockow I, Gehring U, Koletzko S, et al. Respiratory health and individual estimated exposure to traffic-related air pollutants in a cohort of young children. *Occup Environ Med*. 2007;64:8-16
15. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environ Health Perspect*. 2007;115:53-57
16. Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF, et al. Road traffic and adverse effects on respiratory health in children. *Bmj*. 1993;307:596-600
17. Kunzli N, Perez L, Lurmann F, Hricko A, Penfold B, McConnell R. An attributable risk model for exposures assumed to cause both chronic disease and its exacerbations. *Epidemiology*. 2008;19:179-185
18. Kunzli N, Perez L, von Klot S, Baldassarre D, Bauer M, Basagana X, et al. Investigating air pollution and atherosclerosis in humans: Concepts and outlook. *Prog Cardiovasc Dis*. 2011;53:334-343
19. Perez L, Künzli N, Avol E, Hricko A, Lurmann F, Nicholas E, et al. Global goods movement and the local burden of childhood asthma in southern california. . *Am J Public Health. In press*. 2009
20. Zhang J, Yu KF. What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *Jama*. 1998;280:1690-1691
21. WHO. World health organization. Who air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment. Who/sde/phe/oeh/06.02. 2005
22. Schikowski T, Sugiri D, Ranft U, Gehring U, Heinrich J, Wichmann HE, et al. Long-term air pollution exposure and living close to busy roads are associated with copd in women. *Respir Res*. 2005;6:152
23. McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, et al. Traffic, susceptibility, and childhood asthma. *Environ Health Perspect*. 2006;114:766-772
24. Rioux CL, Gute DM, Brugge D, Peterson S, Parmenter B. Characterizing urban traffic exposures using transportation planning tools: An illustrated methodology for health researchers. *J Urban Health*. 2010;87:167-188
25. Brandt SJ, Perez L, Kunzli N, Lurmann F, McConnell R. Costs of childhood asthma due to traffic-related pollution in two california communities. *Eur Respir J*. 2012
26. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, et al. Explaining the decrease in u.S. Deaths from coronary disease, 1980-2000. *N Engl J Med*. 2007;356:2388-2398
27. Unal B, Critchley JA, Capewell S. Explaining the decline in coronary heart disease mortality in england and wales between 1981 and 2000. *Circulation*. 2004;109:1101-1107
28. Anderson HR, Ruggles R, Pandey KD, Kapetenakis V, Brunekreef B, Lai CK, et al. Ambient particulate pollution and the world-wide prevalence of asthma, rhinoconjunctivitis and eczema in children: Phase one of the international study of asthma and allergies in childhood (isaac). *Occup Environ Med*. 2009
29. Salam MT, Gauderman WJ, McConnell R, Lin PC, Gilliland FD. Transforming growth factor- 1 c-509t polymorphism, oxidant stress, and early-onset childhood asthma. *Am J Respir Crit Care Med*. 2007;176:1192-1199
30. Carlsten C, Dybuncio A, Becker A, Chan-Yeung M, Brauer M. Traffic-related air pollution and incident asthma in a high-risk birth cohort. *Occup Environ Med*. 2011;68:291-295
31. Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the american heart association. *Circulation*. 2010;121:2331-2378
32. Nawrot TS, Perez L, Kunzli N, Munters E, Nemery B. Public health importance of triggers of myocardial infarction: A comparative risk assessment. *Lancet*. 2011;377:732-740
33. Rockhill B, Newman B, Weinberg C. Use and misuse of population attributable fractions. *Am J Public Health*. 1998;88:15-19
34. Levine B. What does the population attributable fraction mean? *Prev Chronic Dis*. 2007;4:A14
35. Rothman KJ, Greenland S. Causation and causal inference in epidemiology. *Am J Public Health*. 2005;95 Suppl 1:S144-150

36. Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, et al. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology*. 2005;16:737-743
37. Gordian ME, Haneuse S, Wakefield J. An investigation of the association between traffic exposure and the diagnosis of asthma in children. *J Expo Sci Environ Epidemiol*. 2006;16:49-55
38. Kim JJ, Huen K, Adams S, Smorodinsky S, Hoats A, Malig B, et al. Residential traffic and children's respiratory health. *Environ Health Perspect*. 2008;116:1274-1279
39. Lewis SA, Antoniak M, Venn AJ, Davies L, Goodwin A, Salfield N, et al. Secondhand smoke, dietary fruit intake, road traffic exposures, and the prevalence of asthma: A cross-sectional study in young children. *Am J Epidemiol*. 2005;161:406-411
40. Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *Eur Respir J*. 2003;21:956-963
41. van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res*. 1997;74:122-132
42. Guxens M, Aguilera I, Ballester F, Estarlich M, Fernandez-Somoano A, Lertxundi A, et al. Prenatal exposure to residential air pollution and infant mental development: Modulation by antioxidants and detoxification factors. *Environ Health Perspect*. 2011
43. Lai CK, Beasley R, Crane J, Foliaki S, Shah J, Weiland S. Global variation in the prevalence and severity of asthma symptoms: Phase three of the international study of asthma and allergies in childhood (isaac). *Thorax*. 2009;64:476-483
44. Gehring U, Wijga AH, Brauer M, Fischer P, de Jongste JC, Kerkhof M, et al. Traffic-related air pollution and the development of asthma and allergies during the first 8 years of life. *Am J Respir Crit Care Med*. 2010;181:596-603
45. Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. Short-term effects of pm10 and no2 on respiratory health among children with asthma or asthma-like symptoms: A systematic review and meta-analysis. *Environ Health Perspect*. 2010;118:449-457
46. Andersen ZJ, Wahlin P, Raaschou-Nielsen O, Ketzel M, Scheike T, Loft S. Size distribution and total number concentration of ultrafine and accumulation mode particles and hospital admissions in children and the elderly in copenhagen, denmark. *Occup Environ Med*. 2008;65:458-466
47. Atkinson RW, Anderson HR, Sunyer J, Ayres J, Baccini M, Vonk JM, et al. Acute effects of particulate air pollution on respiratory admissions: Results from aphea 2 project. Air pollution and health: A european approach. *Am J Respir Crit Care Med*. 2001;164:1860-1866
48. Peters A, von Klot S, Heier M, Trentinaglia I, Cyrys J, Hormann A, et al. Particulate air pollution and nonfatal cardiac events. Part i. Air pollution, personal activities, and onset of myocardial infarction in a case-crossover study. *Res Rep Health Eff Inst*. 2005:1-66; discussion 67-82, 141-148
49. D'Ippoliti D, Forastiere F, Ancona C, Agabiti N, Fusco D, Michelozzi P, et al. Air pollution and myocardial infarction in rome: A case-crossover analysis. *Epidemiology*. 2003;14:528-535
50. Lanki T, Pekkanen J, Aalto P, Elosua R, Berglind N, D'Ippoliti D, et al. Associations of traffic related air pollutants with hospitalisation for first acute myocardial infarction: The heapss study. *Occup Environ Med*. 2006;63:844-851
51. Poloniecki JD, Atkinson RW, de Leon AP, Anderson HR. Daily time series for cardiovascular hospital admissions and previous day's air pollution in london, uk. *Occup Environ Med*. 1997;54:535-540
52. Halonen JI, Lanki T, Yli-Tuomi T, Tiittanen P, Kulmala M, Pekkanen J. Particulate air pollution and acute cardiorespiratory hospital admissions and mortality among the elderly. *Epidemiology*. 2009;20:143-153
53. Larrieu S, Jusot JF, Blanchard M, Prouvost H, Declercq C, Fabre P, et al. Short term effects of air pollution on hospitalizations for cardiovascular diseases in eight french cities: The psas program. *Sci Total Environ*. 2007;387:105-112
54. Le Tertre A, Medina S, Samoli E, Forsberg B, Michelozzi P, Boumghar A, et al. Short-term effects of particulate air pollution on cardiovascular diseases in eight european cities. *J Epidemiol Community Health*. 2002;56:773-779
55. Oudin A, Stromberg U, Jakobsson K, Stroh E, Bjork J. Estimation of short-term effects of air pollution on stroke hospital admissions in southern sweden. *Neuroepidemiology*. 2009;34:131-142

Table 1. Concentration-Response Functions (CRFs) used in the evaluation for asthma and related acute morbidities

Outcome	**Unit of CRF**	**Location, age study participants**	**Definition of outcome and/or frequency outcome in study**	**CRF (95% CI)#**	**Study**
Prevalence of underlying chronic diseases					
Asthma prevalence	Residence ≤ 75m of busy roads defined as freeways, other highways, and arterial roads Prevalence exposure=15%	Southern California, 5-7y,	For long-term residents-prevalent asthma. 14%, prevalence, defined as controller medications for asthma in the previous year, diagnosed lifetime asthma with any wheeze in the previous year, severe wheeze in the previous 12	1.64 (1.1-2.44)	23
Exacerbations					
Asthma symptoms among children symptomatic for or diagnosed with asthma	PM_{10} per 10 μg/m3	Meta-analytic review, children	--	1.028 (1.016-1.039)	45
Hospital admission for asthma	PM_{10} per 5 μg/m3,	5-18, Copenhagen, Danemark	ICD10: J45, 46	1.07 (1.00-1.15) (transformed from PM_{25})	46
	PM_{10} per 10 μg/m3	0-14 Aphea study, Europe	ICD9: 490-496	1.012 (1.001-1.023)	47
			Meta-analytic estimate, PM_{10} per 1 ug/m3	1.0013 (1.0002-1.0024)	--

CRF: Concentration-response function; ICD: international classification of diseases.

#When estimate reported as odds ratios (ORs), ORs were corrected with the formula CRF/[1+It(CRF-1)], where It is estimated as the frequency of the outcome in the population [20].

Table 2. Concentration-Response Functions (CRFs) used in the evaluation in relation to coronary heart disease and related acute morbidity

Outcome	Unit of CRF	Location, age study participants	Definition of outcome and/or frequecny	CRF (95% CI)#	Study
Prevalence of underlying chronic diseases					
CHD prevalence	Busy road≤150 m of home (defined as autobahn and federal highways: 10-110∗10^3 veh/day)	North-Rhine-Westphalia, Germany, 45-74y	7.1%, prevalence, defined as self-reported history of MI or application of a coronary stent or angioplasty or bypass	1.85 (1.21-2.84)	11
Exacerbations					
Non-fatal MI	$PM_{2.5}$ per 10 µg/m3	Ausburg 1999-2001, >18	WHO algorithm (includes evaluation chest pain, Q waves examination, levels creatine kinase and other)	1.105 (0.987-1.226)	48
			Transformed to PM_{10} per 1 ug/m3	1.005 (0.999-1.012)	--
Hospital admission for MI	NO_2 per 10 µg/m^3	Rome 1995-1997, >18	ICD9-410	1.026 (1.002-1.052)	49
	NO_2 per 8 µg/m^3	5 european studies, >35y	ICD9: 410	1.005 (1.00-1.01)	50
	NO_2 per 57.3 µg/^{m3}	London 1987-1994, all ages	ICD9:410	1.0274 (1.0084-1.0479)	51
		--	Meta-analytic estimate, NO_2 per 1 ug/m3	1.0005 (1.0002-1.0008)	--
Hospital admission for stroke	PM_{10} per 12 µg/m3	Helsinki 1998-2004, >=65	ICD10: I60-61 and I63-I64	0.9922 (0.069-1.016)	52
	PM_{10} per 10 µg/m^3	9 French cities, >=65	ICD10: I60-I64; G45-G46	1.008 (0.991-1.025)	53
	PM_{10} per 10 µg/m^3	Aphea, 8 european cities, all ages	ICD9: 430-8	1.000 (0.997-1.003)	54
	PM_{10} per 25 µg/m^3	Southwestern Sweden, median age 70y	Ischemic stroke	1.13 (1.04-1.22)	55
			Meta-analytic estimate, PM_{10} per 1 ug/m3	1.0007 (0.9993-1.0020)	--

CRF: Concentration-response function; CHD: coronary heart disease; MI: Myocardial infarction; ICD: international classification of diseases.

#When estimate reported as odds ratios (ORs), ORs were corrected with the formula CRF/[1+It(CRF-1)], where It is estimated as the frequency of the outcome in the population [20].

Table 3. Summary of population and heath baseline frequencies

Outcome	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Sevilla	Stockholm	Valencia	Vienna	10 cities
Asthma											
Total population aged 0-17	261241 (2008)#	49327 (2006)	227402 (2007)	44734 (2007)#	52255 (2002)	439543 (2009)	140373 (2007)#	264960 (2007)	117040 (2001)	291213 (2007)	1890000
Fraction with asthma (asthma ever)##	11.8%	21.3%	7.3%	12.6%	29.2%	12.6%	13.0%	9.3%	11.0%	5.8%	12.8%
Fraction with symptoms of asthma among those with current wheeze##	29.0%	37.0%	39.2%	37.1%	34.2%	32.9%	37.1%	35.6%	36.5%	33.1%	38.0%
Total number of hospitalizations for asthma (ICD-10: J45-J46)	98 (2008)	230 (2006)	356 (2006)	39 (2008)	100 (2007)	482 (2008)	28 (2008)	219#	40 (2002)	149 (2007)	1702
Coronary heart disease (CHD)											
Total population, aged ≥65	249945 (2008)	76113 (2006)	152412 (2007)	42045 (2007)	41314 (2002)	597887 (2009)	111570 (2007)	183329 (2007)	129190 (2001)	269936 (2007)	1850000
Fraction with CHD†	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%	7.1%
Fraction with CHD with non-fatal MI†	2.3% (2006)	2.5%	2.5%	2.5%	2.5%	2.5%	2.5%	2.5%	2.5%	2.7% (2007)	2.5%
Number of hospitalizations for MI (ICD10: I20-I21)	1370 (2008)	416 (2006)	806 (2006)	206 (2008)	469 (2007)	4705 (2008)	651 (2008)	3590‡	908‡	2860 (2007)	15981
Total Number of hospitalizations for stroke (ICD10: I60-I61;I63-I64)	1960 (2008)	578 (2006)	1523 (2006)	266 (2008)	231 (2007)	3374 (2008)	668 (2008)	2513‡	1084‡	2866 (2007)	15062

CHD: cardiovascular disease; MI: Myocardial Infarction; ICD: international classification of diseases. Number in parenthesis is year for which data applies

#0-19

##extrapolated from prevalence given in[43].The prevalence of symptoms of severe asthma in this study is defined as >or=4 attacks of wheeze or >or=1 night per week sleep disturbance from wheeze or wheeze affecting speech in the past 12 months.

†if no year indicated, extrapolated from prevalence given in CRF study [11];

‡no data for the ICD code was available, extrapolated using ratios seen in other cities

Table 4. City specific summaries of population distribution in two distance based buffers around busy roads (i.e. >10000 vehicles per day) and annual mean concentrations of PM_{10} and NO_2 at urban background stations.

Indicator	Unit	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Sevilla	Stockholm##	Valencia	Vienna	10 cities#
Traffic proximity (%)	year	2007	2006	2002	2008	2006	2008	2004	2004	2007-2008	2006	--
	≤75m	56%	29%	37%	14%	23%	22%	20%	14%	44%	36%	31%
	≤150m	77%	59%	64%	28%	47%	43%	38%	30%	71%	62%	53%

Urban background pollution	year	2008	2008	2007	2008	2007	2009	2008	2007	2003	2007	--
PM_{10} annual average ($\mu g/m^3$)		33	27	29	34	32	37	41	17	27	25	30.3
NO_2 annual average ($\mu g/m^3$)		36	29	38	31	28	61	29	13	51	32	39.4

#Population weighted concentrations
##Was developed for the population of Stockholm city only (population of 790,000) while burden is for Stockholm greater area (population of 1.3 million)

Table 5. Estimated cases and percent (95% CI) of lifetime childhood asthma and prevalent coronary heart disease in older adults attributable to near-road traffic-related pollution for 10 cities in Europe#

City	Asthma (0-17)			Coronary Heart Disease (≥65)		
	Estimated with chronic outcome	Attributed to near-road traffic-related pollution##		Estimated with chronic outcome	Attributed to near-road traffic-related pollution##	
		prevalent cases	prevalent fraction		prevalent cases	prevalent fraction
Barcelona	30690	6900 (1400; 11500)	23% (5%; 38%)	17740	6400 (2200; 9000)	36% (13%; 54%)
Bilbao	10500	1200 (200; 2100)	12% (2%; 20%)	5400	1600 (500; 1500)	31% (10%; 47%)

Brussels	16480	2800 (500; 5100)	17% (3%; 31%)	10820	3500 (1100; 5500)	32% (11%; 49%)
Granada	5630	300 (0; 700)	7% (1%; 13%)	2980	500 (100; 600)	17% (5%; 30%)
Ljubljana	15250	1200 (200; 2100)	8% (2%; 14%)	2930	700 (200; 1000)	26% (8%; 42%)
Rome	57360	5700 (1000; 10700)	10% (2%; 19%)	42440	10300 (3200; 8600)	24% (8%; 40%)
Sevilla	17680	1600 (200; 3000)	9% (2%; 17%)	7920	1700 (500; 2600)	22% (7%; 37%)
Stockholm	24640	1700 (300; 3400)	7% (1%; 14%)	13010	2300 (700; 3600)	18% (5%; 31%)
Valencia	12810	2400 (400; 4100)	19% (4%; 33%)	9170	3100 (1100; 3800)	35% (12%; 52%)
Vienna	16890	2800 (500; 5200)	17% (3%; 31%)	19160	6000 (2000; 9200)	31% (11%; 48%)
10 Cities	240730	33200 (6200; 59600)	14% (3%; 25%)	131610	37200 (12100; 56600)	28% (9%; 44%)

#Truncated to zero, when negative values obtained. Estimates rounded to lowest 10th, 1000, or 10,000.

##Distance to busy road depends on outcome: within75m asthma, within 150m for coronary heart disease

Table 6. Estimated yearly exacerbations of childhood asthma (95% CI) attributable to air pollution for 10 cities in Europe#

City	Estimated with outcome	Exacerbations attributable to air pollution among those with asthma due to			Exacerbation attributable to other causes among those with asthma due to near-road traffic-related pollution	Total attributable outcome due to air pollution	
		near-road traffic-related pollution	other causes	total		cases	% with [1] as denominator

Referenced in text	[1]	[2]	[3]	[4]	[5]	[6]	
Episodes symptoms asthma							
Barcelona	8910	39 (4; 85)	135 (55; 235)	175 (75; 285)	1900 (200; 3600)	2100 (400; 3800)	24% (5%; 43%)
Bilbao	3880	9 (0; 24)	100 (40; 170)	110 (45; 185)	400 (0; 900)	500 (0; 1000)	14% (1%; 28%)
Brussels	6460	16 (1; 37)	75 (25; 135)	90 (30; 155)	1100 (100; 2100)	1200 (200; 2200)	19% (4%; 34%)
Granada	2090	3 (0; 8)	45 (15; 75)	45 (15; 80)	100 (0; 200)	100 (0; 300)	9% (3%; 17%)
Ljubljana	5660	9 (-1.2; 25)	105 (40; 180)	115 (40; 195)	400 (-58.8; 1000)	500 (0; 1100)	11% (1%; 23%)
Rome	18870	61 (4; 145)	535 (220; 890)	595 (250; 980)	1800 (100; 3800)	2400 (700; 4500)	13% (4%; 24%)
Sevilla	6560	21 (1; 53)	205 (80; 350)	230 (85; 385)	600 (0; 1200)	800 (200; 1500)	13% (4%; 23%)
Stockholm	8750	0 (0; 0)	0 (0; 0)	0 (0; 0)	600 (0; 1300)	600 (0; 1300)	7% (1%; 16%)
Valencia	4670	10 (0; 22)	40 (15; 75)	50 (20; 85)	800 (100; 1600)	900 (100; 1700)	20% (4%; 37%)
Vienna	5590	8 (1; 19)	40 (15; 70)	50 (20; 80)	900 (100; 1800)	1000 (200; 1800)	18% (4%; 33%)
10 cities	91590	220 (15; 520)	1365 (505; 2375)	1585 (590; 2690)	12400 (1200; 25000)	14000 (2800; 26800)	15% (3%; 29%)
Hospital admissions							
Barcelona	90	0.28 (0.001; 0.69)	0.9 (0.1; 1.9)	1.2 (0.1; 2.3)	21 (2; 38)	22 (4; 40)	23% (4%; 41%)
Bilbao	230	0.12 (0 ;0.36)	1.3 (0.1; 2.5)	1.5 (0.2; 2.7)	8 (0; 18)	9 (0; 20)	4% (0.1%; 9%)
Brussels	350	0.66 (0.01; 1.66)	3.1 (0.3; 6)	3.8 (0.4; 7.1)	61 (10; 113)	65 (14; 117)	18% (4%; 33%)
Granada	30	0.04 (0; 0.12)	0.6 (0; 1.1)	0.6 (0; 1.2)	2 (0; 5)	3 (0; 6)	8% (2%; 16%)
Ljubljana	100	0.12 (0; 0.36)	1.3 (0.1; 2.5)	1.5 (0.2; 2.7)	8 (0; 18)	9 (0; 20)	10% (0.4%; 20%)
Rome	480	1.07 (0; 2.82)	9.3 (1.2; 17.5)	10.4 (1.4; 19.3)	48 (4; 96)	58 (14; 108)	12% (3%; 22%)
Sevilla	20	0.06 (0; 0.18)	0.6 (0; 1.2)	0.7 (0.1; 1.3)	2 (0; 5)	3 (0; 6)	12% (3%; 22%)
Stockholm	210	0 (0; 0)	0 (0; 0)	0 (0; 0)	16 (2; 33)	16 (2; 33)	7% (1%; 15%)
Valencia	40	0.06 (0; 0.15)	0.2 (0; 0.5)	0.3 (0; 0.6)	7 (0; 13)	7 (1; 14)	20% (3%; 35%)
Vienna	140	0.16 (0; 0.39)	0.7 (0.1; 1.4)	0.9 (0.1; 1.7)	25 (4; 46)	26 (5; 47)	18% (4%; 32%)
10 cities	1740	3.1 (0; 8.1)	15 (0; 35)	20 (0; 40)	230 (20; 460)	260 (40; 480)	15% (3%; 28%)

#Truncated to zero, when negative values obtained. Estimates rounded to lowest 10th, 1000, or 10,000

Table 7. Estimated yearly exacerbations of coronary heart disease (95% CI) attributable to air pollution for 10 cities in Europe#

Exacerbations	Estimated exacerbations	Exacerbations attributable to air pollution among those with coronary heart disease due to			Exacerbations attributable to other causes among those with coronary heart disease due to near-road traffic-related pollution	Total attributable exacerbations due to air pollution
		near-road traffic-related pollution	Other	total		
Referenced in text	[1]	[2]	[3]	[4]	[5]	[6]
Non-fatal myocardial events	3260	40 (0; 120)	110 (0; 275)	155 (0; 365)	870 (290; 1460)	1025 (420; 1655)
in % of [1]		1% (0%; 4%)	3% (0%; 8%)	5% (0%; 11%)	27% (9%; 45%)	31% (13%; 51%)
Hospital admission for myocardial infarction	15980	40 (0; 80)	100 (0; 185)	140 (0; 245)	4430 (1510; 7425)	4575 (1640; 7575)
in % of [1]		0.3% (0%; 0.5%)	1% (0%; 1%)	1% (0%; 2%)	28% (9%; 46%)	29% (10%; 47%)
Hospital admission for stroke	15060	15 (0; 630)	30 (0; 1535)	50 (0; 2105)	4200 (1410; 7125)	4250 (930; 7425)
in % of [1]		0.13% (0%; 4%)	0.2% (0%; 10%)	0.3% (0%; 13%)	26% (9%; 45%)	27% (6%; 46%)

#Truncated to zero, when negative values obtained. Estimates rounded to nearest multiplier