OMCeO Udine

Con il Patrocinio di

Corso di Formazione

Inquinamento atmosferico e danni alla salute: cosa devono sapere e cosa devono fare il Medico e l'Odontoiatra

Cosa dice la letteratura e cosa dobbiamo sapere

Giuseppe Sarno

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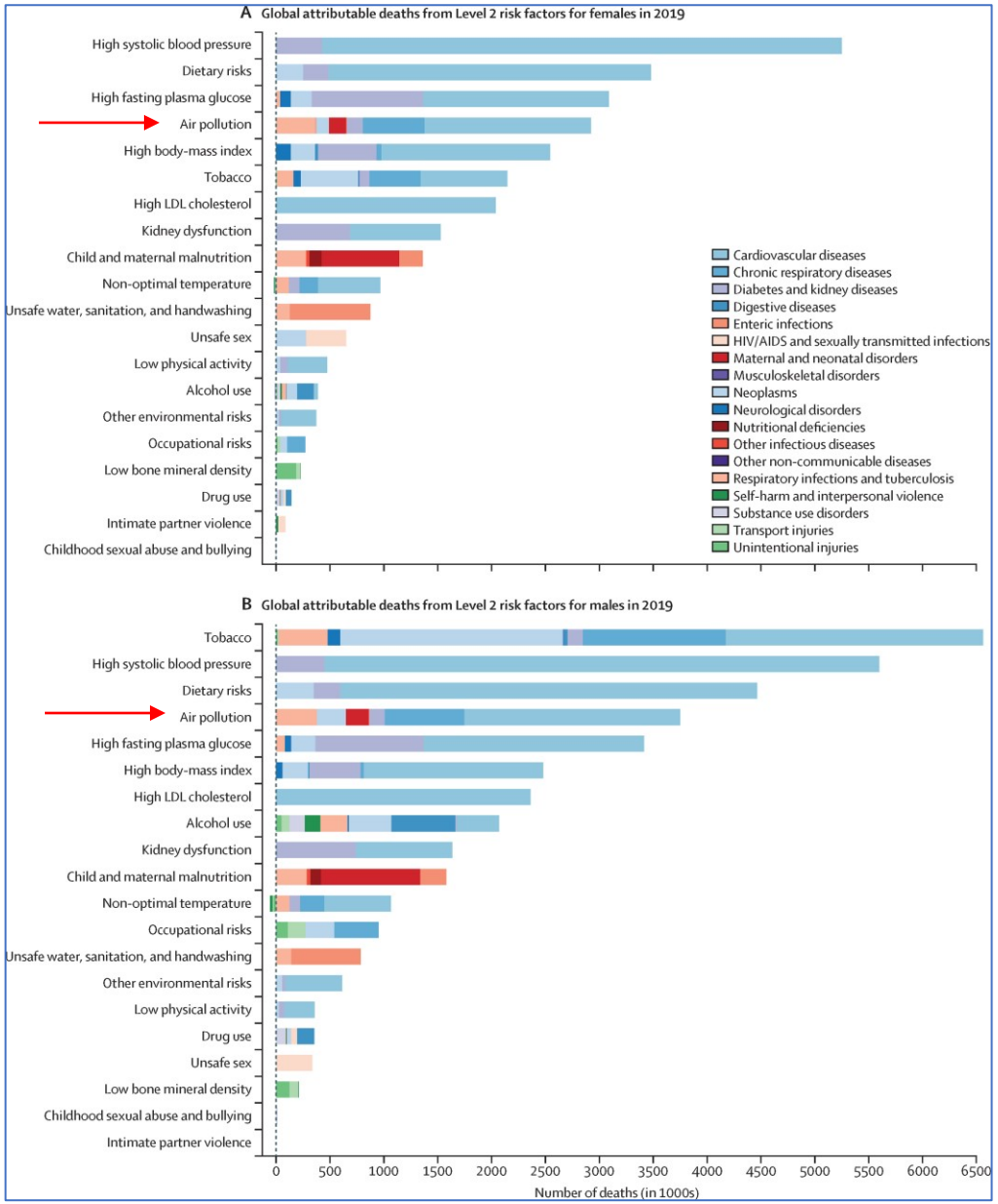
THE LANCET

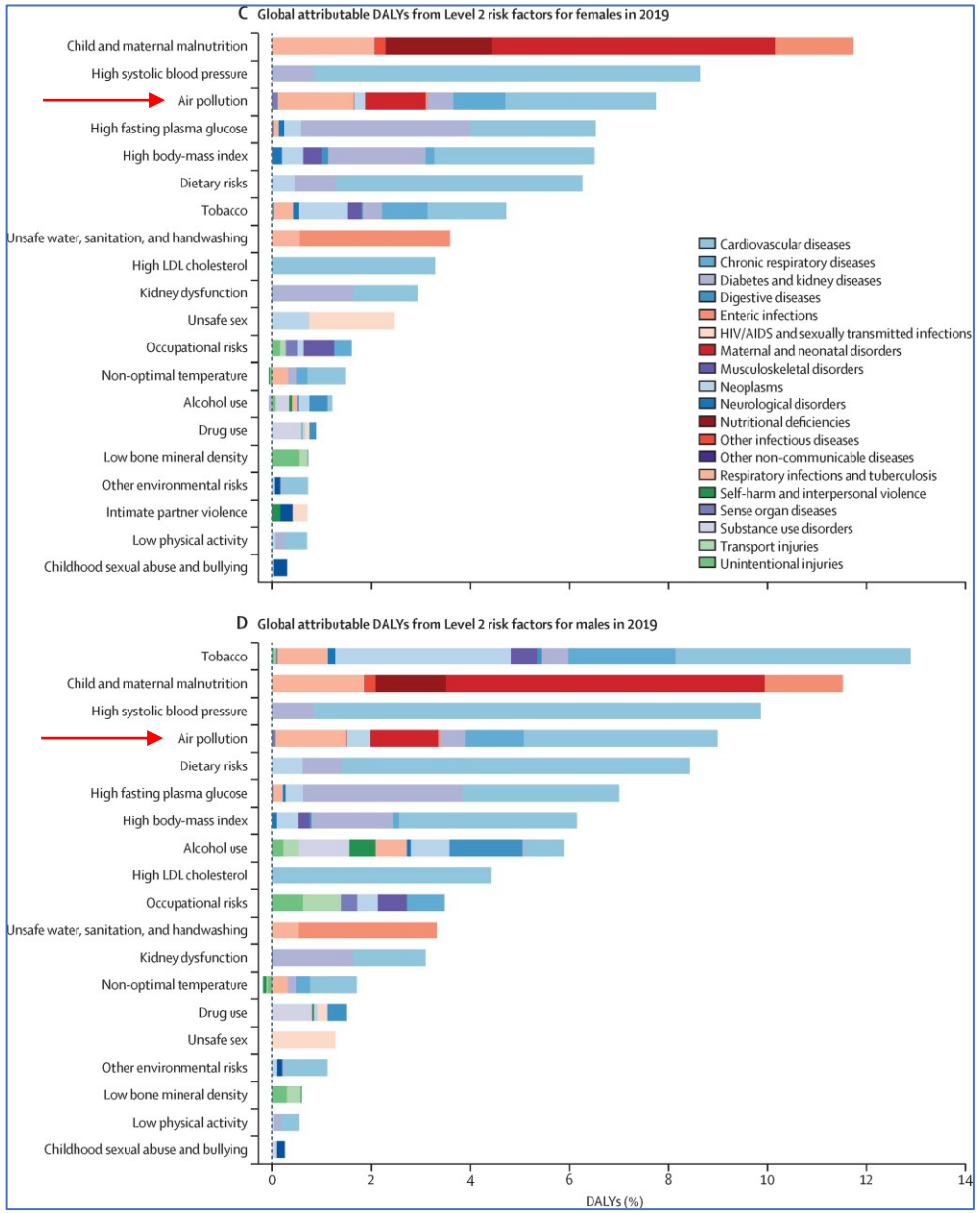
GLOBAL HEALTH METRICS | VOLUME 396, ISSUE 10258, P1223-1249, OCTOBER 17, 2020

Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019

Ambient and household air pollution together currently rank 4th for attributable disease and mortality among 20 major risk factors evaluated in the Global Burden of Disease (GBD) study, only after hypertension, smoking and dietary factors

Global number of deaths and percentage of DALYs attributable to risk factors, by cause and sex, 2019



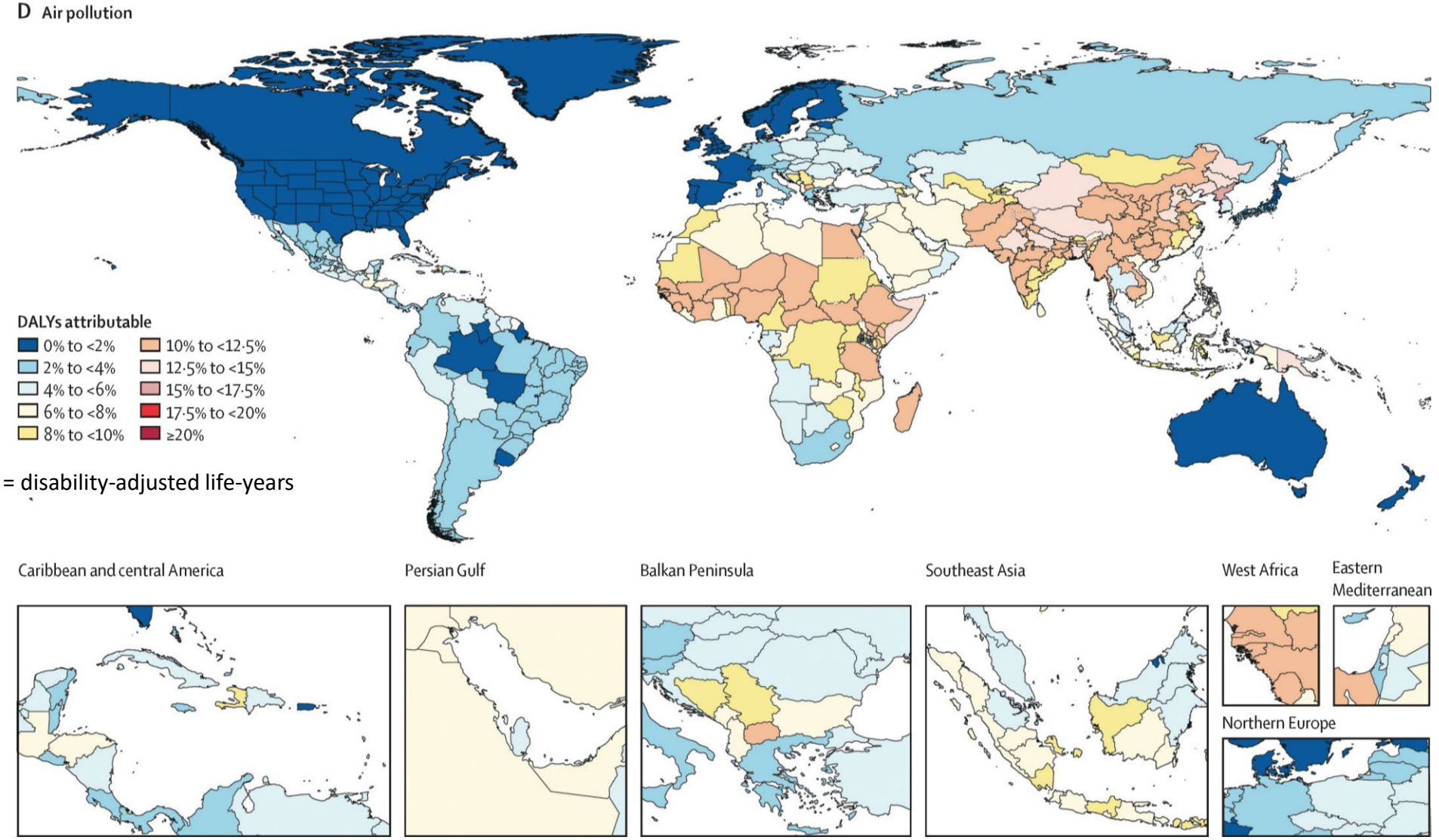


THE LANCET

GLOBAL HEALTH METRICS | VOLUME 396, ISSUE 10258, P1223-1249, OCTOBER 17, 2020

Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019

The burden attributable to air pollution (ambient particulate matter, household air pollution, and ambient ozone pollution).



DALYs = disability-adjusted life-years

AIR POLLUTION – THE SILENT KILLER

Every year, around **7 MILLION DEATHS** are due to exposure from both outdoor and household air pollution.

Air pollution is a major environmental risk to health.
By reducing air pollution levels, countries can reduce:

- **Stroke**
- **Heart disease**
- **Lung cancer, chronic obstructive pulmonary disease, pneumonia and asthma**

REGIONAL ESTIMATES ACCORDING TO WHO REGIONAL GROUPINGS:

- **More than 2 million** in South-East Asia Region
- **More than 2 million** in Western Pacific Region
- **1 million** in Africa Region
- **500 000** deaths in Eastern Mediterranean Region
- **500 000** deaths in European Region
- **More than 300 000** in the Region of the Americas

WHO Air Quality Guidelines set goals to protect millions of lives from air pollution.

CLEAN AIR FOR HEALTH #AirPollution World Health Organization

World Health Organization

World Health Organization

Our Planet Our Health

Clean our air, water & food

#HealthierTomorrow

World Health Day 2022
7 April 2022

Air pollution kills 13 people every minute due to lung cancer, heart disease and strokes. Stop burning fossil fuels like oil, coal and natural gas. #HealthierTomorrow

Nine out of ten people breathe polluted air. Stop burning fossil fuels like oil, coal and natural gas. #HealthierTomorrow

Burning fossil fuels like oil, coal and natural gas causes air pollution. Keep fossil fuels in the ground for a healthy planet and a healthy me. #HealthierTomorrow

Nitrogen dioxide pollution can aggravate respiratory diseases, particularly asthma. Well planned public transport systems, including safe walking and cycling, can improve air quality, mitigate climate change, and deliver additional health gains. #HealthierTomorrow

World Health Organization

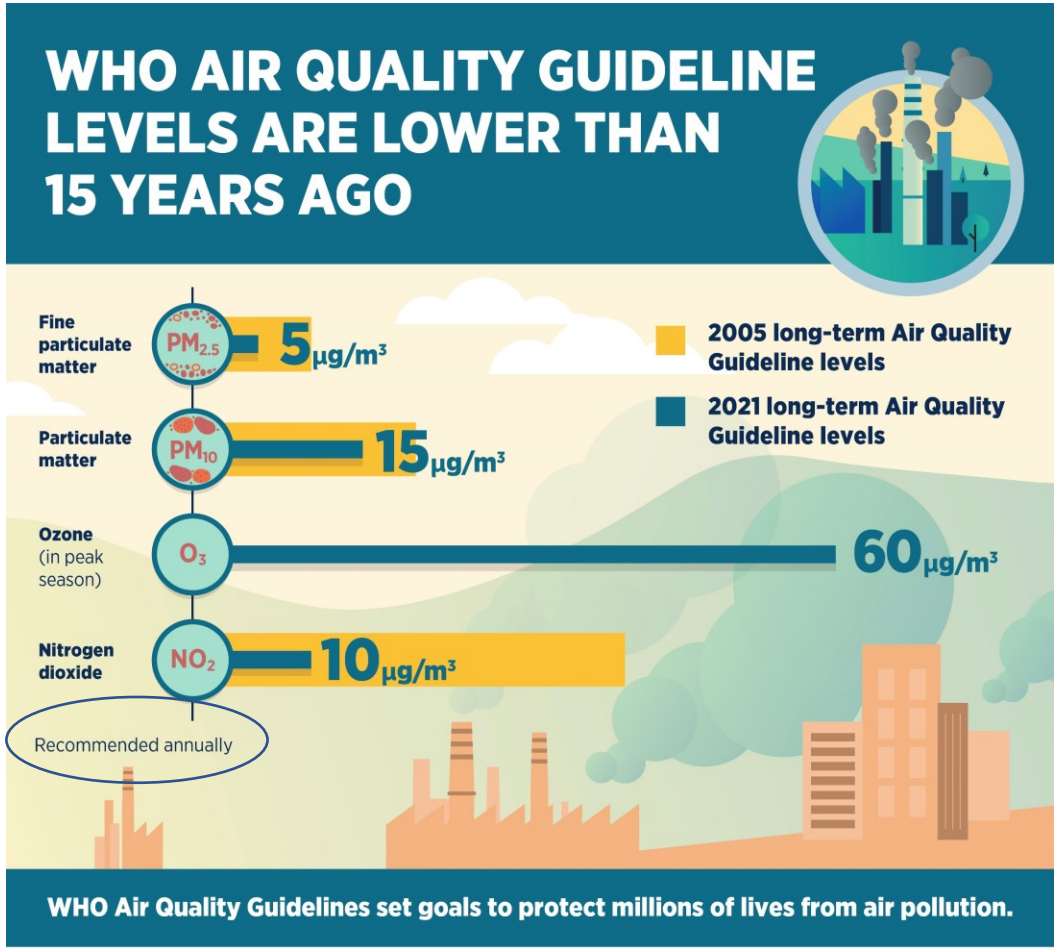
WHO global health quality guidelines

Livelli AQG raccomandati per il 2021 rispetto alle linee guida sulla qualità dell'aria del 2005

Pollutant	Averaging time	2005 AQGs	2021 AQG level
$PM_{2.5}$, µg/m³	Annual	10	5
	24-hour[a]	25	15
PM_{10}, µg/m³	Annual	20	15
	24-hour[a]	50	45
O_3, µg/m³	Peak season[b]	–	60
	8-hour[a]	100	100
NO_2, µg/m³	Annual	40	10
	24-hour[a]	–	25
SO_2, µg/m³	24-hour[a]	20	40
CO, mg/m³	24-hour[a]	–	4

World Health Organization (2021). WHO global health quality guidelines: particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. World Health Organization. https://apps.WHO.int/iris/handle/10665/345329

World Health Organization



CLEAN AIR FOR HEALTH #AirPollution World Health Organization

CLEAN AIR FOR HEALTH #AirPollution World Health Organization

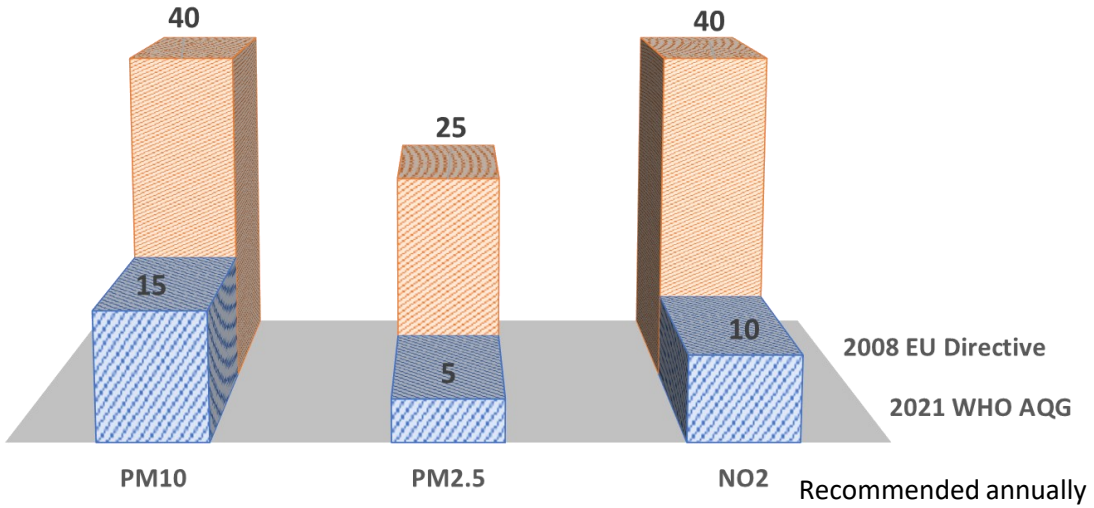
DIRETTIVA 2008/50/CE DEL PARLAMENTO EUROPEO E DEL CONSIGLIO
del 21 maggio 2008
relativa alla qualità dell'aria ambiente e per un'aria più pulita in Europa

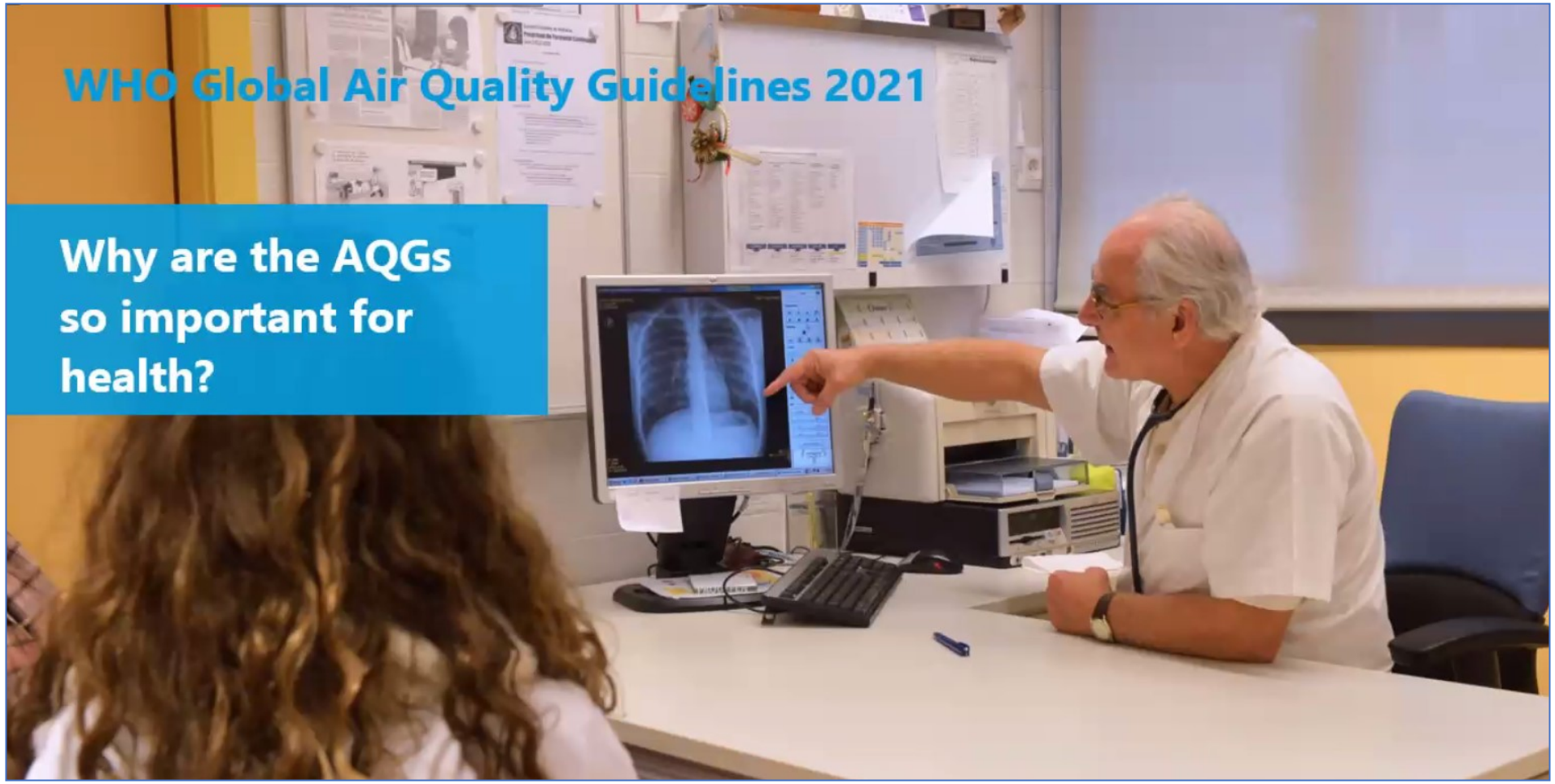
VS

	2008 EU Directive		2021 WHO AQG	
Pollutant	**Averaging time**	**Limit or target value**	**Averaging time**	**AQG level**
$PM_{2.5}$ ($\mu g/m^3$)	Annual	25	Annual	5
			24-hour[d]	15
PM_{10} ($\mu g/m^3$)	Annual	40	Annual	15
	24-hour[a]	50	24-hour[d]	45
O_3 ($\mu g/m^3$)	Daily maximum 8-hour[b]	120	Peak season[e]	60
			24-hour[d]	100
NO_2 ($\mu g/m^3$)	Annual	40	Annual	10
	1-hour[c]	200	1-hour	200
			24-hour[d]	25

a not to be exceeded more than 35 times a year
b not to be exceeded on more than 25 days per year averaged over three years
c not to be exceeded more than 18 times a year
d 99th percentile (i.e. 3–4 exceedance days per year).
e average of daily maximum 8-hour mean O3 concentration in the six consecutive months with the highest six-month running-average O3 concentration.

COMPARISON BETWEEN WHO AIR QUALITY GUIDELINES AND THE EU DIRECTIVE ON AMBIENT AIR QUALITY AND CLEANER AIR




WHO Global Air Quality Guidelines 2021
Why are the AQGs so important for health?

World Health Organization
Regional Office for Europe

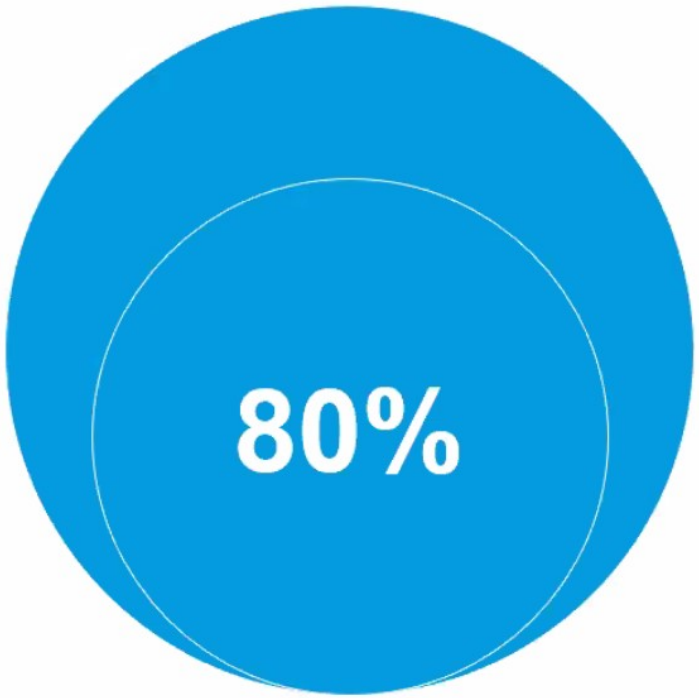
Air pollution in the world

- More than 90% of the global population in 2019 lived in areas where $PM_{2.5}$ concentrations exceeded the 2005 annual WHO AQG of 10 µg/m³.
  - *Many of the countries with the lowest national $PM_{2.5}$ exposure levels were in WHO European Region.*
- The patterns of ambient NO_2 concentrations had the highest population-weighted concentrations in eastern Asia, the Middle East, North America and much of Europe, reflecting emissions from mobile sources propelled by combustion engines.
- Air pollution leads to health-related economic impacts through human health costs and lost labour productivity.

How many premature deaths could be avoided?

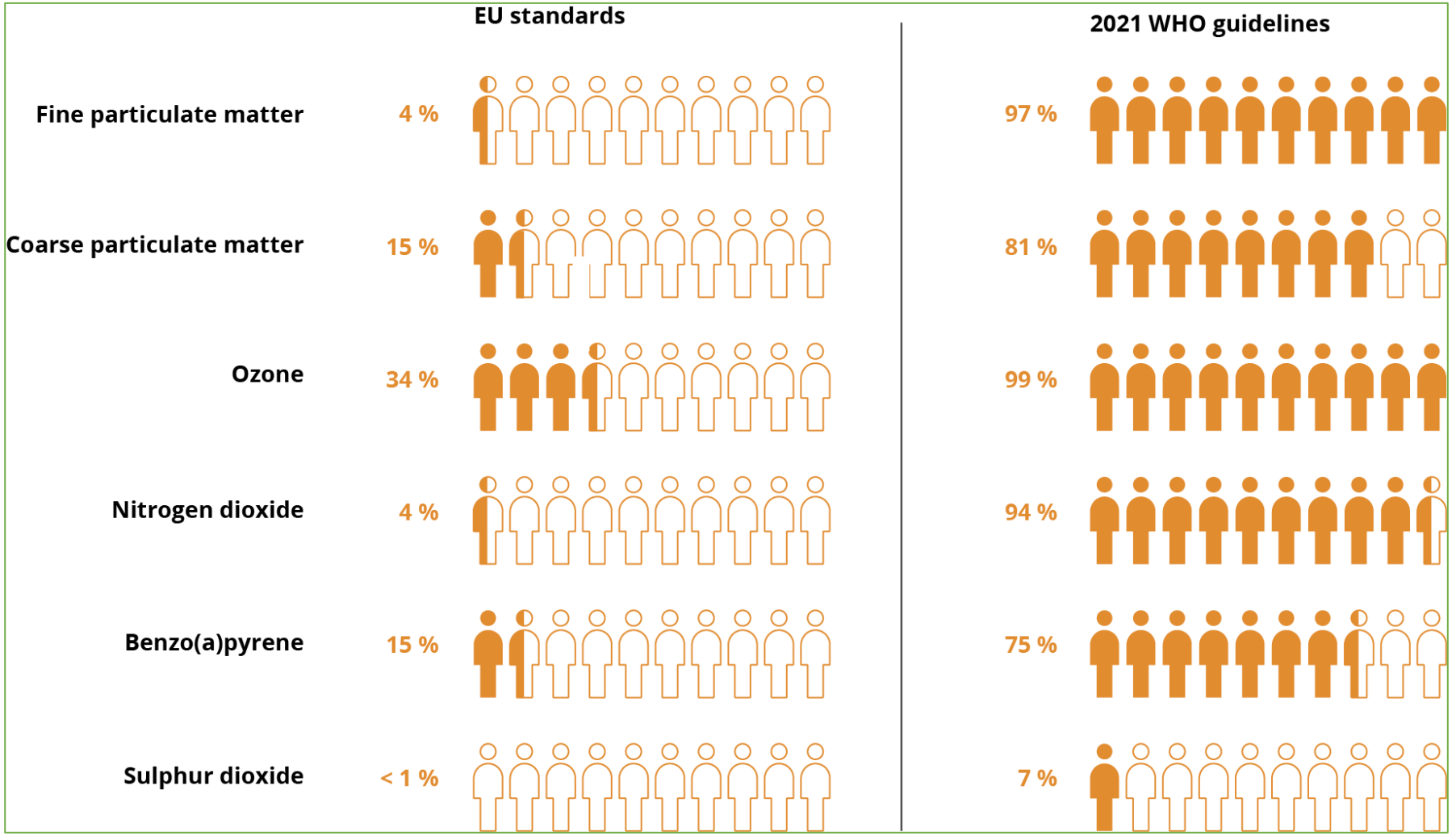
World Health Organization
REGIONAL OFFICE FOR Europe

- WHO performed a rapid scenario analysis to explore the reductions in disease burden attributed to ambient $PM_{2.5}$ globally that would occur if the 2016 concentrations were reduced to the current AQGs (2021). **The estimated burden of disease can be reduced substantially.**
- **Almost 3.3 million premature deaths attributed to ambient $PM_{2.5}$ would have been avoided if the 2021 AQG level had been achieved worldwide in 2016.**
- **66% of premature deaths would have been avoided in the European region (more than 300 000).**
- In high-income countries, where the ambient $PM_{2.5}$ concentrations are already below the interim targets, reaching the AQG level **is needed to significantly reduce the health burden from exposure**.



European Environment Agency

Figure 1. Share of the EU urban population exposed to air pollutant concentrations above EU standards and WHO guidelines in 2019



https://www.eea.europa.eu/publications/air-quality-in-europe-2021

European Environment Agency

Table 1. Theoretical health benefits, in terms of reductions in premature deaths, that would have been attained for the EU-27 in 2019 if the different EU limit values and WHO guidelines for $PM_{2.5}$ had been met across Europe

EU-27	Premature deaths due to $PM_{2.5}$	Reduction in premature deaths on 2019 levels	% reduction in premature deaths on 2019 levels	% reduction in premature deaths on 2005 levels
2019 concentrations	306,700	-	-	33%
EU limit value 25 µg/m^3	306,500	200	0%	33%
EU indicative limit value 20 µg/m^3	303,500	3,200	1%	33%
2021 WHO interim target 3 15 µg/m^3	289,200	17,500	6%	37%
2021 WHO interim target 4 (2005 WHO air quality guideline) 10 µg/m^3	241,400	65,300	21%	47%
2021 WHO air quality guideline 5 µg/m^3	129,400	177,300	58%	72%

Note: The WHO interim targets and guidelines are drawn from the updated guidelines published in 2021.

https://www.eea.europa.eu/publications/health-risks-of-air-pollution/health-impacts-of-air-pollution

European Environment Agency

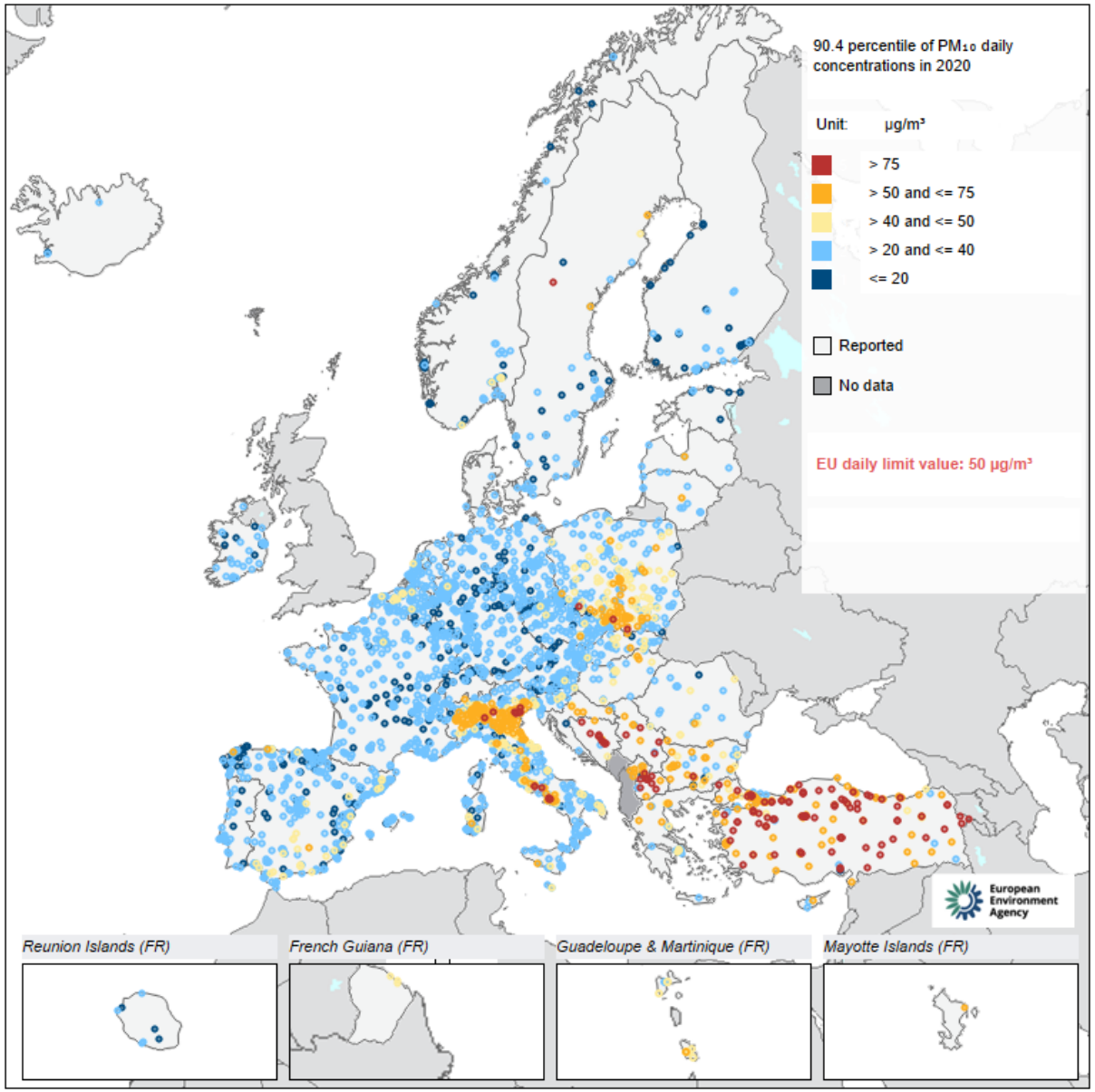
Table 2. Theoretical health benefits, in terms of reductions in premature deaths, that would have been attained in 2019 if the different EU limit values and WHO guidelines for $PM_{2.5}$ had been met, by country

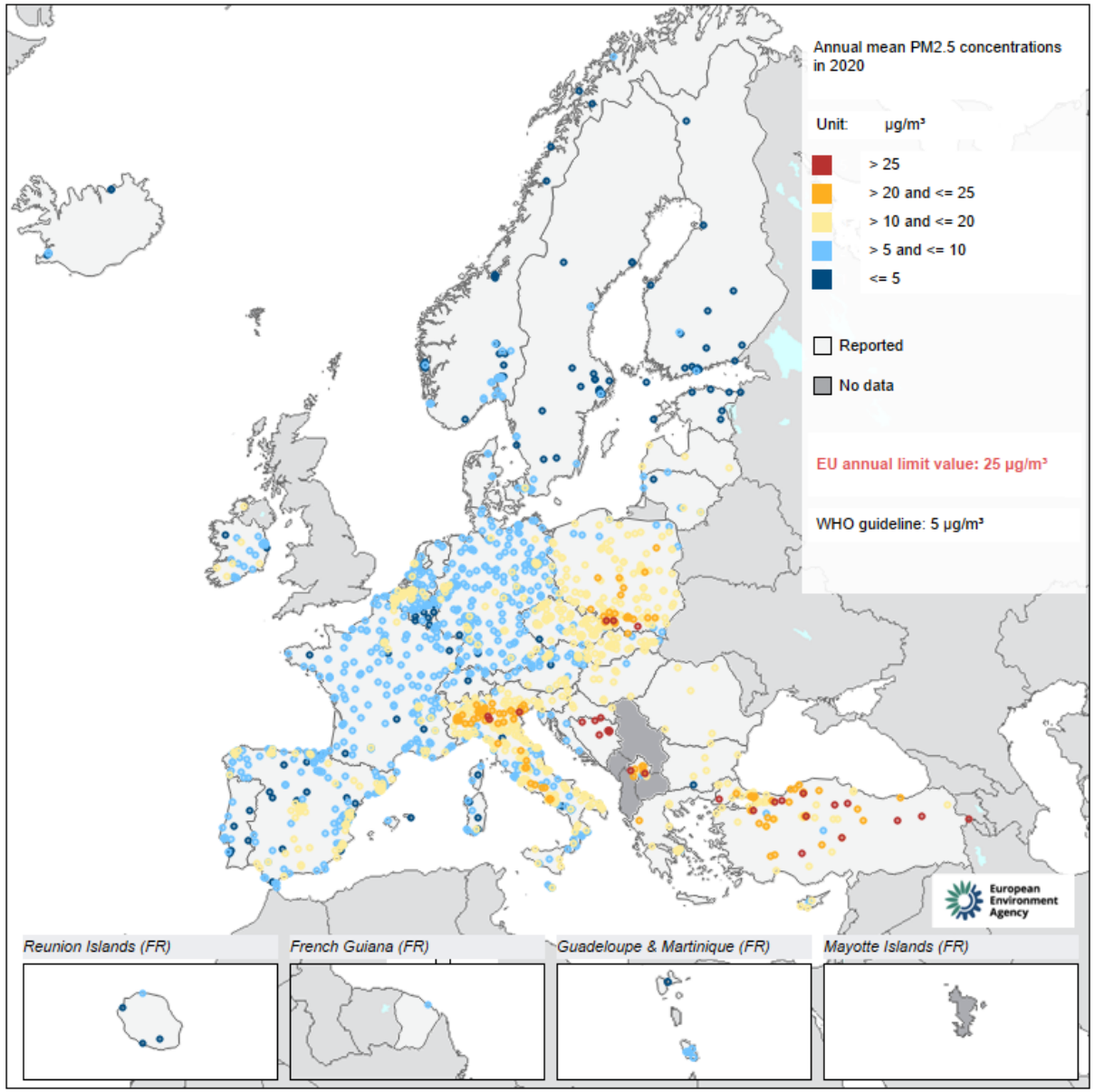
Country	2019 levels	EU limit value 25 µg/m³		EU indicative limit value 20 µg/m³		WHO interim target 3 15 µg/m³		WHO interim target 4 (2005 WHO air quality guideline) 10 µg/m³		WHO air quality guideline 5 µg/m³	
	Premature deaths	Premature deaths	% reduction on 2019 levels	Premature deaths	% reduction on 2019 levels	Premature deaths	% reduction on 2019 levels	Premature deaths	% reduction on 2019 levels	Premature deaths	% reduction on 2019 levels
Hungary	10,400	10,400	0	10,400	0	10,200	2	7,300	30	3,700	64
Ireland	1,300	1,300	0	1,300	0	1,300	0	1,300	0	900	31
Italy	49,900	49,900	0	49,100	2	45,200	9	34,500	31	17,700	65
Latvia	1,600	1,600	0	1,600	0	1,600	0	1,400	13	800	50
Lithuania	2,500	2,500	0	2,500	0	2,500	0	2,100	16	1,100	56
Luxembourg	200	200	0	200	0	200	0	200	0	100	50
Malta	300	300	0	300	0	300	0	200	33	100	67
Netherlands	8,900	8,900	0	8,900	0	8,900	0	8,300	7	4,200	53
Poland	39,300	39,100	1	37,600	4	32,700	17	22,800	42	11,600	70
Portugal	4,900	4,900	0	4,900	0	4,900	0	4,800	2	3,000	39
Romania	21,500	21,500	0	21,300	1	19,600	9	14,300	33	7,300	66
Slovakia	4,200	4,200	0	4,200	0	4,000	5	2,900	31	1,500	64
Slovenia	1,400	1,400	0	1,400	0	1,400	0	1,100	21	600	57
Spain	23,300	23,300	0	23,200	0	23,100	1	20,800	11	11,600	50
Sweden	2,800	2,800	0	2,800	0	2,800	0	2,800	0	2,300	18

https://www.eea.europa.eu/publications/health-risks-of-air-pollution/health-impacts-of-air-pollution

European Environment Agency

Concentrations of PM_{10} and $PM_{2.5}$ in 2020 and in relation to the EU limit value

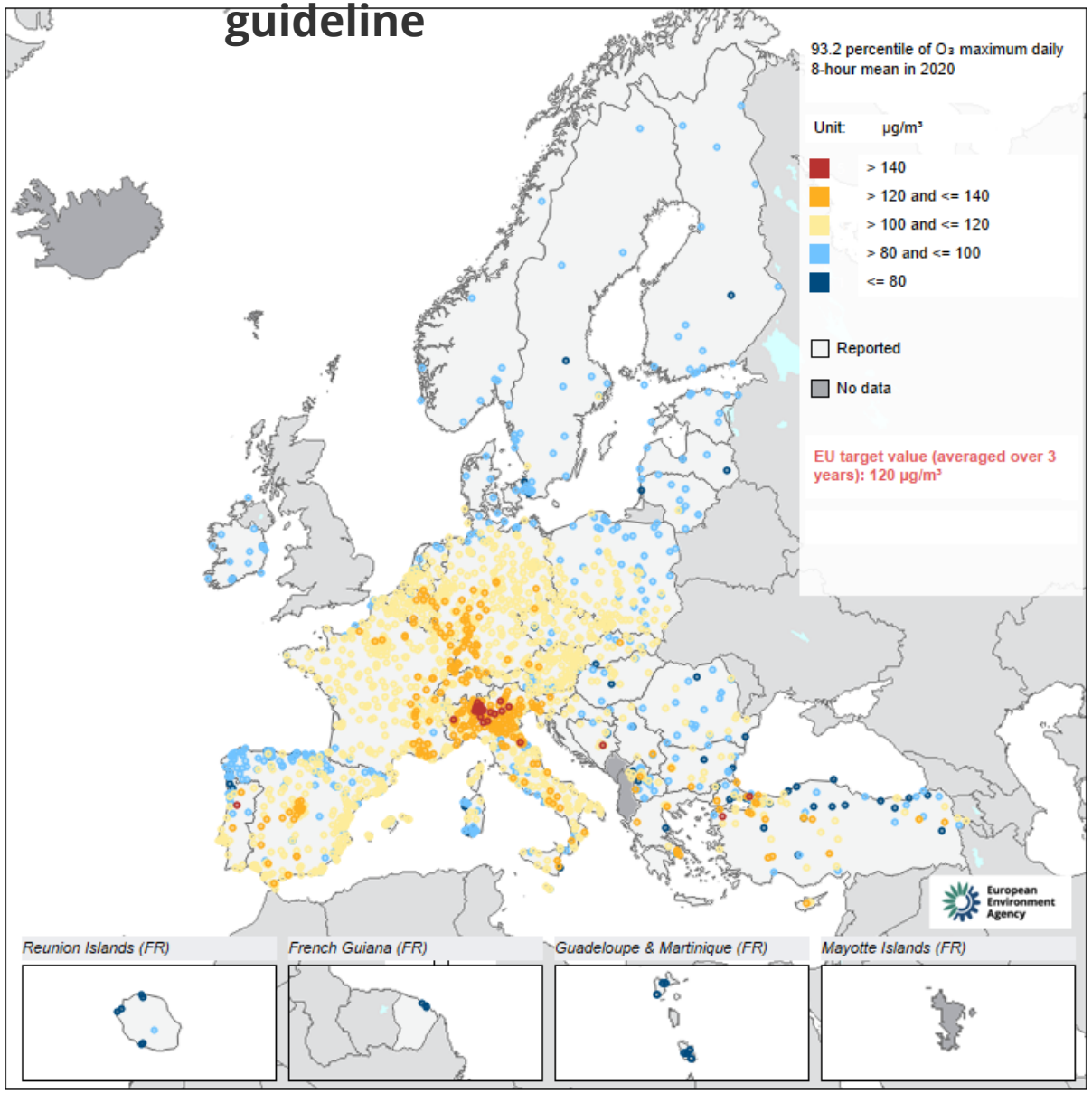


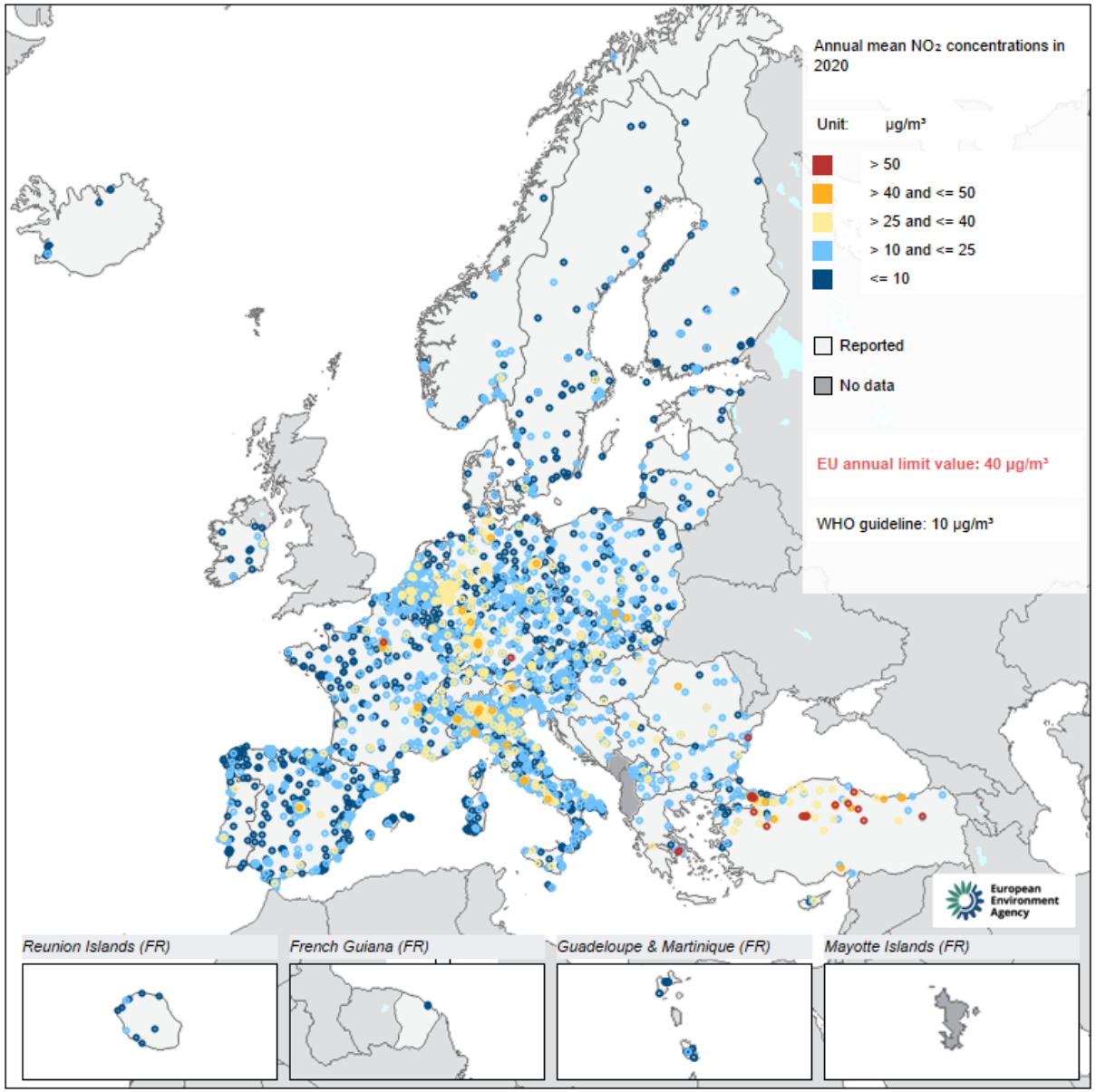


https://www.eea.europa.eu/publications/status-of-air-quality-in-Europe-2022/europes-air-quality-status-2022

European Environment Agency

Concentrations of O_3 and NO_2 in 2020 in relation to the EU limit value and the WHO guideline

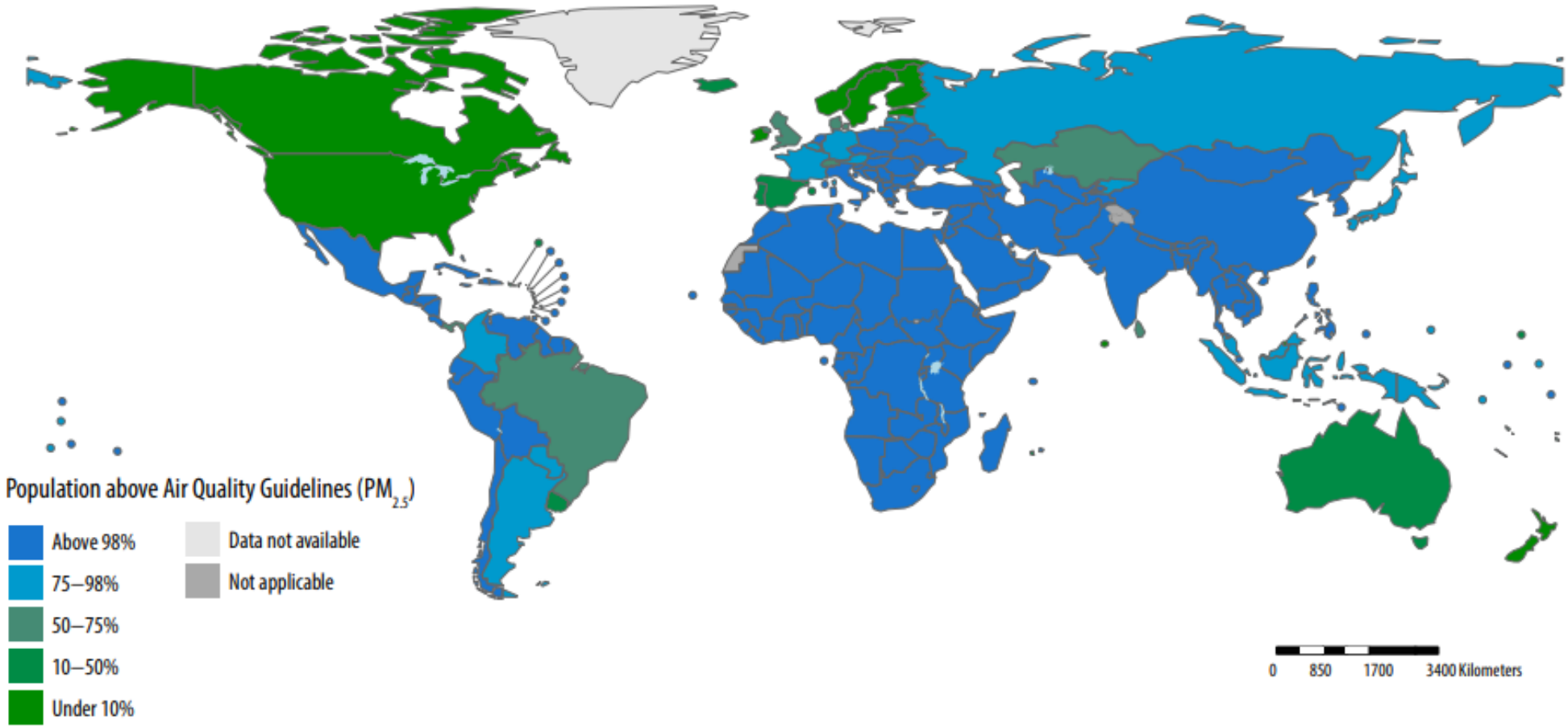




https://www.eea.europa.eu/publications/status-of-air-quality-in-Europe-2022/europes-air-quality-status-2022

World Health Organization

Fig. 1. Proportions of children under 5 years living in areas in which the WHO air quality guidelines ($PM_{2.5}$) are exceeded, by country, 2016



World Health Organization. (2018). Air pollution and child health: prescribing clean air: summary. World Health Organization. https://apps.who.int/iris/handle/10665/275545.

Major outdoor/indoor pollutants and related health effects

Pollutant	Major sources	Health effects
Particulate matter	Outdoor	Lung cancer
	Vehicular traffic Organic matter and fossil fuel combustion Power stations/industry Windblown dust from roadways, agriculture and construction Bushfires/dust storms	Premature death Mortality for cardiorespiratory diseases Reduced lung function Lower airway inflammation Upper airways irritation Neurological, cardiovascular diseases, metabolic disorders
	Indoor	
	Woodstoves Organic matter and fossil fuel combustion for heating/cooking ETS	
Nitrogen dioxide	Outdoor	Exacerbation of asthma
	Vehicular traffic Power stations/industry	Airway inflammation Bronchial hyperresponsiveness Increased susceptibility to respiratory infection Reduced lung function
	Indoor	
	Unvented gas/kerosene appliances	

Viegi G, et al. Indoor and outdoor pollution. In "ERS Handbook, Respiratory Medicine 3rd Edition", Palange P, Rohde G eds., European Respiratory Society, Sheffield (UK) 2019: pp 771-778.

Pollutant	Major sources	Health effects
Ozone	Outdoor	Lung tissue damage
	Sunlight chemical reaction with other pollutants Vehicular traffic Power stations/industry Consumer products	Reduced lung function Reduced exercise capacity Exacerbation of asthma Upper airway and eye irritation
Carbon monoxide	Outdoor	Death/coma at very high levels
	Organic matter and fossil fuel combustion Vehicular traffic Domestic heating	Headache, nausea, breathlessness, confusion/reduced mental alertness
	Indoor	Low birth weight (fetal exposure)
	Organic matter and fossil fuel combustion for heating/cooking Woodstoves Unvented gas/kerosene appliances ETS	

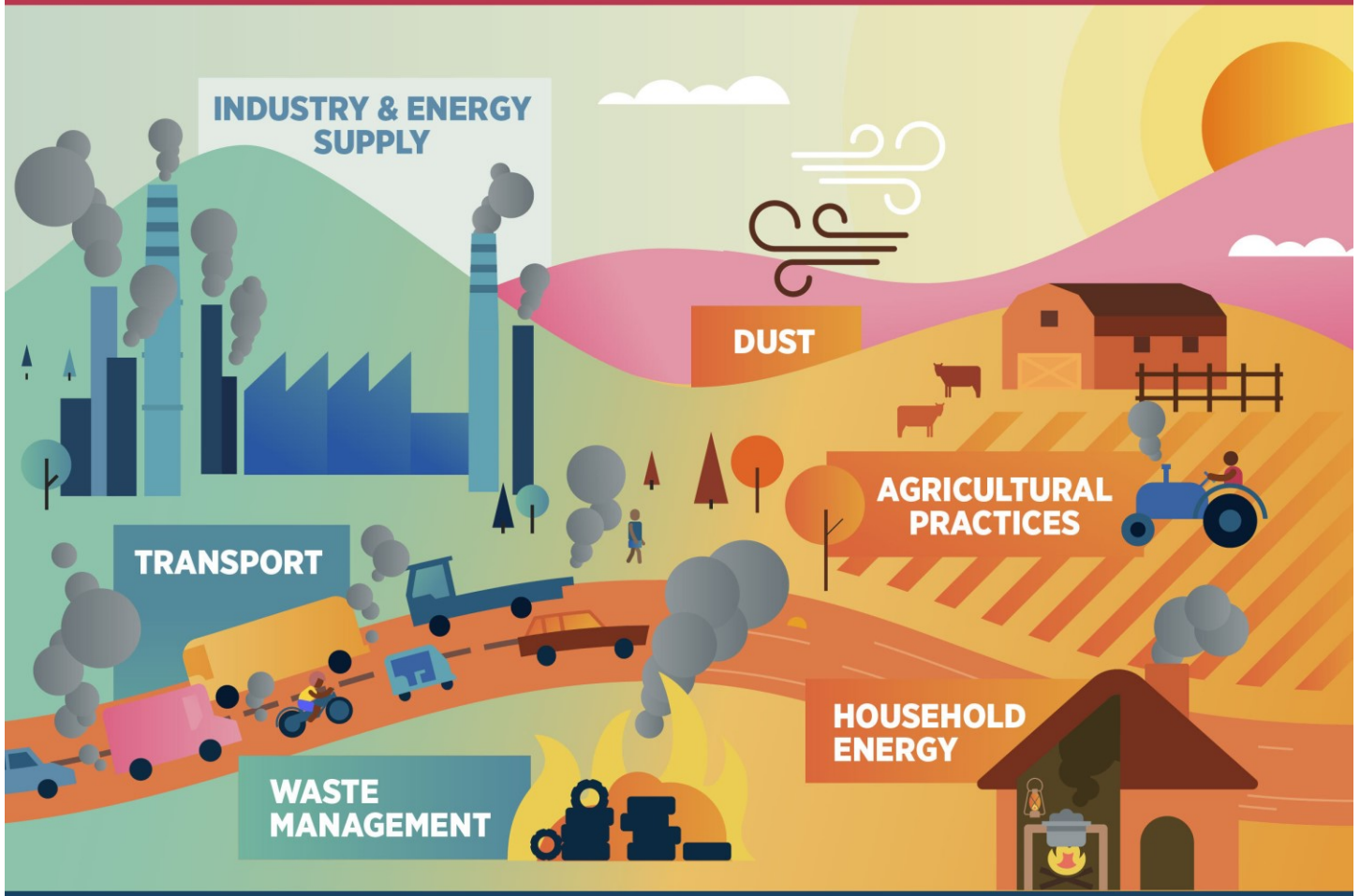
Viegi G, et al. Indoor and outdoor pollution. In "ERS Handbook, Respiratory Medicine 3rd Edition", Palange P, Rohde G eds., European Respiratory Society, Sheffield (UK) 2019: pp 771-778.

Pollutant	Major sources	Health effects
Sulfur dioxide	Outdoor Coal/oil-burning power stations Industry/refineries Diesel engines Metal smelting	Exacerbation of respiratory diseases including asthma Respiratory tract irritation
VOCs	Indoor Building materials and products such as new furniture, solvents, paint, adhesives, insulation Cleaning activities and products Materials for offices	Lung cancer Asthma, dizziness, respiratory and lung diseases Chronic eye, lung or skin irritation Neurological and reproductive disorders

Viegi G, et al. Indoor and outdoor pollution. In "ERS Handbook, Respiratory Medicine 3rd Edition", Palange P, Rohde G eds., European Respiratory Society, Sheffield (UK) 2019: pp 771-778.

World Health Organization

SOURCES OF AIR POLLUTION ARE A GLOBAL CHALLENGE WE MUST TACKLE TOGETHER



WHO Air Quality Guidelines set goals to protect millions of lives from air pollution.

CLEAN AIR FOR HEALTH #AirPollution World Health Organization

European Environment Agency

2 / Some 60 % of SOx come from **energy production and distribution.**

3 / Many natural phenomena, including volcanic eruptions and sand storms, release air pollutants into the atmosphere.

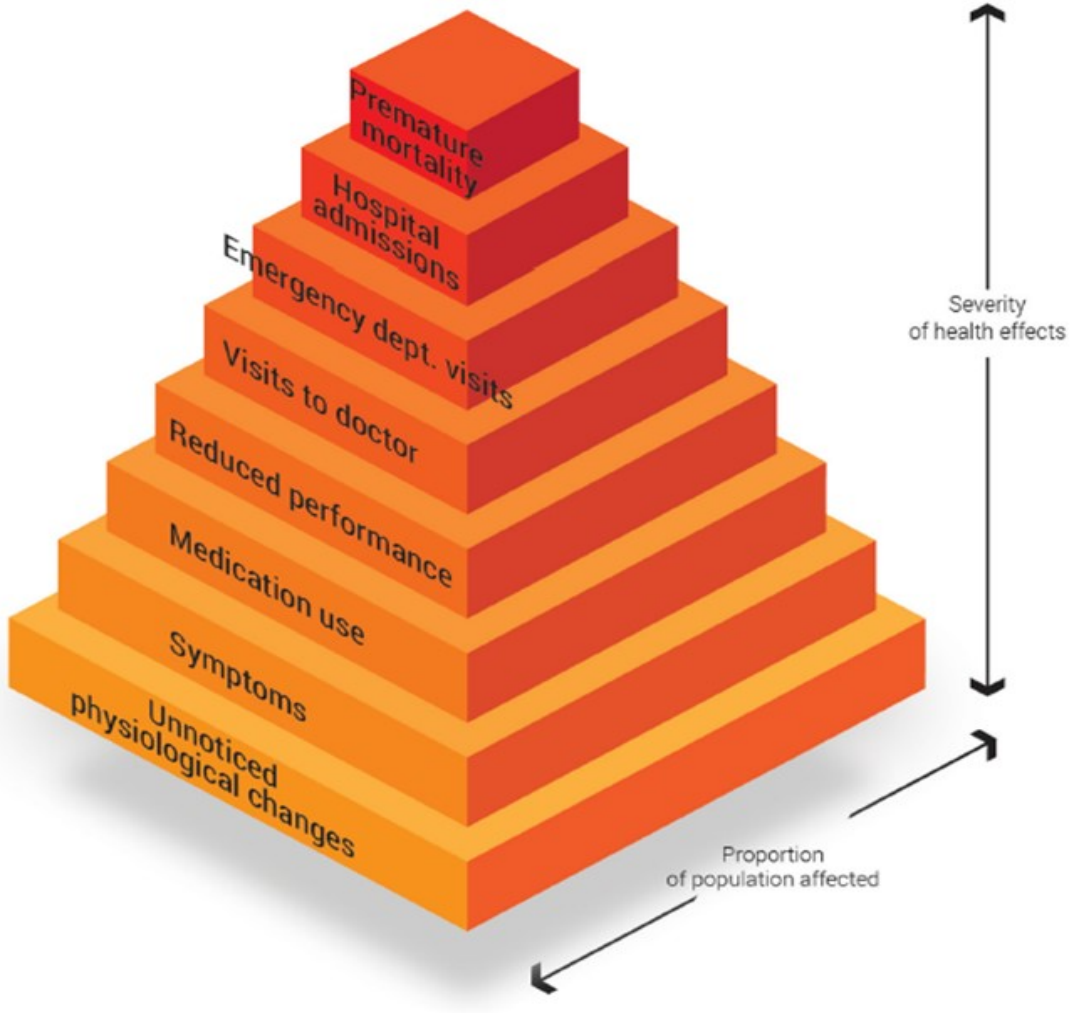
1 / Around 90 % of NH3 emissions and 80% of CH4 emissions come from **agricultural activities**.

6 / Fuel combustion is a key contributor to air pollution — from road transport, households to energy use and production.

5 / More than 40 % of emissions of NOx come from **road transport**. Almost 40 % of primary PM2.5 emissions come from transport.

4 / **Waste (landfills), coal mining and long-distance gas transmission** are sources of methane.

La piramide degli effetti sanitari associati con l'inquinamento atmosferico



Van Brusselen D, et al. PloS ONE 2016; 11;11(5):e0154052.

A joint ERS/ATS policy statement: what constitutes an adverse health effect of air pollution? An analytical framework

George D. Thurston[1], Howard Kipen[2], Isabella Annesi-Maesano[3], John Balmes[4,5], Robert D. Brook[6], Kevin Cromar[7], Sara De Matteis[8], Francesco Forastiere[9], Bertil Forsberg[10], Mark W. Frampton[11], Jonathan Grigg[12], Dick Heederik[13], Frank J. Kelly[14], Nino Kuenzli[15,16], Robert Laumbach[2], Annette Peters[17], Sanjay T. Rajagopalan[18], David Rich[19], Beate Ritz[20], Jonathan M. Samet[21], Thomas Sandstrom[11], Torben Sigsgaard[22], Jordi Sunyer[23] and Bert Brunekreef[13,24]

Eur Respir J 2017;

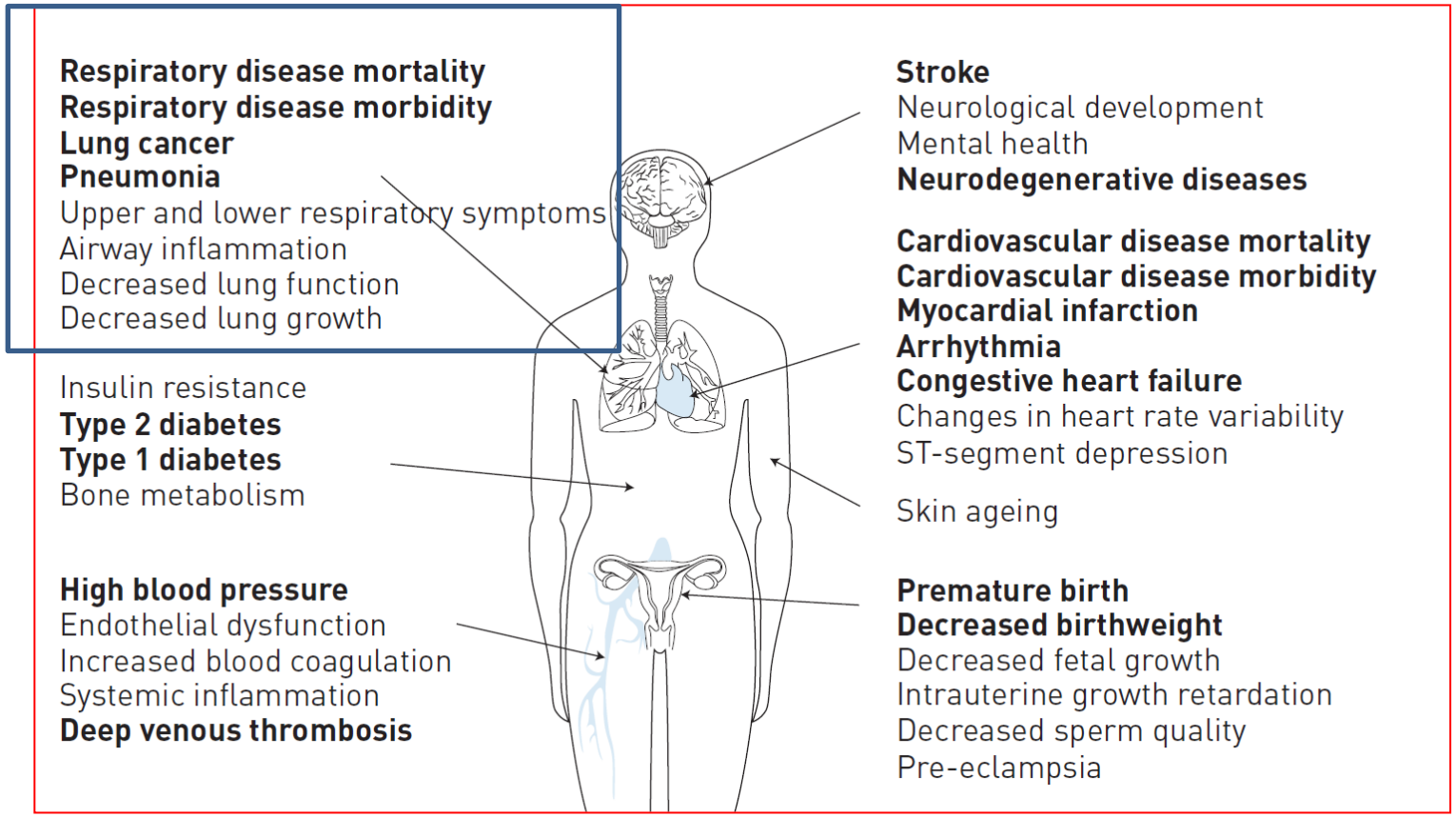
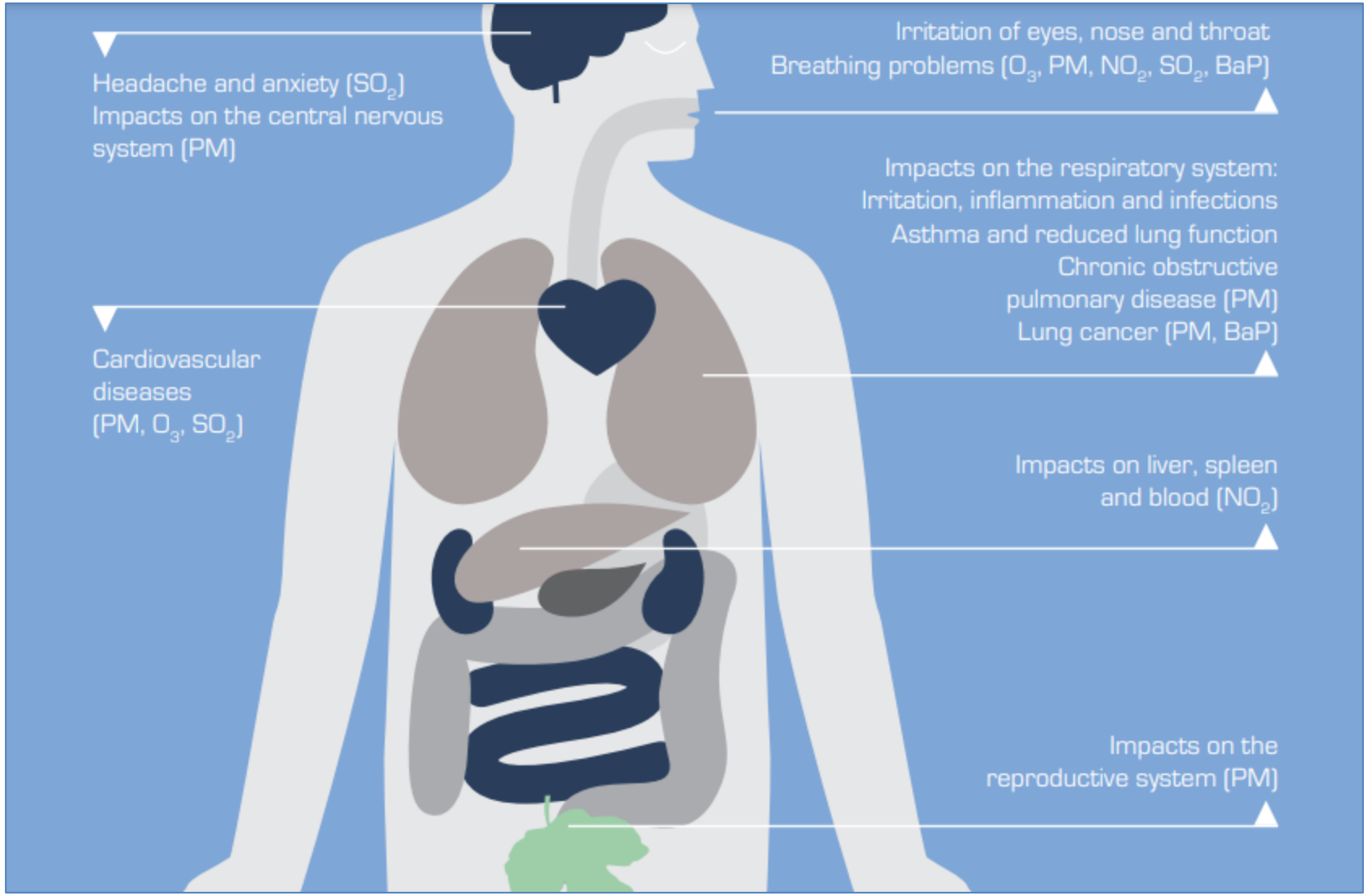
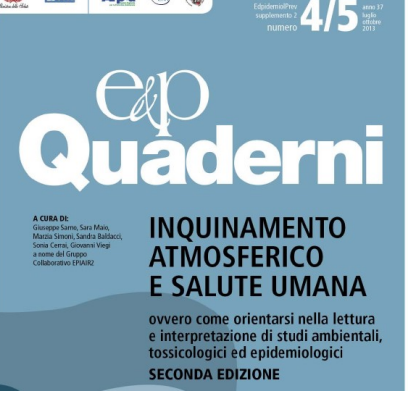


FIGURE 1. Overview of diseases, conditions and biomarkers affected by outdoor air pollution. Bold type indicates conditions currently included in the Global Burden of Disease categories.


Headache and anxiety (SO_2)
Impacts on the central nervous system (PM)
Irritation of eyes, nose and throat
Breathing problems (O_3, PM, NO_2, SO_2, BaP)
Impacts on the respiratory system:
Irritation, inflammation and infections
Asthma and reduced lung function
Chronic obstructive pulmonary disease (PM)
Lung cancer (PM, BaP)
Cardiovascular diseases (PM, O_3, SO_2)
Impacts on liver, spleen and blood (NO_2)
Impacts on the reproductive system (PM)



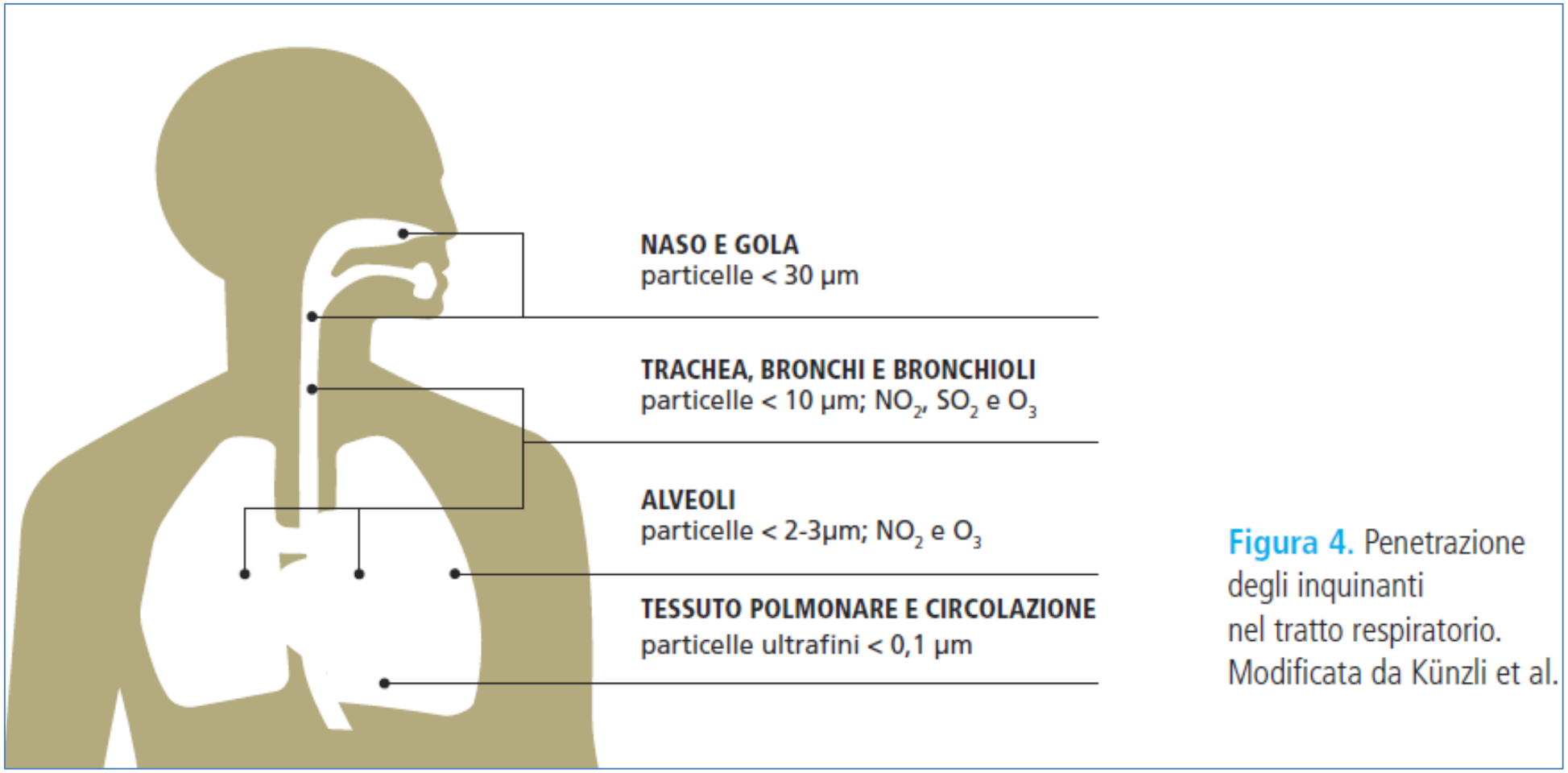
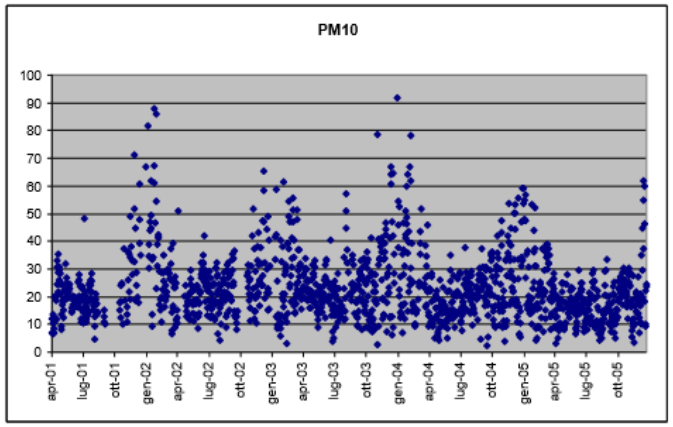


Figura 4. Penetrazione degli inquinanti nel tratto respiratorio. Modificata da Künzli et al.

Sarno G, Maio S, Simoni M, Baldacci S, Cerrai S, Viegi G a nome del Gruppo collaborativo EPIAIR2.
Inquinamento atmosferico e salute umana. Ovvero come orientarsi nella lettura e interpretazione di studi ambientali, tossicologici ed epidemiologici. Edizione seconda. Epidemiol &Prev 2013;4/5(suppl 2):1-86

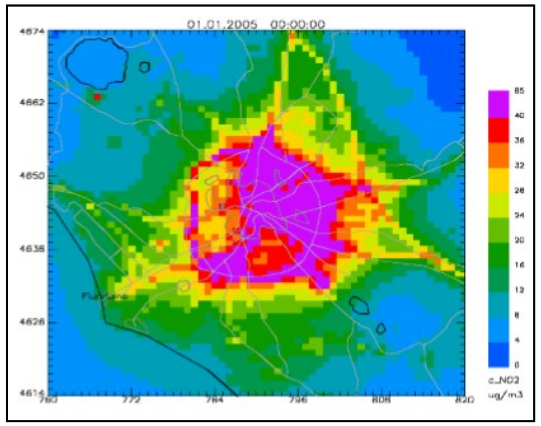
GLI EFFETTI DELL'INQUINAMENTO ATMOSFERICO SONO CLASSIFICATI IN:

1. effetti a breve termine



Effetti acuti, differenze temporali

2. effetti a lungo termine



Effetti cronici, differenze spaziali

Environmental Science and Pollution Research (2021) 28:19615–19628
https://doi.org/10.1007/s11356-021-13208-x

REVIEW ARTICLE

Air pollution exposure—the (in)visible risk factor for respiratory diseases

Gabriel-Petrică Bălă[1] · Ruxandra-Mioara Râjnoveanu[2] · Emanuela Tudorache[1] · Radu Motișan[3] · Cristian Oancea[1]

The goal of review was to summarize the most important air pollutants and their impact on the main respiratory diseases to reduce both short- and the long-term exposure consequences

Table 1 Associations between air pollutants and respiratory diseases

Authors	Disease	Air pollutant association	Ethnicity/nationality
Liang et al. (2019)	COPD	PM2.5	China
Huang et al. (2019)	COPD	PM2.5	Taiwan
Havet et al. (2019)	Asthma	PM10, O_3	France
Cadelis et al. (2014)	Asthma	PM10, PM2.5–10	Caribbean
Akpinar-Elci et al. (2015)	Asthma	PM10, PM2.5–10	Caribbean
Guarnieri and Balmes (2014)	Asthma	PM2.5, PM 10	Meta-analyses
Xing et al. (2019)	Lung cancer	PM2.5, PM10, O_3	China
Hamra et al. (2014)	Lung cancer	PM2.5	Meta-analyses
Gharibvand et al. (2017)	Lung cancer	PM2.5	USA, Canada
Wang et al. (2019a)	Lung cancer	PM2.5	China
Winterbottom et al. (2018)	IPF	PM10	USA
Johannson et al. (2018)	IPF	NO_2, PM2.5, PM10	USA
Johannson et al. (2014)	IPF	O_3, NO_2	South Korea
Nsoh et al. (2019)	Respiratory infections	PM2.5	Cameroon
Z. Zhang et al. (2019)	Respiratory infections	PM2.5, PM2.5–PM10	China
Zheng et al. (2017)	Respiratory infections	PM10, NO_2, SO_2	China
Goeminne et al. (2018)	Bronchiectasis	PM10, NO_2	UK
Garcia-Olivé et al. (2018)	Bronchiectasis	SO_2	Spain
Popovic et al. (2019)	Tuberculosis	PM2.5	Asia, Europe, North America
Zhu et al. (2018)	Tuberculosis	PM10, NO_2, SO_2	China
Lai et al. (2016)	Tuberculosis	PM2.5	Taiwan
Jassal et al. (2012)	Tuberculosis	PM2.5	USA
Li et al. (2019)	Tuberculosis	PM2.5	China
Yao et al. (2019)	Tuberculosis	PM2.5, PM10, O_3, CO	China

Clinical Reviews in Allergy & Immunology (2022) 62:72–89
https://doi.org/10.1007/s12016-020-08830-5

External Environmental Pollution as a Risk Factor for Asthma

Jose Chatkin[1] · Liana Correa[2] · Ubiratan Santos[3]

Table 2 Consequences of outdoor air pollution over allergic rhinitis and asthma (adapted from Eguiluz-Gracia et al. [22])

Environmental factors	Health outcomes
Pollution from traffic and industry (PM_{10}, $PM_{2.5}$, NO, NO_2)	
During childhood	Higher asthma prevalence after the school age
During adulthood	Possibly higher asthma prevalence
Lifelong	Poorer lung function Higher rate of asthma exacerbations Conflicting results on AR onset
Livestock farming (organic dust, toxins form microorganisms, gases like ammonia and methane)	Decreased lung function
Black carbon	Possibly epigenetic changes leading to increased type two inflammation in children
Interaction between air pollutants (PM_{10}, nitrogen oxides) and allergens (pollen, fungal spores)	
Production of more pollen, more allergens per pollen grain, and more PALMs per pollen grain	Potentially, facilitation of IgE sensitization against aeroallergens Higher rate of asthma-related hospitalizations

Table 5 Effects of outdoor air pollutants on asthma outcomes if legal concentrations are exceeded (adapted from Tiotiu et al. [96])

Pollutant	Concentration (μg/m^3)	Asthma symptoms	Exacerbations	Hospitalizations	Lung function
O_3	100 (8-h mean)	-	↑	↑	↓
NO_2	200 (1-h mean)	↑	↑	↑	↓
CO	30 (1-h mean)	-	↑	-	
SO_2	20 (24-h mean)	↑	↑	↑	↓
$PM_{2.5}$	10 (annual mean) 25 (24-h mean)	↑	↑	↑	↓
PM_{10}	10 (annual mean) 50 (24-h mean)	↑	↑	↑	↓

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REVIEW

Issue 1 - "Update on adverse respiratory effects of outdoor air pollution". Part 1): Outdoor air pollution and respiratory diseases: A general update and an Italian perspective

S. De Matteis[a,b], F. Forastiere[c,d], S. Baldacci[e], S. Maio[e], S. Tagliaferro[e], S. Fasola[d], G. Cilluffo[d], S. La Grutta[d], G. Viegi[d,e,*]

Summarize of the results for the systematic reviews that have evaluated exposure to various pollutants and mortality (all causes mortality as well as mortality from respiratory diseases, COPD, ALRI, and lung cancer)

Table 1 Association between long-term and short-term exposure to specific pollutants and mortality. Systematic reviews for the update WHO AQG. Relative risks (RR) are per 10 $\mu g/m^3$ exposure.

Mortality/pollutant	Long-term			Short-term		
	No.of studies	RR	95% CI	No.of studies	RR	95% CI
All-cause mortality (natural mortality)						
PM_{10}	17	1.04	1.03-1.06	66	1.0041	1.0034-1.0049
$PM_{2.5}$	25	1.08	1.06-1.09	29	1.0065	1.0044-1.0086
NO_2	24	1.02	1.01-1.04	54	1.0072	1.0059-1.0085
O_3 (annual exposure)	9	0.97	0.93-1.02			
O_3 (peak exposure)	7	1.01	1.00-1.02			
SO_2		NA		36	1.0059	1.0046-1.0071
Respiratory mortality						
PM_{10}	13	1.12	1.06-1.19	41	1.0091	1.0063-1.0119
$PM_{2.5}$	17	1.1	1.03-1.18	20	1.0073	1.0029-1.0116
NO_2	15	1.03	1.01-1.05		NA	
O_3 (annual exposure)	4	0.99	0.89-1.11		NA	
O_3 (peak exposure)	4	1.02	0.99-1.05			
SO_2				23	1.0067	1.0025-1.0109
COPD mortality						
PM_{10}	5	1.19	0.95-1.49		NA	
$PM_{2.5}$	11	1.11	1.05-1.17		NA	
NO_2	9	1.03	1.01-1.04		NA	
ALRI mortality						
PM_{10}	2	NA			NA	
$PM_{2.5}$	4	1.16	1.01-1.34		NA	
NO_2	5	1.06	1.02-1.10		NA	
Lung Cancer mortality						
PM_{10}	13	1.08	1.04-1.13		NA	
$PM_{2.5}$	15	1.12	1.07-1.16		NA	

ALRI: Acute Lower Respiratory Infection
NA: not available

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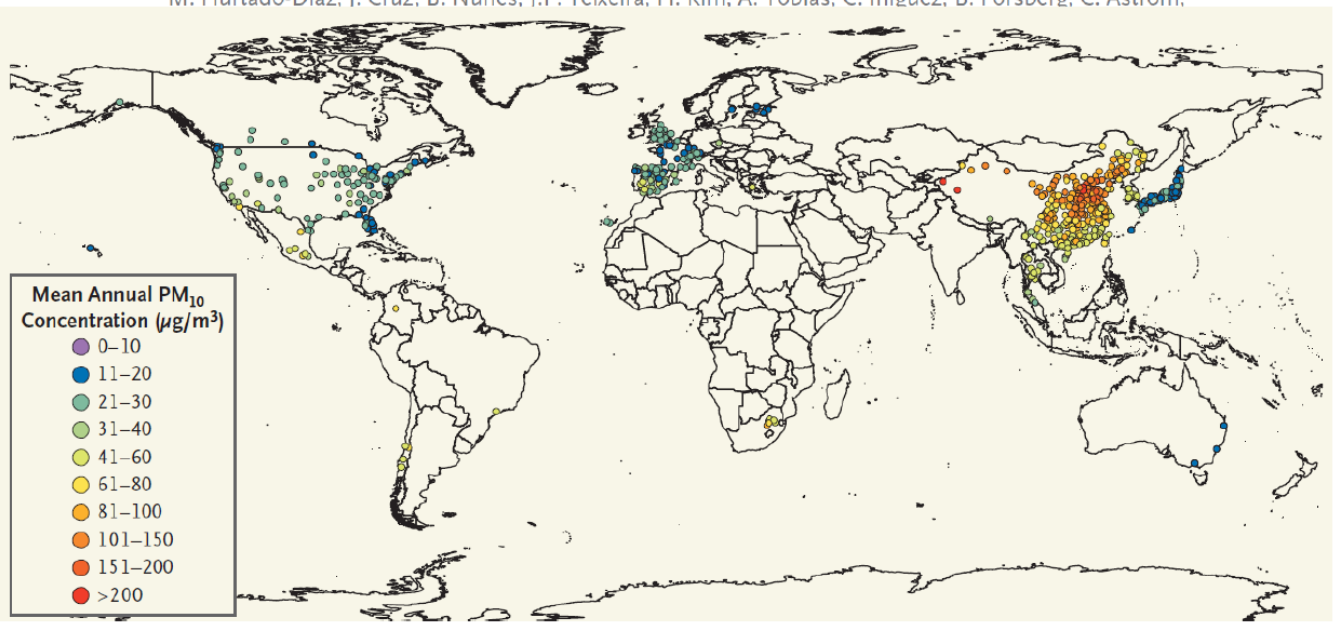
ESTABLISHED IN 1812 AUGUST 22, 2019 VOL. 381 NO. 8

Ambient Particulate Air Pollution and Daily Mortality in 652 Cities

C. Liu, R. Chen, F. Sera, A.M. Vicedo-Cabrera, Y. Guo, S. Tong, M.S.Z.S. Coelho, P.H.N. Saldiva, E. Lavigne, P. Matus, N. Valdes Ortega, S. Osorio Garcia, M. Pascal, M. Stafoggia, M. Scortichini, M. Hashizume, Y. Honda, M. Hurtado-Díaz, I. Cruz, B. Nunes, J.P. Teixeira, H. Kim, A. Tobias, C. Íñiguez, B. Forsberg, C. Åström,



COUNTRY	N CITIES	PM2.5 POOLED EFFECT ESTIMATE % CHANGE IN MORTALITY PER 10 µg/m³
CANADA	25	1.70 (1.17 - 2.23)
CHINA	272	0.41 (0.32 - 0.50)
JAPAN	47	1.42 (1.05 - 1.81)
SPAIN	19	1.96 (1.18 - 2.75)
USA	107	1.58 (1.28 - 1.88)
TOTAL	**499**	**0.68 (0.59 - 0.77)**

Liu C et al. Ambient Particulate Air Pollution and Daily Mortality in 652 Cities. N Engl J Med. 2019

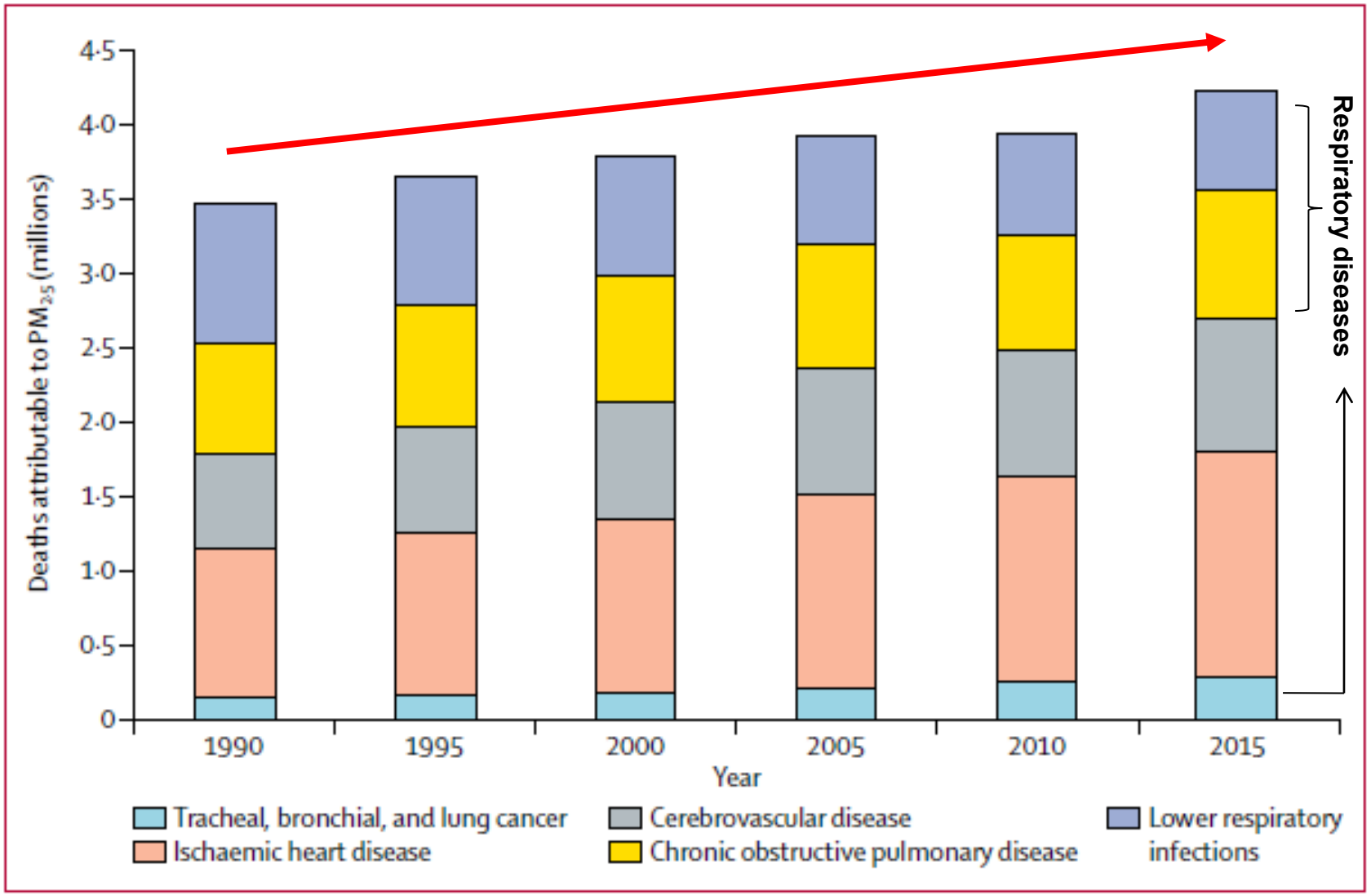


Figure 4: **Deaths attributable to ambient particulate matter pollution by year and cause**

$PM_{2.5}$=particle mass with aerodynamic diameter less than 2·5 μm.

Cohen, Lancet 2017

LETTER

doi:10.1038/nature15371

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

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

17 SEPTEMBER 2015 | VOL 525 | NATURE | 367

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

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

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

Valore doppio rispetto al 2010

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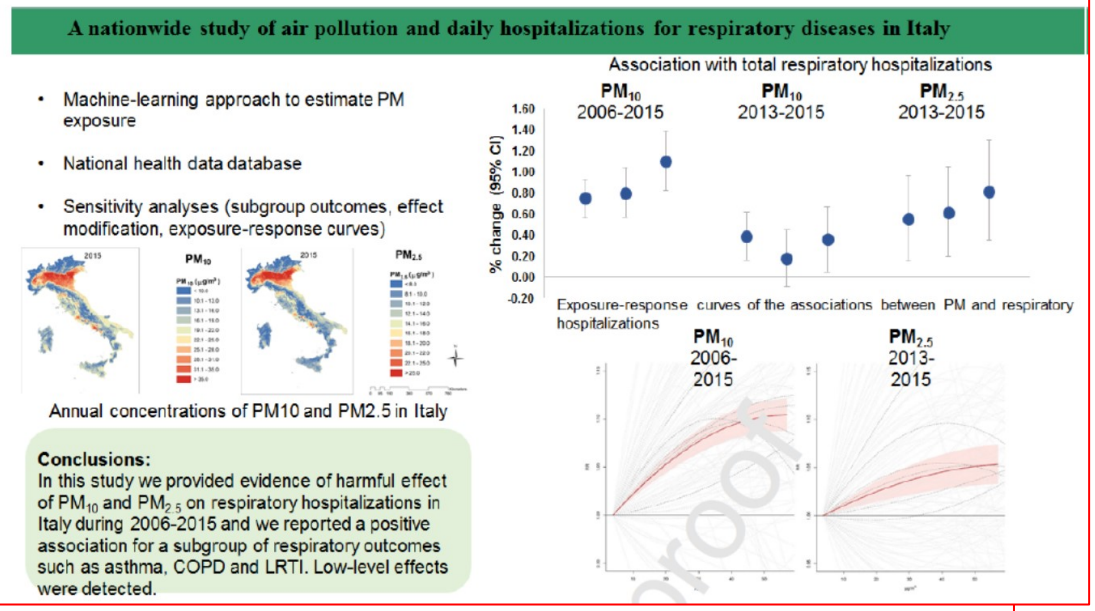
A nationwide study of air pollution from particulate matter and daily hospitalizations for respiratory diseases in Italy

Matteo Renzi [a,*], Matteo Scortichini [a], Francesco Forastiere [b,e], Francesca de' Donato [a], Paola Michelozzi [a], Marina Davoli [a], Claudio Gariazzo [c], Giovanni Viegi [b,d], Massimo Stafoggia [a], on behalf of the BEEP collaborative Group , Carla Ancona [f], Simone Bucci [f], Francesca de' Donato [f], Paola Michelozzi [f], Matteo Renzi [f], Matteo Scortichini [f], Massimo Stafoggia [f], Michela Bonafede [g], Claudio Gariazzo [g], Alessandro Marinaccio [g], Stefania Argentini [h], Roberto Sozzi [h], Sergio Bonomo [i], Salvatore Fasola [i], Francesco Forastiere [i], Stefania La Grutta [i], Giovanni Viegi [i], Achille Cernigliaro [j], Salvatore Scondotto [j], Sandra Baldacci [k], Sara Maio [k], Gaetano Licitra [l], Antonino Moro [l], Paola Angelini [m], Laura Bonvicini [n], Serena Broccoli [n], Marta Ottone [n], Paolo Giorgi Rossi [n], Annamaria Colacci [o], Federica Parmagnani [o], Andrea Ranzi [o], Claudia Galassi [p], Enrica Migliore [p], Lucia Bisceglia [q], Antonio Chieti [q], Giuseppe Brusasca [r], Giuseppe Calori [r], Sandro Finardi [r], Alessandro Nanni [r], Nicola Pepe [r], Paola Radice [r], Camillo Silibello [r], Gianni Tinarelli [r], Francesco Uboldi [r], Giuseppe Carlino [s]



ABSTRACT

Background/aim: The relationship between air pollution and respiratory morbidity has been widely addressed in urban and metropolitan areas but little is known about the effects in non-urban settings. Our aim was to assess the short-term effects of PM10 and PM2.5 on respiratory admissions in the whole country of Italy during 2006–2015.
Methods: We estimated daily PM concentrations at the municipality level using satellite data and spatiotemporal predictors. We collected daily counts of respiratory hospital admissions for each Italian municipality. We considered five different outcomes: all respiratory diseases, asthma, chronic obstructive pulmonary disease (COPD), lower and upper respiratory tract infections (LRTI and URTI). Meta-analysis of province-specific estimates obtained by time-series models, adjusting for temperature, humidity and other confounders, was applied to extrapolate national estimates for each outcome. At last, we tested for effect modification by sex, age, period, and urbanization score. Analyses for $PM_{2.5}$ were restricted to 2013–2015 cause the goodness of fit of exposure estimation.
Results: A total of 4,154,887 respiratory admission were registered during 2006–2015, of which 29% for LRTI, 12% for COPD, 6% for URTI, and 3% for asthma. Daily mean PM_{10} and $PM_{2.5}$ concentrations over the study period were 23.3 and 17 $\mu g/m^3$, respectively. For each 10 $\mu g/m^3$ increases in PM_{10} and $PM_{2.5}$ at lag 0–5 days, we found excess risks of total respiratory diseases equal to 1.20% (95% confidence intervals, 0.92, 1.49) and 1.22% (0.76, 1.68), respectively. The effects for the specific diseases were similar, with the strongest ones for asthma and COPD. Higher effects were found in the elderly and in less urbanized areas.
Conclusions: Short-term exposure to PM is harmful for the respiratory system throughout an entire country, especially in elderly patients. Strong effects can be found also in less urbanized areas.

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Short-term exposure to nitrogen dioxide and mortality: A systematic review and meta-analysis

Mingrui Wang [a], Haomin Li [b], Shiwen Huang [a], Yaoyao Qian [a], Kyle Steenland [a], Yang Xie [c], Stefania Papatheodorou [d, 1], Liuhua Shi [a, *, 1]

ABSTRACT

Background: Ambient air pollution has been characterized as a leading cause of mortality worldwide and has been associated with cardiovascular and respiratory diseases. There is increasing evidence that short-term exposure to nitrogen dioxide (NO_2), is related to adverse health effects and mortality.

Methods: We conducted a systematic review of short-term NO_2 and daily mortality, which were indexed in PubMed and Embase up to June 2021. We calculated random-effects estimates by different continents and globally, and tested for heterogeneity and publication bias.

Results: We included 87 articles in our quantitative analysis. NO_2 and all-cause as well as cause-specific mortality were positively associated in the main analysis. For all-cause mortality, a 10 ppb increase in NO_2 was associated with a 1.58% (95%CI 1.28%–1.88%, $I^2 = 96.3\%$, Eggers' test $p < 0.01$, $N = 57$) increase in the risk of death. For cause-specific mortality, a 10 ppb increase in NO_2 was associated with a 1.72% (95%CI 1.41%–2.04%, $I^2 = 87.4\%$, Eggers' test $p < 0.01$, $N = 42$) increase in cardiovascular mortality and a 2.05% (95%CI 1.52%–2.59%, $I^2 = 78.5\%$, Eggers' test $p < 0.01$, $N = 38$) increase in respiratory mortality. In the sensitivity analysis, the meta-estimates for all-cause mortality, cardiovascular and respiratory mortality were nearly identical. The heterogeneity would decline to varying degrees through regional and study-design stratification.

Conclusions: This study provides evidence of an association between short-term exposure to NO_2, a proxy for traffic-sourced air pollutants, and all-cause, cardiovascular and respiratory mortality.

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Volume 691, 15 November 2019, Pages 549-561

Review

Effects of ambient ozone concentrations with different averaging times on asthma exacerbations: A meta-analysis

Xing Li [a, b], Qing Chen [b], Xueyan Zheng [c], Yongzhi Li [b], Min Han [b], Tao Liu [a], Jianpeng Xiao [a], Lingchuan Guo [a], Weilin Zeng [a], Junfeng Zhang (Jim) [d, e] ✉, Wenjun Ma [a] ✉

Abstract

Background: Mounting evidence suggests that short-term exposure to ozone increases the risk of asthma exacerbations. However, ozone exposures have been assessed using ambient ozone concentrations averaged over different time periods in different studies.

Objective: To evaluate the risks for asthma exacerbations related to ambient ozone measured as 1-hour or 8-hour daily maximum and 24-hour average concentrations.

Methods: Based on a literature search in PubMed, EMBASE and Web of Science, we identified all time-series studies as of December 4th, 2018 and included 47 eligible studies in our analyses. Asthma exacerbation is defined as the risk for emergency room visits or hospital admissions. Pooled relative risks (RRs) and 95% confidence intervals (95%CIs) for a 10 μg/m3 increase in daily ozone concentration were estimated using random effect models. Subgroup analyses and sensitivity analyses were also performed to examine the risks for different seasons, regions and age groups and for the robustness of our main findings.

Results: Significant and similar associations were found for O3-1 h max (RR,1.012; 95%CI, 1.005-1.019) and O3-8 h max (RR, 1.011; 95%CI, 1.007-1.014), while marginal effect was identified for O3-24 h average (RR, 1.005; 95%CI, 0.996-1.014). No significant publication bias but high heterogeneities were observed. During the warm season, ozone was significantly associated with asthma exacerbation. O3-1 h max had the highest RR of 1.014 (95%CI, 1.005-1.024), followed by O3-8 h max (RR, 1.012; 95%CI, 1.009-1.016), while marginal association was identified for O3-24 h avg (RR, 1.008; 95%CI, 0.998-1.017). During the cold season, null associations were identified for all the three averaging times. Variations were also observed in region and age.

Conclusion: Ozone exposure measured as 1-hour or 8-hour daily max were more consistently associated with asthma exacerbations than 24-hour average exposure during the warm season.

Review > Int J Chron Obstruct Pulmon Dis. 2016 Dec 12;11:3079-3091.
doi: 10.2147/COPD.S122282. eCollection 2016.

Major air pollutants and risk of COPD exacerbations: a systematic review and meta-analysis

Jinhui Li [1], Shengzhi Sun [1], Robert Tang [1], Hong Qiu [2], Qingyuan Huang [3], Tonya G Mason [2], Linwei Tian [1]

Abstract

Background: Short-term exposure to major air pollutants (O_3, CO, NO_2, SO_2, PM_{10}, and $PM_{2.5}$) has been associated with respiratory risk. However, evidence on the risk of chronic obstructive pulmonary disease (COPD) exacerbations is still limited. The present study aimed at evaluating the associations between short-term exposure to major air pollutants and the risk of COPD exacerbations.

Methods: After a systematic search up until March 30, 2016, in both English and Chinese electronic databases such as PubMed, EMBASE, and CNKI, the pooled relative risks and 95% confidence intervals were estimated by using the random-effects model. In addition, the population-attributable fractions (PAFs) were also calculated, and a subgroup analysis was conducted. Heterogeneity was assessed by I^2.

Results: In total, 59 studies were included. In the single-pollutant model, the risks of COPD were calculated by each 10 μg/m^3 increase in pollutant concentrations, with the exception of CO (100 μg/m^3). There was a significant association between short-term exposure and COPD exacerbation risk for all the gaseous and particulate pollutants. The associations were strongest at lag0 and lag3 for gaseous and particulate air pollutants, respectively. The subgroup analysis not only further confirmed the overall adverse effects but also reduced the heterogeneities obviously. When 100% exposure was assumed, PAFs ranged from 0.60% to 4.31%, depending on the pollutants. The adverse health effects of SO_2 and NO_2 exposure were more significant in low-/middle-income countries than in high-income countries: SO_2, relative risk: 1.012 (95% confidence interval: 1.001, 1.023); and NO_2, relative risk: 1.019 (95% confidence interval: 1.014, 1.024).

Conclusion: Short-term exposure to air pollutants increases the burden of risk of COPD acute exacerbations significantly. Controlling ambient air pollution would provide benefits to COPD patients.

Keywords: COPD exacerbations; acute exposure; air pollution; meta-analysis.

Meta-Analysis > Occup Environ Med. 2019 Aug;76(8):583-591.
doi: 10.1136/oemed-2018-105420. Epub 2019 Jun 12.

Effect of outdoor particulate air pollution on FEV_1 in healthy adults: a systematic review and meta-analysis

Stefan Edginton [1], Dylan E O'Sullivan [2], Will King [2], M Diane Lougheed [1 2 3]

Abstract

The effect of acute and long-term exposures to outdoor particulate air pollution on lung function in healthy adults is not well established. The objective of this study was to conduct a systematic literature review and meta-analysis of studies that assessed the relationship of outdoor particulate air pollution and lung function in healthy adults. Studies that contained data on outdoor air particulate matter levels (PM_{10} or $PM_{2.5}$) and forced expiratory volume in 1 s (FEV_1) in healthy adults were eligible for inclusion. Effect estimates, in relation to long-term and acute exposures, were quantified separately using random effects models. A total of 27 effect estimates from 23 studies were included in this review. Acute exposures were typically assessed with $PM_{2.5}$, while long-term exposures were predominantly represented by PM_{10} A 10 µg/m3 increase in short-term $PM_{2.5}$ exposure (days) was associated with a -7.02 mL (95% CI -11.75 to -2.29) change in FEV_1 A 10 $\mu g/m^3$ difference in long-term PM_{10} exposure was associated with a -8.72 mL (95% CI -15.39 to -2.07) annual change in FEV_1 and an absolute difference in FEV_1 of -71.36 mL (95% CI -134.47 to -8.24). This study provides evidence that acute and long-term exposure to outdoor particulate air pollution are associated with decreased FEV_1 in healthy adults. Residual confounding from other risk factors, such as smoking, may explain some of the effect for long-term exposures. More studies are required to determine the relationship of long-term exposure to $PM_{2.5}$ and short-term exposure to PM_{10}, which may have different biologic mechanisms.

Meta-Analysis > Environ Res. 2021 Jan;192:110296. doi: 10.1016/j.envres.2020.110296.
Epub 2020 Oct 5.

The effect of acute outdoor air pollution on peak expiratory flow in individuals with asthma: A systematic review and meta-analysis

Stefan Edginton [1], Dylan E O'Sullivan [2], Will D King [2], M Diane Lougheed [3]

Abstract

Objectives: Acute exposures to outdoor air pollution have been shown to reduce lung function in children with asthma, but the effect on adults with asthma has not been established in a meta-analysis. The objective of this study was to conduct a systematic literature review and meta-analysis of studies that assessed the relationship of outdoor air pollution and peak expiratory flow (PEF) in adults with asthma.

Methods: Studies that contained data on outdoor air pollution levels (PM_{10}, $PM_{2.5}$, or NO_2) and PEF in adults with asthma were eligible for inclusion. Effect estimates were quantified for each air pollution measure using random effects models. Heterogeneity was investigated with the Q-test and I^2 statistics. Meta-regression and subgroup analyses were conducted to determine differences in effect by air pollution measures and the inclusion of smokers.

Results: A total of 22 effect estimates from 15 studies were included in this review. A 10 $\mu g/m^3$ increase in acute PM_{10} exposure was associated with a -0.19 L/min (95% CI: 0.30, -0.09) change in PEF. For both PM_{10} and $PM_{2.5}$, the inclusion of current smokers was a significant source of heterogeneity among studies (meta-regression: $p = 0.04$ and $p = 0.03$). Among studies that only included non-smokers, a 10 $\mu g/m^3$ increase in acute exposure to PM_{10} and $PM_{2.5}$ was associated with changes in PEF of -0.25 L/min (95% CI: 0.38, -0.13) and -1.02 L/min (95% CI: 1.79, -0.24), respectively.

Conclusions: This study provides evidence that acute increases in PM_{10} and $PM_{2.5}$ levels are associated with decreases in PEF in adults with asthma, particularly among non-smokers.

Review > J Allergy Clin Immunol. 2021 Jul;148(1):1-14. doi: 10.1016/j.jaci.2021.05.006.

Air pollution and lung function in children

Erika Garcia [1], Mary B Rice [2], Diane R Gold [3]

Box 2. Short-term ambient air pollution exposure and children's lung function

Summary observations

- Short-term exposure to O_3 and particle pollution ($PM_{2.5}$ and PM_{10}) has been associated with lower lung function, including lower FEV_1, FVC, and FEV_1/FVC, in generally healthy children and in those with asthma
- Large wildfires are occurring at increasing frequency and are a major source of PM and other pollutants
- $PM_{2.5}$ levels during wildfires can reach extreme concentrations that far exceed air quality standards and are associated with acute decrements in peak expiratory flow
- Short-term elevations in NO_2 levels outdoors and indoors have been associated with reduction in child lung function

Knowledge gaps

- Need to improve understanding of the extent to which observed acute effects of NO_2 on child lung function are causal, due to confounding by correlated pollutants (eg, $PM_{2.5}$), or represents a pollution mixture that is causal (eg, traffic pollution)
- Need to evaluate the long-term consequences of repeated exposure to wildfire smoke on child lung function

Box 3. Long-term ambient air pollution and children's lung function

Summary observations

- A large number of studies support a relation between early-life or long-term air pollution exposures and subsequent children's lung function level, with the most epidemiologic evidence for $PM_{2.5}$ and NO_2
- There is inconsistent evidence of an effect of early-life or long-term exposure to O_3 on subsequent children's lung function, although animal model studies suggest prenatal or early-life O_3 effects on lung development
- Associations with long-term exposures are more consistent for FEV_1 compared with for FVC, which may indicate greater impacts on airway caliber/airflow obstruction than overall lung size or growth

Knowledge gaps

- Although there is evidence that air pollution exposure at all ages in childhood can have adverse lung function effects, few studies have evaluated whether timing or cumulative period of exposure influences whether pollution effects are fixed or reversible
- Long-term consequences of air pollution on reduced lung function development/growth/trajectories in childhood are not fully understood; studies need to investigate impacts of childhood exposures on lung function in adulthood as well as risk for later respiratory health, such as chronic obstructive pulmonary disease, emphysema, fibrosis, or pulmonary vascular disease
- Need to further assess the toxicity of specific PM components and sources on lung function

Environ Res. 2021 Jun;197:111123. doi: 10.1016/j.envres.2021.111123. Epub 2021 Apr 3.

Global PM2.5-attributable health burden from 1990 to 2017: Estimates from the Global Burden of disease study 2017

Xiang Bu [1], Zhonglei Xie [2], Jing Liu [3], Linyan Wei [3], Xiqiang Wang [3], Mingwei Chen [4], Hui Ren [5]

Table 1
The death cases and age-standardized death rate of PM2.5-attributable diseases in 1990 and 2017, and temporal trends from 1990 to 2017.

Characteristics	1990		2017		1990–2017
	Death cases No.×10^6 (95% UI)	ASR per 100,000 No. (95% UI)	Death cases No.×10^6 (95% UI)	ASR per 100,000 No. (95% UI)	EAPC No. (95% CI)
Overall	4.46(3.99–4.85)	104.79(94.04–114.28)	4.58(4.13–5.03)	59.62(53.70–65.62)	−2.15 (−2.21 to −2.09)
Sex					
Male	2.36(2.12–2.58)	122.91(109.92–135.66)	2.55(2.28–2.83)	72.35(64.81–80.43)	−2 (−2.08 to −1.93)
Female	2.10(1.87–2.32)	90.88(81.05–100.05)	2.03(1.82–2.25)	48.95(43.87–54.00)	−2.39 (−2.45 to −2.33)
Social-demographic index					
Low	0.95(0.80–1.07)	208.97(180.65–237.39)	0.96(0.85–1.06)	138.84(121.79–154.66)	−1.54 (−1.59 to −1.5)
Low-middle	1.08(0.95–1.18)	163.77(147.34–180.45)	1.11(1.00–1.22)	100.22(89.54–110.17)	−1.88 (−1.95 to −1.8)
Middle	1.15(1.03–1.26)	125.63(111.22–138.24)	1.25(1.10–1.40)	62.81(55.06–70.48)	−2.53 (−2.58 to −2.49)
High-middle	0.82(0.73–0.91)	97.25(85.60–107.90)	0.85(0.75–0.95)	50.36(44.37–56.47)	−2.64 (−2.78 to −2.5)
High	0.45(0.38–0.52)	34.51(29.36–40.09)	0.39(0.31–0.46)	15.91(12.76–19.10)	−2.86 (−2.94 to −2.79)
Source of PM2.5					
Ambient	1.75(1.48–2.03)	43.68(36.88–50.85)	2.94(2.50–3.36)	38.15(32.50–43.68)	−0.58 (−0.67 to −0.49)
Household	2.71(2.34–3.06)	61.11(53.03–69.21)	1.64(1.40–1.93)	21.47(18.32–25.19)	−2.15 (−2.21 to −2.09)
PM2.5-attributable diseases					
LRI	1.56(1.30–1.80)	28.65(23.00–32.88)	0.89(0.75–1.04)	12.42(10.00–14.43)	−3.12 (−3.22 to −3.01)
Cancer	0.22(0.17–0.28)	5.45(4.00–6.84)	0.35(0.25–0.45)	4.39(3.00–5.65)	−0.62 (−0.7 to −0.54)
COPD	1.01(0.74–1.24)	27.86(20.00–34.08)	1.00(0.68–1.28)	13.06(8.00–16.78)	−3.08 (−3.21 to −2.96)
Stroke	0.57(0.47–0.66)	14.46(11.00–16.99)	0.68(0.55–0.81)	8.58(6.00–10.26)	−1.98 (−2.09 to −1.88)
IHD	0.99(0.88–1.11)	25.63(22.00–28.74)	1.39(1.23–1.55)	17.62(15.00–19.72)	−1.34 (−1.39 to −1.29)
Diabetes	0.10(0.07–0.12)	2.74(1.00–3.29)	0.28(0.19–0.33)	3.54(2.00–4.23)	0.94 (0.79–1.09)

Table 2
The DALYs and age-standardized DALYs rate of PM2.5-attributable diseases in 1990 and 2017, and its temporal trends from 1990 to 2017.

Characteristics	1990		2017		1990–2017
	DALY cases No.×10^6 (95% UI)	ASR per 100,000 No. (95% UI)	DALY cases No.×10^6 (95% UI)	ASR per 100,000 No. (95% UI)	EAPC No. (95% CI)
Overall	190.27 (166.96–210.83)	3676.99 (3255.40–4029.28)	142.52 (128.77–156.12)	1856.61 (1671.45–2029.71)	−2.58 (−2.64 to −2.51)
Sex					
Male	100.11(88.02–110.97)	3991.33 (3542.08–4394.01)	78.89(71.21–86.76)	2105.53 (1897.89–2313.58)	−2.39(-2.46 to −2.33)
Female	90.17(78.64–100.87)	3404.57 (2975.46–3776.42)	63.63(56.51–69.79)	1628.58 (1444.55–1791.31)	−2.8 (−2.86 to −2.74)
Social-demographic index					
Low	55.09(45.76–63.49)	7205.62 (6156.75–8118.90)	38.93(34.45–43.05)	3920.00 (3495.24–4308.92)	−2.29 (−2.33 to −2.24)
Low-middle	54.14(46.39–60.82)	5470.13 (4840.50–6018.09)	40.26(35.71–44.79)	2897.78 (2587.95–3179.11)	−2.4 (−2.46 to −2.33)
Middle	46.56(41.12–51.70)	3663.28 (3252.78–4034.82)	33.48(29.25–37.29)	1549.78 (1356.56–1725.82)	−3.12 (−3.21 to −3.03)
High-middle	23.76(21.19–26.16)	2452.27 (2183.55–2698.88)	20.90(18.38–23.46)	1191.41 (1045.61–1337.63)	−2.88 (−3 to −2.77)
High	9.92(8.32–11.48)	796.03(668.16–919.39)	8.41(6.48–10.20)	415.56(321.04–503.39)	−2.3 (−2.37 to −2.23)
Source of PM2.5					
Ambient	62.77(52.25–73.40)	1287.52 (1080.36–1493.88)	83.05(71.39–94.31)	1064.92(913.91–1213.48)	−0.85 (−0.78 to −0.7)
Household	127.50 (107.52–146.49)	2389.47 (2036.32–2728.80)	59.47(50.85–68.95)	791.69(675.13–918.75)	−2.64 (−2.58 to −2.51)
PM2.5-attributable diseases					
LRI	113.10(92.00–131.34)	1838.47 (1503.11–2133.03)	44.41(36.83–51.73)	642.18(531.48–748.19)	−3.88 (−4.03 to −3.73)
Cancer	5.69(4.27–7.10)	132.21(99.20–164.91)	7.85(5.61–10.04)	96.18(68.75–122.98)	−1.07 (−1.13 to −1.02)
COPD	25.72(19.01–31.42)	631.46(466.65–770.68)	25.12(17.26–31.94)	315.31(216.40–401.23)	−2.72 (−2.83 to −2.61)
Stroke	14.23(11.79–16.63)	333.93(278.54–390.63)	16.39(13.22–19.51)	201.89(164.10–240.13)	−1.95 (−2.04 to −1.87)
IHD	24.13(21.59–26.88)	565.44(506.00–629.24)	32.09(28.65–35.87)	395.15(352.23–441.32)	−1.33 (−1.37 to −1.28)
Diabetes	6.47(4.32–8.45)	151.35(101.22–197.13)	15.23(10.02–19.88)	187.70(123.45–244.52)	0.88 (0.76–0.99)
Blindness and vision impairment	0.94(0.51–1.43)	24.13(13.10–36.35)	1.44(0.73–2.25)	18.20(9.30–28.50)	−0.99 (−1.14 to −0.84)

Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project

Beelen et al

$PM_{2.5}$

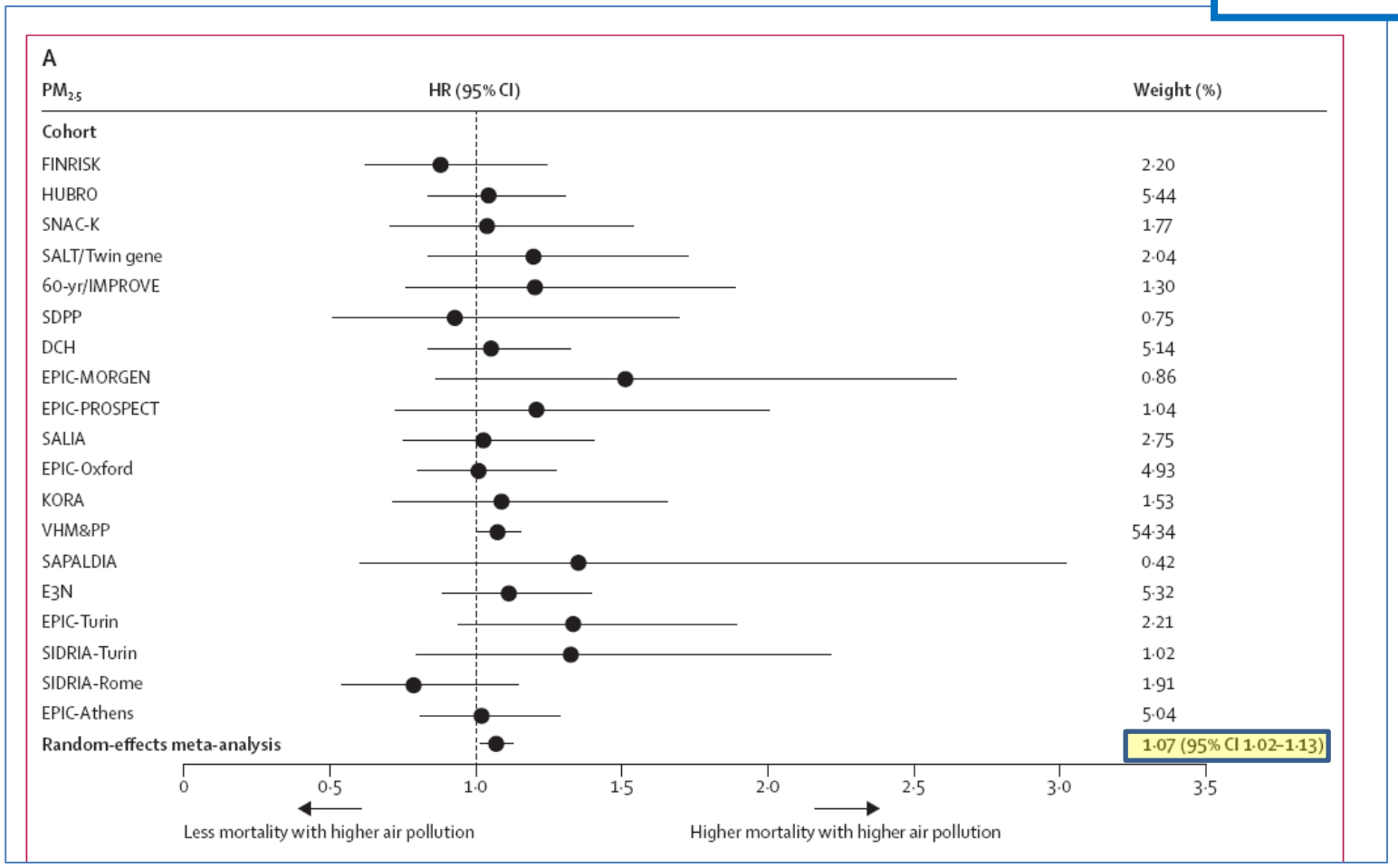


Figure 2: **Adjusted association between natural cause mortality and exposure to $PM_{2.5}$ and NO_2 (with main model 3)—results from cohort-specific analyses and random-effects meta-analyses**

(A) Exposure to $PM_{2.5}$. (B) Exposure to NO_2. HR=hazard ratio. HRs are presented per 5 μg/m³ for $PM_{2.5}$ and per 10 μg/m³ for NO_2. The number of observations was 322 159 in the $PM_{2.5}$ analysis and 367 251 in the NO_2 analysis. Particulate matter concentrations were not available for the EPIC-Umeå, EPIC-Varese, or EPIC-San Sebastian cohorts. For E3N and SAPALDIA, particulate matter concentrations were available for part of the cohort.

Lancet 2014; 383: 785–95

PREVENTING DISEASE THROUGH HEALTHY ENVIRONMENTS

A global assessment of the burden of disease from environmental risks

World Health Organization

LUNG CANCER

17%

Household air pollution
Method: CRA

14%

Ambient air pollution
Method: CRA

7%

Residential Radon
Method: CRA

7%

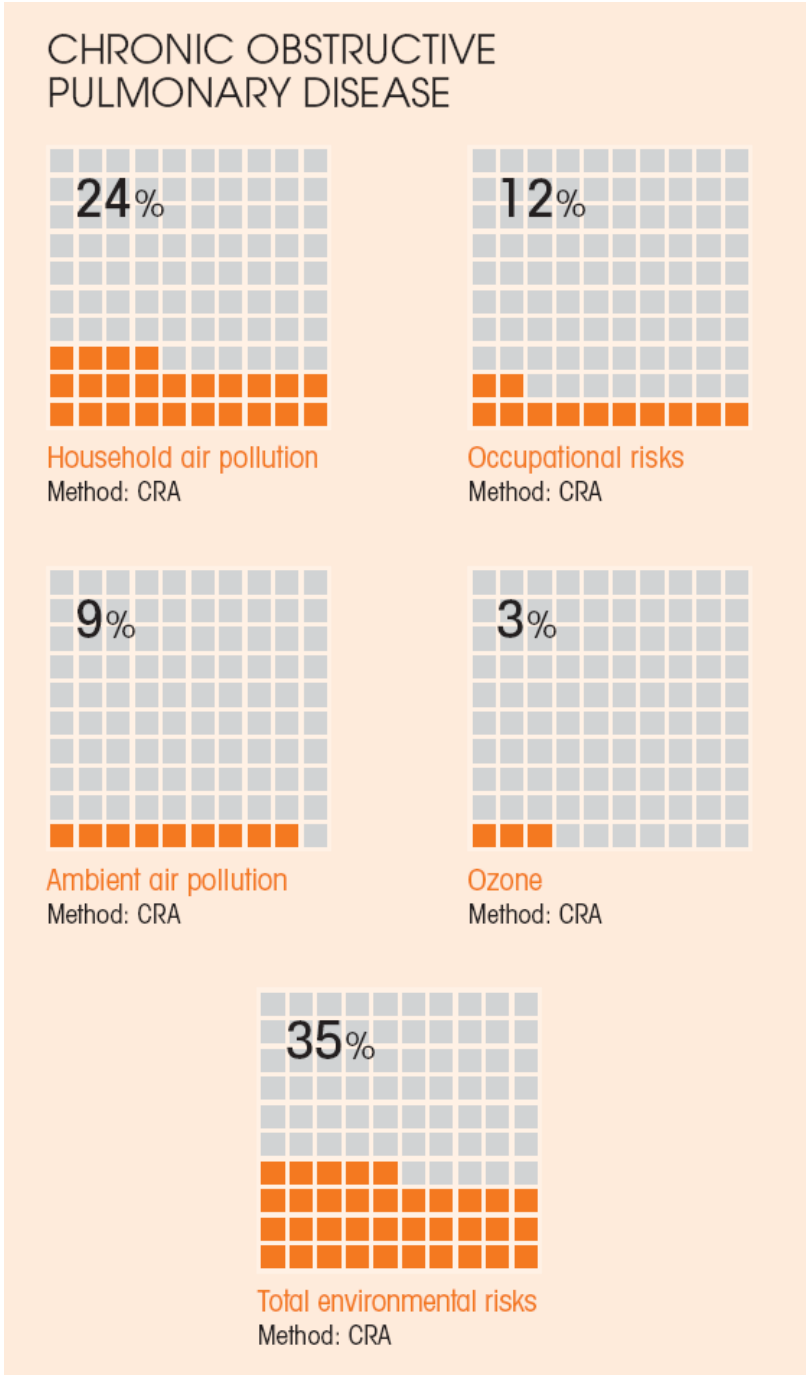
Occupational risks
Method: combination of data from CRAs

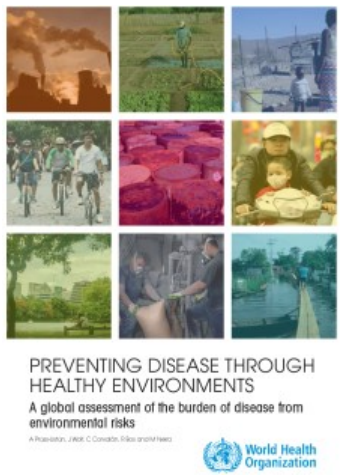
2%

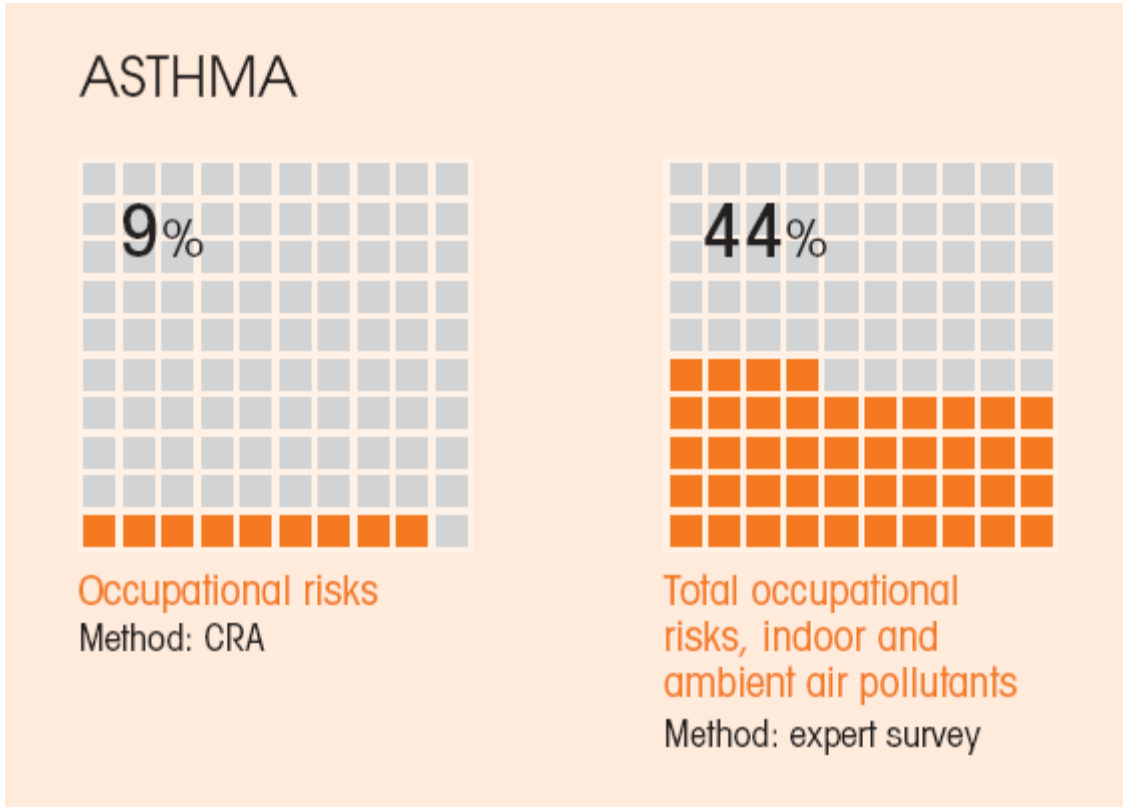
Second-hand tobacco smoke
Method: CRA

14% of lung cancers are attributable to ambient air pollution

Ambient air pollution was estimated to cause 9% of the COPD burden







44% of asthma is attributable to occupational risks, indoor and ambient air pollutants

Meta-Analysis > Environ Res. 2021 Mar;194:110703. doi: 10.1016/j.envres.2020.110703. Epub 2021 Jan 6.

Impact of long-term exposure to ambient air pollution on the incidence of chronic obstructive pulmonary disease: A systematic review and meta-analysis

Jimyung Park [1], Hyung-Jun Kim [2], Chang-Hoon Lee [1], Chang Hyun Lee [3], Hyun Woo Lee [4]

Abstract

Background: It is well known that air pollution causes respiratory morbidity and mortality by inducing airway inflammation. However, whether long-term exposure to air pollution is associated with increased incidence of chronic obstructive pulmonary disease (COPD) is controversial.

Methods: We conducted a systematic review and meta-analysis with a random-effects model to calculate the pooled risk estimates of COPD development per 10 $\mu g/m^3$ increase in individual air pollutants. PubMed, Embase, and Cochrane Library were searched from the date of their inception to August 2019 to identify long-term (at least three years of observation) prospective longitudinal studies that reported the risk of COPD development due to exposure to air pollutants. The air pollutants studied included particulate matter ($PM_{2.5}$ and PM_{10}) and nitrogen dioxide (NO_2).

Results: Of the 436 studies identified, seven met our eligibility criteria. Among the seven studies, six, three, and five had data on $PM_{2.5}$, PM_{10}, and NO_2, respectively. The meta-analysis results showed that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ is associated with increased incidence of COPD (pooled HR 1.18, 95% CI 1.13-1.23). We also noted that a 10 $\mu g/m^3$ increase in NO_2 is marginally associated with increased incidence of COPD (pooled HR 1.07, 95% CI 1.00-1.16). PM_{10} seems to have no significant impact on the incidence of COPD (pooled HR 0.95, 95% CI 0.83-1.08), although the number of studies was too small. Meta-regression analysis found no significant effect modifiers.

Conclusions: Long-term exposure to $PM_{2.5}$ and NO_2 can be associated with increased incidence of COPD.

Eur Respir J. 2021 Jun 4;57(6):2003099. doi: 10.1183/13993003.03099-2020. Print 2021 Jun.

Long-term exposure to low-level air pollution and incidence of asthma: the ELAPSE project

Shuo Liu [1], Jeanette Therming Jørgensen [1], Petter Ljungman [2 3], Göran Pershagen [2 4], Tom Bellander [2 4], Karin Leander [2], Patrik K E Magnusson [5], Debora Rizzuto [6 7], Ulla A Hvidtfeldt [8], Ole Raaschou-Nielsen [8 9], Kathrin Wolf [10], Barbara Hoffmann [11], Bert Brunekreef [12], Maciej Strak [12 13], Jie Chen [12], Amar Mehta [1], Richard W Atkinson [14], Mariska Bauwelinck [15], Raphaëlle Varraso [16], Marie-Christine Boutron-Ruault [16 17], Jørgen Brandt [9 18], Giulia Cesaroni [19], Francesco Forastiere [19], Daniela Fecht [20], John Gulliver [21 22], Ole Hertel [9], Kees de Hoogh [23], Nicole A H Janssen [13], Klea Katsouyanni [24], Matthias Ketzel [9 25], Jochem O Klompmaker [12 13], Gabriele Nagel [26], Bente Oftedal [27], Annette Peters [10 28], Anne Tjønneland [1 8], Sophia P Rodopoulou [24], Evangelia Samoli [24], Doris Tove Kristoffersen [29], Torben Sigsgaard [30], Massimo Stafoggia [2 19], Danielle Vienneau [23], Gudrun Weinmayr [26], Gerard Hoek [12], Zorana Jovanovic Andersen [31 32]

Abstract

Background: Long-term exposure to ambient air pollution has been linked to childhood-onset asthma, although evidence is still insufficient. Within the multicentre project Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE), we examined the associations of long-term exposures to particulate matter with a diameter <2.5 µm ($PM_{2.5}$), nitrogen dioxide (NO_2) and black carbon (BC) with asthma incidence in adults.

Methods: We pooled data from three cohorts in Denmark and Sweden with information on asthma hospital diagnoses. The average concentrations of air pollutants in 2010 were modelled by hybrid land-use regression models at participants' baseline residential addresses. Associations of air pollution exposures with asthma incidence were explored with Cox proportional hazard models, adjusting for potential confounders.

Results: Of 98 326 participants, 1965 developed asthma during a mean follow-up of 16.6 years. We observed associations in fully adjusted models with hazard ratios of 1.22 (95% CI 1.04-1.43) per 5 $\mu g \cdot m^{-3}$ for $PM_{2.5}$, 1.17 (95% CI 1.10-1.25) per 10 $\mu g \cdot m^{-3}$ for NO_2 and 1.15 (95% CI 1.08-1.23) per 0.5×10^{-5} m^{-1} for BC. Hazard ratios were larger in cohort subsets with exposure levels below the European Union and US limit values and possibly World Health Organization guidelines for $PM_{2.5}$ and NO_2. NO_2 and BC estimates remained unchanged in two-pollutant models with $PM_{2.5}$, whereas $PM_{2.5}$ estimates were attenuated to unity. The concentration-response curves showed no evidence of a threshold.

Conclusions: Long-term exposure to air pollution, especially from fossil fuel combustion sources such as motorised traffic, was associated with adult-onset asthma, even at levels below the current limit values.

Review > Environ Int. 2017 Mar;100:1-31. doi: 10.1016/j.envint.2016.11.012. Epub 2016 Nov 21.

Exposure to traffic-related air pollution and risk of development of childhood asthma: A systematic review and meta-analysis

Haneen Khreis [1], Charlotte Kelly [2], James Tate [3], Roger Parslow [4], Karen Lucas [3], Mark Nieuwenhuijsen [5]

Background and objective: The question of whether children's exposure to traffic-related air pollution (TRAP) contributes to their development of asthma is unresolved. We conducted a systematic review and performed meta-analyses to analyze the association between TRAP and asthma development in childhood.

Data sources: We systematically reviewed epidemiological studies published until 8 September 2016 and available in the Embase, Ovid MEDLINE (R), and Transport databases.

Study eligibility criteria, participants, and interventions: We included studies that examined the association between children's exposure to TRAP metrics and their risk of '*asthma*' incidence or lifetime prevalence, from birth to age 18 years old.

Study appraisal and synthesis methods: We extracted key characteristics of each included study using a predefined data items template and these were tabulated. We used the Critical Appraisal Skills Programme checklists to assess the validity of each included study. Where four or more independent risk estimates were available for a continuous pollutant exposure, we conducted overall and age-specific meta-analyses, and four sensitivity analyses for each summary meta-analytic exposure-outcome association.

Results: Forty-one studies met our eligibility criteria. There was notable variability in asthma definitions, TRAP exposure assessment methods and confounder adjustment. The overall random-effects risk estimates (95% CI) were 1.08 (1.03, 1.14) per 0.5×10^{-5} m^{-1} black carbon (BC), 1.05 (1.02, 1.07) per 4 $\mu g/m^3$ nitrogen dioxide (NO_2), 1.48 (0.89, 2.45) per 30 $\mu g/m^3$ nitrogen oxides (NO_x), 1.03 (1.01, 1.05) per 1 $\mu g/m^3$ Particulate Matter <2.5 μm in diameter ($PM_{2.5}$), and 1.05 (1.02, 1.08) per 2 $\mu g/m^3$ Particulate Matter <10 μm in diameter (PM_{10}). Sensitivity analyses supported these findings. Across the main analysis and age-specific analysis, the least heterogeneity was seen for the BC estimates, some heterogeneity for the $PM_{2.5}$ and PM_{10} estimates and the most heterogeneity for the NO_2 and NO_x estimates.

Limitations, conclusions and implication of key findings: The overall risk estimates from the meta-analyses showed statistically significant associations for BC, NO_2, $PM_{2.5}$, PM_{10} exposures and risk of asthma development. Our findings support the hypothesis that childhood exposure to TRAP contributes to their development of asthma. Future meta-analyses would benefit from greater standardization of study methods including exposure assessment harmonization, outcome harmonization, confounders' harmonization and the inclusion of all important confounders in individual studies.

Systematic review registration number: PROSPERO 2014: CRD42014015448.

The meta-analysis showed positive and statistically significant associations between asthma onset and the exposure to BC, NO_2, $PM_{2.5}$ and PM_{10}, with the least heterogeneity detected in the BC and PM analyses and the most detected in the NO_2 and NO_x analyses.

Chronic obstructive pulmonary disease and long-term exposure to traffic-related air pollution: a cohort study

Zorana J. Andersen,[1] Martin Hvidberg,[2] Steen S. Jensen,[2] Matthias Ketzel,[2] Steffen Loft,[3] Mette Sørensen,[1] Anne Tjønneland,[1] Kim Overvad,[4,5] Ole Raaschou-Nielsen[1]

Am J Respir Crit Care Med Vol 183. pp 455–461, 2011

Table 3. Association between traffic-related air pollution and COPD incidence (n = 1 797) among 52 799 Diet, Cancer and Health cohort participants.

50-64 years

	Adjusted for age HR (95% CI)	Adjusted for age, smoking status, duration, intensity and environmental tobacco smoke HR (95% CI)	Fully adjusted* HR (95% CI)
35-year mean (1971-event, censoring, or 27 June 2006)			
NO_2† (µg/m^3)	1.22 (1.15-1.29)	1.08 (1.02-1.14)	1.08 (1.02-1.14)
NO_x† (µg/m^3)	1.16 (1.11-1.22)	1.05 (1.01-1.10)	1.05 (1.01-1.10)
25-year mean (1981- event, censoring, or 27 June 2006)			
NO_2† (µg/m^3)	1.20 (1.13-1.27)	1.06 (1.01-1.13)	1.07 (1.01-1.06)
NO_x† (µg/m^3)	1.14 (1.09-1.19)	1.04 (0.99-1.09)	1.04 (0.99-1.09)
15-year mean (1991- event, censoring, or 27 June 2006)			
NO_2† (µg/m^3)	1.18 (1.11-1.24)	1.05 (0.99-1.11)	1.05 (1.00-1.11)
NO_x† (µg/m^3)	1.12 (1.07-1.17)	1.03 (0.99-1.08)	1.03 (0.99-1.09)
1-year mean at cohort baseline (1993-1997)			
Major road‡ within 50m	1.25 (1.07-1.45)	1.05 (0.90-1.22)	1.04 (0.89-1.21)
Traffic load§ within 200m	1.10 (1.06-1.14)	1.01 (0.98-1.05)	1.01 (0.97-1.05)

COPD, chronic obstructive respiratory disease; HR, hazard ratio; CI, confidence interval; NO_2, nitrogen dioxide; NO_x, nitrogen oxides; *Adjusted for smoking status, smoking duration, smoking intensity, environmental tobacco smoke, body mass index, educational level, occupational exposure, and fruit consumption; † Linear trend per interquartile range; ‡ Road with annual traffic density of 10 000 vehicles or more; § Total number of km traveled within 200m (sum of product of street length and traffic density for each road).

Allergy Asthma Immunol Res. 2015 Jul;7(4):359-66. doi: 10.4168/aair.2015.7.4.359.

Effect of Traffic-Related Air Pollution on Allergic Disease: Results of the Children's Health and Environmental Research

Dal Young Jung [1], Jong Han Leem [2], Hwan Cheol Kim [3], Jeong Hee Kim [4], Seung Sik Hwang [1], Ji Young Lee [1], Byoung Ju Kim [5], Yun Chul Hong [6], Soo Jong Hong [7], Ho Jang Kwon [8]

Purpose: This study evaluated the relationship of living near to main roads to allergic diseases, airway hyperresponsiveness (AHR), allergic sensitization, and lung function in Korean children. **Methods:** A total of 5,443 children aged 6-14 years from 33 elementary schools in 10 cities during 2005-2006 were included in a baseline survey of the Children's Health and Environmental Research. We assessed association of traffic-related air pollution (TAP) exposure with the distance to the nearest main road, total road length of main roads and the proportion of the main road area within the 200-m home area. **Results:** Positive exposure-response relationships were found between the length of the main road within the 200-m home area and lifetime wheeze (adjusted prevalence ratio [PR] for comparison of the longest to the shortest length categories=1.24; 95% CIs, 1.04-1.47; *P* for trend=0.022) and diagnosed asthma (PR=1.42; 95% CIs, 1.08-1.86; *P* for trend=0.011). Living less than 75 m from the main road was significantly associated with lifetime allergic rhinitis (AR), past-year AR symptoms, diagnosed AR, and treated AR. The distance to the main road (*P* for trend=0.001), the length of the main road (*P* for trend=0.041), and the proportion of the main road area (*P* for trend=0.006) had an exposure-response relationship with allergic sensitization. A strong inverse association was observed between residential proximity to the main load and lung function, especially FEV1, FEV1/FVC, and FEF_{25-75}. The length of the main road and the proportion of the main road area were associated with reduced FEV1 in schoolchildren. **Conclusions:** The results of this study suggest that exposure to traffic-related air pollution may be associated with increased risk of asthma, AR, and allergic sensitization, and with reduced lung function in schoolchildren.

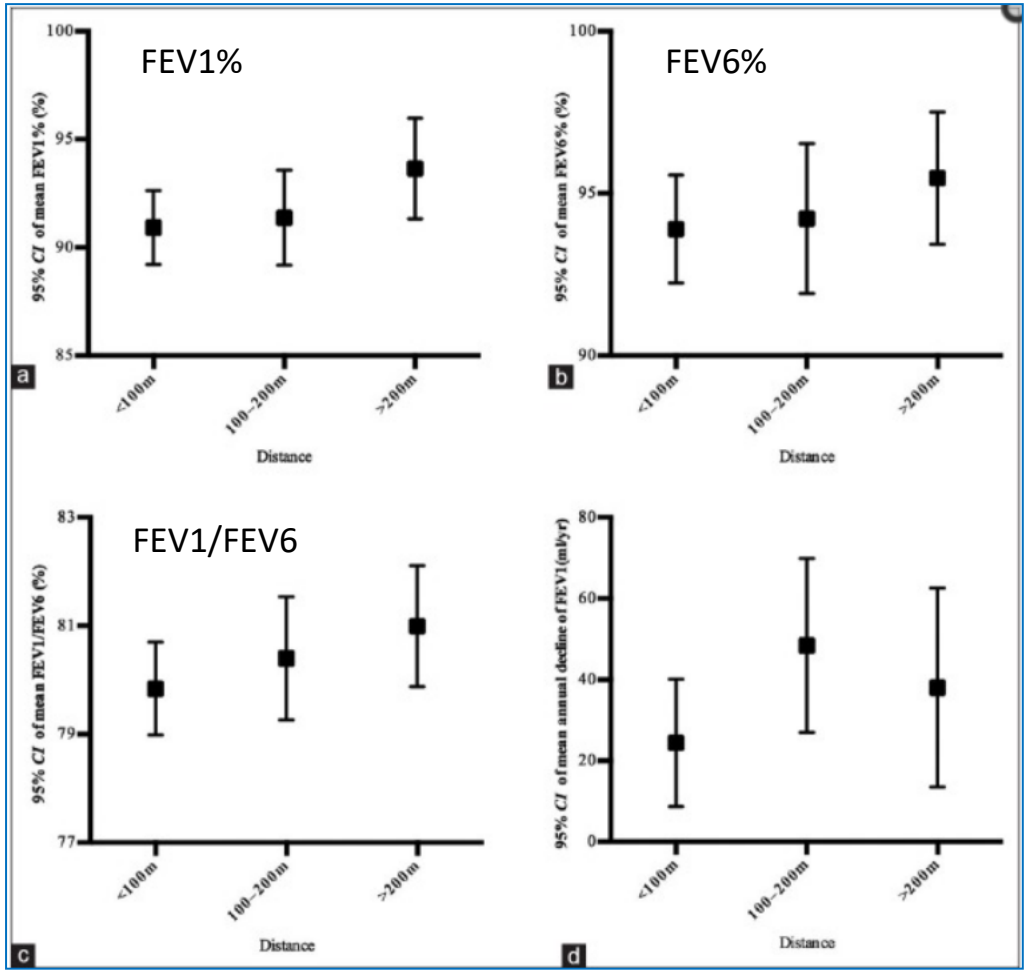
Chin Med J (Engl). 2016 Sep 20; 129(18): 2184–2190. PMCID: PMC5022339
doi: 10.4103/0366-6999.189923

Living near a Major Road in Beijing: Association with Lower Lung Function, Airway Acidification, and Chronic Cough

Zhan-Wei Hu,[1] Yan-Ni Zhao,[1] Yuan Cheng,[1] Cui-Yan Guo,[1] Xi Wang,[1] Nan Li,[1] Jun-Qing Liu,[2] Hui Kang,[2] Guo-Guang Xia,[3] Ping Hu,[3] Ping-Ji Zhang,[3] Jing Ma,[1] Ying Liu,[1] Cheng Zhang,[1] Li Su,[1] and Guang-Fa Wang[1]

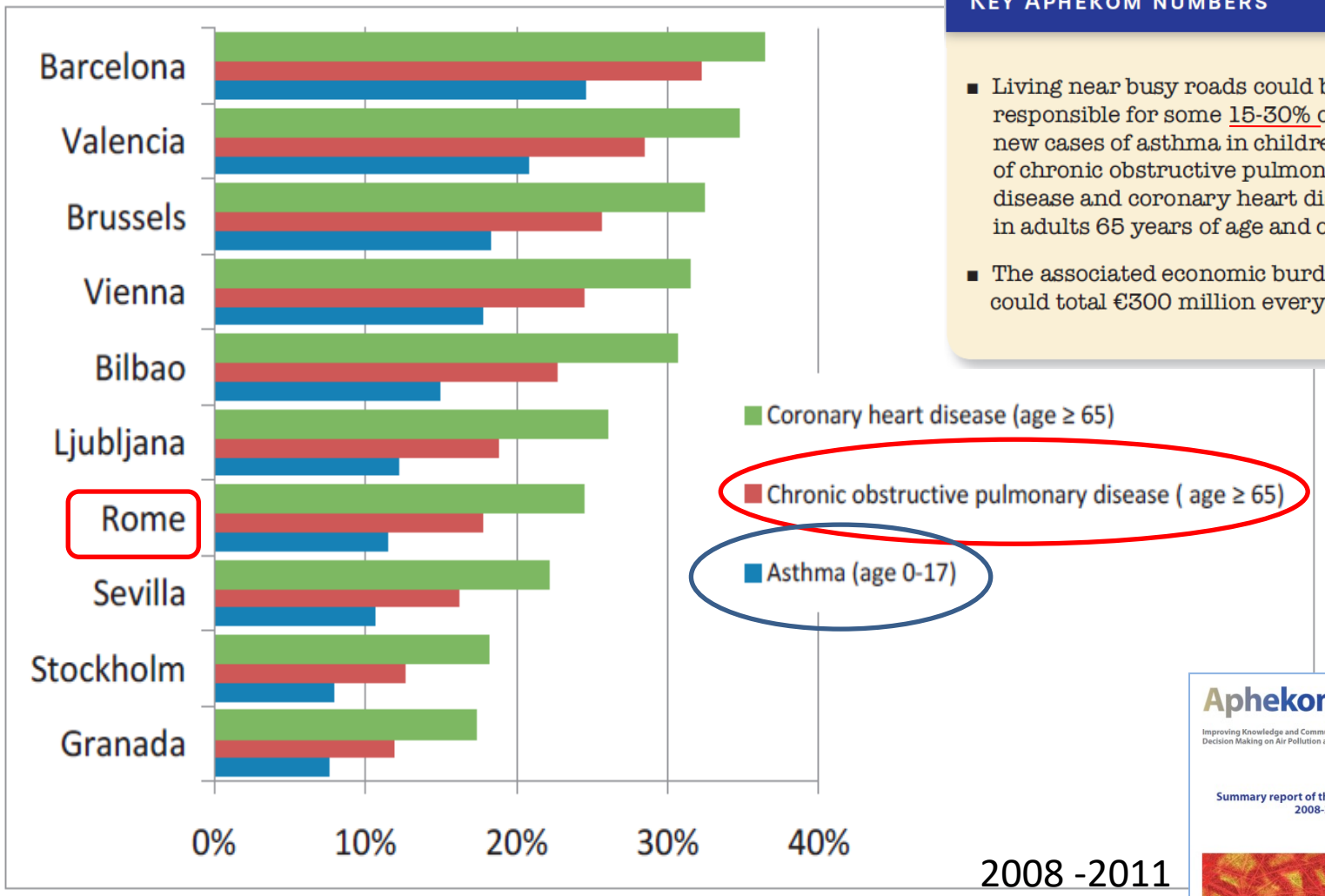
Long-term exposure to traffic-related air pollution in people who live near major roads in Beijing is associated with lower lung function, airway acidification, and a higher prevalence of chronic cough.

1003 participants



Lung function of participants with a home-road distance of <100 m, 100–200 m, and >200 m. (a-c) FEV1%, FEV6%, and FEV1/FEV6 all demonstrate an increasing trend with longer distance. (d) No clear trend can be seen in the annual decline of FEV1 (ml/year) with increased distance. Distance: Home-road distance; FEV1%: Percentage of predicted value of forced expiratory volume in 1 s; FEV6%: Percentage of predicted value of forced expiratory volume in 6 s; *CI*: Confidence interval.

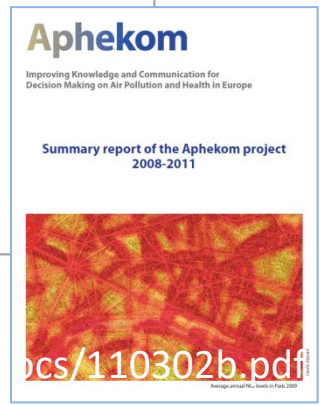
Percentage of population with chronic diseases whose disease could be attributed to living near busy streets and roads in 10 Aphekom cities



Key Aphekom numbers

- Living near busy roads could be responsible for some 15-30% of all new cases of asthma in children; and of chronic obstructive pulmonary disease and coronary heart disease in adults 65 years of age and older
- The associated economic burden could total €300 million every year

2008 -2011



ocs/110302b.pdf

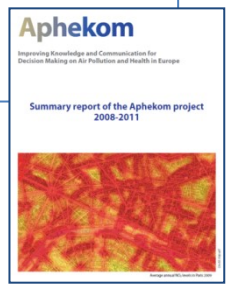
Chronic burden of near-roadway traffic pollution in 10 European cities (APHEKOM network)

Laura Perez(1) (2), Christophe Declercq (3), Carmen Iñiguez (4) (5), Inmaculada Aguilera (5) (6), Chiara Badaloni (8), Ferran Ballester (4) (5), Catherine Bouland (7), Olivier Chanel (9), FB Cirarda (10), Francesco Forastiere (8), Bertil Forsberg (11), Daniela Haluza (12), Britta Hedlund (13), Koldo Cambra (14), Marina Lacasaña (5) (15), Hanns Moshammer (12), Peter Otorepec (16), Miguel Rodríguez-Barranco (15), Sylvia Medina (3), Nino Künzli (1) (2)

ERJ Express. Published on March 21, 2013

APHEKOM

Estimated percent (95% CI) of lifetime childhood asthma attributable to near road traffic-related pollution (**A**) and estimated yearly percent exacerbations (hospital admissions, 95% CI) of childhood asthma attributable to air pollution (**B**) (10 cities in Europe, from original data in Table 5 and 6, respectively).



The impact of prenatal exposure to air pollution on childhood wheezing and asthma: A systematic review

Zhang Hehua[a], Chang Qing[b], Gao Shanyan[b], Wu Qijun[b], Zhao Yuhong[a,b,*]

Environmental Research 159 (2017) 519–530

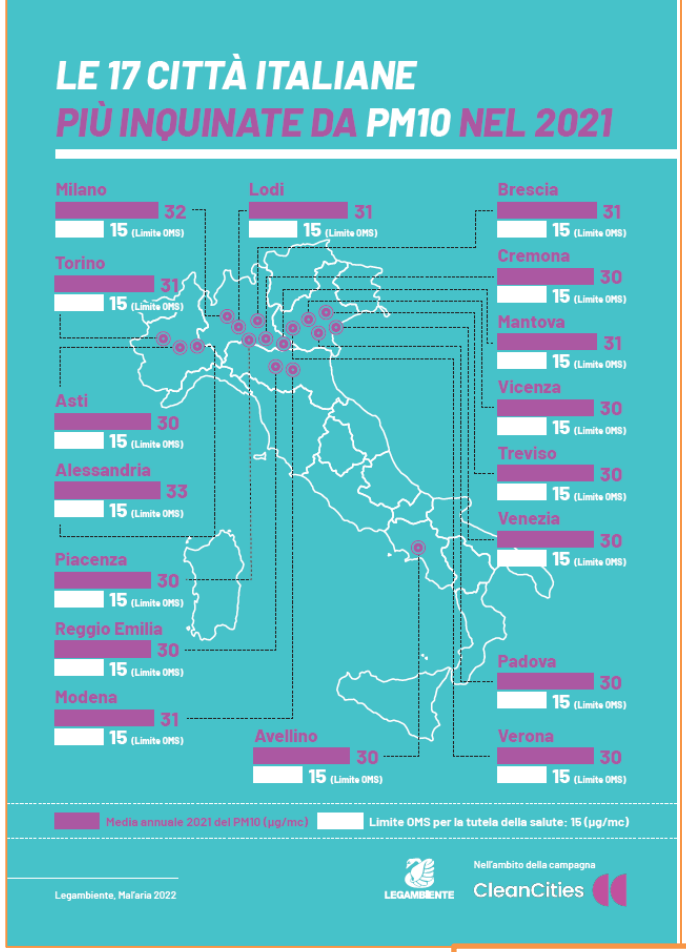
Background and objectives: There has been no clear consensus about whether prenatal exposure to air pollution contributes to the development of wheezing and asthma in children. We conducted a systematic review to analyze the association between exposure to different pollutants during pregnancy and the development of childhood wheezing and asthma.

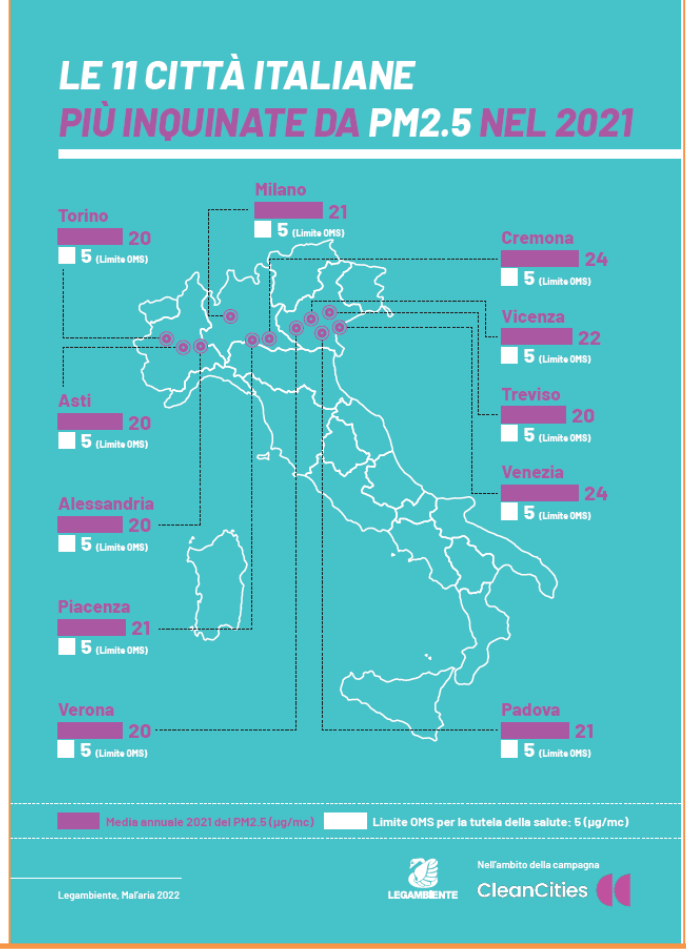
Methods: We systematically reviewed epidemiological studies published through June 6, 2017 available in the MEDLINE and Web of Science databases. We included studies that examined the association between prenatal exposure to any air pollutants except tobacco smoke and the incidence or prevalence of "wheezing" or "asthma" from birth to 14 years of age. We extracted key characteristics of each included study using a template of predefined data items. We used the Critical Appraisal Skills Programme checklists to assess the validity of each included study. We conducted overall and subgroup meta-analyses for each summary exposure-outcome association. Pooled odds ratios (OR) with 95% confidence intervals (CI) were estimated by using a random effects model.

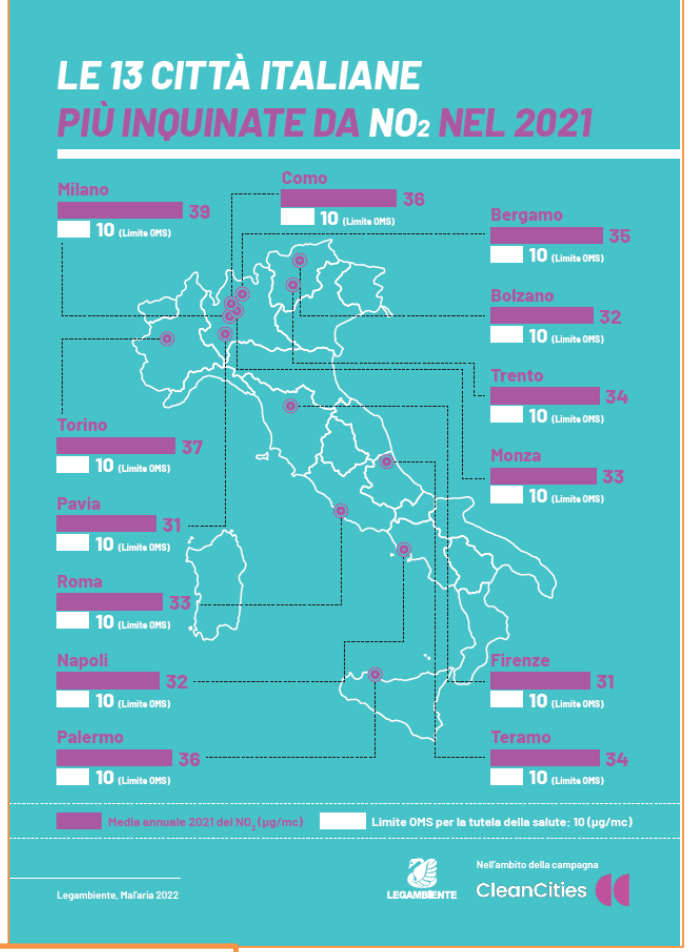
Results: Eighteen studies met our eligibility criteria. There was notable variability in exposure assessment methods. The overall random effects risk estimates (95% CI) of different pollutants were 1.04 (0.94–1.15) aromatic hydrocarbons (PAH), 1.04 (1.01–1.07) NO_2, 1.4 (0.97–2.03) $PM_{2.5}$ for childhood wheeze and 1.07 (1.01–1.14) NO_2, 1 (0.97–1.03) $PM_{2.5}$, 1.02 (0.98–1.07) SO_2, 1.08 (1.05–1.12) PM_{10} for childhood asthma. Minimal heterogeneity was seen for PAH and SO_2, while some heterogeneity was observed for PM_{10}, $PM_{2.5}$ and NO_2.

Conclusions: The overall and subgroup risk estimates from the meta-analyses showed statistically significant associations between prenatal exposures to NO_2, SO_2, and PM_{10} and the risk of wheezing and asthma development in childhood. There is insufficient evidence to show an effect of prenatal exposure to BC, CO, and O_3 on childhood wheezing and asthma. Further studies are needed to examine the individual compounds' effects.

ALCUNE EVIDENZE IN ITALIA







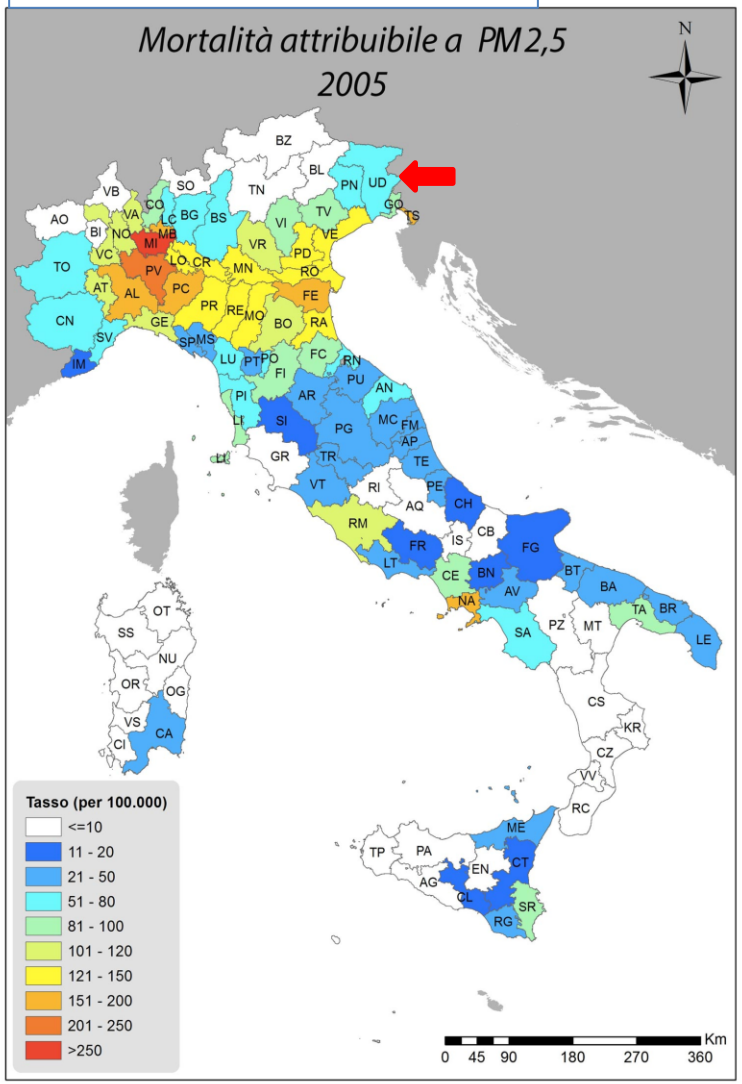
Concentrazione media annuale nel 2021 di Polveri sottili (PM10 e PM2.5) e di Biossido di azoto (NO_2) nelle città capoluogo di provincia

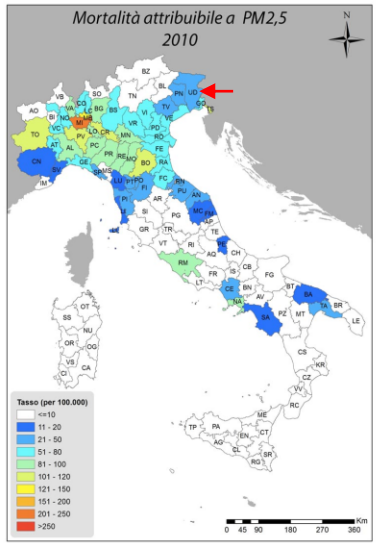
La media annuale è stata calcolata come media delle medie annuali delle singole centraline di monitoraggio ufficiale delle Arpa classificate come urbane (fondo o traffico).

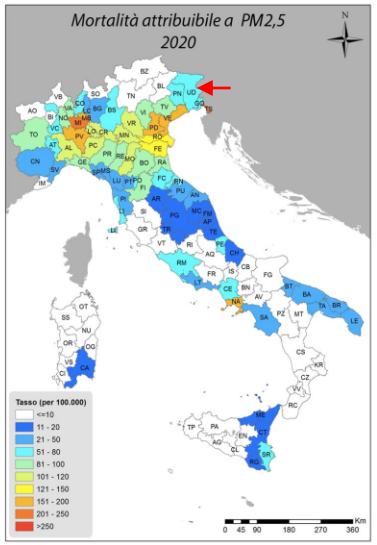
	Concentrazioni medie annuali Anno 2021			Riduzione delle concentrazioni necessarie		
Città	PM10	PM2.5	NO_2	PM10	PM2.5	NO_2
Udine	18	13	17	-17%	-62%	-41%

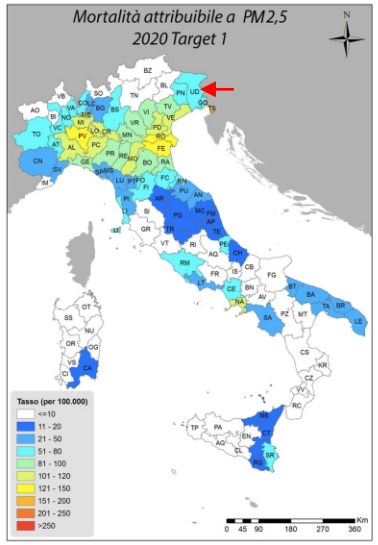
viias

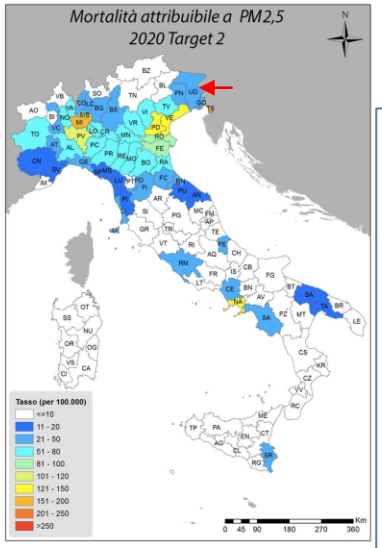
Metodi per la Valutazione Integrata dell'Impatto Ambientale e Sanitario dell'inquinamento atmosferico











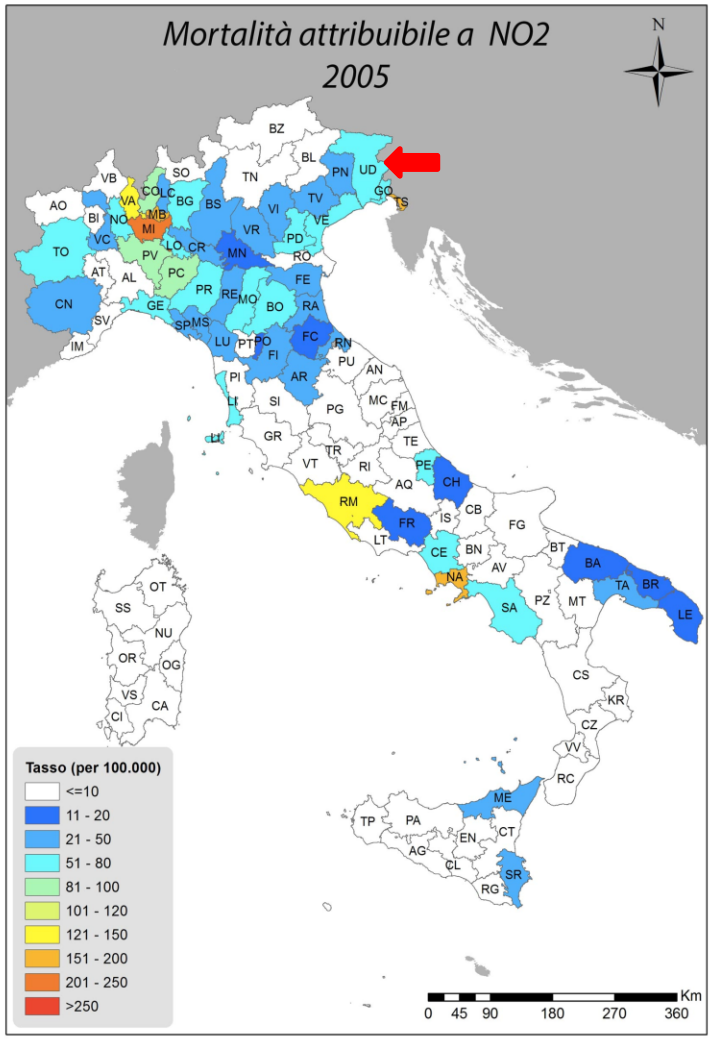
Decessi attribuibili a PM2,5

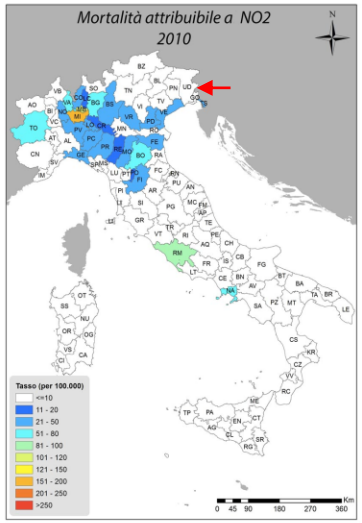
La tabella mostra i decessi per cause naturali attribuibili al Pm2,5 tra la popolazione over 30.

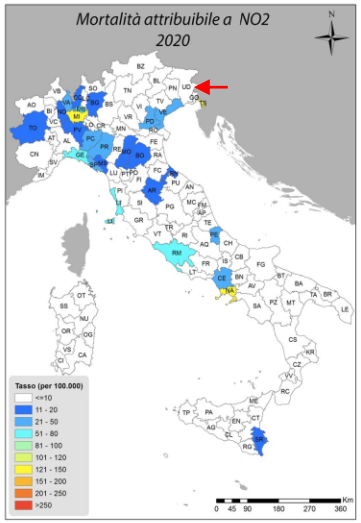
	2005	2020 CLE	2020 Target 1	2020 Target 2
ITALIA	34.552	28.595	23.170	18.511
Nord	22.485	19.536	15.301	13.172
Centro	5.513	4.056	4.004	2.247
Sud e Isole	6.554	5.004	3.865	3.093
Aree urbane	19.358	16.940	12.573	11.652
Aree non urbane	15.194	11.655	10.597	6.860

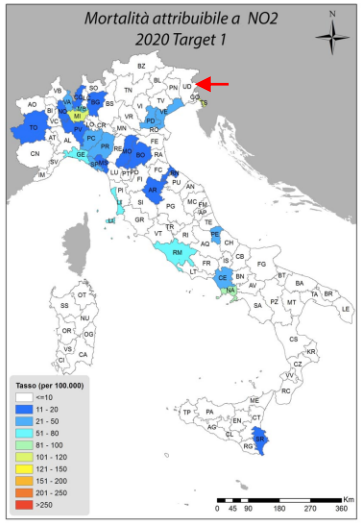
viias

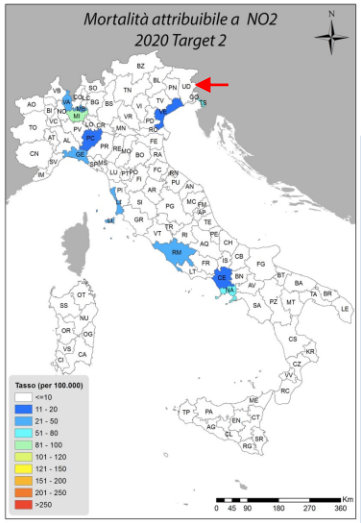
Metodi per la Valutazione Integrata dell'Impatto Ambientale e Sanitario dell'inquinamento atmosferico











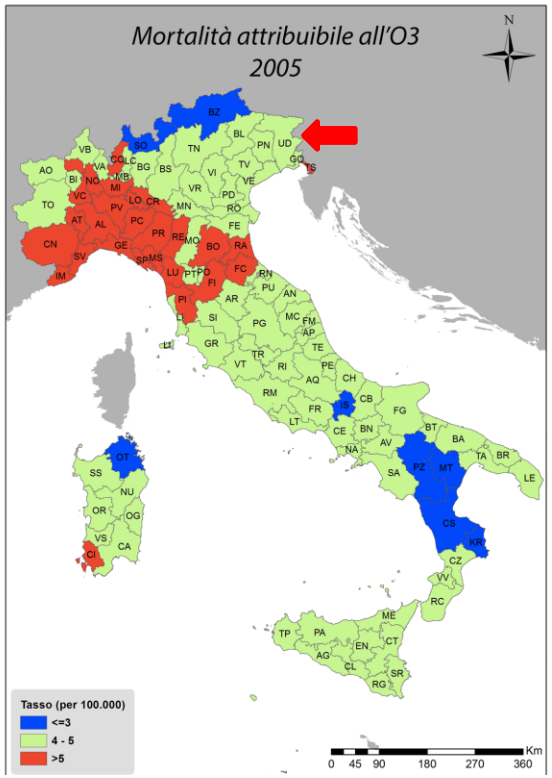
Decessi attribuibili a NO2

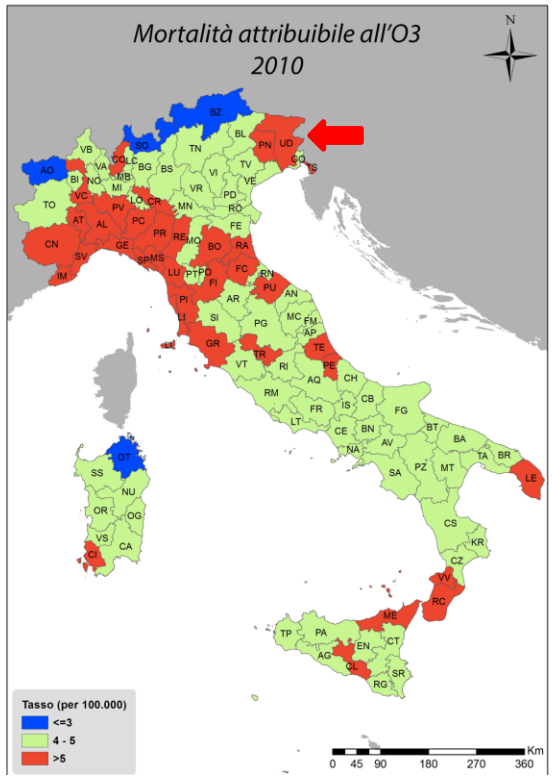
La tabella mostra i decessi per cause naturali attribuibili a biossido di azoto tra la popolazione over 30

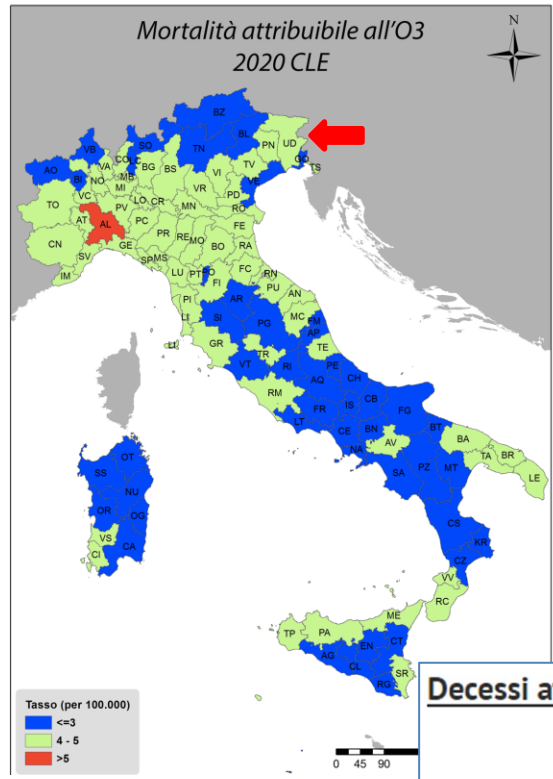
	2005	2020 CLE	2020 Target 1	2020 Target 2
ITALIA	23.387	10.117	9.021	5.247
Nord	14.008	5.615	5.094	2.792
Centro	4.977	1.961	1.959	945
Sud e Isole	4.403	2.541	1.968	1.509
Aree urbane	16.736	7.930	7.081	4.276
Aree non urbane	6.651	2.187	1.940	971

viias

Metodi per la Valutazione Integrata dell'Impatto Ambientale e Sanitario dell'inquinamento atmosferico







Decessi attribuibili a O3

	2005	2010	2020 CLE
ITALIA	1.707	1.858	1.320
Nord	882	898	686
Centro	368	422	295
Sud e Isole	457	538	339
Aree urbane	605	644	489
Aree suburbane ad alta densità	475	532	366
Aree non urbane a media densità	483	526	361
Aree non urbane	144	157	105

Impatto a breve termine dell'inquinamento dell'aria nelle città coperte dalla sorveglianza epidemiologica EpiAir2

Short-term impact of air pollution among Italian cities covered by the EpiAir2 project

Michela Baccini,[1,2] Annibale Biggeri;[1,2] Gruppo collaborativo EpiAir2*

Epidemiol Prev 2013; 37 (4-5): 252-262

Sorveglianza epidemiologica dell'inquinamento atmosferico

CITTÀ	PM_{10} (µg/m³)	DA PER ANNO (ICr80%)	
	MEDIA ANNUALE OSSERVATA	LIMITE 20 µg/m³	LIMITE 40 µg/m³
ANCONA	32,3	5,6 (2,3;9,0)	
BOLOGNA	39,4	36,4 (14,1;57,5)	
BRINDISI	23,1	1,1 (0,5;1,7)	
CAGLIARI	27,6	5,1 (2,2;8,0)	
FERRARA	38,5	16,2 (8,1;25,3)	
FIRENZE	37,2	33,0 (15,1;51,2)	
GENOVA	29,6	34,3 (13,6;54,5)	
MILANO	48,0	134,0 (60,7;204,8)	38,6 (17,4;59,1)
MODENA	42,3	16,0 (4,5;26,6)	1,7 (0,5;2,8)
NAPOLI	35,8	71,0 (35,1;109,4)	
PADOVA	48,4	26,9 (8,9;44,0)	8,0 (2,6;13,1)
PALERMO	35,7	46,9 (23,4;71,5)	
PARMA	36,0	12,1 (4,0;19,8)	
PIACENZA	39,0	10,6 (5,2;16,6)	
PISA	33,1	5,4 (2,2;8,7)	
REGGIO EMILIA	32,3	13,1 (6,2;20,2)	
RIMINI	35,7	9,5 (4,3;14,7)	
ROMA	36,1	202,9 (113,8;305,4)	
TARANTO	28,0	7,0 (3,4;11,0)	
TORINO	51,9	118,6 (66,4;171,7)	44,6 (24,9;64,6)
TREVISO	39,7	8,8 (4,3;13,5)	
TRIESTE	23,4	4,8 (2,3;7,5)	
VENEZIA-MESTRE	46,5	19,7 (5,8;32,5)	4,9 (1,4;8,1)

Tabella 5. Decessi attribuibili (DA) all'effetto a breve termine del PM_{10} sulla mortalità per cause naturali, con relativi intervalli di credibilità all'80% (ICr80%). Scenari controfattuali di 20 µg/m³ (linee guida OMS) e 40 µg/m³ (direttiva dell'Unione europea). Progetto EpiAir2, 2006-2009.

Table 5. Attributable deaths (DA) and 80% credibility intervals (ICr80%) of PM_{10} short-term effect on natural mortality. Counterfactuals scenarios of 20 µg/m³ (WHO air quality guidelines) and 40 µg/m³ (European Union Directive). EpiAir2 project, 2006-2009.

N° di decessi annui che si sarebbero evitati se non fossero stati superati i valori limite

839 decessi

EpiAir2

WWW.EPIPREV.IT

Rassegne e Articoli

e&p anno 37 (4-5) luglio-ottobre 2013

Inquinamento atmosferico e ricoveri ospedalieri urgenti in 25 città italiane: risultati del progetto EpiAir2

Air pollution and urgent hospital admissions in 25 Italian cities: results from the EpiAir2 project

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Cecilia Scarinzi,[1] Ester Rita Alessandrini,[2] Monica Chiusolo,[1] Claudia Galassi,[3] Marco Baldini,[4] Maria Serinelli,[5] Paolo Pandolfi,[6] Antonella Bruni,[7] Annibale Biggeri,[8] Aldo De Togni,[9] Giulia Carreras,[10] Claudia Casella,[11] Cristina Canova,[12] Giorgia Randi,[13] Andrea Ranzi,[14] Caterina Morassuto,[12] Achille Cernigliaro,[15] Simone Giannini,[14] Paolo Lauriola,[14] Fabrizio Minichilli,[16] Bianca Gherardi,[14] Stefano Zauli-Sajani,[14] Massimo Stafoggia,[2] Patrizia Casale,[17] Emilio A.L. Gianicolo,[18] Cinzia Piovesan,[19] Riccardo Tominz,[20] Loredana Porcaro,[21] Ennio Cadum;[1] Gruppo collaborativo EpiAir2*

EPIAIR 2

Inquinamento atmosferico e mortalità in venticinque città italiane: risultati del progetto EpiAir2

Air pollution and mortality in twenty-five Italian cities: results of the EpiAir2 Project

Ester Rita Alessandrini,[1] Annunziata Faustini,[1] Monica Chiusolo,[2] Massimo Stafoggia,[1] Martina Gandini,[2] Moreno Demaria,[2] Antonello Antonelli,[3] Pasquale Arena,[4] Annibale Biggeri,[5] Cristina Canova,[6] Giovanna Casale,[7] Achille Cernigliaro,[8] Elsa Garrone,[9] Bianca Gherardi,[10] Emilio A.L. Gianicolo,[11] Simone Giannini,[10] Claudia Iuzzolino,[12] Paolo Lauriola,[10] Mauro Mariottini,[13] Paolo Pasetti,[14] Giorgia Randi,[15] Andrea Ranzi,[10] Michele Santoro,[16] Vittorio Selle,[17] Maria Serinelli,[18] Elisa Stivanello,[19] Riccardo Tominz,[20] Mariangela Vigotti,[16,21] Stefano Zauli-Sajani,[10] Francesco Forastiere,[1] Ennio Cadum;[2] Gruppo collaborativo EpiAir2*

Epidemiol Prev 2013; 37 (4-5): 220-229

Sorveglianza epidemiologica dell'inquinamento atmosferico

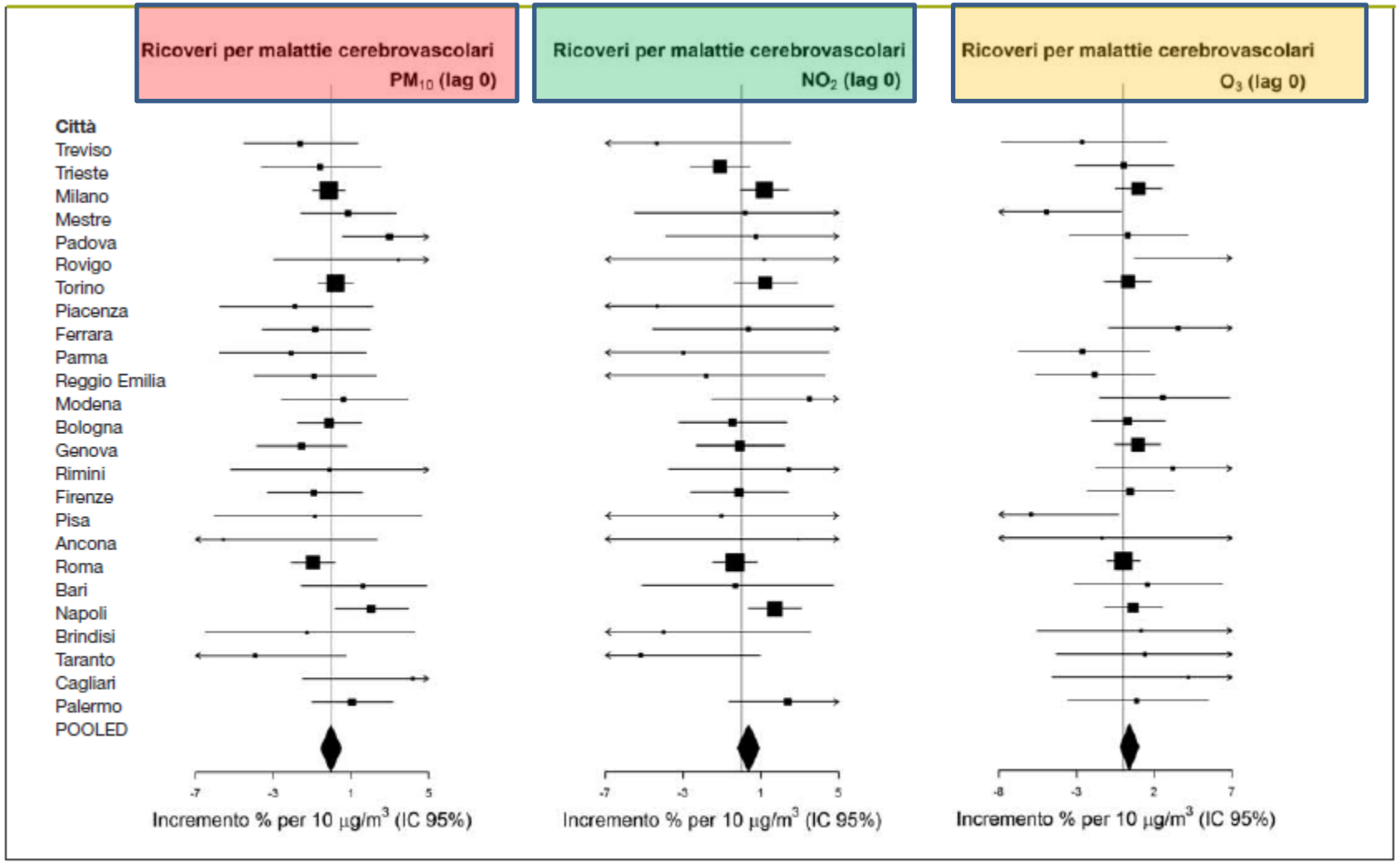


Figura 2. Risultati città-specifici e meta-analitici per le 25 città in studio, relativi all'associazione tra ricoveri per malattie cerebro-vascolari e inquinamento atmosferico, per inquinante: incrementi percentuali di rischio, ed intervalli di confidenza al 95%, corrispondenti a variazioni di 10 µg/m^3 dell'inquinante, 2006-2010 (periodo aprile-settembre per l'ozono).

EPIAIR 2

Scarinzi et al, e&p 2013

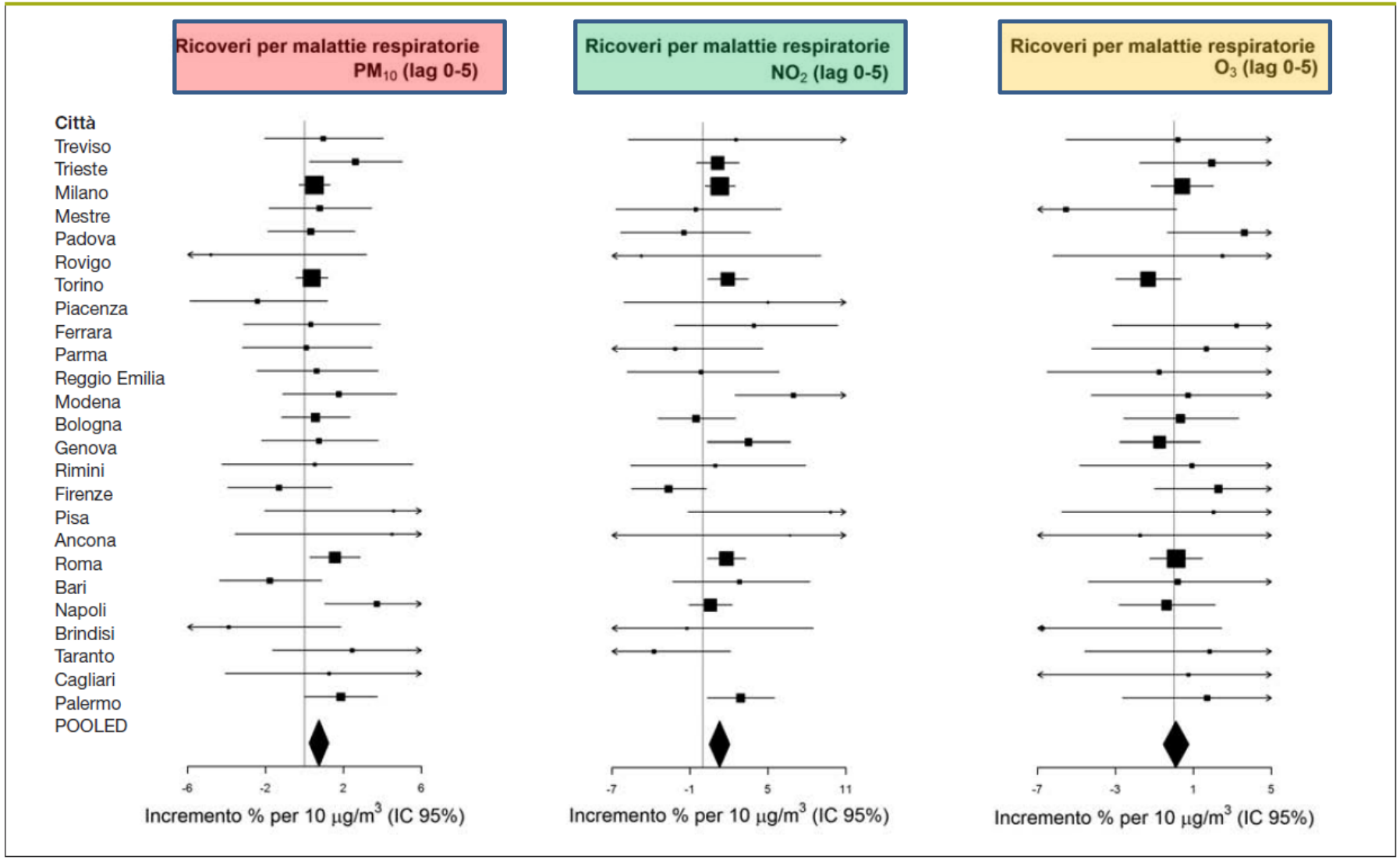
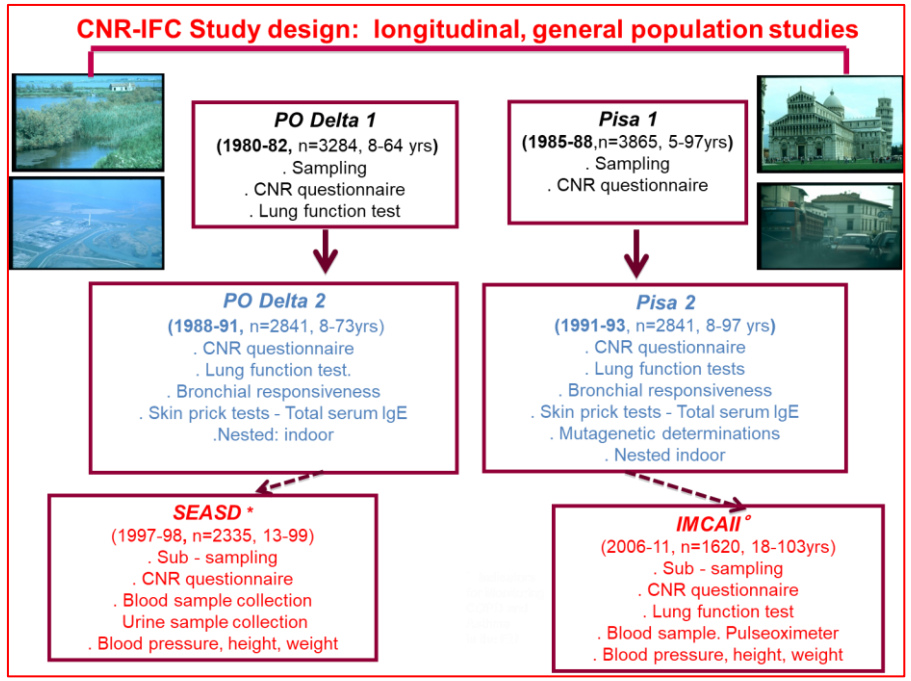


Figura 3. Risultati specifici per città e metanalitici per le 25 città in studio, relativi all'associazione tra ricoveri per malattie respiratorie e inquinamento atmosferico, per inquinante: incrementi percentuali di rischio e intervalli di confidenza al 95% corrispondenti a variazioni di 10 µg/m³ dell'inquinante, 2006-2010 (periodo aprile-settembre per l'ozono).

Figure 3. City-specific and meta-analytical results for the 25 cities under study, for respiratory causes and PM_{10}, NO_2 and O_3. Percent increase and 95%CI for 10 µg/m³ of each pollutant; 2006-2010 (period April-September for ozone).



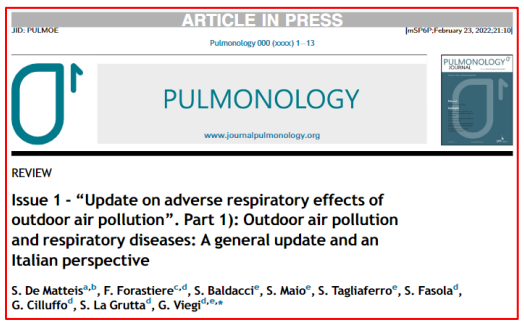
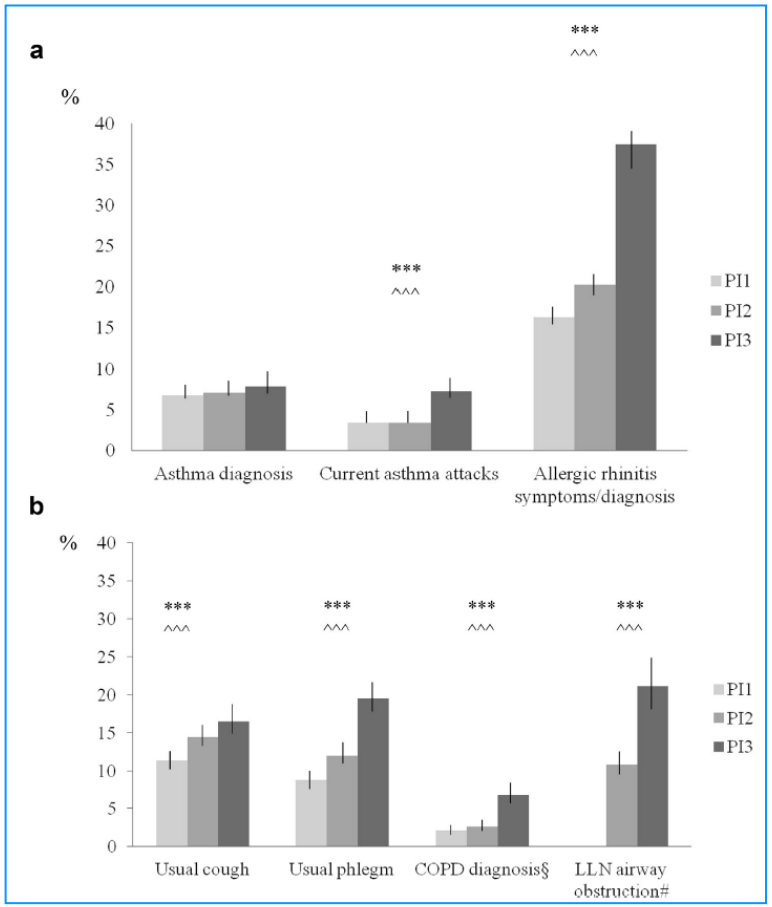


Table 2 Air pollution effects in Italy: I) analytical epidemiological surveys (Po Delta and Pisa studies carried out by CNR).

Study	Study area	Exposure	Health outcome	Health outcome results in the respective study area
Viegi et al, 1991 (ref. 28)	1) Rural area 2) Suburban-traffic area 3) Urban-traffic area 4) Urban-traffic-industry area	Traffic, industry	Chronic cough prevalence (%)	1) 9 2) 10 3) 11 4) 17
			Chronic phlegm prevalence (%)	1) 9 2) 9 3) 7 4) 14
			Attacks of wheezing with dyspnea prevalence (%)	1) 5 2) 8 3) 9 4) 9
			Dyspnea prevalence (%)	1) 14 2) 22 3) 26 4) 28
			Rhinitis prevalence (%)	1) 5 2) 17 3) 13 4) 25
			Chronic bronchitis or emphysema prevalence (%)	1) 2 2) 5 3) 7 4) 8
Viegi et al, 1999 (ref. 37)	Rural area vs urban-suburban area	Traffic, industry	Cough prevalence (%)	Males 25-64 yrs: 15 vs 21 Males > 64 yrs: 18 vs 37 Females 25-64 yrs: 11 vs 15
			Phlegm prevalence (%)	Males <25 yrs: 13 vs 6
			Wheeze prevalence (%)	Males > 64 yrs: 27 vs 39 Females 25-64 yrs: 14 vs 19
			Attacks of wheeze prevalence (%)	Males <25 yrs: 6 vs 12 Males 25-64 yrs: 5 vs 8 Females 25-64 yrs: 4 vs 6
			Dyspnea grade 1 prevalence (%)	Males <25 yrs: 6 vs 2 Males 25-64 yrs: 17 vs 10 Females <25 yrs: 11 vs 3 Females 25-64 yrs: 29 vs 19 Females >64 yrs: 48 vs 33
			Dyspnea grade 2 prevalence (%)	Females <25 yrs: 6 vs 3
			Chronic bronchitis prevalence (%)	Males 25-64 yrs: 3 vs 5
			Emphysema prevalence (%)	Males 25-64 yrs: 2 vs 8 Males > 64 yrs: 7 vs 22
Viegi et al, 2004 (ref. 38)	1) Rural area 2) Urban-suburban area	Traffic, industry	Obstructive lung diseases prevalence (%)	1) 6.9 2) 10.9
Maio et al, 2009 (ref. 39)	Urban-suburban area vs rural area	Traffic, industry	Bronchial hyper-responsiveness (OR, 95% CI)	1.41 (1.13-1.76)

	PI1 (1985-88)	PI2 (1991-93)	PI3 (2009-11)	p-value (PI1 vs PI3)	p-value trend
Asma	6,7	7,1	7,8	n.s.	n.s.
Attacchi d'asma correnti	3,4	3,4	7,2	<0,001	<0,001
Sintomi/diagnosi di rinite allergica	16,2	20,2	37,4	<0,001	<0,001
Tosse usuale	11,4	14,4	16,5	<0,001	<0,001
Espettorato usuale	8,7	12,0	19,5	<0,001	<0,001
BPCO	**2,1**	**2,6**	**6,8**	**<0,001**	**<0,001**
Ostruzione delle vie aeree*	---	10,8	21,1	<0,001	<0,001



La Prevalenza di malattie e sintomi respiratori sta ancora aumentando: il campione di popolazione pisana seguito per 25 anni

Respiratory symptoms/diseases prevalence is still increasing: a 25-yr population study

Respiratory Medicine 110 (2016)

Sara Maio [a, *], Sandra Baldacci [a], Laura Carrozzi [b], Francesco Pistelli [b], Anna Angino [a], Marzia Simoni [a], Giuseppe Sarno [a], Sonia Cerrai [a], Franca Martini [a], Martina Fresta [a], Patrizia Silvi [a], Francesco Di Pede [a], Massimo Guerriero [c], Giovanni Viegi [a, d]

Fig. 2. a. Adjusted prevalence of asthma and allergic rhinitis symptoms/ diagnosis in the three Surveys of the Pisa study. b. Adjusted prevalence of COPD symptoms/ diagnosis and airway obstruction in the three surveys of the Pisa study. # Lower Limit of Normal (LLN) according to ATS/ERS;
*** p-value < 0.001 among the three surveys, by chi square test.
^^^ p-value < 0.001 by chi-square test for trend.

Respiratory symptoms/diseases prevalence is still increasing: a 25-yr population study

Sara Maio [a, *], Sandra Baldacci [a], Laura Carrozzi [b], Francesco Pistelli [b], Anna Angino [a], Marzia Simoni [a], Giuseppe Sarno [a], Sonia Cerrai [a], Franca Martini [a], Martina Fresta [a], Patrizia Silvi [a], Francesco Di Pede [a], Massimo Guerriero [c], Giovanni Viegi [a, d]

Respiratory Medicine 110 (2016) 58–65

Table 3
Risk factors for asthma/allergic rhinitis symptoms/diagnoses: OR and 95% CI.

	Asthma diagnosis	Attacks of asthma	Allergic rhinitis
Survey:			
PI1	1.00	1.00	1.00
PI2	1.08 (0.94–1.25)	0.88 (0.71–1.10)	**1.26 (1.13–1.40)**
PI3	**1.34 (1.09–1.66)**	**1.90 (1.46–2.47)**	**2.98 (2.58–3.44)**
Age	1.000 (0.991–1.001)	**1.010 (1.003–1.020)**	**0.996 (0.992–0.999)**
Sex:			
Females	1.00	1.00	1.00
Males	1.00 (0.80–1.26)	0.91 (0.69–1.21)	0.90 (0.78–1.04)
Work exposure:			
No	1.00	1.00	1.00
Yes	**1.23 (1.03–1.46)**	**1.27 (1.01–1.60)**	**1.37 (1.22–1.55)**
Pack-years:			
0	1.00	1.00	1.00
≤7	1.05 (0.82–1.36)	1.30 (0.92–1.85)	1.08 (0.92–1.28)
8–24	0.97 (0.73–1.27)	1.23 (0.86–1.74)	0.89 (0.75–1.06)
≥24	1.23 (0.92–1.64)	**2.04 (1.47–2.84)**	0.88 (0.73–1.07)
Educational level:			
>13 yrs	1.00	1.00	1.00
9-13 yrs	0.79 (0.52–1.19)	0.83 (0.47–1.47)	0.88 (0.69–1.13)
<8 yrs	1.12 (0.75–1.67)	1.28 (0.75–2.18)	**0.75 (0.59–0.96)**
Area:			
Suburban	1.00	1.00	1.00
Urban	0.89 (0.73–1.10)	1.10 (0.87–1.40)	**1.19 (1.05–1.35)**

PI1 = Pisa 1 survey; PI2 = Pisa 2 survey; PI3 = Pisa 3 survey.
OR and 95% CI from the multivariate generalised estimating equations.
Statistically significant values are represented in bold.

Respiratory symptoms/diseases prevalence is still increasing: a 25-yr population study

Sara Maio [a, *], Sandra Baldacci [a], Laura Carrozzi [b], Francesco Pistelli [b], Anna Angino [a], Marzia Simoni [a], Giuseppe Sarno [a], Sonia Cerrai [a], Franca Martini [a], Martina Fresta [a], Patrizia Silvi [a], Francesco Di Pede [a], Massimo Guerriero [c], Giovanni Viegi [a, d]

Respiratory Medicine 110 (2016) 58–65

Table 4
Risk factors for COPD symptoms/diagnoses and airway obstruction°: OR and 95% CI.

	Usual cough	Usual phlegm	COPD*	LLN airway obstruction#
Survey:				
PI1	1.00	1.00	1.00	
PI2	1.11 (0.98–1.25)	***1.13 (0.99–1.29)***	**1.24 (1.02–1.52)**	1.00
PI3	1.10 (0.93–1.30)	**1.48 (1.25–1.75)**	**1.46 (1.14–1.85)**	**1.78 (1.40–2.27)**
Age	**1.015 (1.011–1.019)**	**1.019 (1.014–1.023)**	**1.050 (1.042–1.058)**	**1.022 (1.013–1.031)**
Sex:				
Females	1.00	1.00	1.00	1.00
Males	0.94 (0.80–1.11)	**1.36 (1.15–1.61)**	**1.55 (1.17–2.05)**	0.76 (0.57–1.01)
Work exposure:				
No	1.00	1.00	1.00	1.00
Yes	**1.25 (1.10–1.44)**	**1.40 (1.22–1.62)**	**1.81 (1.46–2.24)**	1.22 (0.95–1.57)
Pack-years:				
0	1.00	1.00	1.00	1.00
≤7	**1.85 (1.51–2.27)**	**1.80 (1.44–2.24)**	1.26 (0.83–1.91)	**1.81 (1.27–2.57)**
8–23	**2.66 (2.19–3.22)**	**2.67 (2.19–3.26)**	**2.25 (1.62–3.14)**	**2.16 (1.54–3.02)**
≥24	**4.44 (3.64–5.40)**	**4.64 (3.80–5.67)**	**4.45 (3.30–5.99)**	**2.69 (1.89–3.84)**
Educational level:				
>13 yrs	1.00	1.00	1.00	1.00
9-13 yrs	1.25 (0.87–1.79)	0.97 (0.70–1.37)	1.24 (0.66–2.31)	0.94 (0.57–1.55)
≤8 yrs	**1.57 (1.11–2.21)**	1.11 (0.80–1.53)	1.39 (0.77–2.51)	1.06 (0.65–1.73)
Area:				
Suburban	1.00	1.00	1.00	1.00
Urban	***1.14 (0.99–1.31)***	**1.30 (1.12–1.49)**	**1.54 (1.25–1.90)**	0.86 (0.67–1.11)

PI1 = Pisa 1 survey; PI2 = Pisa 2 survey; PI3 = Pisa 3 survey.
° airway obstruction values available in PI2 and PI3 surveys.
* diagnosis of COPD or emphysema or chronic bronchitis computed only in adult subjects.
# Lower Limit of Normal (LLN) according to American Thoracic Society (ATS)/European Respiratory Society (ERS) criterion [18]: forced expiratory volume in the first second (FEV_1)/forced vital capacity (FVC) < 5th percentile of the predicted value.
OR and 95% CI from the multivariate generalised estimating equations.
Statistically significant values are represented in bold. Borderline values are represented in italics.

Geographical information system and environmental epidemiology: a cross-sectional spatial analysis of the effects of traffic-related air pollution on population respiratory health

Daniela Nuvolone[1,2*], Roberto della Maggiore[2], Sara Maio[3], Roberto Fresco[2], Sandra Baldacci[3], Laura Carrozzi[3], Francesco Pistelli[3], Giovanni Viegi[3,4]

Environmental Health 2011

Pisa Study

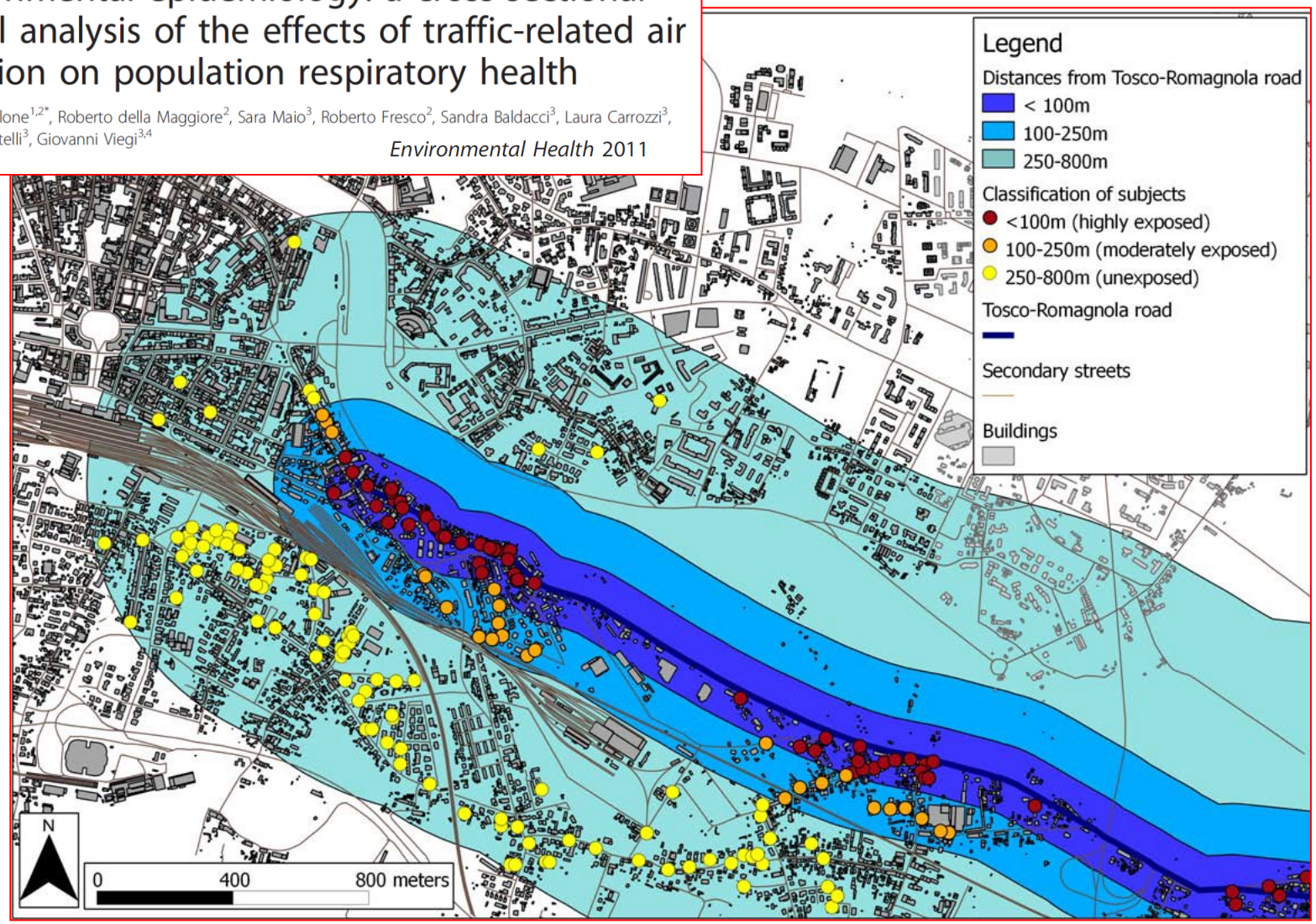


Figure 2 Classification of subjects based on the distance of each home from the main road. Zoomed map representing the classification of subjects according to the distance of each home from the main road. Highly exposed subjects are those living in the buffer area 0-100 m from the road, moderately exposed subjects living in the buffer area 100-250 m and unexposed are those living between 250 and 800 m from the road.

Geographical information system and environmental epidemiology: a cross-sectional spatial analysis of the effects of traffic-related air pollution on population respiratory health

Daniela Nuvolone[1,2*], Roberto della Maggiore[2], Sara Maio[3], Roberto Fresco[2], Sandra Baldacci[3], Laura Carrozzi[3], Francesco Pistelli[3], Giovanni Viegi[3,4]

Environmental Health 2011, **10**:12

Table 6 Effects of distance of residence to main road on respiratory symptoms/diseases and dichotomized test outcomes: OR† and 95% CI

	Males		Females	
	<100 m	100-250 m	<100 m	100-250 m
Persistent wheeze	1.76 * (1.08-2.87)	1.54 # (0.94-2.53)	1.32 (0.76-2.28)	0.77 (0.42-1.42)
Dyspnea	0.88 (0.55-1.41)	0.86 (0.59-1.53)	1.61 ** (1.13-2.27)	1.35 # (0.95-1.93)
COPD	1.80 * (1.03-3.08)	1.21 (0.69-2.13)	1.60 (0.71-3.59)	0.99 (0.39-2.51)
Asthma	1.59 (0.85-2.98)	1.55 (0.83-2.87)	1.68 # (0.97-2.88)	0.58 (0.30-1.15)
Attacks of shortness of breath with wheeze	1.47 (0.87-2.48)	1.20 (0.70-2.04)	1.67 # (0.98-2.84)	0.74 (0.39-1.38)
Skin test_5 mm pos.	1.07 (0.67-1.72)	1.10 (0.70-1.73)	1.83 * (1.11-3.00)	0.95 (0.57-1.60)
FEV_1/FVC% <70%	2.07 * (1.11-3.87)	2.53 ** (1.42-4.53)	1.01 (0.48-2.14)	0.88 (0.41-1.89)
FEV_1/VC% <70%	1.15 (0.63-2.11)	1.76 * (1.02-3.04)	0.84 (0.40-1.72)	0.48 (0.21-1.11)

† OR adjusted for age, educational level, smoking habits, passive smoking exposure, occupational exposure, working position, number of hours spent at home and time of residence, calculated with subjects living between 250-800 m as the reference group.

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, # $0.05 < p < 0.1$ (borderline).

Epidemiol Prev. Nov-Dec 2016;40(6):404-413. doi: 10.19191/EP16.6.P404.120.

[Cancer mortality of residents near a steel factory in Udine (Friuli Venezia Giulia Region, Northern Italy)]

[Article in Italian]

Luigi Castriotta [1], Anica Casetta [2], Fabio Barbone [3 2 4 5]

Objectives: to evaluate the association between distance of residence from a steel factory, a point source of air pollution within an industrial area in Udine (Friuli Venezia Giulia Region, Northern Italy), and cancer mortality.

Design: retrospective study. To evaluate the association between mortality rates and distance from the source, a Stone test analysis for all cases occurred in the entire area was conducted. The significance level was determined using Monte Carlo simulations.

Setting and participants: a GIS mapped residential history of Friuli Venezia Giulia population was completed. Among residents within 5 km from the principal industrial plant of the area from 1989 to 2012, deaths caused by cancer were selected. Furthermore, according to the prevalent wind direction, an analysis was conducted also in a subgroup of residents located into the South-Western quadrant. A set of 8 bands with increasing distance from the point-source was defined. The total population was 37,473 inhabitants.

Main outcome measures: in each band, observed and expected cancer-related deaths (calculated on the basis of mortality rates in the entire area) and standardized mortality ratios (SMRs) were computed to test for decline in risk of mortality at different distances from the main chimney. Stone test, in which a decline in risk of disease with an increase in distance from the source of pollution is tested, was used.

Results: the risk for all cancers and lung cancer was higher than expected. For male residents located within 2 km into the South-Western quarter there were 21 observed deaths from lung cancer vs. 13 expected (SMR: 1.62; p-value=0.02).

Conclusion: despite the fact that in the whole area cancer mortality is not increased, this study seems to support evidence of an excess of cancer deaths, especially lung cancer among males, near a steel factory. However, the study has limitations because of the small number of cases and the lack of individual exposure data and information about confounders (e.g., smoke habits and professional exposure). Therefore, a possible cause-effect interpretation of this association should be considered with caution.

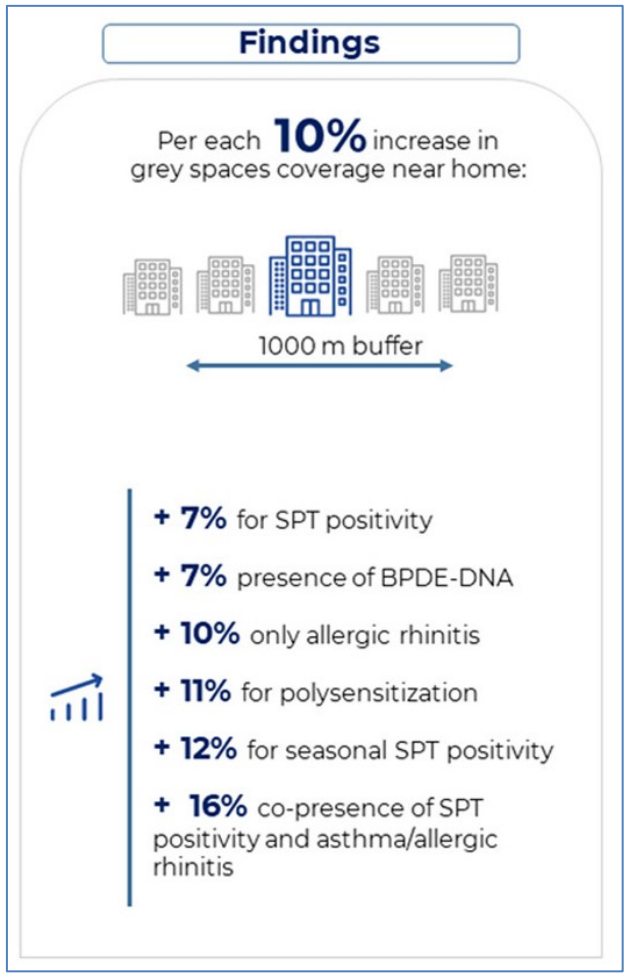
Environ Res. 2022 Apr 15;206:112428. doi: 10.1016/j.envres.2021.112428. Epub 2021 Nov 24.

Urban grey spaces are associated with increased allergy in the general population

S Maio [1], S Baldacci [2], S Tagliaferro [2], A Angino [2], E Parmes [3], J Pärkkä [3], G Pesce [4], C N Maesano [5], I Annesi-Maesano [5], G Viegi [6]

Table 4
Effects of 10% increase in residential exposure to urban greyness on allergic biomarkers/conditions and serum antibodies to BPDE-DNA adducts (n = 2070).

	OR (95% CI)
Allergic biomarkers/conditions	
SPT positivity	1.07 (1.02–1.13)
Reference category: negativity	1.00
Perennial SPT positivity	1.05 (0.98-1.12)
Reference category: negativity	1.00
Seasonal SPT positivity	1.12 (1.05–1.19)
Reference category: negativity	1.00
Type of sensitization:	
polysensitization	1.11 (1.04–1.19)
monosensitization	1.03 (0.96-1.11)
Reference category: negativity	1.00
Asthma/allergic rhinitis co-presence:	
asthma & allergic rhinitis	1.10 (0.98-1.23)
only allergic rhinitis	1.10 (1.04–1.17)
only asthma	*1.07 (0.99-1.15)*
Reference category: neither asthma nor allergic rhinitis	1.00
SPT and asthma/allergic rhinitis co-presence:	
SPT positivity & asthma/allergic rhinitis	1.16 (1.08–1.25)
only SPT positivity	1.02 (0.95-1.09)
only asthma/allergic rhinitis	1.06 (1.00–1.12)
Reference category: neither SPT nor asthma/allergic rhinitis	1.00
Log IgE value:	
≥1.81 kU/L	1.00 (0.95-1.05)
Reference category: < 1.81 kU/L	1.00
Exposure biomarker	
Positivity to serum antibodies to BPDE-DNA adducts	1.07 (1.01–1.14)
Reference category: negativity	1.00



18-yr cumulative incidence of respiratory/allergic symptoms/diseases and risk factors in the Pisa epidemiological study

Sara Maio [a,b,*], Sandra Baldacci [a], Laura Carrozzi [c], Francesco Pistelli [d], Marzia Simoni [a], Anna Angino [a], Stefania La Grutta [e], Vito Muggeo [b], Giovanni Viegi [a,e]

Table 4a
Longitudinal risk factors for asthma/allergic symptom/disease incidence: OR and 95% CI.

	Asthma diagnosis	Asthma attacks	Wheeze	Allergic rhinitis
Smoking habits:				
never	1.0	1.0	1.0	1.0
persistent	0.7 (0.2–3.0)	**2.7 (1.1–6.4)**	1.7 (0.6–4.7)	0.9 (0.5–1.6)
remittent for <18 years	1.1 (0.3–3.6)	1.5 (0.6–3.8)	0.2 (0.0–1.4)	1.1 (0.7–1.9)
remittent for ≥18 years	1.0 (0.4–2.7)	1.4 (0.7–3.1)	1.0 (0.4–2.6)	1.0 (0.7–1.6)
incident	–	0.9 (0.1–7.6)	0.8 (0.1–7.1)	0.7 (0.2–2.1)
Occupational exposure:				
never	1.0	1.0	1.0	1.0
persistent	**4.4 (1.4–13.6)**	1.1 (0.5–2.6)	0.5 (0.1–1.7)	**1.8 (1.1–3.0)**
remittent	–	0.8 (0.2–2.9)	0.3 (0.0–2.6)	0.7 (0.4–1.9)
incident	1.8 (0.7–4.8)	0.9 (0.5–1.9)	1.0 (0.4–2.4)	**1.6 (1.1–2.4)**
Vehicular traffic exposure				
never	1.0	1.0	1.0	1.0
persistent	1.3 (0.3–5.1)	0.6 (0.2–1.6)	1.0 (0.3–2.9)	*1.5 (0.9–2.5)*
remittent	2.4 (0.5–10.2)	0.6 (0.2–2.2)	0.8 (0.2–3.9)	0.8 (0.4–1.6)
incident	2.6 (0.8–8.2)	**2.2 (1.0–4.5)**	1.5 (0.6–3.7)	**1.8 (1.2–2.8)**

Table 4b
Longitudinal risk factors for bronchitic symptom/disease incidence: OR and 95% CI.

	COPD	Usual phlegm	Usual cough	Dyspnoea	AO_{LLN}
Smoking habits:					
never	1.0	1.0	1.0	1.0	1.0
persistent	**5.4 (2.3–12.5)**	**2.9 (1.7–5.1)**	**1.9 (1.0–3.5)**	**1.8 (1.1–3.0)**	**2.7 (1.0–7.4)**
remittent for <18 years	**3.3 (1.4–7.7)**	0.8 (0.5–1.6)	1.0 (0.5–1.9)	1.5 (0.9–2.4)	1.1 (0.4–3.4)
remittent for ≥18 years	**2.4 (1.2–5.1)**	1.1 (0.7–1.7)	1.0 (0.6–1.7)	1.3 (0.9–2.0)	1.2 (0.5–2.8)
incident	–	0.8 (0.2–3.0)	1.7 (0.6–5.1)	0.9 (0.3–2.8)	–
Occupational exposure:					
never	1.0	1.0	1.0	1.0	1.0
persistent	*1.9 (0.9–4.1)*	**1.8 (1.1–3.2)**	1.4 (0.8–2.6)	1.3 (0.8–2.0)	2.0 (0.8–5.2)
remittent	–	0.4 (0.1–1.3)	0.4 (0.1–1.4)	0.8 (0.4–1.7)	1.3 (0.3–5.4)
incident	1.6 (0.9–3.0)	**1.5 (1.0–2.4)**	**1.6 (1.0–2.5)**	**1.9 (1.3–2.8)**	1.1 (0.5–2.6)
Vehicular traffic exposure:					
never	1.0	1.0	1.0	1.0	1.0
persistent	1.7 (0.7–3.9)	1.0 (0.6–1.7)	0.7 (0.4–1.3)	1.0 (0.6–1.6)	*0.4 (0.2–1.1)*
remittent	*2.6 (0.9–7.0)*	1.1 (0.6–2.2)	1.1 (0.6–2.2)	1.0 (0.6–1.9)	0.4 (0.1–1.8)
incident	**2.4 (1.1–5.2)**	1.3 (0.8–2.0)	0.9 (0.6–1.5)	1.2 (0.8–1.8)	0.5 (0.2–1.2)

Int J Environ Res Public Health. 2020 Apr 8;17(7):2540. doi: 10.3390/ijerph17072540.

Effects of Particulate Matter on the Incidence of Respiratory Diseases in the Pisan Longitudinal Study

Salvatore Fasola [1], Sara Maio [2], Sandra Baldacci [2], Stefania La Grutta [1], Giuliana Ferrante [3], Francesco Forastiere [1], Massimo Stafoggia [4], Claudio Gariazzo [5], Giovanni Viegi [1 2], On Behalf Of The Beep Collaborative Group

Table 2. Associations (odds ratio, OR, and 95% confidence intervals (CI)) between risk factors ascertained during the first survey (1991–1993) and cumulative incidences of asthma, rhinitis, Chronic Obstructive Pulmonary Disease (COPD) and chronic phlegm ascertained at the second survey (2009–2011), from multivariable logistic regression models with Firth's correction.

	Asthma	Rhinitis	COPD	Chronic Phlegm
Cumulative incidence:	4/284 (1.4%)	90/264 (34.1%)	29/282 (10.3%)	16/262 (6.1%)
Independent variables:	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
PM_{10} (1 μg/m^3 increase) [1]	- [2]	- [2]	**2.96 (1.50–7.15)**	- [2]
$PM_{2.5}$ (1 μg/m^3 increase) [1]	- [2]	**2.25 (1.07–4.98)**	- [2]	**4.17 (1.12–18.71)**
Age, years (10-year increase)	- [2]	- [2]	**1.87 (1.29–3.02)**	- [2]
Male gender	- [2]	- [2]	- [2]	- [2]
Smoker (ref = non-smoker)	**12.96 (1.25–∞)**	- [2]	**2.99 (1.08–9.39)**	- [2]
Ex-smoker (ref = non-smoker)	4.86 (0.27–∞)	- [2]	1.67 (0.60–4.89)	- [2]
Occupational exposure	- [2]	- [2]	1.91 (0.83–4.79)	**5.41 (1.88–21.79)**

[1] Estimated exposure levels at the residential address for the year 2011, 1 km^2 resolution. [2] Variables excluded by the stepwise selection procedure. Significant odds ratios are reported in bold.

Allergy and asthma: Effects of the exposure to particulate matter and biological allergens

S. Baldacci [a,*,1], S. Maio [a,1], S. Cerrai [a], G. Sarno [a], N. Baïz [b,c], M. Simoni [a], I. Annesi-Maesano [b,c,2], G. Viegi [a,2] on behalf of the HEALS Study

Table 5 Link between allergic diseases and combined exposures.

Study	Country (n, sample)	Health outcome	Single exposure	Combined exposure
Carlsten C, 2011 [114]	Canada (380, children)		OR (95% CI): Dog allergens	OR (95% CI): Dog allergens and NO_2
		Asthma	1.0 (0.2—5.2) NO_2 1.3 (0.4—4.5)	4.8 (1.1—21.5)
Annesi-Maesano I, 2012 [110]	France (36,397, adults)		OR (95% CI): Grass pollen	OR (95% CI): Grass pollens and air pollutants
		Allergic rhinitis	1.08 (1.04—1.11)	1.08 (1.01—1.14)
Cirera L, 2012 [111]	Spain (3939, hospital ER visits)		RR% (95% CI): SO_2	RR% (95% CI): SO_2 and pollens
		Asthma ER visits	5.2 (0.5—10.1) NO_2	5.7 (0.9—10.6) NO_2 and pollens
		Asthma ER visits	2.6 (0.3—5.0)	2.7 (0.4—5.1)
Perzanowski MS, 2013 [112]	US (727, young adults)		RR (95% CI): Cockroach allergens	RR (95% CI): Cockroach allergens and nPAH
		Allergic sensitization	1.15 (1.07—1.25)	1.22 (1.08—1.36)

OR: odds ratio; RR: relative risk; 95% CI: 95% confidence intervals; ER: emergency room; SO_2: sulphur dioxide; NO_2: nitrogen dioxide; nPAH: nonvolatile polycyclic aromatic hydrocarbons.

Influence of residential land cover on childhood allergic and respiratory symptoms and diseases: Evidence from 9 European cohorts

Eija Parmes [a,1,*], Giancarlo Pesce [b,1], Clive E. Sabel [c], Sandra Baldacci [d], Roberto Bono [e], Sonia Brescianini [f], Cristina D'Ippolito [f], Wojciech Hanke [g], Milena Horvat [h], Hilkka Liedes [a], Sara Maio [d], Pierpaolo Marchetti [i], Alessandro Marcon [i], Emanuela Medda [f], Matthieu Molinier [a], Silvia Panunzi [i], Juha Pärkkä [a], Kinga Polańska [g], Julie Prud'homme [b], Paolo Ricci [j], Janja Snoj Tratnik [h], Giulia Squillacioti [e], Maria Antonietta Stazi [f], Cara Nichole Maesano [b,2], Isabella Annesi-Maesano [b,2]

Environmental Research 2019

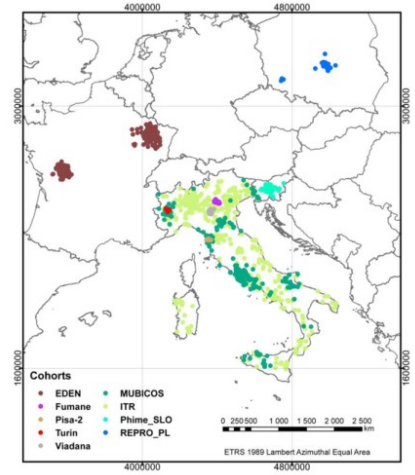


Fig. 1. Geographical distribution of the children included in the analyses differentiated by study.

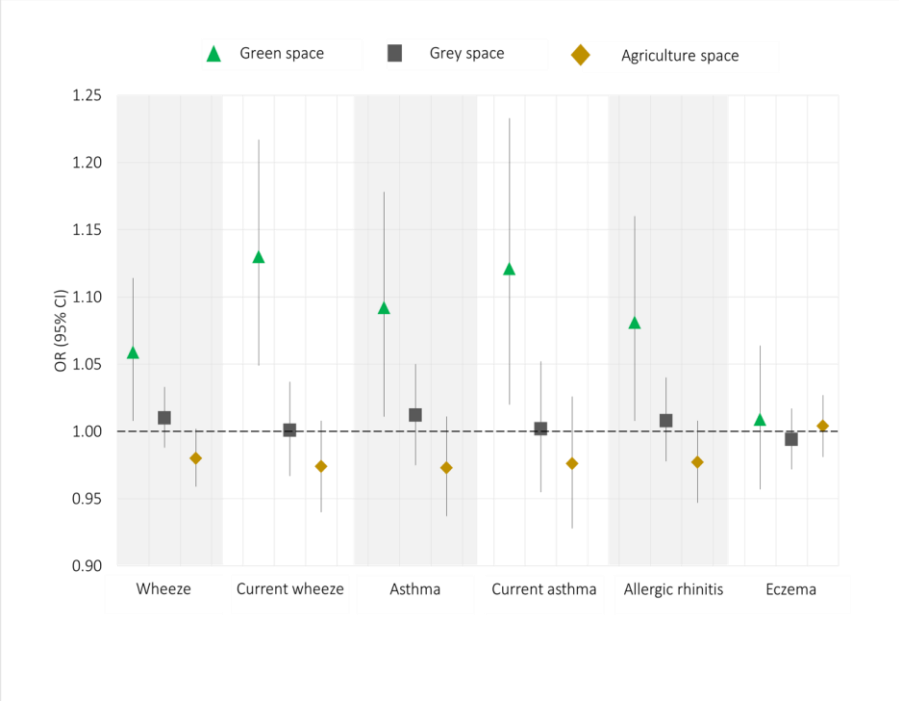


Fig.2: Associations between land coverage within 500m from children's home and allergic and respiratory outcomes. Odds ratios (OR with 95% confidence interval, CI) are estimated for a 10% increase of land covered by green, grey, or agricultural space.

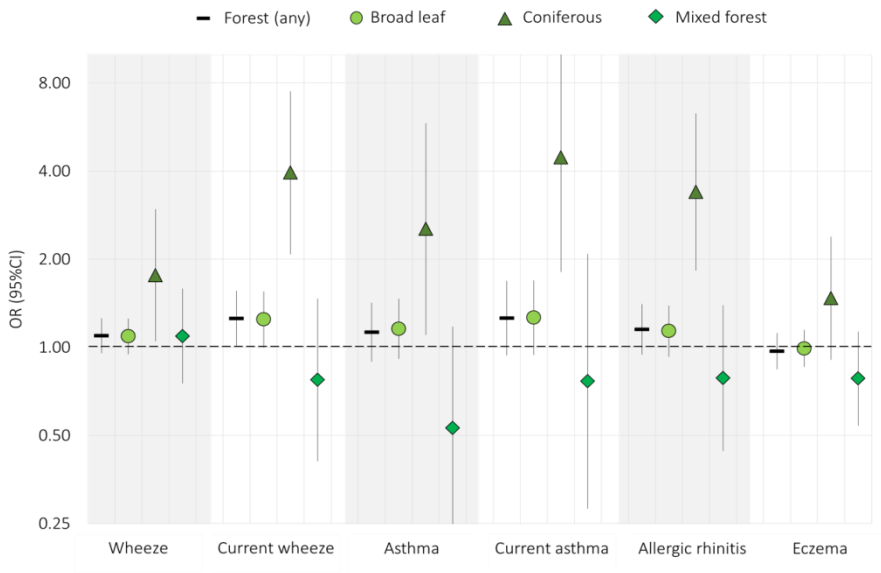


Fig.3: Associations between proximity to forests and respiratory and allergic symptoms. Odds ratios (OR with 95% confidence interval, CI) indicate the risk for children who live within 500 m from a forest vs. those who live further.

Data from 8063 children, aged 3–14 years, were obtained from nine European population-based studies participating in the HEALS project.

Benefici della riduzione dell'inquinamento atmosferico

GLOBAL ACTION PLAN

FOR THE PREVENTION AND CONTROL OF NONCOMMUNICABLE DISEASES

2013-2020

World Health Organization

World Health Organization

TO REDUCE MODIFIABLE RISK FACTORS FOR NONCOMMUNICABLE DISEASES AND UNDERLYING SOCIAL DETERMINANTS THROUGH CREATION OF HEALTH-PROMOTING ENVIRONMENTS

OBJECTIVE 3

TO STRENGTHEN AND ORIENT HEALTH SYSTEMS TO ADDRESS THE PREVENTION AND CONTROL OF NONCOMMUNICABLE DISEASES AND THE UNDERLYING SOCIAL DETERMINANTS THROUGH PEOPLE-CENTRED PRIMARY HEALTH CARE AND UNIVERSAL HEALTH COVERAGE

OBJECTIVE 4

Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study

THE LANCET • Vol 360 • October 19, 2002

Luke Clancy, Pat Goodman, Hamish Sinclair, Douglas W Dockery

1 Settembre 1990: il Governo Irlandese mette al bando il commercio e distribuzione di carbone nella città di Dublino: confronto 6 anni prima e 6 anni dopo.

Riduzione del 35.6% della concentrazione di black smoke e del 11.3% di SO_2.

	Unadjusted % change (95% CI)	p	Adjusted % change* (95% CI)	p
Total				
Non–trauma	–8·0 (–9·8 to –6·2)	<0·0001	–5·7 (–7·2 to –4·1)	<0·0001
Cause-specific				
Cardiovascular	–13·4 (–15·9 to –10·8)	<0·0001	–10·3 (–12·6 to –8·0)	<0·0001
Respiratory	–16·1 (–20·4 to –11·6)	<0·0001	–15·5 (–19·1 to–11·6)	<0·0001
Other	1·4 (–1·6 to 4·6)	0·36	1·7 (–0·7 to 4·2)	0·17
Age-specific				
Younger than age 60 years	–8·1 (–12·3 to –3·7)	<0·0001	–7·9 (–12·0 to –3·6)	<0·0001
Age 60–74 years	–8·6 (–12·3 to –9·6)	<0·0001	–6·2 (–8·8 to –3·5)	<0·0001
Age 75 years or older	–7·6 (–8·1 to –7·0)	<0·0001	–4·5 (–6·7 to –2·3)	<0·0001

*Adjusted in robust Poisson regression for temperature, relative humidity, day of week, respiratory epidemics, and standardised cause-specific death rates in rest of Ireland.

Table 3: **Change in age-standardised total, cause-specific, and age-specific mortality rates for Dublin County Borough for 72 months before and after ban of sale of coal in Dublin**

Studi di intervento durante le Olimpiadi

Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma

Friedman MS, et al. JAMA 2001

Comparazione fra i 17 giorni dei Giochi Olimpici (strategie di trasporto alternativo con riduzione del traffico) e le 4 settimane precedenti e successive.

Riduzione della concentrazione dell'inquinamento atmosferico e degli accessi all'ospedale e al pronto soccorso per attacchi d'asma nei bambini.

Δ -27.9% O_3; Δ -16.1% PM_{10}; Δ -6.8% NO_2

Δ -20-40% ospedalizzazioni

Impact of air pollution control measures and weather conditions on asthma during the 2008 Summer Olympic Games in Beijing.

Li Y, et al. Int J Biometeorol. 2011

Comparazione fra i 41 giorni dei Giochi Olimpici di Pechino (strategie di trasporto alternativo con riduzione del traffico) e le 4 settimane precedenti.

Riduzione della concentrazione dell'inquinamento atmosferico e delle visite mediche per asma negli adulti.

Δ -35% CO; Δ -31-44% PM_{10}; Δ -30% NO_2; Δ -34% O_3

Δ -40% visite mediche

Decline of Ambient Air Pollution Levels and Improved Respiratory Health in Swiss Children

Lucy Bayer-Oglesby,[1] Leticia Grize,[1] Markus Gassner,[2] Kathy Takken-Sahli,[3] Felix H. Sennhauser,[4] Urs Neu,[5] Christian Schindler,[1] and Charlotte Braun-Fahrländer[1]

Environ Health Perspect 113:1632–1637 (2005).

9600 bambini svizzeri (6-15 anni) investigati fra il 1993 e il 2000.

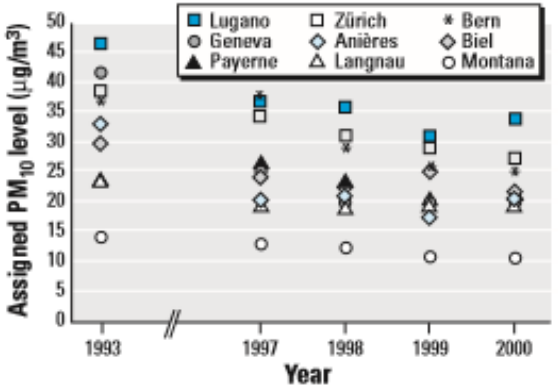


Figure 1. Annual means of PM_{10} levels[a] assigned to children of the first (1993) and second (1997–2000) health assessment phase in nine SCARPOL regions.
[a]Measured with DIGITEL HiVol Samplers. 1993 data converted from Harvard Impactor data.

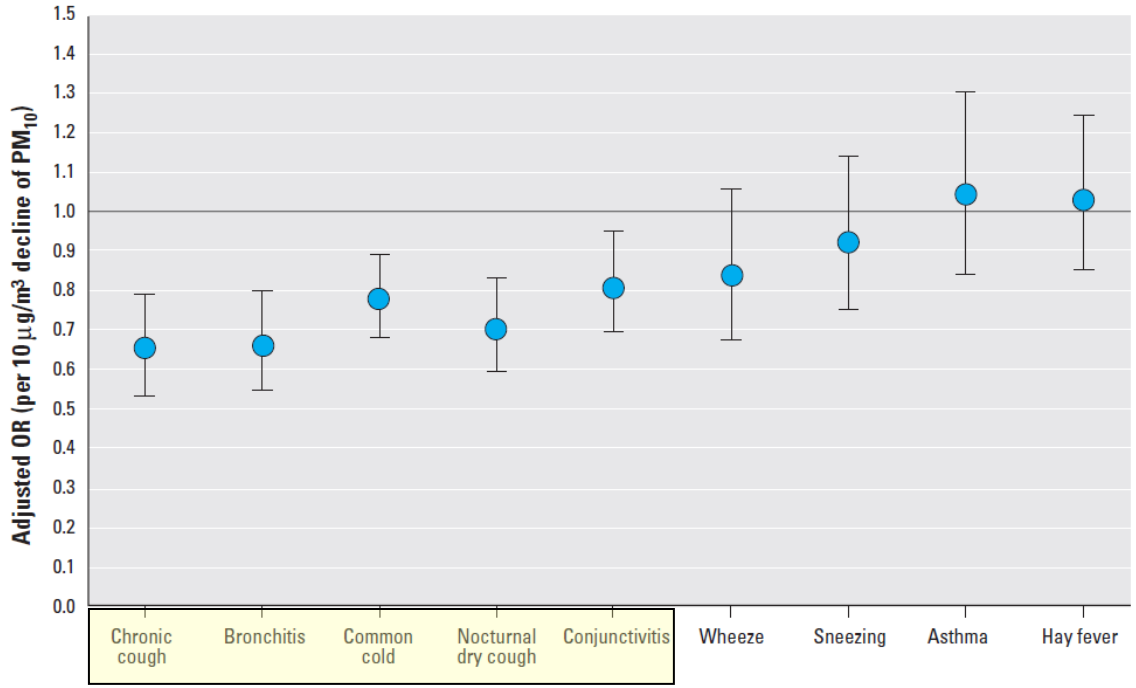


Figure 2. Adjusted ORs[a] and 95% CIs of symptoms and respiratory diseases in SCARPOL associated with a decline of 10 µg/m³ PM_{10} levels.
[a]Adjusted for age, sex, nationality, parental education, number of siblings, farming status, low birth weight, breast-feeding, child who smokes, family history of asthma, bronchitis, and/or atopy, mother who smokes, indoor humidity, mode of heating and cooking, carpeting, pets allowed in bedroom, removal of carpet and/or pets for health reasons, person who completed questionnaire, month when questionnaire was completed, number of days with the maximum temperature < 0°C, belief of mother that there is an association between environmental exposures and children's respiratory health, and region.

Associazioni fra la riduzione della concentrazione di PM_{10} ed una riduzione del rischio di sintomi respiratori, raffreddori e congiuntivite.

Association of Improved Air Quality with Lung Development in Children

W. James Gauderman, Ph.D., Robert Urman, M.S., Edward Avol, M.S., Kiros Berhane, Ph.D., Rob McConnell, M.D., Edward Rappaport, M.S., Roger Chang, Ph.D., Fred Lurmann, M.S., and Frank Gilliland, M.D., Ph.D.

New Engl J Med, 2015

2000 bambini americani investigati a 11 e 15 anni di età.

Politiche di controllo della qualità dell'aria hanno portato alla riduzione dell'inquinamento atmosferico in California.

Table 1. Estimated Differences in 4-Year Lung-Function Growth for Median Decreases in Ambient Pollutant Levels.*

Lung-Function Measurement and Pollutant	Lung-Function Difference at 11 Years of Age		Lung-Function Difference at 15 Years of Age		Growth from 11 to 15 Years of Age	
	Mean (95% CI)	P Value	Mean (95% CI)	P Value	Mean (95% CI)	P Value
	ml		*ml*		*ml*	
FEV_1						
Nitrogen dioxide	119.2 (76.5 to 161.9)	<0.001	210.6 (156.0 to 265.2)	- 14 ppb	91.4 (47.9 to 134.9)	<0.001
Ozone	15.0 (–38.5 to 68.6)	0.58	8.3 (–82.9 to 99.6)		–6.7 (–51.0 to 37.5)	0.77
PM_{10}	87.7 (50.2 to 125.2)	<0.001	153.2 (97.7 to 208.6)	- 8.7 µg/m³	65.5 (27.2 to 103.7)	<0.001
$PM_{2.5}$	100.0 (58.9 to 141.2)	<0.001	165.5 (95.4 to 235.6)	- 12.6 µg/m³	65.5 (17.1 to 113.8)	0.008

La dimensione dell'effetto della riduzione del NO_2 fu più elevata nei bambini asmatici.

Il miglioramento a lungo termine della qualità dell'aria risultò associato alla crescita della funzione polmonare.

World Health Organization

FIRST WHO GLOBAL CONFERENCE ON AIR POLLUTION AND HEALTH

IMPROVING AIR QUALITY, COMBATTING CLIMATE CHANGE – SAVING LIVES

30 October – 1 November 2018

WHO Headquarters, Geneva, Switzerland

save the date

World Health Organization

LET'S ACT TOGETHER ....

BECAUSE THE COST IS FAR TOO HIGH

Air pollution claims 7 million lives a year

Air pollution is a major driver of the non-communicable disease epidemic

Air pollution accelerates climate change

AND WE HAVE SOLUTIONS

Affordable and clean urban, transport, waste & household energy strategies

Health, environment & development sectors can lead the way to change

Organized in collaboration with

UN environment

WMO

CLIMATE & CLEAN AIR COALITION

UNECE

Effetti sanitari dell'inquinamento atmosferico

- **91% della popolazione mondiale, inclusi i bambini, è esposto ad inquinamento atmosferico a livelli inaccettabili.**
- **Sostanziali evidenze scientifiche mostrano che l'inquinamento atmosferico è il fattore che maggiormente contribuisce alle malattie e alla mortalità precoce.**
- **Sono stati mostrati effetti negativi, non solo a livelli elevati di inquinamento, ma anche al di sotto delle linee guida OMS 2005.**
- **L'inquinamento atmosferico provoca effetti sulle persone di tutte le età – dai neonati, ai bambini, fino ai più anziani.**
- **L'inquinamento atmosferico provoca effetti sulle persone con pre-esistenti malattie e con più basso livello socio-economico: le persone più sensibili e vulnerabili sono quelle più a rischio.**
- **E' necessario trovare soluzioni a lungo termine per migliorare la qualità dell'aria.**

Inquinamento atmosferico e cambiamenti climatici

- **L'utilizzo di combustibili fossili è il principale conduttore dei cambiamenti climatici.**
- **Gli stessi fattori che uccidono le persone a causa dell'inquinamento atmosferico stanno determinando cambiamenti climatici.**
- **La riduzione dell'inquinamento atmosferico ha impatti profondi sui cambiamenti climatici e proteggerà milioni di persone.**
- **Le soluzioni esistono: es. ridurre la produzione di energia con l'utilizzo di carbone e passare all'energia da fonti pulite.**
- **I benefici per la società sono enormi se comparati ai costi. Vi sono evidenze che mostrano che i costi della mitigazione potrebbero essere controbilanciati dai benefici per la salute.**

World Health Organization

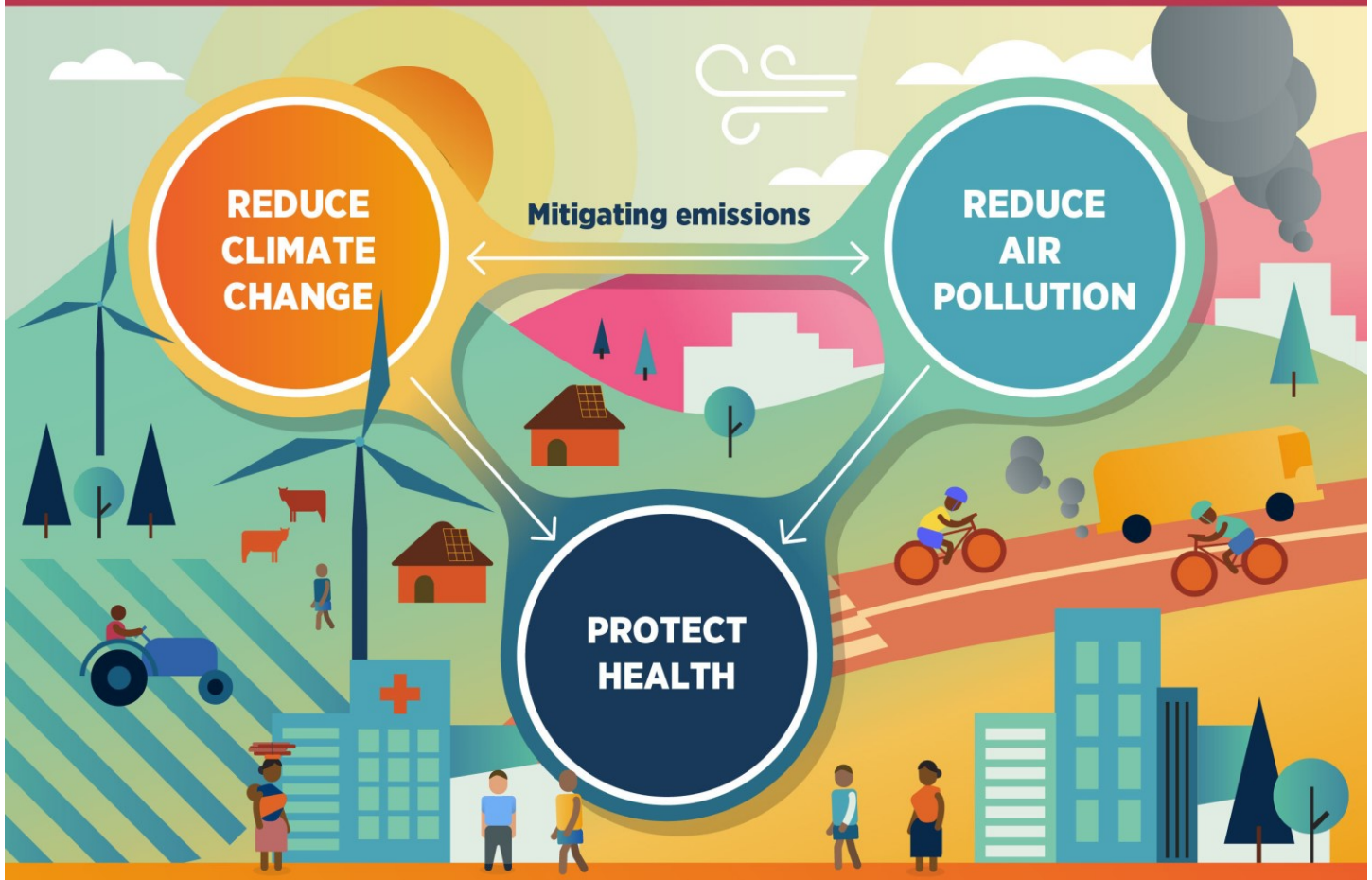
SOLUTIONS

WHO Air Quality Guidelines set goals to protect millions of lives from air pollution.

CLEAN AIR FOR HEALTH #AirPollution World Health Organization

World Health Organization

REDUCING AIR POLLUTION AND MITIGATING CLIMATE CHANGE, TOGETHER HELP TO PROTECT OUR HEALTH



WHO Air Quality Guidelines set goals to protect millions of lives from air pollution.

CLEAN AIR FOR HEALTH #AirPollution World Health Organization

FACTS ABOUT

Reducing Your Exposure to Particle Pollution

California Environmental Protection Agency

1) Ridurre l'esposizione al particolato nei veicoli

- Ridurre gli spostamenti nelle ore di punta e stare lontano dai tubi di scappamento
- Chiudere i finestrini e utilizzare l'impostazione del ricircolo dell'aria (chiudere le prese d'aria) in caso di traffico intenso, ma ventilare periodicamente il veicolo per evitare la sonnolenza dovuta all'accumulo di anidride carbonica esalata.
- Evitare lunghi riscaldamenti (soprattutto in uno spazio chiuso, ad es. garage) e inutili giri al minimo.
- Evitare di fumare negli autoveicoli, soprattutto quando i finestrini sono chiusi.
- Fare manutenzione del proprio veicolo.

FACTS ABOUT

Reducing Your Exposure to Particle Pollution

California Environmental Protection Agency

2) Ridurre l'esposizione indoor

- La più elevata esposizione al particolato indoor avviene mentre si cucina. Usare le cappe di aspirazione.
- Non permettere di fumare in casa.
- Utilizza fuochi elettrici o a gas invece del legno o pellet (fondamentale scegliere camini chiusi e ad alta efficienza)
- Far controllare annualmente stufe e fornelli a gas da un professionista
- Non utilizzare mai bracieri, griglie a carbone o stufe non ventilate all'interno.
- Limitare la combustione di candele e incensi all'interno e utilizzarli solo con una buona ventilazione
- Evitare l'uso di deodoranti per ambienti, prodotti per la pulizia e fragranze
- Garantire un'adeguata ventilazione durante le attività che generano umidità all'interno, come la doccia, la cucina e il lavaggio dei piatti.

FACTS ABOUT

Reducing Your Exposure to Particle Pollution

California Environmental Protection Agency

3) Per ridurre l'ingresso di particolato nella tua casa, intraprendere le seguenti azioni

- Usare gli zerbini e togliersi le scarpe sulla porta.
- Chiudere porte e finestre quando il livello di particelle esterne è elevato. Controllare i livelli di qualità dell'aria attuali e previsti per la tua città.
- Mantenere la casa pulita per evitare la risospensione di particelle da tappeti e pavimenti come terra, pollini, allergeni e peli di animali. Utilizzare un aspirapolvere ad alta efficienza o un aspirapolvere centralizzato e pulire spesso i pavimenti con un straccio umido

FACTS ABOUT

Reducing Your Exposure to Particle Pollution

California Environmental Protection Agency

4) Ridurre l'esposizione al particolato all'aperto

- Evitare l'attività all'aperto quando i livelli di inquinamento esterno sono elevati. Controllare i livelli di qualità dell'aria attuali e previsti per la tua città
- Quando si cammina, si fa jogging, si va in bicicletta e si svolgono altre attività all'aperto, evitare le aree vicine a fonti di inquinamento nocivo da particelle come strade trafficate o autostrade.
- Utilizzare attrezzature elettriche anziché a gas per prato e giardino.

Grazie per l'attenzione

Giuseppe Sarno
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giuseppe.sarno@ifc.cnr.it

Studi condotti dall’ Unità di Epidemiologia Ambientale Polmonare dell’ Istituto di Fisiologia Clinica del CNR di Pisa in collaborazione con il Dr. Mario Canciani riguardanti l’IAQ e la salute respiratoria degli scolari.

(Diapositive aggiuntive)

Eur Respir J. 2010 Apr;35(4):742-9. doi: 10.1183/09031936.00016309. Epub 2010 Jan 14.

School air quality related to dry cough, rhinitis and nasal patency in children

M Simoni [1], I Annesi-Maesano, T Sigsgaard, D Norback, G Wieslander, W Nystad, M Canciani, P Sestini, G Viegi

HESE study

The aim of this study was to assess the effects of IAQ on respiratory health of schoolchildren living in Norway, Sweden, Denmark, France and Italy.

The cross-sectional HESE study involved six operational units in five European countries (Siena and Udine, Italy; Reims, France; Oslo, Norway; Uppsala, Sweden; and Århus, Denmark).
21 schools (46 classrooms) with heterogeneous characteristics were selected.
The study was carried out in 2004–2005, during the heating season, and lasted a full week in each location.

The study protocol included: 1) one standardised questionnaire on school characteristics and IAQ policy completed by the teachers; 2) two standardised questionnaires derived from the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire on characteristics of children (i.e. health conditions, lifestyle, home environment), one filled in by the pupils and the other by their parents; 3) school environmental assessments; and 4) noninvasive clinical tests on a subsample of pupils.

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School air quality related to dry cough, rhinitis and nasal patency in children

M Simoni [1], I Annesi-Maesano, T Sigsgaard, D Norback, G Wieslander, W Nystad, M Canciani, P Sestini, G Viegi

HESE study

Abstract

Controls for indoor air quality (IAQ) in schools are not usually performed throughout Europe. The aim of this study was to assess the effects of IAQ on respiratory health of schoolchildren living in Norway, Sweden, Denmark, France and Italy. In the cross-sectional European Union-funded HESE (Health Effects of School Environment) Study, particulate matter with a 50% cut-off aerodynamic diameter of 10 microm (PM(10)) and CO(2) levels in a day of normal activity (full classroom) were related to wheezing, dry cough at night and rhinitis in 654 children (10 yrs) and to acoustic rhinometry in 193 children. Schoolchildren exposed to PM(10) >50 microg x m(-3) and CO(2) >1,000 ppm (standards for good IAQ) were 78% and 66%, respectively. All disorders were more prevalent in children from poorly ventilated classrooms. Schoolchildren exposed to CO(2) levels >1,000 ppm showed a significantly higher risk for dry cough (OR 2.99, 95% CI 1.65-5.44) and rhinitis (OR 2.07, 95% CI 1.14-3.73). By two-level (child, classroom) hierarchical analyses, CO(2) was significantly associated with dry cough (OR 1.06, 95% CI 1.00-1.13 per 100 ppm increment) and rhinitis (OR 1.06, 95% CI 1.00-1.11). Nasal patency was significantly lower in schoolchildren exposed to PM(10) >50 microg x m(-3) than in those exposed to lower levels. A poor IAQ is frequent in European classrooms; it is related to respiratory disturbances and affects nasal patency.

HESE study

Pediatr Allergy Immunol. 2011 Dec;22(8):843-52. doi: 10.1111/j.1399-3038.2011.01208.x.

Total viable molds and fungal DNA in classrooms and association with respiratory health and pulmonary function of European schoolchildren

Marzia Simoni [1], Gui-Hong Cai, Dan Norback, Isabella Annesi-Maesano, François Lavaud, Torben Sigsgaard, Gunilla Wieslander, Wenche Nystad, Mario Canciani, Giovanni Viegi, Piersante Sestini

Abstract

Indoor molds are associated with adverse respiratory effects in children. Although schools are important exposure sources of molds, objective measurements were more often taken in homes. Our aim was to assess indoor molds in schools and related effects on schoolchildren health. The Health Effects of the School Environment study (HESE) included 21 schools (46 classrooms) in Italy, Denmark, Sweden, Norway, and France and 654 schoolchildren (mean age 10 yr). Information on schoolchildren was collected by standardized questionnaires. Measurements of total viable molds (VM, colony-forming units, cfu/m(3)) and total/specific fungal DNA (cell equivalents, CE/g dust) were taken inside all classrooms in the cold season during normal activities, using the same standardized methodology. Pulmonary function tests were performed on 244 pupils. VM (mean, 320,cfu/m(3)) and total fungal DNA (geometric mean, 2.2 × 10(5) ± 2.1 CE/g dust) were detectable in all classrooms. The levels were significantly higher in buildings with mold/dampness problems. VM, but not fungal DNA, were inversely related to ventilation rate. VM exceeded the maximum standard of 300 cfu/m(3) in 33% of the classrooms. In the past 12 months, dry cough at night (34%) and rhinitis (32%) were the mostly reported. Children exposed to VM levels ≥ 300 cfu/m(3), compared with those exposed to lower levels, showed higher risk for past year dry cough at night (odds ratio, OR: 3.10, 95% confidence interval, CI: 1.61-5.98) and rhinitis (OR: 2.86, 95% CI: 1.65-4.95), as well as for persistent cough (OR: 3.79, 95% CI: 2.40-5.60). Aspergillus/Penicillium DNA was significantly positively associated with wheeze, and Aspergillus versicolor DNA with wheeze, rhinitis, and cough. There were significant inverse associations of Aspergillus versicolor DNA with forced vitality capacity (FVC) and Streptomyces DNA with both FEV(1) and FVC. In conclusion, indoor VM and fungal DNA were commonly found in monitored European schools and adversely related to respiratory health. Schools should be routinely tested through both culturable and non-culturable methods for global indoor molds' evaluation.

HESE study

European Respiratory Society Annual Congress 2012

Title: Relationships between school indoor toluene and respiratory symptoms in children of five European countries (HESE study)

Marzia Simoni, Isabella Annesi-Maesano, Torben Sigsgaard, Gunilla Wieslander, Wenche Nystad, Mario Canciani, Piersante Sestini, Giovanni Viegi

Abstract

Aim: to assess whether indoor toluene may affect respiratory health in schoolchildren. **Methods**: Health status and related risk factors were assessed through questionnaire in 628 children (mean age 10yrs) of five European countries: Sweden, Norway, Denmark, France, Italy (EU-funded HESE Study, Health Effects of School Environment). Measurements of pollutants were performed in 46 classrooms. Toluene was measured by active sampling using charcoal tubes. **Results**: The levels of toluene were relatively low: median concentration was 4.57, significantly higher in France (12.12) than in the other four countries (range: 2.82 in Sweden to 5.09 $\mu g/m^3$ in Italy). Prevalence rates of dry cough at night and wheeze were respectively 35% (range: 17 in Sweden to 48% in Italy) and 13% (range: 10% in Northern countries to 18% in France). Multiple logistic regression, accounting for centre, gender, age, presence of asthma, passive smoking at home, other indoor pollutants (PM_{10}, CO_2, viable moulds) indicated toluene to be associated with higher risk of dry cough (OR 4.37, 95%CI 2.19-8.75 per 1 $\mu g/m^3$ increment) and wheeze (OR 3.24, 1.25-8.45). These associations were significant after further accounting for the fixed effect of the classroom. **Conclusion:** Although toluene levels in classrooms were relatively low, long-term exposure seems to be a risk factor for respiratory health of schoolchildren.

Centro nazionale per la prevenzione e il controllo delle malattie
Network per la prevenzione e la sanità pubblica

Ministero della Salute

CCM Indoor-School study

PROGETTO CCM

Esposizione ad inquinanti indoor: linee guida per la valutazione dei fattori di rischio in ambiente scolastico e definizione delle misure per la tutela della salute respiratoria degli scolari e degli adolescenti (*Indoor-School*)

N° IDENTIFICATIVO DELLA LINEA PROGETTUALE DEL PROGRAMMA CCM: **13**
Area: Sostegno alle Regioni per l'implementazione del PNP e di Guadagnare Salute
Ambito: Prevenzione Universale
Linea progettuale: **Stili e ambienti di vita - Ambiente indoor**

Figura 1 Distribuzione delle UO partecipanti sul territorio nazionale

Periodo: 2010-2013

Popolazione: 53 scuole elementari/medie
153 aule
555 insegnanti
2370 genitori
2289 alunni

- Il Progetto
- Sintesi della relazione finale
- Relazione finale integrale
- Workshop e presentazioni

DISPONIBILI ON LINE AL SEGUENTE INDIRIZZO:
http://www.ccm-network.it/pagina.jsp?id=node/1943