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Aphekom

Improving Knowledge and Communication for Decision Making on Air Pollution and Health in Europe

Guidelines of methods

for

Integrating chronic effects of local-traffic pollution in the air pollution health impact methodology

(WP4)

Deliverable D3

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Figure 1. The burden of air pollution assuming a causal role of air pollution in both disease onset and exacerbations.

Appendix

Appendix 1: Summary of studies on the association between traffic proximity and health outcomes (up to December 2009)

Appendix 2: Summary of studies on the association between traffic related pollutants and health outcomes

Appendix 3: Step by step example to derive the percentage of people living at proximity to “major” roads

Introduction

Many chronic diseases are the product of an underlying pathologic condition and superimposed acute complications (exacerbations). Numerous epidemiological studies have found associations between health outcomes related to exacerbations and air pollution [1-7]. Although still limited, there is sufficient evidence to suggest that air pollution may contribute to some of the chronic pathologies that underlie those exacerbations. For example in the case of childhood asthma, not only exacerbations are triggered by air pollution but most likely the development of the chronic disease is also caused by some air pollutants, in particular the local traffic-related pollutants that occur in high concentrations along busy roads. Pollutants appear to contribute to the development of coronary heart disease (CHD) and possibly to COPD in adults as well. For both chronic diseases, acute exacerbations or events (e.g. myocardial infarctions or hospitalizations due to COPD) have been shown to be associated with daily levels of air pollution.

If one assumes that air pollution affects both the development of chronic disease and its exacerbation, the usual methods to derive attributable risks are inappropriate. First, the traditional approach that generally only evaluates acute morbidities due to air pollution, underestimates the total burden of air pollution because it does not account for part of the burden among those with the chronic disease. Second, the prioritization of preventive strategies may be improved by differentiating the acute and chronic burden attributable to air pollution.

Künzli et al. proposed methods to estimate the attributable risk for exacerbations under a "chronic disease model" [8] using as case study asthma in children due to local traffic-related pollution as the chronic disease of interest [9]. We generalized the model to apply this approach to other chronic diseases potentially associated with air pollution.

Building on standard air pollution HIA methodologies [10-13], this report describes specific calculations and data needs to integrate these new findings into the burden assessment of air pollution, and provide some guidance on limitations one may face when attempting to apply this approach. Deliverable D4 provides a case study for 10 cities in Europe using this approach, and describes in more detail these limitations and related uncertainties to this novel approach.

General approach

Population-attributable fractions (PAF) are commonly used to estimate the health impact of air pollution. PAF is the standard methodology to assess the contribution of a factor to disease using excess risks obtained from epidemiological studies applied to target populations. The derivation of PAFs combines several elements: the concentration–response functions (CRFs; the quantitative association between markers of air pollution and selected outcomes derived from epidemiological studies), the frequency of health conditions in the population of interest, the current population exposure to the selected marker(s) of air pollution, and the population exposure hypothesized after scenarios of air pollution reduction.

The proposed methodological expansion requires combining these different elements for evaluating the burden of both chronic and acute effects of air pollution under a general model of disease proposed and described earlier [8]. The general model of disease, presented in Figure 1, assumes that exposure X (in our case air pollution) is both a cause for the development of the underlying chronic pathologies (or disease) and the triggering of acute events. Under this model, X would first increase the number of people with asthma (Box A in Figure 1) and thereby increase the pool susceptible to acute exacerbations of any etiology. Second, X would trigger acute events (Box C and E) among all susceptible children (i.e. all in Box A and B). However, among children who developed asthma due to chronic exposure to X (Box A), one may attribute all their asthma-related morbidities to factor X, including those acute exacerbations not directly caused by X (Box D). This latter attributable burden has so far been neglected by risk assessors, and attributable cases of Box C and E have not previously been distinguished.

Two major assumptions are made when applying this revised model: (1) one assumes a causal role of pollution – in our example local traffic-related pollutants – in the development of the chronic disease and; (2) those with the chronic disease due to air pollution would not have developed it without this exposure.

Formulas

Detailed revised PAF calculations to integrate chronic effects in the HIA are presented below. Table 1 presents comparison of nomenclature and formulas, using the common and the expanded combined health impact assessment approach, as well as a comparison of the estimated burden of air pollution on exacerbations of symptoms among asthmatic children age 0-17 for 10 European cities, using these approaches (results based on deliverable D4).

Attributable cases of chronic disease due to air pollution (Box A)

The standard attributable fraction formula allows us to calculate an attributable fraction for any chronic disease associated to air pollution:

$$AF_{chron} = p_p (RR_\Delta - 1) / (p_p (RR_\Delta - 1) + 1) \quad (1)$$

where p_p is the proportion of persons exposed and RR_Δ is the concentration-response function for a specified change (Δ) in the ambient concentration.

The attributable number of chronic cases (AN_{chron}) due to air pollution is obtained as the product of the chronic disease in the population expressed as prevalent fraction (P_{chron}), the total population (Pop_{tot}), and the above attributable fraction AF_{chron}:

$$AN_{chron} = Pop_{tot} * P_{chron} * AF_{chron} \quad (2)$$

AN_{chron} is the additional number of cases expected due to the exposure in consideration. Accordingly, one can calculate the attributable number of cases in this population that are due to causes other than air pollution ($AN_{(1\text{-}chron)}$) as is shown in Equation 3.

$AN_{(1- chron)} = Pop_{tot} * P_{chron} * (1-AF_{chron})$ (3)

Annual exacerbations attributable to air pollution (Box C and E)

Using equation (1), the standard attributable fraction formula can also be applied to derive the annual acute exacerbations attributable to air pollution:

$AF_{acute} = p_p (RR_{\Delta}-1) / (p_p (RR_{\Delta}-1) + 1)$ (4)

The attributable number of acute complications (AN_{acute}) can be derived by multiplying the population with the current condition (Pop_{acute}) with the fraction of annual complications attributable to air pollution (AF_{acute}). Pop_{acute} is obtained through the frequency of the acute condition (P_{acute}) applied to the population with the chronic condition ($Pop_{chron}=P_{chron}*Pop_{tot}$). Depending on the definition of "exacerbation", its frequency may be expressed with the incidence (e.g. hospitalizations) or some measure of period prevalence (e.g. chronic symptomatic episodes). Equation 5 shows the formula for AN_{acute} using these parameters.

$AN_{acute}= P_{chron} * Pop_{tot} * P_{acute} * AF_{acute}$ (5)

As presented in Figure 1, AN_{acute} is actually the sum of the two components, Box C and E. By rearranging Equations 3 and 5, one can separate the two components into the attributable number of complications due to air pollution among those for whom the chronic disease is due to air pollution (AN_1, Box C) and the attributable number of complications due to air pollution among those who have the chronic disease due to other factors (AN_2, Box E) as shown in Equation 6 and 7:

$AN_1=AN_{chron} *P_{acute}*AF_{acute}$ (6)

$AN_2= (Pop_{tot} * P_{chron} -AN_{chron}) *P_{acute}*AF_{acute}$ (7)

Exacerbations attributable to causes other than air pollution among cases with the chronic disease due to air pollution (Box D)

Complications attributable to causes other than air pollution need to be considered "attributable to air pollution" if they occur among those who have the chronic disease due to air pollution (AN_3, Box D in Figure 1). This quantity can be estimated with equation 8.

$AN_3= AN_{chron} * P_{acute} * (1-AF_{acute})$ (8)

Total exacerbations due to air pollution (Box C+D+E)

Equations 6, 7 and 8 can be combined to calculate the total burden of complications (Tot AN_{acute}) due to air pollution.

$$\text{Tot } AN_{acute} = AN_1 + AN_2 + AN_3$$
$$= Pop_{tot} * P_{chron} [AF_{chron} * P_{acute} * AF_{acute} (AF_{acute}^{-1} + AF_{chron}^{-1} - 1)] \quad (9)$$

Data needs

Concentration-response functions (RR_Δ)

During the time period in which Aphekom took place, a report from the Health Effect Institute (HEI) revised the existing literature to identify all studies that relate chronic health effects with traffic pollution[14]. We used this review together with updated publications (up to December 2009) to identify studies of air pollution that could be included in the chronic effect part of such expanded models of Health Impact Assessment (HIA). Appendix 1 provides a list of chronic studies using traffic proximity as outcome, and Appendix 2 provides a list of chronic studies using pollutant levels as outcome. Part of the traffic-related chronic health effect evidence stems from studies characterizing proximity to traffic exhaust using markers of exposure such as distance from highways or traffic density or counts within a certain distance of the residence. Although epidemiological studies based on proximity models may be more prone to error than those based on other modeling tools, the integration of these studies in the air pollution health impact evaluation is of relevance because it can improve the policy decision; the information obtained can easily be transposed to relevant air pollution policy considerations, such as urban planning, which is missing in current air quality management strategies. Within the framework of Aphekom, traffic proximity studies for chronic diseases were used as a source of the CRFs to derive the attributable numbers.

For acute outcomes, a large range of effects from hospitalization to cough, to medication are associated with exposure to regional pollutants such as PM2.5, PM10, NO2, or O3. Within our approach, these pollutants are assumed to have acute effects independent from the long-term effects of traffic pollution. Conceptually, one would select acute CRFS related to the chronic disease and conducted on individuals with the chronic diseases. But it is also possible to apply acute CRFS that were developed on total population and for more distal outcomes (e.g. in asthmatic, respiratory disease instead of asthma attacks, or short-term mortality). When several studies are available, meta-analytic estimates can be derived to obtain an acute CRF. Ideally, acute CRFs should be based on populations comparable with those used to derive the chronic CRFs. Moreover, it is generally accepted to base CRFs on peer-reviewed studies.

<u>Limitations:</u>

The number of traffic proximity studies assessing both the long-term effects on chronic pathologies and related acute outcomes among similar populations is rather limited. For example, the exposure assessment is usually inconsistent across studies, possibly resulting in inherent heterogeneities of CRF's; for acute effect studies, the definition of outcomes or the population used may not be consistent across studies, again affecting the CRF's. As a result, with the currently available studies, it may not be possible to derive meta-analytic weighted CRFs based on several studies, which is usually preferred in the risk estimations. Also, using only one study to derive the CRF adds uncertainties with regard to the extrapolation to different populations and geographical areas.

Population exposure (p_p) and scenarios of change of exposure (Δexp)

To evaluate scenarios of exposure contrasts of interest in the traditional approach and to evaluate the superimposed acute effects of air pollution in our study, the RR published and combined or used individually as concentration-response functions (CRFs) has to be modified as follows:

$$RR_{\Delta} = e^{Ln\left(RR_{published} / Unit_{published}\right) x\, \Delta \exp}$$

Where $\mathrm{Unit}_{\mathrm{published}}$ is the unit for which the RR was published or derived, and Δexp is the change in exposure for the scenario under consideration.

For acute outcomes, annual average levels to represent population exposure can be derived from urban background fixed site monitors for one or several years, or population weighted averages can be calculated from existing concentration maps to estimate population exposure. The scenario of change of exposure will depend on the question to answers. For example, a scenario of change of current population exposure to the annual levels recommended by the World Health Organization (WHO) or other assumed reachable background level could be used.

The use of traffic proximity studies as CRFs in the revised approach needs a different methodological approach if it is to be integrated into the HIA compared to the studies based on pollutant concentrations. These studies give dichotomous risk estimates for cases living at a certain distance to busy roads (or other specific type of road) compared to controls living further way. To estimate the burden of traffic pollution for chronic diseases with this CRF, one can assume a scenario for which either no one lived next to those busy roads (i.e. the type of pollution currently occurring at high concentrations along busy roads had been removed). Thus with a dichotomous scenario (i.e. exposed versus not exposed), one needs to know the percent of the population living near traffic. The definition of "near" ought to be comparable to the one used in the original studie(s) used to derive the CRFs (Pp).
Using GIS tools, the distribution of population to traffic proximity can be done as follows. A step by step example is provided in Appendix 3.

- First one needs to distribute the population across the city. Population data is generally provided aggregated at different spatial scales which can vary from district or block depending on the city or country. The population data provided spatially however, doesn't provide information on the spatial distribution of the population within the "unit". But the distance to roadway calculations for populations are sensitive to assumptions regarding the distribution of population. For example, assuming all the population resides at the geographic center of the unit would tend to overestimate the distance to nearest roads because the center is probably the furthest point from the roads in most situations. Assuming the population is uniformly distributed across the unit is a less biased assumption [8]. The distribution of the population across the unit can follow two approaches depending on availability of data.

  ➢ If data is available per census block with no household density available, one can create grids having spaces equal to the average household surface in your city.

For example in Spain the average apartment is about 100 m^2, so we propose to have grids of 10x10m for Spanish cities.

➢ If data is available per census block or building with household density available, grids can be created based on household density. This will determine the amount of spacing of the grid that best fits each census block population.

- Grid points that fall in geographic zones unlikely to have any residents should be removed (e.g. parks, industrial or commercial buildings, schools or other urban features). Grid points that fall onto freeways, roads, rivers, etc should also be removed. Superposition of geo-referenced maps from different origins can also show discrepancies.

- The population at each grid point is then calculated by dividing the total population of the unit by the number of grid points. Similarly, grids points can then be used to derive the distribution of the population by specific age groups or by other strata of interest such as socio-economical status.

- Finally, one can calculate the distance from each population grid point to the closest "major" road to obtain population distributions within distance-based buffers along roads. The selection of roads will depend on the exposure metric used in CRFs. For each "major" road, each direction of travel should represent a separate roadway, and the shortest distance should be estimated from the grid point to the middle of the nearest side of the busy road.

Limitations:

While this method is relatively simple, it requires combining traffic data with population information that may only be partially available in some areas, and requires a series of assumptions whose validity may be difficult to prove. It is likely that the most difficult information to obtain may be traffic data, such as traffic counts for all streets in the area of study of the HIA. Use of road classification to select streets with heavy traffic that match with CRF definitions may be appropriate but consistency should be checked.

Target population (Pop_{tot}) and background risks (P_{chron}, P_{acute})

The target population should ideally be based on the same population as the chronic CRFs for a specific area which should be clearly identified and defined (e.g. city, conurbation, metropolitan area). Local health data should be used that match the outcome definitions in CRFs. All heath data frequencies should be related to the population from the area of the selected study (denominator).

Limitations:

Attributable estimates for exacerbation should be interpreted as episodes attributable per year. Depending on the definition of "exacerbation", their frequency may be expressed with the incidence (e.g. hospitalizations) or some measure of period prevalence (e.g. chronic symptomatic episodes). For chronic outcomes the prevalence is used for background risks and should be interpreted as all current cases that are due to air pollution.

While hospital and mortality data is easily obtainable through local registries, other morbidity prevalence data are generally not available at a local level and/or national level. Thus, extrapolation from literature reviews or from other cities may likely be necessary.

Uncertainties

A major issue for the validity of HIA relates to the expression of uncertainties.[24] Because the model requires combining two set of CRFS provided with confidence intervals, one can provide the range of uncertainty around the estimate of attributable cases, using Monte Carlo simulations to propagate CRFs uncertainty distributions into the attributable risks.[25] However, because there are several other sources of uncertainty that vary across the main assumptions, one needs additional approaches to integrate uncertainties more broadly. Conducting different sets of sensitivity analysis can provide useful information regarding the possible range of the impact caused by the uncertainty in the CRF point estimates. Moreover, stakeholders and expert opinions should be used to further explicit the degree of confidence in assumptions as well as methodological features.

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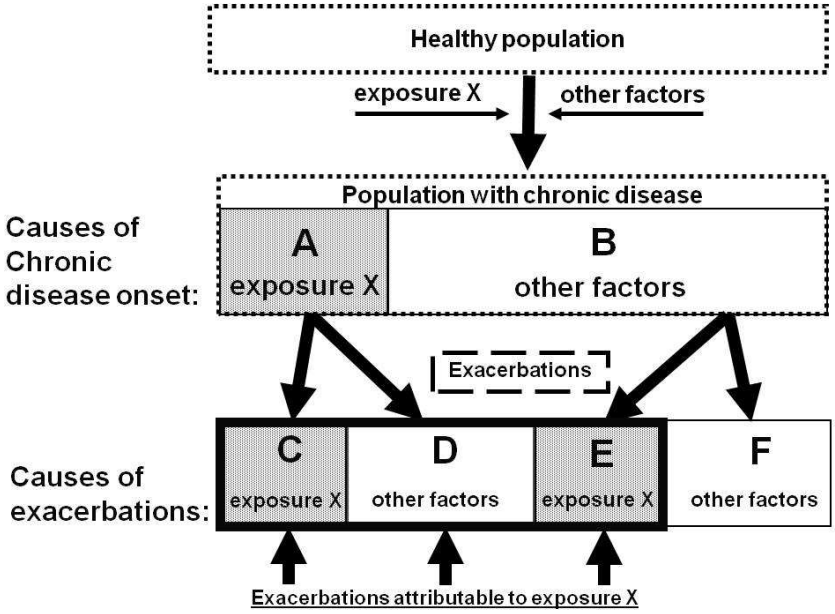
Table 1. Comparison of results, nomenclature, and formulas, using a common and the expanded combined health impact assessment approach to estimate the burden of air pollution on exacerbations of symptoms among asthmatic children age 0-17, in 10 European cities.

MODEL	CHILDHOOD ASTHMA			EXACERBATIONS OF ASTHMA (Bronchitis episodes)					
	Number of children with asthma	Cases attributed to traffic pollution	Cases attributed to other causes	Number of bronchitis exacerbations among asthmatic children	Attributable to air pollution among those with asthma due to…			Exacerbations due to other causes among those with asthma	**Total**
					… traffic pollution	… other factors	… all causes	due to traffic pollution	
Nomenclature in text	Pop_{chron}	AN_{chron}	$AN_{(1\text{-}chron)}$	Pop_{acute}	AN_1	AN_2	$=AN_1 + AN_2$	AN_3	Tot AN_{acute}= $AN_1 + AN_2 + AN_3$
Equation in text	--	(Equ. 2)	(Equ. 3)	--	(Equ. 6)	(Equ. 7)	(Equ. 5)	(Equ. 8)	(Equ. 9)
Location in Figure 1	--	(Box A)	(Box B)	--	(Box C)	(Box E)	(Box C+E)	(Box D)	(Box C+D+E)
TRADITIONAL MODEL									
Number of cases	80,537	0	80,537	4,189	N/A	N/A	400	0	400
Fraction (in %)	100%	0%	100%	100%	--	--	9.6%	0	9.6%
COMBINED MODEL									
Number of cases	80,537	12,046	68,491	4,189	60	340	60+340 =400	570	970
Fraction (in %)	100%	25%	75%	100%	1.4%	8.1%	9.6%	13.6%	23.2%

Figure 1. The burden of air pollution assuming a causal role of air pollution (exposure X) in both disease onset and exacerbations. Modified from Künzli et al. 2008 15. Sizes of the boxes do not reflect the burden.



Appendix 1

Summary of studies on the association between traffic proximity and health outcomes

Appendix 1.1. Summary of studies that use "traffic proximity" as exposure –Cardiovascular outcomes. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Years of Study (N)	Outcome	Selected Effect Estimate	Effect Scale and Metric
Hoffmann et al 2006 [17]	2000-03 (4494) German Ruhr Area	Prevalent Clinical CHD	OR full model: **1.85 (1.21, 2.84)**	Major road≤150 m of home (10-110∗10^3 veh/day)
Hoffmann et al. 2007 [18]	age 45 to 74 years) 3 cities in Germany (Essen, Mülheim, and Bochum)	coronary artery calcification	**1.63 (95%CI, 1.14 to 2.33),** **1.34 (95% CI, 1.00 to 1.79),** 1.08 (95% CI, 0.85 to 1.39),	Compared with participants living >200 m away from a major road, participants living within 50, 51 to 100, and 101 to 200 m
Hoffman et al. 2009 [19]	age 45 to 74 years) 3 cities in Germany (Essen, Mülheim, and Bochum)	ankle-brachial index for peripheral arterial disease	ankle-brachial index of OR=-0.015 (-0.030 to 0.0), OR=-0.002 (-0.021 to 0.016) OR=-0.024 (-0.047 to -0.001) peripheral arterial disease OR of **1.77 (1.01-2.1)**	compared with those living more than 200 m away. Living within 101-200, of a major road 51-100, of a major road and 50 m of a major road Living within 50 m of a major road
Tonne et al 2007 [20]	1995-2003, odd years (5049 cases, 10,277 controls), UK	MI, >=25	OR full model adjusted for spatial autocorr. Cumulative traffic (IQR): **1.06 (1.03, 1.09)** Near major roadway (per km) 1**.06 (1.02, 1.10)** both in same model	Traffic density in 100 m buffer of residence and distance from major roadway
Kan et al. 2008 [21]	1987-1989, 4 US communities, middle-age man and women	Incident Coronary Heart Disease	Adjusted model: 1.09 (0.94-1.26) Adjuste model: **1.03 (1.01-1.05)**	Roadways ≤150 m of home One unit increase of log-transformed traffic density

Appendix 1.2. Summary of studies that use "traffic proximity" as exposure-Doctor-diagnosed asthma incidence Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Birth Year (N) Age of Observation	Effect Estimate – OR (95% CI)	Effect scale and metric
Shima et al 2003 Chiba Prefecture, Japan [22]	1986 (1,858) Age 6-9 (four yr study)	Male: 1.99 (0.79-4.99) Female: 1.74 (0.63-4.81)	≥50m from trunk road (rural=reference)
		Male: **3.77 (1.00-14.16)** Female: 4.03 (0.9-17.69)	0-49m from trunk road (rural=reference)
Morgenstern et al 2007 Munich, Germany [23]	1993-1997 (3,059 age 1; 2,861 age 2) Age 1 Age 2	 1.12 (0.88-1.44) **1.23 (1.00-1.51)**	OR per IQR: Living ≤50m to road

Appendix 1.3. Summary of studies that use "traffic proximity" or other traffic-related metric as exposure-Prevalence of doctor-diagnosed asthma. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Year of Study (N) Age of Observation	Effect Estimate - OR (95% CI)	Effect scale and metric
Gordian et al 2006 Anchorage, Alaska [24]	(671) Age 5-7	 1.40 (0.77-2.55) **2.83 (1.23-6.51)** Children without parental asthma: **2.43 (1.12-5.28)** **5.34 (2.08-13.74)**	Traffic density within 100m of residence Ref = low exposure level (< 4 x 10^6 vehicle meters (vm)) Medium (4 x 10^6 to 8 x 10^6 vm) High (>8 x 10^6 vm) Medium High (traffic density at cross street closest to the child residence)
McConnell et al 2006 southern California [25]	2003 (5,341) Age 5-7	All: **1.29 (1.01-1.66)** No parental asthma: **1.85 (1.11-3.09)** No allergic symptoms: **2.27 (1.04-4.94)** Female long-term residents: **2.51 (1.39-4.54)** Male long-term residents: 0.94 (0.54-1.64)	Residence <75m of a major road
Gauderman et al 2005 southern California [26]	1993 & 1996 (208) Age 10	**1.89 (1.19-3.02)** 1.45 (0.73–2.91)	Distance to freeway OR for the25th percentile compared with the 75th percentile (ie, living closer compared with farther from the freeway). Traffic volume within 150 meters OR for the 75th percentile compared with the 25th percentile
Lewis et al 2005 U.K. [27]	2003 (11,562) Age 4-6	1.0 (ref) 1.14 (0.96-1.36) 1.02 (0.87-1.21) 0.90 (0.69-1.18)	>150 90-149 Distance from main road (meters) 30-89 <30
Janssen et al 2003 Netherlands [28]	1997-99 (2,083) Age 7-12	1.02 (0.42-2.44) 1.04 (0.74-1.45)	Trucks/weekday per 17,000; Cars/weekday, per 120,000 School/home—motorway <100 m versus 100-400 m
Nicolai et al 2003 Munich, Germany [29]	1995-1996 (7,509) Age 5-7 & 9-11	1.19 (0.76-1.87)	High traffic (>30,000 traffic counts per day)
van Vliet et al 1997 South Holland [30]	1993 (1,068) Age 7-12	1.68 (0.68-4.14) 0.54 (0.18-1.60)	Home within 100m of freeway Truck density on freeway

Wjst et al 1993 Munich, Germany [31]	1989/90 (6,537) Age 9-11	Lifetime: 1.06 (0.97-1.16) Present past year: 1.04 (0.89-1.21)	traffic density in school district per 25,000 vehicles / day
Ryan 2005 [32]	Cincinatti, US >6 months old	Infants living very near (<100 m) stop-and-go bus and truck traffic had a significantly increased prevalence of wheezing (adjusted odds ratio, **2.50; 95% CI, 1.15-5.42)**	living near moving truck and bus traffic (highway >50 miles per hour, >1000 trucks daily, <400 m), stop-and-go truck and bus traffic (<50 miles per hour, <100 m), or unexposed and not residing near either.
Kim et al 2008 [33]	2001 California Students grade 3-5 Current asthma (n = 88/724)	 1.00 1.50 (0.67–3.36) **2.33 (1.03–5.28)** 0.60 (0.21–1.69) **2.50 (1.13–5.53)** **2.40 (1.13–5.07)** 1.00 1.39 (0.62–3.11) **2.83 (1.23–6.54)** 1.40 (0.60–3.29) 1.58 (0.69–3.65) 1.16 (0.53–2.54) 1.00 1.23 (0.53–2.83) 1.96 (0.85–4.52) 1.40 (0.60–3.3) **2.37 (1.05–5.36)** **2.14 (1.02–4.52)** **1.43 (1.04–1.54)** **3.80 (1.20–11.71)** 1.87 (0.71–4.90) 1.25 (0.50–3.11) 1.00 1.41 (0.81–2.46) 1.05 (0.58–1.91) 1.00 1.36 (0.51–3.62) 1.00	**Maximum AADT within 150 m (vehicles/day)** 1st quintile (local traffic only) 2nd quintile (up to 7,120) 3rd quintile (7,121–18,900) 4th quintile (18,901–28,657) 5th quintile (28,658–245,000) ≥ 90th percentile (67,000–245,000) **Closest AADT within 150 m (vehicles/day)** 1st quintile (local traffic only) 2nd quintile (up to 5,700) 3rd quintile (5,701–10,534) 4th quintile (10,535–23,800) 5th quintile (23,801–245,000) ≥ 90th percentile (35,100–245,000) **Traffic density within 150 m** 1st quintile 2nd quintile 3rd quintile 4th quintile 5th quintile ≥ 90th percentile **Log distance to freeway/highwayc** **Distance to freeway/highway** ≤ 75 m > 75 to ≤ 150 m > 150 to ≤ 300 m > 300 m **Distance to freeway/highway and wind orientation** ≤ 300 m, downwind ≤ 300 m, upwind > 300 m **Distance to principal artery (excluding those near freeway/highway)** ≤ 75 m > 300 m

Appendix 1.4. Summary of studies that use "traffic proximity" as exposure-Studies on lung function –children. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Year of study/ duration (N) Age of Observation	Group characteristic	Time domain of effect	Effect Estimate (95% CI)	Effect scale and metric
Fritz & Herbarth 2001 Leipzig, Germany [34]	1994/95 (235) Age 4-6	Cross-section Pre-schoolers from 16 day-care centers	Long-term	Centrally heated areas: FVC pred: -3% FEV_1 pred: -15%	'low' vs. 'high' traffic
Gauderman 2007 Southern California [35]	1992-2000 / 8yrs (3677) Age 10-18	Asthma, no asthma	Long-term	**FEV_1 -98 mL (-182 to -15)**	3 categories of road distance, 8-yr lung growth Residence <500 m of a freeway (motorway)
Gauvin et al 2001 4 French metropolitan areas [36]	1998 (~600) Age 4-14	Volunteers, ½ asthmatics	Long-term	FEV_1: -0.23* (±0.04) -0.47# (±0.15) 0.02 (±0.04) -0.001 (±0.01)	Regression slopes (and standard errors) between respiratory function and the index of traffic exposure Traffic index: time-weighted index combining density & distance
Janssen et al 2003 Netherlands [28]	1997-1998 (1,724) Age 7-12	24 schools <400m of busy roads	Long-term	FEV_1<85% predicted 1.19 (0.34-4.23) 1.13 (0.40-3.16)	(distance: not significant) Trucks/day per 17,000 Cars/day per 120,000
Nicolai et al 2003 Munich, Germany [29]	1995-1996 (2019) Age 5-7 & 9-11	school based sample	Long-term	No association; no results reported	Traffic counts - No results
Schikowski et al 2006 Rhine-Ruhr Basin [37]	1985-1994 (2593) Age 54-55	German females	Long-term	**OR=1.79, (1.06-3.02)** For COPD (FEV1/FVC<70%)	Residence ≤ 100 m of a busy road (>10,000 vehicles/day)
Wjst et al 1993 Munich, Germany [31]	1989/90 (4320 with spirometry) Age 9-11	4th graders	Long-term	All estimates negative; significant for: **MEF25: -0.68%(-1.11;-0.25)** **MEF50:-0.72% (-1.25;-0.18)** MEF50/FVC: **-0.66% (-1.20;-0.12)**	Cars passing school district (117 districts) % change per 25,000 cars/day
Roselund et al.. 2009 [38]	Rome 200-1 (n=2107)	Age 9-14	Long-term	**FVC 76ml (8-143)**	Decrease in lung function measure for children living <=20. From busy roads (compared to those living>=200m)

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Appendix 1.5. Summary of studies that use "traffic proximity" as exposure-Studies on respiratory health –adults. Modified from Health Effect Institute report on traffic-related air pollution. (up to September15, 2009).

Author Location	Years of Study (N) Age of Observation	Effect Estimate – Odds ratio (95% CI)	Effect scale and metric
Pujades-Rodriguez et al. 2009 [39]	Nottingham, UK (2644) Age 18-70	No association (wheezing in last year, COPD, bronchial hyper-responsiveness, allergic sensitization, IgE, FEV1)	Distance to main road defined as a motorway (freeway), or 'A' or 'B' class road (principal road as classified by UK Department for Transport
Modig et al 2006 Luleå, Sweden [40]	1995-1999 (138 cases / 136 controls) Age 20-60	Asthma incidence 2.4 (0.9-6.2)	High traffic flow per day (reference = low)
Nitta et al 1993 Western suburbs of Tokyo 3 cross-sectional studies [41]	1979 (1173)	**Chronic wheezing: 2.75 (1.65-4.73)** **Chronic cough: 1.62 (1.07-2.46)** **Chronic phlegm 1.47 (1.03-2.11)** Shortness of breath: 1.41 (0.89-2.24) **Chest cold + phlegm: 1.35 (1.04-1.77)**	Living <20 m versus 20-150 m from heavy traffic road
	1982 (2015)	Chronic wheezing: 1.52 (0.91-2.55) Chronic cough: 1.35 (0.88-2.07) **Chronic phlegm 1.87 (1.31-2.68)** Shortness of breath: 1.42 (0.94-2.15) Chest cold + phlegm: 1.21 (0.91-1.59)	Living <20 m versus 20-150 m from heavy traffic road
	1983 (2023)	Chronic wheezing: 0.94 (1.61-1.42) Chronic cough: 1.45 (0.98-2.13) Chronic phlegm 1.26 (0.94-1.70) **Shortness of breath: 1.66 (1.12-2.48)** Chest cold + phlegm: 0.94 (0.76-1.17)	Living <20 m versus 20-150 m from heavy traffic road
Nakai et al 1999 Tokyo, Japan [42]	1987-1990 (1986) Age 30-59 (females only)	Persistent wheezing: 1.17 (0.59-2.34) 1.00 (0.48-2.04) Chronic cough: **1.87 (1.02-3.42)** **2.18 (1.08-4.42)** Chronic phlegm 1.40 (0.88-2.21) **1.79 (1.07-3.01)** Breathlessness A to B: 0.83 (0.50-1.38) A to C: 1.16 (0.66-2.04)	Zone A: 0-20m -- heavy traffic; B: 20-150m; C: suburban Zone A vs. Zone B Zone A vs. Zone C Zone A vs. Zone B Zone A vs. Zone C Zone A vs. Zone B Zone A vs. Zone C Zone A vs. Zone B Zone A vs. Zone C

Smargiassi et al 2006 Montreal, Canada [43]	2001-2002 (5,805 cases / 39260 controls) Age ≥ 60	All diagnoses (except accidents/CVD) **1.08 (1.00-1.15)** **1.24 (1.12-1.38)** Compared to genitourinary **1.13 (1.02-1.26)** **1.57 (1.32-1.87)**	Traffic intensity during 3-hr-morning peak 1-3160 vehicles/ 3 peak hrs >3160 vehicles 1-3160 vehicles/ 3 peak hrs >3160 vehicles
Livingston et al 1996 London, U.K. [44]	1994 (630 Cases / 5059 controls) Age 16-64	Asthma prescriptions 1.00 (0.84-1.19)	Living >150 m from a main road.
Oosterlee et al 1996 Haarlem, Netherlands [45]	1991 (1117 adults) (See Table 3 for children)	Cross-sectional Doctor diagnosed asthma: 1.02 (0.8-1.9) Current asthma medication: 1.2 (0.4-3.2) Wheeze >1wk, past 2yrs: 1.1 (0.6-1.8) Chronic obstructive pulmonary disease medication, chronic cough, chronic phlegm: all neg.	Living on busy streets (high NO_2) vs. living on calm streets
Morris et al 2000 East London, U.K. [46]	1991-1992 (125 asthma / 124 chronic obstructive airways pairs)	Asthma admissions: 0.78 (0.46-1.32) COPD admissions: 0.94 (0.57-1.54)	Living ≤ 150 m of a main road (motorway A or B)
Schikowski et al 2005 Essen area, Germany [37]	1985-1994 (4262) Age 54-55 (all females)	Doctor diagnosed chronic bronchitis: 1.15 (0.89-1.50) Chronic cough with phlegm: 1.07 (0.83-1.37) **Frequent cough:** **1.14 (1.03-1.49)**	Consecutive cross sectional studies Residential location < 100m of main road (>10,000 cars per day)
Bayer-Oglesbyt et al 2006 Switzerland [47]	1991; 2002 follow-up (8,555 reenrolled; 5,922 never smokers) Age 18-60	Results for never smokers Wheezing & breathing problems: 0.91 (0.79-1.05) 1.15 (0.97-1.35) **1.34 (1.00-1.79)** Wheeze without cold: 0.91 (0.78-1.06) **1.18 (1.01-1.39)** 1.05 (0.76-1.45) Attack of breathlessness 0.88 (0.78-1.00) **1.20 (1.05-1.38)** 1.06 (0.82-1.37) Cough 1.00 (0.91-1.10) 1.08 (0.96-1.21) 0.93 (0.75-1.17) Phlegm 1.01 (0.91-1.12) 1.06 (0.94-1.20) 1.06 (0.84-1.34)	Cross-sectional association 'last 12 months'; ***bold*** = statistically significant Distance to main street (per 100m) Length of main street segments ≤ 200m (per 500 m) Residence ≤ 20 m of a main street Distance to main street (per 100m) Length of main street segments ≤ 200m (per 500 m) Residence ≤ 20 m of a main street Distance to main street (per 100m) Length of main street segments ≤ 200m (per 500 m) Residence ≤ 20 m of a main street Distance to main street (per 100m) Length of main street segments ≤ 200m (per 500 m) Residence ≤ 20 m of a main street Distance to main street (per 100m) Length of main street segments ≤ 200m (per 500 m) Residence ≤ 20 m of a main street Definition as in Vector25 (www.swisstopo.ch): Second-class, first class,

			major road, and highway (Switzerland).
Venn et al 2005 Jimma, Ethiopia [48]	(2179 adults, 2025 households) Children/adults	Wheeze **All subjects: 1.17 (1.01-1.36)** Adults only: 1.14 (0.97-1.33) Nearest road > median traffic **All: 1.26 (1.03-1.53)**	Distance to main road: per 30 m among those living within 150 m 'main road' traffic very low: <1000 vehicles per day light 12 hrs Median flow = 653 vehicles over 12 hour daylight period.
Garshick et al 2003 Southeastern Massachusetts [49]	1988-1992 (2985) US male veterans Mean age 60.6	persistent wheeze: **1.31 (1.00-1.71)** chronic cough: 1.24 (0.92-1.68) chronic phlegm 1.18 (0.88-1.56) persistent wheeze **1.71 (1.22-2.40)** chronic cough: 1.29 (0.87-1.91) chronic phlegm: 1.40 (0.97-2.02)	Residential distance to main roads: ≤50 m versus >400 m Traffic volume (among those within 50 m): <10,000 versus ≥10,000 per day (ref: >50 m) 23% live within 50m

Appendix 1.6. Summary of studies that use "traffic proximity" as exposure-Studies on lung function (only long-term)-adults. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Year of study/ duration (N) Age of Observation	Group characteristic	Effect Estimate (95% CI)	Effect scale and metric
Pujades-Rodriguez et al. 2009 [39]	Nottingham, UK (2644) Age 18-70		No association (wheezing in last year, COPD, bronchial hyper-responsiveness, allergic sensitization, IgE, FEV1)	Distance to main road defined as a motorway (freeway), or 'A' or 'B' class road (principal road as classified by UK Department for Transport
Kan et al 2007 4 US communities [21]	1987-1989 follow-up 1990-1992 (13,972) mean age 54.2	ARIC population sample	FEV_1 (ml): **F: -17.4 (-41.9-7.1)** **M: -43.9 (-85.9- -2)** **F: -29.5 (-52.2- -6.9)** **M: -38.1 (-76.7-0.6)**	Distance to major roads Distance <100m Distance <150m Major road define as: interstate highways (road class 1), state highways (road class 2), major arterial roads (road class 3) and local roads (road class 4).
Schikowski et al 2005 Rhine-Ruhr Basin [37]	1985-1994 (2593) Age 54-55	German females	**OR=1.79, (1.06-3.02)** For COPD (FEV1/FVC<70%)	Residence ≤ 100 m of a busy road (>10,000 vehicles/day)
Sekine et al 2004 Tokyo, Japan [50]	1987-1994 / 8 yrs (733) Age 30-59	Population sample (females only)	Delta FEV1 per year: -20ml -15ml -9 ml p for trend <0.001 *translates very roughly to a -5ml faster decline per year per 11ppb NO_2*	Distance to busy road ≤20m of busy road (47-56ppb NO_2) 20-150m (38-46 ppb NO_2) 'behind roads' (24-36ppb NO_2)
Wjst et al 1993 Munich, Germany [31]	1989/90 (4320 with spirometry) Age 9-11	4th graders	All estimates negative; significant for: **MEF25: -0.68%(-1.11;-0.25)** **MEF50:-0.72% (-1.25;-0.18)** **MEF50/FVC:** **-0.66% (-1.20;-0.12)**	Cars passing school district (117 districts) % change per 25,000 cars/day

Appendix 2

Summary of studies on the association between traffic-related pollutants and health outcomes

Appendix 2.1. Summary of studies of Cardiovascular Morbidity for traffic-related pollutants. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Years of Study (N)	Outcome	Selected Effect Estimate	Effect Scale and Metric
Rosenlund et al 2006 [51]	1992-94 (1397 cases, 1870 controls)	First MI	OR Non-fatal: 0.89 (0.67,1.19) (n=1085) OR In-hosp. death: 1.28 (0.75, 2.17) (n=188) OR Out-hosp. death: 2.17 (1.05, 4.51) (n=84)	NO_2 from dispersion modeled traffic generated pollution (5^{th} – 95^{th} percentile range=30 μg/m^3)

CAC=coronary artery calcification

Appendix 2.2: Summary of studies of doctor-diagnosed asthma incidence for traffic-related pollutants–children. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Birth Year (N) Age of Observation	Effect Estimate – OR (95% CI)	Effect scale and metric
Brauer et al 2002 Netherlands [52]	1996/97 (2,989) 0-12 months 12-24 months 0-24 months Year 2	 1.27 (1.01-1.59) 1.12 (0.88 -1.43) 1.14 (0.94 -1.38) 1.12 (0.88-1.43)	OR per IQR: Per 0.54 x$10^{-5}m^{-1}$ local “soot”
	0-12 months 12-24 months 0-24 months Year 2	1.25 (1.00-1.57) 1.18 (0.88-1.43) 1.16 (0.96 -1.40) 1.18 (0.93-1.51)	Per 10.3 µg/m^3 local NO_2
Brauer et al 2007 Netherlands [53]	1996/97 (2,826) Age 4	 1.30 (0.98-1.71) 1.26 (1.02-1.56)	OR per IQR: Per 0.58 x$10^{-5}m^{-1}$ local “soot”
	Cumulative Year 0-4	1.29 (0.99-1.69) 1.19 (1.04-1.56)	Per 10.6 µg/m^3 local NO_2
Jerrett et al. 2008 Southern California Children Health Study [54]	1983 or 1986 (217) Age 10 years, 8 years follow-up	1.29 (1.07-1.56) (adjusted for humidity, ethnicity, enrolment group, insurance, baseline strata of age and sex)	Per 6.2 ppb measured home outdoor NO2.
Morgenstern et al 2007 Munich, Germany [23]	1993-1997 (3,059 age 1; 2,861 age 2) Age 1 Age 2 Age 1 Age 2	 1.14 (0.88-1.48) 0.85 (0.31-2.34) 1.30 (1.03-1.66) 0.82 (0.33-2.03)	OR per IQR: Per 0.4 x$10^{-5}m^{-1}$ local “soot” Per 8.5 µg/m^3 local NO_2

Appendix 2.3: Summary of studies of prevalence asthma for traffic-related pollutants–children. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Year of Study (N) Age of Observation	Effect Estimate - OR (95% CI)	Effect scale and metric
Kim et al 2004 San Francisco Bay Area [55]	2001 (1,109) Age 8-10	All: 1.05 (0.98-1.12) Long-term residents (LTR): 1.08 (1.0-1.15) LTR girls: 1.19 (1.03-1.36) LTR boys: 1.02 (0.94-1.12)	Per 11.6 ppb nitric oxide (NO)
Gauderman et al 2005 southern California [26]	1993 & 1996 (208) Age 10	1.83 (1.04-3.21)	Per 5.7 ppb NO_2 home outdoor
Janssen et al 2003 Netherlands [28]	1997-99 (2,083) Age 7-12	1.36 (0.62-2.98) 1.39 (0.75-2.56)	Soot per 10 µg/m3 NO_2 per 18 µg/m3
Nicolai et al 2003 Munich, Germany [29]	1995-1996 (7,509) Age 5-7 & 9-11	1.42 (0.92-2.2) 1.28 (0.81-2.01)	Soot (>10.73 µg/m^3): highest tertile versus rest NO_2 (>57.4 µg/m^3): highest tertile versus rest
Hirsch et al 1999 Dresden, Germany [56]	1995-96 (4,378) Age 5-7 & 9-11	1.11 (0.97-1.25) Non-atopic children: 1.32 (1.07-1.62) 1.16 (0.94-1.42) Non-atopic children: 1.49 (1.04-2.16) 1.07 (0.94-1.21) Non-atopic children: 1.29 (1.05-1.59)	Benzene: per 1 µg/m^3 NO_2: per 10 µg/m^3 CO: per 0.2 µg/m^3
van Vliet et al 1997 South Holland [30]	1993 (1,068) Age 7-12	0.22 (0.04-1.13) 0.37 (0.11-1.24)	Black smoke concentration in school NO_2 concentration in school

Appendix 2.4: Summary of studies of respiratory symptoms for traffic-related pollutants-children. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Birth Year (N) Age of Observation	Effect Estimate - OR (95% CI)	Effect scale and metric
DRY COUGH AT NIGHT			
Brauer et al 2002 Netherlands 52	1996/97 (2,969) Age 2	1.02 (0.88-1.17) 1.02 (0.89-1.18)	Per 0.54 x$10^{-5}$$m^{-1}$ local “soot” Per 10.3 µg/m^3 local NO_2
Brauer et al 2007 Netherlands 53	1996/97 (2,830) Age 4	1.14 (1.00-1.31) 1.11 (0.97-1.26)	Per 0.58 x$10^{-5}$$m^{-1}$ local “soot” Per 10.6 µg/m^3 local NO_2
Gehring et al 2002 Munich, Germany 57	1993-1997 (1,607) Age 1	all: 1.27 (1.04-1.55) males: 1.31 (1.04-1.67) females: 1.26 (0.79-1.71) all: 1.36 (1.07-1.74) males: 1.45 (1.07-1.98) females: 1.20 (0.78-1.84)	Per 0.4 x$10^{-5}$$m^{-1}$ local “soot” Per 8.5 µg/m^3 local NO_2
	Age 2 (1,507)	all: 1.16 (0.98 -1.37) males: 1.17 (0.95-1.44) females: 1.12 (0.84-1.48) all: 1.24 (1.02-1.51) males: 1.28 (0.99-1.66) females: 1.17 (0.86-1.60)	Per 0.4 x$10^{-5}$$m^{-1}$ local “soot” Per 8.5 µg/m^3 local NO_2
Morgenstern et al 2007 Munich, Germany 23	1993-1997 (3,037) Age 1	1.09 (0.78-1.51) 1.34 (1.00-1.81) 0.84 (0.61-1.16)	Per 0.4 x$10^{-5}$$m^{-1}$ local “soot” Per 8.5 µg/m^3 local NO_2 Living ≤50m to road
	Age 2	1.18 (0.93-1.50) 1.16 (0.92-1.47)	Per 0.4 x$10^{-5}$$m^{-1}$ local “soot” Per 8.5 µg/m^3 local NO_2
Pierse et al 2006 Leicestershire, U.K. 58	1998 (2,584) follow-up 2001 (2,331)	1998: 1.06 (0.94-1.19 2001: 1.25 (1.06-1.47) Incidence: 1.19 (0.96-1.47) Cough w/o cold: 1998: 1.21 (.107-1.38) 2001: 1.56 (1.32-1.84) Incidence: 1.62 (1.31-2.00)	Per 1 µg/m^3 local PM_{10} (~ per 1 IQR)
Gauderman et al. 2005 Southern California 26	1983 & 1986 (208) Age 10	Current asthma medication use 2.19 (1.20-4.01)	Per 5.7 ppb home outdoor NO_2
Krämer et al 2000 West Germany 59	1987 (317) Age 9	Bronchial asthma ever 1.82 (0.36-9.36)	Per 10 µg/m^3 urban outdoor NO_2
Mukala et al 1996 Helsinki, Finland 60	1985-1988 (172) Age 3-6	Prevalence: Cough 1.66 (1.03-2.68) Nasal symp: 1.97 (1.10-3.52) Incidence: Cough 1.15 (0.83-1.59) Nasal symp: 1.23 (0.91-1.67) Spring: Prevalence of cough:	Comparison of center area versus suburban area Center area = 27.4 µg/m^3 median NO_2 exposure Suburban area = 18.2 µg/m^3 median NO_2 exposure Personal NO_2 tertiles:

		1.17 (0.58-2.40) / 2.18 (0.9-5.33) Incidence of cough: 1.24 (0.75-2.07) / 1.72 (0.85-3.49)	11.0-17.8 µg/m^3 = reference 17.9-26.5 / 26.6-45.8 µg/m^3
Janssen et al 2003 Netherlands [28]	1985-1992 (2,083) Age 7-12	Current phlegm: 2.41$^{\#}$ (0.96-6.04) 1.72 (0.82-3.62) Current conjunctivitis: 2.54* (1.15-5.60) 2.60** (1.38-4.90)	Soot per 10 µg/m^3 NO_2 per 18 µg/m^3 Soot per 10 µg/m^3 NO_2 per 18 µg/m^3
Nicolai et al 2003 Munich, Germany [29]	1984-1991 (7,509) Age 5-7 & 9-11	Cough: 1.60** (1.14-2.23)	NO_2: highest tertile (>57.4µg/m3) versus rest;
Hirsch et al 1999 Dresden, Germany [56]	1984-1991 (4,316=atopic children) (4,306=non-atopic) Age 5-7 & 9-11	Morning cough All children: 1.22 (1.04-1.44) Non-atopic children: 1.42 (1.10-1.84) Bronchitis All children: 1.23 (1.11-1.38) Non-atopic children: 1.37 (1.17-1.62)	per 10 µg/m^3 NO_2 (exposure at home) All models adjusted for SES, maternal smoking, birth weight, sex, age, pets, dampness nod other covariates
Braun-Fahrländer et al 1992 Zurich and Basel, Switzerland [61]	1980-1986 (625) Age 0-5	Upper respire symptoms 1.19 (0.99-1.42) Duration of symptoms: 1.13 (1.01-1.27) Duration of episodes of breathing difficulty 1.50 (1.04-2.16)	per 20 µg/m^3 home outdoor NO_2
Van Roosbroeck et al. 2008 [62]	1987-92 (1843) Age 7-12	Phlegm: 5.29 (1.24-22.62) 3.82 (1.03-14.21) Conjunctivitis: 5.06 (1.02-24.96) 6.60 (1.33-32.77)	Soot per 9.3 ug/m3 NO2 per 17.6 ug/m3 Soot per 9.3 ug/m3 NO2 per 17.6 ug/m3

Appendix 2.5: Summary of health services and other asthma outcomes for traffic-related pollutants-children. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Years of Study (N) Age of Observation	Effect Estimate - OR (95% CI)	Effect scale and metric
Pershagen et al 1995 Stockholm, Sweden [63]	1986-88 (197 cases / 350 controls) Age 4 months – 4 years	Wheezing bronchitis (case/control): males: 1.7 (0.9-3.3) females: 1.7 (0.6-4.4) males: 1.0 (0.5-1.8) females: 1.5 (0.6-3.6) males: 0.7 (0.4-1.3) females: 2.7 (1.1-6.8) p-trend boys: 0.1; girls 0.02	Time-weighted outdoor NO_2: (Reference: <35 µg/m^3 NO_2) 35-45 µg/m^3 NO_2 46-70 µg/m^3 NO_2 >70 µg/m^3 NO_2
Ising et al 2004 Osterode, Germany [64]	2001 (401) Age 5-12	Pediatrician contacts due to: *Asthma* 1.41 (0.83-2.37) 4.22 (2.79-8.16) *Frequent Bronchitis* 1.95 (1.11-3.42) 13.8 (7.19-26.4)	Exposure to traffic emissions control: 10.7 µg/m^3 NO_2, 0.06 mg/(m^2xd) sedimented dust Medium: 11.8 µg/m^3 NO_2, 0.1 mg/(m^2xd) dust High: 26.0 µg/m^3 NO_2, 0.36 mg/(m^2xd) dust Medium: 11.8 µg/m^3 NO_2, 0.1 mg/(m^2xd) dust High: 26.0 µg/m^3 NO_2, 0.36 mg/(m^2xd) dust

Appendix 2.6: Summary of asthma outcomes for traffic-related pollutants-adults. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Years of Study (N) Age of Observation	Effect Estimate – Odds ratio (95% CI)	Effect scale and metric
Künzli et al. 2009 Switzerland	1991-2002 Age 18-60	Asthma incidence : 1.30 (1.05-1.61)	1 ug/m3 of traffic-related PM10
Jaquemin et al. 2009 European Respiratory Health Survey (17 centers) 65	1999-2001	Self-reported asthma incidence: 1.43 (1.02 to 2.01)	per 10 ug/m3 NO2.
Modig et al 2006 Luleå, Sweden 40	1995-1999 (138 cases / 136 controls) Age 20-60	Asthma incidence 2.4 (0.9-6.2) 1.1 (0.9-1.2) Positive skin test: 1.2 (1.0-1.3)	 High traffic flow per day (reference = low) Outdoor NO_2, measured 1 wk 0.5m from home Outdoor NO_2, measured 1 wk 0.5m from home
Sunyer et al 2006 European Community Respiratory Health Survey (10 countries) 66	1991-1993, follow-up 2000-2002 (1634)	All women 1.76 (1.04-2.98) 2.71 (1.03-7.16) Women >16yrs educated: 1.90 (1.23-2.93) 5.81 (1.22-27.7) All men: 0.85 (0.56-1.31) 0.99 (0.40-2.46)	Prevalence of chronic phlegm Per each increase in 30 µg/m^3 home outdoor NO_2 high to low: <20 µg/m^3 NO_2 vs. > 50 µg/m^3 NO_2 Per each increase in 30 µg/m^3 home outdoor NO_2 high to low: <20 µg/m^3 vs. > 50 µg/m^3 Per each increase in 30 µg/m^3 home outdoor NO_2 high to low: <20 µg/m^3 vs. > 50 µg/m^3
Oosterlee et al 1996 Haarlem, Netherlands 45	1991 (1117 adults) (See Table 3 for children)	Cross-sectional Doctor diagnosed asthma: 1.2 (0.8-1.9) Current asthma medication: 1.2 (0.4-3.2) Wheeze >1wk, past 2yrs: 1.1 (0.6-1.8) Chronic obstructive pulmonary disease medication, chronic cough, chronic phlegm: all neg.	 Living on busy streets (high NO_2) vs. living on calm streets

OR adjusted for covariates unless noted. # p<0.10 * p<0.05 **p<0.01

Appendix 2.7: Summary of lung-function for traffic-related pollutants-children and adults. Modified from Health Effect Institute report on traffic-related air pollution [16]. (up to September15, 2009).

Author Location	Year of study/ duration (N) Age of Observation	Group characteristic	Time domain of effect	Effect Estimate (95% CI)	Effect scale and metric
Brunekreef et al 1997 6 cities in Netherlands [67]	1995 / cross-section (877) Age 7-12	13 schools	Long-term	FEV_1 % change -3.7 (-7.2;-0.2)	[BS] in school (per 10 µg/m^3)
Fritz & Herbarth 2001 Leipzig, Germany [34]	1994/95 (235) Age 4-6	Cross-section Pre-schoolers from 16 day-care centers	Long-term	Coal-heated areas: FVC pred: -5% FEV_1 pred: -9% Centrally heated areas: FVC pred: -3% FEV_1 pred: -15%	Benzene (spatial measurements) 'low' vs. 'high' traffic
Hirsch, 1999 Dresden, Germany [56]	1995/96 (981) Age 5-7 & 9-11	random sample of 4th graders	Long-term	POR for $FEF_{25-75\%}$<70% pred.: 1.27 (1.03-1.58) FEV_1<85% pred.: 1.19 (0.86-1.64)	Benzene (spatial measures, home outdoor) Per +1 µg·m^3
Hogervorsrt et al 2006 Maastricht, Netherlands [68]	2002 / cross-section (342) Age 8-13	Six schools,	Long-term	Only sign. result: OH-formation of PM2.5 on FEV1: -1.57 (-2.88;-0.24)	PM radical-generating capacity, PM mass 1x10-6 unit increase of peak area / m3 air
Hong et al 2005 Incheon, Korea [69]	2002 (293) university students Age ~23 to 27	Student sample	Long-term	FEV_1 – 0.0063** (0.0035) FEF_{25-75}: -0.0140* (0.007)	Regression coefficients (SE) Personal NO_2 exposure (1-week mean) with pulmonary function
Schindler, 1998 [70]	1991 Cross-section (7641) (personal NO_2) Age 18-60	Random population sample	Long-term	Personal NO_2: FVC: -0.74% (-1.41%; -0.07%) FEV1: -0.26% (-1.03%; 0.52%)	Measured personal and home outdoor NO_2 (N=423) and assigned by neighborhood zone Per 10 µg/m3
Nordling et al. 2008 4 Swedish municipalities [71]	1994-1996 (2565) Age 4 years	Birth cohort with peak flow measured at age 4 years	Cross-section	-5.36 (0.1067—0.053) -3.08 (-6.84-0.68)	Traffic-PM10 per IQR=6 ug/m3 Traffic-NOx per IQR=44 ug/m3

Appendix 3

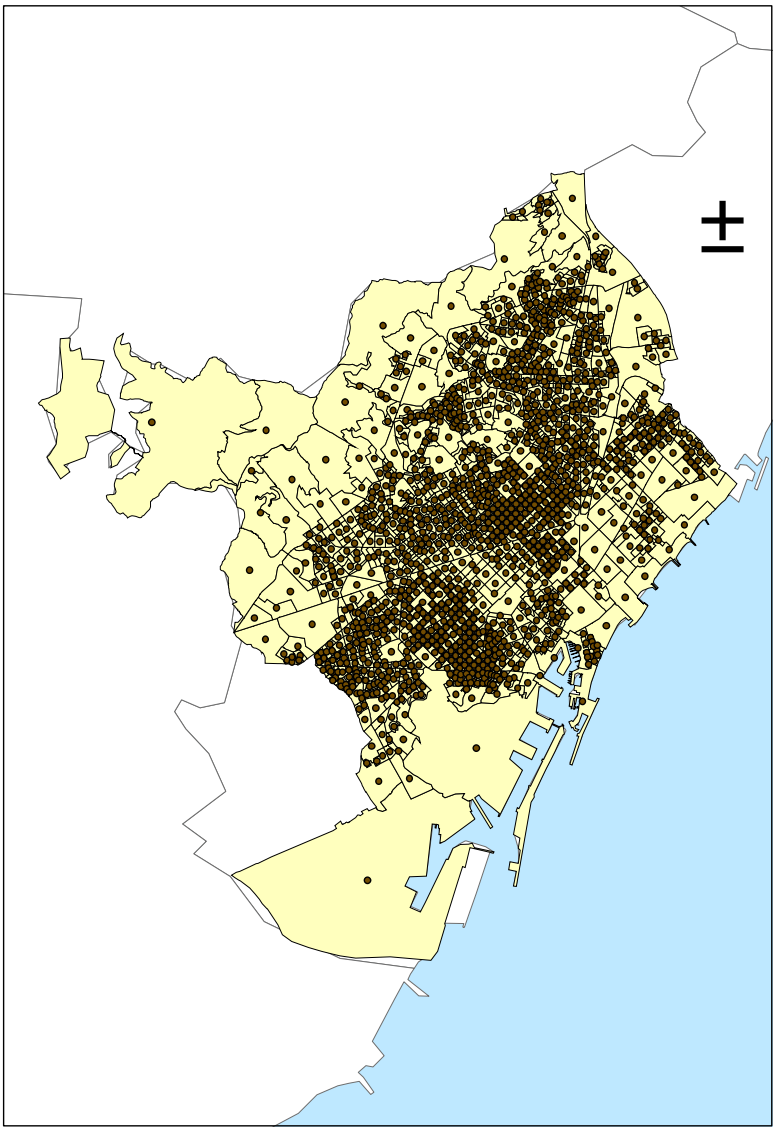
Step by step example to derive the percentage of people living in proximity to "major" roads

Example: Barcelona

Data availability:
Traffic data
Classification of all roads by categories
Counts per day for all streets by modelling
Total population distribution by census block

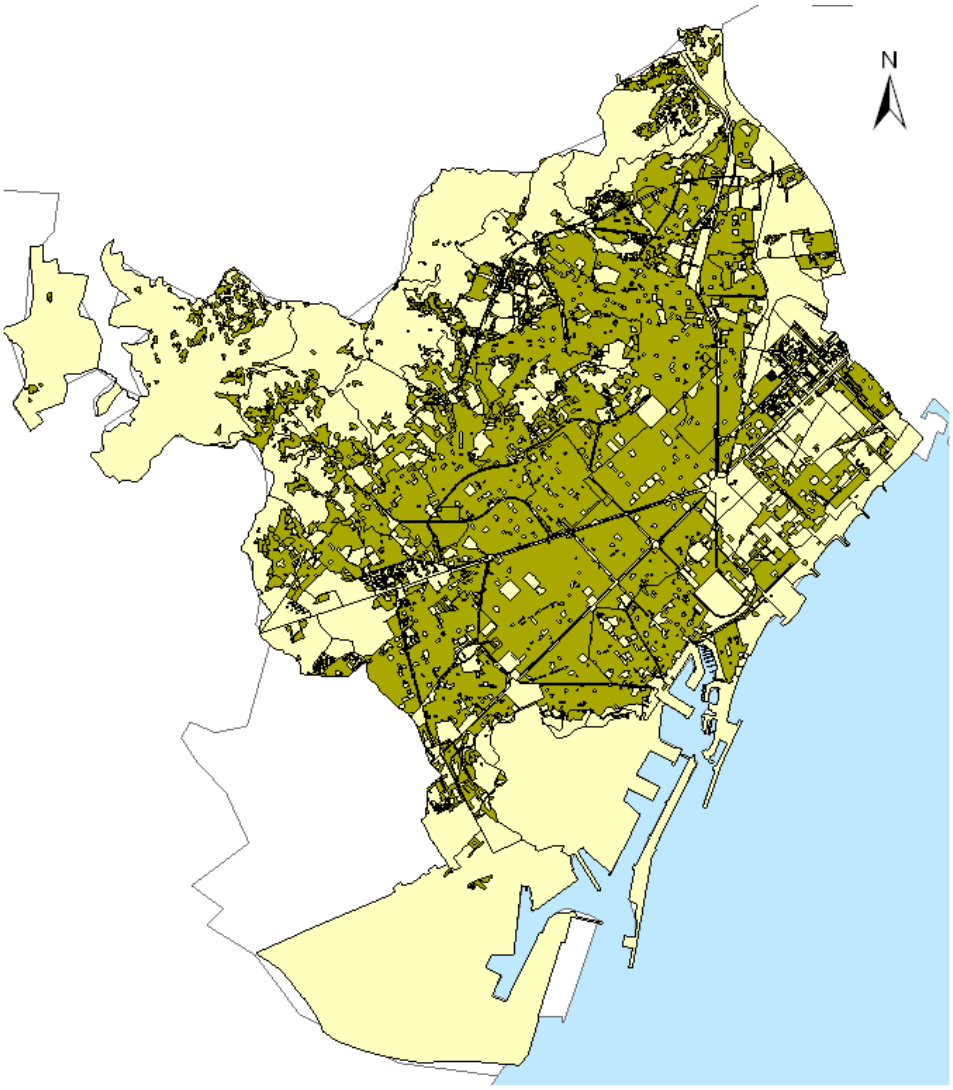
Step 1

- Overlay census section areas and population data



Step 2

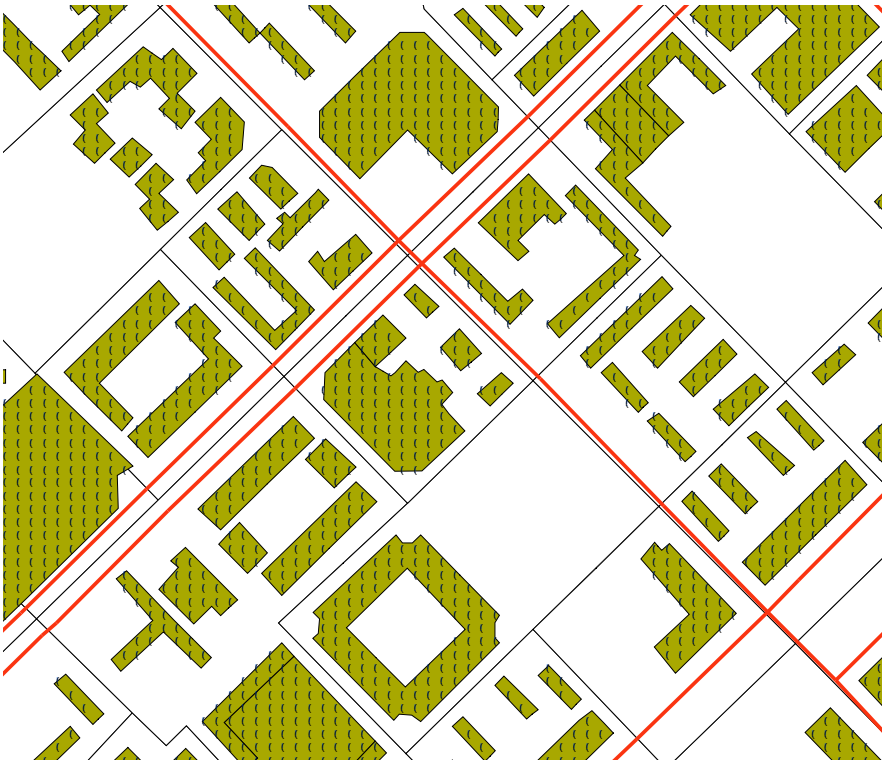
- Add urban land cover
- Remove geographical features where no residency is possible such as
  - Public buildings (schools, hospitals, commercial units…)
  - Industrial area
  - Green areas (parks)
  - Commercial areas



Step 3

- Build a grid of points over the urban land cover (minus feature removed). Grid will depend on household density in area of study or estimated household area (in Barcelona we used a grid of 10x10m).

- Spread the total population of the census over the number of grid points (within census)



Step 4
Selection of major road according to definition used for Concentration-Response Functions (i.e all roads or road segments with vehicle per day >10,000)

Step 5
Calculate distance of each grid point from nearest road classified as “major road” to obtain distribution table as below

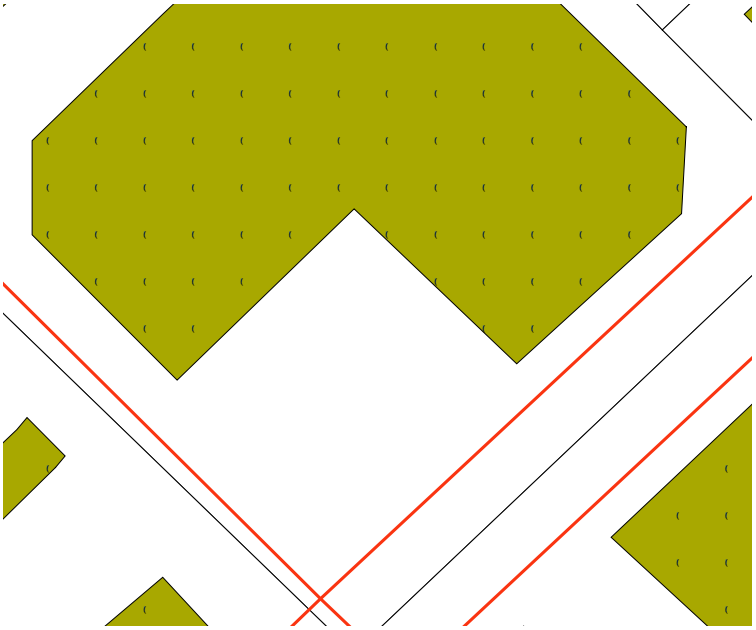


Table of the frequency distribution from nearest major road (grouped by specific distance)

Distance from major road	Population (all ages)	%
≤20m	823942	51.2
21-50m	552052	34.3
51-75m	144613	9.0
76-100m	50918	3.2
101-150m	38768	2.4
201-300m	292	0.0
301-400m	47	0.0
401-500m	0	0.0
> 500m	51	0.0

Process can be repeated from step 5 for different subgroups of population (e.g. by age or SES indicators).