

CARTEEH

Weekly GRA Meeting





What I've learned from the papers

- What are Census blocks?
- What is the question of interest?
- Is there an association between the risk factor and outcome? (1-2)
- How is the risk factor measured? (3-4)
- How is the exposure to the risk factor quantified ? (5)
- How is the burden calculated ? (6-7)



What is the question of interest?



What is the question of interest?

- What is the Burden of disease of incidence/prevalence of childhood asthma due to traffic related air pollution (TRAP)?
- Burden of disease include (but not limited to):
 - Exacerbations
 - Hospitalizations
 - Mortalities
 - Cost



Is there an association between the risk factor and outcome?

2 papers

ABSTRACT

It is widely accepted that air pollution can exacerbate asthma in those who already have the condition. What is less clear is whether air pollution can contribute to the initiation of new cases of asthma. Mechanistic evidence from toxicological studies, together with recent information on genes that predispose towards the development of asthma, suggests that this is biologically plausible, particularly in the light of the current understanding of asthma as a complex disease with a variety of phenotypes. The epidemiological evidence for associations between ambient levels of air pollutants and asthma prevalence at a whole community level is unconvincing; meta-analysis confirms a lack of association. In contrast, a meta-analysis of cohort studies found an association between asthma incidence and within-community variations in air pollureview suggests an association of asthma prevalence with exposure to traffic, although only in those living very close to heavily trafficked roads carrying a lot of trucks. Based on this evidence, the UK's Committee on the Medical Effects of Air Pollutants recently concluded that, overall, the evidence is consistent with the possibility that outdoor air pollution might play a role in causing asthma in susceptible individuals living very close to busy roads carrying a lot of truck traffic. Nonetheless, the effect on public health is unlikely to be large: air pollutants are likely to make only a small contribution, compared with other factors, in the development of asthma, and in only a small proportion of the population.

Key words: air pollution, asthma, incidence, review, vehicle emission.

2012

Gowers, A.M., Cullinan, P., Ayres, J.G., ANDERSON, H., Strachan, D.P., Holgate, S.T., Mills, I.C. and Maynard, R.L., 2012. Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. Respirology, 17(6), pp.887-898.

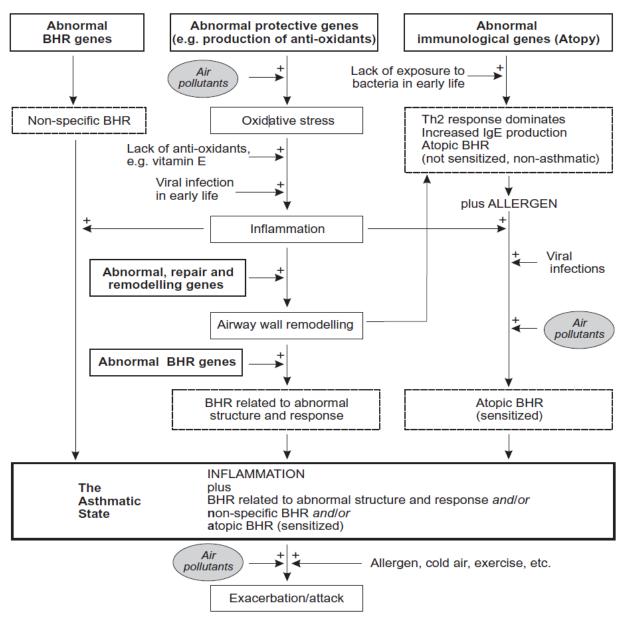


Figure 1 Hypothetical network of causation of asthma in relation to air pollution exposure. NB: Air pollutants could also upregulate or downregulate genes involved in these processes. BHR, bronchial hyperresponsiveness; IgE, immunoglobulin E; Th2, Type 2 helper T cells. Source: COMEAP (2010).³

- The main conclusions of this systematic literature review were that:
- 1 asthma prevalence is associated with reported exposure to truck traffic;
- 2 proximity to traffic appears to be less strongly and less consistently associated with measures of asthma prevalence than exposure to heavy goods vehicle traffic;
- 3 the evidence seems to suggest that the association with asthma prevalence and traffic-related air pollution occurs only in those living very close to the roadsides;
- 4 there is no association between objective markers of allergic sensitization and traffic-related air pollution.

Conclusion

- No Association between community level air pollution and incidence of asthma
- Evidence seems to suggest and association with traffic-related air pollution.

Exposure to traffic-related air pollution and risk of development of childhood asthma: A systematic review and meta-analysis



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ABSTRACT

Background and objective: The question of whether children's exposure to traffic-related air pollution (TRAP) contributes to their development of asthma is unresolved. We conducted a systematic review and performed meta-analyses to analyze the association between TRAP and asthma development in childhood.

Data sources: We systematically reviewed epidemiological studies published until 8 September 2016 and available in the Embase, Ovid MEDLINE (R), and Transport databases.

Study eligibility criteria, participants, and interventions: We included studies that examined the association between children's exposure to TRAP metrics and their risk of 'asthma' incidence or lifetime prevalence, from birth to age 18 years old.

Study appraisal and synthesis methods: We extracted key characteristics of each included study using a predefined data items template and these were tabulated. We used the Critical Appraisal Skills Programme checklists to assess the validity of each included study. Where four or more independent risk estimates were available for a continuous pollutant exposure, we conducted overall and age-specific meta-analyses, and four sensitivity analyses for each summary meta-analytic exposure-outcome association.

Results: Forty-one studies met our eligibility criteria. There was notable variability in asthma definitions, TRAP exposure assessment methods and confounder adjustment. The overall random-effects risk estimates (95% CI) were 1.08 (1.03, 1.14) per 0.5×10^{-5} m⁻¹ black carbon (BC), 1.05 (1.02, 1.07) per $4 \,\mu\text{g/m}^3$ nitrogen dioxide (NO₂), 1.48 (0.89, 2.45) per 30 $\mu\text{g/m}^3$ nitrogen oxides (NO_x), 1.03 (1.01, 1.05) per $1 \,\mu\text{g/m}^3$ Particulate Matter <2.5 μ m in diameter (PM_{2.5}), and 1.05 (1.02, 1.08) per $2 \,\mu\text{g/m}^3$ Particulate Matter <10 μ m in diameter (PM₁₀).

Sensitivity analyses supported these findings. Across the main analysis and age-specific analysis, the least heterogeneity was seen for the BC estimates, some heterogeneity for the $PM_{2.5}$ and PM_{10} estimates and the most heterogeneity for the NO_2 and NO_x estimates.

Limitations, conclusions and implication of key findings: The overall risk estimates from the meta-analyses showed statistically significant associations for BC, NO₂, PM_{2.5}, PM₁₀ exposures and risk of asthma development. Our findings support the hypothesis that childhood exposure to TRAP contributes to their development of asthma. Future meta-analyses would benefit from greater standardization of study methods including exposure assessment harmonization, outcome harmonization, confounders' harmonization and the inclusion of all important confounders in individual studies.

Systematic review registration number: PROSPERO 2014: CRD42014015448.

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- Results : 41 studies
- BC 1.08 (1.03 1.14) per 0.5 × 10–5 m–1 NO2 1.05 (1.02 – 1.07) per 4 μg/m3
- NOx 1.48 (0.89 2.54) per 30 μg/m₃
- PM2.5 1.03 (1.01 1.05) *per 1 μg/m*3
- PM10 1.05 (1.02 1.08) per 2 μg/m₃
- 2016



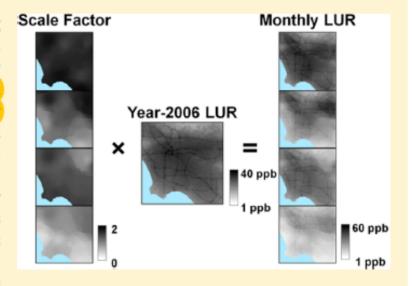
How is the risk factor measured?

2 papers

National Spatiotemporal Exposure Surface for NO₂: Monthly Scaling of a Satellite-Derived Land-Use Regression, 2000–2010

Matthew J. Bechle, Dylan B. Millet, and Julian D. Marshall*,

ABSTRACT: Land-use regression (LUR) is widely used for estimating within-urban variability in air pollution. While LUR has recently been extended to national and continental scales, these models are typically for long-term averages. Here we present NO₂ surfaces for the continental United States with excellent spatial resolution (~100 m) and monthly average concentrations for one decade. We investigate multiple potential data sources (e.g., satellite column and surface estimates, high- and standard-resolution satellite data, and a mechanistic model [WRF-Chem]), approaches to model building (e.g., one model for the whole country versus having separate models for urban and rural areas, monthly LURs versus temporal scaling of a spatial LUR), and spatial interpolation methods for temporal scaling factors (e.g., kriging versus inverse distance weighted). Our core approach uses NO₂ measurements from U.S. EPA monitors



(2000–2010) to build a spatial LUR and to calculate spatially varying temporal scaling factors. The model captures 82% of the spatial and 76% of the temporal variability (population-weighted average) of monthly mean NO₂ concentrations from U.S. EPA monitors with low average bias (21%) and error (2.4 ppb). Model performance in absolute terms is similar near versus far from monitors, and in urban, suburban, and rural locations (mean absolute error 2–3 ppb); since low-density locations generally experience lower concentrations, model performance in relative terms is better near monitors than far from monitors (mean bias 3% versus 40%) and is better for urban and suburban locations (1–6%) than for rural locations (78%, reflecting the relatively clean conditions in many rural areas). During 2000–2010, population-weighted mean NO₂ exposure decreased 42% (1.0 ppb [~5.2%] per year), from 23.2 ppb (year 2000) to 13.5 ppb (year 2010). We apply our approach to all U.S. Census blocks in the contiguous United States to provide 132 months of publicly available, high-resolution NO₂ concentration estimates.

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• Develop a spatiotemporal (space & time) model to estimate No2 concentrations using different data sources (Satellite Imagery)

• Space : Continental US (48 states)

• Time : Temporal scaling using monthly averages for 10 years

Prediction method: Kriging

• Estimation: Population weighted mean NO2

Table 2. Model Performance for Final Spatial LUR Model

	R^2	adj R²	mean error (ppb)	mean abs error (ppb)	mean bias (%)	mean abs bias (%)
all	0.79	0.79	-0.30	2.3	18	34
urban	0.76	0.76	-0.78	2.4	-1	18
rural	0.50	0.49	0.39	2.4	57	75
population- weighted	0.81	0.81	-0.71	2.4	-1	17

 Comparing urban vs rural vs population weighted prediction

• Population weighted performed the best (R2 = 0.81)

Table 3. Summary of Monthly Mean NO₂ Estimates Using Kriging Temporal Scaling^a (2000–2010)

	mean spatial R^2	mean temporal R^2	$\begin{array}{c} \text{spatiotemporal} \\ R^2 \end{array}$	mean concn (ppb)	mean error (ppb)	mean abs error (ppb)	mean bias (%)	mean abs bias (%)	mean (IQR^b) distance to nearest monitor (km)
all									
population- weighted	0.82	0.76	0.85	19.6	-0.3	2.7	2	18	32
unweighted	0.81	0.73	0.84	12.4	-0.05	2.4	21	38	48 (13-50)
distance to nearest monitor									
<10 k	0.80	0.72	0.82	16.3	-0.5	2.6	3	22	6 (4-8)
<25 km	0.82	0.73	0.84	15.4	-0.1	2.5	12	28	13 (8-18)
25-50 km	0.70	0.79	0.77	10.6	-0.06	2.1	23	40	36 (29-40)
>50 km	0.71	0.69	0.75	9.5	0.1	2.2	40	57	133 (65-158)
urban classification									
urban	0.76	0.76	0.80	16.2	-0.5	2.7	1	20	12 (7-16)
suburban	0.76	0.76	0.81	13.6	-0.02	2.4	6	22	30 (21-38)
rural	0.63	0.63	0.69	6.0	0.4	2.0	78	95	104 (36-136)

[&]quot;All values except those in the first row are unweighted metrics. "IQR is the interquartile range.

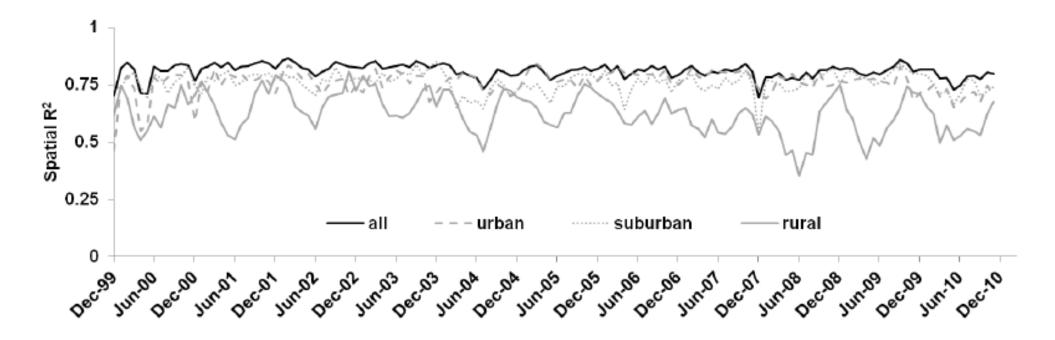


Figure S8. Spatial R^2 for each month. Solid black line shows spatial R^2 for all valid monitors for that month. Long-dash, short-dash, and grey lines show urban, suburban, and rural monitor locations.



How is the exposure to the risk factor quantified?

1 papers

An Attributable Risk Model for Exposures Assumed to Cause Both Chronic Disease and its Exacerbations

Nino Künzli,*†‡ Laura Perez,† Fred Lurmann,§ Andrea Hricko,‡ Bryan Penfold,§ and Rob McConnell‡

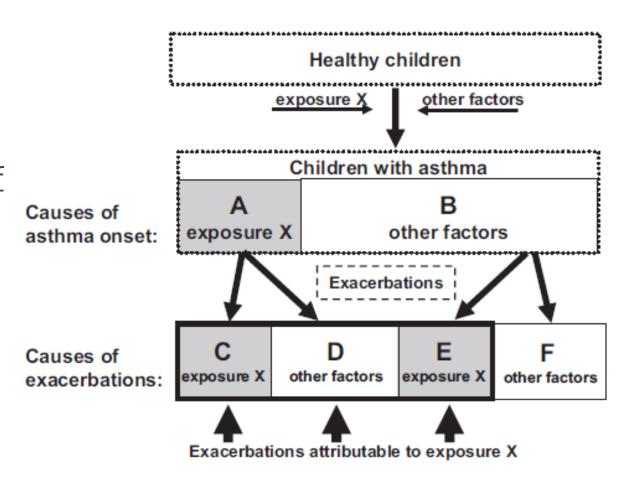
obstructive lung diseases, or atherosclerosis. For exposures affecting both the development of chronic disease and its exacerbation, the usual methods to derive attributable risks (AR) are inappropriate.

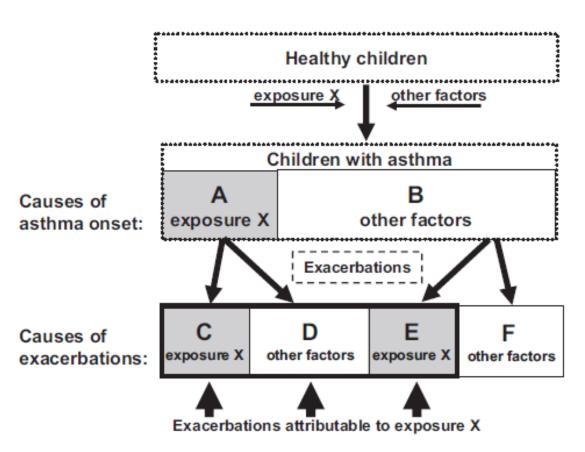
Methods: We expand traditional risk assessment methods to estimate the AR for exacerbations under a "chronic disease model." We

- Traditional Attributable Risk (AR) models for chronic diseases are inappropriate
- Reduce the burden of disease attributable to the exposure causing the disease
- Expand the AR model
- Provides the formulas for calculation
- 2008

Chronic disease and subsequent exacerbations

• If an exposure to a "Risk factor" has a causal association with the development of the chronic disease, then all subsequent exacerbations of the chronic disease related to the exposure (directly or indirectly) should be included in the estimation of the burden of disease.





- Traditional AR models only account for exacerbation directly induced by the "exposure X" (cells C + E)
 - Unaccounting for indirect exacerbations
 - Underestimating the true burden of the exposure
- "Chronic AR Model" accounts for all exacerbations directly/indirectly induced by "exposure X" (cells C + D + E)



How is the burden calculated?

2 papers



Global Goods Movement and the Local Burden of Childhood Asthma in Southern California

Laura Perez, MS, Nino Künzli, MD, PhD, Ed Avol, MS, Andrea M. Hricko, MPH, Fred Lurmann, MS, Elisa Nicholas, MD, Frank Gilliland, MD, PhD, John Peters, MD, ScD, and Rob McConnell, MD

Objectives. As part of a community-based participatory research effort, we estimated the preventable burden of childhood asthma associated with air pollution in the southern California communities of Long Beach and Riverside.

Methods. We calculated attributable fractions for 2 air pollution reduction scenarios to include assessment of the newly recognized health effects associated with residential proximity to major roads and impact from ship emissions.

Results. Approximately 1600 (9%) of all childhood asthma cases in Long Beach and 690 (6%) in Riverside were attributed to traffic proximity. Ship emissions accounted for 1400 (21%) bronchitis episodes and, in more modest proportions, health care visits for asthma. Considerably greater reductions in asthma morbidity could be obtained by reducing nitrogen dioxide and ozone concentrations to levels found in clean coastal communities.

Conclusions. Both Long Beach and Riverside have heavy automobile traffic corridors as well as truck traffic and regional pollution originating in the Los Angeles–Long Beach port complex, the largest in the United States. Community-based quantitative risk analyses can improve our understanding of health problems and help promote public health in transportation planning. (Am J Public Health. 2009;99:S622–S628. doi:10.2105/AJPH.2008.154955)

- Tested the AR Model for Chronic disease
- Exposure : No2 & O3
- Outcome
 - Prevalent asthma among children
 - Bronchitis exacerbation
 - Clinic visits
 - ER visits
 - Hospital admission
- Attributable burden

Derivation of population attributable risk fraction

- Concentration response function
- Frequency of health condition in the population of interest
- Current population exposure
- Population exposure hypothesized after exposure (source) reduction

Concentration response function

TABLE 1—Health Outcomes and Concentration Response Functions (CRFs) Among Children: Long Beach and Riverside, CA

Outcome	Unit of CRF	Age, y	CRF (95% CI)	Corrected CRF (95% CI)	Study
Bronchitis episodes among those					
with asthma					
NO_2	1 ppb (24-h annual average)	9-13	1.070 (1.020, 1.130)	1.042 (1.012, 1.076)	McConnell et al. 12
0 ₃	1 ppb (10:00 AM-6:00 PM average)	9-13	1.060 (1.000, 1.120)	1.057 (1.000, 1.113)	McConnell et al. 12
Clinic visits for asthma					
NO_2	24 ppb (24-h average)	0-14	1.061 (1.012, 1.113)	1.055 (1.011, 1.102)	Hajat et al. 13
0 ₃	50 ppb (24-h average)	2-14	1.054 (1.013, 1.096)	1.049 (1.012, 1.086)	Ostro et al.14
Emergency department visits					
for asthma					
NO_2	50 μg/m³ (27 ppb³) (24-h average)	≤15	1.026 (1.006, 1.049)	1.024 (1.006, 1.045)	Sunyer et al.15
0 ₃	10 ppb (daily 1-h max)	1-16	1.024 (1.015, 1.033)	1.022 (1.014, 1.030)	Ostro et al.2
Hospital admissions for asthma					
NO_2	27.1 μg/m ³ (14.4 ppb ^a) (24-h average)	≤15	1.079 (1.054, 1.090)	1.079 (1.054, 1.090)	Lee et al.16
0 ₃	23 μg/m³ (11.5 ppb ^b) (8-h mean)	≤15	1.060 (1.041, 1.079)	1.060 (1.041, 1.079)	Lee et el.16
Prevalent asthma: traffic exposure	Living < 75 m from busy road for long-term residents	5-7	1.64 (1.10, 2.44)	1.515 (1.086, 2.059)	McConnell et al. ¹⁰

Note. CI = confidence interval; NO_2 = nitrogen dioxide; O_3 = ozone. Published CRFs were corrected with the formula CRF/[1+I_t(CRF-1)], where I_t is the frequency of the outcome in the population. ^aConversion factor for NO_2 : 1 ppb = 1.88 μ g/m³.

bConversion factor for O₃: 1 ppb=2 µg/m³.

Frequency of health condition in the population of interest

TABLE 2—Population Baseline Frequencies and Exposure Data Among Children: Long Beach and Riverside, CA

Description	Long Beach, CA	Riverside, CA	Data Source
Population or baseline frequencies			
Total population of children aged 0-17 y	136 181	76 491	2000 US Census Bureau
Fraction of children with asthma (prevalence) ^a	0.1284	0.1488	CHS ^b
Fraction of children with asthma with reported bronchitis symptoms ^c	0.387	0.387	McConnell et al. ¹²
Fraction of children with reported clinic visits for asthma ^d	0.710	0.7521	CHS ^b
Fraction of children with asthma with reported emergency department visits for asthma ^e	0.581	0.3793	CHS ^b
Number of hospital admissions for asthma (ICD-9, 493)	264	120	California Breathing, 2003 ^f

Current population exposure & reduction scenario

Population exposure—current conditions			
NO ₂	33 ppb	26 ppb	CHS Web page ¹⁷
0 ₃	29 ppb	57 ppb	CHS Web page ¹⁷
Exposi	ire reduction from current	levels	
Scenario 1: no ship emissions			
NO ₂	-5.9 ppb	-2.0 ppb	Vutukuru and Dabdub ⁴
0 ₃	Not considered	Not considered	
Scenario 2: clean communities			
NO ₂	-18 ppb	-11 ppb	Clean CHS communities ¹⁷
0 ₃	Not considered	-27 ppb	Clean CHS communities ¹⁷

Population exposure hypothesized after exposure (source) reduction

Table 3-Number of Outcomes Attributable to Air Pollution Per Year Among Children: Long Beach and Riverside, CA

			Scenario 1: No	Ship Emissions			Scenario 2: Clea	n Communities	
		Attributable	Attributable	Tota	ll ^b	Attributable	Attributable	Tota	al ^b
Community and Outcome	Baseline Estimate, No.	to Air Pollution, No. (95% CI)	No. (95% CI)	(No. Cases)	% (95% CI)	to Air Pollution, No. (95% CI)	to Other Causes, ^a No. (95% CI)	No. Cases (95% CI)	% (95% CI)
				Traffic exposure					
Long Beach: asthma cases	17 486	1600 (1500, 1800)	NA	1600 (1500, 1800)	9.2 (8.6, 10.3)	1600 (1500, 1800)	NA	1600 (1500, 1800)	9.2 (8.6, 10.3)
Riverside: asthma cases	11 382	690 (630, 750)	NA	690 (630, 750)	6.1 (5.5, 6.6)	690 (630, 750)	NA	690 (630, 750)	6.1 (5.5, 6.6)
				NO ₂					
Long Beach									
Bronchitis episodes among those with asthma	6767	1400 (400, 2300)	500 (400, 600)	1900 (980, 2700)	28.1 (14.5, 39.9)	3400 (1200, 4900)	310 (170, 530)	3700 (1700, 5100)	54.7 (25.1, 75.4)
Emergency room visits for asthma	10 166	<mark>54</mark> (7, 100)	940 (860, 1020)	1000 (910, 1100)	9.8 (9.0, 10.8)	160 (20, 300)	930 (860, 1000)	1100 (950, 1200)	10.8 (9.3, 11.8)
Clinic visits for asthma	12 410	160 (30, 290)	1100 (1000, 1200)	1300 (1200, 1400)	10.5 (9.7, 11.3)	500 (90, 860)	1100 (1000, 1200)	1600 (1200, 2000)	12.9 (9.7, 16.1)
Hospital admissions for asthma	264	10 (8, 12)	24 (22, 26)	34 (31, 36)	12.9 (11.7, 13.6)	30 (24, 35)	22 (20, 24)	51 (46, 57)	19.3 (17.4, 21.6)
Riverside									
Bronchitis episodes among those with asthma	4405	340 (90, 590)	250 (220, 270)	590 (350, 820)	13.4 (7.9, 18.6)	1600 (470, 2400)	170 (120, 240)	1700 (710, 2500)	38.6 (16.1, 56.8)
Emergency room visits for asthma	4317	8 (1, 15)	260 (240, 280)	270 (240, 290)	6.3 (5.6, 6.7)	42 (5, 73)	260 (240, 280)	300 (260, 340)	6.9 (6.0, 7.9)
Clinic visits for asthma	8560	40 (7, 70)	510 (470, 560)	550 (500, 600)	6.4 (5.8, 7.0)	200 (40, 370)	500 (460, 550)	710 (550, 870)	8.3 (6.4, 10.2)
Hospital admissions for asthma	120	2 (1, 2)	4 (4, 4)	6 (5, 6)	5.0 (4.2, 5.0)	8 (7, 10)	4 (3, 4)	12 (11, 14)	10.0 (9.2, 11.7)
				Ozone					
Riverside									
Bronchitis episodes among those with asthma	4405	NC	NC	NC	NC	3100 (170, 4200)	80 (14, 250)	3200 (420, 4200)	72.6 (9.5, 93.3)
Emergency room visits for asthma	4317	NC	NC	NC	NC	250 (160, 330)	245 (223, 267)	508 (417, 595)	11.8 (9.7, 13.8)
Clinic visits for asthma	8560	NC	NC	NC	NC	220 (20, 410)	250 (220, 270)	490 (410, 580)	5.7 (4.8, 6.8)
Hospital admissions for asthma	120	NC	NC	NC	NC	12 (9, 16)	4 (3, 4)	16 (13, 19)	13.3 (10.8, 15.8)

Note. CI = confidence interval; NA = not applicable; NC = not considered. Uncertainty distributions obtained by Monte Carlo simulations.

^aAmong those with asthma caused by pollution.

^bNot to be summed from other columns.

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

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

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 population attributable risk fraction

Derivation of attributable cases required combining concentration-response function (CRF)

between exposures and the respective health outcome of interest (obtained from published

literature), an estimate of the distribution of selected exposures in the target population, and

information about the frequency of the assessed morbidities.

• Concentration – response function

Frequency of health condition in the population of interest

Current population exposure

Estimating the attributable burden

Concentration response function

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

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

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

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

Frequency of health condition in the population of interest

Table 3. Summary of population and heath baseline frequencies

Outcome	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Sevilla	Stockholm	Valencia	Vienna	10 cities
Asthma											
Total population aged 0-17	261241 (2008)#	49327 (2006)	227402 (2007)	44734 (2007)#	52255 (2002)	439543 (2009)	140373 (2007)#	264960 (2007)	117040 (2001)	291213 (2007)	1890000
Fraction with asthma (asthma ever)##	11.8%	21.3%	7.3%	12.6%	29.2%	12.6%	13.0%	9.3%	11.0%	5.8%	12.8%
Fraction with symptoms of asthma among those with current wheeze##	29.0%	37.0%	39.2%	37.1%	34.2%	32.9%	37.1%	35.6%	36.5%	33.1%	38.0%
Total number of hospitalizations for asthma (ICD-10: J45-J46)	98 (2008)	230 (2006)	356 (2006)	39 (2008)	100 (2007)	482 (2008)	28 (2008)	219#	40 (2002)	149 (2007)	1702

Current population exposure

Table 4. City specific summaries of population distribution in two distance based buffers around busy roads (i.e. >10000 vehicles per day) and annual mean concentrations of PM₁₀ and NO₂ at urban background stations.

Indicator	Unit	Barcelona	Bilbao	Brussels	Granada	Ljubljana	Rome	Sevilla	Stockholm##	Valencia	Vienna	10 cities#
Traffic proximity (%)	year	2007	2006	2002	2008	2006	2008	2004	2004	2007-2008	2006	
	≤75m	56%	29%	37%	14%	23%	22%	20%	14%	44%	36%	31%
	≤150m	77%	59%	64%	28%	47%	43%	38%	30%	71%	62%	53%
Urban												
background	year	2008	2008	2007	2008	2007	2009	2008	2007	2003	2007	
pollution												
PM ₁₀ annual avera	ge $(\mu g/m^3)$	33	27	29	34	32	37	41	17	27	25	30.3
NO ₂ annual averag	ge $(\mu g/m^3)$	36	29	38	31	28	61	29	13	51	32	39.4

Estimating the attributable burden

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

		Asthma (0-17)	
C't-	Estimated with	Attributed to near-ro	oad traffic-related
City	chronic	pollutio	on##
	outcome	prevalent cases	prevalent fraction
Barcelona	30690	6900 (1400; 11500)	23% (5%; 38%)
Bilbao	10500	1200 (200; 2100)	12% (2%; 20%)
10 Cities	240730	33200 (6200; 59600)	14% (3%; 25%)

Estimating the attributable burden

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

	Estimated	Exacerbations attr	ibutable to air pollution asthma due to	among those with	Exacerbation attributable to other causes among those	Total attributable or pollut	
City	with outcome	near-road traffic- related pollution	other causes	total	with asthma due to near- road traffic-related pollution	cases	% with [1] as denominator
Referenced in text	[1]	[2]	[3]	[4]	[5]	[6]	
Episodes symptoms asthma							
Barcelona	8910	39 (4; 85)	135 (55; 235)	175 (75; 285)	1900 (200; 3600)	2100 (400; 3800)	24% (5%; 43%
Bilbao	3880	9 (0; 24)	100 (40; 170)	110 (45; 185)	400 (0; 900)	500 (0; 1000)	14% (1%; 28%
Brussels	6460	16 (1; 37)	75 (25; 135)	90 (30; 155)	1100 (100; 2100)	1200 (200; 2200)	19% (4%; 34%
Granada	2090	3 (0; 8)	45 (15; 75)	45 (15; 80)	100 (0; 200)	100 (0; 300)	9% (3%; 17%
Ljubljana	5660	9 (-1.2; 25)	105 (40; 180)	115 (40; 195)	400 (-58.8; 1000)	500 (0; 1100)	11% (1%; 23%
Rome	18870	61 (4; 145)	535 (220; 890)	595 (250; 980)	1800 (100; 3800)	2400 (700; 4500)	13% (4%; 24%
Sevilla	6560	21 (1; 53)	205 (80; 350)	230 (85; 385)	600 (0; 1200)	800 (200; 1500)	13% (4%; 23%
Stockholm	8750	0 (0; 0)	0 (0; 0)	0 (0; 0)	600 (0; 1300)	600 (0; 1300)	7% (1%; 16%
Valencia	4670	10 (0; 22)	40 (15; 75)	50 (20; 85)	800 (100; 1600)	900 (100; 1700)	20% (4%; 37%
Vienna	5590	8 (1; 19)	40 (15; 70)	50 (20; 80)	900 (100; 1800)	1000 (200; 1800)	18% (4%; 33%
10 cities	91590	220 (15; 520)	1365 (505; 2375)	1585 (590; 2690)	12400 (1200; 25000)	14000 (2800; 26800)	15% (3%; 29%
Hospital admissions							
Barcelona	90	0.28 (0.001; 0.69)	0.9 (0.1; 1.9)	1.2 (0.1; 2.3)	21 (2; 38)	22 (4; 40)	23% (4%; 419
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%; 339
Granada	30	0.04 (0; 0.12)	0.6 (0; 1.1)	0.6 (0; 1.2)	2 (0; 5)	3 (0; 6)	8% (2%; 16%
Ljubljana	100	0.12 (0; 0.36)	1.3 (0.1; 2.5)	1.5 (0.2; 2.7)	8 (0; 18)	9 (0; 20)	10% (0.4%; 20
Rome	480	1.07 (0; 2.82)	9.3 (1.2; 17.5)	10.4 (1.4; 19.3)	48 (4; 96)	58 (14; 108)	12% (3%; 22%
Sevilla	20	0.06 (0; 0.18)	0.6 (0; 1.2)	0.7 (0.1; 1.3)	2 (0; 5)	3 (0; 6)	12% (3%; 229
Stockholm	210	0 (0; 0)	0 (0; 0)	0 (0; 0)	16 (2; 33)	16 (2; 33)	7% (1%; 15%
Valencia	40	0.06 (0; 0.15)	0.2 (0; 0.5)	0.3 (0; 0.6)	7 (0; 13)	7 (1; 14)	20% (3%; 359
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%; 329
10 cities	1740	3.1 (0; 8.1)	15 (0; 35)	20 (0; 40)	230 (20; 460)	260 (40; 480)	15% (3%; 289
July 22, 2012 F00	iter text here						

July 22, 2012



Part 2 - Data set



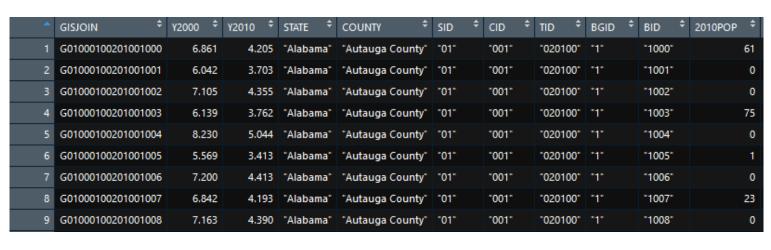
35 July 22, 2012 Footer text here

Viewing the data set

*	GISJOIN ‡	Y2000 ÷	Y2010 ÷
1	G01000100201001000	6.861	4.205
2	G01000100201001001	6.042	3.703
3	G01000100201001002	7.105	4.355
4	G01000100201001003	6.139	3.762
5	G01000100201001004	8.230	5.044
6	G01000100201001005	5.569	3.413
7	G01000100201001006	7.200	4.413
8	G01000100201001007	6.842	4.193
9	G01000100201001008	7.163	4.390
10	G01000100201001009	6.305	3.864

- "processed/BechleLUR_annual2000-2010_2010blocks.csv"
- Processed LUR data set
- NO2 levels by census block
 - Each row represents a census block or observation
- Year 2000 & 2010
- GISJOIN is the unique identifier

Viewing the data set



 "processed/US_Census_data_blockinf o_201oblocks.csv"

- Geographical information
 - State
 - County
 - SID, CID, TID ...etc ?? (BGID Block group id, BID – Block identifier?)
- 2010 population by census block ??

Merging the two sets

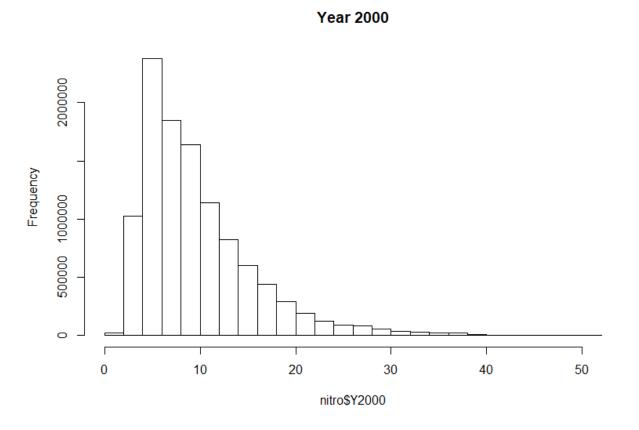
	*	GISJOIN ‡	Y2000 ‡	Y2010 ‡	STATE \$	COUNTY ‡	SID :	÷	CID	÷	TID ‡	BGID	‡	BID	‡	2010POP	‡
	1	G01000100201001000	6.861	4.205	"Alabama"	"Autauga County"	°01°		*001*		"020100"	111		"1000"			61
	2	G01000100201001001	6.042	3.703	"Alabama"	"Autauga County"	°01°		*001*		"020100"	717		"1001"			0
	3	G01000100201001002	7.105	4.355	"Alabama"	"Autauga County"	*01*		*001*		"020100"	717		"1002"			0
	4	G01000100201001003	6.139	3.762	"Alabama"	"Autauga County"	*01*		*001*		"020100"	"1"		"1003"			75
	5	G01000100201001004	8.230	5.044	"Alabama"	"Autauga County"	*01*		*001*		"020100"	717		"1004"			0
	6	G01000100201001005	5.569	3.413	"Alabama"	"Autauga County"	*01*		*001*		"020100"	"1"		"1005"			1
	7	G01000100201001006	7.200	4.413	"Alabama"	"Autauga County"	*01*		*001*		*020100*	717		"1006"			0
	8	G01000100201001007	6.842	4.193	"Alabama"	"Autauga County"	*01*		*001*		"020100"	111		"1007"			23
	9	G01000100201001008	7.163	4.390	"Alabama"	"Autauga County"	*01*		*001*		°020100°	717		"1008"			0
1	10	G01000100201001009	6.305	3.864	"Alabama"	"Autauga County"	°01°		°001°		"020100"	111		"1009"			1
1	11	G01000100201001010	6.593	4.041	"Alabama"	"Autauga County"	°01°		°001°		"020100"	717		~1010~			0

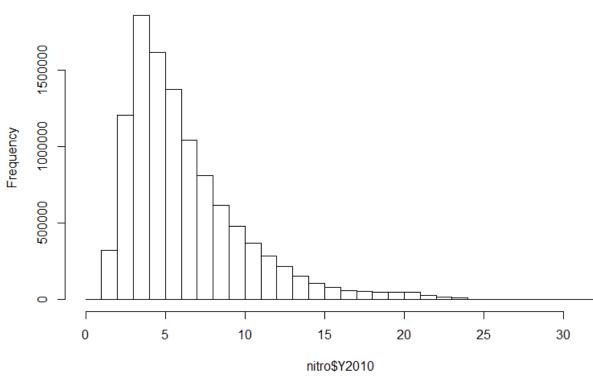
Summary stats

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$GISJOIN
[1] 11007989
$Y2000
[1] 11007989
$Y2010
[1] 11007989
```

- Number of observations = 11,007,989
- Na's = 114,332
- Mean 2000 = 9.69
- Mean 2010 = 6.32

```
$Y2000
   Min. 1st Qu.
                 Median
                           Mean 3rd Qu.
                                            Max.
                                                    NA's
           5.34
                   8.19
                           9.69
                                           51.24
  1.16
                                  12.26
                                                 114332
$Y2010
                 Median
                           Mean 3rd Qu.
                                                    NA's
   Min. 1st Qu.
                                            Max.
   0.72
           3.62
                   5.29
                           6.32
                                    7.90
                                           31.14
                                                  114332
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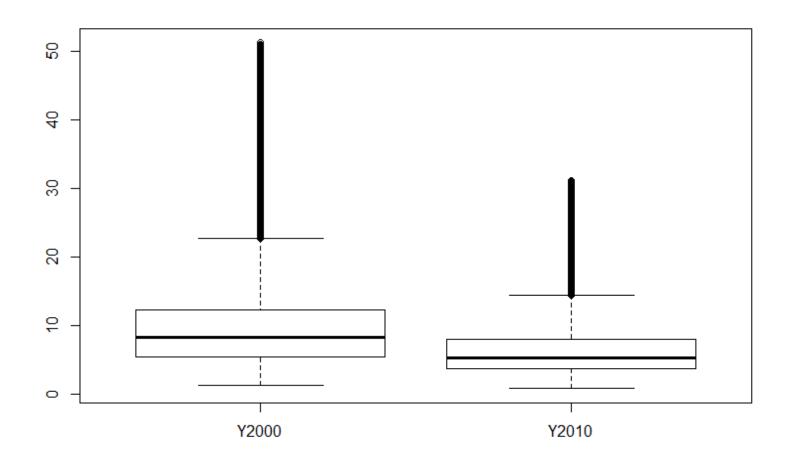




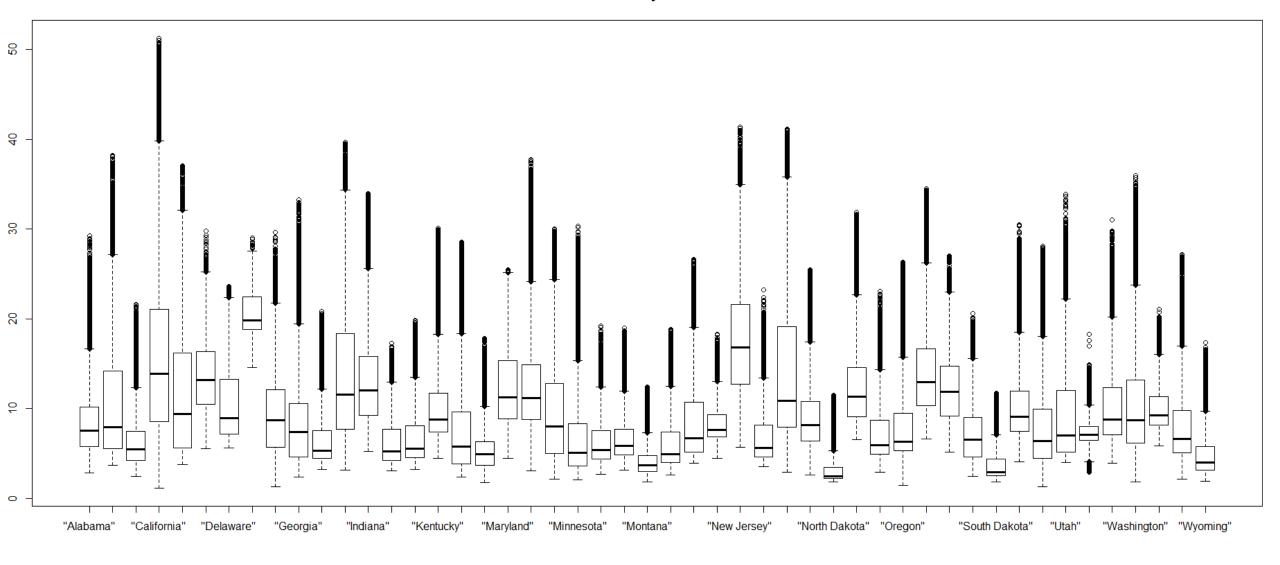
Year 2010

40

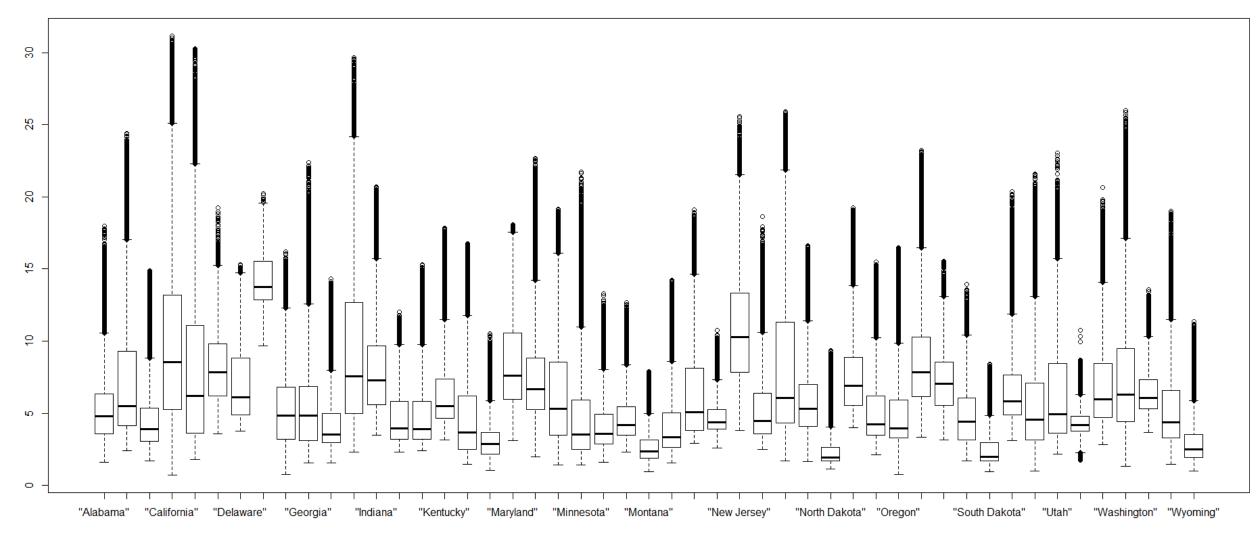
Boxplot



No2 variability Year 2000



No2 variability Year 2010



43