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

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ABSTRACT   Recent epidemiological research suggests that near road traffic-related pollution may cause chronic disease, as well as exacerbation of 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.

Derivation of attributable cases required combining concentration–response function 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.

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.

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

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APHEKOM network: pollution near busy roads is a large, preventable source of chronic disease and acute episode triggers http://ow.ly/mkpRH

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 unravelling the effects of air pollution on health. Two findings in particular have led 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 hospitalisations, bronchitis symptoms, myocardial infarctions or restricted activity days) [1, 6, 7]. However, chronic morbidity due to air pollution also heavily impacts health and the healthcare system in addition to acute effects. Thus, the burden of chronic disease due to air pollution has been grossly underestimated.

Secondly, 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}, $PM_{2.5}$ (particulate matter up to 10 μm or 2.5 μm in size) 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 characterise 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 underestimated 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 (PAFs) 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 per day, was selected as a marker of long-term exposure to near-road traffic-related pollution (tables 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 hospitalisation 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 the odds ratios of published studies, to avoid overestimating the true relative risks in our final estimations [24]. 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: 75 m for asthma and 150 m 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 per 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

TABLE 1 Concentration–response functions (CRFs) used in the evaluation for asthma and related acute morbidities

Outcome	Unit of CRF	Location, age of study participants	Definition of outcome and/or frequency outcome in study population	CRF (95% CI)[#]	Study
Prevalence of underlying chronic diseases					
Asthma prevalence	Residence ⩽75 m of busy roads defined as freeways, other high-ways, and arterial roads Prevalence exposure 15%	Southern CA, USA, 5–7 years	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 months	1.64 (1.1–2.44)	[20]
Exacerbations					
Asthma symptoms among children symptomatic for or diagnosed with asthma	PM_{10} per 10 µg·m^{-3}	Meta-analytic review, children		1.028 (1.016–1.039)	[21]
Hospital admission for asthma	PM_{10} per 5 µg·m^{-3}	Copenhagen, Denmark, 5–18 years	ICD10: J45, 46	1.07 (1.00–1.15) (transformed from $PM_{2.5}$)	[22]
	PM_{10} per 10 µg·m^{-3}	APHEA study, Europe, 0–14 years	ICD9: 490–496	1.012 (1.001–1.023)	[23]
			Meta-analytic estimate, PM_{10} per 1 µg·m^{-3}	1.0013 (1.0002–1.0024)	

PM_{10}: particulate matter up to 10 µm; ICD: international classification of diseases; $PM_{2.5}$: particulate matter up to 2.5 µm; APHEA: Air pollution and health, a European approach. [#]: when estimates were reported as odds ratios, odds ratios were corrected with the formula CRF/(1+It(CRF-1)), where It is estimated as the frequency of the outcome in the population [24].

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 of study participants	Definition of outcome and/or frequency outcome in study population	CRF (95% CI)[#]	Study
Prevalence of underlying chronic diseases					
CHD prevalence	Busy road $\leqslant$150 m from home (defined as autobahn and federal highways: 10-110 $\times 10^3$ vehicles per day) Prevalence exposure 31.4%	North-Rhine-Westphalia, Germany, 45–74 years	7.1%, prevalence, defined as self-reported history of MI or application of a coronary stent or angioplasty or bypass surgery	1.85 (1.21–2.84)	[11]
Exacerbations					
Non-fatal MI	$PM_{2.5}$ per 10 $\mu g \cdot m^{-3}$	Ausburg 1999–2001, >18 years	WHO algorithm (includes evaluation chest pain, Q waves examination, levels creatine kinase and other)	1.105 (0.987–1.226)	[25]
			Transformed to PM_{10} per 1 $\mu g \cdot m^{-3}$	1.005 (0.999–1.012)	
Hospital admission for MI	NO_2 per 10 $\mu g \cdot m^{-3}$	Rome 1995–1997, >18 years	ICD9-410	1.026 (1.002–1.052)	[26]
	NO_2 per 8 $\mu g \cdot m^{-3}$	5 European studies, >35 years	ICD9: 410	1.005 (1.00–1.01)	[27]
	NO_2 per 57.3 $\mu g \cdot m^{-3}$	London 1987–1994, all ages	ICD9:410	1.0274 (1.0084–1.0479)	[28]
			Meta-analytic estimate, NO_2 per 1 $\mu g \cdot m^{-3}$	1.0005 (1.0002–1.0008)	
Hospital admission for stroke	PM_{10} per 12 $\mu g \cdot m^{-3}$	Helsinki 1998–2004, $\geqslant$65 years	ICD10: I60-61 and I63–I64	0.9922 (0.069–1.016)	[29]
	PM_{10} per 10 $\mu g \cdot m^{-3}$	9 French cities, $\geqslant$65 years	ICD10: I60–I64; G45–G46	1.008 (0.991-1.025)	[30]
	PM_{10} per 10 $\mu g \cdot m^{-3}$	APHEA, 8 European cities, all ages	ICD9: 430–8	1.000 (0.997–1.003)	[31]
	PM_{10} per 25 $\mu g \cdot m^{-3}$	Southwestern Sweden, median age 70 years	Ischaemic stroke	1.13 (1.04–1.22)	[32]
			Meta-analytic estimate, PM_{10} per 1 $\mu g \cdot m^{-3}$	1.0007 (0.9993–1.0020)	

CHD: coronary heart disease; MI: myocardial infarction; $PM_{2.5}$: particulate matter up to 2.5 µm; PM_{10}: particulate matter up to 10 µm; WHO: World Health Organization; ICD: international classification of diseases; APHEA: Air pollution and health, a European approach. [#]: when estimates were reported as odds ratios, odds ratios were corrected with the formula CRF/(1+It(CRF-1)), where It is estimated as the frequency of the outcome in the population [24].

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 $\mu g \cdot m^{-3}$ as the scenario reference point. For PM_{10}, this reflects the World Health Organization (WHO) guideline value [34]. For both PM_{10} and NO_2, the scenario reference point reflects an annual mean not yet reached in any of the participating

TABLE 3 Summary of population and heath baseline frequencies

Outcome	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Seville	Stockholm	Valencia	Vienna	10 cities
Asthma											
Total population aged 0-17	261 241 (2008)##	49 327 (2006)	227 402 (2007)	44 734 (2007)##	52 255 (2002)	439 543 (2009)	140 373 (2007)##	264 960 (2007)	117 040 (2001)	291 213 (2007)	1 890 000
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 hospitalisations for asthma¶	98 (2008)	230 (2006)	356 (2006)	39 (2008)	100 (2007)	482 (2008)	28 (2008)	219##	40 (2002)	149 (2007)	1702
CHD											
Total population, aged ≥65 years	249 945 (2008)	76 113 (2006)	152 412 (2007)	42 045 (2007)	41 314 (2002)	597 887 (2009)	111 570 (2007)	183 329 (2007)	129 190 (2001)	269 936 (2007)	1 850 000
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 hospitalisations for MI§	1370 (2008)	416 (2006)	806 (2006)	206 (2008)	469 (2007)	4705 (2008)	651 (2008)	3590¶¶	908¶¶	2860 (2007)	15 981
Total number of hospitalisations for stroke‡	1960 (2008)	578 (2006)	1523 (2006)	266 (2008)	231 (2007)	3374 (2008)	668 (2008)	2513¶¶	1084¶¶	2866 (2007)	15 062

Data are presented as n or % (study year). CHD: coronary heart disease; MI: myocardial infarction; ICD: international classification of diseases. #: extrapolated from prevalence given in [33], the prevalence of symptoms of severe asthma in this study is defined as ≥4 attacks of wheeze or ≥1 night per week sleep disturbance from wheeze or wheeze affecting speech in the past 12 months; ¶: ICD-10: J45-J46; +: if no year indicated, extrapolated from prevalence given in CRF study [11]; §: ICD10: I20-I21; ‡: ICD10: I60-I61;I63-I64; ##: 0–19 years; ¶¶: no data for the ICD code was available, extrapolated using ratios seen in other cities.

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 $\mu g \cdot m^{-3}$. 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 10 cities.

Results

Table 4 summarises 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 75 m of a busy road and 53% within 150 m of such roads. Across the 10 cities, the annual weighted mean for PM_{10} was 30 $\mu g \cdot m^{-3}$ and 39 $\mu g \cdot m^{-3}$ 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 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 were attributable to near-roadway traffic-related pollution (28% of all older adults with CHD, with a 95% confidence interval 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 basis. 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 1585 episodes of asthma symptoms (1.7%) and 20 asthma hospitalisations (1.1%) (table 6). 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 hospitalisations (table 6) 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 myocardial infarctions, 28% of myocardial infarction hospitalisations, and 26% of stroke hospitalisations were attributable to causes other than air pollution

TABLE 4 City-specific summaries of population distribution in two distance based buffers around busy roads (*i.e.* >10 000 vehicles per day) and annual mean concentrations of PM_{10} and NO_2 at urban background stations

Indicator	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Seville	Stockholm[#]	Valencia	Vienna	10 cities[¶]
Traffic proximity (%)											
Year	2007	2006	2002	2008	2006	2008	2004	2004	2007–2008	2006	
⩽75 m	56%	29%	37%	14%	23%	22%	20%	14%	44%	36%	31%
⩽150 m	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 $\mu g \cdot m^{-3}$	33	27	29	34	32	37	41	17	27	25	30.3
NO_2 annual average $\mu g \cdot m^{-3}$	36	29	38	31	28	61	29	13	51	32	39.4

PM_{10}: particulate matter up to 10 μm. [#]: was developed for the population of Stockholm city only (population of 790 000) while burden is for Stockholm greater area (population of 1 300 000); [¶]: population weighted concentrations.

TABLE 5 Estimated cases and per cent 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 years)			Coronary heart disease (⩾65 years)		
	Estimated with chronic outcome n	Attributed to near-road traffic-related pollution[¶]		Estimated with chronic outcome n	Attributed to near-road traffic-related pollution[¶]	
		Prevalent cases n (95% CI)	Prevalent fraction % (95% CI)		Prevalent cases n (95% CI)	Prevalent fraction % (95% CI)
Barcelona	30 690	6900 (1400–11 500)	23 (5–38)	17 740	6400 (2200–9000)	36 (13–54)
Bilbao	10 500	1200 (200–2100)	12 (2–20)	5400	1600 (500–1500)	31 (10–47)
Brussels	16 480	2800 (500–5100)	17 (3–31)	10 820	3500 (1100–5500)	32 (11–49)
Granada	5630	300 (0–700)	7 (1–13)	2980	500 (100–600)	17 (5–30)
Ljubljana	15 250	1200 (200–2100)	8 (2–14)	2930	700 (200–1000)	26 (8–42)
Rome	57 360	5700 (1000–10 700)	10 (2–19)	42 440	10 300 (3200–8600)	24 (8–40)
Seville	17 680	1600 (200–3000)	9 (2–17)	7920	1700 (500–2600)	22 (7–37)
Stockholm	24 640	1700 (300–3400)	7 (1–14)	13 010	2300 (700–3600)	18 (5–31)
Valencia	12 810	2400 (400–4100)	19 (4–33)	9170	3100 (1100–3800)	35 (12–52)
Vienna	16 890	2800 (500–5200)	17 (3–31)	19 160	6000 (2000–9200)	31 (11–48)
10 cities	240 730	33 200 (6200–59 600)	14 (3–25)	131 610	37 200 (12 100–56 600)	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: within 75 m for asthma, within 150 m for coronary heart disease.

among those who suffer CHD due to near-road traffic-related pollution (table 6). 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 75 m of busy roads, and 53% within 150 m. This distribution is consistent with results from other epidemiological studies in Europe [11, 35]. Notably, however, this distribution is very different from urban areas in the USA, such as Long Beach and Riverside, where a lower percentage of the population lives in close proximity to busy roads [20, 36]. 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, PM10 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 as 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 [37].

Several studies in high income countries estimate that as much as 50% of the reduction in deaths from coronary diseases over the past 20 years can be attributed to preventive measures, such as lowering cholesterol levels, smoking prevalence, blood pressure, and physical inactivity [38, 39]. 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, these 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

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	%[¶]
Episodes of symptoms of 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	18 870	61 [4–145]	535 [220–890]	595 [250–980]	1800 [100–3800]	2400 [700–4500]	13 [4–24]
Seville	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	91 590	220 [15–520]	1365 [505–2375]	1585 [590–2690]	12 400 [1200–25 000]	14 000 [2800–26 800]	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]
Seville	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 [1–14]	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 10000. [¶]: % calculated with ''estimated with outcome'' as the denominator.

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, 40]. 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 that is very hard to explain with uncontrolled biases or confounding factors [41]. 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 [37]. Furthermore, the Dutch birth cohort study followed children up until 8 years old, and found no evidence supporting postponed onset, validating our assumption [42].

In the case of myocardial infarction, there is also a large amount of evidence that acute events are related to high daily levels of air pollution [5, 43, 44]. 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. However, 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 myocardial infarction, such as oxidative stress and inflammation [43]. 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 myocardial infarction 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 multi-causal model implies that it is sufficient to eliminate or remove any one of the factors contributing to the disease in order to prevent it [45]. 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 [46, 47]. 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 behavioural 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-analytical estimate, because the available studies differ largely in their approach to defining exposure metrics (e.g. chosen cut-off of distance and type of roads selected). While living close to busy roads was positively associated with asthma prevalence in children from both American [48–50] and European studies [12, 16, 51–53] the local urban layout, meteorology, fuel type, vehicle fleet or population susceptibility may differ, and potentially influence the exposure-response

TABLE 7 Estimated yearly exacerbations of coronary heart disease 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		
Non-fatal myocardial events	3260	40 (0–120)	110 (0–275)	155 (0–365)	870 (290–1460)	1025 (420–1655)
Proportion of estimated exacerbations %		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)
Proportion of estimated exacerbations %		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)
Proportion of estimated exacerbations %		0.13 (0–4)	0.2 (0–10)	0.3 (0–13)	26 (9–45)	27 (6–46)

Data are presented as n, n (95% CI) or % (95% CI) of the number of estimated exacerbations. [#]: truncated to zero, when negative values obtained. Estimates rounded to nearest multiplier.

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. [20] 2006 risk estimate in our analysis, derived for Southern Californian children. In the USA, 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 USA, and our use of methodology developed in the USA 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, 54]. Asthma is most likely under-diagnosed, thus the prevalence used to derive attributable cases may be too low [33]. We may 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 socioeconomic 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, 55]; 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 that 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 emphasise 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.

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