Des polluants atmosphériques qui altèrent notre santé

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

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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 citadins européens sont exposées à des niveaux de pollution dépassant les normes de qualité de l'air de l'UE

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

Source : EEA 2013

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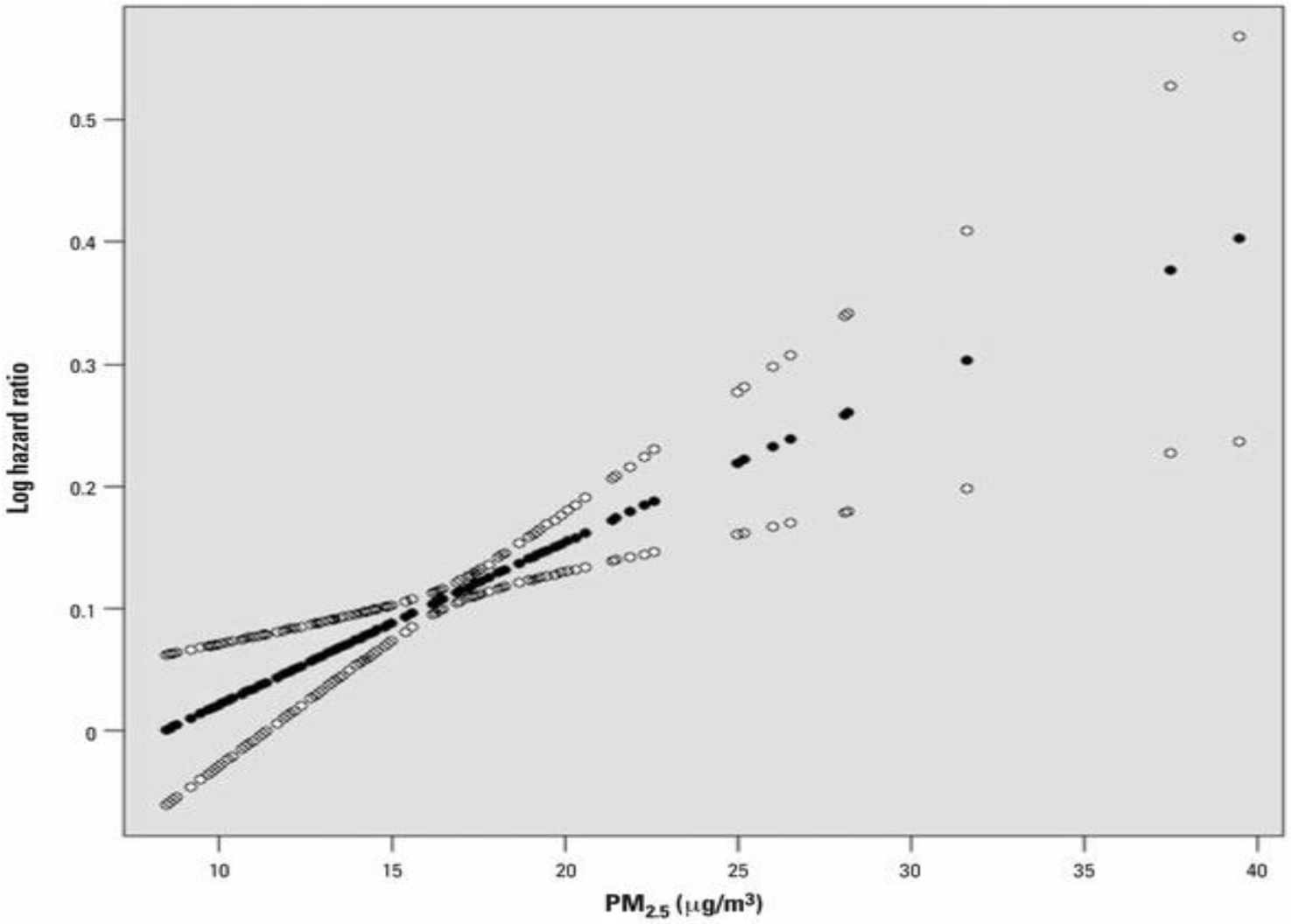
Relation linéaire sans seuil



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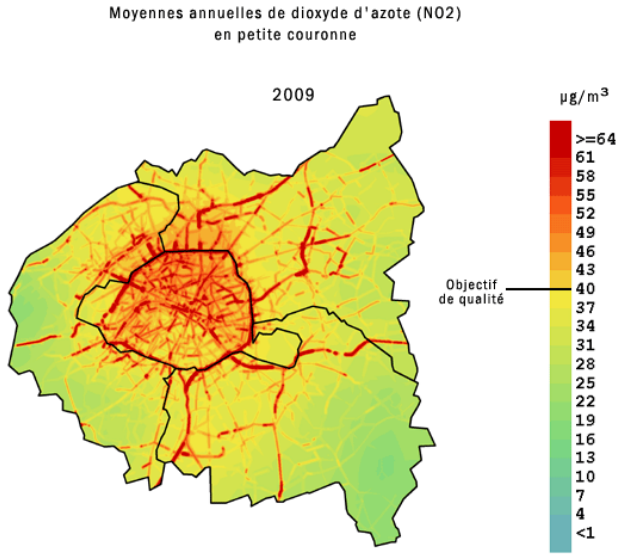
Des inégalités d'exposition

- 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



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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 cardio-vasculaires, 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 ?

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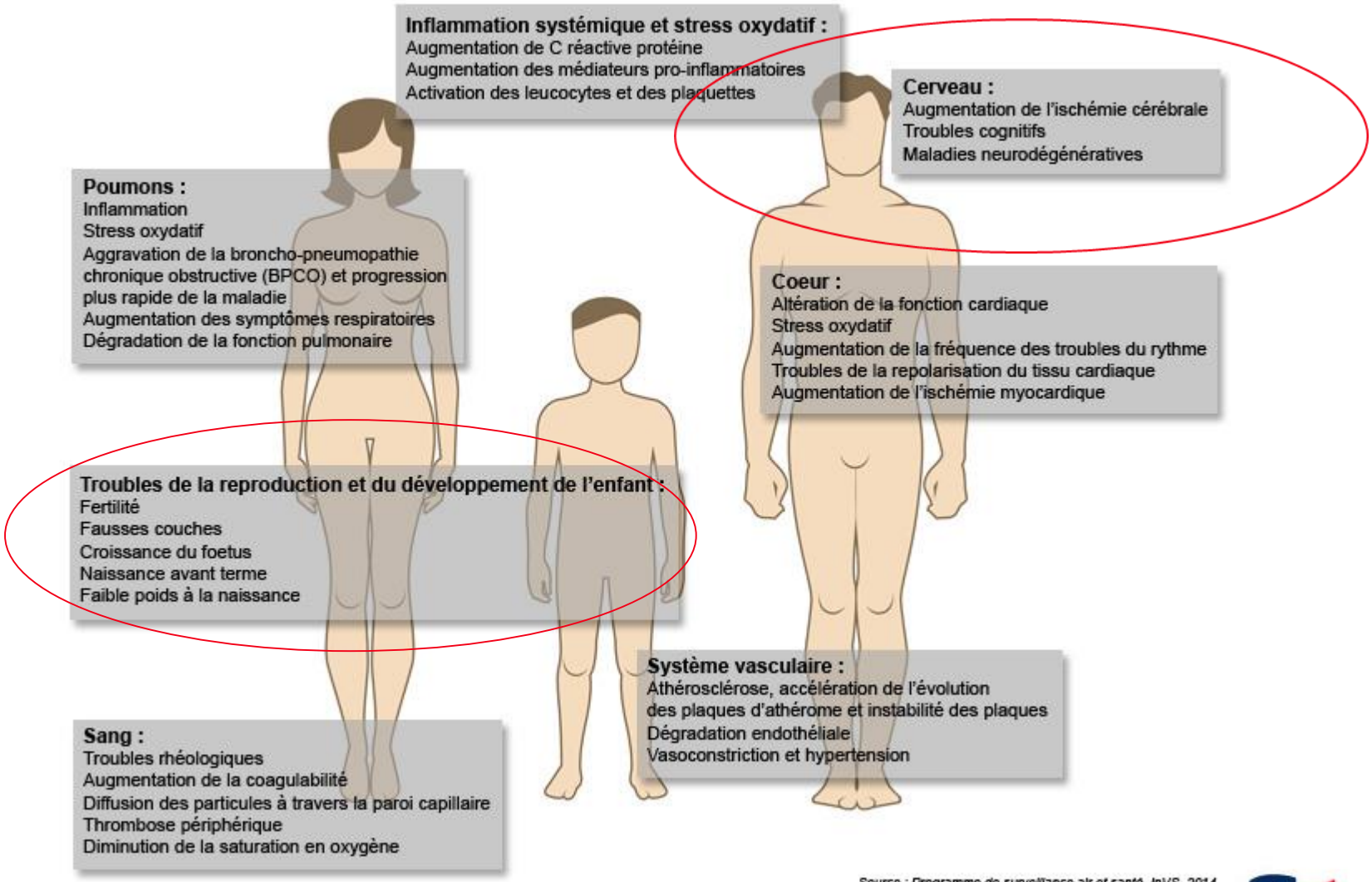
Effets sur la santé de la pollution atmosphérique



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

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Effets sur l'appareil respiratoire

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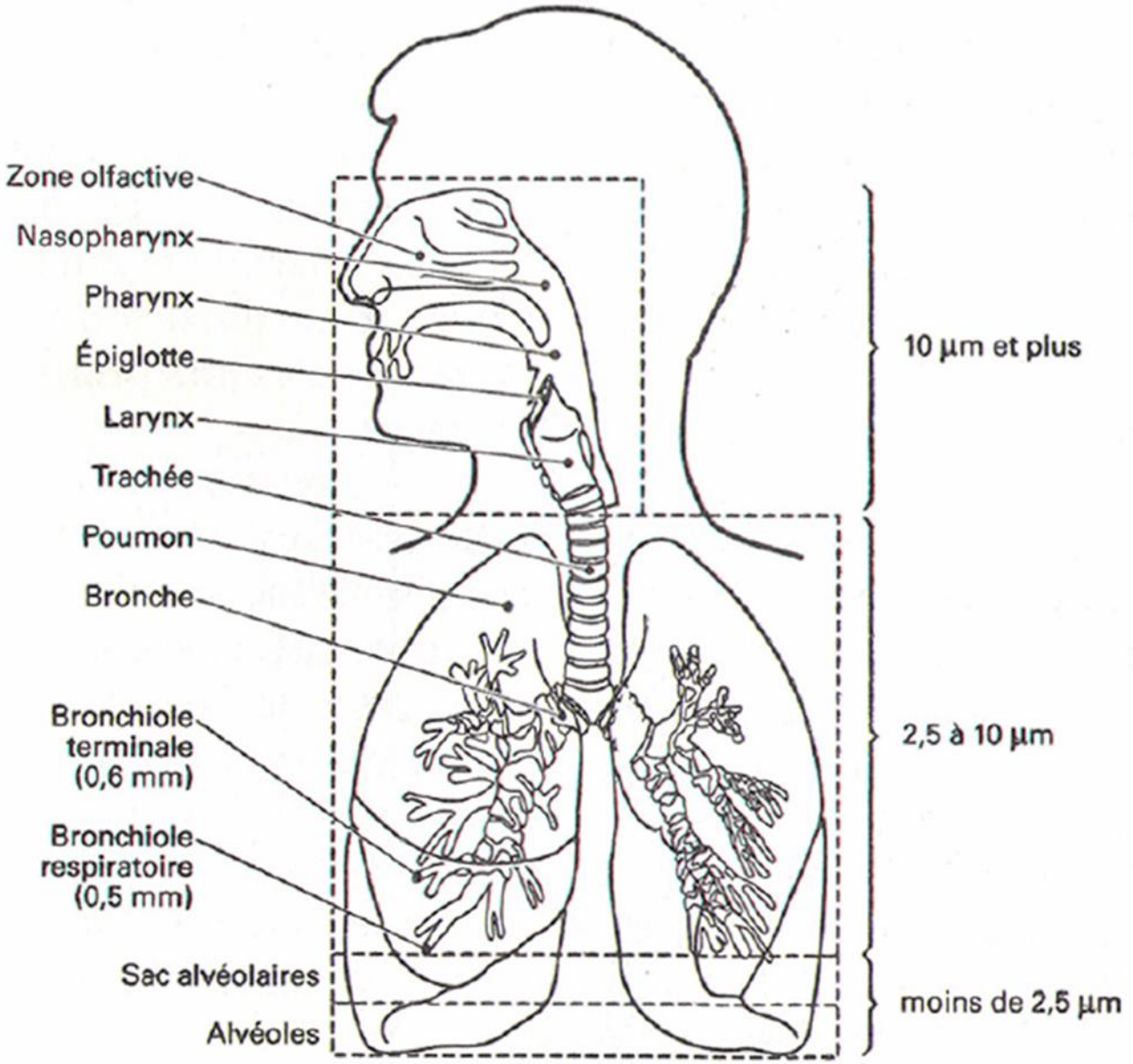
Pénétration des PM selon leur taille



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

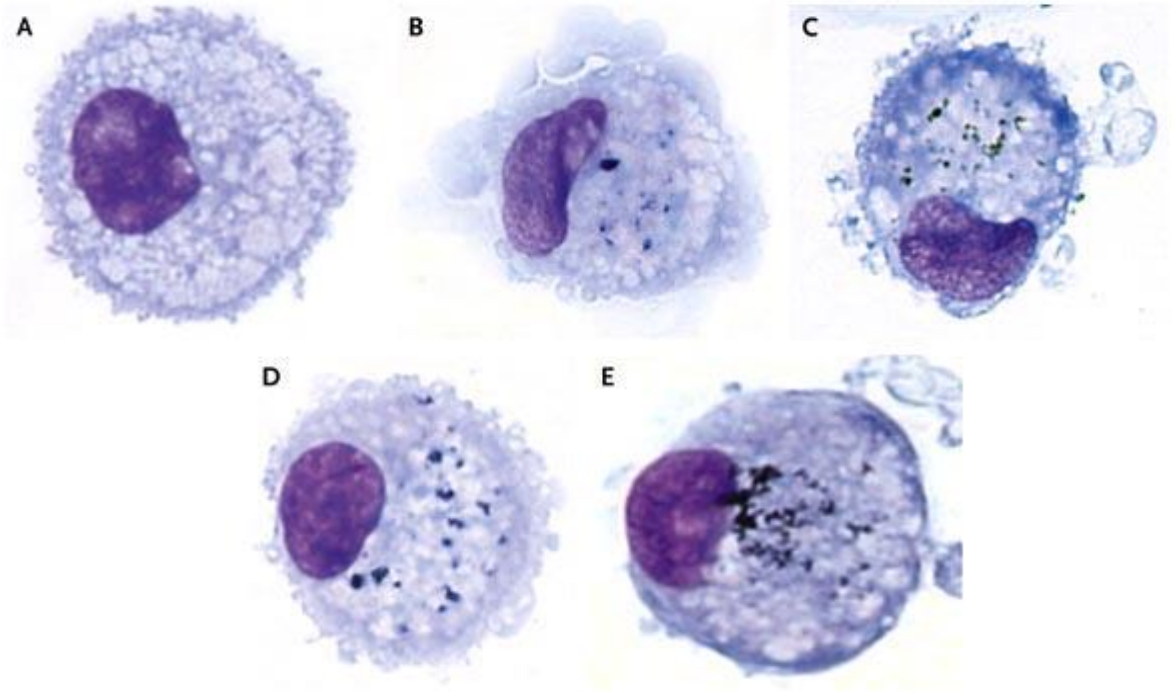

Figure 1. Representative Images of Carbon in Airway Macrophages from Healthy Children. Panel A shows a macrophage with no carbon. Increasing levels of carbon are shown in Panels B through E.

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Pollution atmosphérique et développement de l'appareil respiratoire

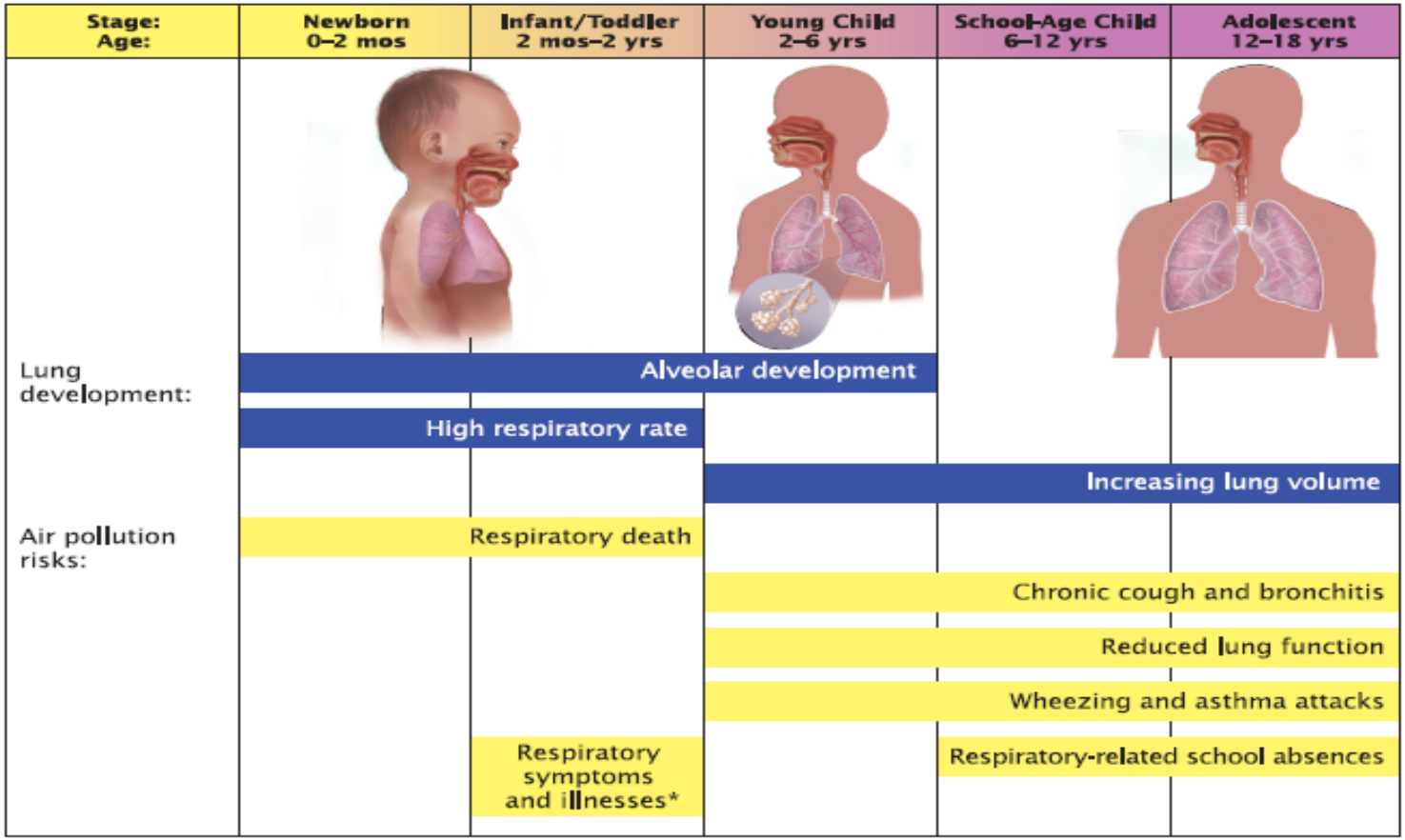

Figure 2. Air pollution effects on the developing respiratory system.

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

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

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

Research | Children's Health

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

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

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Childhood Incident Asthma and Traffic-Related Air Pol and School

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

[1]University of Southern California, Los Angeles, California, USA; [2]St. Michael's Hospital, Toronto, Ontario, Canada; [3]University of California, Berkeley, California, USA; [4]Sonoma Technology, Inc, Petaluma, California, USA; [5]Swiss Tropical and Public Health Institute, Basel and University of Basel, Switzerland; [6]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.

The Southern California Children's Health Study (CHS) was designed to investigate the chronic effects of air pollution on respiratory health. Previous analyses have found associations of asthma with residential distance to major roads and modeled and measured pol-

Methods: Parent-r during 3 years of fol asthma- and wheezing assessed traffic-relate distance from home (NO$_2$), and particulat study communities. central site pollutants

Results: Asthma risk homes [HR 1.51; 95 1.06–1.98). Ambient increased risk (HR 2. sures, there were ind home, whereas the es

Conclusions: Traffi development of asthn

Key words: air poll 118:1021–1026 (201

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

Pollutant	HR[a] (95% CI)
NO_2	2.17 (1.18–4.00)
PM_{10}	1.35 (0.64–2.85)
$PM_{2.5}$	1.66 (0.91–3.05)
O_3	0.76 (0.38–1.54)

[a]HR (95% CI) across the range of exposure in the 13 communities (23.6 ppb for NO_2, 43.9 μg/m^3 PM_{10}, 17.4 μg/m^3 $PM_{2.5}$, and 30.3 ppb for 1000–1800 hours O_3), 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.[a]

Central site pollutant	HR[b] (95% CI) for ambient pollutant, adjusted for TRP at home and school	HR[b] (95% CI) for home TRP, adjusted for school TRP and ambient pollutant	HR[b] (95% CI) for school TRP, adjusted for home TRP and ambient pollutant
NO_2	1.37 (0.69–2.71)	1.46 (1.16–1.84)*	1.45 (1.03–2.06)
PM_{10}	1.40 (0.62–3.17)	1.46 (1.16–1.85)*	1.53 (1.10–2.12)*
$PM_{2.5}$	1.30 (0.66–2.56)	1.48 (1.19–1.85)*	1.49 (1.07–2.08)
O_3	1.01 (0.49–2.11)	1.50 (1.20–1.86)*	1.54 (1.10–2.14)*

[a]Mutually adjusted across each row (i.e., effect of each community pollutant was examined separately in a model including both home and school TRP). [b]HR (95% CI) for central-site pollutants scaled across the range of exposure in the 13 communities (23.6 ppb for NO_2, 43.9 μg/m^3 PM_{10}, 17.4 μg/m^3 $PM_{2.5}$, and 30.3 ppb for 1000–1800 hours O_3); 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_2), distance to nearest major road and the onset and incidence of asthma

	Onset asthma		Incident asthma	
	OR	95% CI	OR	95% CI
Crude#				
NO_2 per 10 μg·m^{-3}	1.39	1.05–1.84	1.36	0.92–2.02
Distance to nearest major road <50 m¶	2.81	1.66–4.76	3.55	1.78–7.01
Fully adjusted+				
NO_2 per 10 μg·m^{-3}	1.46	1.07–1.99	1.54	1.00–2.36
Distance to nearest major road <50 m¶	2.92	1.70–4.98	3.88	1.93–7.82

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

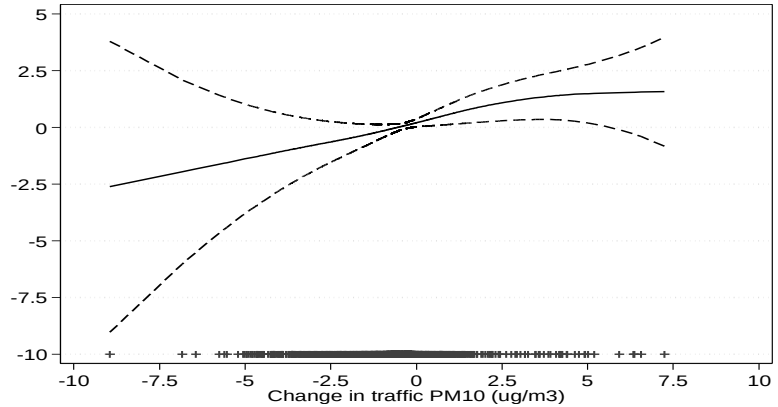

Figure 2: Association between change in traffic-related PM_{10} and adult-onset asthma (log-hazard with 95%-confidence interval) among SAPALDIA never-smokers. (Generalized additive model, adjusted for age, sex, atopy at baseline, BMI at baseline, bronchial hyperreactivity at baseline, maternal allergies.) 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 NO2 and new onset of self-reported asthma in adults.

Jacquemin B, Sunyer J, Forsberg B, Aguilera I, Briggs D, García-Esteban R, Götschi T, Heinrich J, Järvholm 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 (NO2) estimates, as a marker of local traffic-related pollution. We obtained this information from the 1-km background NO2 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 NO2 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 NO2 et incidence d'asthme chez l'adulte (OR 1,43; IC 95% 1.2 à 2.1 par 10 μg/m^3)

Kuenzli et al. SAPALDIA Study, Thorax 2009

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Centre international de Recherche sur le (

Organisation mondiale de la Santé

COMMUNIQUE DE I
N° 221

Le Centre International de Recherche sur le Cancer (CIRC) classe les particules fines diesel carcinogènes certains pour l'homme

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)

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

La pollution atmosphérique une des prem de décès par cancer,

Lyon/Genève, 17 octobre 2013 – Agence spécialisée sur le canc international de Recherche sur le Cancer (CIRC), a annoncé au comme ca

Après avc experts m suffisantes 1). Ils ont

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

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 Xun, 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_{10}), less than 2·5 µm ($PM_{2.5}$), and between 2·5 and 10 µm (PM_{coarse}), soot ($PM_{2.5absorbance}$), 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_{10} (hazard ratio [HR] 1·22 [95% CI 1·03–1·45] per 10 µg/m³). For $PM_{2.5}$ the HR was 1·18 (0·96–1·46) per 5 µg/m³. The same increments of PM_{10} and $PM_{2.5}$ 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 201
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July 10, 2013
http://dx.doi.org/
S1470-2045(13)7
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Effets sur le système cardiovasculaire

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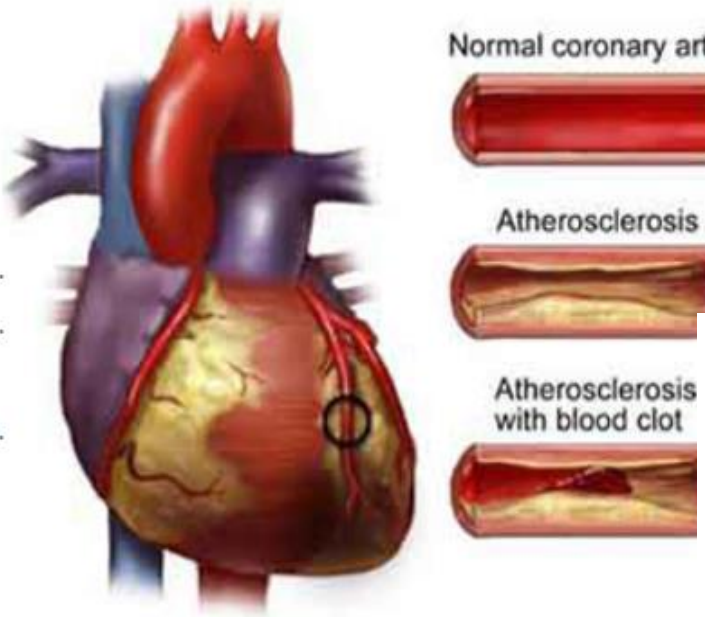

Pollution atmosphérique et maladies coronariennes

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

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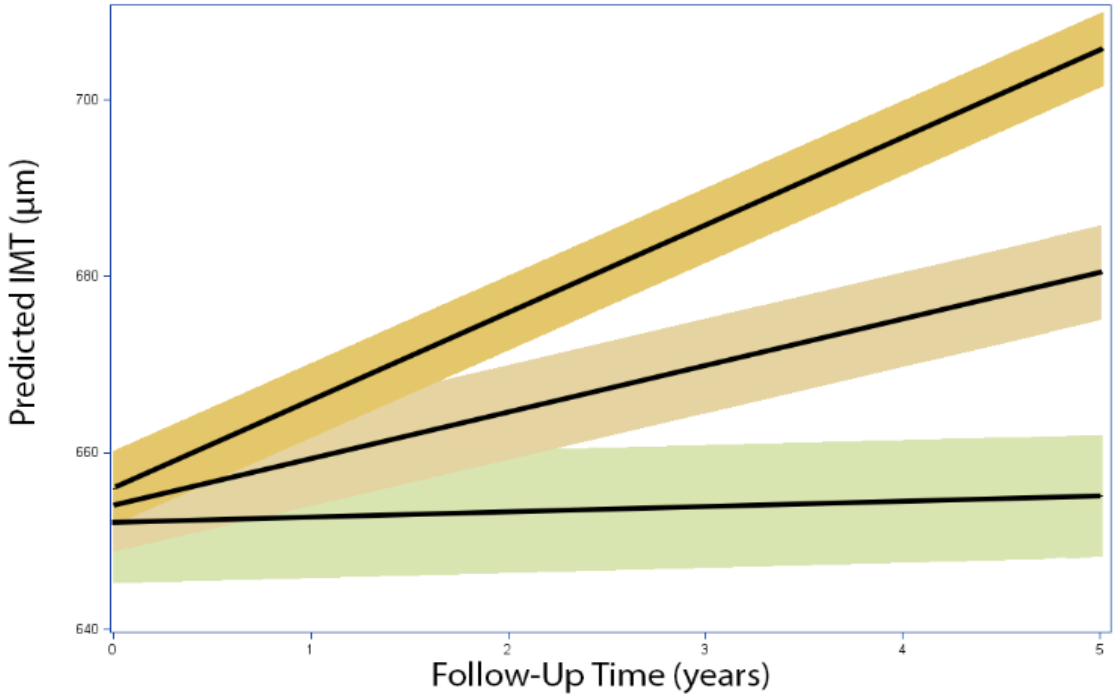
8 Division of Epidemiology and Community Health, University of Minnesota, Minneapolis,
iology, Columbia University Medical Center, New York, New York, United States of America,
eattle, Washington, United States of America



en linked to cardiovascular disease, possibly via accelerated progression of the intima-medial thickness (IMT) of the common g-term $PM_{2.5}$ concentrations in participants from the Multi-Ethnic

, enrolled 6,814 participants at the baseline exam (2000–2002), with ound examinations between 2000 and 2005 (mean follow-up: 2.5 aseline and between ultrasounds using a spatio-temporal model. ined using mixed models adjusted for confounders including age, ors. Among 5,362 participants (5% of participants had missing data) higher levels of residential $PM_{2.5}$ during the follow-up period were er IMT progressions among persons in the same metropolitan area. T progression without adjustment for metropolitan area (0.4 µm/y reas showed positive associations. Greater reductions in $PM_{2.5}$ over ated with slowed IMT progression (−2.8 µm/y [95% CI −1.6 to clude the use of a surrogate measure of atherosclerosis, some loss concentrations prior to 1999.

that higher long-term $PM_{2.5}$ concentrations are associated with $PM_{2.5}$ are related to slower IMT progression. These findings, even d literature on air pollution and the progression of atherosclerotic the full 10 years of follow-up in this cohort, these findings will help

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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 μm in diameter ($PM_{2.5}$) 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 $PM_{2.5}$ 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, $PM_{2.5}$ 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

thebmj

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BMJ 2014;348:f7412 doi: 10.1136/bmj.f7412 (Published 21 January 2014)

Etude de cohorte anglaise montre des liens entre l'exposition à long terme aux particules et NO_2 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

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

Effets sur le développement fœtal

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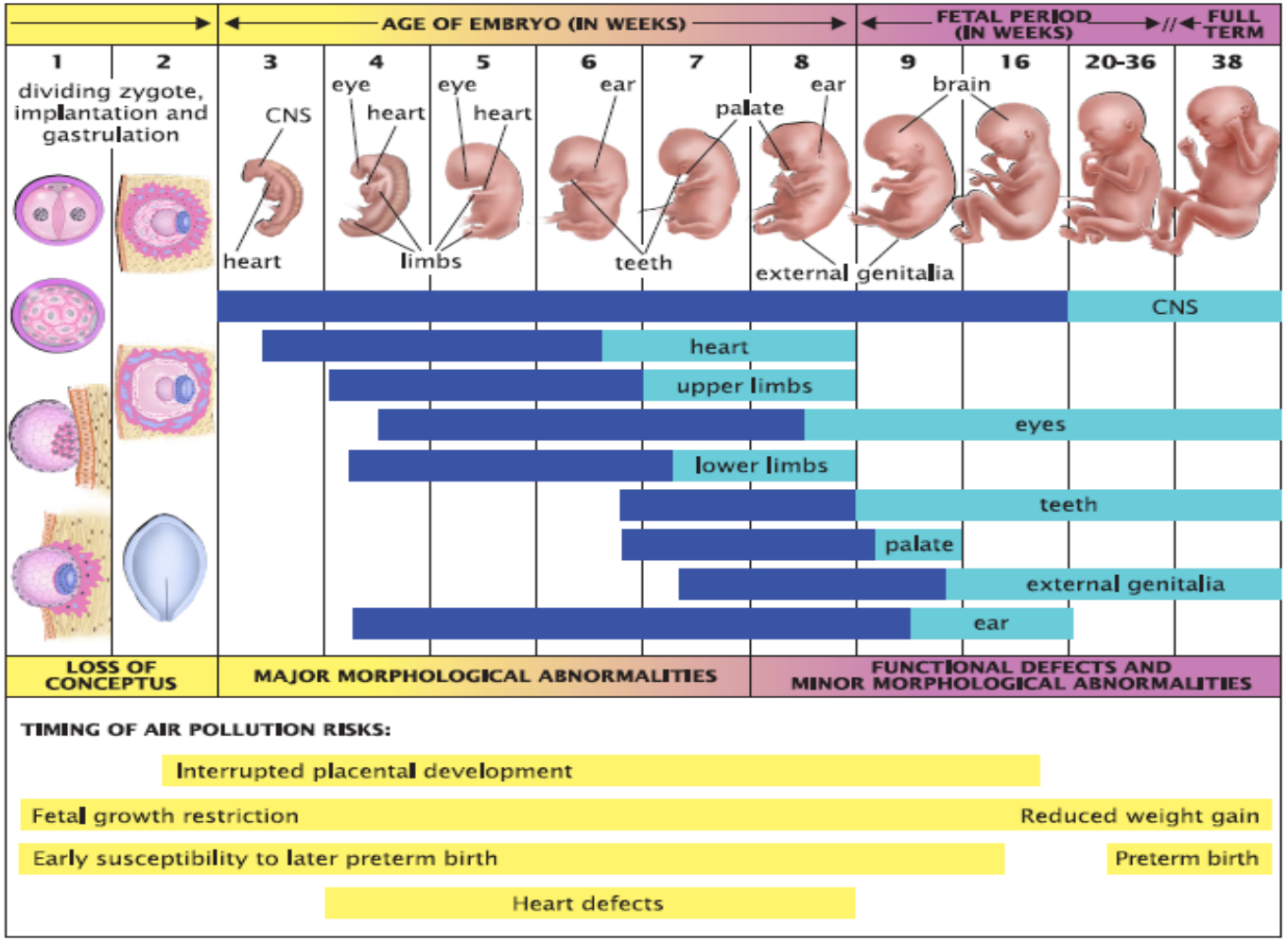
Pollution atmosphérique et développement fœtal



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,

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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 Gruzieva, Barbara Heude, Gerard Hoek, Kees de Hoogh, Edith H van den Hooven, Siri E Håberg, Vincent WV 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 74 178 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 ($PM_{2.5}$), less than 10 µm (PM_{10}), 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 $PM_{2.5}$ absorbance and concentrations of nitrogen dioxide (NO_2) 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/m³ increase in concentration of $PM_{2.5}$ 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 $PM_{2.5}$ limit of 25 µg/m³ (OR for 5 µg/m³ increase in participants exposed to concentrations of less than 20 µg/m³ 1·41, 95% CI 1·20–1·65). PM_{10} (OR for 10 µg/m³ increase 1·16, 95% CI 1·00–1·35), NO_2 (OR for 10 µg/m³ 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 $PM_{2.5}$ concentration to 10 µg/m³ 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.

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Effets sur le système nerveux central

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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,[1] Virginia Rauh,[1] Robin M. Whyatt,[1] Lori Hoepner,[1] Dana Barr,[3] Yi-Hsuan Tu,[1] David Caman

[1]Columbia Center for Children's Environmental Health, Mailman USA; [2]Department of Statistics, National Cheng Kung University, Prevention, National Center for Environmental Health, Division o and Environmental Chemistry, Southwest Research Institute, San

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 developpement à 3 ans, diminution du QI à 5 ans, et des symptômes d'anxiété/depression et troubles de l'attention à 6 et 7ans.

Our prospective cohort study of non children in New York City is evaluatin polycyclic aromatic hydrocarbons (PAI the pathogenesis of neurobehavioral dis evaluate the effects on child mental and PAHs monitored during pregnancy by by the Child Behavior Checklist. We graphic factors and prenatal exposure t associated with psychomotor developn exposure to PAHs (upper quartile) wa [β = −5.69; 95% confidence interval (C mental delay were also significantly gr 2.89; 95% CI, 1.33 to 6.25; $p = 0.01$). × PAH effect on mental development Further adjustment for lead did not alt by ethnicity. The results require con recently encountered in New York City 3 years of age, with implications for s

PLOS

OPEN ACCESS PEER-REVI

RESEARCH ARTICLE

Early-Life Exp Problems

Frederica P. Perera, Shuang Wang, Virginia F

Published: November 05,

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- Discussion
- Conclusions
- Acknowledgments
- Author Contributions
- References

Reader Comments (0)

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.

CrossMark

Subject Areas

ADHD | Anxiety | Aromatic hydroc | Behavior | Behavioral diso | Children

Pollution atmosphérique et maladies neurodégénératives

Hindawi Publishing Corporation
Journal of Environmental and Public Health
Volume 2012, Article ID 472751, 9 pages
doi:10.1155/2012/472751

Review Article

Air Pollution, Oxidative Stress, and Alzheimer's Disease

Paula Valencia Moulton[1] and Wei Yang[1,2]

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Received 18 September 2011; Accepted 3 January 2012

Academic Editor: Pam R. Factor-Litvak

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.

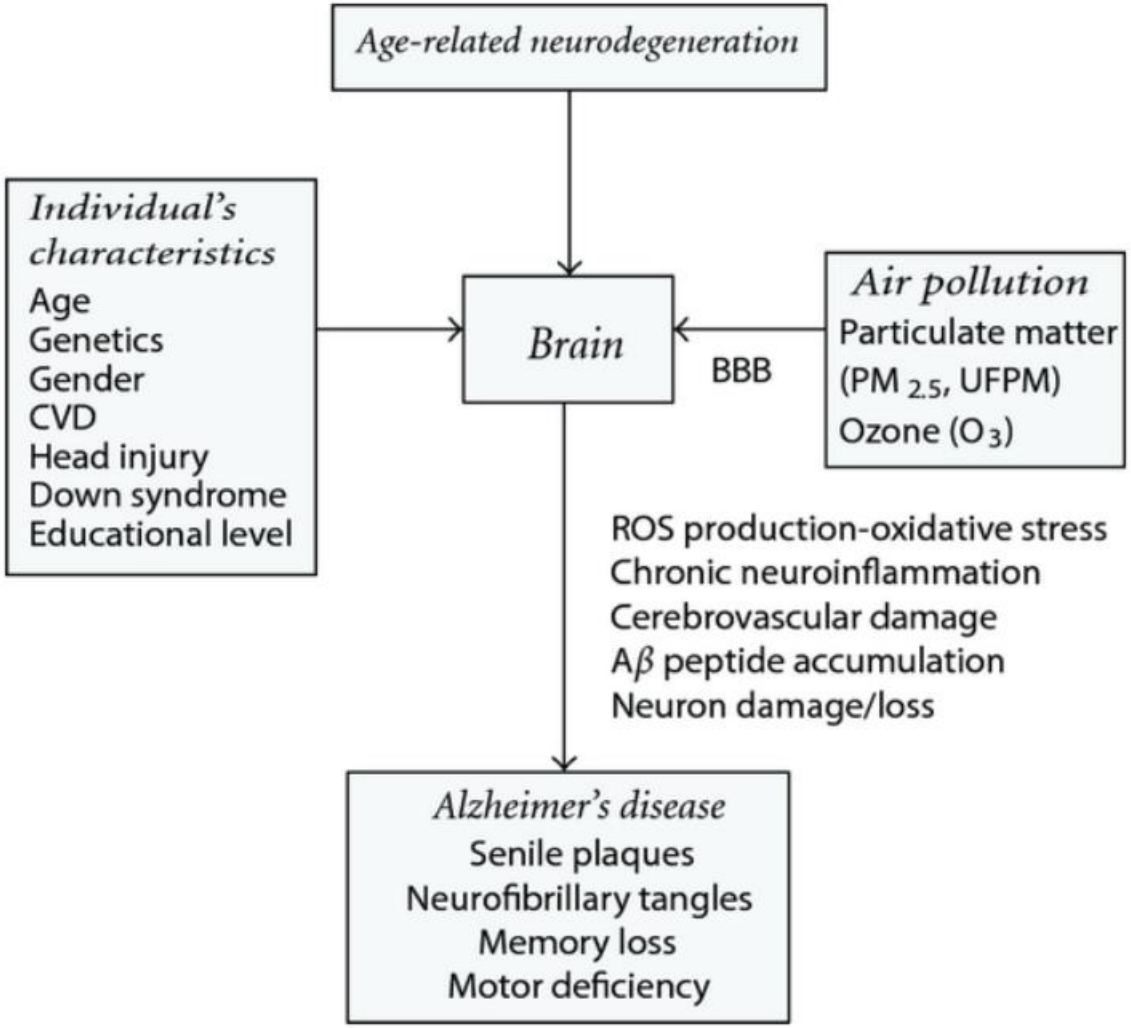

Figure 1: Interacting factors in the causality of Alzheimer's disease [1, 4–11, 17, 19, 28, 35, 72, 73]. Alzheimer's disease is the result of the interaction of aging, genetic predisposition, and environmental exposures such as air pollution in the etiology and pathogenesis of the disease. Air pollution 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.

Valencia Moulton P, Yang W, JEPH, 2012

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Pollution atmosphérique et autisme

Residential Proximity to Freeways and Autism in the CHARGE Study

Heather E. Volk,[1] Irva Hertz-Picciotto,[2] Lora Delwiche,[2] Fred Lurmann,[3] and Rob McConnell[4]

[1]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; [2]Department of Public Health Sciences, University of California–Davis, Davis, California, USA; [3]Sonoma Technology Inc., Petaluma, California, USA; [4]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 sociodemo
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 associated 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[1,2,3], **Tara Kerin**[1], **Fred Lurmann**[4], **Irva Hertz-Picciotto**[5], **Rob McConnell**[1], and **Daniel B. Campbell**[3,6,7]

[1]Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA

[2]Department of Pediatrics, Children's Hospital Los Angeles, University of Southern California, Los Angeles, CA

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Effets sur l'obésité

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

ehp ENVIRONMENTAL HEALTH PERSPECTIVES

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Current Issue

CHILDREN'S HEALTH ADVANCE PUBLICATION

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,[1] Ernest Shen,[1] Frank D. Gilliland,[1] Michael Jerrett,[2] Jennifer Wolch,[3] Chih-Chieh Chang,[1] Frederick Lurmann,[4] and Kiros Berhane[1]

Author Affiliations close

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

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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

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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

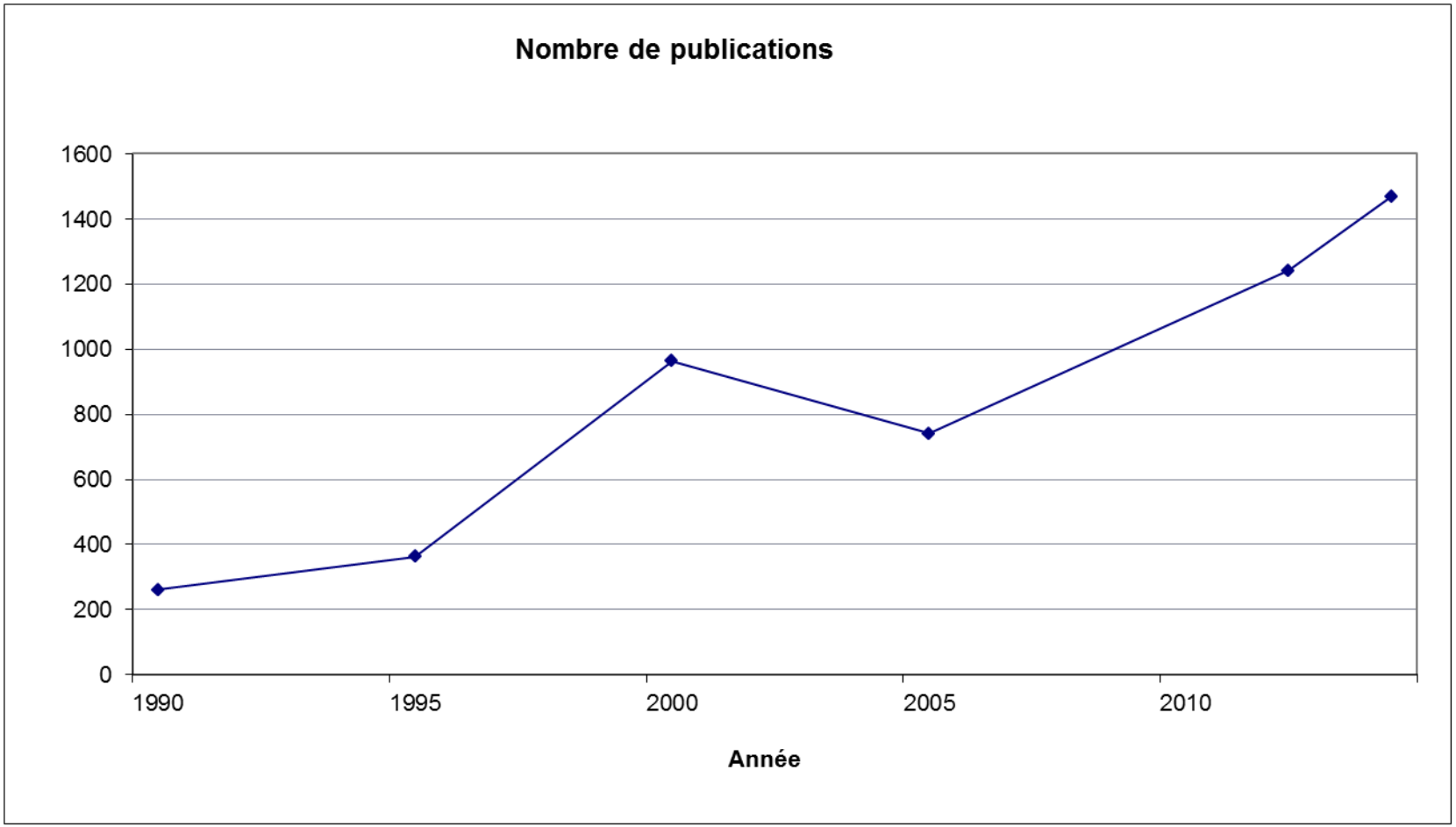
Ozone

- 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 »

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Quelles conséquences en termes de santé publique ?

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Dernières estimations de l'OMS au niveau mondial

mars 2014

Organisation mondiale de la Santé

nées et stiques | 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 mondia – 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.

Organisation mondiale de la Santé

Centre des médias | Publications | Pays | Programmes et projets | À propos de l' OMS

Centre des médias

Qualité de l'air ambiant (extérieur) et santé

Aide-mémoire N°313

Mars 2014

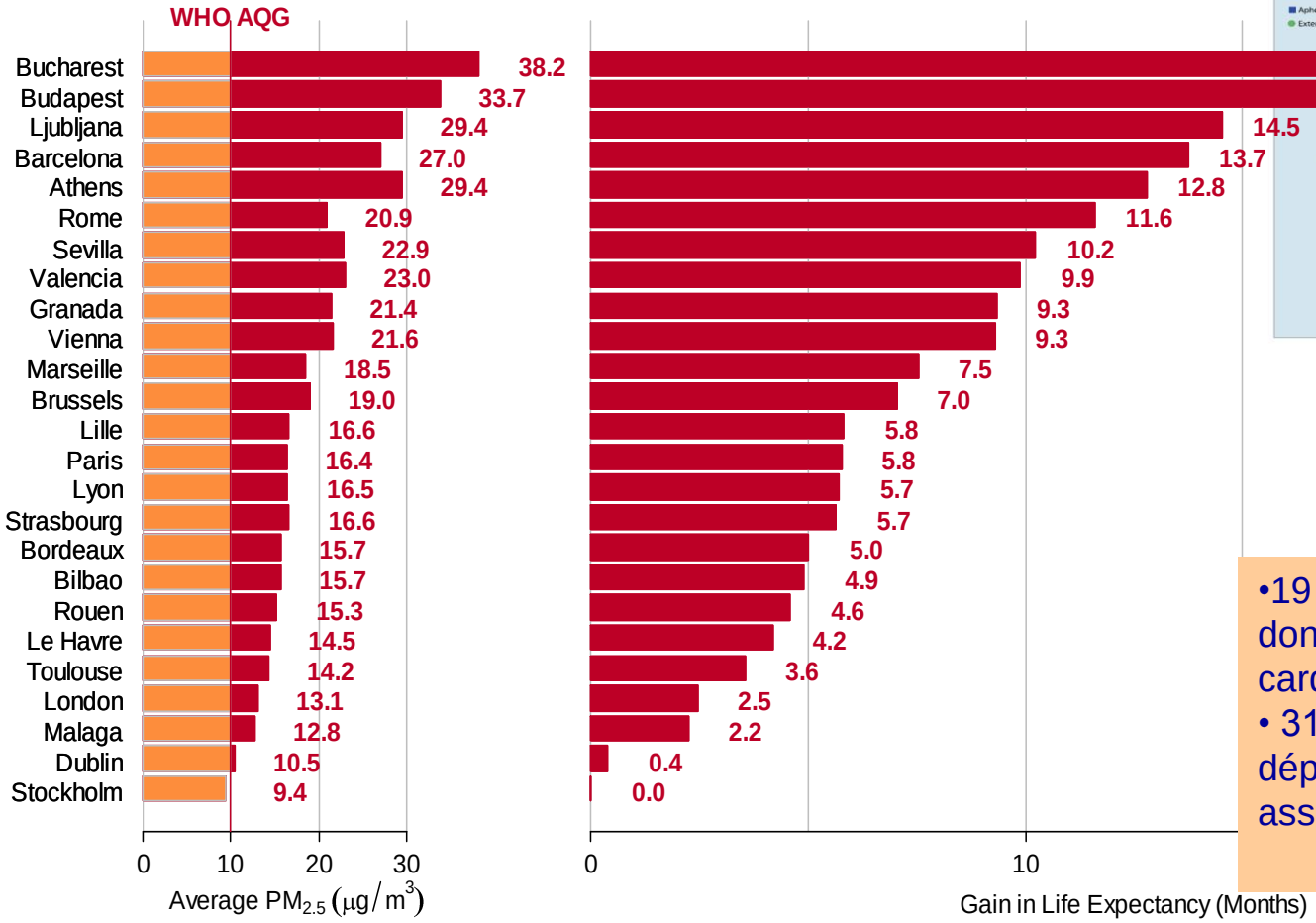
Principaux faits

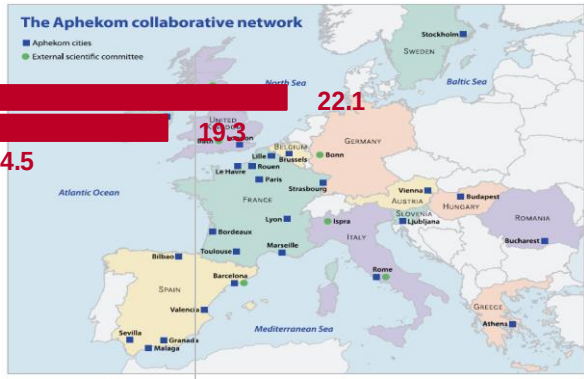
- La pollution de l'air représente un risque environnemental majeur pour la santé. En diminuant les niveaux de pollution atmosphérique, les pays peuvent réduire la charge de morbidité imputable aux accidents vasculaires cérébraux, aux cardiopathies, au cancer du poumon et aux affections respiratoires, 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 $PM_{2.5}$ respectaient la valeur OMS de 10 µg/m^3





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

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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)

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-Texier, 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

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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

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Episodes de pollution

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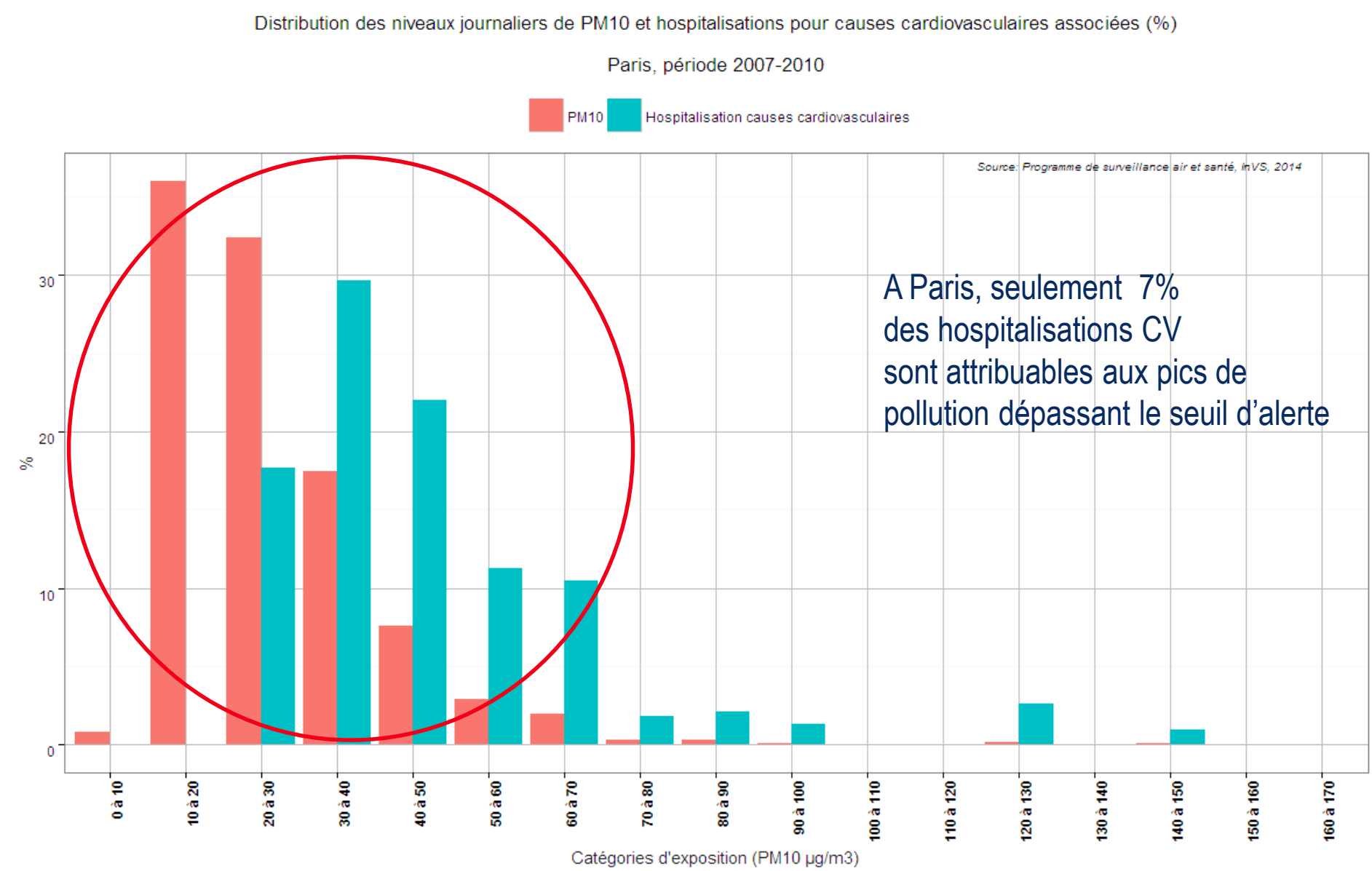
Episodes de pollution

- Météo favorable à une accumulation des polluants atmosphériques

- Seuils réglementaires (PM_{10}) :
  - Information : 50 µg/m^3 (en moyenne journalière)
  - Alerte : 80 µg/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 entrainer le décès

Poids relatif des épisodes de pollution sur la santé



A Paris, seulement 7%
des hospitalisations CV
sont attribuables aux pics de
pollution dépassant le seuil d'alerte

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)

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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*)

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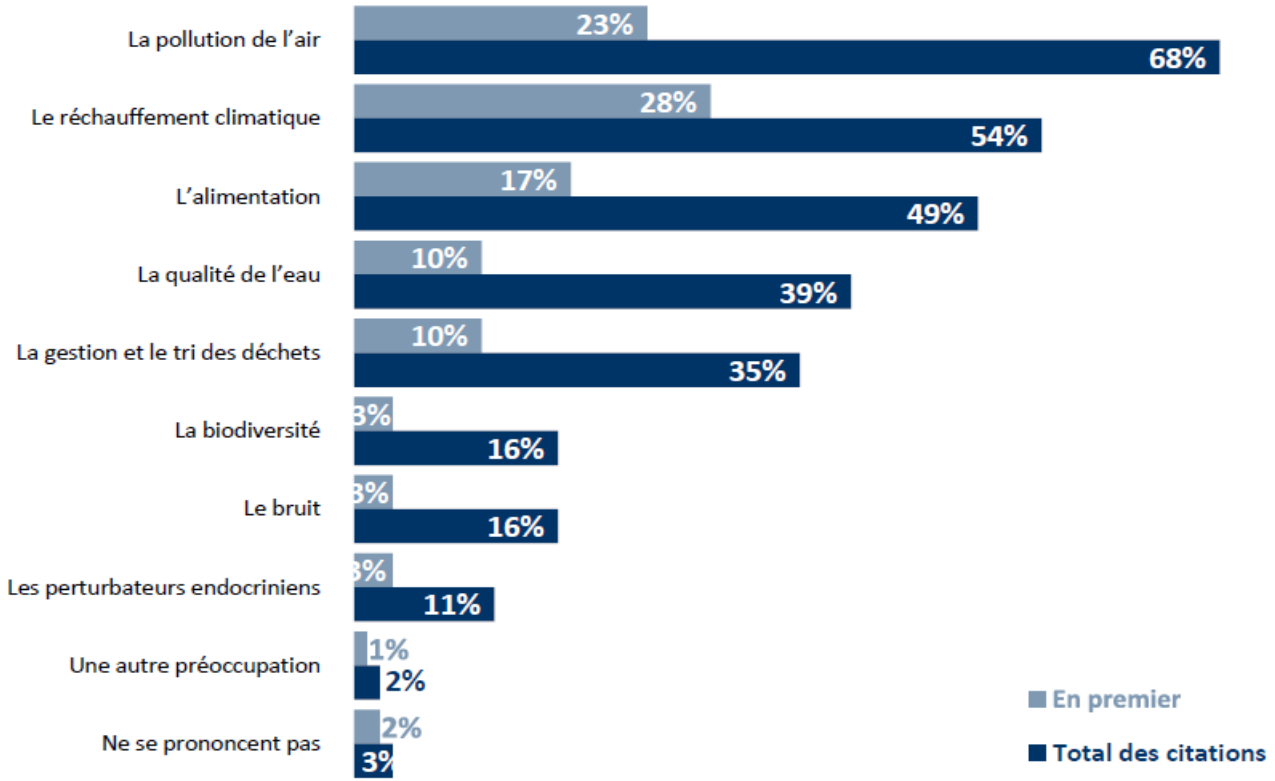
Que pense la population ?

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 S(
ENVIRONMENTAL EPIDE[
Website: www.ise

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

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

Dear Minister Touraine,

The European Chapter of the International Socie
you regarding the French National Plan on En
consultation, and in particular regarding the effort

INTERNATIONAL SOCIETY FOR ENVIRONMENTAL EPIDEMIOLOGY-EUROPE
Website: www.iseepi.org

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)

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 Royal,

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 ($PM_{2.5}$). As you know, the current EU regulation, with a yearly limit of 25 micrograms/m^3 for $PM_{2.5}$, is twice as high as the current regulation in the USA (set to 12 micrograms/m^3) and two and a half times as high as the value of 10 micrograms/m^3 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 ?

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Merci

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