Impact of air pollution on Health



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Air pollution is one of the world's leading risk factors for death

Air pollution is responsible for 5 million deaths each year

Air pollution – the combination of outdoor and indoor particulate matter, and ozone – is a risk factor for many of the leading causes of death including heart disease, stroke, lower respiratory infections, lung cancer, diabetes and chronic obstructive pulmonary disease (COPD).

The Institute for Health Metrics and Evaluation (IHME) in its *Global Burden of Disease* study provide estimates of the number of deaths attributed to the range of risk factors for disease.¹

In the visualization we see the number of deaths per year attributed to each risk factor. This chart is shown for the global total, but can be explored for any country or region using the "change country" toggle.

Air pollution is one of the leading risk factors for death. In low-income countries it tops the list. In 2017, it was responsible for an estimated 5 million deaths globally. That means it contributed to 9% – nearly 1-in-10 – deaths.



https://ourworldindata.org/air-pollution

FIGURE 3. Particle matter (PM)₁₀ and PM_{2.5} in cities of Latin America and the Caribbean and their situation compared with the World Health Organization–Air Quality Guidelines (WHO-AQG), 2010–2014







Pollution exposure => Health effect

Since the 1990s epidemiological studies (Dockery et al., Shwartz et al., Pope et al....)



Different types of exposure :

London smog (1952) : 12000 deaths

-Short term (2 - 40 days) : better known (Daily Concentrations/Daily Mortality) -Long term (many years) => more severe effect and less wellknown (ex. => long-term exposure/mortality (Pope et al. 2002)), exposure/life expectancy (WHO 2003)

Dose-response functions (RR) : Correlation between polluant concentrations and health response hospital admissions/emergency visits for different types of diseases (morbidity) and premature deaths (mortality) : determination from time series (more frequent, short-term) and cohort studies (expensive, both short and long-term). Calculated with Poisson regression.

Cohort: More important effect for young children, elder people and sick people

HEALTH ENDPOINT	POLLUTANT	POP SECTOR	FUNCTION	SOURCE
Respiratory hospital admissions - daily exposures	PM10	All persons	1.20 x 10 ⁻⁵	Ostro 1994 as referenced in World Bank 1998
Respiratory hospital admissions - daily exposures	SO_2	All persons	2.01 x 10 ⁻⁶	Maddison 1997 as referenced in WB 1998
Respiratory hospital admissions - daily exposures	NO_2	All persons	1.65 x 10 ⁻⁶	Maddison 1997 as referenced in WB 1998
Cardiovascular hospital admission - daily exposures	PM10	All persons	1.01 x 10 ⁻⁷	Dockery et al 1989
Daily Mortality - daily exposures	PM10	>=65 years	4.42 x 10 ⁻⁷	EXMOD - as referenced by Nelson 2000
Daily Mortality - daily exposures	PM10	<65 years	2.35 x 10 ⁻⁸	EXMOD - as referenced by Nelson 2000
Daily Mortality - daily exposures	SO_2	>=65 years	1.01 x 10 ⁻⁸	Watkiss and Holland - functions collated for application by the European Commission DG Environment
Daily Mortality - daily exposures	SO_2	<65 years	1.38 x 10 ⁻⁹	Watkiss and Holland - functions collated for application by the European Commission DG Environment
Chronic Bronchitis - annual exposures	PM10	children (<5 years)	1.61 x 10 ⁻³	Dockery et al 1989
Chronic Bronchitis - annual exposures	PM10	adults (20 years+)	4.90 x 10 ⁻⁵	Abbey et al 1995
Restricted activity days (RAD) -daily exposures	РМ	20-65 years	1.60 x 10 ⁻⁴	Rowe et al 1994 as referenced in van Horen 1996
Minor restricted activity days (MRAD) - daily exposures	SO_2	20-65 years	9.76 x 10 ⁻³	Watkiss and Holland - functions collated for application by the European Commission DG Environment

Example of dose-response functions (review from Scorgie et al., 2004)

What about dose-response functions in Latin American cities?

2 big reviews :

- Fajersztajn et al., 2017 (https://doi.org/10.1007)
- ✓ 1628 studies reviewed. Nine elected for the qualitative analysis and seven for the quantitative analyses.
- Each 10 μg/m³ increments in daily PM_{2.5} concentrations are significantly associated with increased risk for respiratory and cardiovascular mortality in all-ages (polled RR = 1.02, 95% CI and RR = 1.01, 95% CI).
- Cifuentes et al., 2005 (http://www.iadb.org/sds/env)

Exposure	Type of Endpoint		Endpoint (specific cause)	City/Country providing C-R functions
			All cause	USA
Long.town	Premature Mortality Illness or Disease		Cardiopulmonary	USA
Long-term			Lung cancer	USA
			Chronic Bronchitis	USA
			All cause mortality	Several LA cities / USA
	Premature Mortality		Respiratory causes	USA
			CVD causes	USA
			Cardiovascular disease (ICD9 390-429)	USA
		Hospital Admissions	Asthma	USA
			Dysrhytmias (ICD9 427)	USA
			Respiratory Causes (ICD9 460-519)	Sao Paulo/USA
			Pneumonia (ICD9 480-487)	Sao Paulo/USA
			Asthma (ICD9 493)	Sao Paulo
	Medical		Cardiovascular disease	Sao Paulo
	Actions		Ischemic Heart Disease	USA
Short-term		Emergency	Respiratory Causes	Santiago
		Room Visits	Pneumonia and Influenza	USA
			Pneumonia (ICD9 480-486)	Santiago
			Lower-RSP	Santiago
			Upper RSP symptoms (ICD9 460, 465, 487)	Santiago
		Medical Visits	Asthma (ICD9 493)	Juarez
	Illness	or Disease	Asthma Attacks	USA
	Inness	of Distast	Acute Bronchitis	USA
			Work Loss Days (WLD)	USA
	Days with	Restriction in	Restricted Activity Days (RAD)	USA
	A	ctivity	Minor Restricted Activity Days (MRAD)	USA
			Shortness of Breath Days	USA

Mortality impacts in LAC : Time series studies

Higher RR(PM10) values for Infants and Elder Co-pollutant : few studies for Elder only Risk increased when co-pollutant

Heterogeneous results between the cities due to different factors (demography, culture, Socio-economy, mobility, sampling)

group C	ity Co-Pollutant	studies	Metric	90 Illereas	(95% CI)	References
All Ages						
	All none	6	FE	0.41%	(0.32% - 0.51%)	Borja-Aburto 1997, O'Neill 2004, Castilloios 2000, Cifuentes 2000, Ostro
			RE	0.61%	(0.26% - 0.97%)	1996, Gouveia 2000b
	none	4	FE	0.70%	(0.57% - 0.82%)	Borja-Aburto 1997, Castillejos 2000,
			RE	0.87%	(0.55% - 1.19%)	Cituentes 2000, Osito 1990
	O3, SO2; O3	4	FE	0.43%	(0.33% - 0.54%)	Borja-Aburto 1997, Castillejos 2000,
			RE	0.91%	(0.38% - 1.44%)	Cituentes 2000, Ostro 1996
Mexico C	ity none	3	FE	0.24%	(0.09% - 0.38%)	Borja-Aburto 1997, O'Neill 2004-b,
			RE	0.89%	-(0.05% - 1.85%)	Castillejos 2000
	O3, SO2; O3	2	FE	1.35%	(0.89% - 1.82%)	Borja-Aburto 1997, Castillejos 2000
			RE	1.37%	(0.85% - 1.89%)	
Santia	igo none	2	FE	0.63%	(0.49% - 0.76%)	Cifuentes 2000, Ostro 1996
			RE	0.64%	(0.47% - 0.80%)	
	03	2	FE	0.38%	(0.27% - 0.49%)	Cifuentes 2000, Ostro 1996
			RE	0.55%	(0.03% - 1.07%)	
Elder 65+ y	т					
	All None	5	FE	0.66%	(0.51% - 0.81%)	Castillejos 2000, Ostro 1996, Sanhueza
			RE	0.83%	(0.48% - 1.17%)	1998, Gouveia 2000b, Saldiva 1995
	O3, SO2; O3	3	FE	0.56%	(0.37% - 0.75%)	Borja-Aburto 1997, Castillejos 2000,
			RE	1.00%	(0.24% - 1.77%)	Sanhueza 1998
México C	ity O3, SO2; O3	2	FE	1.35%	(0.76% - 1.95%)	Borja-Aburto 1997, Castillejos 2000
			RE	1.35%	(0.76% - 1.95%)	
Santia	igo None	2	FE	0.61%	(0.44% - 0.78%)	Ostro 1996, Sanhueza 1998
			RE	0.69%	(0.30% - 1.08%)	
Sao Pa	ulo none	2	FE	0.71%	(0.37% - 1.06%)	Gouveia 2000b, Saldiva 1995
			RE	0.81%	(0.13% - 1.51%)	
Infant < 18	vr					
	All None	3	FE	2.73%	(1.55% - 3.92%)	Loomis 1999, Linn 2000, Nishioka 2004
			RE	2.94%	(1.35% - 4.56%)	
Sao Pa	ulo None	2	FE	2.37%	(1.05% - 3.72%)	Linn 2000, Nishioka 2004
			RE	2.59%	(0.54% - 4.69%)	
	O3, SO2; O3	2	FE	3.20%	(1.29% - 5.16%)	Loomis 1999, Nishioka 2004
			RE	3.20%	(1.29% - 5.16%)	

Table IV-3 Summary estimates from the Meta-analysis of Latin-American studies of the effects of PM10 on

All Cause Mortality

Note: FE = Fixed effects estimate, RE= Random Effects Estimate. Mid estimates shown in bold, 95% Confidence interval (percentile 2.5 to percentile 97.5 is shown in parenthesis)

Cifuentes et al., 2005

Mortality impacts in LAC : Time series studies



PAPA : Asian cities (HEI 2004) APHEA2 : 29 european cities (Atkinson et al., 2001) NMAPS : 90 US cities (Samet et la., 2000) Stieb study : 109 cities worldwide (Stieb et al., 2002)

All ages : results from LAC comparable to European and Stieb studies Elderly : Stieb slightly bigger than LAC

Cifuentes et al., 2005

Mortality impact in LAC : Time series

ESCALA project (supported by HEI) : 1997-2005 Romieu et al., 2012 Cifuentes et al. : Chili Gouveia et al. : Brazil Romieu et al. : Mexico 9 cities/ PM10 and 03



Figure 5. All-natural-cause, all-age mortality. Risk percent changes (95% CI) per 10-µg/m³ increase of PM₁₀ and O₃, using DLM 0-3 for low, medium, and high SES levels, in São Paulo and Rio de Janeiro, Brazil; Santiago, Chile; and Mexico City, México (1997-2005). Note: the y-axes of Figures 5-10 are scaled differently. Sources: Appendix Tables I.9–I.12, available on the HEI Web site.

Small but significant effect of daily exposure to PM10 and ozone on daily mortality Link with socioeconomical data

Mortality impact in LAC : no cohort studies in 2005

Table IV-5 Risk estimates reported in the studies of the long-term exposure to particulate matter explain entries – central & confidence interval

Cause Location	Age Group	o Cohort	C-R Specif.	Expos met	sure ric	% change in deaths for given change in PM2.5 (*)		or given (*)	Source
All Cause mortality									
6 US cities	>24	8,192	Linear	Avg PM ₂	2.5	13.2%	(4.1% -	23.1%)	Dockery, Pope III et al. 1993
151 US cities (ACS cohort)	>30	552,138 adults	Linear	Median PM _{2.5}		5.7%	(1.5% -	10.0%)	Pope III, Thun et al. 1995
California, USA	>27	6,338 non smoking	Linear	Avg PM ₁₀		0.1% (0.1% - 0.1%) Abbey, Nishino et		Abbey, Nishino et al. 1999	
Cardio-pulmonary n	nortality								
151 US cities	> 30	552,138	Linear	Avg.	79-83	5.9%	(1.5% -	10.5%)	Pone III Burnett et al 2002
(ACS cohort)		adults		PM _{2.5}					Pope III, Bullett et al. 2002
				Avg PM ₂	2.5	9.3%	(3.3% -	15.8%)	
			Log- Linear	Avg. PM _{2.5}	79-83	1.2%	(0.3% -	2.0%)	Cohen, Anderson et al.
				Avg PM ₂	2.5	1.6%	(0.6% -	2.5%)	
Lung cancer mor	tality								
151 US cities	> 30	552,138	Linear	Avg.	79-83	8.2%	(1.1% -	15.8%)	D
(ACS cohort)		adults		PM _{2.5}					Pope III, Burnett et al. 2002
				Avg PM ₂	2.5	13.5%	(4.4% -	23.4%)	
			Log-	Avg.	79-83	1.7%	(0.3% -	3.1%)	Cohen Anderson et al. 2004
			Linear	PM _{2.5}					Colleit, Allueisoli et al. 2004
				Avg PM ₂	2.5	2.3%	(0.9% -	3.8%)	

(*) For linear CR the PM2,5 change considered is 10 ug/m3. For the log-linear CR, the change considered is 10% For example, if PM2.5 decreases 20%, the % change in lung cancer mortality, based on average PM2.5 (last line), equals -4.6% (2.3 *(-20%/10%))

Cifuentes et al., 2005

Morbidity impacts in LAC : time series study



Figure IV-2. Percentage increase in baseline effects per 10µg/m³ of PM₁₀ for morbidity endpoints in Latin-American studies

Cifuentes et al., 2005

Morbidity impacts in LAC : time series study

Short term effects of AP on respiratory morbidity in Colombia / 2011-2014 NO_2 , O_3 , SO_2 , CO, PM2.5 and PM10 Greater % increase for PM2.5, PM10 and NO_2 Maximum for 5-9 yrs old and >60 yrs old



Figure 1. Percentage increases in emergency department visits by children for respiratory diseases associated with PM_{2.5} concentrations, per age group, in the four cities in Colombia, 2011–2014. Point estimates and 95% confidence intervals for age groups of children in years.

Other morbidity effects (not exhaustive):

Air pollution effect on Asthma morbidity in LAC cities Forno et al. 2015, Wong et al. 2013 ISAAC study : 46500 children Impact on lung function Link with open fire cooking systems and Asthma Importance of vitamin C and D levels on oxidative stress

Air pollution effect on Neural system during neonatal and post-natal period Gouveia et al., 2018 Medeiros et al., 2009 Calederon-Garciduenas et al. 2015 (Mexico city) MacIntyre et al. 2014 : 10 european birth cohorts

Air pollution on **Diabetes** : Bowe et al., 2018 (<u>https://doi.org/10.1016/S2542-5196(18)30140-2</u>), The 2016 global and national burden of diabetes mellitus attributable to $PM_{2.5}$ air pollution (from a cohort study with US veterans).

Mechanisms of damage? : Focus on aerosols and biological studies

Respiratory system penetration linked to particle size: inhalable, thoracic, respirable, ultra-fine



Adapted from H. Cachier, 2009



Mechanisms of defense and biological damage



Oxidative stress and inflammation : primary mechanisms to explain the impact of air pollution on health (Korten et al. 2017)

Biological impact of particles



Adapted from A. Baeza,

Main important characteristics of particules responsible for biological effects

Size and properties (solubility, hygroscopicity)

- Penetration
- Deposition
- Clearance

Surface reactivity



Rétention (Churg, 2000)



Chemical composition

•Metals •organics •inorganics

Adapted from A. Baeza,

How to measure : biological measurements



Adapted from A. Baeza,

Other inflammatory tracers

- Out of cells : particle pro-oxydant capacity (DTT chemical test for PAHs) or Reactive oxygen species measurements (H2O2, fluorometry).
- Urines : see table
- Blood :glutathion tests

Pollutants	PAH Gazolin		Diesel	NO2	Metals	Oxidative stress
Biological tracer	Urinary 1- hydroxypyre ne	urinary trans, trans-muconic acid	Urinary 8-OHdG	urinary hydroxyproline ratio	Blood test: Ag⁺, Pb²+, Cd²+	Ratio GSH/GSSH (blood)
Methodology	HPLC ou kit ELISA	HPLC	ELISA Kit	hypronosticon test	ICP-MS	ELISA Kit
Reference	(Llop et al., 2008)	(Ong et al., 1996) (Boogaard et van Sittert, 1995)	(Lee et al., 2010)	(Perdelli et al., 2002)	(Shirali et al., 2004)	(Lee et al., 2010)

Adapted from G. Uzu, 2010

Characteristics of i	identified studies.			
Study	Study location	Characteristics of subjects	Sampling technique	Exposure assessment
Bunn et al. (2001)	Leicester, UK	Healthy children (mean age 3 months to 16 years, $n=22)$	BAL	Traffic. Distance from home to major road.
Kulkarni et al. (2005)	Gondar, Ethiopia and Leicester, UK	Women $(n = 10)$ and children $(n = 10)$ using biomass fuels for cooking from Ethiopia. Adults $(n = 10)$ and children $(n = 10)$ without exposure to biomass smoke from UK	IS	Biomass smoke. Kitchen and biomass fuels classified into different categories.
Kulkarni et al. (2006)	Leicester, UK	Healthy children (mean age 11.5 years, $n=64)$ and asthmatic children (mean age 13.6 years, $n=9)$	IS	Traffic. Modeled annual mean level of primary PM ₁₀ at children's residence.
Fullerton et al. (2009)	Blantyre, Malawi	Healthy adults ($n = 57$)	BAL	Biomass smoke. Fuels for heating and cooking were categorized.
Jacobs et al. (2010)	Leuven, Belgium	Adults with diabetes $(n = 137)$	IS	Traffic. Recent (2 h) outdoor and indoor PM. Modeled PM ₁₀ level (1 d, 1 wk, 1 m, 6 m, 1 yr) at subjects' residence.
Jacobs et al. (2011)	Leuven, Belgium	Adults with diabetes $(n = 137)$	IS	Traffic. Recent (2 h) outdoor and indoor PM. Distance from subjects' residence to major road was calculated.
Kalappanavar et al. (2012)	Davangere, India	Children from industrial area (mean age 13.5 years, $n=300)$ and children from a "green" zone $^a\!(mean$ age 13.6 years, $n=300)$	IS	"Ambient air quality monitoring instrument" one week before study: PM_{10} 1403 µg/m ³ (industrial area) vs 315 µg/m ³ ("green" area).
Nwokoro et al. (2012)	London, UK	Adult cyclists ($n = 14$) and non-cyclists ($n = 14$)	IS	Traffic. Personal exposure to black carbon monitored by aethalometer on a working day. Background PM ₁₀ measured by monitoring station.
Brugha et al. (2014)	London, UK	Healthy children (mean age 9.3 years, $n = 47$), children with mild asthma (mean age 9.4 years, $n = 13$), children with moderate to severe asthma (mean age 11.6 years, $n = 36$)	IS	Traffic. Modeled residential PM _{2.5} level and distance between home and main road.

Table 1

A new biomarker ...



Bai et al., 2015 : Carbon loading in airway macrophages as a biomarker for an individual exposure

to particulate matter => Novel approach to assess personal exposure to combustion-derived particles (9 studies in Europ, adults and children)

Measure the Inhaled particles phagocytosed by airway macrophages of the epithelial surfaces in the alveoli. Macrophages are collected from BAL : bronchoalveolar lavage (invasive) or IS : induced sputum (non invasive)

In vivo studies : respiratory parameters with spirometry



In vitro biological measurements : focus on POM/GM-CSF citokyne dose-response



Better response for BK2 than for DK => due to POM solubility at Bamako?

	BK2	DK
WSOC/OC	0.8	0.25

Incomplete sources (two stroke emissions and domestic fires) at Bamako (BK2) with relatively more OC and WSOC => bigger inflammatory responses than diesel sources at Dakar (DK).

Biological measurements in LAC

	Mean (% COHb) ^a	S.D. (% COHb) ^a	%<2.5 ^b	%>2.5°	Range (% COHb)
Before	4.9*	4.3	45	55	1.05–13.88
After	1.0	0.14	100	0.0	0.65–1.30

*p<0.05 when compared after intervention.</p>

Torres-Dosal, Perez-Maldonado et al., et al., 2008 : Mexico => Wood as energy used by 27M of people Health risk reductions linked to 3 measures :

soot removal on roofs and walls, floor paving and new wood-stove with a chimney

Biological measurements in LAC

N. Brucker et al. / Science of the Total Environment 463-464 (2013) 884-893





Brucker et al., 2013 : Porto Aligre (South of Brazil) Biomarker of exposure to PAH from traffic on 60 subjects (with urinary and blood tests)

« The » relationship to calculate Excess mortality numbers At all scales

Un example: Excess mortalities due to 2005 to 2030 emission change (Δ PM2.5) => Use the following relationships (Anenberg et al., 2010, Lelieveld et al., 2013) $\Delta death=y0^*(1-exp-\beta\Delta PM2.5)^*POP$

where

y0 is the baseline mortality rate (WH0) for different mortalities

POP is the exposed population (>30yrs)

l is the concentration response factor

 $RR=(exp \ \beta^* \Delta PM2.5)$

Given by the mode

RR is the relative risk for a 10µm/m3 PM2.5 increase

Here : RR (all causes) : 1.06 ; RR (lung cancer) : 1.06; RR (cardiopulmonary : 1.11); RR (respiratory diseases : 1.06) (=> miminim of litterature range)

An example of Aerosol/Health Regional modeling



Liousse et al., 2020

Table 2. Estimated annual mortalities ± 1 SD due to anthropogenic O₃ and PM_{2.5}, assuming natural background only or LCTs (33.3 ppb for O₃ and 5.8 µg/m³ for PM_{2.5}) (× 1,000).

	O ₃ resp	iratory	PM _{2.5} cardi	opulmonary	PM _{2.5} lung cancer		
	Background	Threshold	Background	Threshold	Background	Threshold	
Africa	63 ± 34	45 ± 30	154 ± 44	52 ± 33	3±1	1±1	
North America	35 ± 17	25 ± 15	124 ± 37	65 ± 30	17 ± 7	10 ± 5	
Europe	41 ± 21	23 ± 17	586 ± 149	383 ± 143	47 ± 17	31 ± 14	
Asia	543 ± 253	370 ± 220	2,584 ± 618	1,991 ± 603	152 ± 53	122 ± 47	
South America	18 ± 9	8±6	48 ± 15	16 ± 9	2 ± 1	1 ± 1	
Oceania	1±1	0 ± 0	2 ± 1	0 ± 0	0 ± 0	0 ± 0	
World	700 ± 335	470 ± 288	3,499 ± 864	2,506 ± 816	222 ± 80	164 ± 68	

SDs reflect uncertainty in the CRF and simulated present-day concentrations (SD = 25% of simulated concentration).

Anenberg et al., 2010

Table I Excess mortality attributable to ambient air pollution^a

	Mortality	Deaths per	YLL	LLE	LLE Avoidable A (years) LLE (years) m (>	Avoidable mortality (×10 ³ /year)	Mortal	Mortality for disease categories (×10 ³ /year)					
	(×10 [°] /year)	100 000 (year ⁻¹)	(×10°/year)	(years)			LRI	COPD	LC	CEV	IHD	Other NCD	
Africa	957	81	40.0	3.1	0.7	230	378	36	7	113	224	199	
East Asia	3112	196	67.4	3.9	3.0	2403	204	511	300	738	779	580	
South Asia	2809	119	83.6	3.3	1.9	1660	478	509	61	383	981	397	
West Asia	544	94	14.6	2.3	1.0	241	50	27	19	76	292	80	
Europe	790	133	14.3	2.2	1.7	608	54	49	54	64	313	256	
Australia	14	47	0.3	0.8	0.2	3	0.6	0.8	0.9	0.6	4	7	
North America	360	74	7.5	1.4	1.1	294	24	40	24	14	112	146	
South America	207	42	5.3	1.0	0.5	115	30	14	6	14	63	80	
World	8793	120	233	2.9	1.7	5554	1218	1187	472	1403	2768	1745	

Avoidable LLE and mortality were calculated by removing anthropogenic emissions in the model. Australia also includes other islands of Oceania. Data for all countries, including 95% uncertainty intervals, are given in the Supplementary material online, *Tables* (overall uncertainty about ±50%).

CEV, cerebrovascular disease; COPD, chronic obstructive pulmonary disease; IHD, ischaemic heart disease; LC, lung cancer; LLE, loss of life expectancy; LRI, lower respiratory infections; NCD, non-communicable diseases; YLL, years of life lost.

^aExcess mortality expresses the number of deaths over a given period that would not occur in the absence of exposure.

Lelieveld et al., 2013, 2020

26 LAC cities (86 M people) If PM10 = norms : 13500 avoided deaths per year (2-2.6% of death avoided/yr) If PM10 decrease of 10μg/m³ : 12-25% of deaths avoided

Cifuentes et al., 2005

Mexico City; Santiago; and São Paulo. 2000-2020 : 2 scenarios Modest changes in fossil fuel use Avoided in the three cities : 156,000 deaths, 4 million asthma attacks, 300,000 children's medical visits, and almost 48,000 cases of chronic bronchitis

Bell et al., 2005

Take-home messages

- New proxys for health such as aerosol oxydant capacity are really important for long-term crossed air pollution and health studies
- Need to integrate sociological factors within the calculation of dose response functions (people vulnerability to air pollution)
- Do not separe urban studies on health and climate (we need winwin results).
- Reconciling epidemiological studies with process (biological) studies : need more cohort studies with personal exposure measurements (exposology) : a way to act on source mitigation.

The inhaled aerosol is not the same than in alveoli



Thanks for your attention

