Journée PAPPEI 2015

Dr Isabella ANNESI-MAESANO

Directeur de recherche INSERM

Directeur EPAR i-PLESP INSERM and UPMC S

orbonne Universités

isabella.annesi-maesano@inserm.fr

Ecce Terra
Observatoire des Sciences de l'Univers
UPMC - INSU

UPMC
SORBONNE UNIVERSITÉS

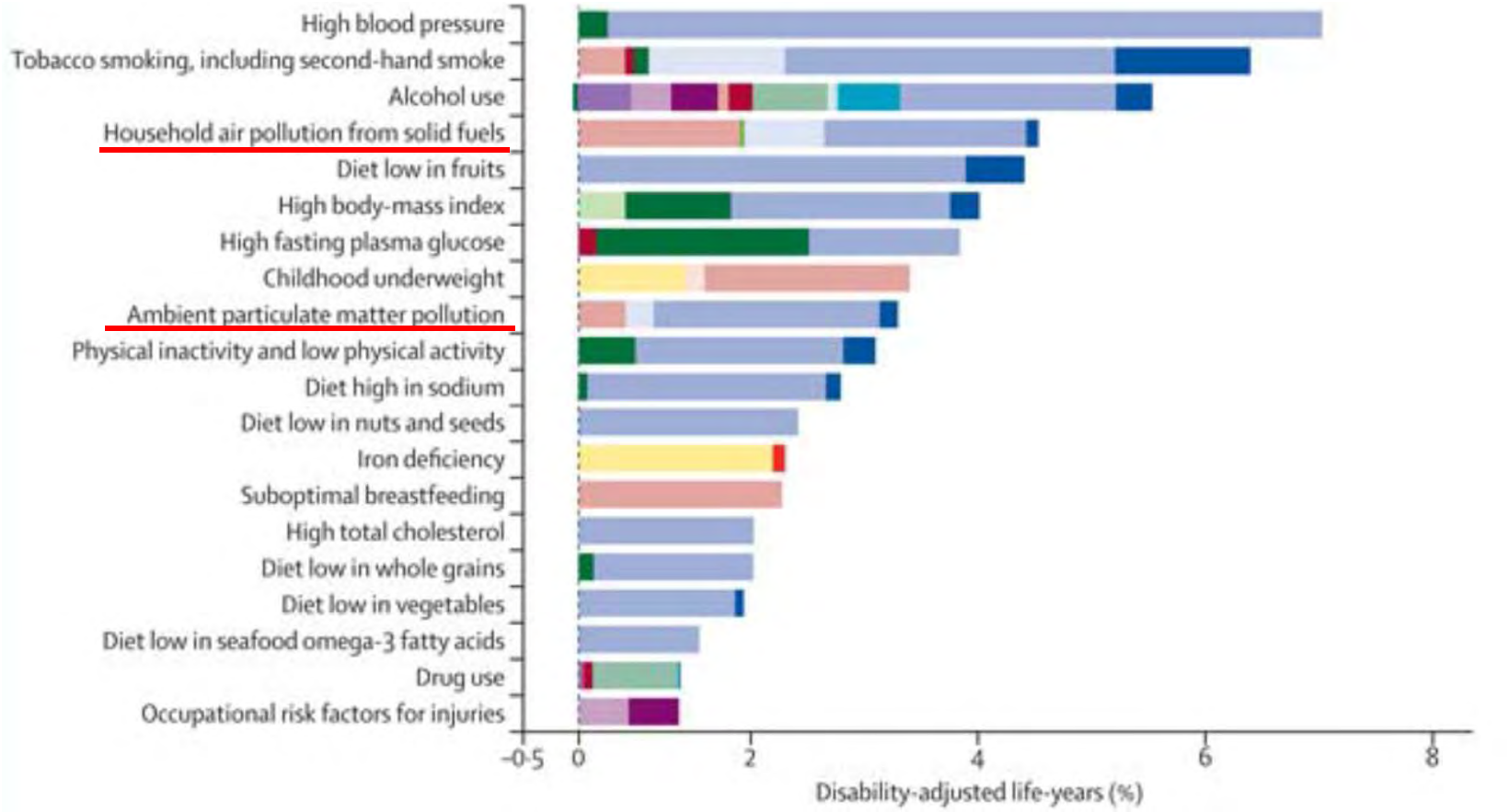
Instituts thématiques
Inserm
Institut national
de la santé et de la recherche médicale

EPARI
EPidemiology
Allergic and
Respiratory
Diseases

AUTRES EFFETS DE LA POLLUTION ATMOSPHÉRIQUE.

Avant de commencer

Global Burden of Disease 2010



DALYs= sum of years lived with disability [YLD] and years of life lost [YLL]

http://www.healthmetricsandevaluation.org/

Lancet 2012

LETTER

doi:10.1038/nature15371

The contribution of outdoor air pollution sources to premature mortality on a global scale

J. Lelieveld[1,2], J. S. Evans[3,4], M. Fnais[5], D. Giannadaki[2] & A. Pozzer[1]

Assessment of the global burden of disease is based on epidemiological cohort studies that connect premature mortality to a wide range of causes[1–5], including the long-term health impacts of ozone and fine particulate matter with a diameter smaller than 2.5 micrometres ($PM_{2.5}$)[3–9]. It has proved difficult to quantify premature mortality related to air pollution, notably in regions where air quality is not monitored, and also because the toxicity of particles from various sources may vary[10]. Here we use a global atmospheric chemistry model to investigate the link between premature mortality and seven emission source categories in urban and rural environments. In accord with the global burden of disease for 2010 (ref. 5), we calculate that outdoor air pollution, mostly by $PM_{2.5}$, leads to 3.3 (95 per cent confidence interval 1.61–4.81) million premature deaths per year worldwide, predominantly in Asia. We primarily assume that all particles are equally toxic[5], but also include a sensitivity study that accounts for differential toxicity. We find that emissions from residential energy use such as heating and cooking, prevalent in India and China, have the largest impact on premature mortality globally, being even more dominant if carbonaceous particles are assumed to be most toxic. Whereas in much of the USA and in a few other countries emissions from traffic and power generation are important, in eastern USA, Europe, Russia and East Asia agricultural emissions make the largest relative contribution to $PM_{2.5}$, with the estimate of overall health impact depending on assumptions regarding particle toxicity. Model projections based on a business-as-usual emission scenario indicate that the contribution of outdoor air pollution to premature mortality could double by 2050.

Table 1 | Premature mortality related to $PM_{2.5}$ and O_3 for the population <5 and ≥30 years old

WHO region	Year	Population ($\times 10^6$)	Mortality attributable to air pollution (deaths $\times 10^3$)						
			$PM_{2.5}$					O_3	Total
			ALRI < 5 yr	IHD ≥ 30 yr	CEV ≥ 30 yr	COPD ≥ 30 yr	LC ≥ 30 yr	COPD ≥ 30 yr	
Africa	2010	809	90	55	77	11	2	2	237
	2050	1,807	158	185	262	38	5	12	660
Americas	2010	930	0	44	8	4	7	5	68
	2050	1,191	0	75	15	7	11	11	119
Eastern Mediterranean	2010	602	56	115	86	12	5	12	286
	2050	1,021	66	321	246	37	13	40	723
Europe	2010	867	1	239	95	13	27	6	381
	2050	886	1	307	156	18	37	11	530
Southeast Asia	2010	1,762	64	327	250	124	15	82	862
	2050	2,332	104	865	807	419	48	227	2,470
Western Pacific	2010	1,812	19	299	794	209	107	35	1,463
	2050	1,861	16	413	1,120	309	155	57	2,070
World	2010	6,783	230	1,079	1,311	374	161	142	3,297
	2050	9,098	346	2,166	2,604	828	270	358	6,572

Regions are defined by the World Health Organization, see Extended Data Table 1. Results for 2050 are based on a business-as-usual scenario.

Lilieveld, Nature 2015

For a universal climate agreement

Climate change?

50 years ago

Climate change? YES!

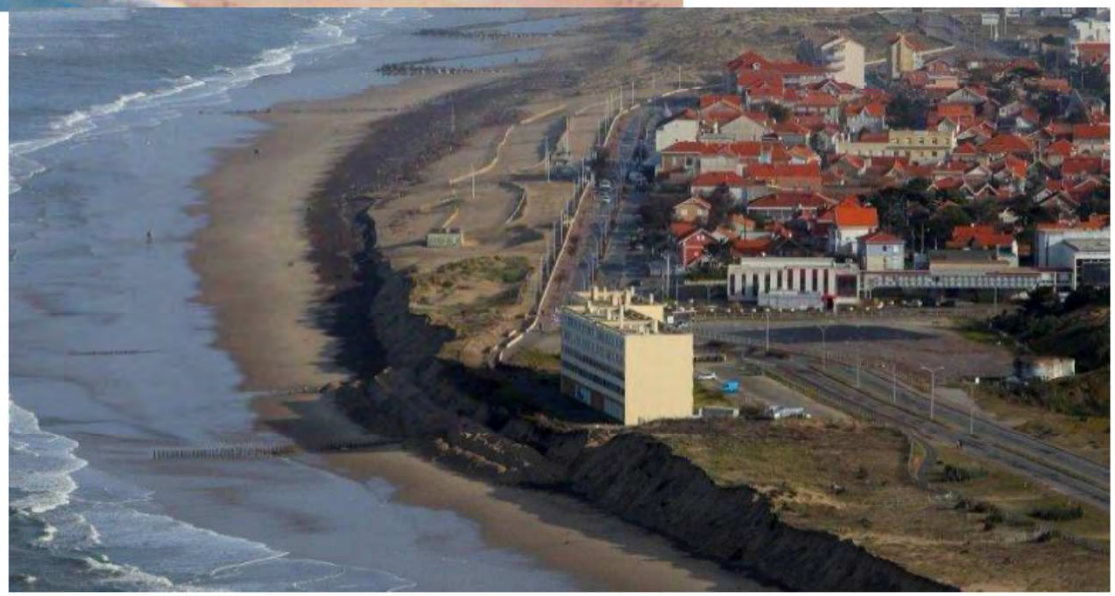
50 years ago

Now

élévation du niveau des mers

Le Signal - Soulac sur Mer

1975



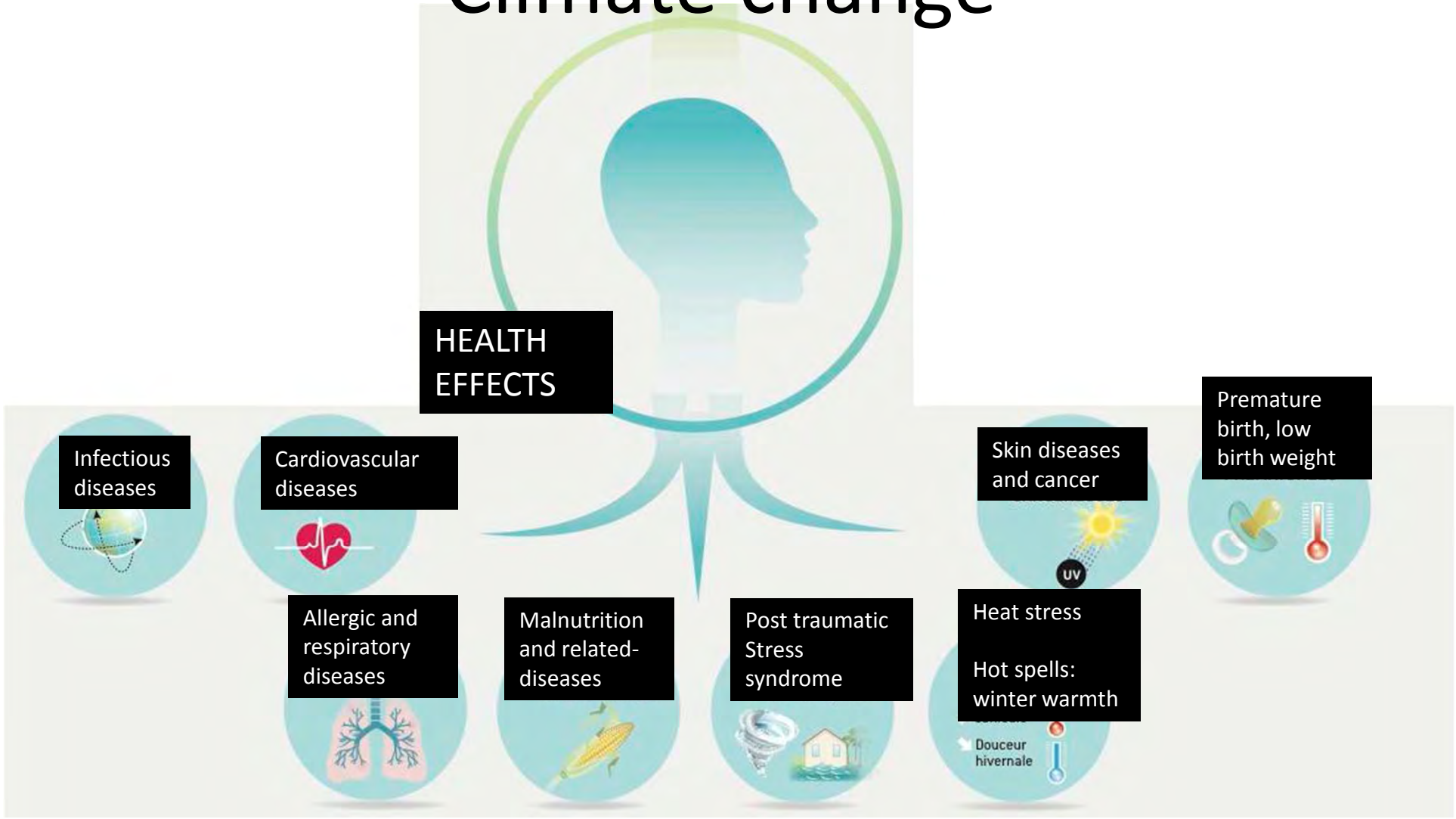
2015

50m par décennie

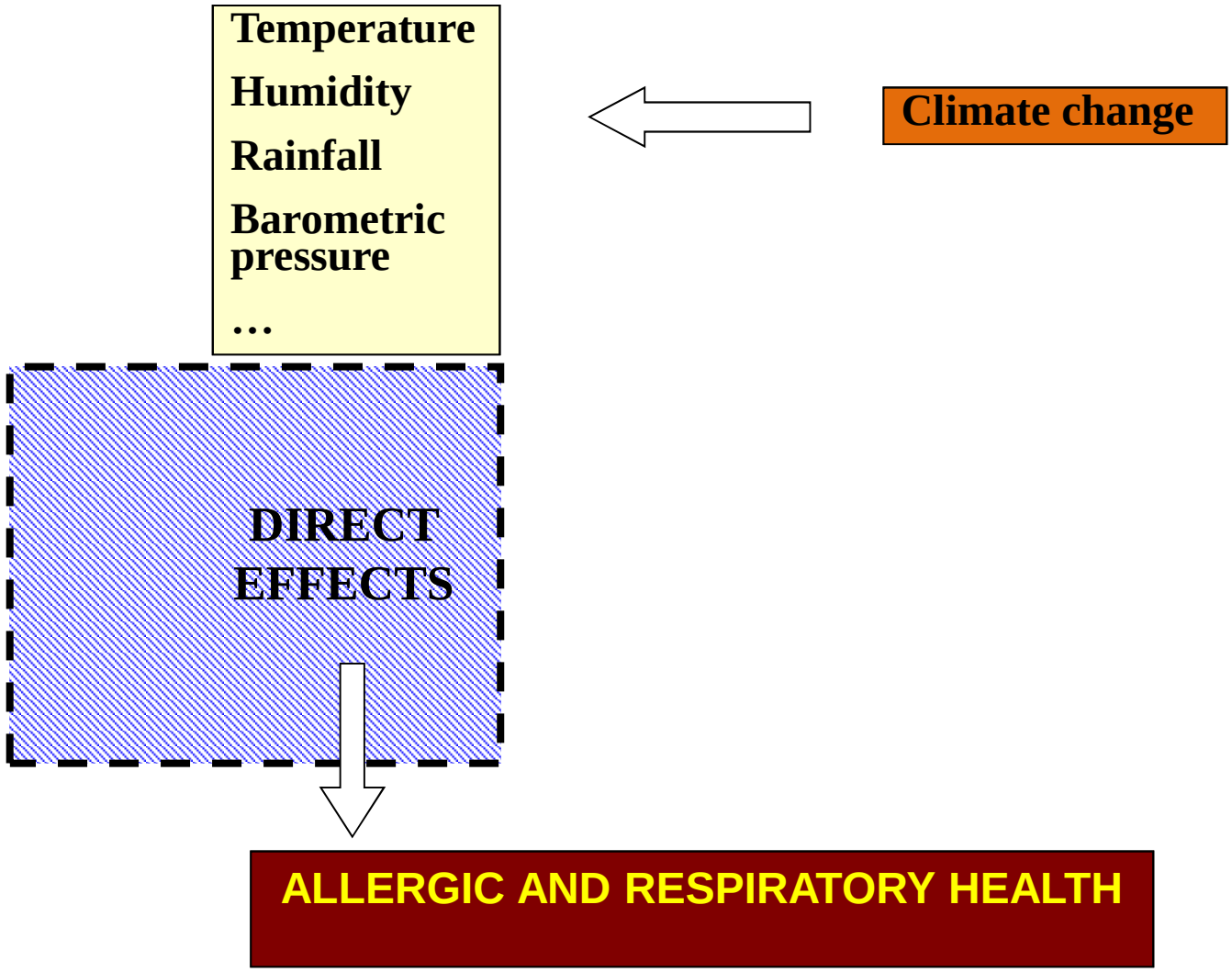
Vision of UN Secretary-General on Climate Change

- "Climate change is a serious threat to development everywhere"
- "Today, the time for doubt has passed. The IPCC has unequivocally affirmed the warming of our climate system, and linked it directly to human activity"
- "Slowing or even reversing the existing trends of global warming is the defining challenge of our ages"
- "Galvanising international action on global warming as one of main priorities as Secretary General"

Climate change



Comment les changements climatiques agissent sur la pollution atmosphérique et la santé respiratoire?



Chronic Diseases

- **Direct:** increased morbidity and/or mortality from weather evolution and extreme weather events (evidence for hot spells, extreme cold, temperature, humidity…) for chronic diseases

CLIMATE CHANGE AND ALLERGIC AND RESPIRATORY HEALTH . ACTUAL CHANGES

- **Virtually certain**
  - **Increased cardio-respiratory morbidity**
  - **Increased cardio-respiratory mortality**
  - **Increased infections (but probably in the case of cold-related infections)**
- **Likely**
  - **Increased AVC morbidity and related respiratory co-morbidity**
- **To be further confirmed**
  - **Diminished respiratory infections due to warmer and fewer colder days**

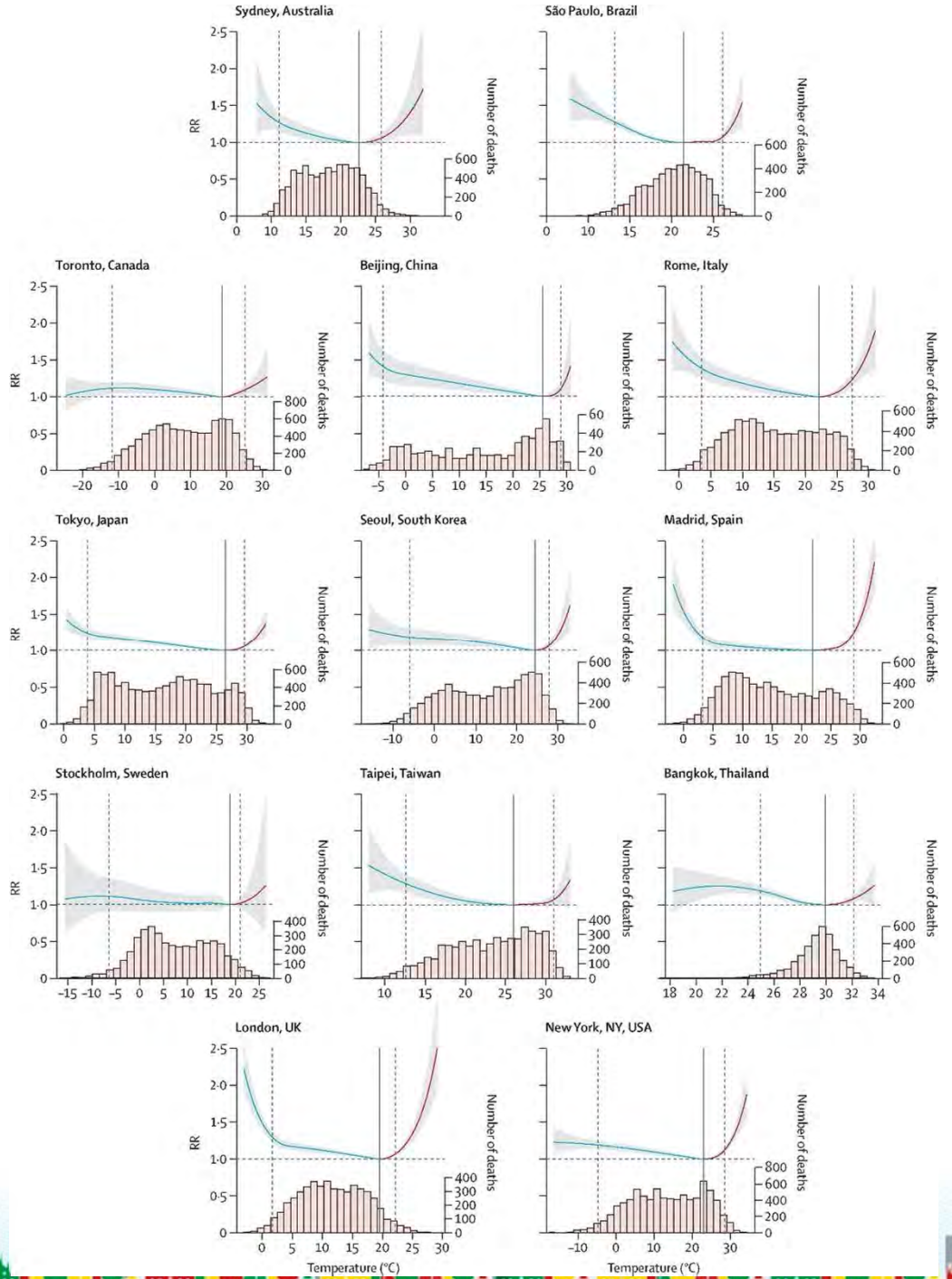
Mortality risk attributable to high and low ambient temperature

Exposure–response associations in representative cities of 13 countries, with related temperature distributions

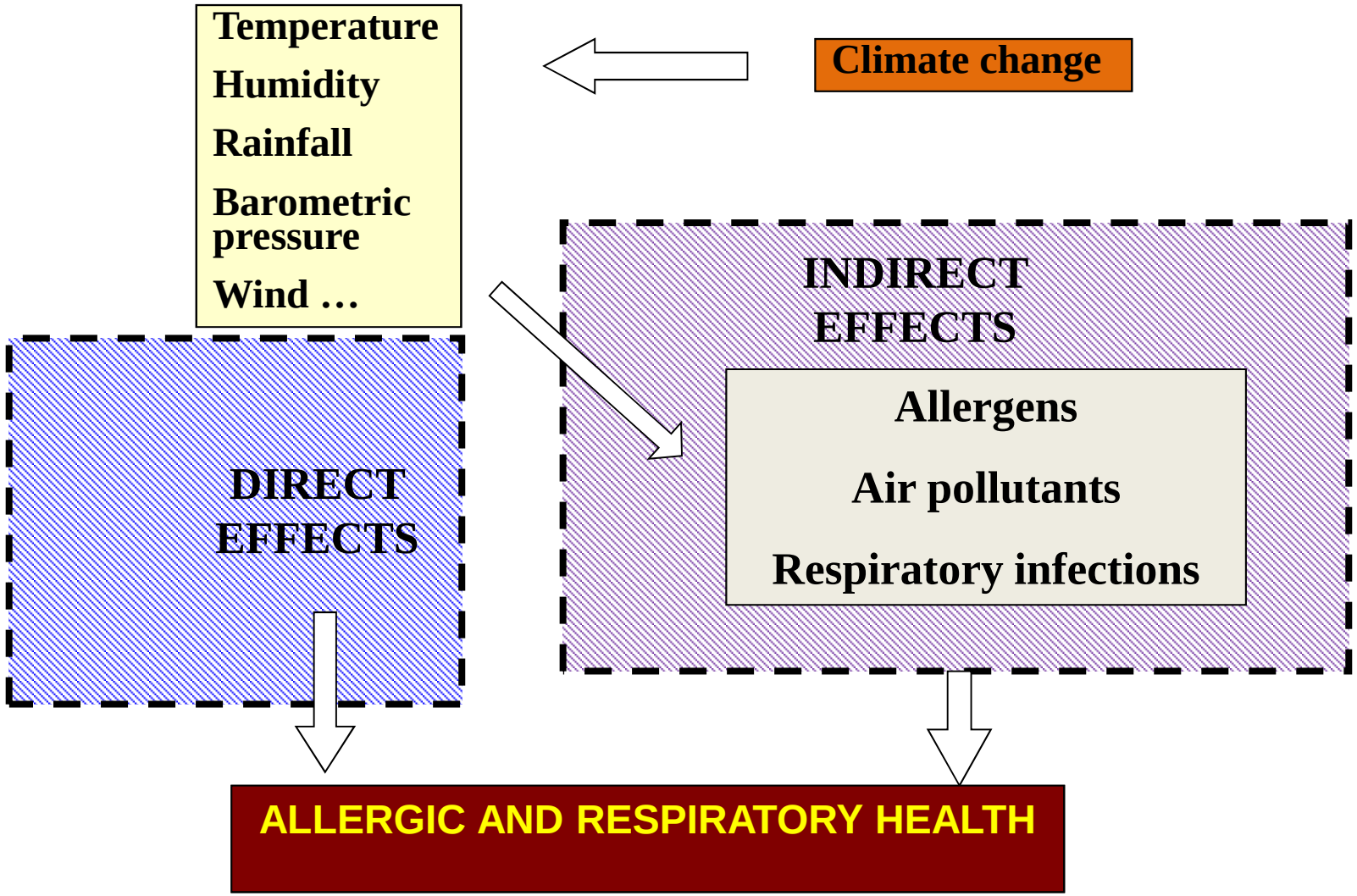
OF NOTE: Africa is missing

Extreme cold and hot temperatures were responsible for 0·86% (0·84–0·87) of total mortality.

7·71% (95% empirical CI 7·43–7·91) of mortality was attributable to non-optimum temperature



The Lancet DOI: (10.1016/S0140-6736(15)62114-0)



Chronic Diseases

- **Direct:** increased morbidity and/or mortality from weather evolution and extreme weather events (evidence for hot spells, extreme cold, temperature, humidity…) for chronic diseases
- **Indirect:** increased morbidity and/or mortality

CLIMATE CHANGE-RELATED RISK FACTORS FOR ALLERGIC AND RESPIRATORY HEALTH: ACTUAL CHANGES

AIR POLLUTION

- **Virtually certain**
  - **Increased outdoor ozone level**
  - **Increased outdoor PM level**
  - **Increased natural aerosol sources**
- **Likely**
  - **Removal by precipitation of PM level at local level**
- **To be further confirmed**
  - **Increase of indoor air pollutants**

→ INCREASED IMPACT ON HEALTH EFFECTS

Climate change: impact on air pollution and related health effects

- Outdoor ozone: Overall increase in concentrations in high-income countries, but with wide regional differences

  → 1,500 more ozone-associated deaths annually by the year 2020 in UK

  UK Department of Health, 2008

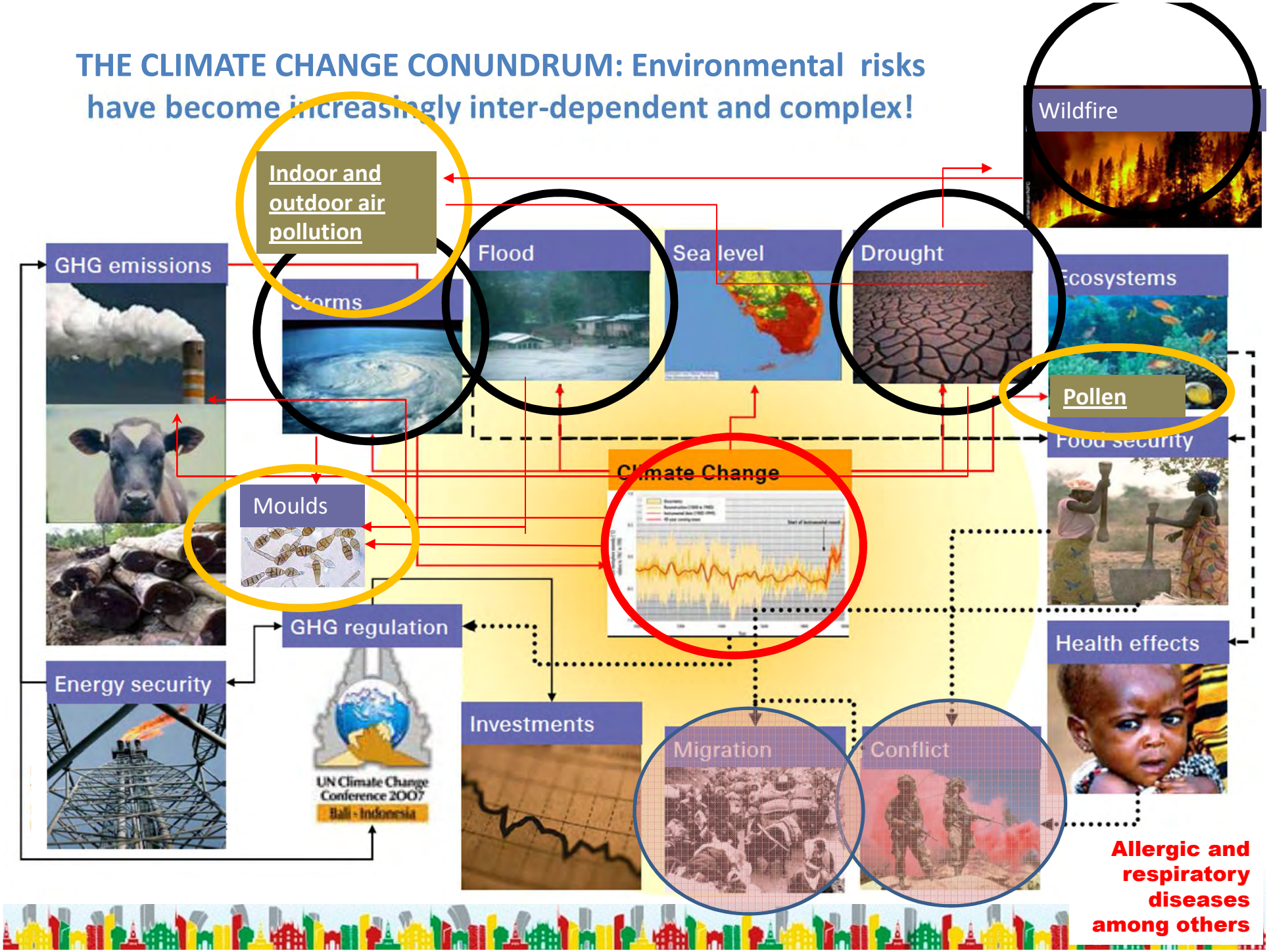
- Outdoor PM: Local and transboundary increase in concentrations
  - Transport , urbanisation, heating
  - Desertification and a higher frequency of forest fires may increase transboundary transport of particles

  → Projections on increased mortality and respiratory health effects

- Indoor air pollution: Overall increase in biomass use and other air pollutants

  No projections

THE CLIMATE CHANGE CONUNDRUM: Environmental risks have become increasingly inter-dependent and complex!



Pyramide des effets de la pollution atmosphérique

- Effets à **court terme** et effets à **long terme,** mais continuité entre les 2 types d'effets

Plus la gravité des effets diminue plus le nombre de gens touchés augmente

Les PM de taille inférieure à 2,5 micromètres ($PM_{2,5}$) sont les plus dangereuses

I. Annesi-Maesano & W. Dab, Le livre de l'Interne. Pneumologie. Lavoisier Editeur 2013

What constitutes an adverse effect of air pollution?
Forthcoming **Joint ERS/ATS statement**

Rationale:

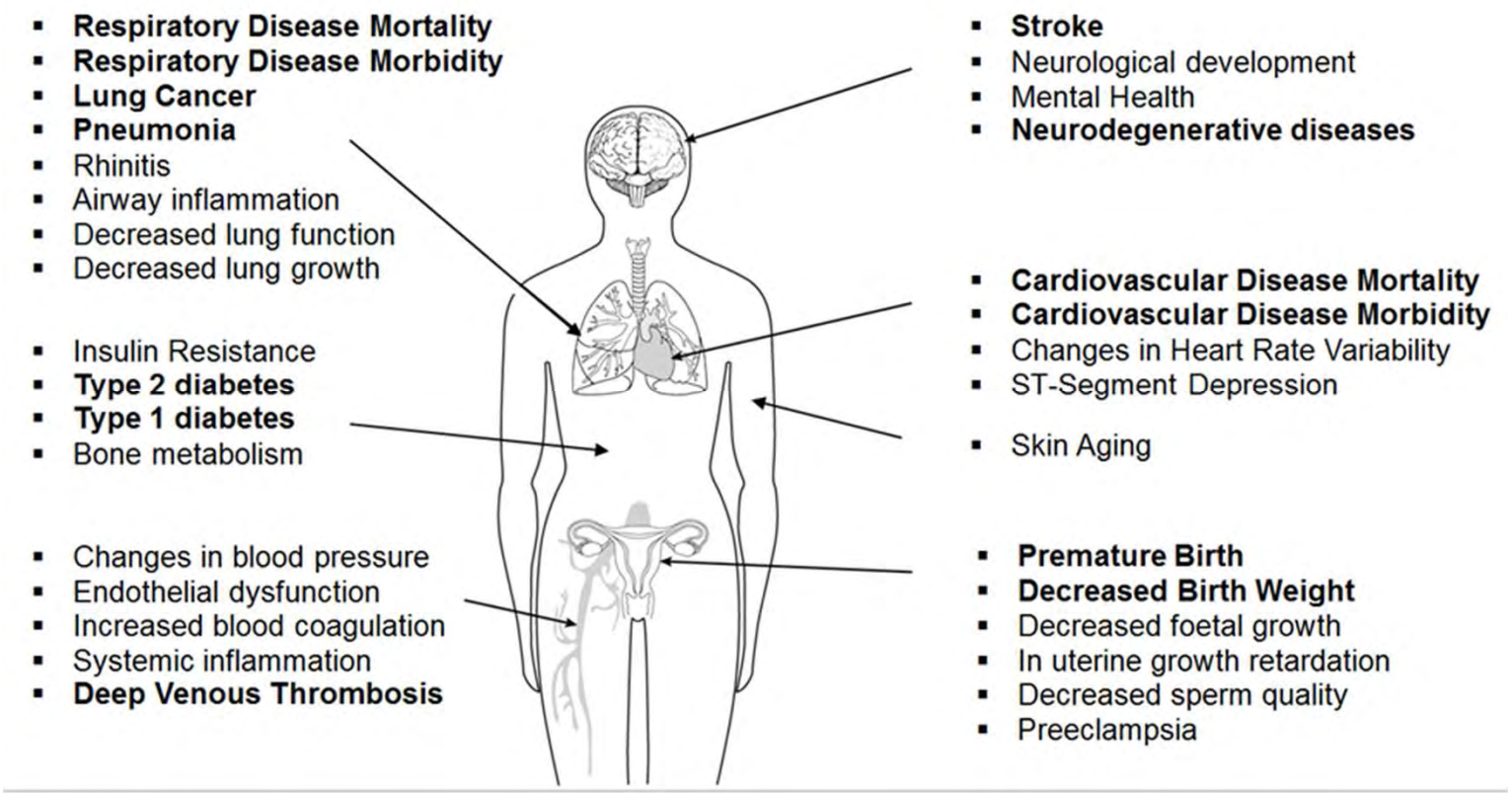
- Whether in the developed or developing world, the aim of air quality management is to limit or avoid any adverse impact of air pollution on the public's health. Thus, there is a need to identify those effects that are considered "adverse" and to separate them from those effects not considered adverse, thus focusing control measures on the pollutants causing, and populations experiencing, the greatest health impacts.
- 2000 Statement, "What Constitutes an Adverse Health Effect of Air Pollution" focused largely on impacts to the respiratory system. **However, since that time, new toxicological, clinical, and epidemiological studies have identified a wider range of health effects, often occurring at low levels of air pollution exposure (especially true for effects on the cardiovascular system).**
- New evidence is accumulating for adverse effects of air pollution on the Central Nervous System, reproduction and development, and certain metabolic outcomes.

→ the American Thoracic Society and the European Respiratory Society now update the ATS 2000 Statement to address these new scientific findings

New considerations for Assessing Adversity of Effects

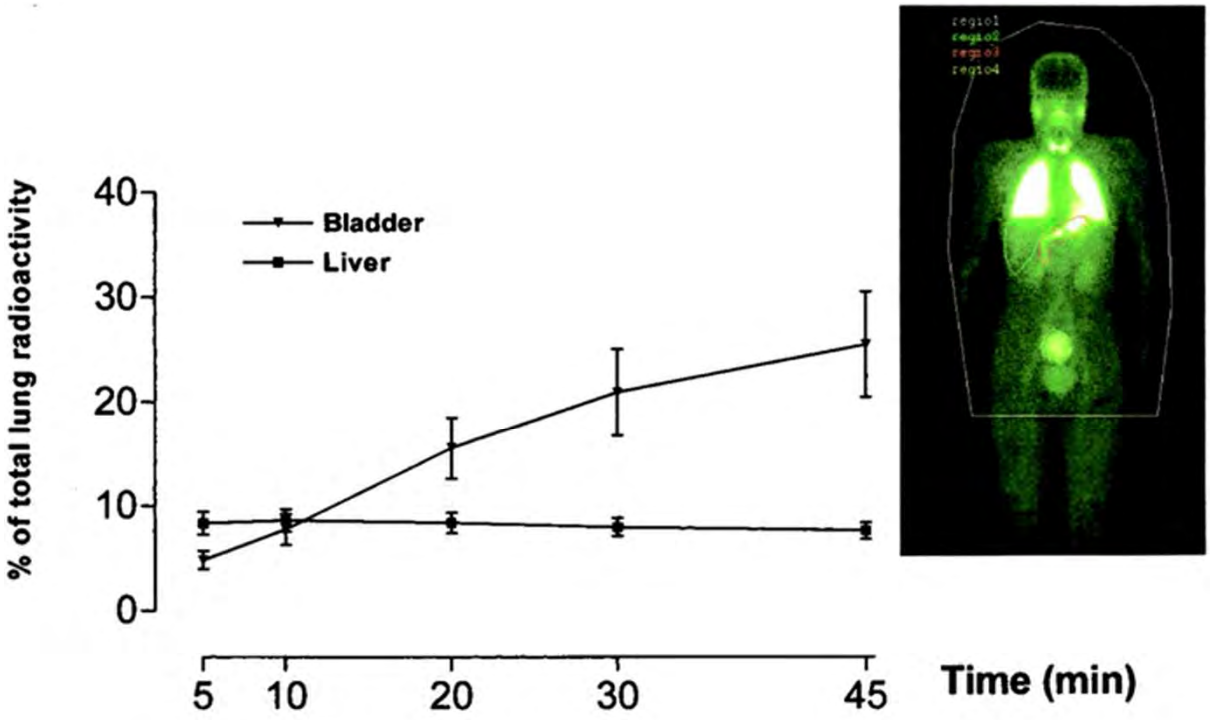
1.Persistence of Effect	*How persistent over time is the effect? Generally,, persistent effects are of greater concern, although short term reversible changes may increase risk for triggering acute adverse events, such as MI*
2. Population Risk	*Is there a shift in the population risk distribution of an adverse event?*
3. Susceptibility	*Are individuals with preexisting health conditions or geneti changes more affected?*
4. Medical/Functional Significance	*Is there evidence of one or more of the following:(1) interference with the activity of the affected person or persons, (2) incapacitating illness, (3) permanent injury, and/or (4) progressive dysfunction, (5) reduced quality of life?*

Overview of potential diseases, conditions and biomarkers affected by ambient air pollution



+ leukemia (Filippini, J Environ Sci Health C Environ Carcinog Ecotoxicol Rev., 2015)

PM penetration



Nemmar et al. Circulation 2002;105:411

Exacerbation for IPF and air pollution

Lucile SESE, Hilario NUNES, Dominique VALEYRE, Isabella ANNESI-MAESANO et les membres de la cohorte COFI

Table 2: Short effect of air pollution on exacerbations

Exposure	HR (95%CI)	p-value
O_3	1.0234 (1.0005 - 1.0468)	0.045*
NO_2	0.9915 (0.9667-1.0169)	0.509
PM_{10}	0.9750(0.9382-1.0132)	0.197
$PM_{2.5}$	1.0037(0.9462-1.0648)	0.902

Adjusted on: age, FVC, DLCO, tabac

ERJ (soumis)

Pollution atmosphérique et mortalité chez des patients FPI

IPF suite

Lucile SESE, Hilario NUNES, Dominique VALEYRE, Isabella ANNESI-MAESANO et les membres de la cohorte COFI

Tableau 5 : Cumulative air pollution exposure and mortality

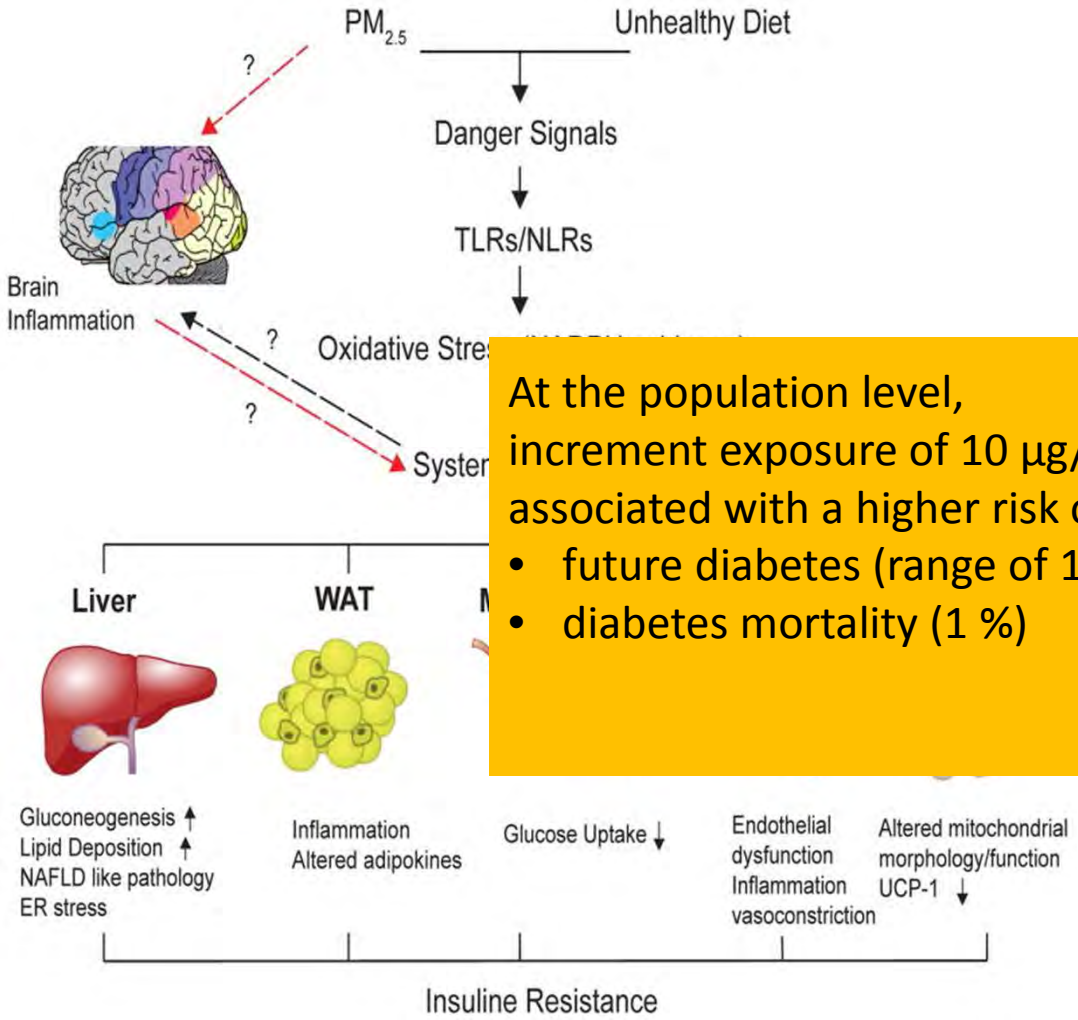
Exposition (upper quartile)	Dead (n=109)	Alive (n=83)	p-value
O_3 > 47 ug/m^3	33(30.8)	14(18.2)	0.05
$N0_2$ > 36 ug/m^3	36(33.3)	15(19.5)	0.03*
PM_{10} > 27 ug/m^3	50(46.7)	11(14.3)	<0.01*
$PM_{2.5}$ > 20 ug/m^3	20(42.6)	9(23.1)	0.06

Table 6: Association of cumulative air pollution exposure and mortality

Exposure	HR (95%CI)	p-value
O_3	0.9884(0.960-1.017)	0.4253
$N0_2$	1.0015(0.977-1.026)	0.9024
PM_{10}	1.0724 (1.007-1.142)	0.0295*
$PM_{2.5}$	1.230 (1.1136-1.358)	<0.001*

Ajusté: age, CVF, DLCO, tabac

Hypothesized mechanisms of air-pollution mediated type II DM/insulin resistance



At the population level,
increment exposure of 10 µg/m^3 of $PM_{2.5}$
associated with a higher risk of:

- future diabetes (range of 10 to 27 %)
- diabetes mortality (1 %)

Esposito, Endocrine 2015

E. Thiering & J. Heinrich
Trends Endocrinol Metab 2015

TLR Toll like receptor, NLR Nod like receptor
NADPH oxidase (nicotinamide adenine dinucleotide phosphate-oxidase)
BAT Brown Adipose Tissue WAT white adipose tissue
Non-alcoholic fatty liver disease (**NAFLD**)

J Environ Sci Health C Environ Carcinog Ecotoxicol Rev. 2015;33(1):36-66. doi: 10.1080/10590501.2015.1002999.

A review and meta-analysis of outdoor air pollution and risk of childhood leukemia.

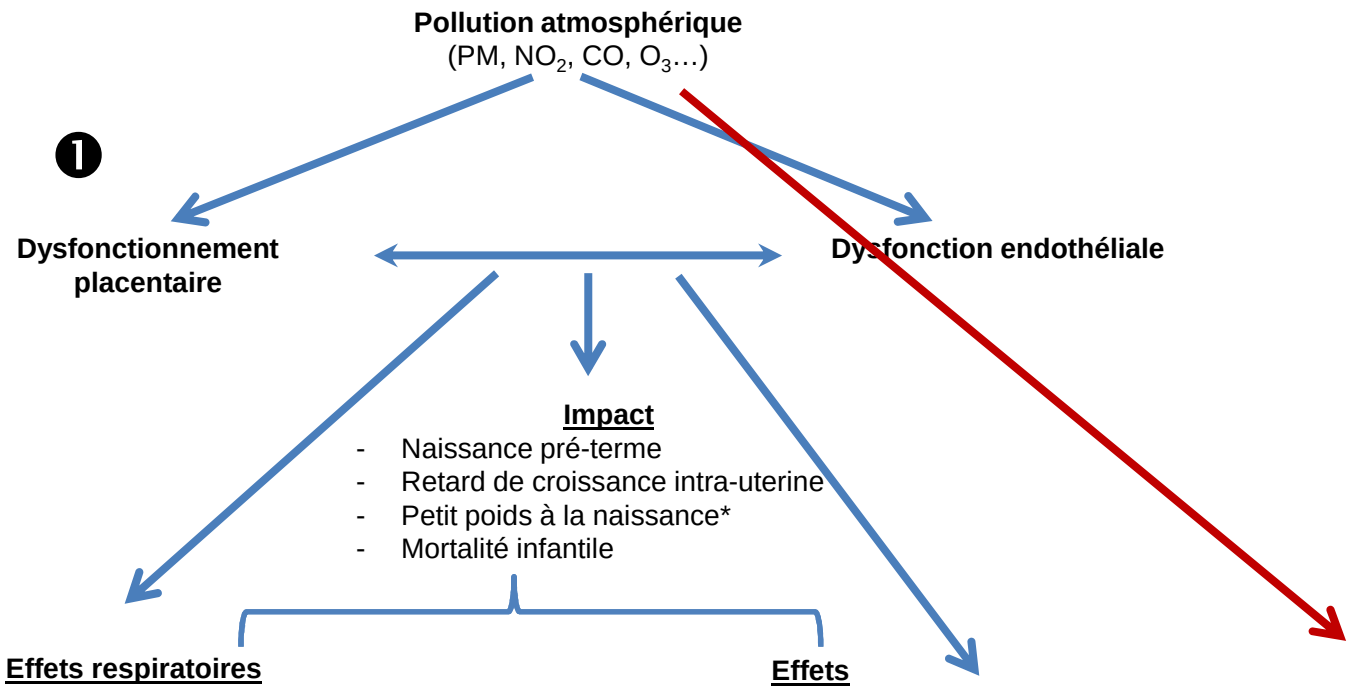
Filippini T[1], Heck JE, Malagoli C, Del Giovane C, Vinceti M.
Author information

Abstract

Leukemia is the most frequent malignant disease affecting children. To date, the etiology of childhood leukemia remains largely unknown. Few risk factors (genetic susceptibility, infections, ionizing radiation, etc.) have been clearly identified, but they appear to explain only a small proportion of cases. Considerably more uncertain is the role of other environmental risk factors, such as indoor and outdoor air pollution. We sought to summarize and quantify the association between traffic-related air pollution and risk of childhood leukemia, and further examined results according to method of exposure assessment, study quality, leukemia subtype, time period, and continent where studies took place. After a literature search yielded 6 ecologic and 20 case-control studies, we scored the studies based on the Newcastle-Ottawa Scale. The studies assessed residential exposure to pollutants from motorized traffic by computing traffic density in the neighboring roads or vicinity to petrol stations, or by using measured or modeled nitrogen dioxide and benzene outdoor air levels. Because heterogeneity across studies was observed, random-effects summary odds ratios (OR) and 95% confidence intervals (CI) were reported. Whenever possible we additionally conducted stratified analyses comparing acute lymphoblasticleukemia (ALL) and acute myeloid leukemia (AML). Limiting the analysis to high-quality studies (Newcastle-Ottawa Scale ≥ 7), those using traffic density as the exposure assessment metric showed an increase in childhood leukemia risk in the highest exposure category (OR = 1.07, 95% CI 0.93-1.24). However, we observed evidence of publication bias. Results for NO2 exposure and benzene showed an OR of 1.21 (95% CI 0.97-1.52) and 1.64 (95% CI 0.91-2.95) respectively. When stratifying by leukemia type, the results based upon NO2 were 1.21 (95% CI 1.04-1.41) for ALL and 1.06 (95% CI 0.51-2.21) for AML; based upon benzene were 1.09 (95% CI 0.67-1.77) for ALL and 2.28 (95% CI 1.09-4.75) for AML. Estimates were generally higher for exposures in the postnatal period compared to the prenatal period, and for European studies compared to North American studies. Overall, our results support a link between ambient exposure to traffic pollution and childhood leukemia risk, particularly due to benzene

Cela commence dès la vie *in utero* voire la préconception

Effets précoces de la pollution atmosphérique



*1.05 (0.99-1.12) per 10 µg/m^3 $PM_{2.5}$ to 1.10 (1.05-1.15) per 20 µg/m^3 PM_{10}

Stieb DM et al. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. Environ Res. 2012

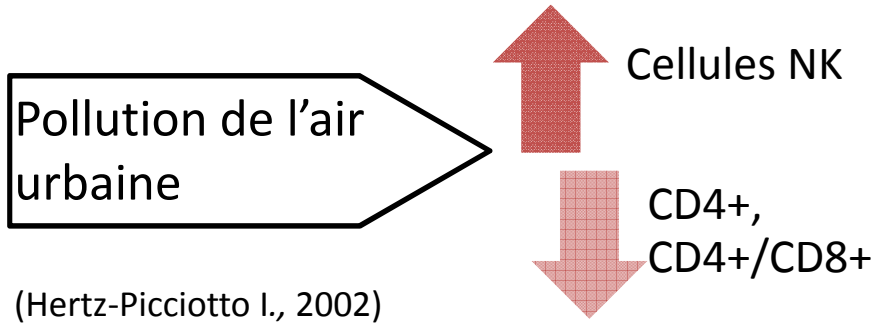
❷

Action sur le système immunitaire:

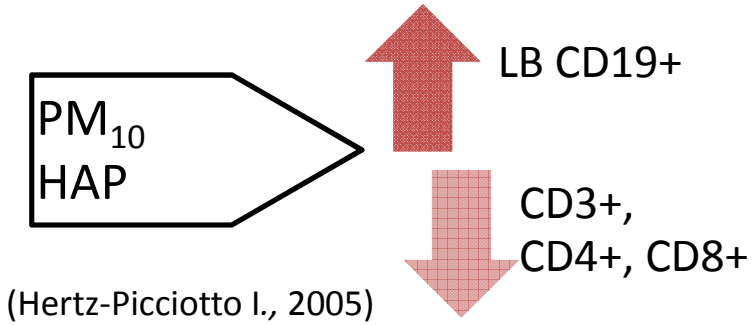
Diminution des lymphocytes T Reg (%CD4+CD25+) en function de l'exposition de la mère avant et après la grossesse aux particules fines

Hertz-Picciotto, Baïz

Liens entre exposition précoce à polluants atmosphériques et développement du système immunitaire



(Hertz-Picciotto I., 2002)



(Hertz-Picciotto I., 2005)

Hertz-Picciotto I. et al. Air pollution and distribution of lymphocyte immunophenotypes in cord and maternal blood at delivery, *Epidemiology, 13:172-183, 2002;*

Hertz-Picciotto I., Herr CEW, Yap PH., Dostál M., Shumway RH., Ashwood P., Lipsett M., Joad JP., Pinkerton KE., Sram RJ. Air pollution and Lymphocytes Phenotype Proportions in Cord Blood. *Environ Health Perspect*., Vol 113, number 10, 2005).

Exposition maternelle à la pollution atmosphérique et distribution des immunophénotypes lymphocytaires dans le sang de cordon ombilical . Etude de Cohorte EDEN (Baiz et al. BMC Perinat Care 2012)

Exposition croissante de la femme au **NO_2** (N=370)

CD8+ (β=**+0.71** [-0.02; 1.44]),
NK **(log)** (β=**+0.09** [0.01; 0.17])

Exposition croissante de la femme aux **PM_{10}** (N=370)

CD8+(β=**+1,52** [0,21; 2,83]),
NK(β=**+0.22** [0.02; 0.42])

CD4+CD25+ (β=**-0.82** [-1.53; -0.11]),
CD4+/CD8+ (β=**-0.28** [-0.59; 0.03]),
CD3+ (β=**-2.85** [-5.67; -0.03])

Exposition croissante de la femme au **benzène** (N=57)

CD4+CD25+ (β=**-0.76** [-1.62 to 0.10])

AIR POLLUTION AND ARLY LIFE EVENTS

Author, year of publication	Prenatal exposure	Main results
Slama et al,[21] 2009	Benzene	Reduced BW and HC
Aguillera et al,[10] 2010	BTEX	Reduced BPD
	NO_2	Reduced growth in HC, AC, BPD, and EFW for those women who spent more time outdoor
Llop et al,[23] 2010	NO_2	Increased risk for PB
	Benzene	Increased risk for PB
Ballester et al,[19] 2010	NO_2	Reduced length, weight, and HC at birth Positive association with SGA
Newman et al,[61] 2010	Active MTS	Positive association with SGA and reduced BW
	Passive MTS	No associations with birth outcomes
Estarlich et al,[16] 2011	NO_2	Reduced length and weight at birth
	Benzene	No association
Gehring et al,[25] 2011	NO_2	Highest BW and lowest risk of SGA No association with PB
Gehring et al,[15] 2011	NO_2 and $PM_{2.5}$	No association between air pollutants and term BW
Iñiguez et al,[17] 2012	NO_2	Reduced BPD, AC, and EFW Reduced neonatal length and HC
van den Hooven et al,[14] 2012	NO_2 and PM_{10}	Reduced fetal growth and BW Positive association with SGA and PB
Iñiguez et al,[58] 2012	MTS	
	Active	Reduced BPD, AC, FL, and EFW
	Passive	Reduced growth on BPD
Sunyer et al,[65] 2012	MTS	
	Active/passive	Reduced BW, increased risk for SGA Reduced BW for asthmatic mothers (no statistical significance)
Choi and Perera,[22] 2012	PAH	PA – maternal obesity exacerbated the risk of exposure to PAH on reduced BW
Ritz et al,[22] 2014	TRAP, NO, NO_x, NO_2, CO, and distance to monitoring station	Reduced BPD

Abbreviations: BW, birth weight; HC, head circumference; BTEX, aromatic hydrocarbons (benzene, toluene, ethylbenzene, m-/p-xylene, and o-xylene); BPD, biparietal diameter; NO_2, nitrogen dioxide; AC, abdominal circumference; EFW, estimated fetal weight; SGA, small for gestational age; MTS, maternal tobacco smoking; PB, premature birth; PM, particulate matter; FL, femur length; PAH, polycyclic aromatic hydrocarbons; TRAP, twin reversed arterial perfusion; NO, nitric oxide; NO_x, nitrogen oxides; CO, carbon monoxide.

Published in final edited form as:
Environ Res. 2014 October ; 134: 198–204. doi:10.1016/j.envres.2014.08.002.

Ha et al.

The effects of air pollution on adverse birth outcomes

Highlights

- Prenatal exposure to $PM_{2.5}$ may increase the risk of term LBW, PTD and VPTD.
- The effects of $PM_{2.5}$ on ABOs are strongest during the second trimester.
- Prenatal O_3 exposure may increase the risk of PTD and VPTD.

The protective association between O_3 and term LBW needs further investigation.

prenatal exposure to air pollutants including particulate matter with aerodynamic diameter less than 2.5 micrometer ($PM_{2.5}$) and ozone (O_3) on the risk of adverse birth outcomes (ABOs) including term low birth weight (LBW), preterm delivery (PTD) and very PTD (VPTD).

Epidemiology. 2015 Sep 30. [Epub ahead of print]

Air pollution and cognitive development at age seven in a prospective Italian birth cohort.

Porta D[1], Narduzzi S, Badaloni C, Bucci S, Cesaroni G, Colelli V, Davoli M, Sunyer J, Zirro E, Schwartz J, Forastiere F.
Author information

- [1]a Department of Epidemiology of the Lazio Regional Health Service, Rome, Italy b Centre for Research in Environmental epidemiology (CREAL), Barcelona, Spain c Department of Environmental Health, Harvard School of Public Health, Boston, MA, USA.

Abstract

BACKGROUND:
Early life exposure to air pollution has been linked with cognitive impairment in children, but the results have not been conclusive. We analyzed the association between traffic-related air pollution and cognitive function in a prospective birth cohort in Rome.

METHODS:
A cohort of 719 newborns was enrolled in 2003-2004 as part of the GASPII project. At 7 years of age, 474 children took the Wechsler Intelligence Scale for Children-III to assess their cognitive development in terms of IQ composite scores. Exposure to air pollutants (NO2, PMcoarse, PM2.5, PM2.5 absorbance) at birth was assessed using land use regression models. We also considered variables indicating traffic intensity. The effect of environmental pollution on IQ was evaluated performing a linear regression model for each outcome, adjusting for gender, child age at cognitive test, maternal age at delivery, parental educational level, siblings, socio-economic status, maternal smoking during pregnancy, tester. To account for selection bias at enrolment and during follow-up, the regression models were weighted for the inverse probabilities of participation and follow-up.

RESULTS:
A 10 µg/m higher NO2 exposure during pregnancy was associated with 1.4 fewer points (95%CI:-2.6 to -0.20) of Verbal IQ, and 1.4 fewer points (95%CI:-2.7 to -0.20) of Verbal Comprehension IQ. Similar associations were found for traffic intensity in a 100mt buffer around home. Other pollutantsshowed negative associationswith larger confidence intervals.

CONCLUSIONS:
Consistent with previous evidence, this study suggests an association of exposure to NO2 and traffic intensity with the verbal area of cognitive development.

Neurological and psychiatric conditions associated with air pollution

.
Alzheimer's disease and other dementias
Parkinson's disease
Reduced cognitive function in adults
Reduced neurodevelopment in children
Depression
Anxiety disorders

Examples of markers of neurological effects.

Structural brain damage at functional magnetic resonance imaging (FMRI)
Neurobehavioral testing
Cognitive function testing


EDC
Körsbärstomat 199,00
Plastkasse liten 9,95
Rågbröd 2,00
Ägg 12-pack Kronäg 16,95
Pant 21,95
-4,00
Total 304,70
Moms% Moms Netto Brutto
25,00 0,40 1,60 2,00
12,00 11,11
6,00
skin resurfacing cleanser
Building materials
Dietary, cook wares and packages
Sources are everywhere… everywhere!

Four reasons for emerging public health concern for EDC-exposure

The ubiquity of exposure

Low doses/non-monotonic

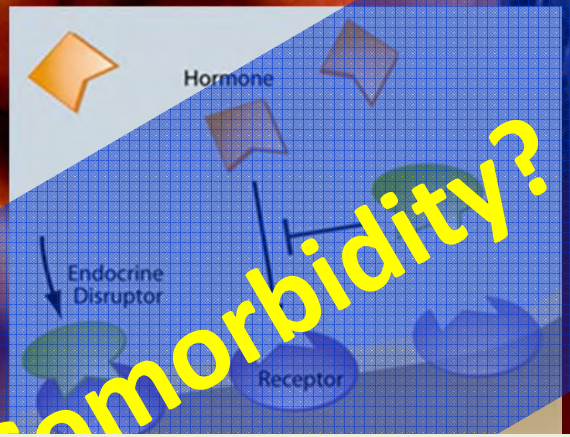
The persistence of effects

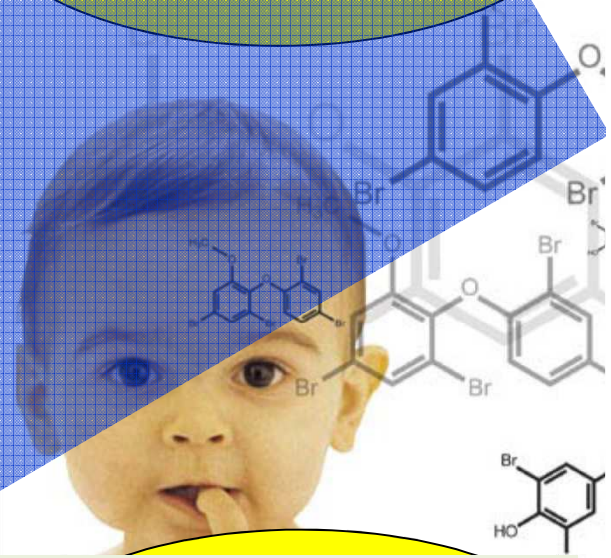
The wide range of health effects

Immunological effects

Reproductive health

Respiratory health



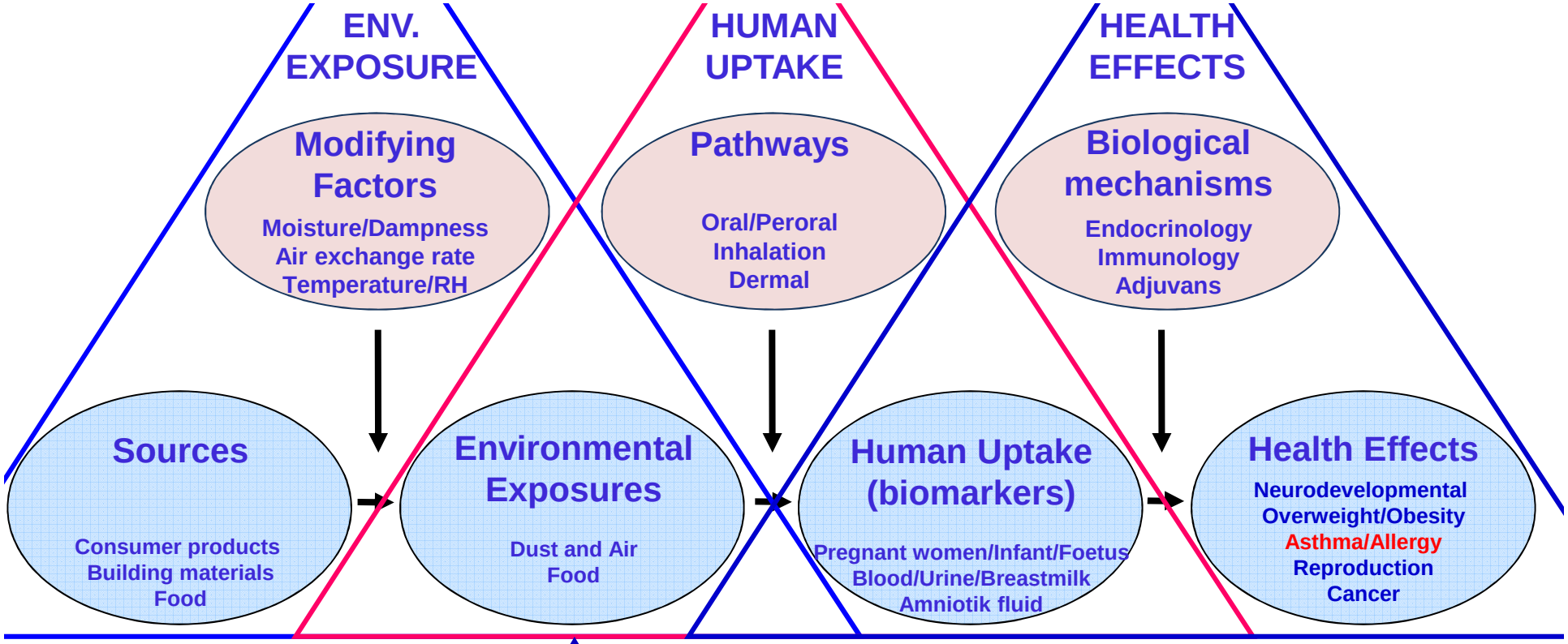


Metabolic synd
incl. diabet

emorbidity?

ginning a lifetime of vulne
ro exposures to endocrine-disru

Studies on urinary metabolites or objective assessments of EDCs



PVC, Polish
Bornehag et al., 2005; Kolarik et al., 2008a

Phthalates in Indoor Air/Dust
Adibi et al., 2003, Adibi et al., 2008; Kolossa-Gehring et al., 2011; Hsu et al., 2011; Bekö et al., 2013

PVC
Carlstedt et al., 2013; Shu et al., 2013B *in manuscript*; Shu et al., 2013C *in manuscript*

PVC
Jaakkola et al., 1999; Oie et al., 1999; Jaakkola et al., 2000; 2004; Bornehag et al., 2005; Kolarik et al., 2008b; Larsson et al., 2009; Choi et al., 2011; Shu et al., 2013A

Phthalates in Indoor Air/Dust
Bornehag et al., 2004; Kolarik et al., 2008b; Hsu et al., 2011; Callesen et al., 2011; Kishi et al., 2012

Phthalate metabolites in urine
Hsu et al., 2011; Just et al., 2012; Callesen et al., 2013; Bertelsen et al., 2013; Whyatt et al., 2013; Hoppin et al., 2013

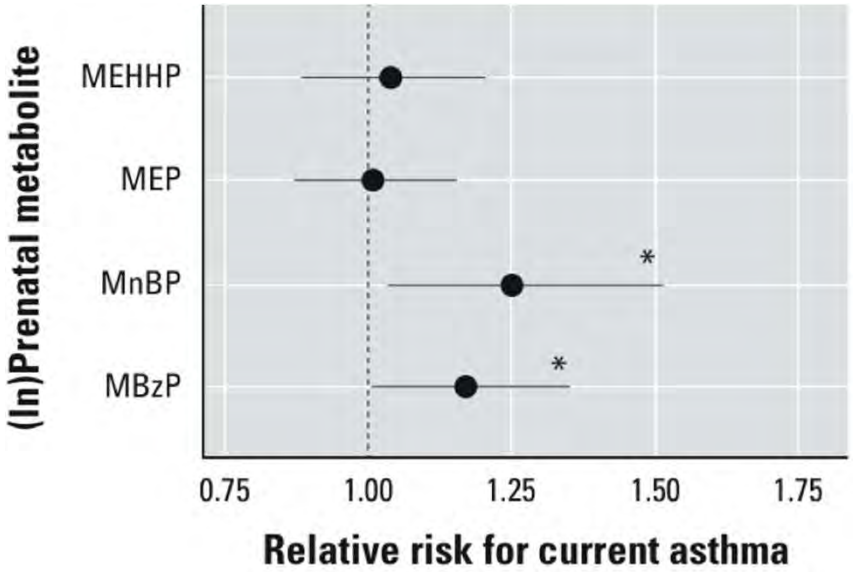
Effect of prenatal exposure to phthalates on child's asthma

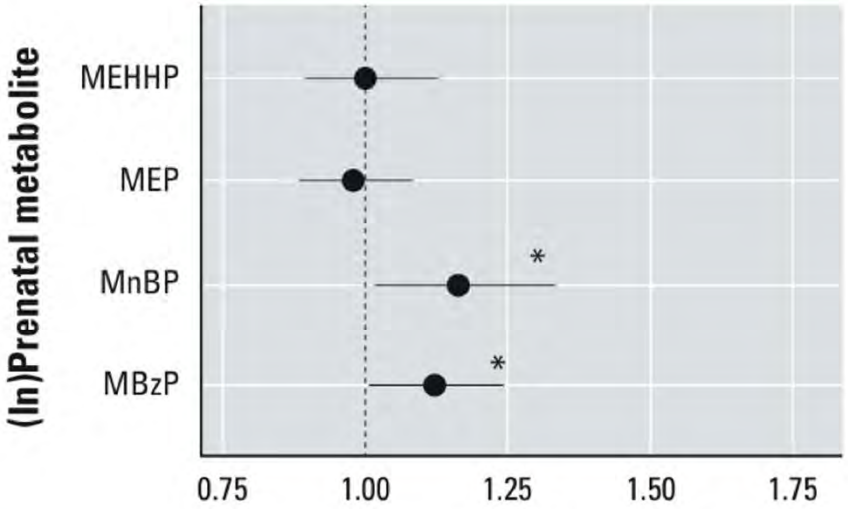
- Phthalates are a class of synthetic compounds used widely in polyvinyl chloride plastics, in cosmetics, and in building materials.

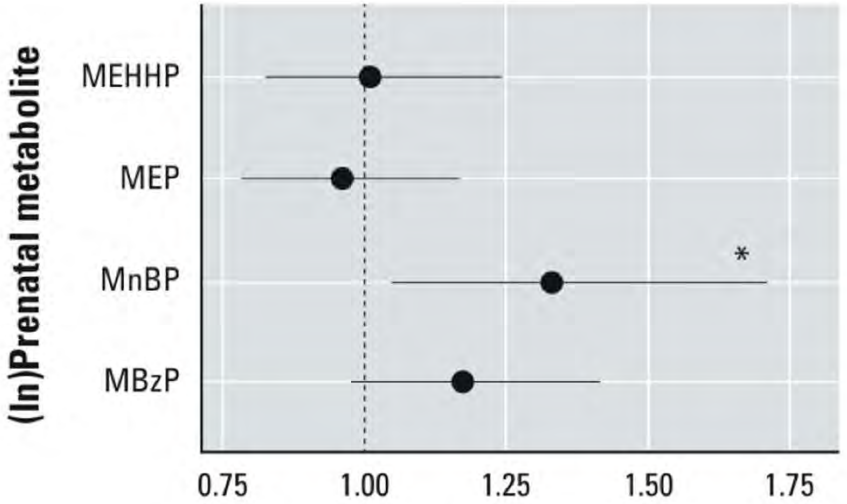
- Prenatal exposure to two phthalates - chemicals commonly used in plastics and cosmetics, such as skin moisturizers - may significantly increase the risk of childhood asthma.

Source : Whyatt Rmet al, Environ Health Perspect. 2014 Oct;122(10):1141-6

Asthma in Inner-City Children at 5-11 years of age and prenatal exposure to Phthalates, Whyatt, EHP, 2014







EDCs exposure in pregnancy and lung function

371 mother-child pairs

To our knowledge, this is the first study to estimate the effect of prenatal phenol and phthalate exposures on the lung function measured in childhood. Our results did not indicated association between phenols or phthalate metabolite exposures, considered as continuous, and lung function parameters at 5 years. However we observed borderline association with BPA exposure by a small but statistically significant impairment in FVC% predicted with an increase in exposure to BPA. In addition, when exposures were categorized in tertiles, we observed lower FEV_1% (-2.64%, 95% CI, -5.33; 0.05) in the medium MCNP tertile compared to the lowest.

I. Annesi-Maesano Submitted

Biological plausibility for EDC

- Modulation of immune system (balance lymphocytes Th1 and Th2)
- Th2 promoted antigen specific production of IgE and IgG1
- Eosinophilic inflammation of the lung and the airways
- Oxydative stress
- ….

EFFETS SUR LA SANTÉ DES ÉMISSIONS DE MOTEURS DIESEL. APPROCHE ÉPIDÉMIOLOGIQUE.

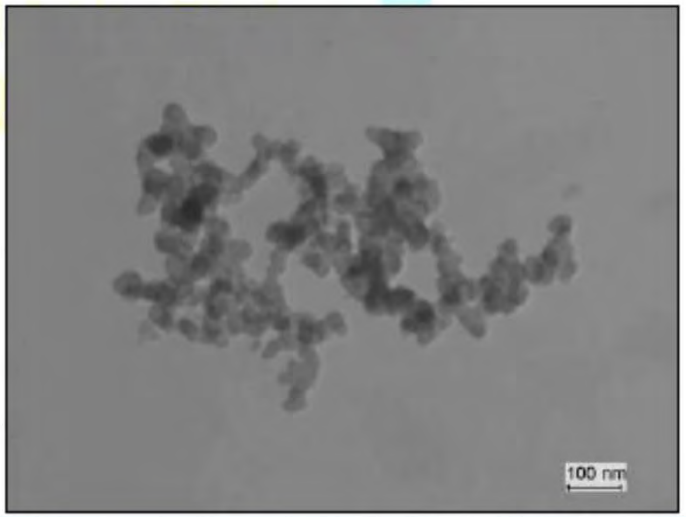
Le moteur diesel

L'inventeur : Rudolf Diesel (1858 – 1913)

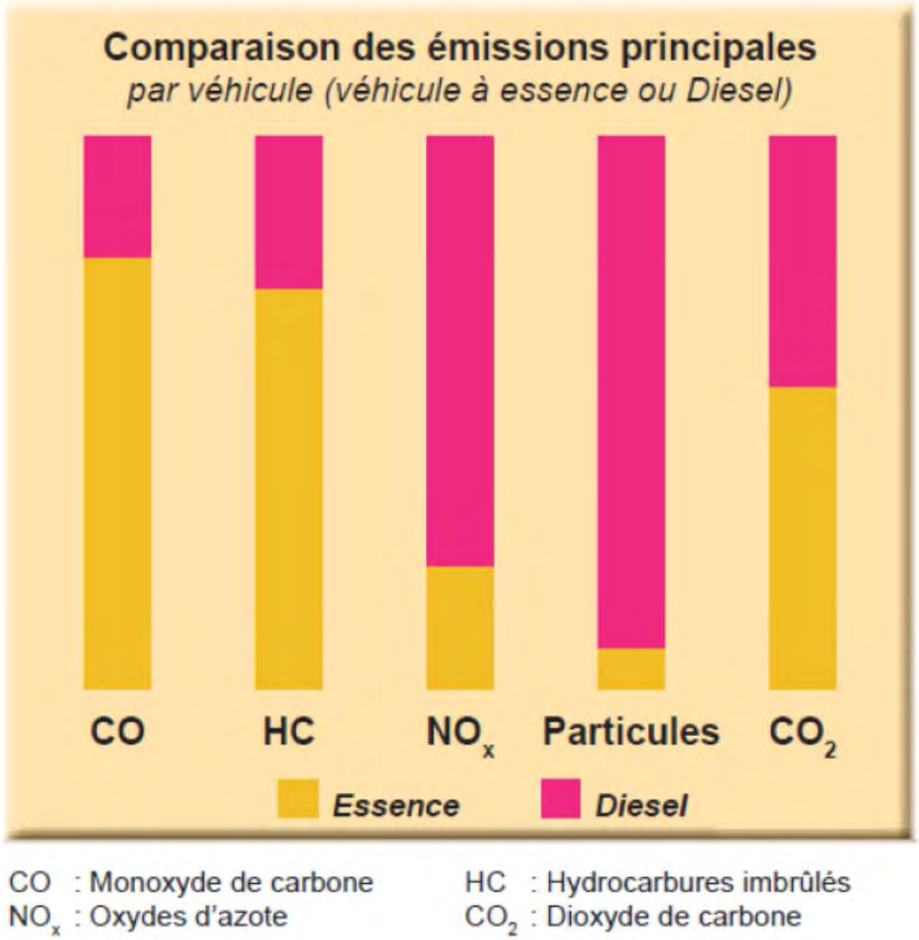
Un moteur à combustion interne avec allumage par compression pouvant utiliser comme carburant des huiles lourdes et des huiles végétales et non du gazole

Les émissions diesel

La combustion du carburant dans le moteur diesel produit une pollution chimiquement complexe de gaz et de vapeurs auxquels s'ajoutent une phase particulaire.



Moteur diesel et essence



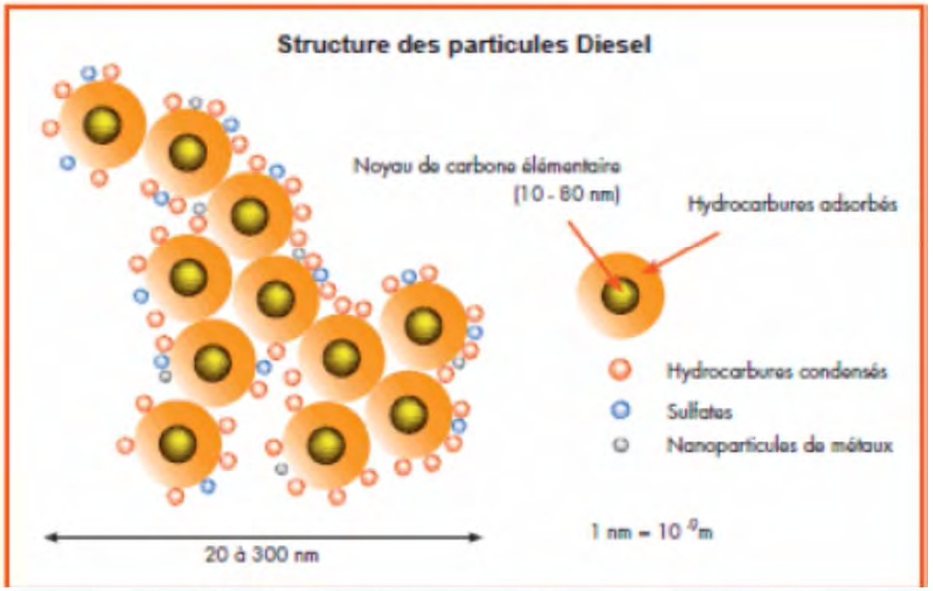
Les moteurs diesel produisent beaucoup plus de particules (et d'oxydes d'azote) que les moteurs à essence.

Autres composantes de la phase gazeuse: eau, aldéhydes, hydrocarbures de bas poids moléculaire (ex. toluène), HAP légers (2-4 cycles) non cancérogènes

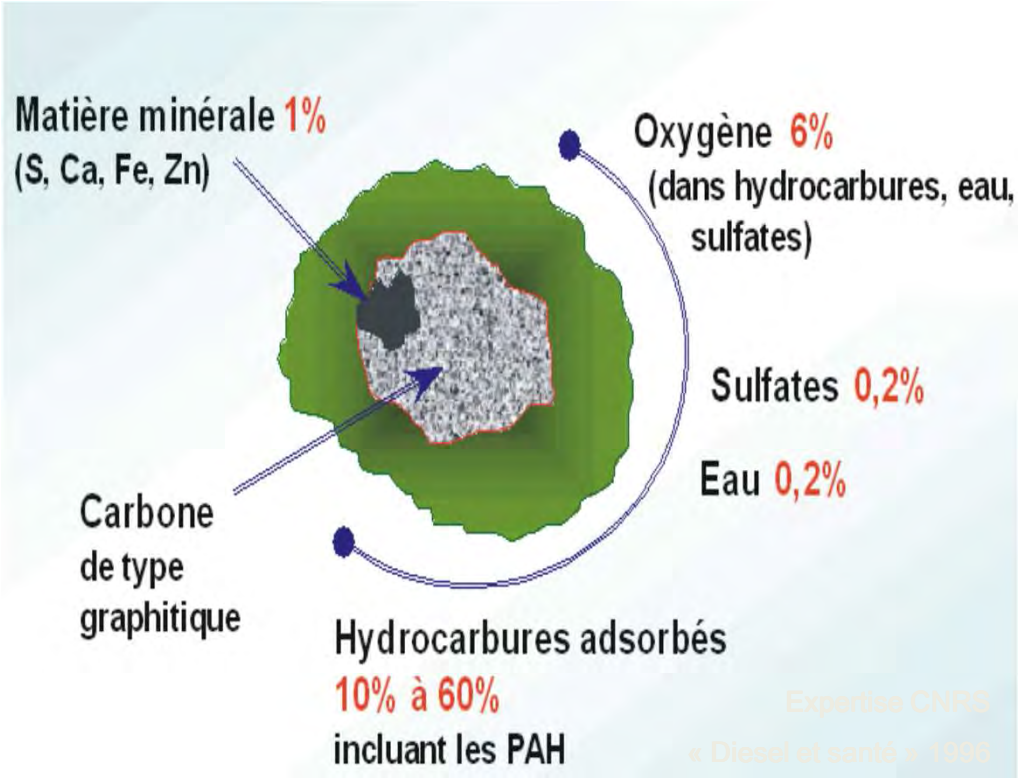
Les émissions diesel

Principaux composants des particules diesel :

- **Carbone élémentaire** (fraction insoluble) : **50-75 %** de la masse des particules
- **Carbone organique** (fraction soluble) : **19-43 %** de la masse des particules. Contient divers composés organiques légers ou lourds, notamment des HAP et des nitro-HAP, dont certains sont cancérogènes (benzo(a)pyrène , 1-nitro-pyrène)
- Sulfates : 1-4 %
- Métaux (1-5 %)
- autres composés…



Composition moyenne du DIESEL



Source : CNRS

Les effets sur la santé

- A court terme
  - Effets irritants (oculaires, respiratoires (asthme…), cutanés)
  - Céphalées, nausées
  - Intoxication au CO (très rare)
- A long terme
  - Réaction inflammatoire des voies aériennes
  - Toux et expectoration chroniques
  - Asthma, BPCO
  - Effets génétoxiques et cencérogènes
  - Effets sur le système CDV envisagés mais non prouvés

Diesel Exhaust Emissions-Health Effects

- Short-term: breathing in diesel fumes can cause coughing, itchy or burning eyes, chest constriction, wheezing, and difficulty breathing.
- Long- term exposure to diesel may increase the risk of asthma
- Long term exposure to diesel fumes may increase the risk of lung cancer and possibly bladder cancer.
- There is additional evidence that the fine particles in DPM can aggravate heart problems.

Diesel and asthma. Evidence.

- Increased asthma occurrence (McConnell, 2006)
- Traffic related pollutants are associated with airway inflammation reduced lung function, and reduced lung development (Gauderman 2012)
- Increased respiratory symptoms (Gauderman, 2005, Ryan 2007)
- Increased doctor visits and use of medications (English, 1999)
- Increased risk of ER and/or hospitalizations
- Cause or contribute to new-onset asthma
- Interact with allergens to increase intensity of allergic response in susceptible individuals
  - DEP can disrupt the regulation of the immune system in sensitive individuals, which can increase their risk of having allergic reactions to other substances in their environments

Diesel and asthma. Mechanisms.

- Irritant effect
- Immunologic effect
- Adjuvant effect

MECANISMES

Diesel

- Adjuvant de la réponse allergique
- Induit un stress oxydatif:
  - Les radicaux libres de l'oxygène interagissent avec les lipides, les protéines et l'ADN, aboutissant à des lésions cellulaires
  - Effet toxique direct / épithélium respiratoire

(Baulig, Toxicol In Vitro 2003)

- Le diesel augmente la synthèse d'IgE
  - Diesel seul : ↑ IgE totales
  - Diesel + allergène : ↑ IgE spécifiques

  (Diaz-Sanchez; J Immunol 1997)
- Le diesel modifie le profil de cytokines
  - ↑ IL-4, IL-5, IL-10, IL-13

  (Diaz-Sanchez , JCI…1996-1999)
- Exposition de volontaires sains !!!
  - ↑ neutrophiles, ↑ CD4+ et CD8+ T cells
  - ↑↑ IL-5 mRNA

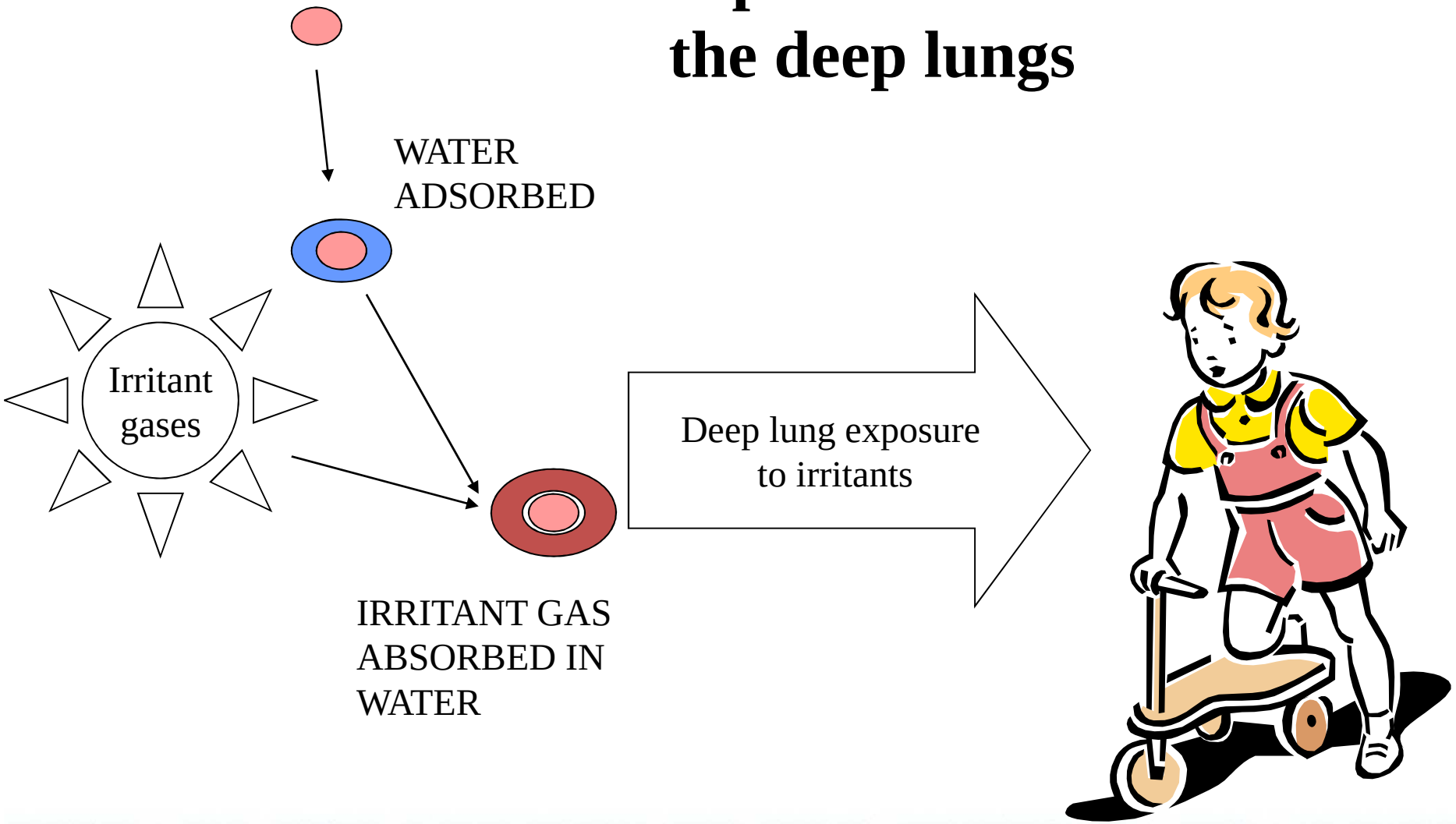
  (Salvi S. AJRCCM 1999)

Diesel (mais aussi O_3, NO_2)

- Réponse inflammatoire non spécifique des voies aériennes

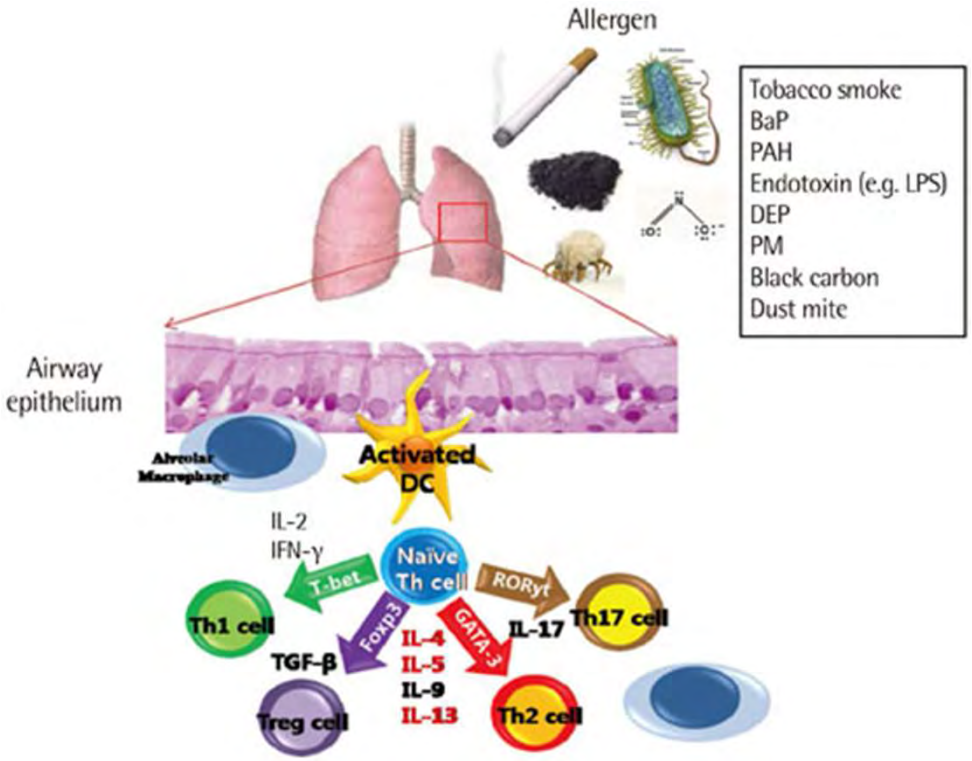
- Les particules diesel peuvent fixer des allergènes
  - Chat, pollens
- Les antigènes peuvent être modifiés dans leur conformation, et parfois augmenter l'immunogénicité

How particles increase exposure of irritants to the deep lungs



Diesel as an airway irritantant

- Diesel contains 40 toxic air pollutants & many are irritants
- Trigger oxidative/inflammatory cascade
- Reactive airways dysfunction syndrome or irritant-induced asthma
- Railroad workers riding behind locomotive at increased risk of asthma



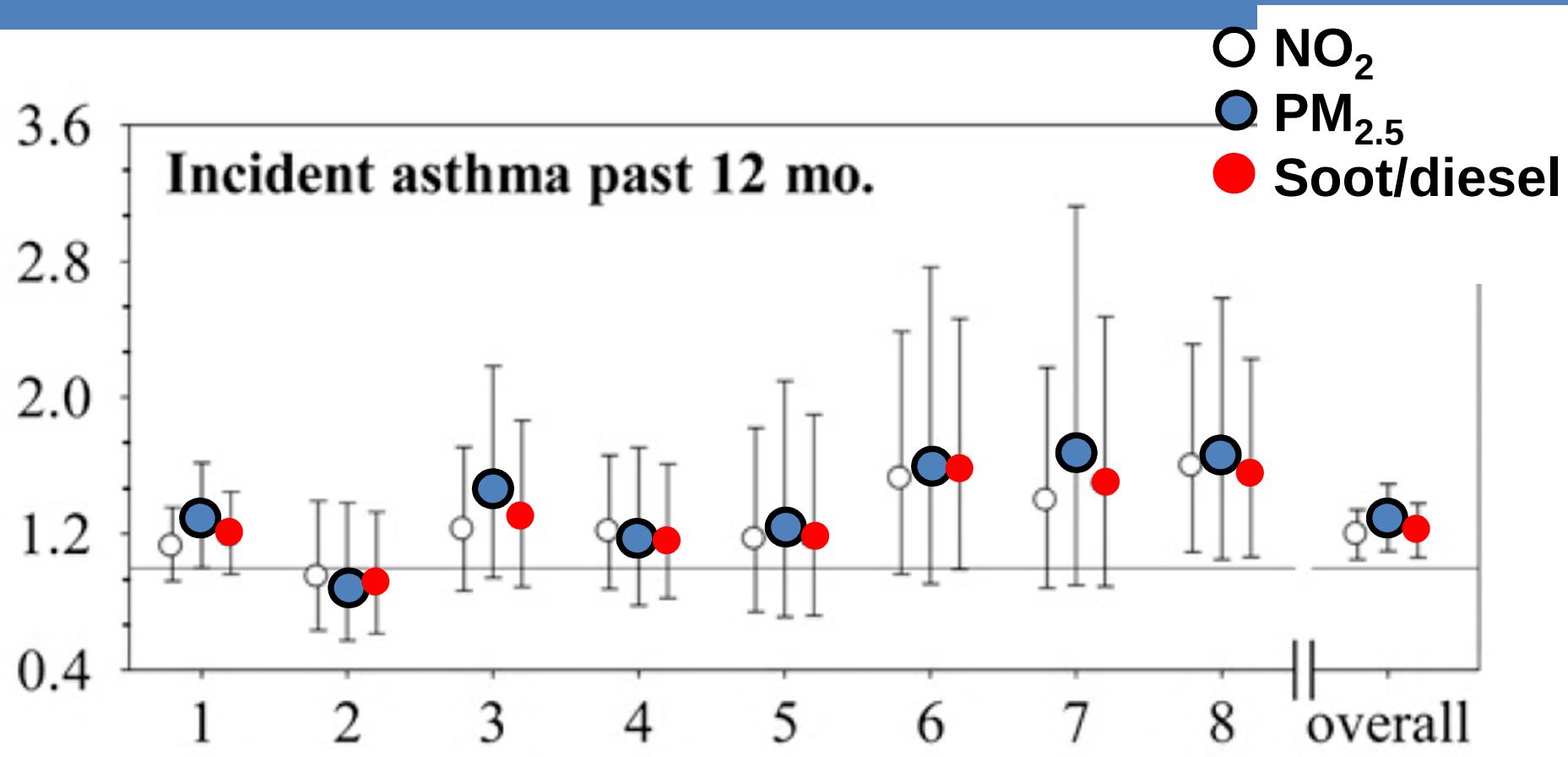
Diesel and immune changes

- Diesel exhaust exposure promotes
  - IgE release
  - Interleukin release
  - Eosinophil production
- Increases in TH2 cells

Does air pollution induce new cases of asthma? Taking into account birth cohorts!

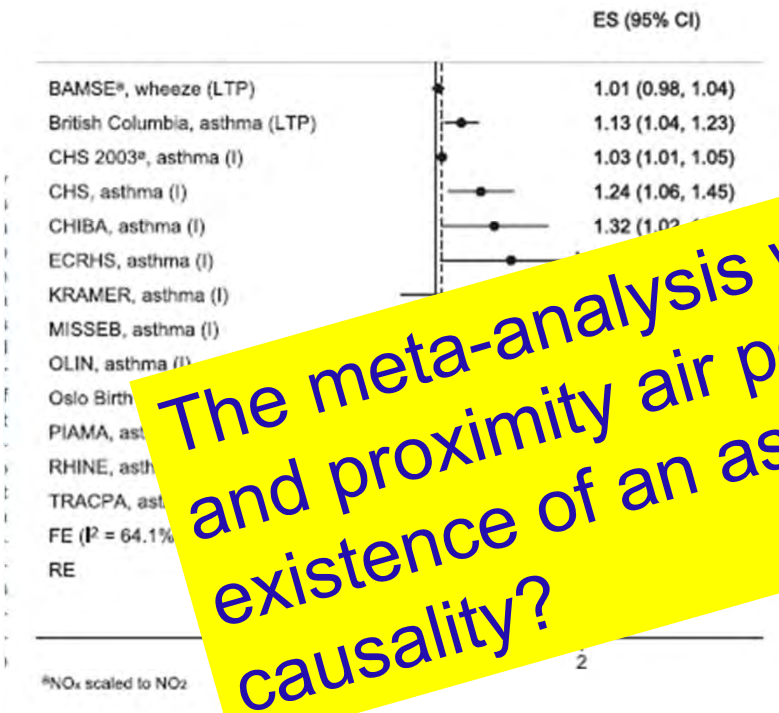
Traffic-related pollution at birth is associated with the onset of childhood asthma during the first 8 years of life (Dutch children)

Gehring et al, AJRCCM 2010

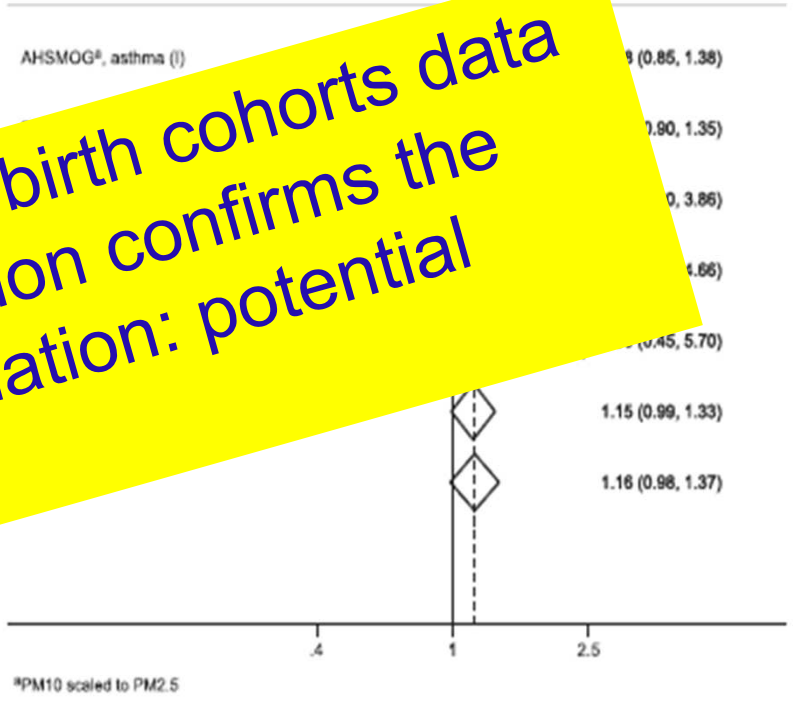


Air pollution exposure and asthma incidence
Birth cohort studies

- NO_2



- $PM_{2.5}$

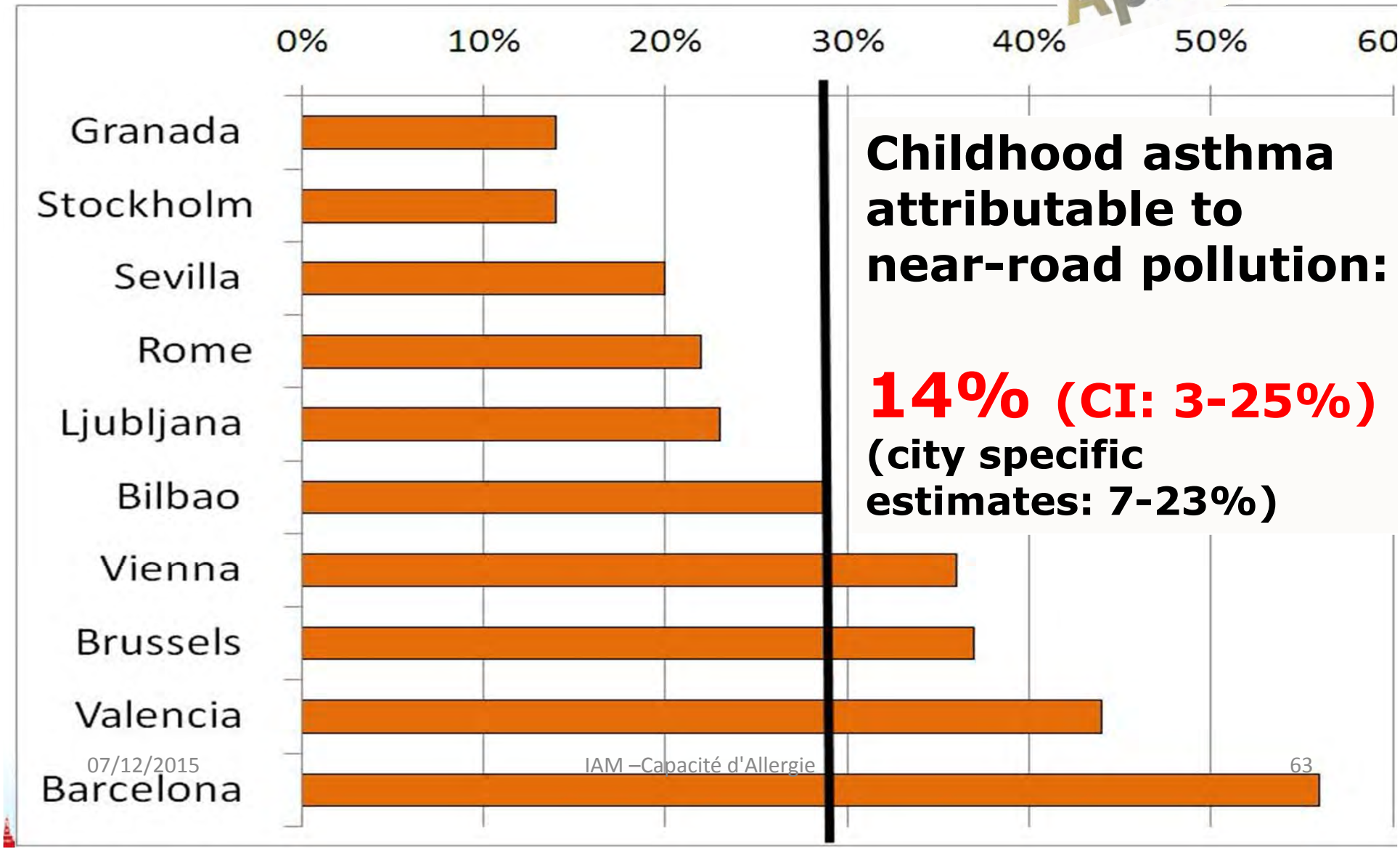


The meta-analysis with birth cohorts data and proximity air pollution confirms the existence of an association: potential causality?

Gower, Respirology 2012

% of people living within 75 m of roads with >10’000 vehicles per day!

Perez et al, - Eur Respir J –2013

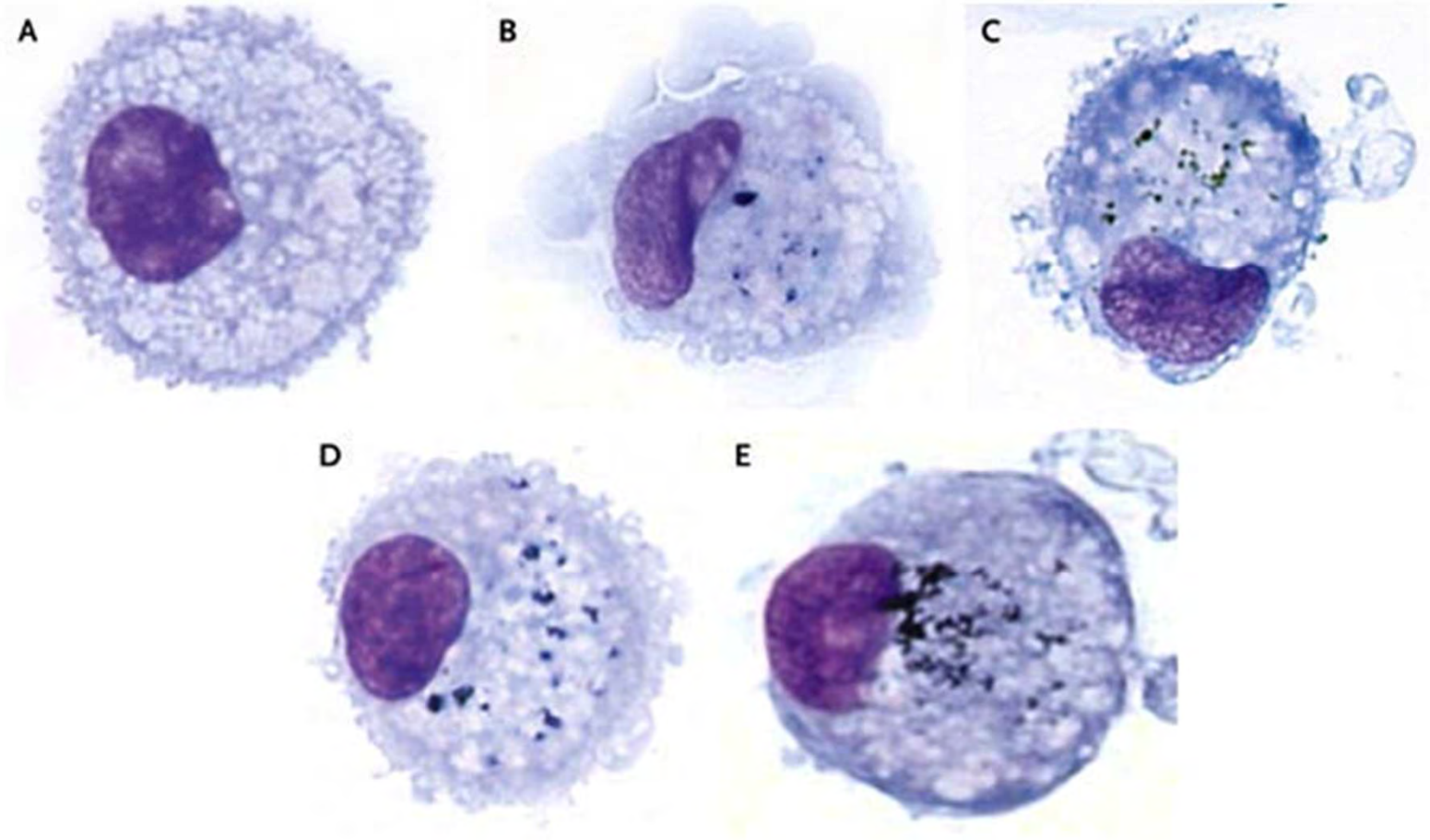


Childhood asthma attributable to near-road pollution:

14% (CI: 3-25%)

(city specific estimates: 7-23%)

Macrophages chargés en carbone chez des enfants



Relation entre la Modélisation Annuelle en PM_{10} et les Macrophages chargés en carbone

Kulkarni N et al. N Engl J Med 2006;355:21-30

Air pollution and allergic sensitisation (specific IgE)

The meta-analysis with birth cohorts data and modeled air pollution do not show a significant link

…13 Oct 3. pii: S0091-
…2. doi:
…016/j.jaci.2013.07.048. [Epub ahead of print]

Meta-analysis of air pollution exposure association with allergic sensitization in European birth cohorts.

Gruzieva O,

Overall, air pollution exposure was not associated with sensitization to any common allergen, with odds ratios ranging from 0.94 (95% CI, 0.63-1.40) for a $1 \times 10^{-5} \cdot m^{-1}$ increase in measurement of the blackness of $PM_{2.5}$ filters to **1.26 (95% CI, 0.90-1.77) for a 5 µg/m³ increase in $PM_{2.5}$ exposure at birth address**. Further analyses did not provide consistent evidence for a modification of the air pollution effects by sex, family history of atopy, or moving status.

Les effets cancérogènes

- Première évaluation par le CIRC (1988) → classement groupe 2A (cancérogène probable pour l'homme)
  - Chez l'animal: « preuves suffisantes » d'une association causale entre l'exposition aux émissions diesel et le risque de cancer pulmonaire
  - Chez l'homme: « preuves limitées » d'un effet cancérogène pulmonaire
    - Etudes épidémiologiques: risque relatif de KP modéré
    - Nombreux biais ou facteurs de confusion potentiels: tabagisme, exposition à l'amiante
- Deuxième évaluation par le CIRC (2012) → classement des émissions diesel dans le groupe 1 (cancérogène certain pour l'homme)
  - « preuves suffisante » pour le lien entre le KP et l'exposition aux émissions diesel chez l'homme
  - « preuves limitées » pour le lien entre diesel et K de la vessie (résultats significatifs chez les travailleurs mais négatifs dans les études de cohortes)

PM and Lung cancer

Raaschou-Nielsen et al. Lancet Oncology 2013

- European Study of Cohorts for Air Pollution Effects (ESCAPE)
- Meta-analysis of 17 cohort studies in 9 European countries including almost 313 000 people, 2095 with lung cancer during the average 13 years of follow up Air pollution concentration estimated a t the home addresses using land-use regression models.
- **For every increase of 5 micrograms per cubic meter of $PM_{2.5}$ pollution, the risk of lung cancer rose by 18%, and for every increase of 10 micrograms per cubic meter in PM_{10} pollution the risk increased by 22%, with stronger effects indicated for adenocarcinomas**.

Traffic-related exposure studies on lung cancer. PM, particulate matter.

Study	Date	Location	Pollutants	Subject/location	Results
Vineis *et al.* [2006]	1993–1998	10 European countries	NO_2	adults aged 35–74 residing near heavy traffic roads	46% increase in lung cancer
Chiu *et al.* [2006]	1994–2003	Taiwan	PM10, SO_2, NO_2	females	28% increased risk of lung cancer
Edwards *et al.* [2006]	2000–2004	Teesside, northeast England	PM10, SO_2, NO_2	women aged <80 years living for >25 years close to highly industrialized area	83% increased risk of lung cancer
Loomis *et al.* [2014]	2003–2012	31 provincial capital cities in China	PM2.5	71,000 adults	increased risk of lung cancer
Raaschou-Nielsen *et al.* [2013]	2008–2011	17 separate European cohorts	PM10	312,944 adults	increased risk of lung cancer

PM, particulate matter.

Polosa et al 2014

Environmental Science & Technology

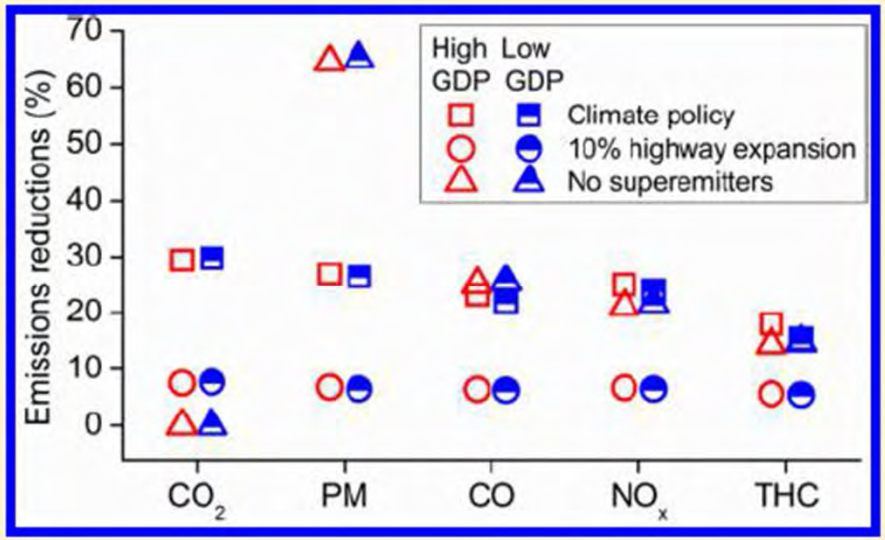
Article

pubs.acs.org/est

Emission Projections for Long-Haul Freight Trucks and Rail in the United States through 2050

Liang Liu,[†] Taesung Hwang,[†,a] Sungwon Lee,[‡] Yanfeng Ouyang,[†] Bumsoo Lee,[‡] Steven J. Smith,[§] Fang Yan,[∥] Kathryn Daenzer,[⊥] and Tami C. Bond*[,†]

ABSTRACT: This work develops an integrated model approach for estimating emissions from long-haul freight truck and rail transport in the United States between 2010 and 2050. We connect models of macroeconomic activity, freight demand by commodity, transportation networks, and emission technology to represent different pathways of future freight emissions. Emissions of particulate matter (PM), carbon monoxide (CO), nitrogen oxides (NO_x), and total hydrocarbon (THC) decrease by 60%−70% from 2010 to 2030, as older vehicles built to less-stringent emission standards retire. Climate policy, in the form of carbon tax that increases apparent fuel prices, causes a shift from truck to rail, resulting in a 30% reduction in fuel consumption and a 10%−28% reduction in pollutant emissions by 2050, if rail capacity is sufficient. Eliminating high-emitting conditions in the truck fleet affects air pollutants by 20% to 65%; although these estimates are highly uncertain, they indicate the importance of durability in vehicle engines and emission control systems. Future infrastructure investment will be required both to meet transport demand and to enable actions that reduce emissions of air and climate pollutants. By driving the integrated model framework with two macroeconomic scenarios, we show that the effect of carbon tax on air pollution is robust regardless of growth levels.



Adopting Clean Fuels and Technologies on School Buses

Pollution and Health Impacts in Children

Sara D. Adar[1], Jennifer D'Souza[1], Lianne Sheppard[2,3], Joel D. Kaufman[2,4,5], Teal S. Hallstrand[4], Mark E. Davey[6], James R. Sullivan[2], Jordan Jahnke[7], Jane Koenig[2], Timothy V. Larson[2,8], and L. J. Sally Liu[2,6†]

Am J Respir Crit Care Med Vol 191, Iss 12, pp 1413–1421, Jun 15, 2015

Abstract

Rationale: More than 25 million American children breathe polluted air on diesel school buses. Emission reduction policies exist, but the health impacts to individual children have not been evaluated.

Methods: Using a natural experiment, we characterized the exposures and health of 275 school bus riders before, during, and after the adoption of clean technologies and fuels between 2005 and 2009. Air pollution was measured during 597 trips on 188 school buses. Repeated measures of exhaled nitric oxide (F_{ENO}), lung function (FEV_1, FVC), and absenteeism were also collected monthly (1,768 visits). Mixed-effects models longitudinally related the adoption of diesel oxidation catalysts (DOCs), closed crankcase ventilation systems (CCVs), ultralow-sulfur diesel (ULSD), or biodiesel with exposures and health.

Measurements and Main Results: Fine and ultrafine particle concentrations were 10–50% lower on buses using ULSD, DOCs, and/or CCVs. ULSD adoption was also associated with reduced F_{ENO} (−16% [95% confidence interval (CI), −21 to −10%]), greater changes in FVC and FEV_1 (0.02 [95% CI, 0.003 to 0.05] and 0.01 [95% CI, −0.006 to 0.03] L/yr, respectively), and lower absenteeism (−8% [95% CI, −16.0 to −0.7%]), with stronger associations among patients with asthma. DOCs, and to a lesser extent CCVs, also were associated with improved F_{ENO}, FVC growth, and absenteeism, but these findings were primarily restricted to patients with persistent asthma and were often sensitive to control for ULSD. No health benefits were noted for biodiesel. Extrapolating to the U.S. population, changed fuel/technologies likely reduced absenteeism by more than 14 million/yr.

Conclusions: National and local diesel policies appear to have reduced children's exposures and improved health.

Keywords: particulate matter; air pollution; asthma; absenteeism; lung function

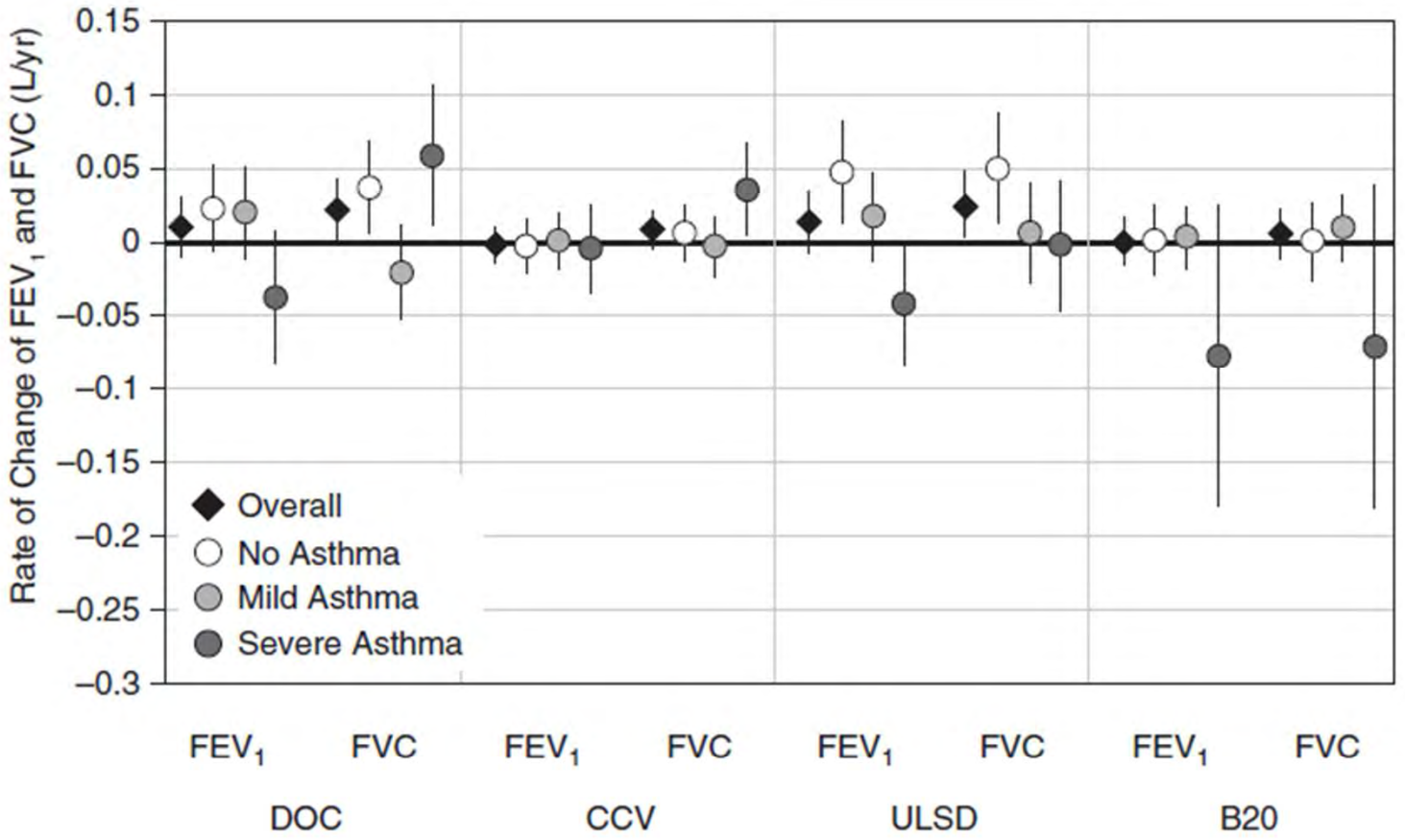


Figure 4. Adjusted associations (percent difference, 95% confidence interval) between rate of change in lung function over time and clean air technologies and fuels among all students and by asthma status. Models were adjusted for age, sex, race/ethnicity, height, weight, asthma status, ambient temperature, relative humidity, fine particulate matter (≤2.5-μm diameter), district flu prevalence, individual report of a cold or flu, within–school year time trend, and random subject effect. B20 = biodiesel; CCV = crankcase ventilation system; DOC = diesel oxidative catalyst; ULSD = ultralow-sulfur diesel.

Adar, AJRCCM 2015

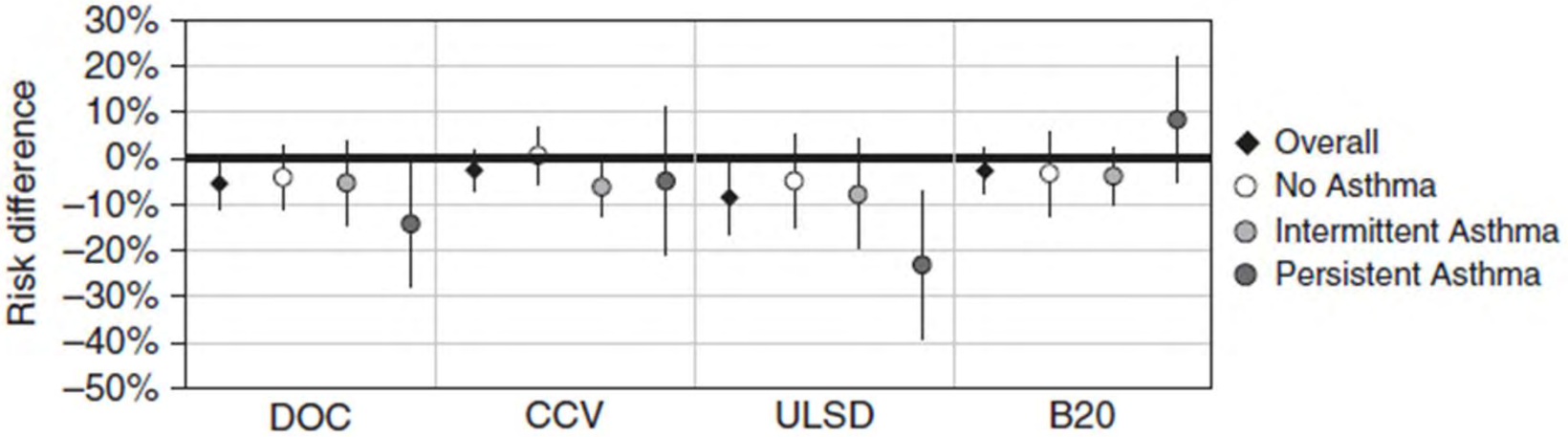


Figure 5. Adjusted associations (risk difference, 95% confidence interval) for any absenteeism in the past month as a function of clean air technologies and fuels among all students and by asthma status. Models were adjusted for age, sex, race/ethnicity, asthma status, ambient temperature, relative humidity, fine particulate matter (≤2.5-μm diameter), district flu prevalence, within–school year time trend, and random subject effect. B20 = biodiesel; CCV = crankcase ventilation system; DOC = diesel oxidative catalyst; ULSD = ultralow-sulfur diesel.

Adar, AJRCCM 2015

The association between greenness and traffic-related air pollution at schools

Payam Dadvand [a,b,c,*], Ioar Rivas [a,b,c,d], Xavier Basagaña [a,b,c], Mar Alvarez-Pedrerol [a,b,c], Jason Su [e], Montserrat De Castro Pascual [a,b,c], Fulvio Amato [d], Michael Jerret [e], Xavier Querol [d], Jordi Sunyer [a,b,c], Mark J. Nieuwenhuijsen [a,b,c]

Science of the Total Environment 523 (2015) 59–63

A B S T R A C T

Greenness has been reported to improve mental and physical health. Reduction in exposure to air pollution has been suggested to underlie the health benefits of greenness; however, the available evidence on the mitigating effect of greenness on air pollution remains limited and inconsistent. We investigated the association between greenness within and surrounding school boundaries and monitored indoor and outdoor levels of traffic-related air pollutants (TRAPs) including NO_2, ultrafine particles, black carbon, and traffic-related $PM_{2.5}$ at 39 schools across Barcelona, Spain, in 2012. TRAP levels at schools were measured twice during two one-week campaigns separated by 6 months. Greenness within and surrounding school boundaries was measured as the average of satellite-derived normalized difference vegetation index (NDVI) within boundaries of school and a 50 m buffer around the school, respectively. Mixed effects models were used to quantify the associations between school greenness and TRAP levels, adjusted for relevant covariates. Higher greenness within and surrounding school boundaries was consistently associated with lower indoor and outdoor TRAP levels. Reduction in indoor TRAP levels was partly mediated by the reduction in outdoor TRAP levels. We also observed some suggestions for stronger associations between school surrounding greenness and outdoor TRAP levels for schools with higher number of trees around them. Our observed reduction of TRAP levels at schools associated with school greenness can be of public importance, considering the burden of health effects of exposure to TRAPs in schoolchildren.

Table 3

Adjusted regression coefficients (RC) and 95% confidence intervals (CI) indicating the change in air pollutant levels associated with one IQR[a] increase in average of NDVI surrounding (50 m buffer) the school boundaries separately for schools with surrounding tree counts lower and higher than median school surrounding tree counts (91 trees).

Outdoor levels	School surrounding greenness (average NDVI)			
	Lower tree count		Higher tree count	
	RC (95% CI)	p	RC (95% CI)	p
NO_2[b]	−2.7 (−7.9, 2.5)	0.303	−5.2 (−10.3, −0.1)	0.047
UFP (LDSA)[c]	−6.7 (−12.1, −1.2)	0.016	−10.0 (−17.4, −2.6)	0.008
Black carbon[c]	−0.17 (−0.39, −0.04)	0.011	−0.35 (−0.60, −0.11)	0.004
Traffic-related $PM_{2.5}$[c]	−0.7 (−1.6, 0.2)	0.108	−2.3 (−4.0, −0.6)	0.009

[a] 0.060 and 0.102 for lower and higher tree content schools respectively.

[b] Adjusted for weekly average of background level of that pollutant, meteorological indicators (temperature, humidity, and precipitation), monitor placement (floor and orientation), and traffic indicators (squared distance to the nearest major road, product of traffic intensity on the nearest road and inverse of distance to the nearest road, and total length of roads (all types) in a 1000 m buffer around the school).

[c] Adjusted for weekly average of background level of that pollutant, meteorological indicators (temperature, humidity, and precipitation), monitor placement (floor and orientation), and traffic indicators (the product of traffic intensity on the nearest road and inverse of distance to the nearest road and total traffic load (all road types) in a 50 m buffer around the school).

NDVI=Normalized Difference Vegetation Index

Dadvand, Sci Total Environ 2015

What to tell patients?

Stop running outdoors?
Ware a mask?
Use air cleaners?
Avoid opening the windows?
Drink more orange juice?
Change your apartment?
Change your genes?

ACC.15
TCT@ACC-iR | Innovation in Intervention

A883
JACC March 17, 2015
Volume 65, Issue 10S

Heart Failure and Cardiomyopathies

RESPIRATORY MASK-FILTER PREVENTS CARDIOVASCULAR EFFECTS ASSOCIATED WITH DIESEL EXHAUST EXPOSURE: A RANDOMIZED, PROSPECTIVE, DOUBLE-BLIND, CONTROLLED, CROSSOVER STUDY IN HEART FAILURE (FILTER-HF TRIAL)

Poster Contributions
Poster Hall B1
Saturday, March 14, 2015, 3:45 p.m.-4:30 p.m.

Session Title: Advances in Heart Failure Therapies: From Diuretics to VADs and Transplant
Abstract Category: 14. Heart Failure and Cardiomyopathies: Clinical
Presentation Number: 1146-213

Authors: *Jefferson L. Vieira, Guilherme V. Guimaraes, Paulo A. de Andre, Paulo H.N Saldiva, Edimar A. Bocchi, Heart Institute (InCor), University of Sao Paulo Medical School, Sao Paulo, Brazil*

Background: Air pollution is linked to heart failure (HF) risk factors and decompensation.

Methods: We tested the effects of a mask-filter in a randomized, controlled, double-blind trial, including 26 HF patients and 15 healthy volunteers exposed in 3 apart sessions to clean air, diluted diesel exhaust (DE at 300 µg/m3) or mask-filtered diluted DE. The primary endpoint was endothelial function by reactive hyperemia index (RHI), and the secondary endpoints were submaximal 6-minute cardiopulmonary test (6mwt) and serum biomarkers.

Results: All HF patients were under standard therapy, with LVEF 30 ± 6% and in I-II functional class (85%). Mask-filter intervention reduced DE from 325 to 25µg/m3. Healthy volunteers had higher RHI. In HF group, DE-exposure decreased RHI from 2.17 [1.8-2.5] to 1.72 [1.5-2.2] (p=0.002), and arterial stiffness from 14% to 10% (p=0.007). Mask-filter intervention prevented endothelial dysfunction (RHI 1.72 [1.5-2.2] vs 2.06 [1.5-2.6]; p=0.019). In all subjects, the 6mwt distance decreased during DE-exposure (from 243.3±13m to 220.8±14m in HF group; p=0.030, and from 292.3±19m to 252.7±20m in healthy volunteers; p=0.032). HF patients walked even less with the filter (209.2±15.1m; p=0.025), as they perceived a greater effort of breathing. This effect might be attributed to a increase on inspired CO2 (p=0.031). Oxygen-uptake declined in healthy volunteers during DE-exposure (p=0.009) but also during mask-filter intervention. HF patients had higher troponin, C-reactive protein and BNP (p<0.05), but only BNP increased during DE-exposure (from 47 [17-118] to 66 [26-155] pg/ml; p=0.004). This effect was significantly prevented by the mask-filter intervention (from 66 [26-155] to 44 [20-110] pg/ml; p=0.015).

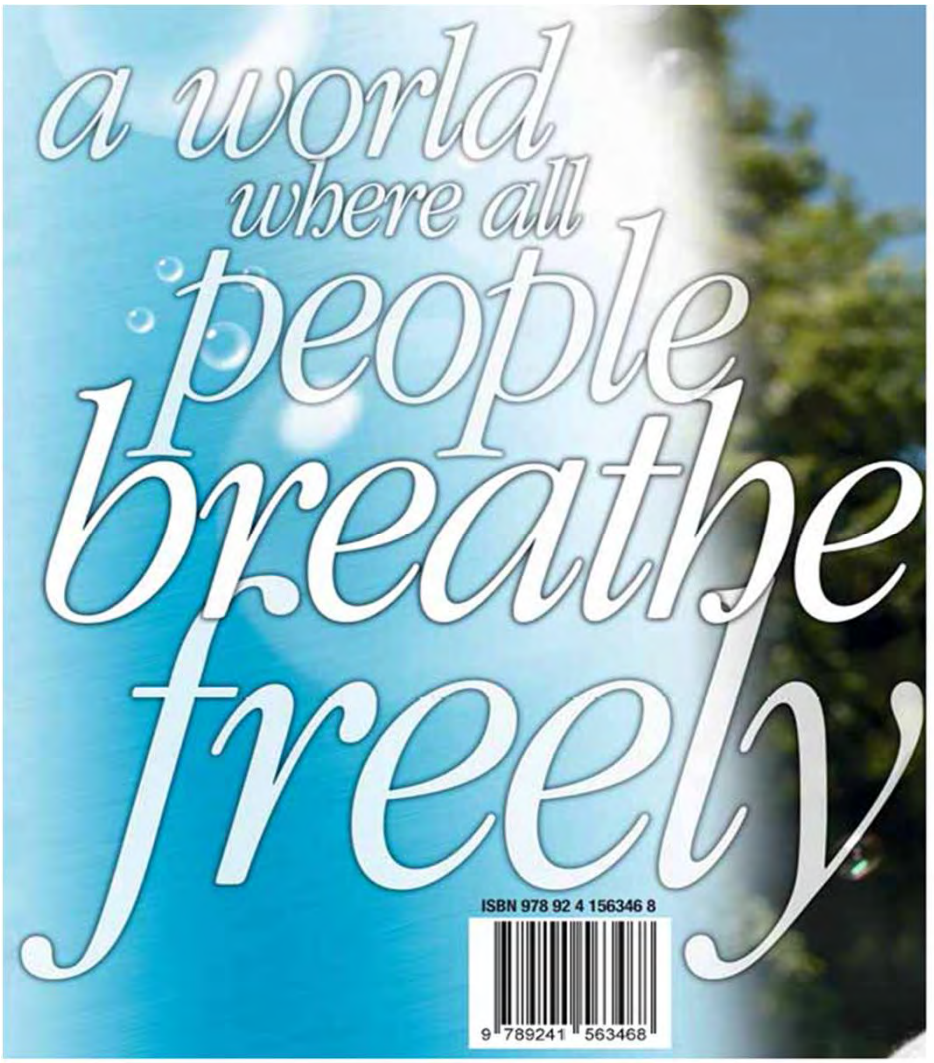
Conclusion: To our knowledge, this is the first trial to support the hypothesis that endothelial dysfunction and BNP increment related to DE-exposure, both predictors of HF morbidity, could be favorably influenced by a mask-filter intervention. Given these potential benefits, the widespread use of mask-filters in HF subjects exposed to traffic-derived air pollution may have substantial public health impact and reduce HF burden.

(ClinicalTrials.gov NCT01960920)

What to tell patients?

Stop running outdoors?
Ware a mask?
Use air cleaners?
Avoid opening the windows?
Drink more orange juice?
Change your apartment?
Change your genes?

isabella.annesi-maesano@inserm.fr



Merci

Mortalité liée à la pollution atmosphérique

WHO 2014
~ 7 Millions de morts
Mortalité due à la pollution de l'extérieur:
40% – maladies ischémiques;
40% – AVC;
11% – BPCO;
6% – Cancer du poumon;
3% – Infections respiratoires infantiles;
Mortalité due à la pollution de l'intérieur:
34% – AVC;
26% – maladies ischémiques;
22% – BPCO;
12% – Infections respiratoires infantiles;
6% – Cancer du poumon.

→ Importante morbidité

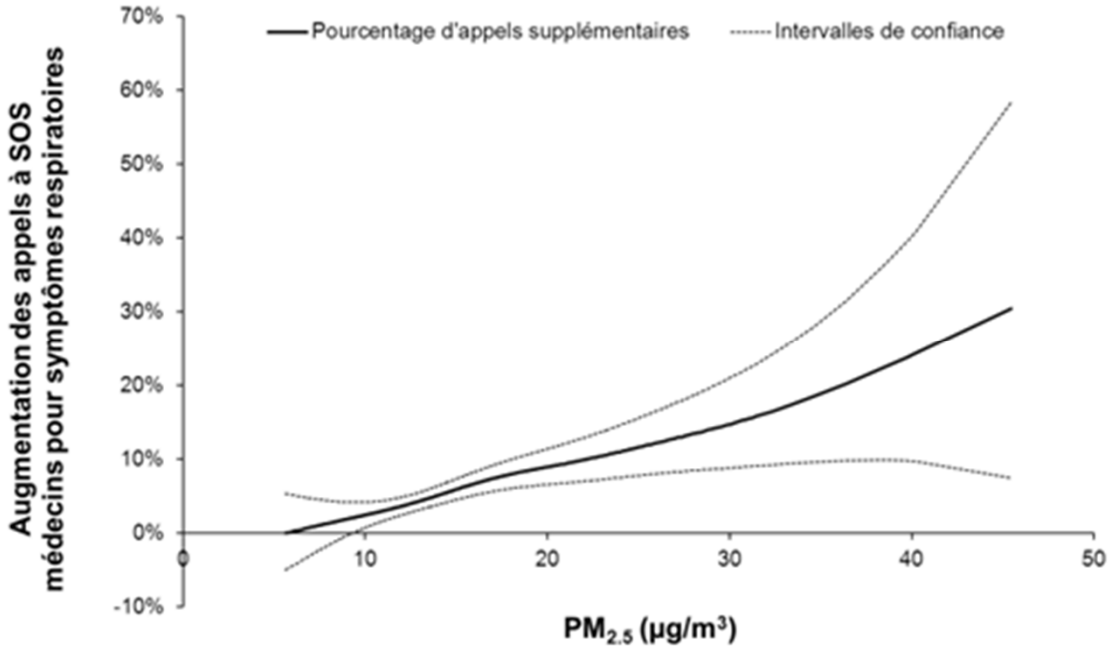
Plus la gravité des effets diminue plus le nombre de gens touchés augmente

Les PM de taille inférieure à 2,5 micromètres ($PM_{2,5}$) sont les plus dangereuses

I. Annesi-Maesano & W. Dab, Le livre de l'Interne. Pneumologie. Lavoisier Editeur 2013

Illustration de l'absence de seuil des effets sanitaires de la pollution atmosphérique : quel que soit le niveau d'exposition (10, 20, 40 µg/m³), on observe une augmentation de 0,6% des appels à SOS médecins pour symptômes respiratoires lorsque les concentrations ambiantes de $PM_{2,5}$ augmentent d'1 µg/m³

Source : ORS Île-de-France



Article original

Évaluation à minima du coût de la pollution atmosphérique pour le système de soin français

Christophe Rafenberg[1,2]
Gilles Dissaut[3,4]
Isabella Annesi-Maesano[1,2]
[1] INSERM

Résumé. Les évaluations réalisées en matière de coûts de la pollution de l'air se fondent le plus souvent sur une approche socio-économique et sur les coûts intangibles (valeur de la vie ou de la souffrance par exemple). Ce type d'évaluations est un sujet de controverses tant il est délicat de fixer ces valeurs en dehors d'un sujet de recherche

ERS 2015

Article original

Évaluation à minima du coût de la pollution atmosphérique pour le système de soin français

Christophe Rafenberg[1,2]
Gilles Dissaut[3,4]
Isabella Annesi-Maesano[1,2]
[1] INSERM

Résumé. Les évaluations réalisées en matière de coûts de la pollution de l'air se fondent le plus souvent sur une approche socio-économique et sur les coûts intangibles (valeur de la vie ou de la souffrance par exemple). Ce type d'évaluations est un sujet de controverses tant il est délicat de fixer ces valeurs en dehors d'un sujet de recherche

ERS 2015

Nature de la pathologie		Nombre annuel de nouveaux cas attribuables à l'environnement		Coût annuel pour le système de soin des nouveaux cas attribuables à l'environnement	
		Valeur basse	Valeur haute	Valeur basse	Valeur haute
Broncho-pneumopathie chronique obstructive (BPCO)		26 800	40 200	123,7 millions €	186 m€
Bronchite chronique (BC)		134 000 cas		113,4 millions €	
Bronchite aiguë (BA)		Enfants 450 218/Adultes 500 000		170,4 millions €	
Asthme (As)		400 000 cas	1 400 000 cas	315 millions €	1 102,4 millions €
Cancers	Voies respiratoires basses	1 608	4 020	87 millions €	249 millions €
	Voies respiratoires hautes	76	380		
Hospitalisations	Causes respiratoires	13 796		155 millions €	
	Causes cardiovasculaires	19 761			
TOTAL				1 milliard €	2 milliards €

Article original

Évaluation à minima du coût de la pollution atmosphérique pour le système de soin français

Christophe Rafenberg[1,2]
Gilles Dissaut[3,4]
Isabella Annesi-Maesano[1,2]
[1] INSERM

Résumé. Les évaluations réalisées en matière de coûts de la pollution de l'air se fondent le plus souvent sur une approche socio-économique et sur les coûts intangibles (valeur de la vie ou de la souffrance par exemple). Ce type d'évaluations est un sujet de controverses tant il est délicat de fixer ces valeurs en dehors d'un sujet de recherche

Table 4. Breakdown of asthma cases attributable to the environment, by severity and by costs.

Nombre de cas d'asthmes attribuables à l'environnement	Très léger à léger 39 %	Modéré 43 %	Sévère 18 %	Total
Valeur basse (10 %)	156 000 cas	172 000 cas	72 000 cas	400 000 cas
Valeur haute (35 %)	546 000 cas	602 000 cas	252 000 cas	1 400 000 cas
Coût de traitement	443 €	764 €	1536 €	
Valeur basse	69 ,1 millions €	131,4 millions €	110,6 millions €	311, 1 millions €
Valeur haute	242 millions €	460 millions €	387 millions €	1089 millions €
Passages aux urgences imputables à l'environnement	191 € par passage			
	Valeur basse	20 000		3,8 millions €
	Valeur haute	70 000		13,4 millions €
Valeur basse du coût total de l'asthme	315 millions €			
Valeur haute du coût total de l'asthme	1102,4 millions €			