

Contaminación Atmosférica y Salud

Líneas de investigación y minimización de impactos

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Ministerio de Economía y Competitividad

La contaminación del aire produce **más de 9 millones de muertes prematuras al año en todo el mundo**. El doble de la mortalidad global atribuible a malnutrición materno-filial y la mitad de la atribuible al tabaco (The Lancet, 2012).

LA SALUD Y LA CARGA DE MORBILIDAD PROVOCADA
POR LA CONTAMINACIÓN DEL AIRE



de todas las muertes
por cáncer de pulmón



de las muertes por
enfermedad pulmonar
obstructiva crónica (EPOC)



de las muertes por
accidente cerebrovascular



de las muertes por
cardiopatía

»El impacto supera los 3,7 billones de euros al año (6,2% de la riqueza del planeta)

CIUDADES QUE EXCEDEN LOS LÍMITES ESTABLECIDOS EN LAS DIRECTRICES DE LA OMS SOBRE INOCUIDAD DEL AIRE*

60%

Más del 60% de las ciudades y municipios de los países europeos de ingresos altos

90%

Más del 90% de las ciudades y municipios de los países de ingresos bajos y medianos de Europa, América Latina, Asia y Oriente Medio

35%

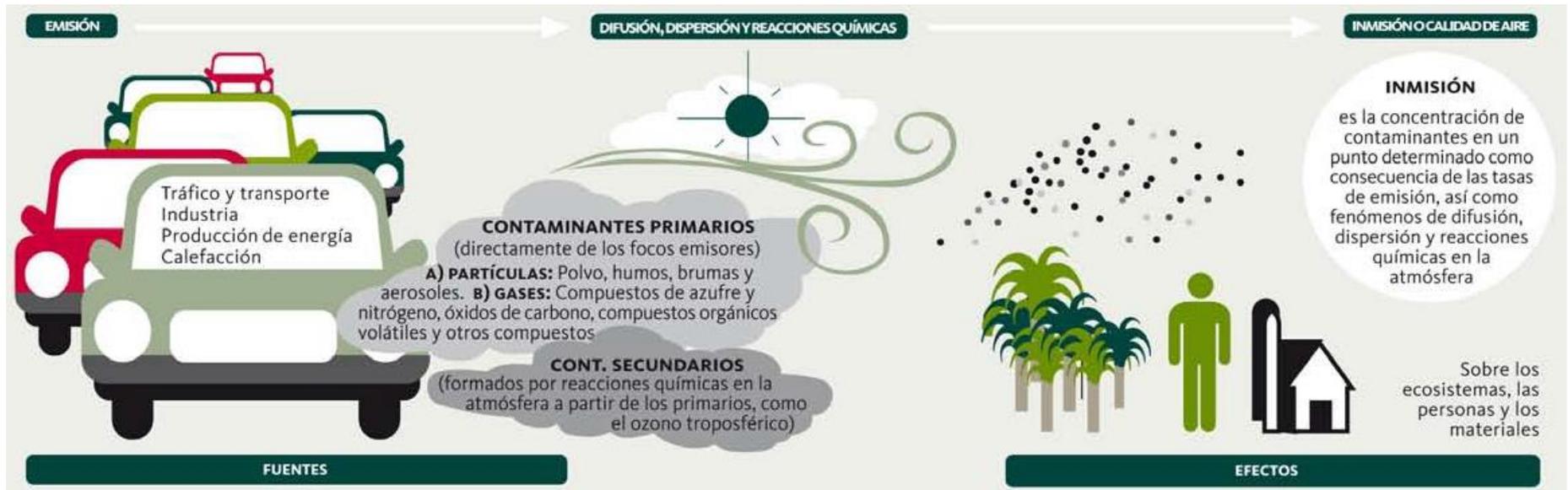
Más del 35% de las ciudades y municipios de los países de ingresos altos de Asia

17%

El 17% de las ciudades y municipios de América del Norte

*De las 3000 ciudades y municipios que monitorean y comunican sus niveles de contaminación del aire

Cada día una persona respira de media un volumen de 12.000 litros de aire... que no elige



EFFECTOS RESPIRATORIOS



Síntomas

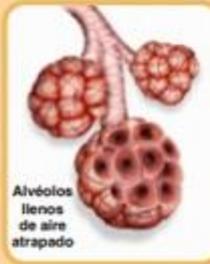
- Tos
- Respiración sibilante
- Flema
- Falta de aire
- Opresión en el pecho

Aumento de enfermedades y muerte prematura causado por:

- Asma
- Bronquitis (aguda o crónica)
- Enfisema
- Neumonía

Desarrollo de otras enfermedades

- Bronquitis crónica
- Envejecimiento prematuro de los pulmones



Alvéolos llenos de aire atrapado

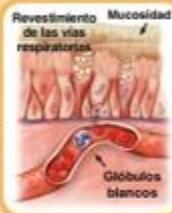
Cómo los contaminantes causan síntomas

Efectos en la función pulmonar

- Estrechamiento de las vías respiratorias (broncoconstricción)
- Reducción del flujo de aire

Inflamación de las vías respiratorias

- Afluencia de glóbulos blancos
- Producción anormal de mucosidad
- Acumulación de líquido e hinchazón (edema)
- Muerte y eliminación de las células que revisten las vías respiratorias



Mayor susceptibilidad a infección respiratoria



Normal



Pulmón con infección

EFFECTOS CARDIOVASCULARES



Síntomas:

- Opresión en el pecho
- Dolor de pecho (angina de pecho)
- Palpitaciones
- Falta de aire
- Fatiga inusual

Aumento de enfermedades y muerte prematura causado por:

- Enfermedad de las arterias coronarias
- Ritmos cardíacos anormales
- Insuficiencia cardíaca congestiva

Cómo los contaminantes pueden causar síntomas

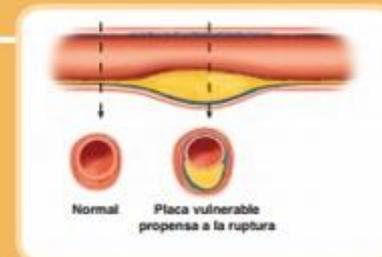


Efectos en la función cardiovascular

- Baja oxigenación de los glóbulos rojos
- Ritmos cardíacos anormales
- Alteración de la actividad cardíaca controlada por el sistema nervioso autónomo

Inflamación vascular

- Mayor riesgo de formación de coágulos
- Estrechamiento de los vasos sanguíneos (vasoconstricción)
- Mayor riesgo de ruptura de la placa aterosclerótica



Normal Placa vulnerable propensa a la ruptura

Gradiente de efectos en la salud

Rango de efectos que pueden producirse por la exposición a la contaminación atmosférica y número de personas afectadas por estos efectos.



Una gran proporción de la población experimenta estos efectos

Gravedad de los efectos sobre la salud

Una proporción más pequeña de la población experimenta estos efectos



Grupos más vulnerables a la contaminación atmosférica

- Niños
- Enfermos crónicos: diabetes, patologías de pulmón y corazón
- Ancianos
- Bajo nivel educativo/socioeconómico



- » Asociada a síntomas respiratorios (tos, picor, irritación de las vías respiratorias)

- » Exacerbación de enfermedades respiratorias
 - Aumento del riesgo **infección pulmonar** : Afecta a la función de los macrófagos alveolares y su capacidad de expeler agentes patógenos.
 - ✓ Empeoramiento de Neumonías / Bronquitis



Fig. 2. The synergistic pathological and immunological interactions between air pollutants and viral infections

Effect	Pollutant	Infection
Bronchoconstriction	+	+++
Bronchial hyperresponsiveness	+	+++
Inflammatory mediator release	++	+++
Ciliary dyskinesia	++	+++
Inflammatory cell activation	++	+++
Epithelial damage	++	++/±
T lymphocyte function	++	+++
Alveolar macrophage function	+++	++
Interaction with allergens	++	+++
↑ Epithelial-derived cytokines	++	+++
↓ Macrophage-derived cytokines	++	+

'mild'	+
'moderate'	++
'severe'	+++

Reproduced by permission from Chauhan & Johnston (4).

» Asociada a síntomas respiratorios
(tos, picor, irritación de las vías respiratorias)

» Exacerbación de enfermedades respiratorias

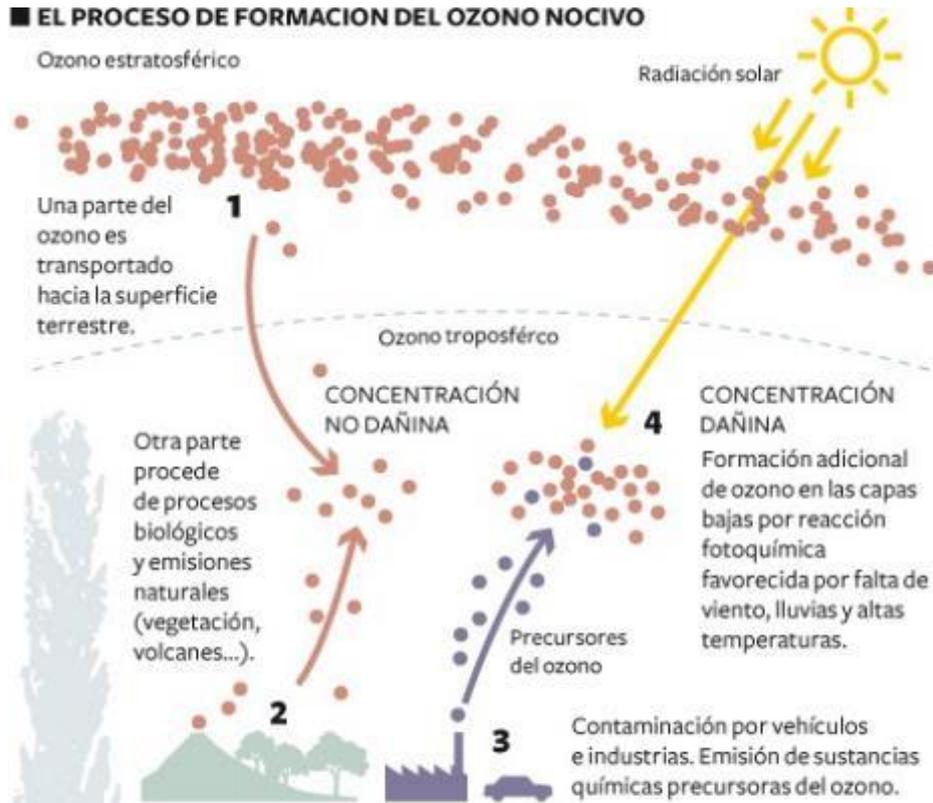
- Aumento del riesgo **infección pulmonar** : Afecta a la función de los macrófagos alveolares y su capacidad de expeler agentes patógenos.
 - ✓ Empeoramiento de Neumonías / Bronquitis
- Aumenta la sensibilidad al alérgeno y la respuesta inflamatoria
 - ✓ Empeoramiento del Asma
- ❖ Deterioro de la función pulmonar
- ❖ Enfermedades crónicas (EPOC)

» **Precursores del ozono troposférico**

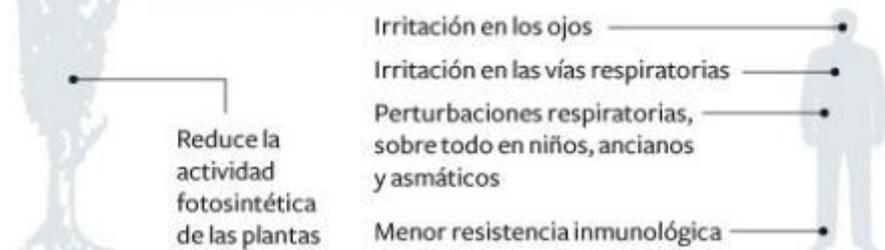


Ozono Troposférico (O3)

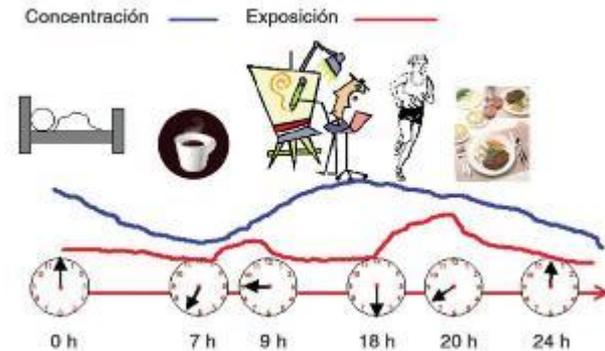
EL PROCESO DE FORMACION DEL OZONO NOCIVO



EFFECTOS NEGATIVOS



EXPOSICIÓN DE LA POBLACIÓN AL O₃



Contaminante secundario:



Máximo en la periferia de las ciudades

Morbi-mortalidad & Material Particulado

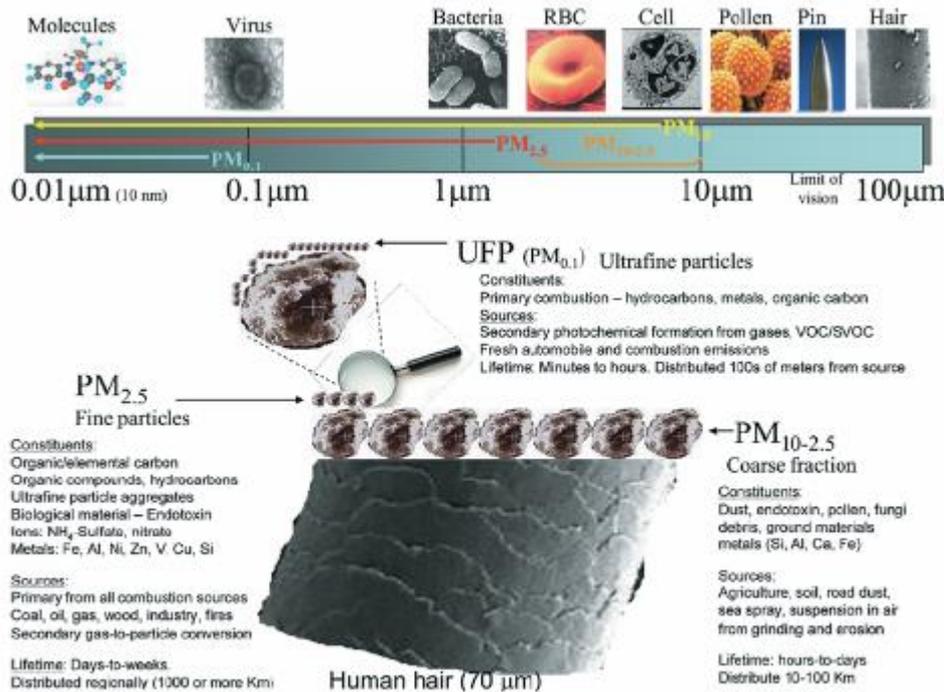
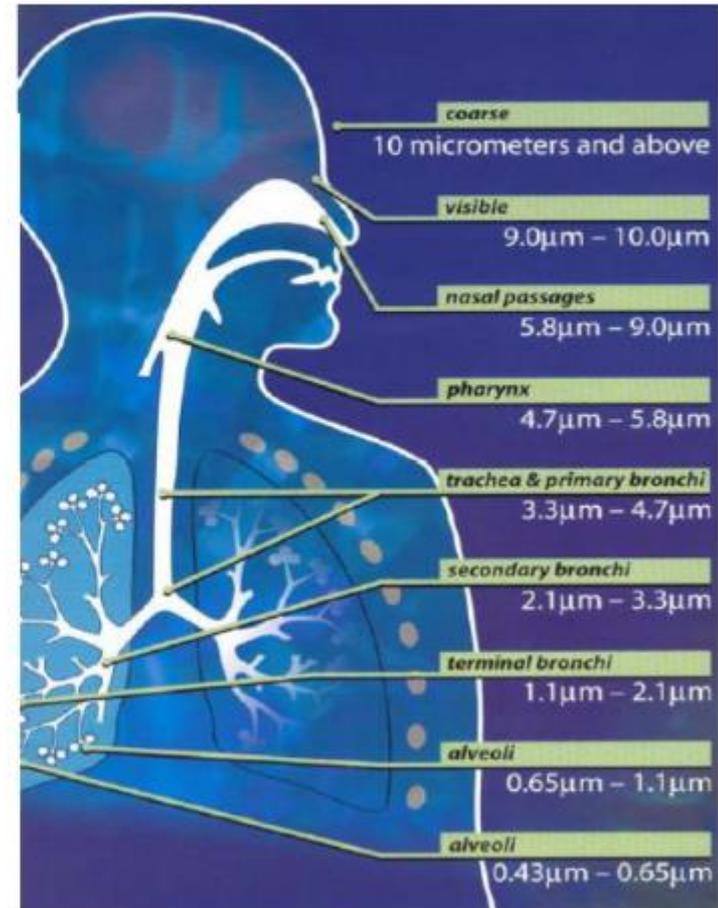


Figure 1 Size, sources and composition of PM air pollution

RBC, red blood cell; SVOC, semi-volatile organic carbons; UFP, ultra-fine particles; VOC, volatile organic carbons.





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IARC Scientific Publication No. 161
Air Pollution and Cancer
(ePUB format)



IARC SCIENTIFIC PUBLICATION NO. 161: AIR POLLUTION AND CANCER

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La OMS clasifica la contaminación del aire como causa de cáncer

La Agencia de Investigación del Cáncer incluye la polución atmosférica en la lista de agentes con efectos cancerígenos demostrados | En 2010 hubo 223.000 muertes por cáncer de pulmón en todo el mundo por la polución

		Animals			
		Sufficient	Limited	Inadequate	Suggesting lack
Humans	Sufficient	1	1	1	1
	Limited	2A (1)	2B (2A)	2B (2A)	2B (2A)
	Inadequate	2B (1, 2A, 3)	3 (2B)	3	3 (4)
	Suggesting lack	3 (1)	3	3	4

- IARC has classified outdoor air pollution as *carcinogenic to humans* (Group 1).
- There is *sufficient evidence* that exposure to outdoor air pollution causes **lung cancer** (Group 1).
- Particulate matter was evaluated separately and was also classified as *carcinogenic to humans* (Group 1).



IMPACTO EN SALUD ATRIBUIBLE

Contaminación Atmosférica



La contaminación ha matado a 93.000 personas en España en una década

Investigadores de la Escuela Nacional de Sanidad ponen cifras al impacto de las emisiones de los coches en las ciudades

MANUEL ANSEDE

22 JUN 2018 - 10:55 CEST



Un panel luminoso en la M-30 indica la activación del protocolo anticontaminación en Madrid. VÍCTOR SAINZ

IN ENGLISH

Pollution has killed 93,000 people in Spain in the last decade

“Hay que restringir el tráfico”, sentencia la bióloga [Cristina Linares](#), investigadora de la Escuela Nacional de Sanidad, en Madrid. No es una propuesta, sino un llamamiento desesperado a la acción. El equipo de Linares acaba de calcular que la [contaminación atmosférica](#) ha provocado la muerte prematura de unas 93.000 personas en España a lo largo de una década. Son los primeros estudios con datos de todas las provincias españolas.



Evaluation of short-term mortality attributable to particulate matter pollution in Spain



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ABSTRACT

According to the WHO, 3 million deaths are attributable to air pollution due to particulate matter (PM) world-wide. However, there are no specific updated studies which calculate short-term PM-related cause specific mortality in Spain. The objective is to quantify the relative risks (RRs) and attributable risks (ARs) of daily mortality associated with PM₁₀ concentrations, registered in Spanish provinces and to calculate the number of PM-related deaths. We calculated daily mortality due to natural (ICD-10: A00 R99), circulatory (ICD-10: I00 I99) and respiratory causes (ICD-10: J00 J99) for each province across the period 2000–2009. Mean daily concentrations of PM₁₀, NO₂ and O₃ was used. For the estimate of RRs and ARs, we used generalised linear models with a Poisson link. A meta-analysis was used to estimate RRs and ARs in the provinces with statistically significant results. The overall RRs obtained for these provinces, corresponding to increases of 10 µg/m³ in PM₁₀ concentrations were 1.009 (95% CI: 1.006 1.011) for natural, 1.026 (95% CI: 1.019 1.033) for respiratory, and 1.009 (95% CI: 1.006 1.012) for circulatory-cause mortality. This amounted to an annual overall total of 2683 deaths (95% CI: 852 4354) due to natural, 651 (95% CI: 359 1026) due to respiratory, and 556 (95% CI: 116 1012) due to circulatory causes, with 90% of this mortality lying below the WHO guideline values. This study provides an updated estimate of the effect had by this type of pollutant on causes of mortality, and constitutes an important basis for reinforcing public health measures.

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1. Introduction

Particulate matter (PM) consists of a complex mix of solid and liquid particles of organic and inorganic substances suspended in the air. The main components of PM are sulphates, nitrates, ammonium, sodium chloride, “black carbon”, mineral dust, organic matter and water (Querol et al., 2012). The respirable particles, PM₁₀ (aerodynamic diameter of less than 10 microns) and PM_{2.5} (aerodynamic diameter of less than 2.5 microns), are the types of PM which have the greatest health impact (WHO, 2013). According to a recent WHO study (WHO, 2016), in 2012 some 3 million deaths world-wide were estimated to be attributable to PM-related air pollution, with 193,000 of these occurring in Europe and 7000 in Spain.

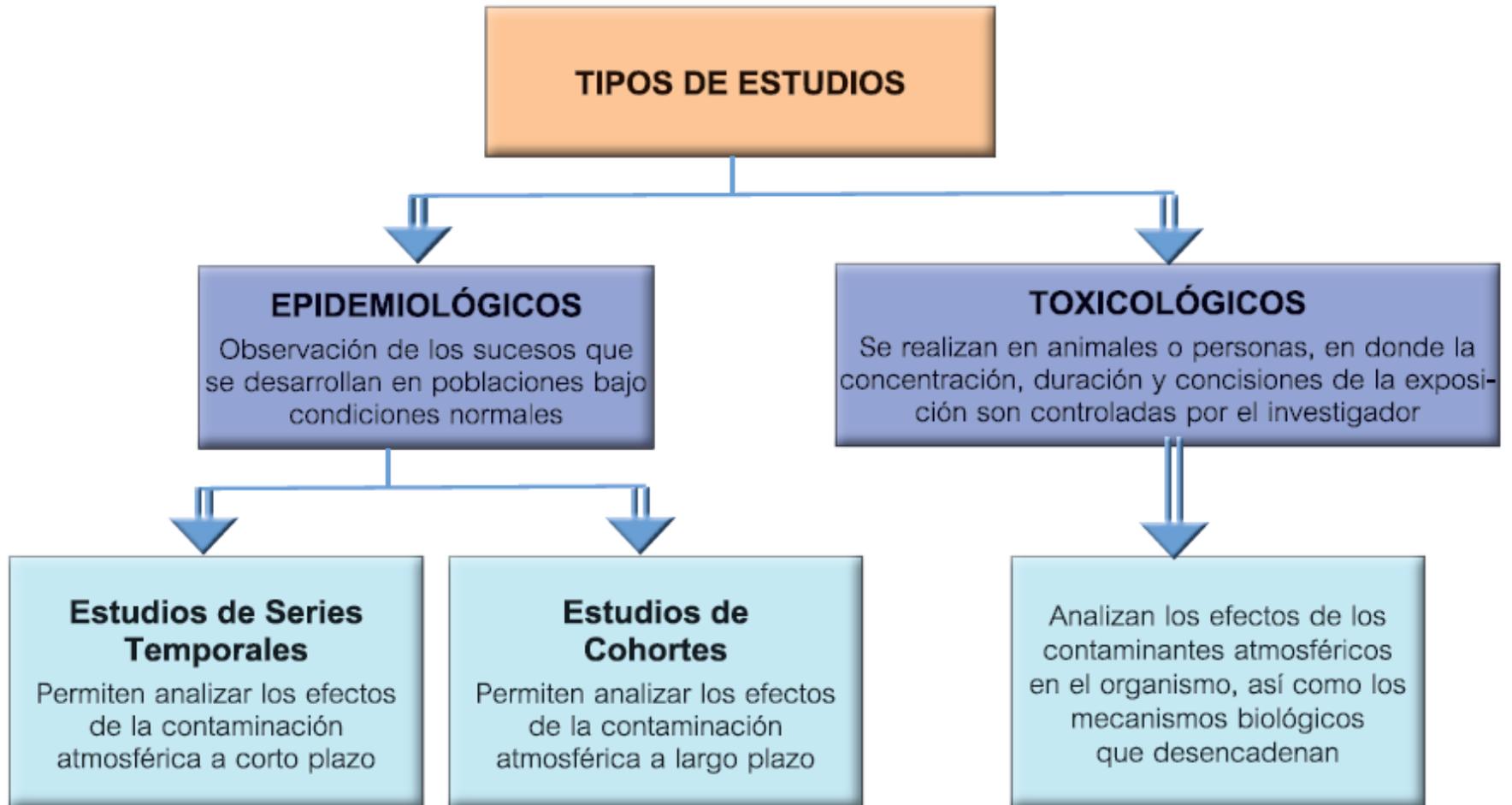
The health effects of PM are especially well documented, with a distinction being drawn between two types of effects, short- and

long-term. Cohort studies designed to detect the long-term effects on population health, link exposure to PM to an increased risk of death (Dockery et al., 1993; Pope et al., 1995; Miller et al., 2007; Beelen et al., 2008a; Ostro et al., 2010), even for very low PM_{2.5} concentrations (Crouse et al., 2012). Although the principal causes of mortality associated with long-term exposure to PM are some types of cancer (Beelen et al., 2008b), recently the International Agency for Research on Cancer (IARC) classified PM_{2.5} as a carcinogen (Loomis et al., 2013); equally notable are its effects on cardiovascular (Brook et al., 2010; Dominici et al., 2006) and respiratory causes (Dominici et al., 2006; Guaita et al., 2011; Kim et al., 2012), with clearly established physiopathological mechanisms (Brook et al., 2010; Rückerl et al., 2011). Recent studies suggest other types of health outcomes, in which PM is associated with other types of diseases (Rückerl et al., 2011). Hence, PM has been found to have an effect on diabetes (Brook et al., 2008), neurological development in children (Freire et al., 2010) and neurological disorders in adults (Ranft et al., 2009).

There are also numerous studies which associate short-term exposure to PM –both PM₁₀ and PM_{2.5}– with morbidity and

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Estudios Epidemiológicos



Mortalidad anual a corto plazo atribuible Contaminación Atmosférica Química España (2000-2009)

	Naturales (IC 95%)	Respiratorias (IC 95%)	Circulatorias (IC 95%)
PM10	2683 (852 4354)	651 (358 1026)	556 (116 1012)
NO2	6085 (3371 9180)	997 (460 1524)	1977 (828 3197)
O3	499 (277 717)	126 (54 194)	167 (39 292)
Total	9267 (4500 14251)	1774 (872 2747)	2700 (983 4501)

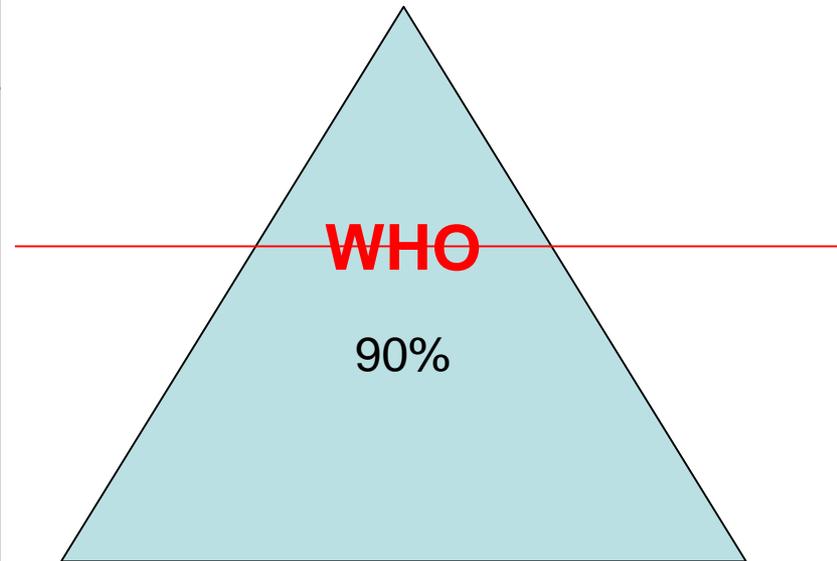
OMS: 7.000 muertes anuales

Mortalidad anual tabaco: 50.000

Mortalidad accidentes tráfico en 2015: 1.126

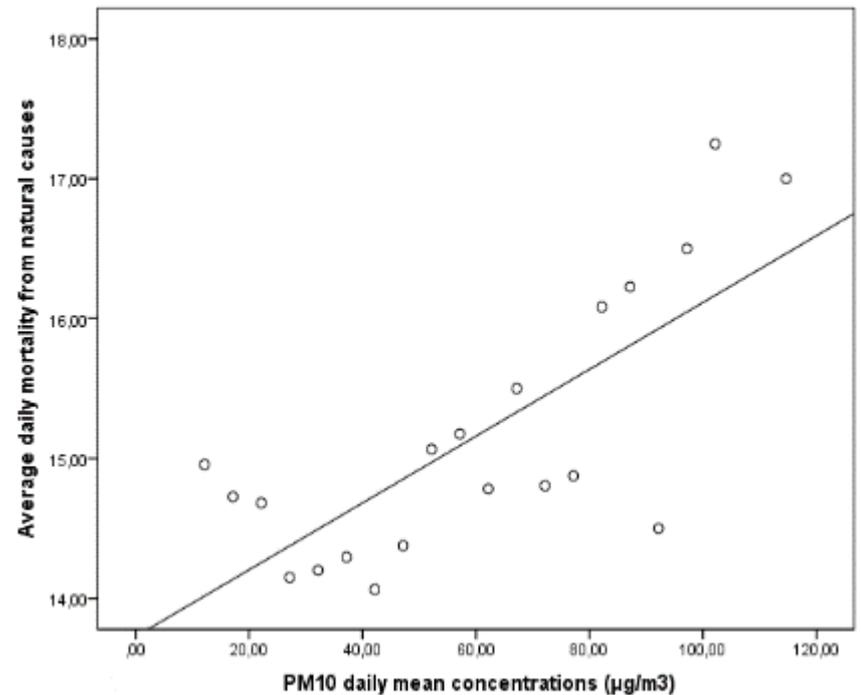
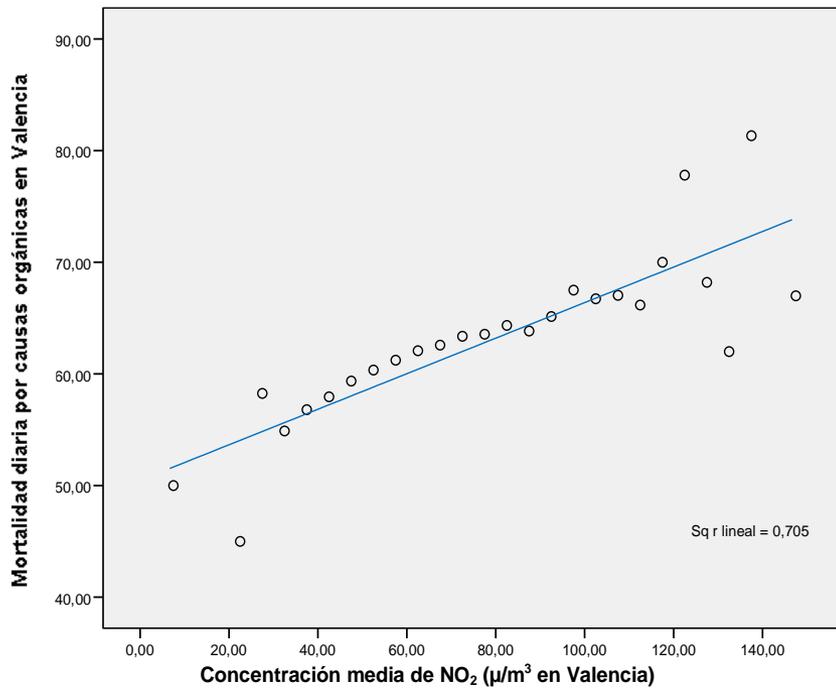
NO₂ 1,8% / PM 1% / O₃ 0,2%

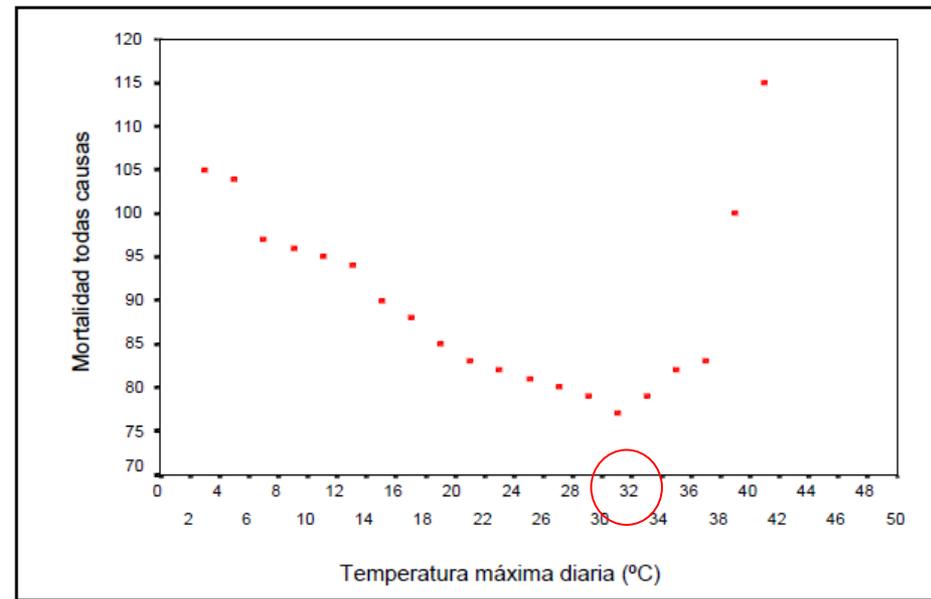
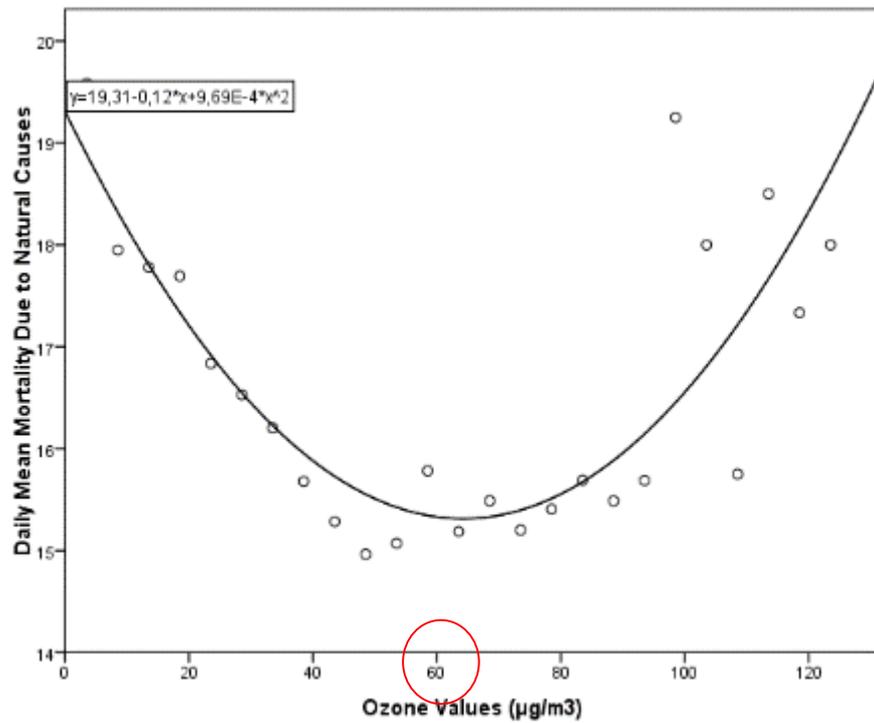
City	Mortality Attributable without threshold OMS*	IC 95% Mortality Attributable without threshold WHO*	Mortality Attributable with threshold WHO*	IC 95% Mortality Attributable with threshold WHO*	
España	26830	(8525 - 43538)	2292	(882 - 3820)	
Natural Causes	Albacete	975	(79 - 1864)	119	(10 - 278)
	Bilbao	1551	(553 - 2544)	107	(43 - 267)
	Córdoba	2790	(1608 - 3967)	469	(270 - 667)
	Guadalajara	1328	(905 - 1748)	84	(57 - 111)
	León	1247	(37 - 2449)	96	(3 - 189)
	Madrid	2798	(771 - 4818)	155	(43 - 267)
	Oviedo	2963	(1221 - 4699)	541	(223 - 858)
	Pamplona	1523	(550 - 2489)	74	(27 - 121)
	Salamanca	1244	(278 - 2202)	50	(11 - 88)
	Santander	2116	(417 - 3304)	92	(18 - 143)
	San Sebastián	3110	(2074 - 4138)	134	(89 - 178)
	Toledo	1109	(158 - 2055)	106	(15 - 196)
	Valencia	1858	(252 - 3457)	50	(7 - 93)
	Vitoria	488	(32 - 940)	29	(2 - 55)
Zaragoza	1729	(590 - 2864)	186	(64 - 309)	
España	6509	(3581 - 10256)	499	(266 - 781)	
Respiratory Causes	Bilbao	362	(69 - 651)	26	(5 - 47)
	Córdoba	835	(412 - 1253)	145	(72 - 218)
	Guadalajara	272	(132 - 410)	21	(10 - 32)
	Madrid	1720	(1460 - 2845)	100	(85 - 166)
	Santander	963	(548 - 1371)	48	(27 - 68)
	Sevilla	785	(271 - 1292)	61	(21 - 100)
	San Sebastián	774	(462 - 1080)	39	(23 - 54)
	Toledo	551	(224 - 872)	54	(22 - 85)
	Zamora	247	(3 - 482)	5	(1 - 11)
España	5558	(1159 - 10123)	472	(116 - 827)	
Circulatory Causes	Bilbao	777	(249 - 1300)	53	(17 - 88)
	Madrid	717	(203 - 1231)	42	(12 - 72)
	Oviedo	1173	(380 - 1962)	218	(71 - 365)
	Pamplona	626	(98 - 1149)	29	(5 - 54)
	Salamanca	635	(165 - 1100)	26	(7 - 45)
	Santander	619	(15 - 1217)	28	(1 - 55)
	San Sebastián	562	(26 - 1091)	24	(1 - 46)
Toledo	449	(23 - 1073)	52	(2 - 102)	



Mortalidad atribuible a las concentraciones de PM₁₀ por causas naturales, respiratorias y circulatorias según capitales de provincia y toda España en el periodo de estudio 2000-2009. En el supuesto de una relación sin umbral y una relación con efecto a partir del valor límite de la WHO (*limit value = 50 µg/m³ diario).

Diagrama de dispersión entre la concentración media de NO_2 y PM_{10} ($\mu\text{g}/\text{m}^3$) y la mortalidad por todas las causas durante el período 2000-2009





EVOLUCIÓN DE Líneas De Investigación EN EFECTOS EN SALUD ATRIBUIBLES A LA CONTAMINACIÓN ATMOSFÉRICA



POBLACIÓN ADULTA

- DIFERENTES TIPOS DE CÁNCER

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Research Article

Cancer
Epidemiology,
Biomarkers
& Prevention

Cancer Mortality Risks from Long-term Exposure to Ambient Fine Particle

Chit Ming Wong¹, Hilda Tsang¹, Hak Kan Lai¹, G. Neil Thomas², Kin Bong Lam³, King Pan Chan¹, Qishi Zheng¹, Jon G. Ayres⁴, Siu Yin Lee⁵, Tai Hing Lam¹, and Thuan Quoc Thach¹

Abstract

Background: Few studies have assessed long-term effects of particulate matter (PM) with aerodynamic diameter < 2.5 μm ($\text{PM}_{2.5}$) on mortality for causes of cancer other than the lung; we assessed the effects on multiple causes. In Hong Kong, most people live and work in urban or suburban areas with high-rise buildings. This facilitates the estimation of $\text{PM}_{2.5}$ exposure of individuals, taking into account the height of residence above ground level for assessment of the long-term health effects with sufficient statistical power.

Methods: We recruited 66,820 persons who were ≥ 65 in 1998 to 2001 and followed up for mortality outcomes until 2011. Annual concentrations of PM at their residential addresses were estimated using $\text{PM}_{2.5}$ concentrations measured at fixed-site monitors, horizontal-vertical locations, and satellite data. We

used Cox regression model to assess the HR of mortality for cancer per 10 $\mu\text{g}/\text{m}^3$ increase of $\text{PM}_{2.5}$.

Results: $\text{PM}_{2.5}$ was associated with increased risk of mortality for all causes of cancer [HR, 1.22 (95% CI, 1.11–1.34)] and for specific cause of cancer in upper digestive tract [1.42 (1.06–1.89)], digestive accessory organs [1.35 (1.06–1.71)] in all subjects; breast [1.80 (1.26–2.55)] in females; and lung [1.36 (1.05–1.77)] in males.

Conclusions: Long-term exposures to $\text{PM}_{2.5}$ are associated with elevated risks of cancer in various organs.

Impact: This study is particularly timely in China, where compelling evidence is needed to support the pollution control policy to ameliorate the health damages associated with economic growth. *Cancer Epidemiol Biomarkers Prev*; 25(5); 839–45. ©2016 AACR.

POBLACIÓN ADULTA

• CÁNCER DE MAMA

Research

A Section 508-conformant HTML version of this article is available at <https://doi.org/10.1289/EHP1742>.

Long-Term Exposure to Ambient Air Pollution and Incidence of Postmenopausal Breast Cancer in 15 European Cohorts within the ESCAPE Project

Zorana J. Andersen,¹ Massimo Stafoggia,^{2,3} Gudrun Weinmayr,⁴ Marie Pedersen,^{1,5} Claudia Galassi,⁶ Jeanette T. Jørgensen,¹ Anna Oudin,⁷ Bertil Forsberg,⁷ David Olsson,⁷ Bente Oftedal,⁸ Gunn Marit Aasvang,⁸ Geir Aamodt,⁸ Andrei Pyko,³ Göran Pershagen,³ Michal Korek,³ Ulf De Faire,³ Nancy L. Pedersen,⁹ Claes-Göran Östenson,¹⁰ Laura Fratiglioni,¹¹ Kirsten T. Eriksen,⁵ Anne Tjønneland,⁵ Petra H. Peeters,^{12,13} Bas Bueno-de-Mesquita,^{13,14,15} Michelle Plusquin,¹³ Timothy J. Key,¹⁶ Andrea Jaensch,⁴ Gabriele Nagel,^{4,17} Alois Lang,¹⁸ Meng Wang,¹⁹ Ming-Yi Tsai,^{19,20,21} Agnes Fournier,^{22,23} Marie-Christine Boutron-Ruault,^{22,23} Laura Baglietto,^{22,23} Sara Grioni,²⁴ Alessandro Marcon,²⁵ Vittorio Krogh,²⁴ Fulvio Ricceri,^{6,26} Carlotta Sacerdote,⁶ Enrica Migliore,⁶ Ibon Tamayo-Uria,^{27,28,29} Pilar Amiano,^{28,30} Miren Dorronsoro,^{28,30} Roel Vermeulen,^{12,13,31} Ranjeet Sokhi,³² Menno Keuken,³³ Kees de Hoogh,^{20,21} Rob Beelen,^{31,34} Paolo Vineis,^{13,35} Giulia Cesaroni,² Bert Brunekreef,^{12,28} Gerard Hoek,^{12,31} and Ole Raaschou-Nielsen^{5,36}

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RESULTS: Of 74,750 postmenopausal women included in the study, 3,612 developed breast cancer during 991,353 person-years of follow-up. We found positive and statistically insignificant associations between breast cancer and PM_{2.5} (hazard ratio (HR) = 1.08 [95% confidence interval (CI): 0.77, 1.51] per 5 µg/m³), PM₁₀ [1.07 (95% CI: 0.89, 1.30) per 10 µg/m³], PM_{coarse} [1.20 (95% CI: 0.96, 1.49 per 5 µg/m³), and NO₂ [1.02 (95% CI: 0.98, 1.07 per 10 µg/m³), and a statistically significant association with NO_x [1.04 (95% CI: 1.00, 1.08) per 20 µg/m³, *p* = 0.04].

CONCLUSIONS: We found suggestive evidence of an association between ambient air pollution and incidence of postmenopausal breast cancer in European women. <https://doi.org/10.1289/EHP1742>

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³⁵Molecular and Epidemiology Unit, Human Genetics Foundation (HuGeF), Torino, Italy

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POBLACIÓN ADULTA

- **DIABETES**

Curr Environ Health Rep. 2014 September 1; 1(3): 275–286. doi:10.1007/s40572-014-0017-9.

Ambient Air Pollution and Type 2 Diabetes: A Systematic Review of Epidemiologic Research

Sung Kyun Park and Weiye Wang

Departments of Epidemiology and Environmental Health Sciences, University of Michigan, School of Public Health, Ann Arbor, MI, U.S.A

- **ANSIEDAD**

BMJ 2015;350:h1510 doi: 10.1136/bmj.h1510 (Published 24 March 2015)

Page 1 of 2



EDITORIALS

Air pollution, stroke, and anxiety

Particulate air pollution is an emerging risk factor for an increasing number of common conditions

Michael Brauer *professor*

School of Population and Public Health, University of British Columbia, Vancouver, BC, Canada

POBLACIÓN ADULTA

• ENFERMEDADES NEURODEGENERATIVAS

Living near major roads and the incidence of dementia, Parkinson's disease, and multiple sclerosis: a population-based cohort study

Hong Chen, Jeffrey C Kwong, Ray Copes, Karen Tu, Paul J Villeneuve, Aaron van Donkelaar, Perry Hystad, Randall V Martin, Brian J Murray, Barry Jessiman, Andrew S Wilton, Alexander Kopp, Richard T Burnett

Summary

Background Emerging evidence suggests that living near major roads might adversely affect health. However, little is known about its relationship with the incidence of dementia, Parkinson's disease, and multiple sclerosis. We sought to investigate the association between residential proximity to major roadways and the incidence of these neurological diseases in Ontario, Canada.

Methods In this population-based cohort study, we assembled two population-based cohorts: one of adults aged 20–50 years (about 4.4 million; multiple sclerosis cohort) and all adults aged 55–84 years (dementia or Parkinson's disease cohort) who resided in Ontario, Canada on April 1, 2001. We assessed each individual's proximity to major roadways based on their residential postal-code address at cohort inception. Incident diagnoses of dementia, Parkinson's disease, and multiple sclerosis were ascertained from provincial health administrative databases with validated algorithms. We assessed the association between traffic proximity and incident dementia, Parkinson's disease, and multiple sclerosis using Cox proportional hazards models, adjusting for individual and contextual factors such as diabetes, brain injury, and other factors. We did various sensitivity analyses, such as adjusting for access to neurologists and pollutants, and restricting to never movers and urban dwellers.

Findings Between 2001, and 2012, we identified 243 611 incident cases of dementia, 31 577 cases of Parkinson's disease, and 9247 cases of multiple sclerosis. The adjusted hazard ratio (HR) of incident dementia was 1.12 (95% CI 1.06–1.18) for people living less than 50 m from a major traffic road (95% CI 1.06–1.08), 1.04 (1.02–1.05) for 50–100 m, 1.00 (0.99–1.01) for 101–200 m, and 1.00 (0.99–1.01) for 201–300 m versus further than 300 m (*p* for trend = 0.001). These associations were robust to sensitivity analyses and seemed stronger among urban residents, especially those who lived in major cities (HR 1.12, 95% CI 1.10–1.14 for people living <50 m from a major traffic road), and who never moved (1.12, 95% CI 1.10–1.14 for people living <50 m from a major traffic road). No association was found with Parkinson's disease or multiple sclerosis.

NO₂ y enfermedad de Parkinson (EHP 2015)



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<http://www.ehponline.org>

Traffic-Related Air Pollution and Parkinson's Disease in
Denmark: A Case-Control Study

Beate Ritz, Pei-Chen Lee, Johnni Hansen,
Christina Funch Lassen, Matthias Ketznel, Mette Sørensen,
and Ole Raaschou-Nielsen

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• ENFERMEDADES NEURODEGENERATIVAS

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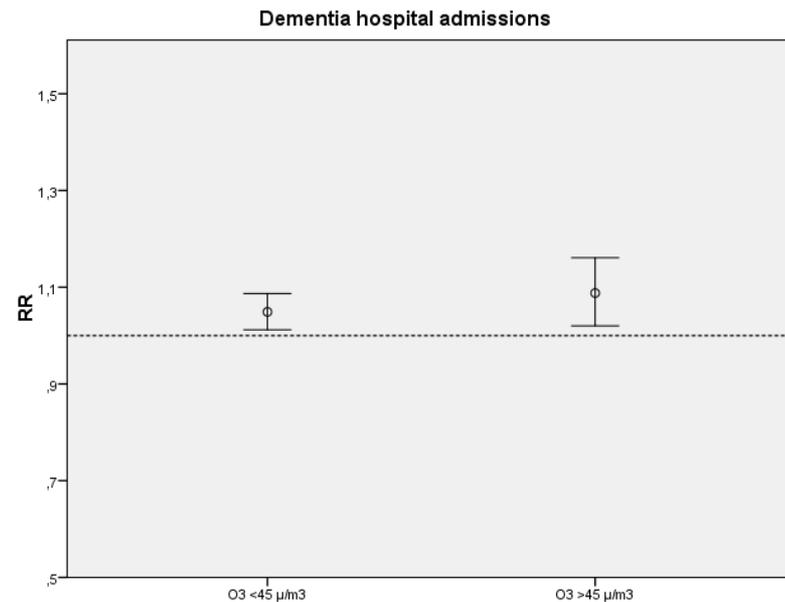
Short-term association between environmental factors and hospital admissions due to dementia in Madrid



C. Linares^a, D. Culqui^b, R. Carmona^a, C. Ortiz^a, J. Díaz^{a,*}

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POBLACIÓN ADULTA

• ENFERMEDADES NEURODEGENERATIVAS

Science of the Total Environment 592 (2017) 451–457



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Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv



Association between environmental factors and emergency hospital admissions due to Alzheimer's disease in Madrid



D R Culqui^{a,b,c,e}, C Linares^d, C Ortiz^d, R Carmona^d, J Díaz^d

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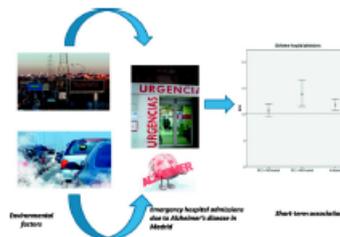
^c Evaluation and Intervention Methods Service from Barcelona Public Health Agency, Spain

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HIGHLIGHTS

- Alzheimer's disease (AD) is the most common cause of dementia among older adults
- Air pollutants may be risk factors regarding the decompensation of AD
- PM_{2.5} concentrations are associated with the development and the exacerbation of AD
- Heat waves can exacerbate Alzheimer's hospital admissions
- More epidemiologic studies will be needed to confirm the relation between AD and environmental factors

GRAPHICAL ABSTRACT



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ABSTRACT

Introduction: There are scarce studies of time series that analysed the short-term association between emergency hospital admissions due to Alzheimer's disease (AD) and environmental factors. The objective is to analyse the effect of heat waves, noise and air pollutants on urgent hospital admissions due to AD in Madrid.

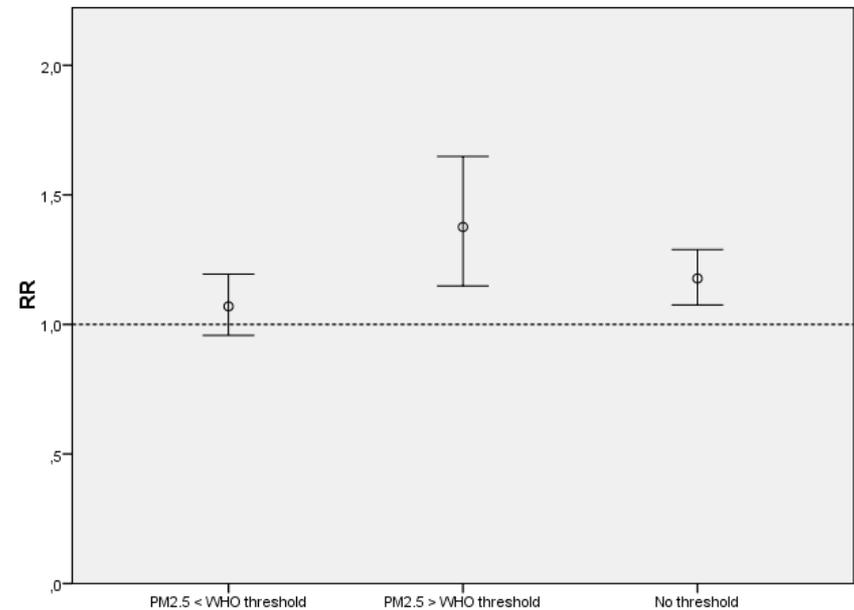
Methods: Longitudinal ecological time series study was performed. The dependent variable was the emergency AD hospital admissions occurred in Madrid during the period 2001–2009. Independent variables were: Daily mean concentrations (µg/m³) of air pollutants (PM_{2.5} and PM₁₀; O₃ and NO₂); maximum daily temperature (°C) and daily and night noise levels (dB(A)). Relative Risk (RR) for an increment in interquartile range, and Attributable Risk (AR) values were calculated through GIM with Poisson link.

Results: Our findings indicated that only PM_{2.5} concentrations at lag 2 with a RR: 1.38 (95% CI: 1.15–1.65); AR 27.5% (95% CI: 13.0–39.4); and heat wave days at lag 3 with a RR: 1.30 (95% CI: 1.12–1.52); AR 23.1% (95% CI: 10.7–34.2) were associated with AD hospital admissions.

Conclusion: A reduction in AD patients' exposure levels to PM_{2.5} and special care of such patients during heat wave periods could result in a decrease in both emergency AD admissions and the related health care costs.

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Alzheimer hospital admissions



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El CREAL Programas de investigación Publicaciones Docencia Asesoramiento

Portada > Noticias

NOTICIAS

Los niños que van a escuelas cercanas al tráfico muestran un menor desarrollo cognitivo

Nota de prensa

miércoles, 4 de marzo de 2015



El desarrollo cognitivo de los niños expuestos a la contaminación del aire cerca de las escuelas, según un estudio publicado en *Environmental Health Perspectives*.

Muchas escuelas están sufriendo con picos de contaminación del aire, un objetivo de la investigación es estudiar si la exposición a los niveles de contaminación asociada con el desarrollo cognitivo.

Se sospechaba que la exposición a la contaminación del aire en estudios previos de partículas ultrafinas procedentes de la combustión de combustibles fósiles expresaba una expresión elevada de comportamiento anómalo.

La exposición al aire contaminado relacionado con el tráfico y el desarrollo del cerebro se desarrolla rápidamente, se ha relacionado con funciones ejecutivas, como la memoria de trabajo y el estriado, han mostrado respuestas inflamatorias con el tráfico.



A Section 508-compliant HTML version of this article is available at <http://dx.doi.org/10.1289/EHP118>

Autism Spectrum
Pollution
A Nested Case-Control Study

Raanan Raz
Allan C.

Perinatal Exposure to Traffic-Related Air Pollution and Autism Spectrum Disorders

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BACKGROUND: Studies from the United States indicate that exposure to air pollution in early life is associated with autism spectrum disorders (ASD) in children, but the evidence is not consistent with European data.

OBJECTIVE: We aimed to investigate the association between exposure to air pollution from road traffic and the risk of ASD in children, with careful adjustment for socioeconomic and other confounders.

METHOD: Children born and residing in Stockholm, Sweden, during 1993–2007 with an ASD diagnosis were identified through multiple health registers and classified as cases ($n = 5,136$). A randomly selected sample of 18,237 children from the same study base constituted controls. Levels of nitrogen oxides (NO_x) and particulate matter with diameter $\leq 10 \mu\text{m}$ (PM_{10}) from road traffic were estimated at residential addresses during mother's pregnancy and the child's first year of life by dispersion models. Odds ratios (OR) and 95% confidence intervals (CI) for ASD with or without intellectual disability (ID) were estimated using logistic regression models after conditioning on municipality and calendar year of birth as well as adjustment for potential confounders.

RESULT: Air pollution exposure during the prenatal period was not associated with ASD overall

matter (PM) (Becerra et al. 2013; Jung et al. 2013; Kalkbrenner et al. 2010, 2015; Raz et al. 2015; Windham et al. 2006; Volk et al. 2013), metals (Kalkbrenner et al. 2010; Palmer et al. 2009; Windham et al. 2006), and other hazardous air pollutants (Kalkbrenner et al. 2010; Windham et al. 2006) have been linked to a modestly increased risk of ASD, which may be explained by systemic inflammation affecting the central nervous system development *in utero* (Allen et al. 2014). However, a recent European meta-analysis could not confirm this from those exposed to NO_x and PM during perinatal life (Guxens et al. 2016).

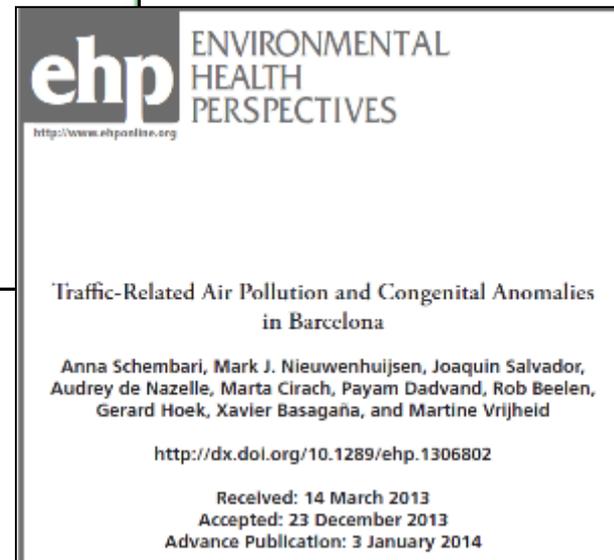
Research

POBLACIÓN PRENATAL



The screenshot shows the website of the Centro de Investigación en Epidemiología Ambiental (CREAL). The main navigation bar includes: El CREAL, Programas de investigación, Publicaciones, Docencia, Asesoramiento, and Herramientas y Recursos. The news section is titled 'NOTICIAS' and features an article from February 7, 2014, titled 'La contaminación del aire puede afectar las malformaciones congénitas del corazón'. The article text states: 'Existen diversos estudios epidemiológicos que sugieren que la contaminación causada por el tráfico puede afectar negativamente la salud de los recién nacidos. En esta ocasión, los investigadores del CREAL encontraron que el riesgo de la coartación de la aorta, un defecto congénito del corazón, aumenta en un 15% entre las poblaciones expuestas a los niveles más altos de contaminación del aire en Barcelona. Este estudio, publicado en la revista Environmental Health Perspectives por la investigadora Anna Schembari, representa un avance en los resultados previos publicados en un meta-análisis anterior realizado por el mismo grupo del CREAL, centro de investigación de la alianza ISGlobal.' A red box highlights a concluding sentence: 'Los investigadores también concluyen que la exposición a la contaminación del aire relacionada con el tráfico, como el NO2 y partículas, puede aumentar el riesgo de otros defectos de nacimiento no estudiados previamente: el riesgo de defectos del sistema digestivo y de la pared abdominal. No se encontraron asociaciones para los muchos otros grupos de defectos congénitos estudiados como: defectos del tubo neural, defectos en el sistema respiratorio, las hendiduras faciales, defectos en el sistema urinario, hipospadias y defectos por reducción de extremidades.' Below the article, there is a 'CONTACTO' section with contact information for Gisela Sanmartín.

Los niños tienen un riesgo elevado de enfermedad relacionada con la contaminación e incluso la exposición a dosis extremadamente bajas de contaminantes durante los periodos de especial vulnerabilidad en la vida intrauterina y la primera infancia puede conducir a la enfermedad, invalidez y muerte en la infancia y a lo largo de toda la vida (Lancet 2017)



The image shows the front cover of the journal 'Environmental Health Perspectives'. The logo 'ehp' is prominent on the left, with 'ENVIRONMENTAL HEALTH PERSPECTIVES' to its right. The URL 'http://www.ehponline.org' is printed below the logo. The title of the article, 'Traffic-Related Air Pollution and Congenital Anomalies in Barcelona', is centered. Below the title, the authors are listed: Anna Schembari, Mark J. Nieuwenhuijsen, Joaquin Salvador, Audrey de Nazelle, Marta Cirach, Payam Davdand, Rob Beelen, Gerard Hoek, Xavier Basagaña, and Martine Vrijheid. At the bottom, the DOI link 'http://dx.doi.org/10.1289/ehp.1306802' is provided, along with the dates: 'Received: 14 March 2013', 'Accepted: 23 December 2013', and 'Advance Publication: 3 January 2014'.

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<http://www.ehponline.org>

Outdoor Air Pollution, Preterm Birth, and Low Birth Weight: Analysis of the World Health Organization Global Survey on Maternal and Perinatal Health

Nancy L. Fleischer, Mario Merialdi, Aaron van Donkelaar, Felipe Vadillo-Ortega, Randall V. Martin, Ana Pilar Betran, João Paulo Souza, and Marie S. O'Neill

<http://dx.doi.org/10.1289/ehp.1306837>

Received: 22 March 2013

Accepted: 4 February 2014

Advance Publication: 7 February 2014



Proyecto
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Áreas de
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Familias
participantes

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Differences in Birth Weight Associated with the 2008 Beijing Olympic Air Pollution Reduction: Results from a Natural Experiment

David Q. Rich, Kaibo Liu, Jinliang Zhang, Sally W. Thurston, Timothy P. Stevens, Ying Pan, Cathleen Kane, Barry Weinberger, Pamela Ohman-Strickland, Tracey J. Woodruff, Xiaoli Duan, Vanessa Assibey-Mensah, and Junfeng Zhang

<http://dx.doi.org/10.1289/ehp.1408795>

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POBLACIÓN PRENATAL

Environmental Pollution 218 (2016) 1154–1161



Contents lists available at ScienceDirect

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journal homepage: www.elsevier.com/locate/envpol



Impact of air pollution and temperature on adverse birth outcomes: Madrid, 2001–2009[☆]



Virginia Arroyo^{a, b}, Julio Díaz^a, Rocío Carmona^a, Cristina Ortiz^a, Cristina Linares^{a, *}

^a National School of Public Health, Carlos III Institute of Health, Madrid, Spain

^b University of Salamanca Health Care Complex (Complejo Asistencial Universitario de Salamanca/CAUSA), Salamanca, Spain

Table 2

Relative risks (RRs) and Proportional attributable risks (PARs) obtained in the Poisson models for the variables of low birth weight, premature birth and late foetal death: Madrid, 2001–2009. Shown in brackets are the explanatory lags for each significant environmental variable.

Low birth weight (<2500 g)	Wk. gest. ^a (trimester)	RR			PAR (%)	Cumulative PAR (%)	p-value
NO ₂ (lag 14)	23 (2nd Tr.)	1.011	1.007	1.014	1.06	2.45 ^b	0.000
NO ₂ (lag 20)	17 (2nd Tr.)	1.014	1.011	1.017	1.39	–	0.000
O ₃ (lag 25)	12 (1st Tr.)	1.005	1.001	1.008	0.46	–	0.005
Leqd (lag 0)	37 (3rd Tr.)	1.014	1.008	1.023	1.36	8.84 ^c	0.000
Leqd (lag 16)	21 (2nd Tr.)	1.044	1.037	1.052	4.25	–	0.000
Leqd (lag 34)	3 (1st Tr.)	1.033	1.027	1.040	3.23	–	0.000
Tmax (lag 14)	23 (2nd Tr.)	1.003	1.002	1.004	0.31	–	0.000
Premature birth (<37 wk gest.)	Wk. gest. (natural no.) ^a	RR			PAR (%)	Cumulative PAR (%)	p-value
PM _{2.5} (lag 20)	17 (2nd Tr.)	1.026	1.018	1.034	2.53	–	0.000
O ₃ (lag 25)	12 (1st Tr.)	1.011	1.007	1.014	1.05	–	0.000
Leqd (lag 16)	21 (2nd Tr.)	1.031	1.024	1.039	3.05	–	0.000
Leqn (lag 1)	36 (3rd Tr.)	1.020	1.012	1.028	1.95	–	0.000
Late foetal death (<first 24 h of life)	Wk. gest. (natural no.) ^a	RR			PAR (%)	Cumulative PAR (%)	p-value
NO ₂ (lag 4)	33 (3rd Tr.)	1.028	1.023	1.033	2.72	–	0.000
PM _{2.5} (lag 6)	31 (3rd Tr.)	1.115	1.103	1.126	10.27	–	0.000
O ₃ (lag 13)	24 (2nd Tr.)	1.155	1.147	1.163	13.40	–	0.000
Tmax (lag 20)	17 (2nd Tr.)	1.012	1.010	1.013	1.17	–	0.000
Tmin (lag 2)	35 (3rd Tr.)	1.037	1.035	1.039	3.55	–	0.000

^a 37 weeks of gestation (i.e., duration of a full-term pregnancy) less the numerical value of the corresponding lag.

^b Cumulative PAR for NO₂.

^c Cumulative PAR for diurnal noise level (Leqd).

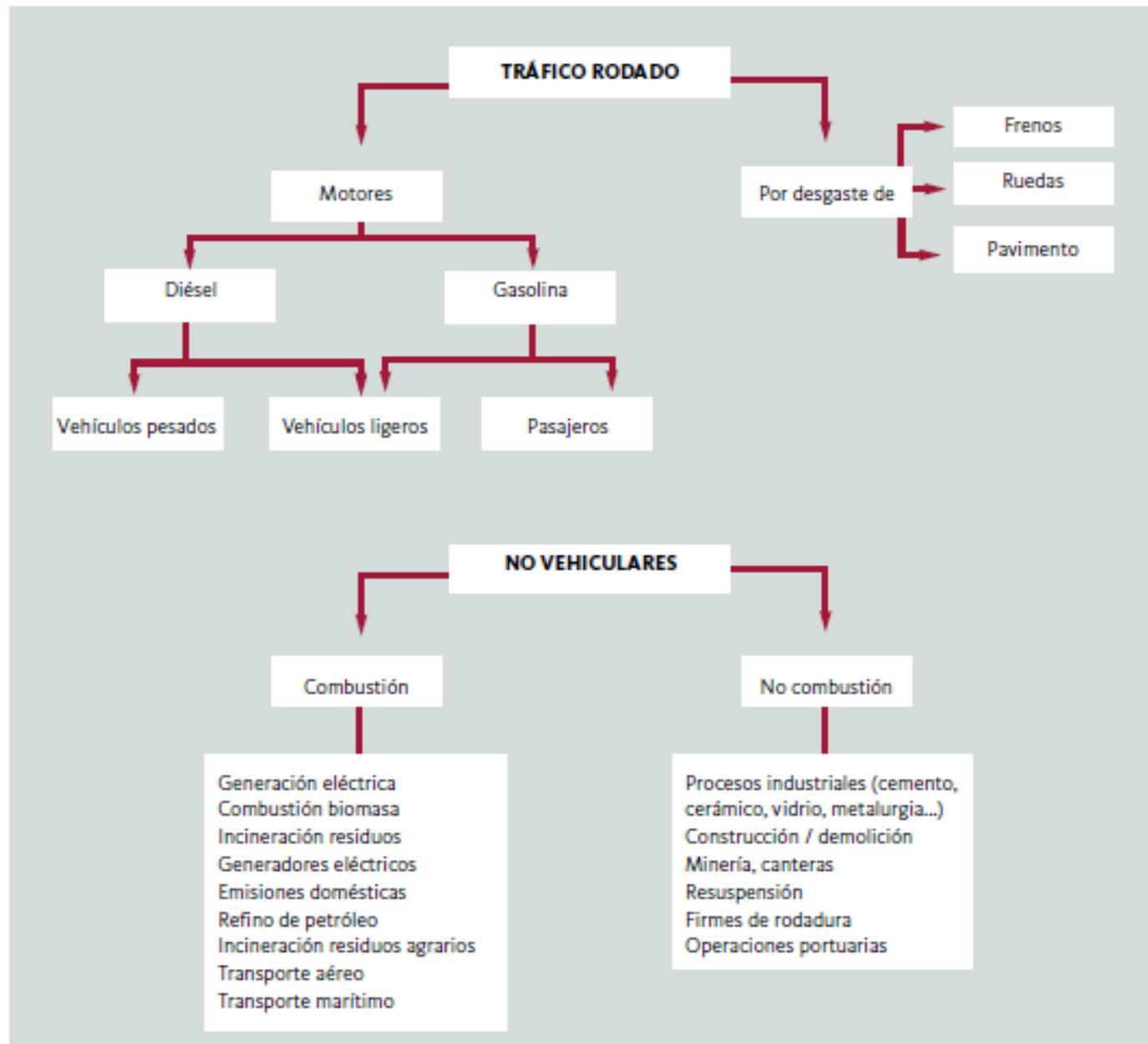
EVOLUCIÓN EN LÍNEAS DE INVESTIGACIÓN

POBLACIÓN PRENATAL

Town/City	First Trimester			Second Trimester			Third Trimester		
	Lags with association	Wk. gest. ^a	Month gest.	Lags with association	Wk. gest. ^a	Month gest.	Lags with association	Wk. gest. ^a	Month gest.
Albacete	*	*	*	NO2 (lag 19)	18	5	*	*	*
Almería	PM10 (lag 35)	2	1	*	*	*	PM10 (lag 0)	37	9+
Ávila	*	*	*	*	*	*	NO2 (lag 4)	33	9
Burgos	PM10 (lag 35)	2	1	NO2 (lag 13)	24	6	*	*	*
Cáceres	PM10 (lag 30)	7	2	*	*	*	*	*	*
Castellón	*	*	*	*	*	*	NO2 (lag 8)	29	8
Córdoba	*	*	*	*	*	*	NO2 (lag 0)	37	9+
Granada	*	*	*	NO2 (lag 21)	16	4	*	*	*
Jaén	PM10 (lag 24)	13	3	*	*	*	PM10 (lag 5)	32	8
León	*	*	*	*	*	*	O3h (lag 0)	37	9+
Madrid	*	*	*	NO2 (lag 14)	23	6	*	*	*
Murcia	*	*	*	*	*	*	PM10 (lag 5)	32	8
Oviedo	*	*	*	*	*	*	PM10 (lag 4)	33	9
Pamplona	*	*	*	*	*	*	O3h (lag 5)	32	8
P.Mallorca	NO2 (lag 36)	1	1	PM10 (lag 12)	25	7	PM10 (lag 9)	28	7
S.C.Tenerife	PM10 (lag 25)	12	3	*	*	*	*	*	*
	PM10 (lag 27)	10	3	*	*	*	*	*	*
	PM10 (lag 31)	6	2	*	*	*	*	*	*
Soria	*	*	*	NO2 (lag 16)	21	6	*	*	*
Tarragona	*	*	*	NO2 (lag 23)	14	4	*	*	*
Valladolid	NO2 (lag 32)	5	2	*	*	*	*	*	*
	PM10 (lag 34)	3	1	*	*	*	*	*	*
Vitoria	*	*	*	NO2 (lag 19)	18	5	PM10 (lag 2)	35	9
Zamora	PM10 (lag 36)	1	1	PM10 (lag 21)	16	4	NO2 (lag 0)	37	9+
	*	*	*	*	*	*	NO2 (lag 6)	31	8
Zaragoza	*	*	*	PM10 (lag 12)	25	7	*	*	*

^a 37 weeks of gestation (i.e., duration of a full-term pregnancy) less the numerical value of the corresponding lag.

MEDIDAS DE MINIMIZACIÓN DE IMPACTOS



MEDIDAS TRAFICO RODADO URBANO

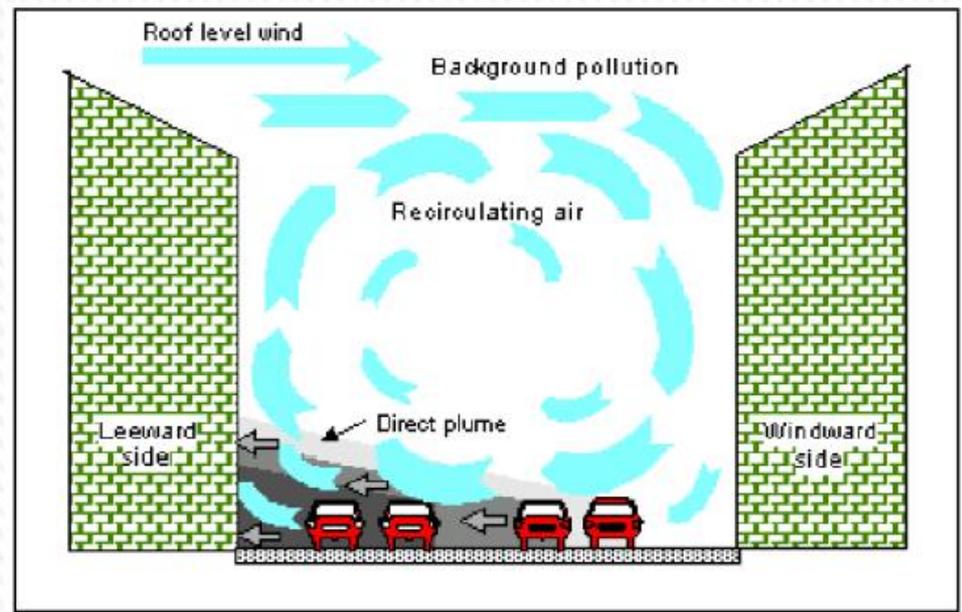
Reducción del transporte privado

- ✓ Incentivar el transporte público o incentivar vehículos con varios pasajeros
- ✓ Diseñar las ciudades y áreas periféricas con infraestructura de transporte adecuado
- ✓ Peajes urbanos (zonas céntricas)
–Zonas de bajas emisiones
- ✓ Favorecer el uso de vehículos ecológicos (bicicleta)

MEDIDAS TRAFICO RODADO URBANO

Reducción del transporte privado

- ✓ Incentivar el transporte público o incentivar vehículos con varios pasajeros
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MEDIDAS DE MINIMIZACIÓN DE IMPACTOS

Los programas de reducción de la contaminación atmosférica a corto y largo plazo mejoran la salud de la población

LOS JUEGOS OLÍMPICOS DE VERANO EN ATLANTA, EEUU (1996)

Durante la celebración de los Juegos Olímpicos de 1996, la ciudad de Atlanta implantó una estrategia de transporte que condujo a una reducción de la congestión del tráfico. Durante más de 10 semanas antes, 17 días durante y 4 semanas después de los Juegos se registraron los datos del número de visitas al hospital, número de hospitalizaciones por asma y otros problemas de salud, calidad del aire, condiciones meteorológicas y uso del transporte público. La calidad del aire incluyó PM_{10} , dióxido de nitrógeno y ozono.

Los resultados de los análisis mostraron un significativo descenso en el número (41,6%) de ataques agudos de asma durante los Juegos Olímpicos. En ese período, la calidad del aire mejoró ostensiblemente, con una reducción significativa de las concentraciones de ozono (28%, desde $163 \mu\text{g}/\text{m}^3$ a $117 \mu\text{g}/\text{m}^3$ de media del máximo diario de 1 hora), monóxido de carbono (22%, desde $1,80 \text{ mg}/\text{m}^3$ a $1,47 \text{ mg}/\text{m}^3$ de media de ocho horas) y PM_{10} (16%, desde $36,7 \mu\text{g}/\text{m}^3$ a $30,8 \mu\text{g}/\text{m}^3$ de media diaria). Los picos de tráfico por las mañanas durante los días laborables fueron reducidos en un 22,5%, considerando como referencia el periodo anterior al inicio de los Juegos Olímpicos. Los picos diarios de las concentraciones de ozono estuvieron significativamente correlacionados con los niveles de tráfico.

LOS JUEGOS OLÍMPICOS DE VERANO EN PEKÍN, CHINA (2008)

El gobierno chino prometió unos Juegos Olímpicos limpios pese a que la ciudad es una de las más contaminadas del mundo y los atletas albergaban temores a que la contaminación de la ciudad afectara a su rendimiento físico. Se implantaron estándares muy estrictos para mejorar la calidad del aire durante la celebración de los Juegos.

Un estudio científico mostró la reducción de visitas por asma en el hospital Chaoyang de Pekín paralela a la bajada de los niveles de contaminantes. La media de estas visitas fue de 12,5 adultos al día del 1 al 30 de junio, es decir, antes de que se aplicaran medidas de control de la contaminación atmosférica. En cambio, con las medidas de control implantadas durante los Juegos, estas visitas se redujeron hasta 7,3 en el mismo hospital.

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