

# Des polluants atmosphériques qui altèrent notre santé

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Journée scientifique Météo et Climat "Pollution  
atmosphérique et impacts sanitaires"  
24 novembre 2014



# Sommaire

- Quelques généralités
- Effets de la pollution atmosphérique sur la santé
- Conséquences en termes de santé publique
- Episodes de pollution
- Perspectives



# Quelques généralités



# Polluants atmosphériques et santé : une relation complexe

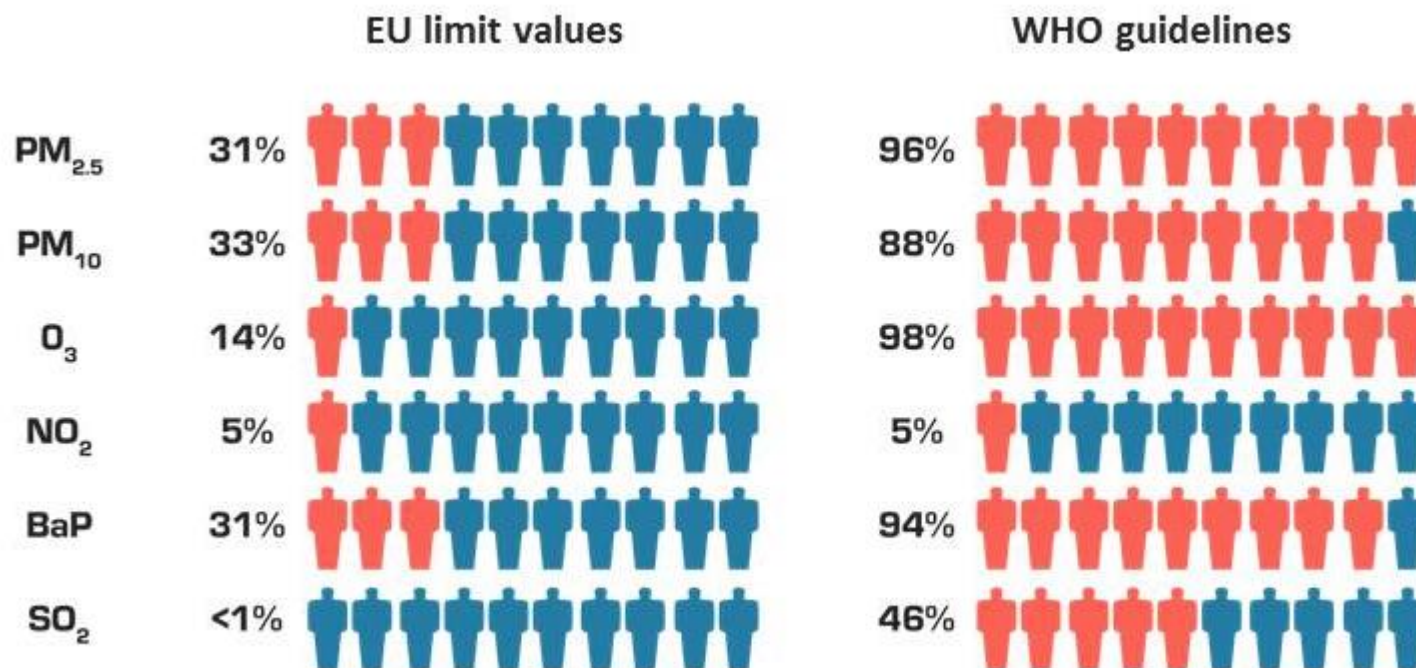
Du fait de :

- grand nombre de polluants dans l'air qui peuvent en outre réagir entre eux pour former des polluants secondaires
- hétérogénéité de l'exposition dans le temps et dans l'espace, et hétérogénéité de la sensibilité pour l'individu
- multifactorialité des maladies susceptibles d'être liées à la pollution atmosphérique
- Faiblesse des risques individuels mais grand impact à l'échelle de la population générale (toute la population est exposée)

# Population urbaine européenne exposée en 2011 à des niveaux de pollution considérés nuisibles pour la santé

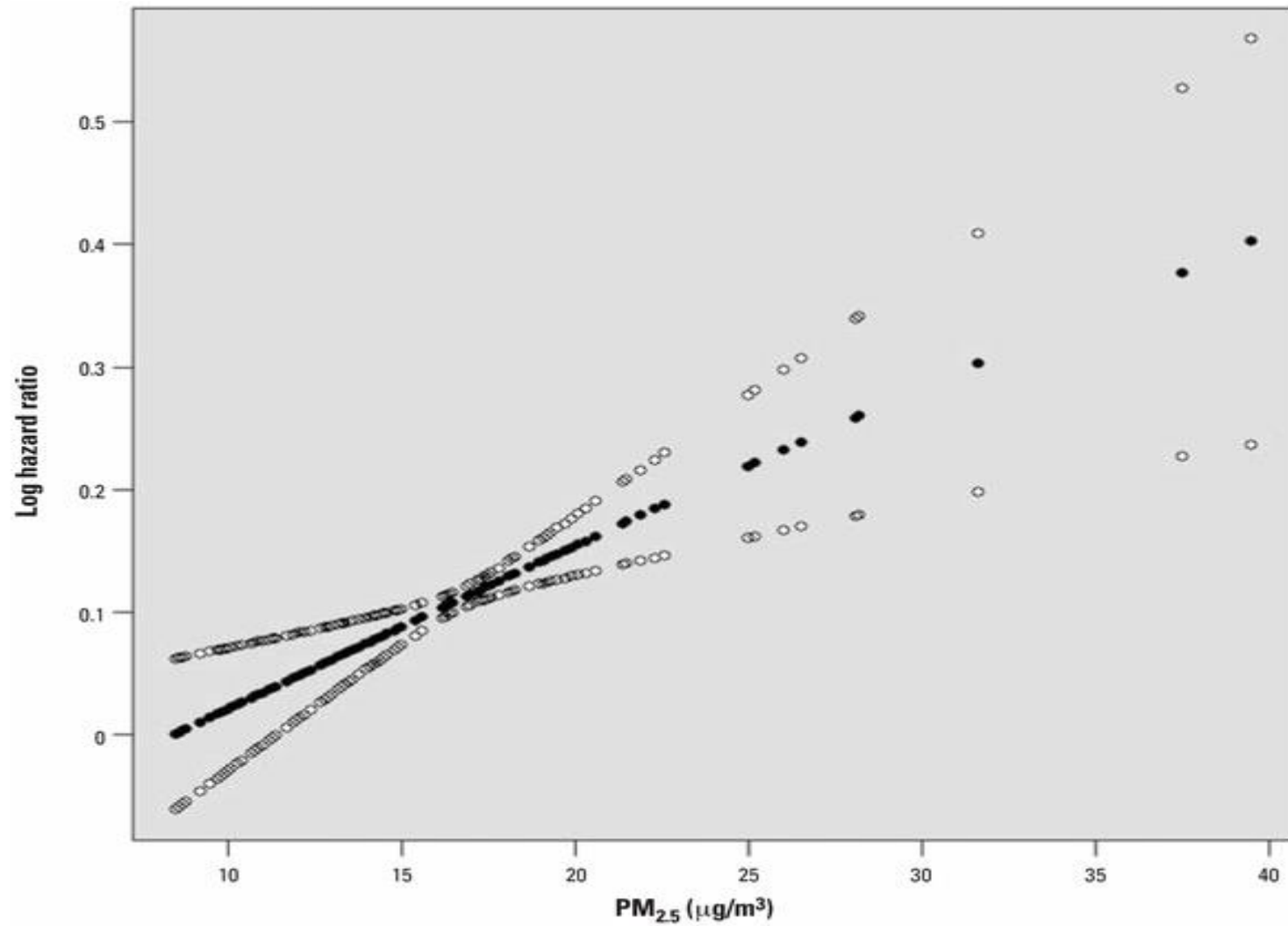
Jusqu'à 1/3 des citoyens européens sont exposés à des niveaux de pollution dépassant les normes de qualité de l'air de l'UE

Et environ 90% des citoyens européens sont exposés à des niveaux de pollution jugés nuisibles pour la santé par les valeurs guides de l'OMS



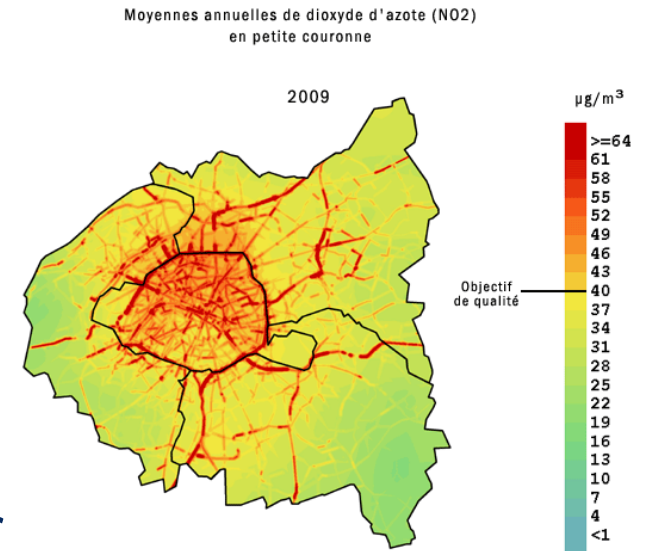
Source : EEA 2013

# Relation linéaire sans seuil



# Des inégalités d'exposition

- Forte hétérogénéité intra-urbaine de l'exposition
- Contribution de la pollution atmosphérique aux inégalités sociales de santé
  - inégalités sociales de l'exposition à la pollution
  - inégalités sociales de vulnérabilité aux effets sur la santé de la pollution





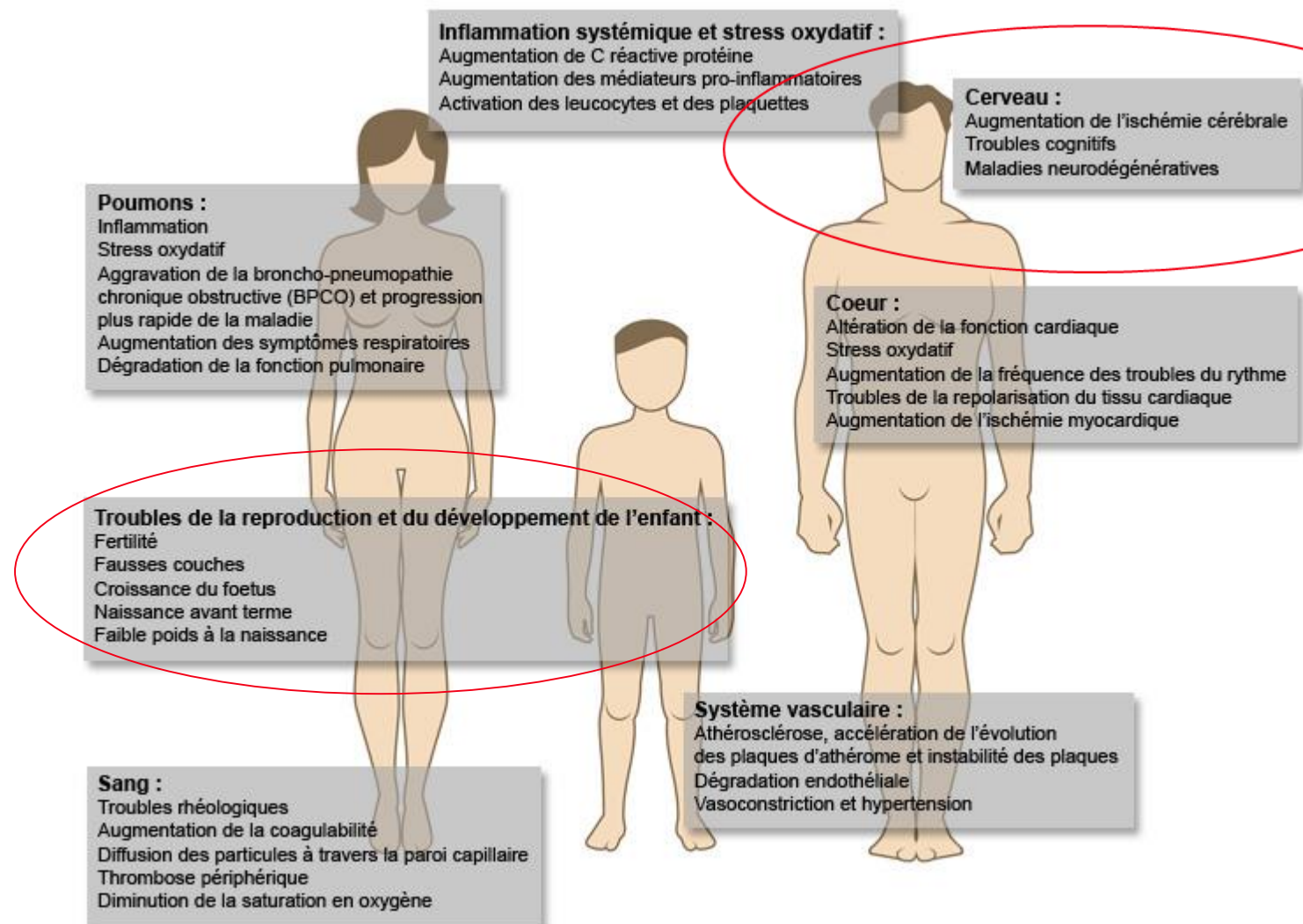
# Des populations plus sensibles ou vulnérables, et plus exposées que d'autres

- Femmes enceintes
- Nourrissons et enfants de moins de 5 ans dont les poumons ne sont pas complètement formés
- Personnes âgées, plus sensibles en raison du vieillissement et de la présence de pathologies chroniques
- Personnes souffrant de pathologies chroniques (maladies respiratoires chroniques, allergies, asthme, maladies cardiovasculaires, diabète)
- Fumeurs, dont l'appareil respiratoire est déjà irrité par le tabac
- Personnes pratiquant une activité sportive en extérieur soumises à une exposition plus importante (augmentation de la ventilation)



Quels effets sur la santé ?

# Effets sur la santé de la pollution atmosphérique

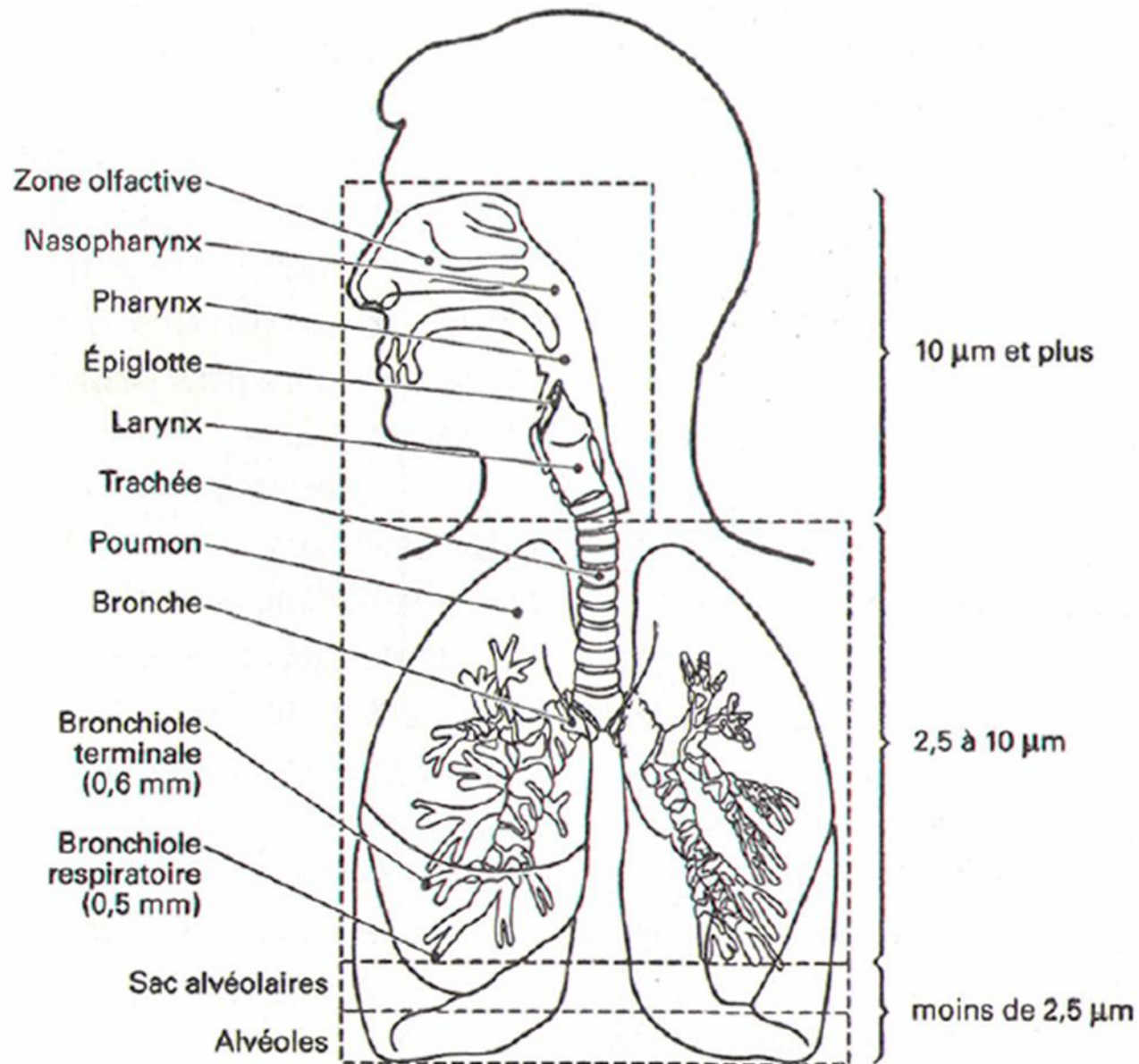


Source : Programme de surveillance air et santé, InVS, 2014

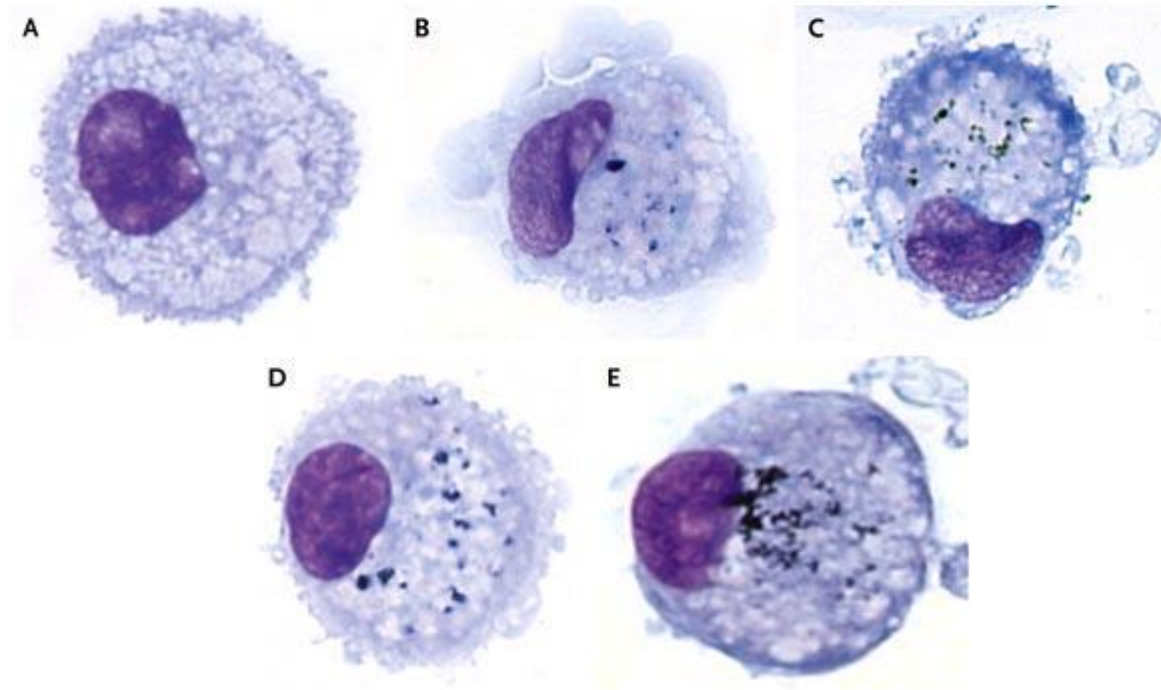


# Effets sur l'appareil respiratoire

# Pénétration des PM selon leur taille

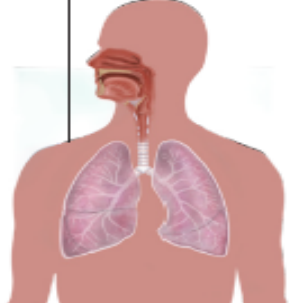


# Présence de particules de carbone dans les macrophages alvéolaires chez les enfants (Kulkarni et al. NEJM 2006)



**Figure 1.** Representative Images of Carbon in Airway Macrophages from Healthy Children. Panel A shows a macrophage with no carbon. Increasing levels of carbon are shown in Panels B through E.

# Pollution atmosphérique et développement de l'appareil respiratoire

| Stage:<br>Age:          | Newborn<br>0–2 mos                                                                | Infant/Toddler<br>2 mos–2 yrs             | Young Child<br>2–6 yrs                                                              | School-Age Child<br>6–12 yrs        | Adolescent<br>12–18 yrs                                                             |
|-------------------------|-----------------------------------------------------------------------------------|-------------------------------------------|-------------------------------------------------------------------------------------|-------------------------------------|-------------------------------------------------------------------------------------|
| Lung<br>development:    |  |                                           |  |                                     |  |
|                         | Alveolar development                                                              |                                           |                                                                                     |                                     |                                                                                     |
|                         | High respiratory rate                                                             |                                           |                                                                                     |                                     |                                                                                     |
| Air pollution<br>risks: |                                                                                   |                                           | Increasing lung volume                                                              |                                     |                                                                                     |
|                         | Respiratory death                                                                 |                                           |                                                                                     |                                     |                                                                                     |
|                         |                                                                                   |                                           | Chronic cough and bronchitis                                                        |                                     |                                                                                     |
|                         |                                                                                   |                                           | Reduced lung function                                                               |                                     |                                                                                     |
|                         |                                                                                   |                                           | Wheezing and asthma attacks                                                         |                                     |                                                                                     |
|                         |                                                                                   | Respiratory<br>symptoms<br>and illnesses* |                                                                                     | Respiratory-related school absences |                                                                                     |

\*Air pollution exposure has also been more recently linked to respiratory symptoms and illnesses in early life including cough, bronchitis, wheeze and ear infections

Figure 2. Air pollution effects on the developing respiratory system.

*Air pollution impacts on infants and children, B. Beate and M. Wilhelm,  
J. Dowdalls (medical illustrations), UCLA, 2008*

# Liens entre pollution automobile et incidence d'asthme chez l'enfant

Research | Children's Health

## Traffic-Related Air Pollution and Asthma Onset in Children: A Prospective Cohort Study with Individual Exposure Measurement

Michael Jerrett,<sup>1</sup> Ketan Shankardass,<sup>2</sup> Kiros Berhane,<sup>2</sup> W. James Gauderman,<sup>2</sup> Nino Künzli,<sup>3</sup> Edward Avol,<sup>2</sup> Frank Gilliland,<sup>2</sup> Fred Lurmann,<sup>4</sup> Jassy N. Molitor,<sup>5</sup> John T. Molitor,<sup>5</sup> Duncan C. Thomas,<sup>2</sup> John Peters,<sup>2</sup> and Rob McConnell<sup>2</sup>

<sup>1</sup>School of Public Health, Division of Environmental Health Science, University of California, Berkeley, California, USA; <sup>2</sup>Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California, USA; <sup>3</sup>Center for Research in Environmental Epidemiology (CREAL), Institut Municipal d'Investigació Mèdica (IMIM), Barcelona, Spain; <sup>4</sup>Sonoma Technology Inc., Petaluma, California, USA; <sup>5</sup>Department of Epidemiology and Public Health, School of Medicine, Imperial College, London, UK

*L'incidence de l'asthme chez l'enfant positivement associée à la pollution d'origine automobile (RR = 1,29 IC95% : 1,07-1,56)*

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## Childhood Incident Asthma and Traffic-Related Air Pollution and School

Rob McConnell,<sup>1</sup> Talat Islam,<sup>1</sup> Ketan Shankardass,<sup>2</sup> Michael Jerrett,<sup>3</sup> Fred Lurmann,<sup>4</sup> Frank Gilliland,<sup>1</sup> Jim Gauderman,<sup>1</sup> Ed Avol,<sup>1</sup> Nino Künzli,<sup>5</sup> Ling Yao,<sup>6</sup> John Peters,<sup>1</sup> and Kiros Berhane<sup>1</sup>

<sup>1</sup>University of Southern California, Los Angeles, California, USA; <sup>2</sup>St. Michael's Hospital, Toronto, Ontario, Canada; <sup>3</sup>University of California, Berkeley, California, USA; <sup>4</sup>Sonoma Technology, Inc, Petaluma, California, USA; <sup>5</sup>Swiss Tropical and Public Health Institute, Basel and University of Basel, Switzerland; <sup>6</sup>United Health Group, City of Hope Hospital Medical Center, Los Angeles, California, USA

**BACKGROUND:** Traffic-related air pollution has been associated with adverse cardiorespiratory effects, including increased asthma prevalence. However, there has been little study of effects of traffic exposure at school on new-onset asthma.

**OBJECTIVES:** We evaluated the relationship of new-onset asthma with traffic-related pollution near homes and schools.

**METHODS:** Parent-reported incident asthma during 3 years of follow-up and wheezing were assessed traffic-related distance from home (NO<sub>2</sub>), and particulate matter (PM<sub>10</sub> and PM<sub>2.5</sub>) at central site pollutants.

**RESULTS:** Asthma risk increased with distance from home (HR 1.51; 95% CI 1.06–1.98). Ambient increased risk (HR 2.0; 95% CI 1.06–1.98). At school, there were no associations.

**CONCLUSIONS:** Traffic-related air pollution is associated with development of asthma.

**KEY WORDS:** air pollution, asthma, children, traffic-related air pollution.

**Table 4.** Association of new-onset asthma with community central site pollutant measurements.

| Pollutant         | HR <sup>a</sup> (95% CI) |
|-------------------|--------------------------|
| NO <sub>2</sub>   | 2.17 (1.18–4.00)         |
| PM <sub>10</sub>  | 1.35 (0.64–2.85)         |
| PM <sub>2.5</sub> | 1.66 (0.91–3.05)         |
| O <sub>3</sub>    | 0.76 (0.38–1.54)         |

<sup>a</sup>HR (95% CI) across the range of exposure in the 13 communities (23.6 ppb for NO<sub>2</sub>, 43.9 µg/m<sup>3</sup> PM<sub>10</sub>, 17.4 µg/m<sup>3</sup> PM<sub>2.5</sub>, and 30.3 ppb for 1000–1800 hours O<sub>3</sub>), adjusted for race/ethnicity and for baseline hazards strata of age at study entry and sex with random effects for community and school.

**Table 5.** Mutually adjusted associations of new-onset asthma with community central site pollutant measurements and nonfreeway TRP at home and school.<sup>a</sup>

| Central site pollutant | HR <sup>b</sup> (95% CI) for ambient pollutant, adjusted for TRP at home and school | HR <sup>b</sup> (95% CI) for home TRP, adjusted for school TRP and ambient pollutant | HR <sup>b</sup> (95% CI) for school TRP, adjusted for home TRP and ambient pollutant |
|------------------------|-------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------|
| NO <sub>2</sub>        | 1.37 (0.69–2.71)                                                                    | 1.46 (1.16–1.84)*                                                                    | 1.45 (1.03–2.06)                                                                     |
| PM <sub>10</sub>       | 1.40 (0.62–3.17)                                                                    | 1.46 (1.16–1.85)*                                                                    | 1.53 (1.10–2.12)*                                                                    |
| PM <sub>2.5</sub>      | 1.30 (0.66–2.56)                                                                    | 1.48 (1.19–1.85)*                                                                    | 1.49 (1.07–2.08)                                                                     |
| O <sub>3</sub>         | 1.01 (0.49–2.11)                                                                    | 1.58 (1.20–1.86)*                                                                    | 1.54 (1.10–2.14)*                                                                    |

<sup>a</sup>Mutually adjusted across each row (i.e., effect of each community pollutant was examined separately in a model including both home and school TRP). <sup>b</sup>HR (95% CI) for central-site pollutants scaled across the range of exposure in the 13 communities (23.6 ppb for NO<sub>2</sub>, 43.9 µg/m<sup>3</sup> PM<sub>10</sub>, 17.4 µg/m<sup>3</sup> PM<sub>2.5</sub>, and 30.3 ppb for 1000–1800 hours O<sub>3</sub>); household nonfreeway TRP was deviated from school, scaled to the IQR for home exposure (8 ppb from Table 2). \**p* < 0.01.

# Liens entre pollution automobile et incidence d'asthme chez l'adulte

Modig et al. – RHINE Study, ERJ 2009

**TABLE 4** Associations between exposures to nitrogen dioxide (NO<sub>2</sub>), distance to nearest major road and the onset and incidence of asthma

|                                                   | Onset asthma |           | Incident asthma |           |
|---------------------------------------------------|--------------|-----------|-----------------|-----------|
|                                                   | OR           | 95% CI    | OR              | 95% CI    |
| <b>Crude<sup>#</sup></b>                          |              |           |                 |           |
| NO <sub>2</sub> per 10 µg·m <sup>-3</sup>         | 1.39         | 1.05–1.84 | 1.36            | 0.92–2.02 |
| Distance to nearest major road <50 m <sup>†</sup> | 2.81         | 1.66–4.76 | 3.55            | 1.78–7.01 |
| <b>Fully adjusted<sup>‡</sup></b>                 |              |           |                 |           |
| NO <sub>2</sub> per 10 µg·m <sup>-3</sup>         | 1.46         | 1.07–1.99 | 1.54            | 1.00–2.36 |
| Distance to nearest major road <50 m <sup>†</sup> | 2.92         | 1.70–4.98 | 3.88            | 1.93–7.82 |

OR: odds ratio; CI: confidence interval. <sup>#</sup>: includes the same population as in the fully adjusted model; <sup>†</sup>: yes versus no, major road is defined as ≥8,000 vehicles per 24 h; <sup>‡</sup>: including city, sex, body mass index, age, smoking history, water damage or mould in the home at any time during the last 8 yrs.

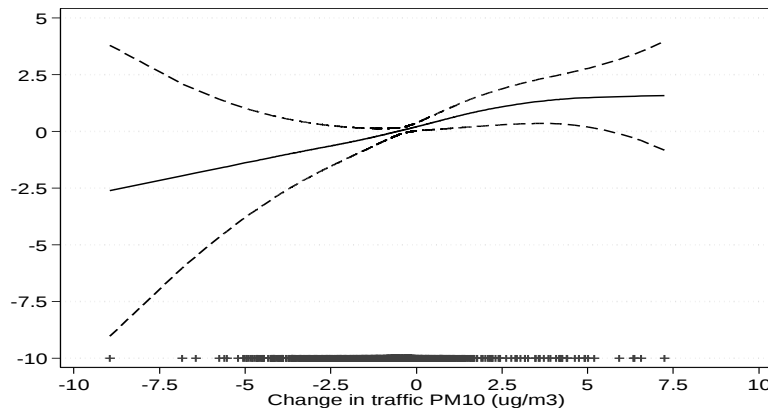


Figure 2: Association between change in traffic-related PM<sub>10</sub> and adult-onset asthma (log-hazard with 95%-confidence interval) among SAPALDIA never-smokers. (Generalized additive model, adjusted for age, sex, atopy at baseline, BMI at baseline, bronchial hyperreactivity at baseline, maternal allergies.) The symbols (+) on the X-axis indicate observations.

*Deux études, en Suède et en Suisse, suggèrent que les niveaux élevés de pollution automobile à proximité du lieu de domicile augmentent le risque d'incidence d'asthme chez l'adulte*

Jacquemin et al. ERHS, Epidemiology 2009

## Home outdoor NO<sub>2</sub> and new onset of self-reported asthma in adults.

Jacquemin B, Sunyer J, Forsberg B, Aquilera I, Briggs D, Garcia-Esteban R, Götschi T, Heinrich J, Järnholm B, Jarvis D, Vienneau D, Künzli N.

Centre for Research in Environmental Epidemiology, Barcelona, Spain. benedicte.jacquemin@inserm.fr [corrected]

### Erratum in

Epidemiology. 2009 Jul;20(4):630.

### Abstract

**BACKGROUND:** Few studies have investigated new onset of asthma in adults in relation to air pollution. The aim of this study is to investigate the association between modeled background levels of traffic-related air pollution at the subjects' home addresses and self-reported asthma incidence in European adult population.

**METHODS:** Adults from the European Respiratory Health Survey were included (n = 4185 from 17 cities). Subjects' home addresses were geocoded and linked to outdoor nitrogen dioxide (NO<sub>2</sub>) estimates, as a marker of local traffic-related pollution. We obtained this information from the 1-km background NO<sub>2</sub> surface modeled in APMoSPHERE (Air Pollution Modelling for Support to Policy on Health and Environmental Risk in Europe). Asthma incidence was defined as reporting asthma in the follow-up (1999 to 2001) but not in the baseline (1991 to 1993).

**RESULTS:** A positive association was found between NO<sub>2</sub> and asthma incidence (odds ratio 1.43; 95% confidence interval = 1.02 to 2.01) per 10 microg/m. Results were homogeneous among centers (P value for heterogeneity = 0.59).

**CONCLUSIONS:** We found an association between a marker of traffic-related air pollution and asthma incidence in European adults.

*Une association positive entre NO<sub>2</sub> et incidence d'asthme chez l'adulte (OR 1,43; IC 95% 1.2 à 2.1 par 10 µg/m<sup>3</sup>)*

Kuenzli et al. SAPALDIA Study, Thorax 2009

## La pollution atmosphérique une des principales causes de décès par cancer,

Un groupe de travail du Centre International de Recherche sur le Cancer (CIRC) vient de classer les particules fines diesel carcinogènes certains pour l'homme (Groupe 1). L'exposition à ces particules est associée à une augmentation du risque de cancer du **poumon**. En 1988, le CIRC avait déjà classé ces particules comme carcinogènes probable (Groupe 2A) pour l'homme.

(13/06/2012)

Lyon/Genève, 17 octobre 2013 – Agence spécialisée sur le cancer international de Recherche sur le Cancer (CIRC). a annoncé que L'étude du CIRC porte sur une cohorte de plus de 12 000 personnes. Elle montre que le risque de cancer du poumon est environ trois fois plus important chez les sujets les plus exposés. L'étude a également mis en évidence des éléments plus

Après avoir consulté des experts médicaux, les experts ont conclu que la pollution atmosphérique est une cause importante de décès par cancer.

## Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE)

➔ Lect additionnel de ces données dans le monde

aux cas de cancer du

Ole Raaschou-Nielsen, Zorana J Andersen, Rob Beelen, Evangelia Samoli, Massimo Stafoggia, Gudrun Weinmayr, Barbara Hoffmann, Paul Fischer, Mark J Nieuwenhuijsen, Bert Brunekreef, Wei W Xu, Klea Katsouyanni, Konstantina Dimakopoulou, Johan Sommar, Bertil Forsberg, Lars Modig, Anna Oudin, Bente Oftedal, Per E Schwarze, Per Nafstad, Ulf De Faire, Nancy L Pedersen, Claes-Göran Östenson, Laura Fratiglioni, Johanna Penell, Michal Korek, Göran Pershagen, Kirsten T Eriksen, Mette Sørensen, Anne Tjønneland, Thomas Ellermann, Marloes Eeftens, Petra H Peeters, Kees Meliefste, Meng Wang, Bas Bueno-de-Mesquita, Timothy J Key, Kees de Hoogh, Hans Concin, Gabriele Nagel, Alice Vilier, Sara Grioni, Vittorio Krogh, Ming-Yi Tsai, Fulvio Ricceri, Carlotta Sacerdote, Claudia Galassi, Enrica Migliore, Andrea Ranzi, Giulia Cesaroni, Chiara Badaloni, Francesco Forastiere, Ibon Tamayo, Pilar Amiano, Miren Dorronsoro, Antonia Trichopoulou, Christina Bamia, Paolo Vineis\*, Gerard Hoek\*

### Summary

**Background** Ambient air pollution is suspected to cause lung cancer. We aimed to assess the association between long-term exposure to ambient air pollution and lung cancer incidence in European populations.

**Methods** This prospective analysis of data obtained by the European Study of Cohorts for Air Pollution Effects used data from 17 cohort studies based in nine European countries. Baseline addresses were geocoded and we assessed air pollution by land-use regression models for particulate matter (PM) with diameter of less than 10 µm (PM<sub>10</sub>), less than 2.5 µm (PM<sub>2.5</sub>), and between 2.5 and 10 µm (PM<sub>coarse</sub>), soot (PM<sub>2.5-salt</sub>), nitrogen oxides, and two traffic indicators. We used Cox regression models with adjustment for potential confounders for cohort-specific analyses and random effects models for meta-analyses.

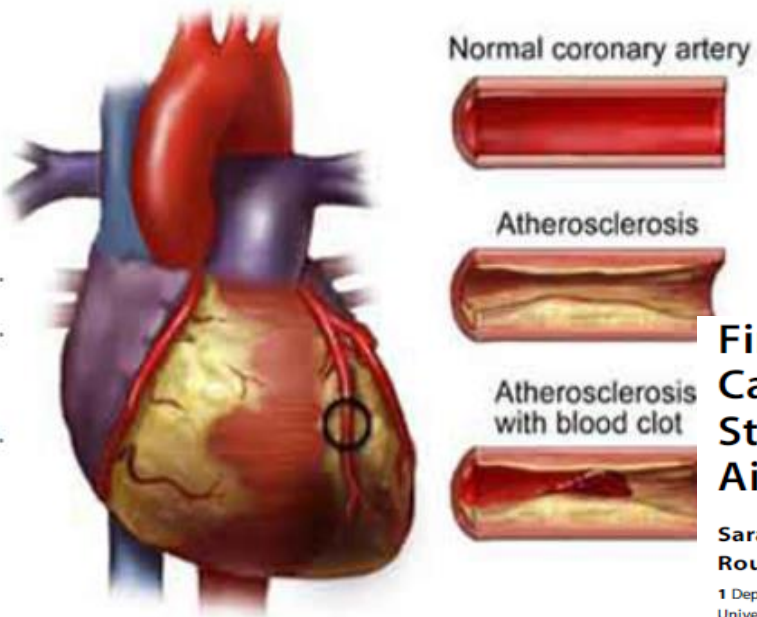
**Findings** The 312944 cohort members contributed 4013131 person-years at risk. During follow-up (mean 12.8 years), 2095 incident lung cancer cases were diagnosed. The meta-analyses showed a statistically significant association between risk for lung cancer and PM<sub>10</sub> (hazard ratio [HR] 1.22 [95% CI 1.03–1.45] per 10 µg/m<sup>3</sup>). For PM<sub>2.5</sub> the HR was 1.18 (0.96–1.46) per 5 µg/m<sup>3</sup>. The same increments of PM<sub>10</sub> and PM<sub>2.5</sub> were associated with HRs for adenocarcinomas of the lung of 1.51 (1.10–2.08) and 1.55 (1.05–2.29), respectively. An increase in road traffic of 4000 vehicle-km per day within

**Lancet Oncol 2013**  
Published Online  
July 10, 2013  
[http://dx.doi.org/10.1016/S1473-0161\(13\)60000-0](http://dx.doi.org/10.1016/S1473-0161(13)60000-0)  
See [Comment](#) page  
\*Joint last author  
See Online for related multimedia content  
Danish Cancer Society, Copenhagen  
(O Raaschou-Nielsen, Z J Andersen PhD, K T Eriksen PhD, N A Tjønneland DM, Epidemiology and Public Health Department of P



# Effets sur le système cardiovasculaire

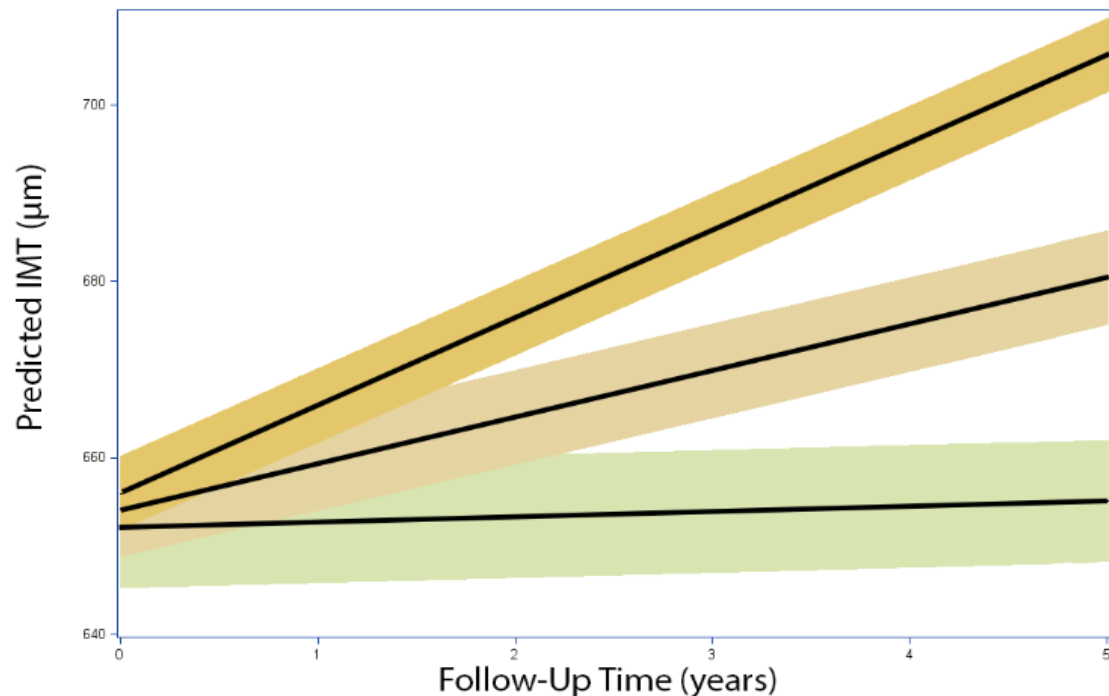
# Pollution atmosphérique et maladies coronariennes



## Fine Particulate Air Pollution and the Progression of Carotid Intima-Medial Thickness: A Prospective Cohort Study from the Multi-Ethnic Study of Atherosclerosis and Air Pollution

Sara D. Adar<sup>1,2\*</sup>, Lianne Sheppard<sup>2,3</sup>, Sverre Vedal<sup>2</sup>, Joseph F. Polak<sup>4</sup>, Paul D. Sampson<sup>5</sup>, Ana V. Diez Roux<sup>1</sup>, Matthew Budoff<sup>6,7</sup>, David R. Jacobs, Jr.<sup>8</sup>, R. Graham Barr<sup>9</sup>, Karol Watson<sup>7</sup>, Joel D. Kaufman<sup>2,10</sup>

<sup>1</sup> Department of Epidemiology, University of Michigan, Ann Arbor, Michigan, United States of America, <sup>2</sup> Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, Washington, United States of America, <sup>3</sup> Department of Biostatistics, University of Washington, Seattle, Washington, United States of America, <sup>4</sup> Department of Radiology, Tufts Medical Center, Boston, Massachusetts, United States of America, <sup>5</sup> Department of Statistics, University of Washington, Seattle, Washington, United States of America, <sup>6</sup> Los Angeles Biomedical Research Institute, Los Angeles, California, United States of America, <sup>7</sup> Division of Cardiology, University of Washington, Seattle, Washington, United States of America, <sup>8</sup> Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, Minnesota, United States of America, <sup>9</sup> Division of Cardiology, Columbia University Medical Center, New York, New York, United States of America, <sup>10</sup> Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, Washington, United States of America



7 µg/m<sup>3</sup>

5 µg/m<sup>3</sup>

3 µg/m<sup>3</sup>

been linked to cardiovascular disease, possibly via accelerated progression of the intima-medial thickness (IMT) of the common carotid artery. Higher long-term PM<sub>2.5</sub> concentrations in participants from the Multi-Ethnic Study of Atherosclerosis (MESA) were associated with greater IMT progression.

We enrolled 6,814 participants at the baseline exam (2000–2002), with follow-up examinations between 2000 and 2005 (mean follow-up: 2.5 years). We used mixed models adjusted for confounders including age, sex, race, education, and smoking to estimate the association between baseline and between ultrasounds using a spatio-temporal model. Among 5,362 participants (5% of participants had missing data) higher levels of residential PM<sub>2.5</sub> during the follow-up period were associated with greater IMT progressions among persons in the same metropolitan area. For each 1 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>, IMT progression without adjustment for metropolitan area (0.4 µm/y) showed positive associations. Greater reductions in PM<sub>2.5</sub> over time were associated with slowed IMT progression (−2.8 µm/y [95% CI −1.6 to −4.0]).

These findings suggest that higher long-term PM<sub>2.5</sub> concentrations are associated with greater IMT progression. These findings, even after adjustment for metropolitan area and the progression of atherosclerosis over the full 10 years of follow-up in this cohort, these findings will help inform public health strategies to reduce the burden of cardiovascular disease.



## AHA Scientific Statement

### Particulate Matter Air Pollution and Cardiovascular Disease An Update to the Scientific Statement From the American Heart Association

Robert D. Brook, MD, Chair; Sanjay Rajagopalan, MD; C. Arden Pope III, PhD;  
Jeffrey R. Brook, PhD; Aruni Bhatnagar, PhD, FAHA; Ana V. Diez-Roux, MD, PhD, MPH;  
Fernando Holguin, MD; Yuling Hong, MD, PhD, FAHA; Russell V. Luepker, MD, MS, FAHA;  
Murray A. Mittleman, MD, DrPH, FAHA; Annette Peters, PhD; David Siscovick, MD, MPH, FAHA;  
Sidney C. Smith, Jr, MD, FAHA; Laurie Whitsel, PhD; Joel D. Kaufman, MD, MPH; on behalf of the  
American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in  
Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism

**Abstract**—In 2004, the first American Heart Association scientific statement on “Air Pollution and Cardiovascular Disease” concluded that exposure to particulate matter (PM) air pollution contributes to cardiovascular morbidity and mortality. In the interim, numerous studies have expanded our understanding of this association and further elucidated the physiological and molecular mechanisms involved. The main objective of this updated American Heart Association scientific statement is to provide a comprehensive review of the new evidence linking PM exposure with cardiovascular disease, with a specific focus on highlighting the clinical implications for researchers and healthcare providers. The writing group also sought to provide expert consensus opinions on many aspects of the current state of science and updated suggestions for areas of future research. On the basis of the findings of this review, several new conclusions were reached, including the following: Exposure to PM  $<2.5\ \mu\text{m}$  in diameter (PM<sub>2.5</sub>) over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; longer-term exposure (eg, a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between PM<sub>2.5</sub> exposure and cardiovascular morbidity and mortality. This body of evidence has grown and been strengthened substantially since the first American Heart Association scientific statement was published. Finally, PM<sub>2.5</sub> exposure is deemed a modifiable factor that contributes to cardiovascular morbidity and mortality. (*Circulation*. 2010;121:2331-2378.)

**Key Words:** AHA Scientific Statements ■ atherosclerosis ■ epidemiology ■ prevention



# Avis scientifique de l'AHA sur les effets de la pollution particulaire sur les maladies cardiovasculaires

Brook et al. (Circulation 2010)

## Exposition à court terme aux $PM_{2.5}$ (heures, semaines)

- peut précipiter la mortalité et la morbidité CV (ischémie du myocarde, infarctus myocarde, insuffisance cardiaque, arythmies, accidents ischémiques cérébraux)

## Exposition à long terme aux $PM_{2.5}$ (années)

- Augmente le risque de mortalité CV de façon plus importante que l'exposition à CT
- RR de mortalité CV encore plus importants pour des populations particulières et des pathologies spécifiques
- La réduction de l'espérance de vie associée aux  $PM_{2.5}$  est principalement due à l'excès de mortalité CV
- La réduction des niveaux de PM peut diminuer la mortalité CV dans un délai de seulement quelques années
- Pas de seuil de protection détecté
- Les effets des expositions à LT sur la morbidité CV moins consistants

**Etude de cohorte anglaise** montre des liens entre l'exposition à long terme aux particules et NO<sub>2</sub> et le développement d'insuffisance cardiaque. *Long-term exposure to outdoor air pollution and incidence of cardiovascular diseases. Atkinson et al. Epidemiology, 2013 Jan;24(1):44-53.*

## RESEARCH

## Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project



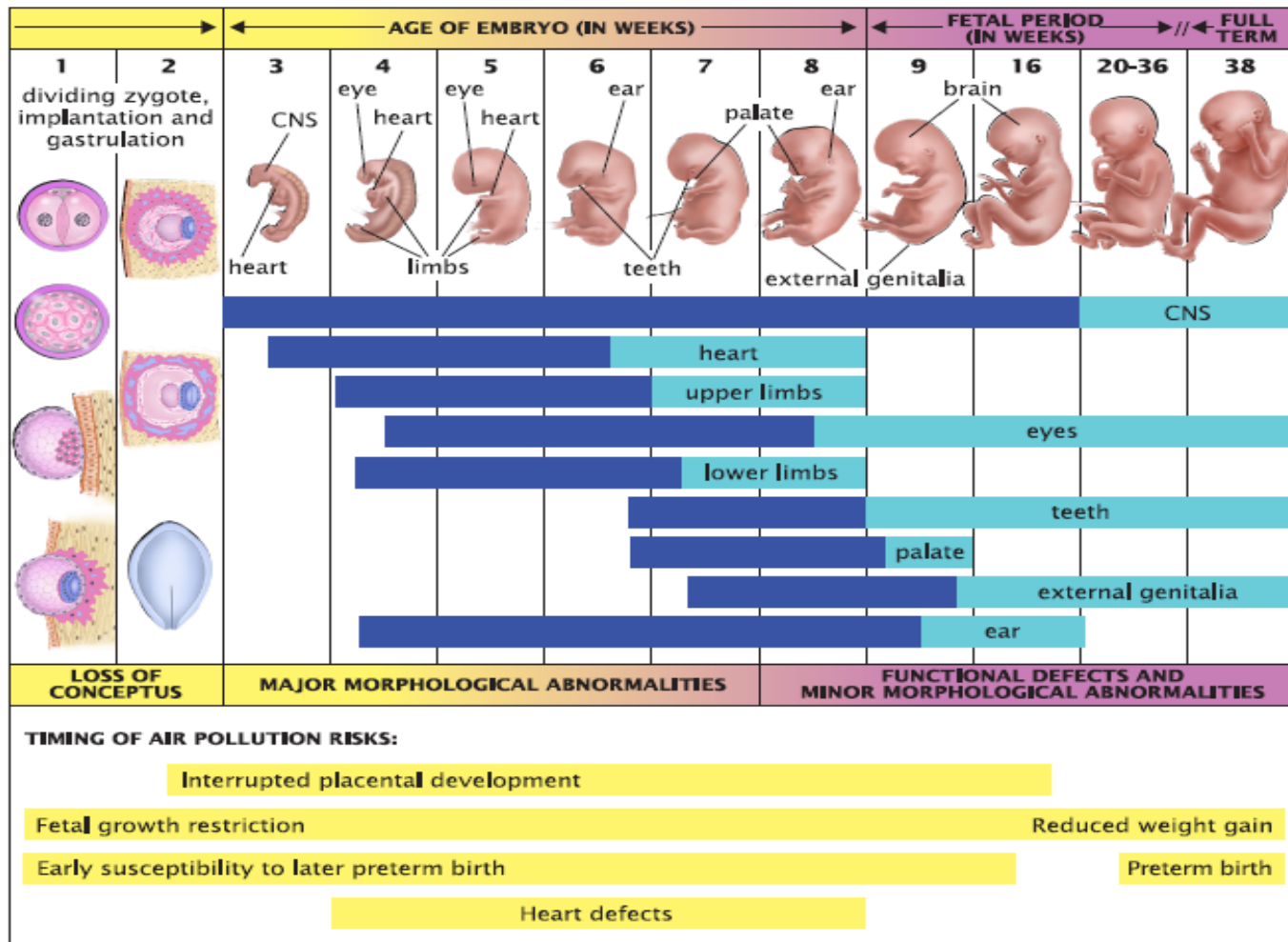
OPEN ACCESS

Giulia Cesaroni *senior researcher*<sup>1</sup>, Francesco Forastiere *research director*<sup>1</sup>, Massimo Stafoggia *senior researcher*<sup>1</sup>, Zorana J Andersen *associate professor in epidemiology*<sup>2,3</sup>, Chiara Badaloni *research fellow*<sup>1</sup>, Rob Beelen *senior researcher*<sup>4</sup>, Barbara Caracciolo *researcher*<sup>5,6</sup>, Ulf de Faire *senior professor of cardiovascular epidemiology*<sup>7</sup>, Raimund Erbel *professor*<sup>8</sup>, Kirsten T Eriksen *researcher*<sup>2</sup>, Laura Fratiglioni *professor in geriatric epidemiology*<sup>5,9,10</sup>, Claudia Galassi *medical epidemiologist*<sup>11</sup>, Regina Hampel *research fellow*<sup>12</sup>, Margit Heier *research fellow*<sup>12,13</sup>, Frauke Hennig *research fellow*<sup>14</sup>, Agneta Hilding *researcher*<sup>15</sup>, Barbara Hoffmann *professor*<sup>14,16</sup>, Danny Houthuijs *senior researcher*<sup>17</sup>, Karl-Heinz Jöckel *professor*<sup>18</sup>, Michal Korek *doctoral student*<sup>7</sup>, Timo Lanki *chief researcher*<sup>19</sup>, Karin Leander *researcher*<sup>7</sup>, Detlef K E Meerpolder *professor*<sup>20</sup>, Enrica Migliore



# Effets sur le développement fœtal

# Pollution atmosphérique et développement foetal



Note: Blue bars indicate time periods when major morphological abnormalities can occur, while light blue bars correspond to periods at risk for minor abnormalities and functional defects.

Figure 1. Fetal development and timing of air pollution risks.

*Air pollution impacts on infants and children, B. Beate and M. Wilhelm, J. Dowdalls (medical illustrations), UCLA, 2008,*

# Ambient air pollution and low birthweight: a European cohort study (ESCAPE)

Marie Pedersen, Lise Giorgis-Allemand, Claire Bernard, Inmaculada Aguilera, Anne-Marie Nybo Andersen, Ferran Ballester, Rob M J Beelen, Leda Chatzi, Marta Cirach, Asta Danileviciute, Audrius Dedele, Manon van Eijsden, Marisa Estarlich, Ana Fernández-Somoano, Mariana F Fernández, Francesco Forastiere, Ulrike Gehring, Regina Grazuleviciene, Olena Gruzdeva, Barbara Heude, Gerard Hoek, Kees de Hoogh, Edith H van den Hooven, Siri E Håberg, Vincent W V Jaddoe, Claudia Klümper, Michal Korek, Ursula Krämer, Aitana Lerchundi, Johanna Lepeule, Per Nafstad, Wenche Nystad, Evridiki Patelarou, Daniela Porta, Dirkje Postma, Ole Raaschou-Nielsen, Peter Rudnai, Jordi Sunyer, Euripides Stephanou, Mette Sørensen, Elisabeth Thiering, Derek Tuffnell, Mihály J Varró, Tanja G M Vrijkotte, Alet Wijga, Michael Wilhelm, John Wright, Mark J Nieuwenhuijsen, Göran Pershagen, Bert Brunekreef, Manolis Kogevinas\*, Rémy Slama\*

## Summary

**Background** Ambient air pollution has been associated with restricted fetal growth, which is linked with adverse respiratory health in childhood. We assessed the effect of maternal exposure to low concentrations of ambient air pollution on birthweight.

**Methods** We pooled data from 14 population-based mother–child cohort studies in 12 European countries. Overall, the study population included 74178 women who had singleton deliveries between Feb 11, 1994, and June 2, 2011, and for whom information about infant birthweight, gestational age, and sex was available. The primary outcome of interest was low birthweight at term (weight <2500 g at birth after 37 weeks of gestation). Mean concentrations of particulate matter with an aerodynamic diameter of less than 2.5 µm (PM<sub>2.5</sub>), less than 10 µm (PM<sub>10</sub>), and between 2.5 µm and 10 µm during pregnancy were estimated at maternal home addresses with temporally adjusted land-use regression models, as was PM<sub>2.5</sub> absorbance and concentrations of nitrogen dioxide (NO<sub>2</sub>) and nitrogen oxides. We also investigated traffic density on the nearest road and total traffic load. We calculated pooled effect estimates with random-effects models.

**Findings** A 5 µg/m<sup>3</sup> increase in concentration of PM<sub>2.5</sub> during pregnancy was associated with an increased risk of low birthweight at term (adjusted odds ratio [OR] 1.18, 95% CI 1.06–1.33). An increased risk was also recorded for pregnancy concentrations lower than the present European Union annual PM<sub>2.5</sub> limit of 25 µg/m<sup>3</sup> (OR for 5 µg/m<sup>3</sup> increase in participants exposed to concentrations of less than 20 µg/m<sup>3</sup> 1.41, 95% CI 1.20–1.65). PM<sub>10</sub> (OR for 10 µg/m<sup>3</sup> increase 1.16, 95% CI 1.00–1.35), NO<sub>2</sub> (OR for 10 µg/m<sup>3</sup> increase 1.09, 1.00–1.19), and traffic density on nearest street (OR for increase of 5000 vehicles per day 1.06, 1.01–1.11) were also associated with increased risk of low birthweight at term. The population attributable risk estimated for a reduction in PM<sub>2.5</sub> concentration to 10 µg/m<sup>3</sup> during pregnancy corresponded to a decrease of 22% (95% CI 8–33%) in cases of low birthweight at term.

**Interpretation** Exposure to ambient air pollutants and traffic during pregnancy is associated with restricted fetal growth. A substantial proportion of cases of low birthweight at term could be prevented in Europe if urban air pollution was reduced.



# Effets sur le système nerveux central

# Pollution atmosphérique et développement neurologique chez l'enfant

Research | Children's Health

## Effect of Prenatal Exposure to Airborne Polycyclic Aromatic Hydrocarbons on Neurodevelopment in the First 3 Years of Life among Inner-City Children

Frederica P. Perera,<sup>1</sup> Virginia Rauh,<sup>1</sup> Robin M. Whyatt,<sup>1</sup> Mary C. Fox,<sup>1</sup> Joseph A. Sparano,<sup>1</sup> Lori Hoepner,<sup>1</sup> Dana Barr,<sup>3</sup> Yi-Hsuan Tu,<sup>1</sup> David Camann,<sup>1</sup> et al.

<sup>1</sup>Columbia Center for Children's Environmental Health, Mailman School of Public Health, New York, New York, USA; <sup>2</sup>Department of Statistics, National Cheng Kung University, Tainan, Taiwan; <sup>3</sup>Department of Environmental Health Sciences, National Center for Environmental Health, Division of Environmental and Environmental Chemistry, Southwest Research Institute, San Antonio, Texas, USA

Our prospective cohort study of non-Hispanic Black children in New York City is evaluating the pathogenesis of neurobehavioral disorders. We evaluated the effects on child mental and behavioral outcomes of PAHs monitored during pregnancy by the Child Behavior Checklist. We evaluated the effects of geographic factors and prenatal exposure to PAHs (upper quartile) on psychomotor development. Prenatal exposure to PAHs (upper quartile) was associated with psychomotor development delay (mean difference [MD] = -5.69; 95% confidence interval [CI], -10.38 to -0.99;  $p = 0.01$ ). PAH effect on mental development was not significant (MD = 2.89; 95% CI, -1.33 to 6.25;  $p = 0.01$ ). Further adjustment for lead did not alter results. The results require confirmation in other populations recently encountered in New York City.



OPEN ACCESS  
RESEARCH ARTICLE

### Early-Life Exposure to Airborne Polycyclic Aromatic Hydrocarbons and Child Neurodevelopment

Frederica P. Perera, Virginia Rauh, Robin M. Whyatt, Mary C. Fox, Joseph A. Sparano, Lori Hoepner, Dana Barr, Yi-Hsuan Tu, David Camann, et al.

Published: November 05, 2014

Article



- Abstract
- Introduction
- Methods
- Results
- Discussion
- Conclusions
- Acknowledgments
- Author Contributions
- References

Reader Comments (0)

Une nouvelle étude de Columbia University (nov 2014) trouve des liens entre HAP et troubles de l'attention à l'âge de 9 ans. Une étude précédente (2012) associait l'exposition prénatal aux HAP à des troubles cognitifs et du comportement, incluant des associations avec du retard de développement à 3 ans, diminution du QI à 5 ans, et des symptômes d'anxiété/depression et troubles de l'attention à 6 et 7 ans.

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Subject Areas

- ADHD
- Anxiety
- Aromatic hydrocarbons
- Behavior
- Behavioral disorders
- Children

## Abstract

### Importance

Polycyclic aromatic hydrocarbons are widespread urban air pollutants from combustion of fossil fuel and other organic material shown previously to be neurotoxic.

### Objective

In a prospective cohort study, we evaluated the relationship between Attention Deficit Hyperactivity Disorder behavior problems and prenatal polycyclic aromatic hydrocarbon exposure, adjusting for postnatal exposure.

## *Review Article*

## **Air Pollution, Oxidative Stress, and Alzheimer's Disease**

**Paula Valencia Moulton<sup>1</sup> and Wei Yang<sup>1,2</sup>**

<sup>1</sup> *The Environmental Sciences and Health Graduate Program, University of Nevada, Reno, NV 89557-0186, USA*

<sup>2</sup> *School of Community Health Sciences, University of Nevada, Reno, NV 89557-0274, USA*

Correspondence should be addressed to Wei Yang, weiyang@unr.edu

Received 18 September 2011; Accepted 3 January 2012

Academic Editor: Pam R. Factor-Litvak

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Alzheimer's disease (AD) is the most common form of dementia affecting millions of people worldwide and will continue to affect millions more with population aging on the rise. AD causality is multifactorial. Known causal factors include genetic predisposition, age, and sex. Environmental toxins such as air pollution (AP) have also been implicated in AD causation. Exposure to AP can lead to chronic oxidative stress (OS), which is involved in the pathogenesis of AD. Whereas AP plays a role in AD pathology, the epidemiological evidence for this association is limited. Given the significant prevalence of AP exposure combined with increased population aging, epidemiological evidence for this link is important to consider. In this paper, we examine the existing evidence supporting the relationship between AP, OS, and AD and provide recommendations for future research on the population level, which will provide evidence in support of public health interventions.

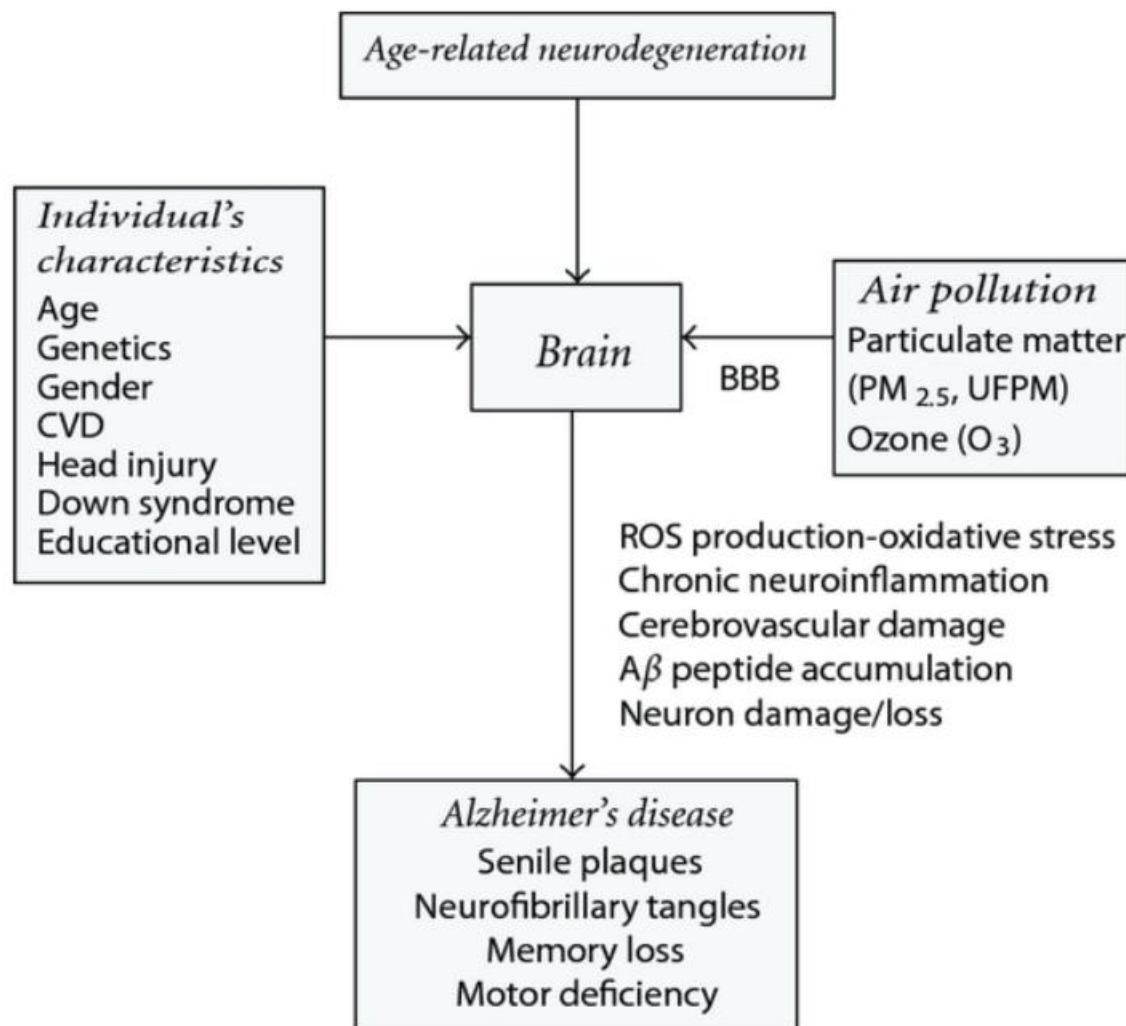


Figure 1: Interacting factors in the causality of Alzheimer's disease [1, 4–11, 17, 19, 28, 35, 72, 73]. Alzheimer's disease is the result of the interaction of aging, genetic predisposition, and environmental exposures such as air pollution in the etiology and pathogenesis of the disease. Air pollution is a prevalent environmental source of ROS that impacts the brain through the multiple pathways accelerating the development and clinical manifestation of Alzheimer's disease. BBB: blood-brain barrier; ROS: reactive oxygen species; CVD: cardiovascular disease.

# Pollution atmosphérique et autisme

## Residential Proximity to Freeways and Autism in the CHARGE Study

Heather E. Volk,<sup>1</sup> Irva Hertz-Picciotto,<sup>2</sup> Lora Delwiche,<sup>2</sup> Fred Lurmann,<sup>3</sup> and Rob McConnell<sup>4</sup>

<sup>1</sup>Departments of Preventive Medicine and Pediatrics, Zilkha Neurogenetic Institute, Keck School of Medicine, Children's Hospital Los Angeles, University of Southern California, Los Angeles, California, USA; <sup>2</sup>Department of Public Health Sciences, University of California–Davis, Davis, California, USA; <sup>3</sup>Sonoma Technology Inc., Petaluma, California, USA; <sup>4</sup>Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California, USA

**BACKGROUND:** Little is known about environmental causes and contributing factors for autism. Basic science and epidemiologic research suggest that oxidative stress and inflammation may play a role in disease development. Traffic-related air pollution, a common exposure with established effects on these pathways, contains substances found to have adverse prenatal effects.

**OBJECTIVES:** We examined the association between autism and proximity of residence to freeways and major roadways during pregnancy and near the time of delivery, as a surrogate for air pollution exposure.

**METHODS:** Data were from 304 a Childhood Autism Risks from G address recorded on the birth cert history obtained by questionnaire roads were calculated using ArcGI imity to freeways and major roads

**RESULTS:** Adjusting for sociodem time of delivery was more likely b (OR) = 1.86; 95% confidence inte proximity to a freeway during the socioeconomic and sociodemogra near other major roads at birth wa

**CONCLUSIONS:** Living near a freev measured air pollutants is needed.

levels. Additionally, autism has been associ- ated with estimated regional concentrations of hazardous air pollutants, including arsenic and nickel, and with diesel PM exposure in early childhood (Windham et al. 2006).

Thus, an emerging literature suggests that near roadways, traffic-related air pollutants

Published in final edited form as:

*Epidemiology*. 2014 January ; 25(1): 44–47. doi:10.1097/EDE.0000000000000030.

## Interaction of the MET Receptor Tyrosine Kinase Gene and Air Pollution Exposure in Autism Spectrum Disorder

Heather E. Volk<sup>1,2,3</sup>, Tara Kerin<sup>1</sup>, Fred Lurmann<sup>4</sup>, Irva Hertz-Picciotto<sup>5</sup>, Rob McConnell<sup>1</sup>, and Daniel B. Campbell<sup>3,6,7</sup>

<sup>1</sup>Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA

<sup>2</sup>Department of Pediatrics, Children's Hospital Los Angeles, University of Southern California, Los Angeles, CA

<sup>3</sup>Zilkha Neurogenetic Institute, Keck School of Medicine, University of Southern California, Los Angeles, CA



# Effets sur l'obésité

# Interaction tabagisme passif-pollution atmosphérique pendant la grossesse et développement de l'obésité chez l'enfant

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*Environ Health Perspect*; DOI:10.1289/ehp.1307031

## **A Longitudinal Cohort Study of Body Mass Index and Childhood Exposure to Secondhand Tobacco Smoke and Air Pollution: The Southern California Children's Health Study**

Rob McConnell,<sup>1</sup> Ernest Shen,<sup>1</sup> Frank D. Gilliland,<sup>1</sup> Michael Jerrett,<sup>2</sup> Jennifer Wolch,<sup>3</sup> Chih-Chieh Chang,<sup>1</sup> Frederick Lurmann,<sup>4</sup> and Kiros Berhane<sup>1</sup>

Author Affiliations [close](#)

<sup>1</sup>Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California, USA; <sup>2</sup>Department of Environmental Health, School of Public Health, University of California, Berkeley, California, USA; <sup>3</sup>Department of City and Regional Planning, College of Environmental Design, University of California, Berkeley, California, USA; <sup>4</sup>Sonoma Technology, Inc, Petaluma, California, USA



# Review of evidence on health aspects of air pollution – **REVIHAAP Project**

World Health Organization 2013

Principales références

[http://www.euro.who.int/\\_data/assets/pdf\\_file/0020/182432/e96762-final.pdf](http://www.euro.who.int/_data/assets/pdf_file/0020/182432/e96762-final.pdf)



# Depuis les “WHO Air Quality Guidelines” de 2005

## Quoi de neuf ?

---

**PM10:** confirmation des études antérieures (effets à court et long termes)

### **PM2,5**

- Confirmation des effets à court et long termes sur la mortalité et la morbidité
- Meilleure connaissance des mécanismes d'action sur le système cardio-vasculaire (études toxicologiques, épidémiologiques, cliniques)
- Nouvelles études reliant exposition à long terme et athérosclérose, faible poids à la naissance, naissances prématurées, maladies respiratoires chez l'enfant
- Etudes émergentes sur l'altération de la fonction cognitive et le diabète

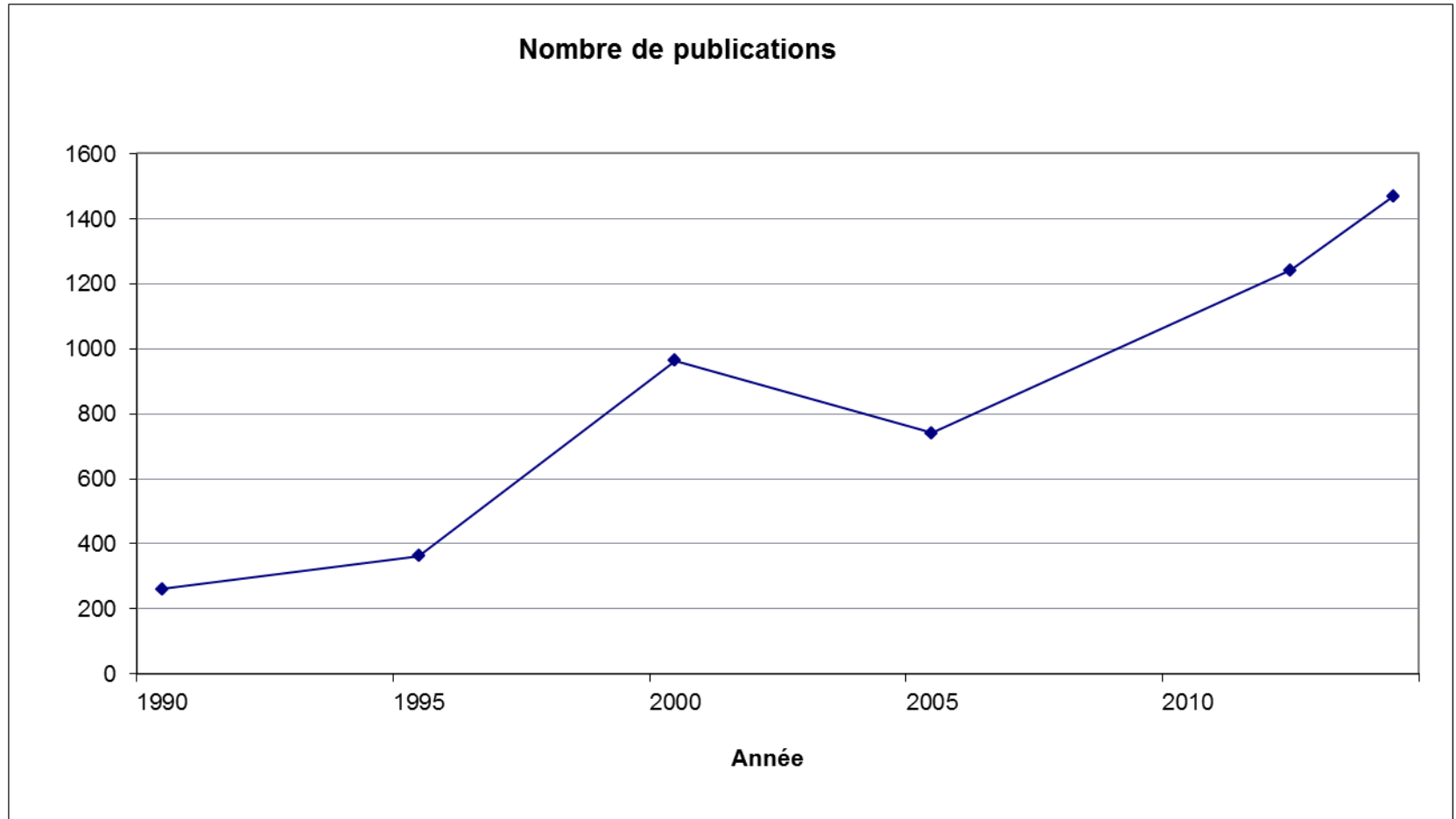
### **Ozone**

- Confirmation des effets respiratoires et cardiovasculaires à court terme
- Nouvelles études sur effets à long terme (mortalité respiratoire, incidence de l'asthme)

### **NO2**

- Effets propres au NO2 et meilleure connaissance des mécanismes d'action à court terme sur l'appareil respiratoire
- Effets à long terme sur mortalité cardio-respiratoire, symptômes et fonction respiratoire de l'enfant mais effets difficiles à individualiser des autres polluants

# Polluants atmosphériques et santé : de plus en plus d'études



Recherche PubMed: « air pollution » NOT  
« indoor » AND health AND « date  
publication »



Quelles conséquences  
en termes de santé publique ?

# Dernières estimations de l'OMS au niveau mondial

## mars 2014

[Nouvelles et statistiques](#)[Centre des médias](#)[Publications](#)[Pays](#)[Programmes et projets](#)[À propos de l'OMS](#)[Centre des médias](#)[Publications](#)[Pays](#)[Programmes et projets](#)[À propos de l'OMS](#)

### Centre des médias

## 7 millions de décès prématurés sont liés à la pollution de l'air chaque année

Communiqué de presse

25 MARS 2014 | GENÈVE - Dans de nouvelles estimations publiées aujourd'hui, l'Organisation mondiale de la Santé (OMS) indique que près de 7 millions de personnes sont décédées prématurément en 2012 – une sur huit au niveau mondial – du fait de l'exposition à la pollution de l'air. Ces chiffres représentent plus du double des estimations précédentes et confirment que la pollution de l'air est désormais le principal risque environnemental pour la santé dans le monde. On pourrait sauver des millions de vies en luttant contre la pollution de l'air.

### Centre des médias

## Qualité de l'air ambiant (extérieur) et santé

Aide-mémoire N°313

Mars 2014

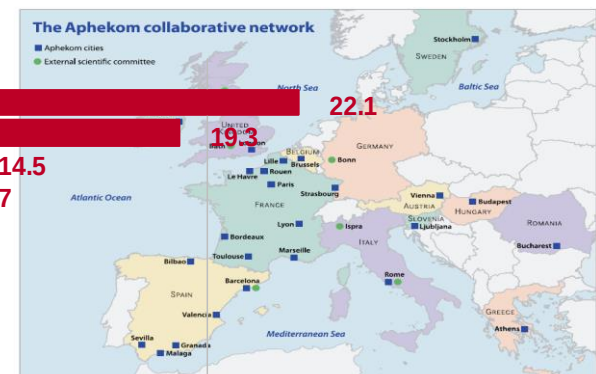
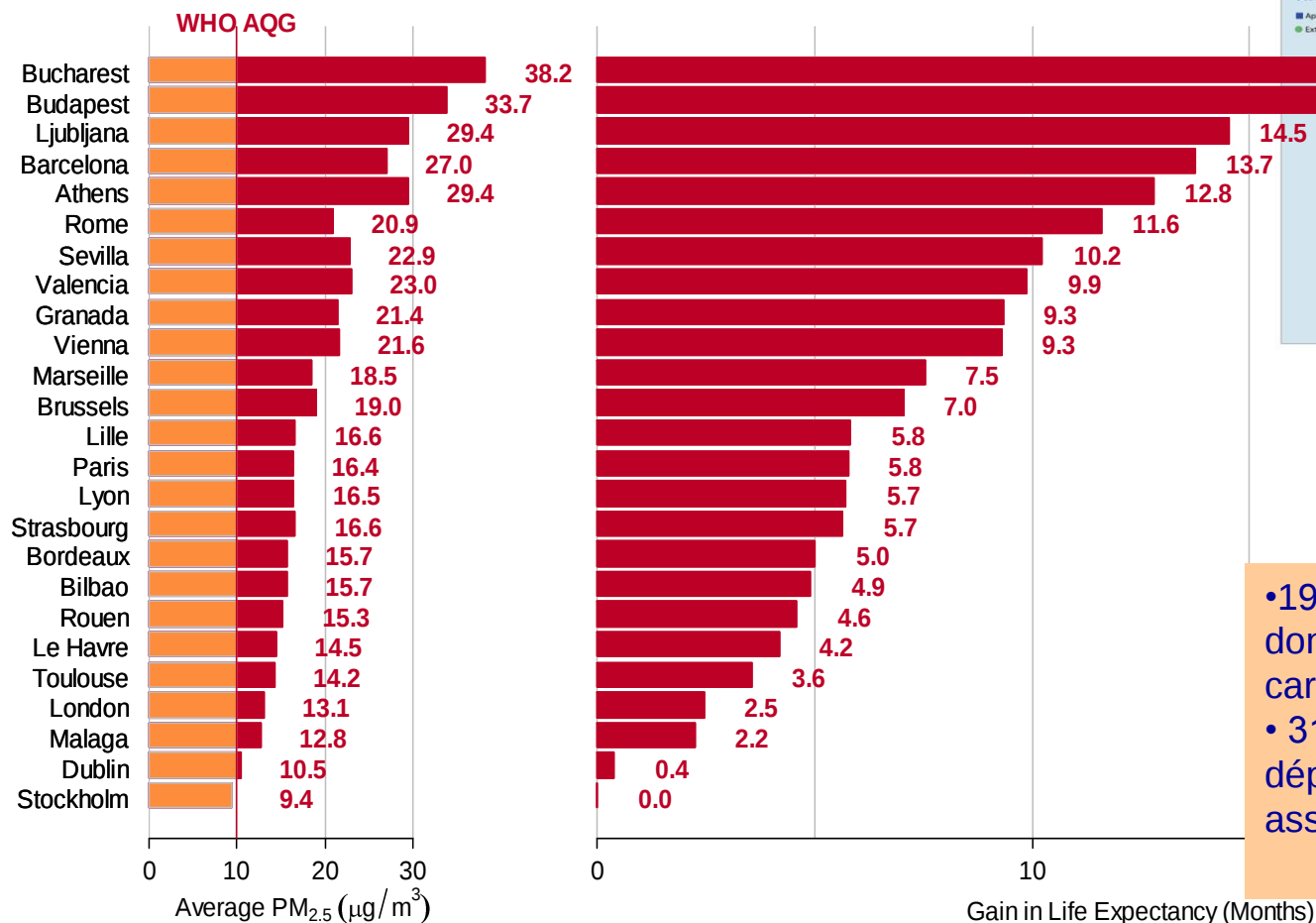
### Principaux faits

- La pollution de l'air représente un risque environnemental majeur pour la santé. En diminuant les niveaux de pollution atmosphérique, les pays peuvent réduire la charge de morbidité imputable aux accidents vasculaires cérébraux, aux cardiopathies, au cancer du poumon et aux affections respiratoires, chroniques ou aiguës, y compris l'asthme.
- La santé cardiovasculaire et respiratoire de la population, à court et long termes, est inversement proportionnelle au niveau de la pollution atmosphérique.
- Les *Lignes directrices OMS relatives à la qualité de l'air* évaluent les effets de la pollution atmosphérique sur la santé et donnent des valeurs seuils au-delà desquelles elle lui est nuisible.
- En 2012, on estimait à 3,7 millions le nombre de décès prématurés provoqués dans le monde par la pollution ambiante (de l'air extérieur) dans les zones urbaines et rurales.

# Aphekom en Europe

Improving Knowledge and Communication for Decision Making on  
Air Pollution and Health in Europe (2008-2011)

Gain en espérance de vie à 30 ans dans 25 villes d'Aphekom si les concentrations annuelles de PM<sub>2.5</sub> respectaient la valeur OMS de 10 µg/m<sup>3</sup>



- 19 000 décès prématurés dont 15 000 décès pour causes cardiovasculaires
- 31.5 milliards d'euros en dépenses de santé et coûts associés



# En France dans les années 2000

**La pollution de l'air était responsable d'environ 19 000 à 44 000 décès prématurés chaque année**  
(Künzli et al., Lancet, 2000)

ARTICLES

---

## **Public-health impact of outdoor and traffic-related air pollution: a European assessment**

*N Künzli, R Kaiser, S Medina, M Studnicka, O Chanel, P Filliger, M Herry, F Horak Jr, V Puybonnieux-Textier, P Quénel, J Schneider, R Seethaler, J-C Vergnaud, H Sommer*

---

### **Summary**

**Background** Air pollution contributes to mortality and morbidity. We estimated the impact of outdoor (total) and traffic-related air pollution on public health in Austria,

action in Europe. Our results, which have also been used for economic valuation, should guide decisions on the assessment of environmental health-policy options.

*Lancet* 2000; **356**: 795–801



# Comparaison à d'autres risques en France

- **42 000 morts par an attribuables à la pollution** (programme CAFE Cost-Benefit Analysis 2005)
- **3 700 décès sur nos routes** en 2013
- **70 000 morts par an attribués au tabac**

Nous choisissons d'être ou non fumeur mais nous ne choisissons pas l'air qu'on respire. Nous respirons **TOUS**, populations sensibles ou pas, le contenu (bon et mauvais) de l'air



# Episodes de pollution



# Episodes de pollution

- Météo favorable à une accumulation des polluants atmosphériques
- Seuils réglementaires ( $PM_{10}$ ) :
  - Information :  $50 \mu\text{g}/\text{m}^3$  (en moyenne journalière)
  - Alerte :  $80 \mu\text{g}/\text{m}^3$  (en moyenne journalière)
- Effets exacerbés sur la santé :
  - Bénins : toux, hypersécrétion nasale, expectoration, essoufflement, irritation nasale des yeux et de la gorge...
  - Graves : hospitalisations pour causes cardiovasculaire ou respiratoire pouvant entraîner le décès

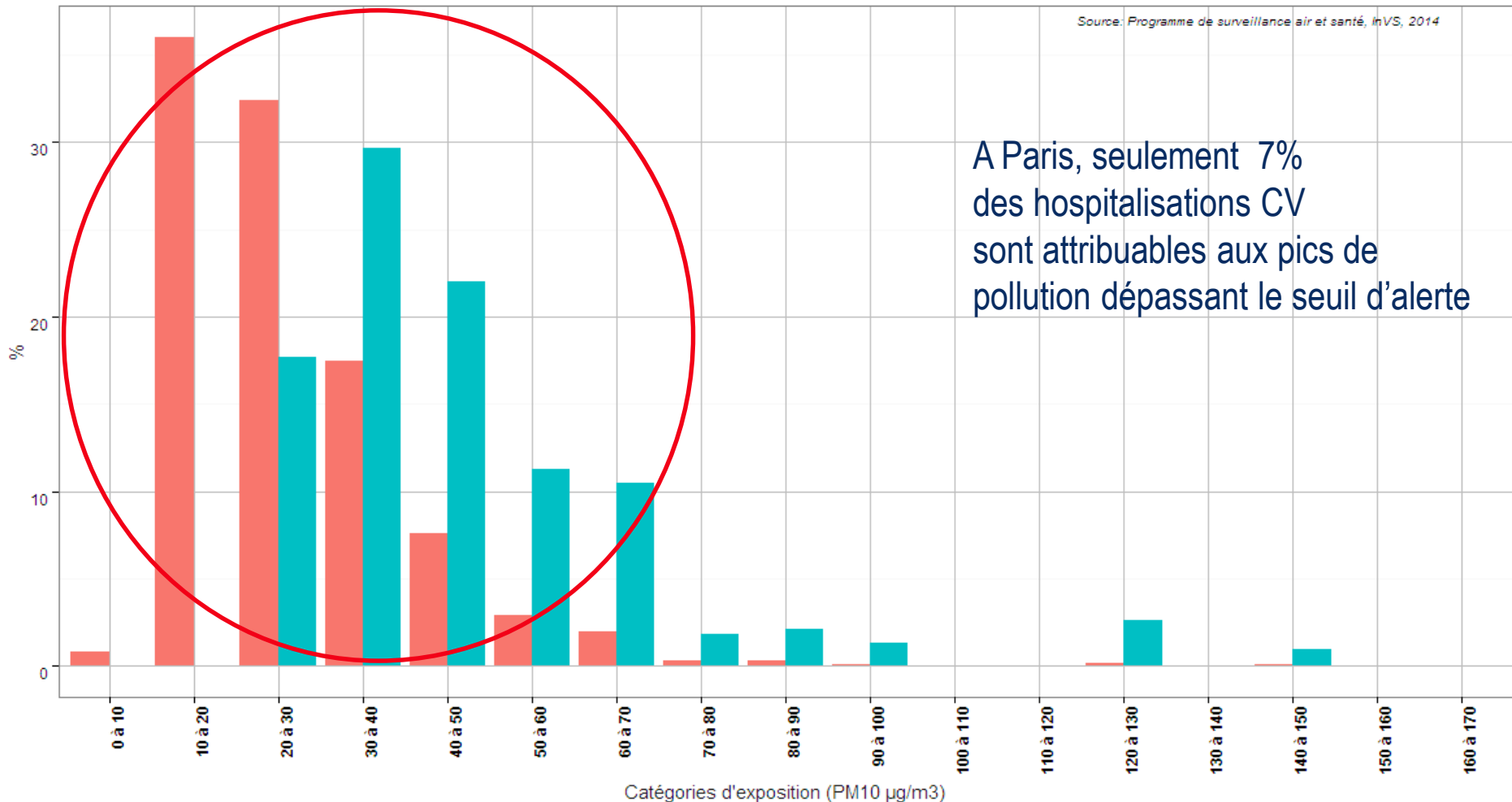
# Poids relatif des épisodes de pollution sur la santé

Distribution des niveaux journaliers de PM10 et hospitalisations pour causes cardiovasculaires associées (%)

Paris, période 2007-2010

PM10 Hospitalisation causes cardiovasculaires

Source: Programme de surveillance air et santé, InVS, 2014





# Quel est le message à retenir ?

**Gérer les pics de pollution ne suffit pas  
Il faut agir aussi et surtout sur la  
pollution de fond  
et les sources de pollution  
(émissions de polluants et de précurseurs)**



# Sait-on tout ce qu'il faut savoir ?

Pour être plus utiles à la décision individuelle et collective, il y a besoin de poursuivre la surveillance des effets sur la santé de la pollution :

- Etudes épidémiologiques plus globales sur les sources de pollution
- Travaux d'évaluation d'impact sanitaire sur les scénarios énergétiques
- Evaluation pluridisciplinaires d'actions mises en place pour réduire les niveaux de pollution
- Un meilleur partage des connaissances avec ceux qui en ont besoin

Plus d'échanges avec météorologistes, climatologues, urbanistes, professionnels de la communication, ... (*barrières scientifiques, culturelles, de langage, mais bons exemples : projet AC-HIA*)

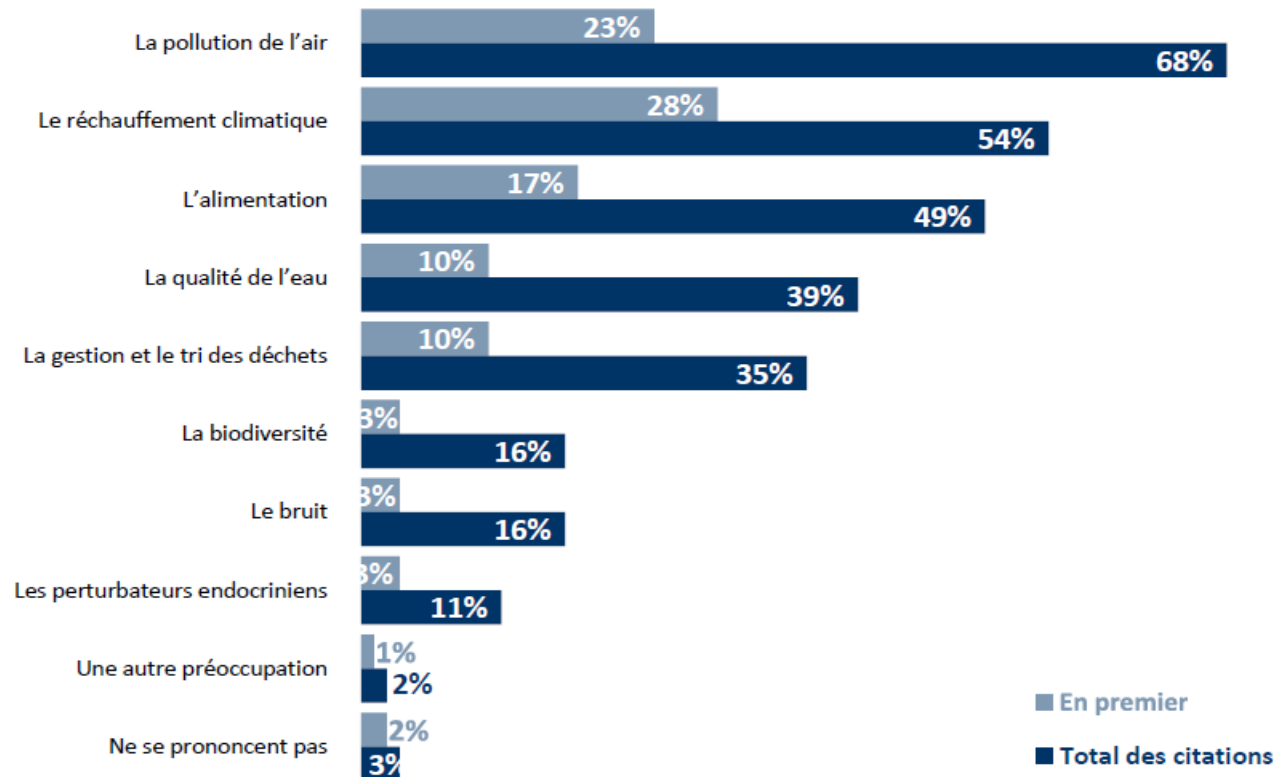
# Que pense la population ?

Sondage IFOP pour Airparif, Octobre 2014



## Les principales préoccupations environnementales

**QUESTION :** De manière générale, en matière d'environnement, qu'est-ce qui vous préoccupe le plus ? En premier ? En deuxième ? En troisième ?



# Que pensent les scientifiques ?

*Société internationale d'épidémiologie environnementale, Octobre 2014*



INTERNATIONAL SOCIETY FOR  
ENVIRONMENTAL EPIDEMIOLOGY

Website: [www.isee.org](http://www.isee.org)



INTERNATIONAL SOCIETY FOR  
ENVIRONMENTAL EPIDEMIOLOGY-EUROPE

Website: [www.iseeepi.org](http://www.iseeepi.org)

Prof. Manolis Kogevinas, MD, PhD, *Chair*  
Prof. Barbara Hoffmann, MD, PhD, *Vice-Chair*

Prof. Manolis Kogevinas, MD, PhD, *Chair*  
Prof. Barbara Hoffmann, MD, PhD, *Vice-Chair*

October 16, 2014

Re: 3rd French National Plan on Environmental Health (PNSE3)

Re: 3rd French National Plan on Environmental Health (PNSE3)

To : Madame Marisol Touraine  
Ministre des Affaires Sociales, de la Santé et des Droits des Femmes  
14, avenue Duquesne  
75350 PARIS 07 SP  
France

To : Madame Ségolène Royal  
Ministre de l'Ecologie, du Développement durable et de l'Energie  
246, boulevard Saint-Germain  
Hôtel de Roquelaure  
75007 Paris, France

Dear Minister Touraine,

Dear Minister Royal,

The European Chapter of the International Society for Environmental Epidemiology is pleased to participate in your consultation, and in particular regarding the effort

We, as scientists, are concerned by the fact that PNSE3 does not include clear and ambitious goals in terms of reducing the exposure of the French population to outdoor pollutants such as fine particulate matter (PM<sub>2.5</sub>). As you know, the current EU regulation, with a yearly limit of 25 micrograms/m<sup>3</sup> for PM<sub>2.5</sub>, is twice as high as the current regulation in the USA (set to 12 micrograms/m<sup>3</sup>) and two and a half times as high as the value of 10 micrograms/m<sup>3</sup> recommended by the World Health Organization. Aiming to comply with the EU regulation (by 2020) is therefore far from being protective for your populations. An ambitious reduction in the mean exposure of the French population, even those currently below the EU yearly limit, could avoid thousands of death and many more disease cases every year, with a considerable monetary gain.

We are looking forward to assisting you, and the citizens of your country, in proving that comprehensive efforts to clean the air have a positive impact on health and wellbeing and urge you to strongly support the reduction of air pollution exposures, below the current EU regulatory levels.

Yours sincerely,

Prof. Manolis Kogevinas, Chair of the European Chapter, International Society for Environmental Epidemiology, CREAL Barcelona.

Prof. Barbara Hoffmann, Vice-Chair of the European Chapter, International Society for Environmental Epidemiology, Leibnitz Research Institute for Environmental Medicine, Düsseldorf.



**Fait-on assez ?**



**Merci**