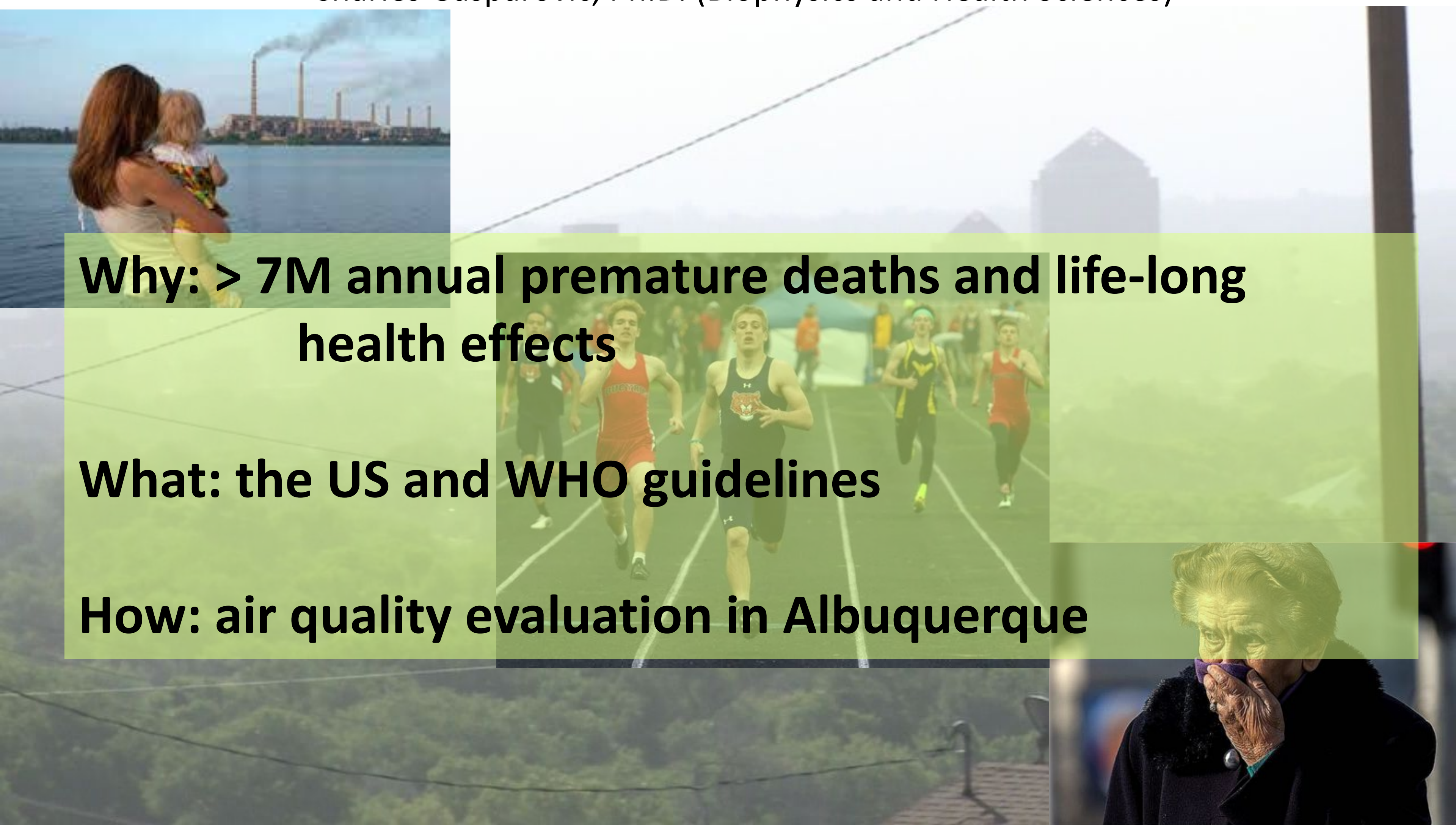
WHO Air Pollution Guidelines

What are they and why?

Charles Gasparovic, Ph.D. (Biophysics and Health Sciences)



Why: > 7M annual premature deaths and life-long health effects

What: the US and WHO guidelines

How: air quality evaluation in Albuquerque

Multicenter Study > Lancet Oncol. 2013 Aug;14(9):813-22.

BMJ. 2014; 348: f7412. PMCID: PMC3898420
Published online 2014 Jan 21. doi: 10.1136/bmj.f7412 PMID: 24452269

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

THE

Findings: The 312 944 cohort members contributed 4 013 131

Results 5157 participants experienced incident events. A 5 μg/m^3 increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (hazard ratio 1.13, 95% confidence interval 0.98 to 1.30), and a 10 μg/m^3 increase in estimated annual mean PM_{10} was associated with a 12% increased risk of coronary events (1.12, 1.01 to 1.25) with no evidence of heterogeneity between cohorts. Positive associations were detected below the current annual European limit value of 25 μg/m^3 for $PM_{2.5}$ (1.18, 1.01 to 1.39, for 5 μg/m^3 increase in $PM_{2.5}$) and below 40 μg/m^3 for PM_{10} (1.12, 1.00 to 1.27, for 10 μg/m^{3}increase in PM_{10}). Positive but non-significant associations were found with other pollutants.

between lung cancer and nitrogen oxides concentration (HR 1·01 [0·95-1·07] per 20 μg/m(3)) or traffic intensity on the nearest street (HR 1·00 [0·97-1·04] per 5000 vehicles per day).

Premature mortality due to air pollution in European cities: a health impact assessment

Sasha Khomenko, Marta Cirach, Evelise Pereira-Barboza, Natalie Mueller, Jose Barrera-Gómez, David Rojas-Rueda, Kees de Hoogh, Gerard Hoek, Mark Nieuwenhuijsen

Summary

Background Ambient air pollution is a major environmental cause of morbidity and mortality worldwide. Cities are generally hotspots for air pollution and disease. However, the exact extent of the health effects of air pollution at the city level is still largely unknown. We aimed to estimate the proportion of annual preventable deaths due to air pollution in almost 1000 cities in Europe.

Methods We did a quantitative health impact assessment for the year 2015 to estimate the effect of air pollution exposure ($PM_{2·5}$ and NO_2) on natural-cause mortality for adult residents (aged ≥20 years) in 969 cities and 47 greater cities in Europe. We retrieved the cities and greater cities from the Urban Audit 2018 dataset and did the analysis at a 250 m grid cell level for 2015 data based on the global human settlement layer residential population. We estimated the annual premature mortality burden preventable if the WHO recommended values (ie, 10 μg/m³ for $PM_{2·5}$ and 40 μg/m³ for NO_2) were achieved and if air pollution concentrations were reduced to the lowest values measured in 2015 in European cities (ie, 3·7 μg/m³ for $PM_{2·5}$ and 3·5 μg/m³ for NO_2). We clustered and ranked the cities on the basis of population and age-standardised mortality burden associated with air pollution exposure. In addition, we did several uncertainty and sensitivity analyses to test the robustness of our estimates.

Findings Compliance with WHO air pollution guidelines could prevent 51213 (95% CI 34036–68682) deaths per year for $PM_{2·5}$ exposure and 900 (0–2476) deaths per year for NO_2 exposure. The reduction of air pollution to the lowest measured concentrations could prevent 124729 (83332–166535) deaths per year for $PM_{2·5}$ exposure and 79435 (0–215165) deaths per year for NO_2 exposure. A great variability in the preventable mortality burden was observed by city, ranging from 0 to 202 deaths per 100000 population for $PM_{2·5}$ and from 0 to 73 deaths for NO_2 per 100000 population when the lowest measured concentrations were considered. The highest $PM_{2·5}$ mortality burden was estimated for cities in the Po Valley (northern Italy), Poland, and Czech Republic. The highest NO_2 mortality burden was estimated for large cities and capital cities in western and southern Europe. Sensitivity analyses showed that the results were particularly sensitive to the choice of the exposure response function, but less so to the choice of baseline mortality values and exposure assessment method.

Interpretation A considerable proportion of premature deaths in European cities could be avoided annually by lowering air pollution concentrations, particularly below WHO guidelines. The mortality burden varied considerably between European cities, indicating where policy actions are more urgently needed to reduce air pollution and achieve sustainable, liveable, and healthy communities. Current guidelines should be revised and air pollution concentrations

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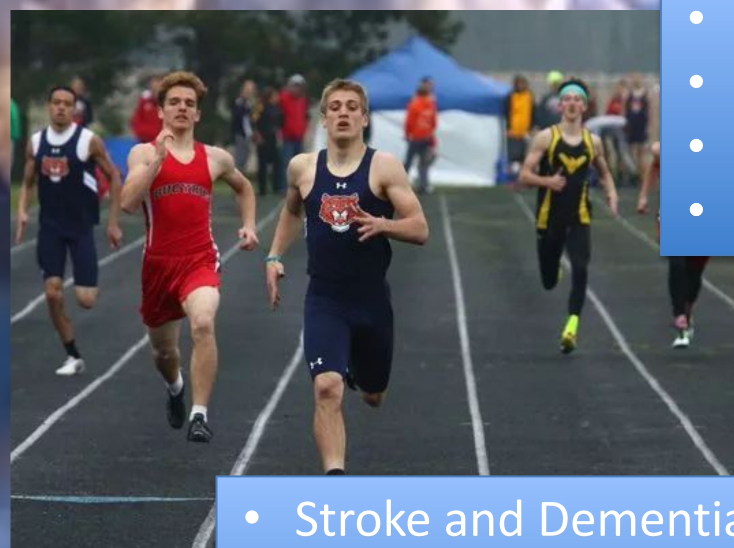
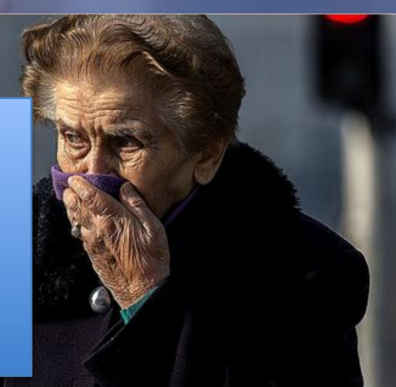
A killer stalking us throughout life

- Birth Defects
- Premature Births
- Pulmonary Development



- Asthma
- Brain Development
- Learning and Memory
- Childhood cancers



- Stroke and Dementias
- Pulmonary Diseases
- Heart diseases
- Cancer

WHO releases new repository of resources for air quality management

Report calls for further cross-governmental action and capacity building on air pollution in cities

7 September 2022 | Departmental news | Geneva | Reading time: 2 min (665 words)

- **There is growing scientific evidence of the harm air pollution exposure has on human health.**
- **Countries and cities should set targets to meet WHO air quality guidelines and include health in cost-benefit analysis of air quality management.**
- **The new WHO repository aims to be a one-stop-shop for tools and guidance documents related to air quality policies, monitoring methods, funding opportunities and educational programs from UN agencies and international institutions.**

https://www.who.int/news/item/07-09-2022-who-releases-new-repository-of-resources-for-air-quality-management

National Ambient Air Quality Standards (NAAQS)
Clean Air Act (1963, 1970 and amendments 1971-2016)

Pollutant	Type	Standard	Averaging Time	Form
Sulfur dioxide (SO_2)	Primary	.075 ppm (195 µg/m³)	1-hour	99th aver
Particulate matter (PM_{10})	Primary	150 µg/m³	24-hour	Not t over
Particulate matter ($PM_{2.5}$)	Primary	12 µg/m³	annual	Annu
	Primary	35 µg/m³	24-hour	98th
Carbon monoxide (CO)	Primary	35 ppm (40 mg/m³)	1-hour	Not t
	Primary	9 ppm (10 mg/m³)	8-hour	Not t
Ozone (O_3)	Primary	0.12 ppm (235 µg/m³)	1-hour	expe hour to or
	Primary	0.070 ppm (140 µg/m³)	8-hour	Annu avera
Nitrogen dioxide (NO_2)	Primary	0.053 ppm (100 µg/m³)	annual	Annu
Lead (Pb)	Primary	0.15 µg/m³	Rolling 3 mo	Not t

WHO

SO_2	500 µg/m³	- 10 min
	40 µg/m³	- 24h
PM_{10}	45 µg/m³	- 24h
	15 µg/m³	- annual
$PM_{2.5}$	15 µg/m³	- 24h
	5 µg/m³	- annual
CO	100 mg/m³	- 15 min
	35 mg/m³	- 1 h
	10 mg/m³	- 8 h
	4 mg/m³	- 24 h
O_3	100 µg/m³	- 8h
NO_2	200 µg/m³	- 1h
	25 µg/m³	- 24h
	10 µg/m³	- annual

	Del Norte HS	Jefferson	S. Valley	Foothills	N. Valley
year average	5	11	8	6	11
yr ave <30	5	10	8	5	11
daysPM2.5>5	156	342	264	129	336
daysPM2.5>12	12	93	53	23	131
daysPM2.5>20	2	27	14	3	34
daysPM2.5>35	1	3	2	1	2

	Del Norte HS	Jefferson	S. Valley	Foothills	N. Valley
year average	23	47	52	19	45
yr ave <100	23	42	46	19	40
daysPM10>15	260	341	334	181	343
daysPM10>20	174	310	314	104	313
daysPM10>50	16	126	143	11	109
daysPM10>100	1	18	25	1	16
daysPM10>150	0	2	4	0	1

$$I = \frac{I_{high} - I_{low}}{C_{high} - C_{low}}(C - C_{low}) + I_{low}$$

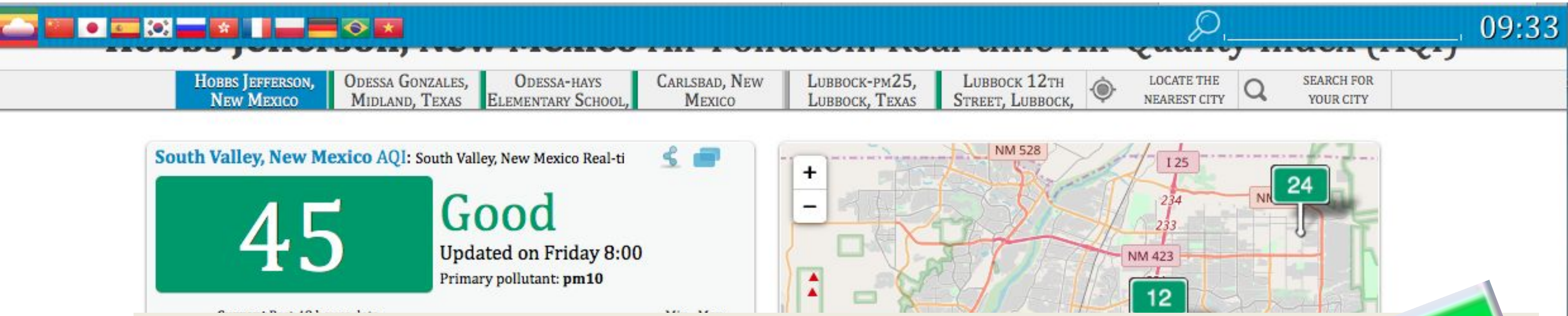
where:

- I = the (Air Quality) index,
- C = the pollutant concentration,
- C_{low} = the concentration breakpoint that is ≤ C,
- C_{high} = the concentration breakpoint that is ≥ C,
- I_{low} = the index breakpoint corresponding to C_{low},
- I_{high} = the index breakpoint corresponding to C_{high}

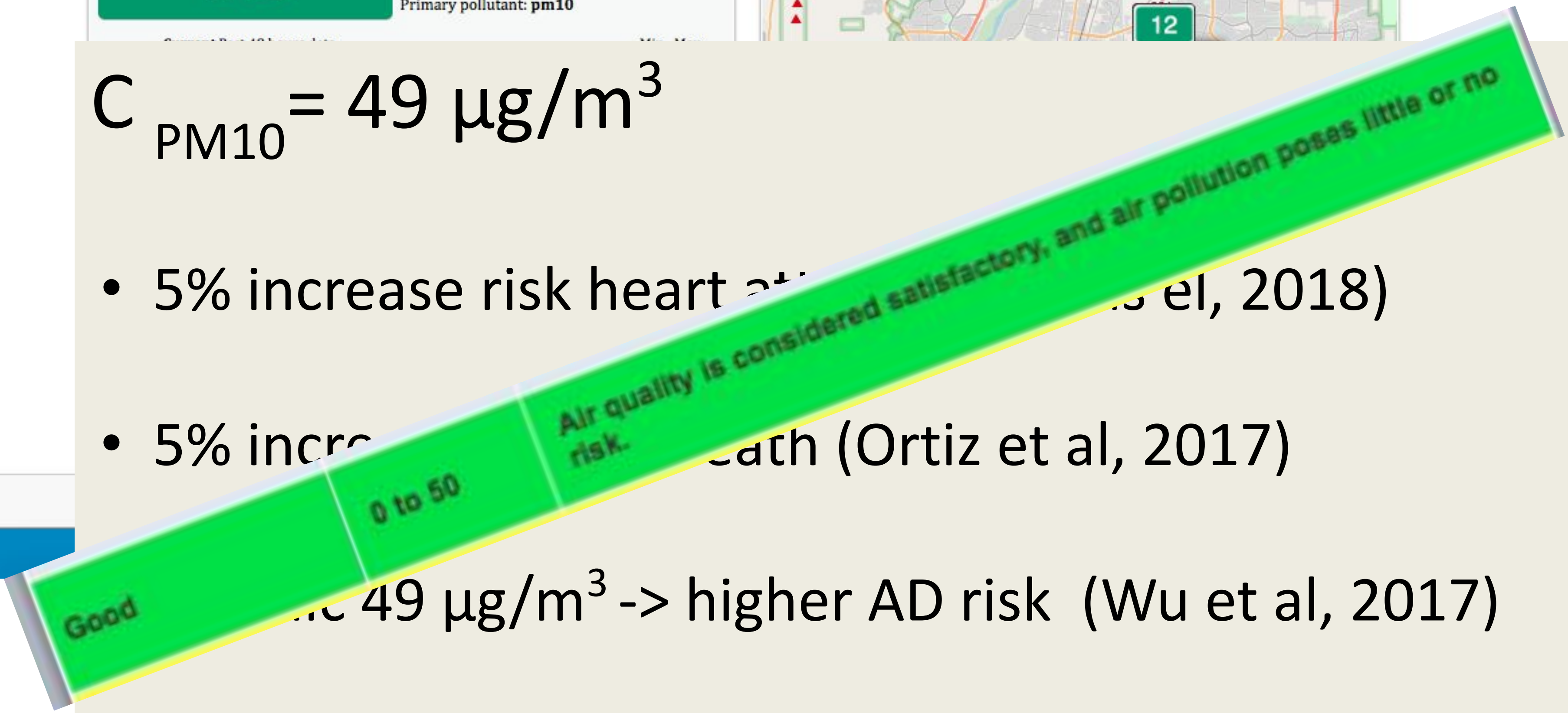
$$C = C_{low} + (C_{high}-C_{low})(I-I_{low})/(I_{igh}-I_{low})$$

O_3 (ppb)	O_3 (ppb)	$PM_{2.5}$ (µg/m³)	PM_{10} (µg/m³)	CO (ppm)				
C_{low} - C_{high} (avg)	C_{low} - C_{high} (avg)	C_{low}- C_{high} (avg)	C_{low} - C_{high} (avg)	C_{low} - C_{high} (a				
0-54 (8-hr)	-	0.0-12.0 (24-hr)	0-54 (24-hr)	0.0-4.4 (8-hr)				
55-70 (8-hr)	-	12.1-35.4 (24-hr)	55-154 (24-hr)	4.5-9.4 (8-hr)				
71-85 (8-hr)	125-164 (1-hr)	35.5-55.4 (24-hr)	155-254 (24-hr)	9.5-12.4 (8-hr)				Groups
86-105 (8-hr)	165-204 (1-hr)	55.5-150.4 (24-hr)	255-354 (24-hr)	12.5-15.4 (8-h				
106-200 (8-hr)	205-404 (1-hr)	150.5-250.4 (24-hr)	355-424 (24-hr)	15.5-30.4 (8-h				
-	405-504 (1-hr)	250.5-350.4 (24-hr)	425-504 (24-hr)	30.5-40.4 (8-hr)	[illegible]	1250-1649 (1-hr)	301-400	Hazardous
-	505-604 (1-hr)	350.5-500.4 (24-hr)	505-604 (24-hr)	40.5-50.4 (8-hr)	805-1004 (24-hr)	1650-2049 (1-hr)	401-500	

PM_{10} (µg/m³)	AQI
C_{low} - C_{high} (avg)	Category
0-54 (24-hr)	Good
55-154 (24-hr)	Moderate
155-254 (24-hr)	Unhealthy for Sensitive Groups
255-354 (24-hr)	Unhealthy
355-424 (24-hr)	Very Unhealthy
425-504 (24-hr)	Hazardous
505-604 (24-hr)	

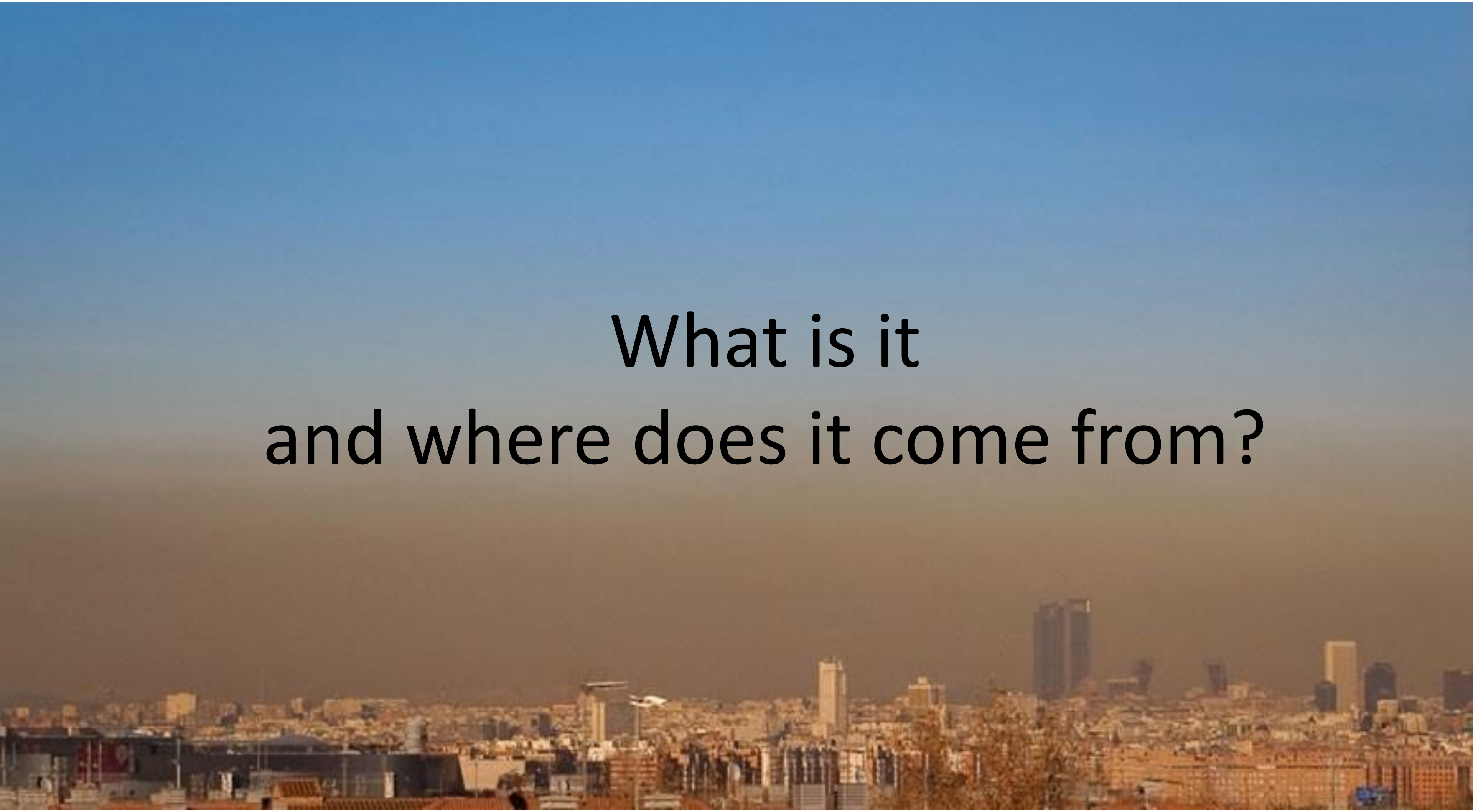


$$C_{PM10} = 49\ \mu g/m^3$$

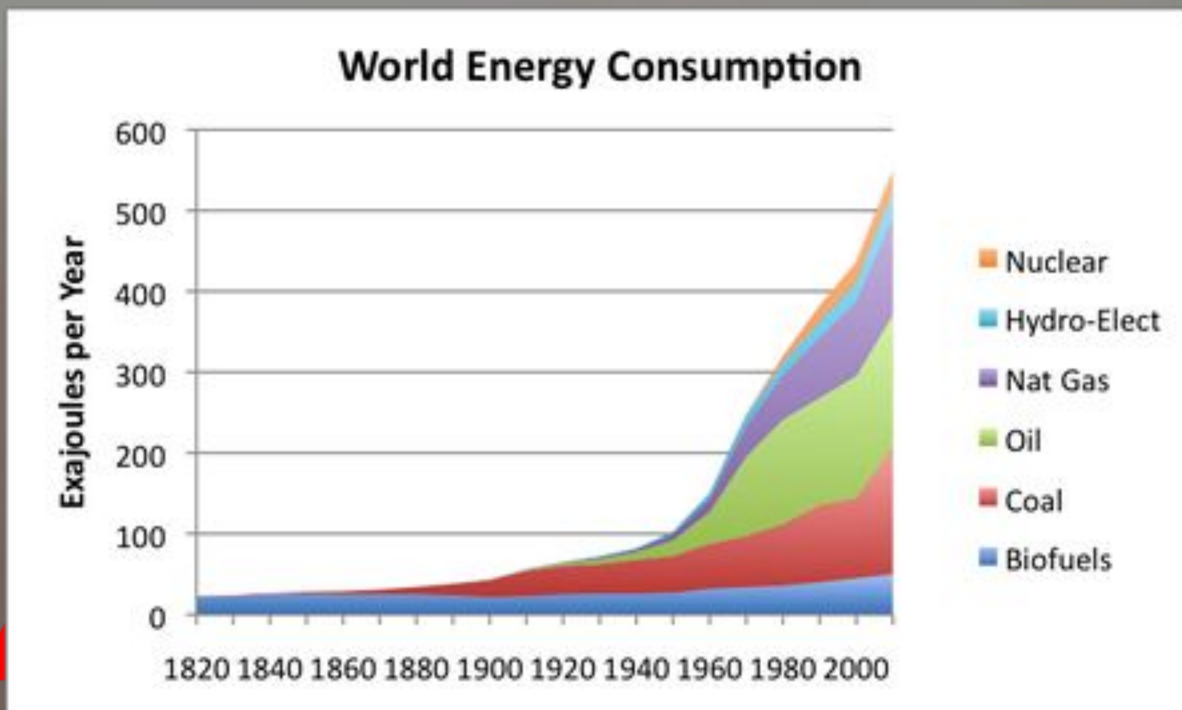


- 5% increase risk heart at… …el, 2018)
- 5% incr… …eath (Ortiz et al, 2017)
- …c 49 µg/m³ -> higher AD risk (Wu et al, 2017)

Supplement 1:
Major Measured Pollutants


What is it
and where does it come from?

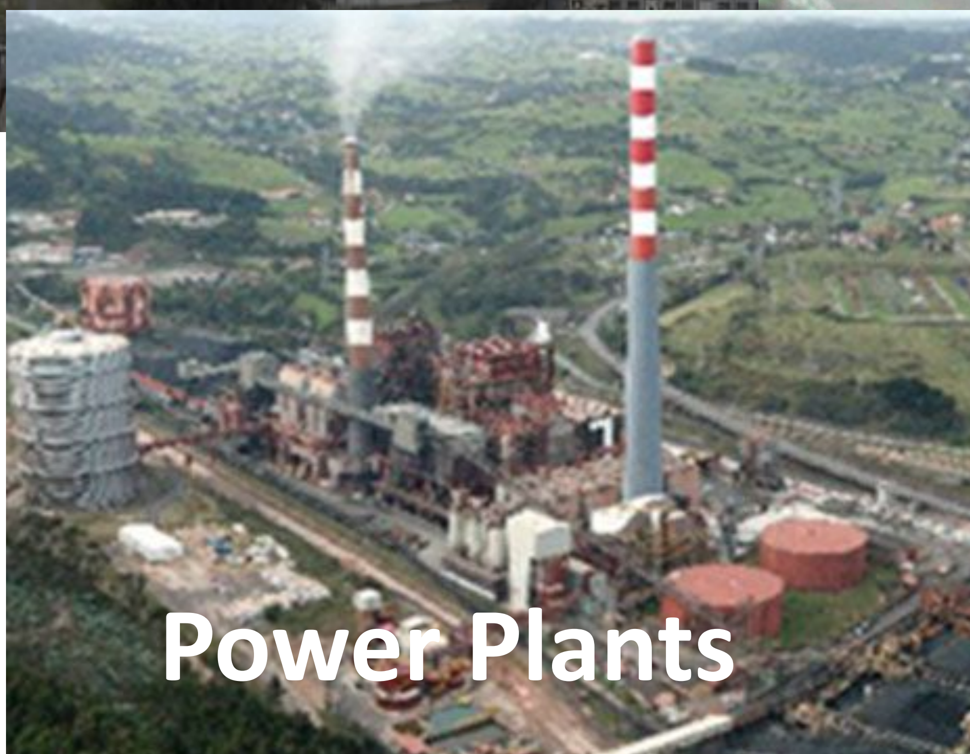
Planet of the Carbon Burners



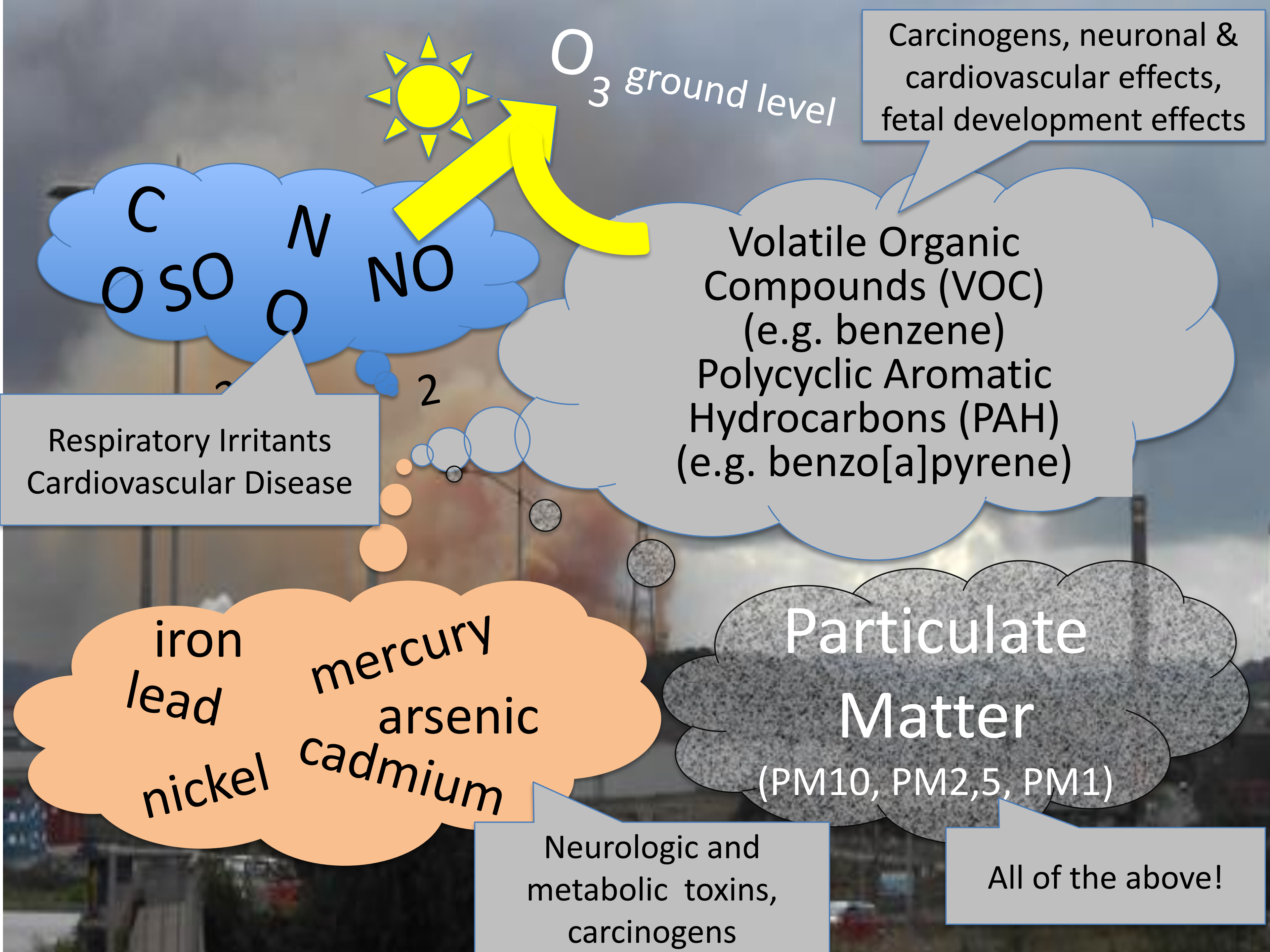
https://ourfiniteworld.com/2012/03/12/world-energy-consumption-since-1820-in-charts/

1963 Clean Air Act **2016**

Major Human Sources



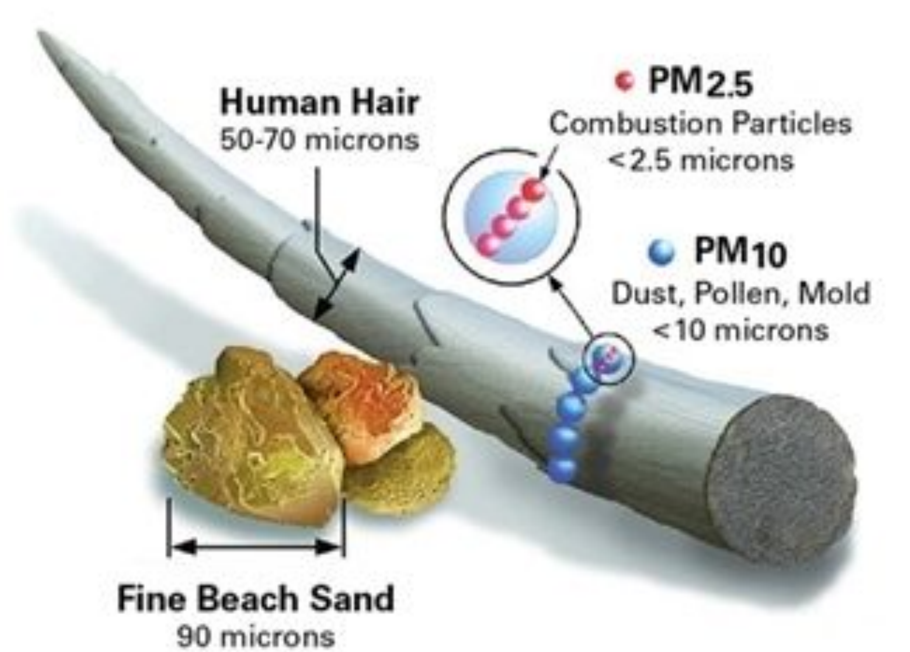
Agriculture, Construction
Fossil Fuel Extraction
Commercial/Institutional
Homes/Buildings
Waste disposal

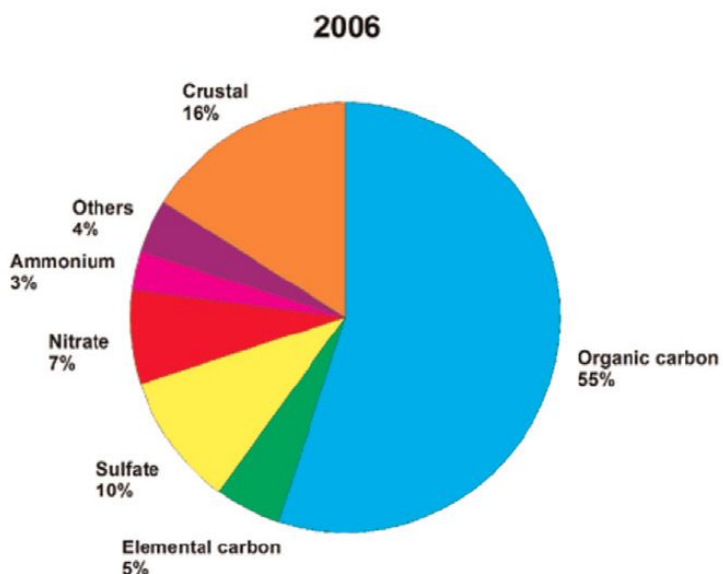


O_3 ground level
Carcinogens, neuronal & cardiovascular effects, fetal development effects
C
O
SO
N
O
NO
2
Respiratory Irritants
Cardiovascular Disease
Volatile Organic Compounds (VOC)
(e.g. benzene)
Polycyclic Aromatic Hydrocarbons (PAH)
(e.g. benzo[a]pyrene)
iron
lead
mercury
arsenic
nickel
cadmium
Particulate Matter
(PM10, PM2,5, PM1)
Neurologic and metabolic toxins, carcinogens
All of the above!

Anthropogenic Particulate Matter:

- Elemental carbon, gases and salts of sulfur, nitrogen, ammonia, metals, benzene, benzo[a]pyrene, toluene and other volatile organic compounds.
- Sizes of <0.1 micrometers can enter the blood circulation via the alveoli, macrophages, or GI tract.
- Evidence of PM entering the brain via the olfactory nerve.





Calderón-Garcidueñas et al., 2016

Supplement 2:
General Health Effects


World Health Organization
Health Topics
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News
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Air pollution
News release: 9 out of 10 people worldwide breathe polluted air
2 May 2018, Geneva – Air pollution levels remain at dangerously high levels in many parts of the world. New data reveals that 9 out of 10 people breathe air containing high levels of pollutants. like black carbon which penetrate deep into the lungs and cardiovascular system.
WHO estimates that around 7 million people die every year from exposure to fine particles in polluted air that lead to diseases such as stroke, heart disease, lung cancer, chronic obstructive pulmonary diseases and respiratory infections, including pneumonia.
9 out of 10 people worldwide breathe polluted air but more countries are taking action
Neuf personnes sur 10 respirent un air pollué dans
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Air quality
Reduction of national emissions
The EU and international air pollution policy
Air Pollution from the main sources
Publications
Infographics
Useful Links
Clean Air
Clean air is essential to our health and to th
breathe has deteriorated considerably - mai
burning of fossil fuels and biomass, as well
towns and cities which, in turn, can lead to
The human toll for poor air quality is worse
of premature death in Europe, with over 40
or exacerbating asthma and respiratory pro
vulnerable groups such as children, asthma
nitrogen pollution (eutrophication) and acid
To counter this, the European Union has set
negative impacts on, and risks to, human h
improve air quality by controlling emissions
integrating environmental protection require
NCBI
Resources
How To
PubMed.gov
US National Library of Medicine National Institutes of Health
PubMed
"air pollution"
Create RSS
Create alert
Advanced
Article types
Clinical Trial
Review
Customize ...
Text availability
Abstract
Free full text
Full text
Publication dates
clear
5 years
10 years
From 2007/01/01 to 2019/12/31
Species
Humans
Other Animals
Clear all
Show additional filters
Format: Summary
Sort by: Most Recent
Per page: 20
Send to
Best matches for "air pollution":
Air pollution during pregnancy and lung development in the child.
Korten I et al. Paediatr Respir Rev. (2017)
Air pollution and Parkinson's disease - evidence and future directions.
Palacios N et al. Rev Environ Health. (2017)
Comparison of Health Impact of Air Pollution Between China and Other Countries.
Tian L et al. Adv Exp Med Biol. (2017)
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1. Household air pollution as an important factor in the complex relationship between altitude and COPD.
Basagaña X.
Eur Respir J. 2019 Feb 7;53(2). pii: 1802454. doi: 10.1183/13993003.02454-2018. Print 2019 Feb. No abstract available.
PMID: 30759422
Similar articles

Toxicologic Pathology, 36:289-310, 2008

ISSN: 0192-6233 print / 1533-1601 online
DOI: 10.1177/0192623307313011

Long-term Air Pollution Exposure Is Associated with Neuroinflammation, an Altered Innate Immune Response, Disruption of the Blood-Brain Barrier, Ultrafine Particulate Deposition, and Accumulation of Amyloid β-42 and α-Synuclein in Children and Young Adults

Lilian Calderón-Garcidueñas,[1,2] Anna C. Solt,[3] Carlos Henríquez-Roldán,[4] Ricardo Torres-Jardón,[5] Bryan Nuse,[2] Lou Herritt,[2] Rafael Villarreal-Calderón,[6] Norma Osnaya,[1] Ida Stone,[2] Raquel García,[1] Diane M. Brooks,[2] Angelica González-Maciel,[1] Rafael Reynoso-Robles,[1] Ricardo Delgado-Chávez,[7] and William Reed[8]

Air pollution is a serious environmental problem. We investigated whether residency in cities with high air pollution is associated with neuroinflammation/neurodegeneration in healthy children and young adults who died suddenly. We measured mRNA cyclooxygenase-2, interleukin-1β, and CD14 in target brain regions from low (n = 12) or highly exposed residents (n = 35) aged 25.1 ± 1.5 years. Upregulation of cyclooxygenase-2, interleukin-1β, and CD14 in olfactory bulb, frontal cortex, substantia nigrae and vagus nerves; disruption of the blood-brain barrier; endothelial activation, oxidative stress, and inflammatory cell trafficking were seen in highly exposed subjects. Amyloid β42 (Aβ42) immunoreactivity was observed in 58.8% of apolipoprotein E (APOE) 3/3 < 25 y, and 100% of the APOE 4 subjects, whereas α-synuclein was seen in 23.5% of < 25 y subjects. Particulate material (PM) was seen in olfactory bulb neurons, and PM < 100 nm were observed in intraluminal erythrocytes from lung, frontal, and trigeminal ganglia capillaries.

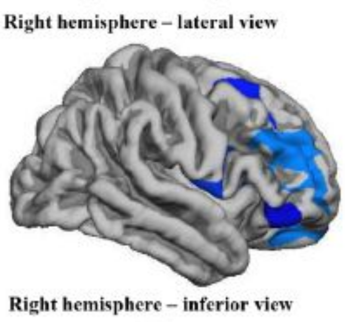
Exposure to air pollution causes neuroinflammation, an altered brain innate immune response, and accumulation of Aβ42 and α-synuclein starting in childhood. Exposure to air pollution should be considered a risk factor for Alzheimer's and Parkinson's diseases, and carriers of the APOE 4 allele could have a higher risk of developing Alzheimer's disease if they reside in a polluted environment.

Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children (6-10 y.o.)

Mònica Guxens, Małgorzata J. Lubczyńska, Ryan L. Muetzel, Albert Dalmau-Bueno, Vincent W.V. Jaddoe, Gerard Hoek, Aad van der Lugt, Frank C. Verhulst, Tonya White, Bert Brunekreef, Henning Tiemeier, and Hanan El Marroun

Biological Psychiatry August 15, 2018; 84:295–303

A Fine particles exposure



CONCLUSIONS: Exposure to fine particles during fetal life was related to child brain structural alterations of the cerebral cortex, and these alterations partially mediated the association between exposure to fine particles during fetal life and impaired child inhibitory control. Such cognitive impairment at early ages could have significant long-term consequences.

Neurod

2618 young students (~8.5 y) were tested for memory and attention 4 times over 1 year. PM2.5 was measured during twice over 1-week periods and analyzed for source. Traffic-related PM2.5 correlated inversely with preformance on the tests.

Research | Children's Health

Neurodevelopmental Deceleration by Urban Fine Particles from Different Emission Sources: A Longitudinal Observational Study

Xavier Basagaña,[1,2,3] Mikel Esnaola,[1,2,3] Ioar Rivas,[1,2,3,4] Fulvio Amato,[4] Mar Alvarez-Pedrerol,[1,2,3] Joan Forns,[1,2,3,5] Mònica López-Vicente,[1,2,3] Jesús Pujol,[6,7] Mark Nieuwenhuijsen,[1,2,3] Xavier Querol,[4] and Jordi Sunyer[1,2,3,8]

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2715 young students (7-10 años) showed less improvements in cognitive tasks during 1 year if they were exposed to the highest levels of elemental carbon, NO_2, y PM2.5 in the study.

EDICINE

RESEARCH ARTICLE

Association between Traffic-Related Air Pollution in Schools and Cognitive Development in Primary School Children: A Prospective Cohort Study

Jordi Sunyer*[1,2,3,4], Mikel Esnaola[1,2,3], Mar Alvarez-Pedrerol[1,2,3], Joan Forns[1,2,3], Ioar Rivas[1,2,3,5], Mònica López-Vicente[1,2,3], Elisabet Suades-González[1,2,3,6], Maria Foraster[1,2,3], Raquel Garcia-Esteban[1,2,3], Xavier Basagaña[1,2,3], Mar Viana[5], Marta Cirach[1,2,3], Teresa Moreno[5], Andrés Alastuey[5], Núria Sebastian-Galles[2], Mark Nieuwenhuijsen[1,2,3], Xavier Querol[5]

1 Centre for Research in Environmental Epidemiology (CREAL), Barcelona, Catalonia, Spain, 2 Pompeu Fabra University, Barcelona, Catalonia, Spain, 3 Consortium for Biomedical Research in Epidemiology and Public Health (CIBERESP), Madrid, Spain, 4 Institut Hospital del Mar d'Investigacions Mèdiques–Parc de Salut Mar, Barcelona, Catalonia, Spain, 5 Institute of Environmental Assessment and Water Research

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ORIGINAL CONTRIBUTION

Long-Term Exposure to Particulate Matter Air Pollution Is a Risk Factor for Stroke

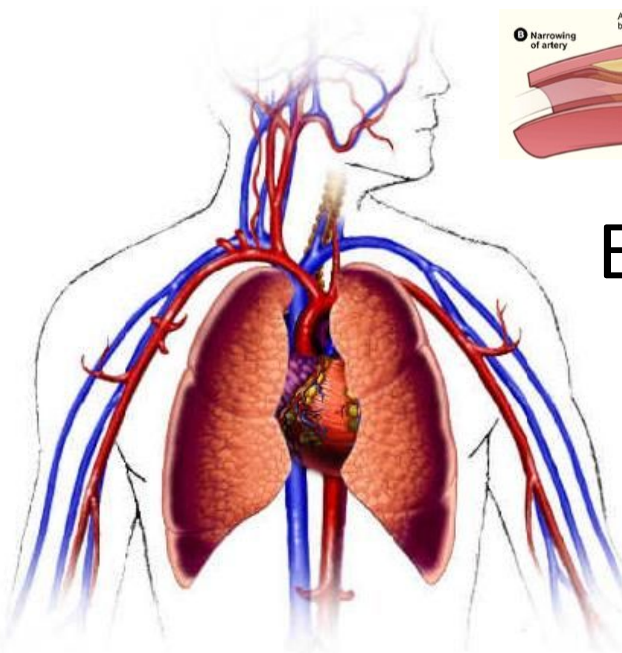
Meta-Analytical Evidence

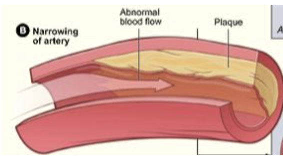
Hans Scheers, Lotte Jacobs, Lidia Casas, Benoit Nemery and Tim S. Nawrot

Download PDF

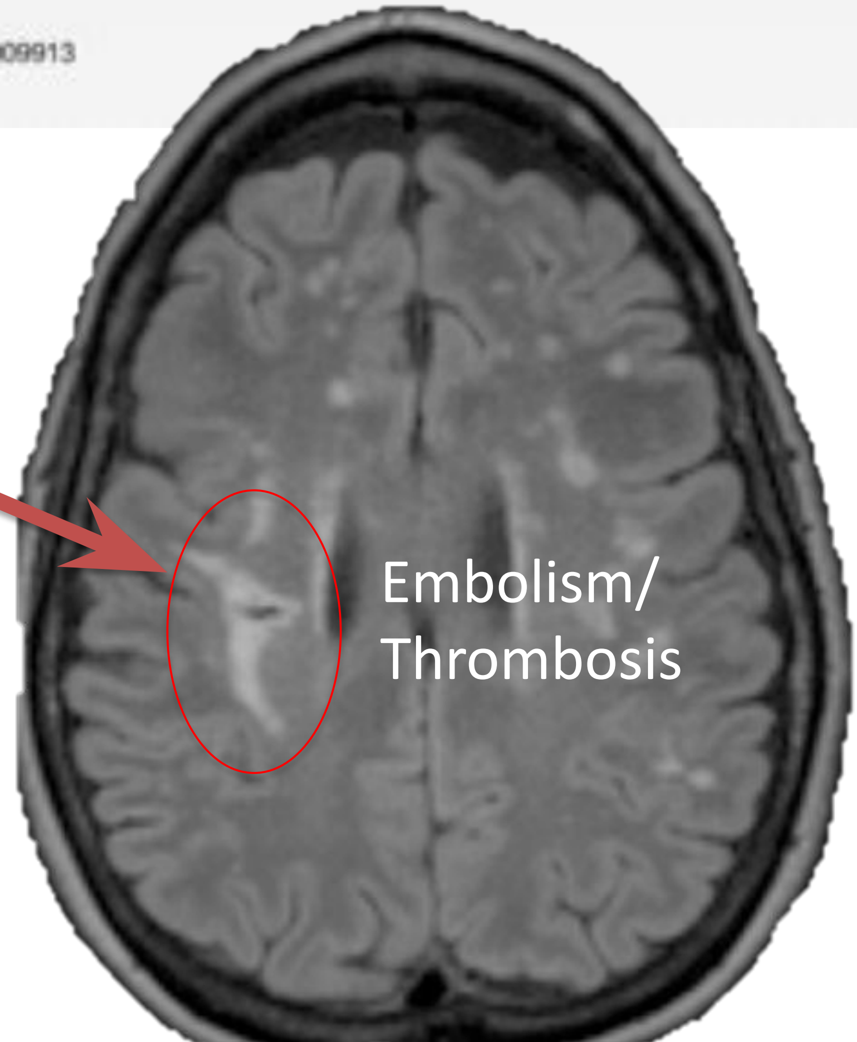
DOI http://dx.doi.org/10.1161/STROKEAHA.115.009913
Stroke. 2015;46:3058-3066
Originally published October 13, 2015

PM → Arterioesclerosis





Emboli



Published in final edited form as:
Arch Intern Med. 2012 February 13; 172(3): 219–227. doi:10.1001/archinternmed.2011.683.

Exposure to Particulate Air Pollution and Cognitive Decline in Older Women

Dr. Jennifer Weuve, MPH, ScD, Dr. Robin C. Puett, MPH, PhD, Dr. Joel Schwartz, PhD, Dr. Jeff D. Yanosky, MS, ScD, Dr. Francine Laden, MS, ScD, and Dr. Francine Grodstein, ScD

130,978 adults in London aged 50–79 years with no history of dementia. Average NO2, PM2.5 and O3 levels during 2004 were estimated. Traffic intensity, distance from major road and night-time noise levels were estimated at the subject postal codes and linked to clinical data. Diagnoses of dementia were noted during **2005–2013**. **There was a positive exposure response relationship between dementia and all measures of air pollution except O3.**

BMJ Open

Are noise and air pollution related to the incidence of dementia? A cohort study in London, England

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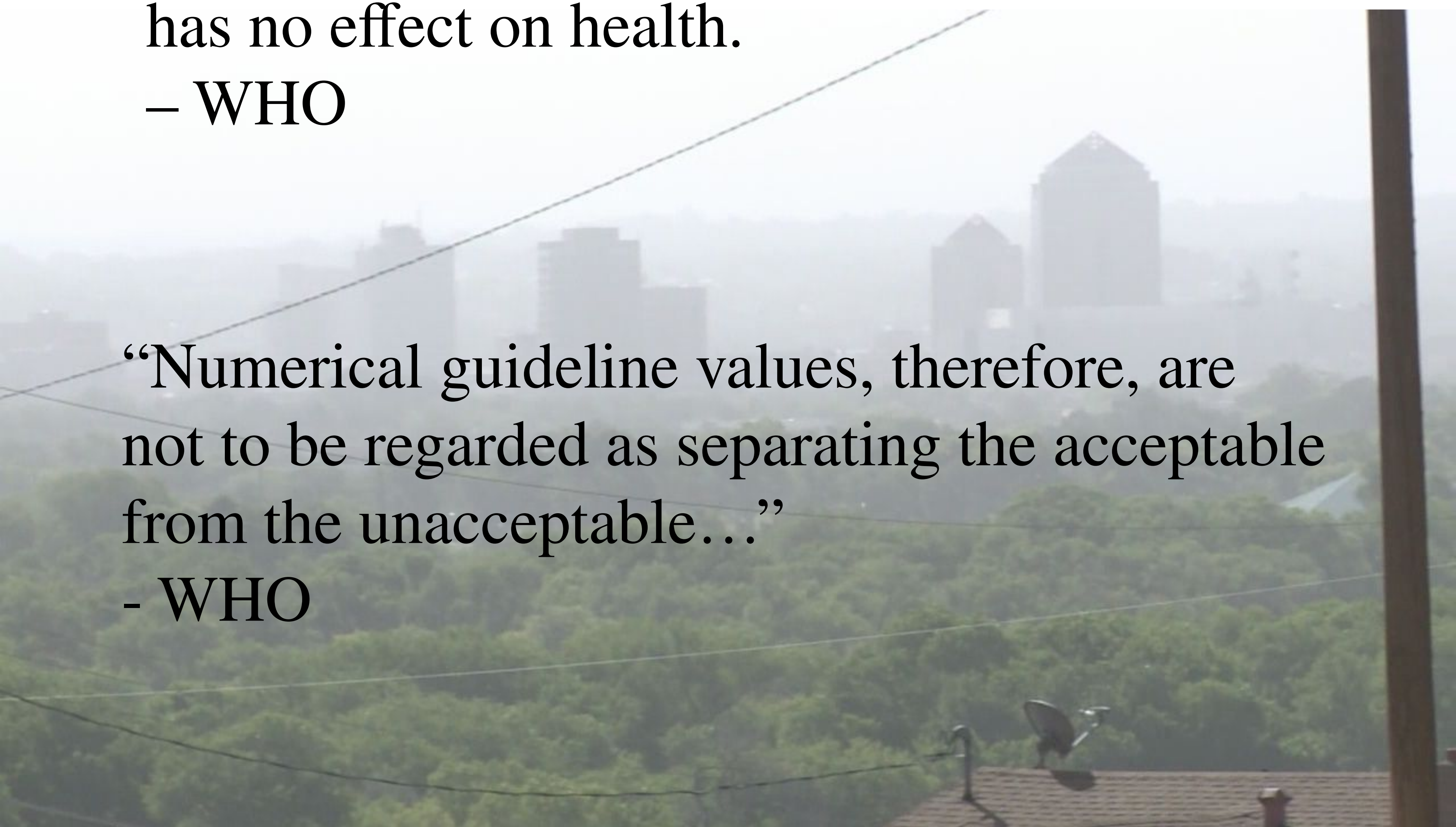
How much is healthy?
GREENPEACE

There is no threshold below which a pollutant has no effect on health.
– WHO



"Numerical guideline values, therefore, are not to be regarded as separating the acceptable from the unacceptable…"
- WHO

All Medicare beneficiaries (60,925,443 persons)min the continental United States from the years 2000 through 2012,

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Air Pollution and Mortality in the Medicare Population

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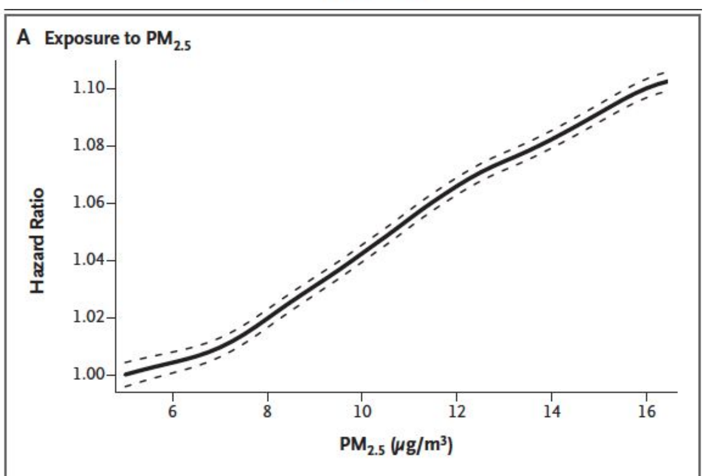

RESULTS

Increases of 10 μg per cubic meter in $PM_{2.5}$ and of 10 ppb in ozone were associated with increases in all-cause mortality of 7.3% (95% confidence interval [CI], 7.1 to 7.5) and 1.1% (95% CI, 1.0 to 1.2), respectively. When the analysis was restricted to person-years with exposure to $PM_{2.5}$ of less than 12 μg per cubic meter and ozone of less than 50 ppb, the same increases in $PM_{2.5}$ and ozone were associated with increases in the risk of death of 13.6% (95% CI, 13.1 to 14.1) and 1.0% (95% CI, 0.9 to 1.1), respectively. For $PM_{2.5}$, the risk of death among men, blacks, and people with Medicaid eligibility was higher than that in the rest of the population.

JAMA | Original Investigation

Association of Short-term Exposure to Air Pollution With Mortality in Older Adults

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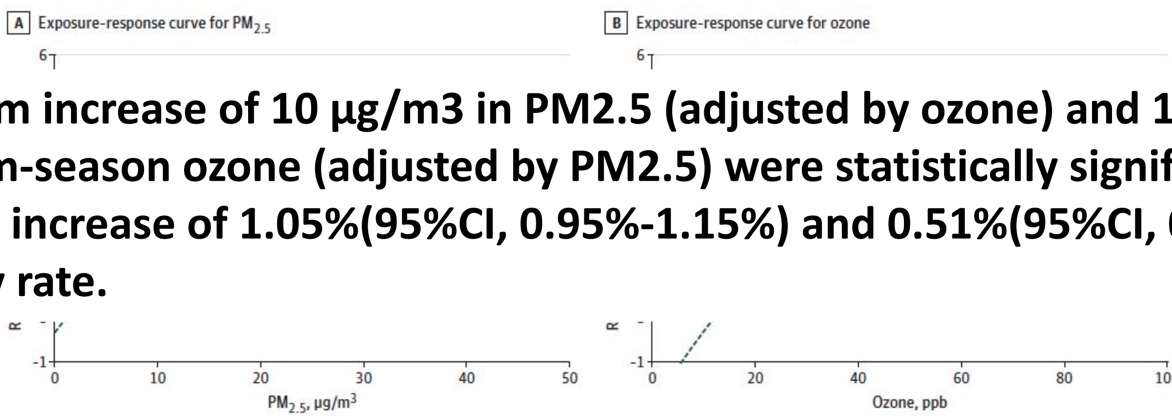
Figure 5. Estimated Exposure-Response Curves for Short-term Exposures to Fine Particulate Matter ($PM_{2.5}$) and Ozone



Each short-term increase of 10 μg/m3 in PM2.5 (adjusted by ozone) and 10 parts per billion (10−9) in warm-season ozone (adjusted by PM2.5) were statistically significantly associated with a relative increase of 1.05%(95%CI, 0.95%-1.15%) and 0.51%(95%CI, 0.41%-0.61%) in daily mortality rate.

A 2-pollutant analysis with separate penalized splines on $PM_{2.5}$ (A) and ozone (B) was conducted to assess the percentage increase in daily mortality at various pollution levels. Dashed lines indicate 95% CIs. The mean of daily exposure on the same day of death and 1 day prior (lag 01-day) was used as metrics of exposure to $PM_{2.5}$ and ozone. Analysis for ozone was restricted to the warm season (April to September). Ppb indicates parts per billion.

CONCLUSIONS AND RELEVANCE In the US Medicare population from 2000 to 2012, short-term exposures to $PM_{2.5}$ and warm-season ozone were significantly associated with increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated.

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Short-term exposure to air pollutants increases the risk of ST elevation myocardial infarction and of infarct-related ventricular arrhythmias and mortality

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Results: The daily rate of hospital admissions for STEMI was associated with increases in PM 2.5, PM 10, lead and NO2 concentrations. VA incidence and mortality were associated with increases in PM 2.5 and PM 10 concentrations. In the most specific cohort, BCN (Barcelona) Attended & Resident, STEMI incidence was associated with increases in PM 2.5 (1.009% per 10 $\mu g/m^3$) and PM 10 concentrations (1.005% per 10 $\mu g/m^3$). VA was associated with increases in PM 2.5 (1.021%) and PM 10 (1.015%) and mortality was associated with increases in PM 2.5 (1.083%) and PM 10 (1.045%).

Conclusions: Short-term exposure to high levels of PM 2.5 and PM 10 is associated with increased daily STEMI admissions and STEMI-related VA and mortality. Exposure to high levels of lead and NO2 is associated with increased daily STEMI admissions, and NO2 with higher mortality in STEMI patients.

Global mortality from outdoor fine particle pollution generated by fossil fuel combustion: Results from GEOS-Chem

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ABSTRACT

The burning of fossil fuels – especially coal, petrol, and diesel – is a major source of airborne fine particulate matter ($PM_{2.5}$), and a key contributor to the global burden of mortality and disease. Previous risk assessments have examined the health response to total $PM_{2.5}$, not just $PM_{2.5}$ from fossil fuel combustion, and have used a concentration-response function with limited support from the literature and data at both high and low concentrations. This assessment examines mortality associated with $PM_{2.5}$ from only fossil fuel combustion, making use of a recent meta-analysis of newer studies with a wider range of exposure. We also estimated mortality due to lower respiratory infections (LRI) among children under the age of five in the Americas and Europe, regions for which we have reliable data on the relative risk of this health outcome from $PM_{2.5}$ exposure. We used the chemical transport model GEOS-Chem to estimate global exposure levels to fossil-fuel related $PM_{2.5}$ in 2012. Relative risks of mortality were modeled using functions that link long-term exposure to $PM_{2.5}$ and mortality, incorporating nonlinearity in the concentration response. We estimate a global total of 10.2 (95% CI: −47.1 to 17.0) million premature deaths annually attributable to the fossil-fuel component of $PM_{2.5}$. The greatest mortality impact is estimated over regions with substantial fossil fuel related $PM_{2.5}$, notably China (3.9 million), India (2.5 million) and parts of eastern US, Europe and Southeast Asia. The estimate for China predates substantial decline in fossil fuel emissions and decreases to 2.4 million premature deaths due to 43.7% reduction in fossil fuel $PM_{2.5}$ from 2012 to 2018 bringing the global total to 8.7 (95% CI: −1.8 to 14.0) million premature deaths. We also estimated excess annual deaths due to LRI in children (0–4 years old) of 876 in North America, 747 in South America, and 605 in Europe. This study demonstrates that the fossil fuel component of $PM_{2.5}$ contributes a large mortality burden. The steeper concentration-response function slope at lower concentrations leads to larger estimates than previously found in Europe and North America, and the slower drop-off in slope at higher concentrations results in larger estimates in Asia. Fossil fuel combustion can be more readily controlled than other sources and precursors of $PM_{2.5}$ such as dust or wildfire smoke, so this is a clear message to policymakers and stakeholders to further incentivize a shift to clean sources of energy.

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Microglial priming through the lung–brain axis: the role of air pollution–induced circulating factors

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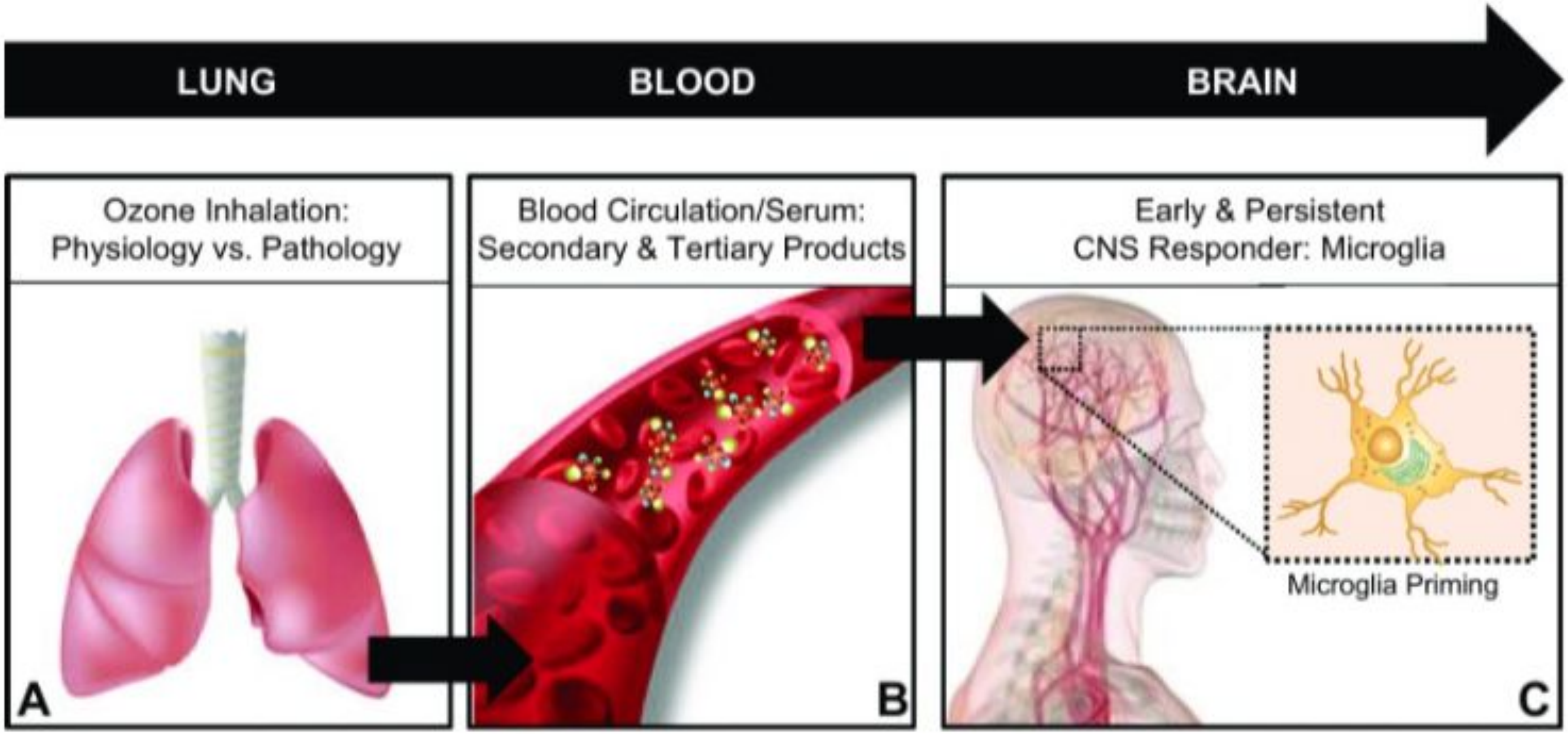

The lung–brain axis. Circulating factors in response to air pollutants prime microglia. Increasing evidence supports the

Supplement 3:
Air Quality Guidelines

العربية 中文 Français Русский Español

New WHO Global Air Quality Guidelines aim to save millions of lives from air pollution

Air pollution is one of the biggest environmental threats to human health, alongside climate change.

What are the US air quality standards?

National Ambient Air Quality Standards (NAAQS)
Clean Air Act (1963, 1970 and amendments 1971-2016)

Pollutant	Type	Standard	Averaging Time	
Sulfur dioxide (SO_2)	Primary	.075 ppm (195 µg/m³)	1-hour	
	Secondary	0.5 ppm (1,300 µg/m³)	3-hour	
Particulate matter (PM_{10})	Primary and Secondary	150 µg/m³	24-hour	
Particulate matter ($PM_{2.5}$)	Primary	12 µg/m³	annual	
	Secondary	15 µg/m³	annual	
	Primary and Secondary	35 µg/m³	24-hour	
Carbon monoxide (CO)	Primary	35 ppm (40 mg/m³)	1-hour	
	Primary	9 ppm (10 mg/m³)	8-hour	
Ozone (O_3)	Primary and Secondary	0.12 ppm (235 µg/m³)	1-hour	hourly a to or less than 1
	Primary and Secondary	0.070 ppm (140 µg/m³)	8-hour	
Nitrogen dioxide (NO_2)	Primary and Secondary	0.053 ppm (100 µg/m³)	annual	
Lead (Pb)	Primary and Secondary	0.15 µg/m³	Rolling 3 mo	

EU

SO_2	350 µg/m³	-1 h
	125 µg/m³	- 24 h
PM_{10}	50 µg/m³	- 24 h
	40 µg/m³	- annual
$PM_{2.5}$	25 µg/m³	- annual
CO	10 mg/m³	- 8 h
O_3	120 µg/m³	- 8 h
NO_2	200 µg/m³	- 1h
	40 µg/m³	- annual
Lead	0.5 µg/m³	- annual

But what is considered “good” air in Albuquerque NM USA?

The Air Quality Index?

Air Quality Index Levels of Health Concern	Numerical Value	Meaning
Good	0 to 50	Air quality is considered satisfactory, and air pollution poses little or no risk.
Moderate	51 to 100	Air quality is acceptable; however, for some pollutants there may be a moderate health concern for a very small number of people who are unusually sensitive to air pollution.
Unhealthy for Sensitive Groups	101 to 150	Members of sensitive groups may experience health effects. The general public is not likely to be affected.
Unhealthy	151 to 200	Everyone may begin to experience health effects; members of sensitive groups may experience more serious health effects.
Very Unhealthy	201 to 300	Health alert: everyone may experience more serious health effects.
Hazardous	301 to 500	Health warnings of emergency conditions. The entire population is more likely to be affected.