

Environmental and Occupational Lung Disease

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Environmental and occupational lung disease

- Review major determinants of air quality and air pollution
- Discuss lung health effects of outdoor and indoor air pollution
- Discuss major causes of occupational lung disease

Pollutant	Outdoor Sources	Indoor Sources	Adverse Effects
Sulfur oxides	Power plants, oil refineries, smelters	Kerosene space heaters	Bronchoconstriction
Oxides of nitrogen	Motor vehicle exhaust, power plants, oil refineries	Gas stoves and furnaces, kerosene space heaters	Airway injury (respiratory bronchiolitis), impaired lung defenses, enhanced response to allergen
Ozone	Motor vehicle exhaust	Aircraft cabins, welding, copiers, ozone generators	Airway injury (respiratory bronchiolitis), decreased lung function, exacerbations of asthma, enhanced response to allergen
Particulate matter	Motor vehicle exhaust, power plants	Tobacco and wood smoke	Exacerbations of asthma and chronic obstructive pulmonary disease, increased cardiopulmonary mortality
Radon		Residential basements	Lung cancer
Polycyclic hydrocarbons	Diesel exhaust	Tobacco smoke	Lung cancer

Sulfur oxides

- Produced by the combustion of sulfur contained in fossil fuels such as coal and crude oil
- Environmental sources are power plants, oil refineries, smelters, and paper pulp mills
- In the United States, sulfur dioxide levels are generally higher in the northeastern and midwestern states, primarily because of the use of high-sulfur-content coal in power plants
- Sulfur dioxide is a clear, highly water-soluble gas, effectively absorbed by the mucosal surfaces of the upper airways
- Sulfur dioxide undergoes chemical reactions with water, trace metals, and other pollutants to form a variety of particulate aerosols

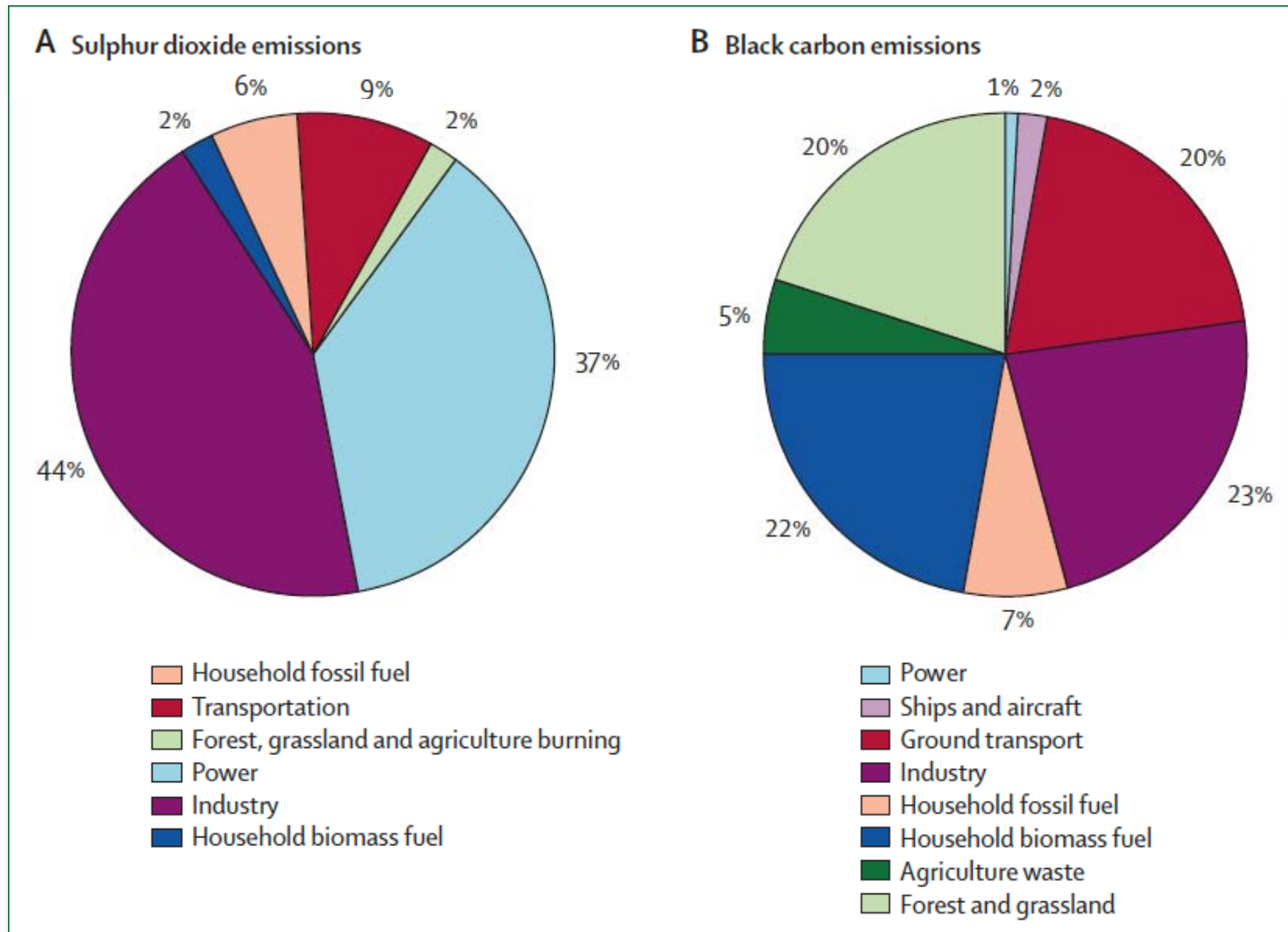
Ozone and nitrogen oxides

- Primarily products of the action of sunlight on emissions of internal combustion engines
- Most important of these products: unburned hydrocarbons and NO_2 , an oxidation product of nitrogen oxide, itself a product of the fixation of atmospheric nitrogen with oxygen under conditions of high-temperature combustion
- Ultraviolet irradiation of this mixture in the atmosphere results in a complex series of chemical reactions, producing ozone, alkyl nitrates, peroxyacyl nitrates, alcohols, ethers, acids, and peroxyacids
- This mixture of pollutants typifies the "smog" of areas with large numbers of automobiles and abundant sunlight, such as the Los Angeles basin
- These gases are relatively insoluble and poorly absorbed by the upper airways. A high proportion of the dose inhaled thus reaches the peripheral portion of the lungs and can cause injury at any site from the upper airways to the alveoli

Particulates

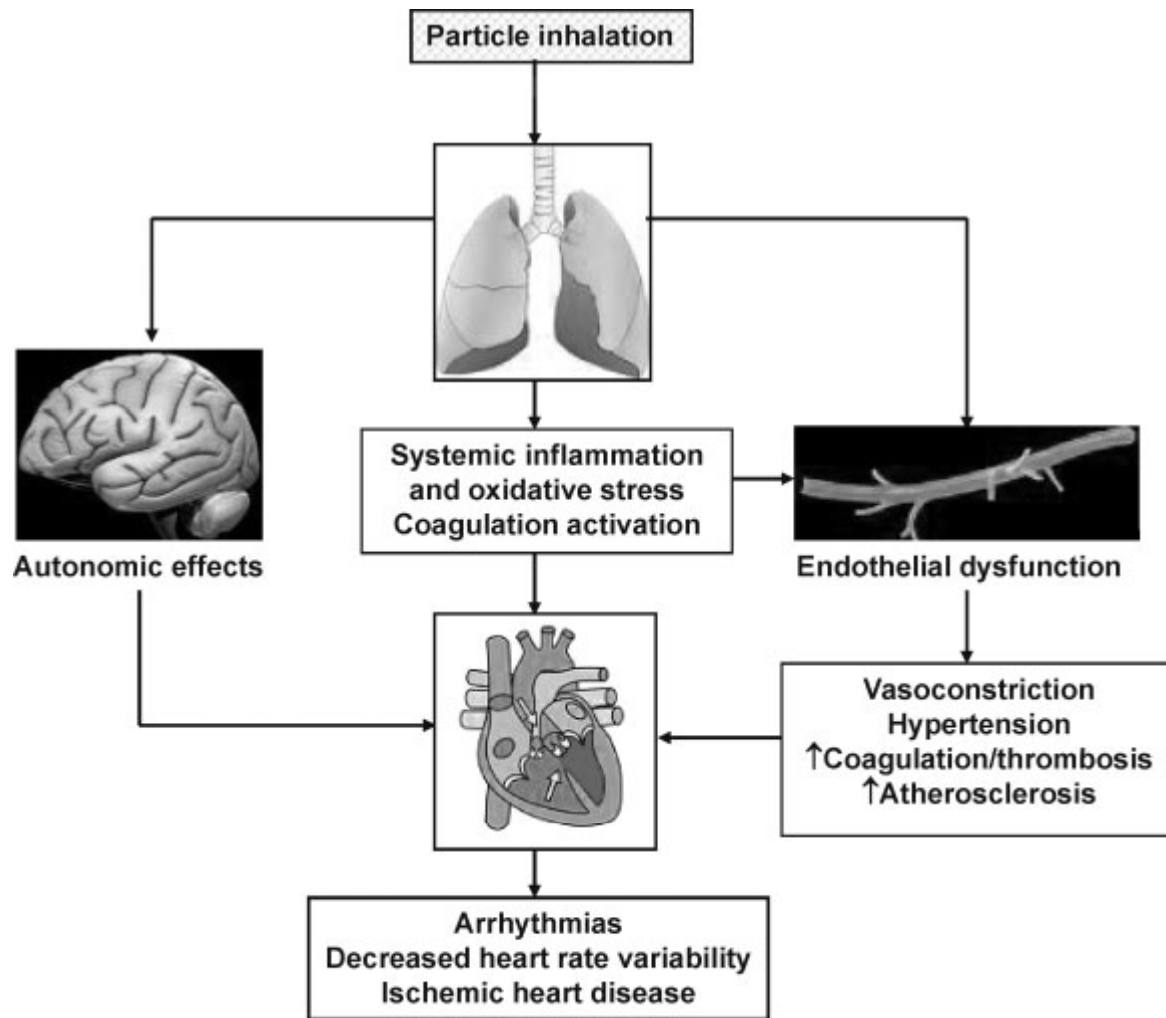
- Atmospheric particulate air pollution arises from a variety of sources, both natural (e.g., sea spray, windblown dust) and synthetic sources (e.g., power plants, motor vehicles)
- Particles classified as "fine" ($<2.5 \mu\text{m}$, $\text{PM}_{2.5}$) can travel for long distances with atmospheric half-lives of days to weeks
 - "Fine" mode particles are composed mainly of varying amounts of water and six major components (sulfates, acids, nitrates, elemental carbon, organic carbon, and trace metals)
- "Coarse" particles ($>2.5 \mu\text{m}$) travel for short distances with atmospheric half-lives of minutes to hours.
 - "Coarse" mode particles are composed primarily of crustal (rock, soil), biologic (pollen, spores), and industrial components;

Relative contributions of human sources to sulfur dioxide (A) and black carbon (B) emissions



U.S. air quality standards

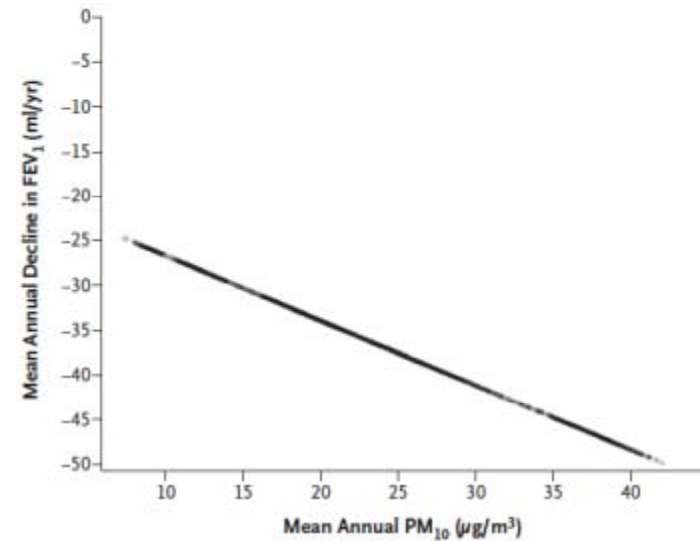
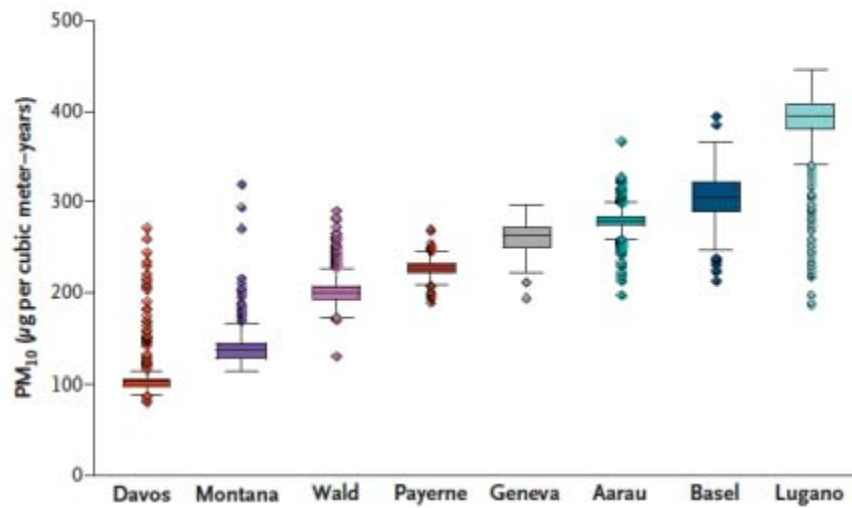
Pollutant	Standard	Averaging Period
Carbon monoxide	9 ppm (10 mg/m ³)	8 hr
	35 ppm (40 mg/m ³)	1 hr
Nitrogen dioxide	0.053 ppm (100 µg/m ³)	1 yr
Ozone	0.12 ppm (235 µg/m ³)	1 hr
	0.08 ppm (157 µg/m ³)	8 hr
Particulate <10 µm (PM ₁₀)	50 µg/m ³	1 yr
	150 µg/m ³	24 hr
Particulate <2.5 µm (PM _{2.5})	15 µg/m ³	1 yr
	65 µg/m ³	24 hr
Sulfur dioxide	0.03 ppm (80 µg/m ³)	1 yr
	0.14 ppm (365 µg/m ³)	24 hr
Lead	1.5 µg/m ³	3 mo



Air pollution



Effect of PM₁₀ exposure on lung function decline in adults



Downs SH et al. N Engl J Med 2007;357:2338-47.

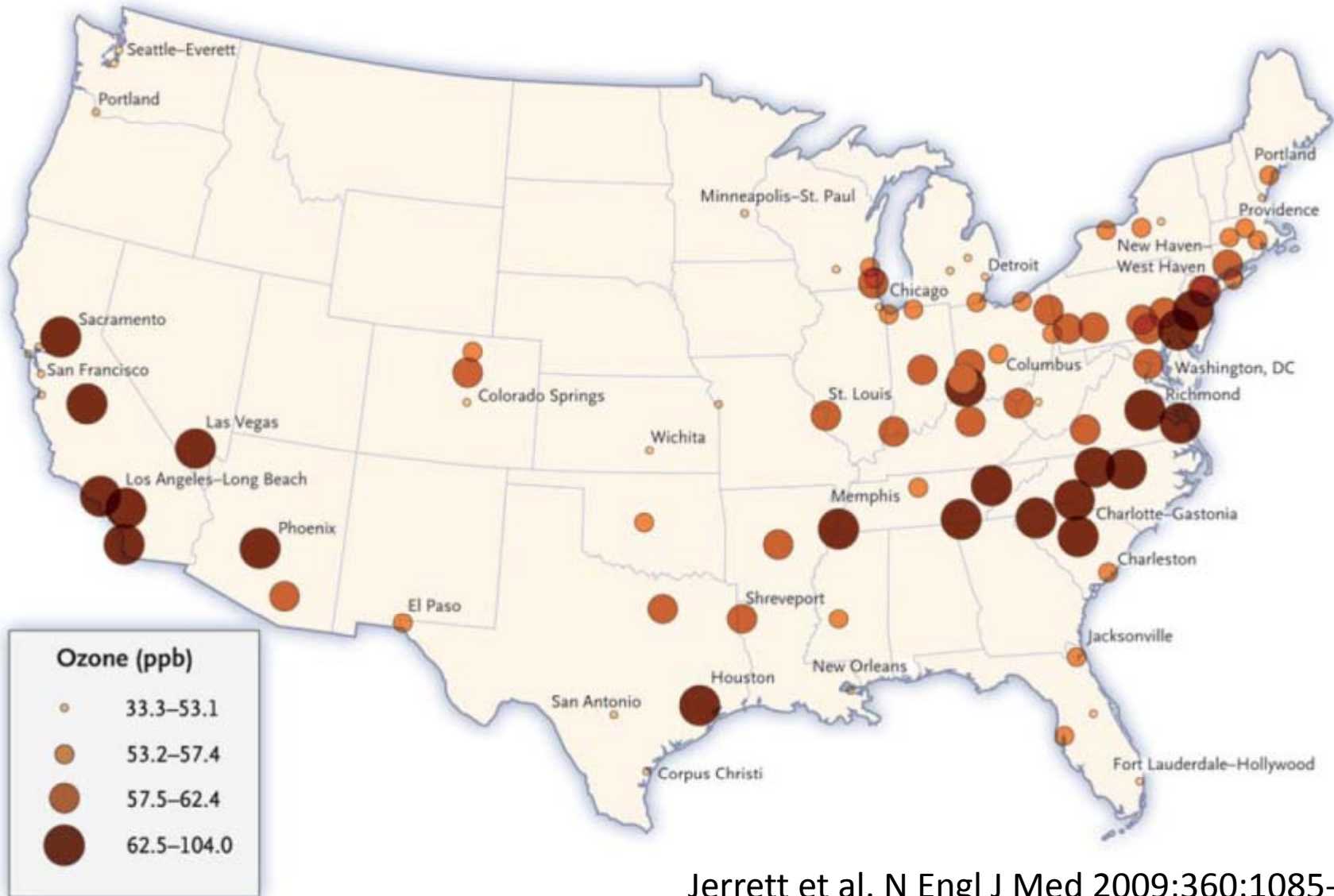
Exposure to PM_{2.5} and lung cancer risk

- American Cancer Society/Cancer Prevention II Study has followed 1.2 million people since 1982
- Cause of death through 1998 was examined in relation to air pollution in metropolitan areas throughout the United States
- Risk of mortality for every 10 µg/m³ increase in PM_{2.5} :

Cause of Mortality	Adjusted RR (95% CI)*		
	1979-1983	1999-2000	Average
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other cause	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

Pope et al. JAMA. 2002;287(9):1132-1141

Average ozone concentrations 1977-2000, US



Jerrett et al. N Engl J Med 2009;360:1085-95

Relative Risk of Death Attributable to a 10-ppb Change in the Ambient Ozone Concentration

Cause of Death	Single-Pollutant Model†			Two-Pollutant Model‡	
	Ozone (96 MSAs)	Ozone (86 MSAs)	PM _{2.5} (86 MSAs)	Ozone (86 MSAs)	PM _{2.5} (86 MSAs)
	<i>relative risk (95% CI)</i>				
Any cause	1.001 (0.996–1.007)	1.001 (0.996–1.007)	1.048 (1.024–1.071)	0.989 (0.981–0.996)	1.080 (1.048–1.113)
Cardiopulmonary	1.014 (1.007–1.022)	1.016 (1.008–1.024)	1.129 (1.094–1.071)	0.992 (0.982–1.003)	1.153 (1.104–1.204)
Respiratory	1.029 (1.010–1.048)	1.027 (1.007–1.046)	1.031 (0.955–1.113)	1.040 (1.013–1.067)	0.927 (0.836–1.029)
Cardiovascular	1.011 (1.003–1.023)	1.014 (1.005–1.023)	1.150 (1.111–1.191)	0.983 (0.971–0.994)	1.206 (1.150–1.264)
Ischemic heart disease	1.015 (1.003–1.026)	1.017 (1.006–1.029)	1.211 (1.156–1.268)	0.973 (0.958–0.988)	1.306 (1.226–1.390)

Jerrett et al. N Engl J Med 2009;360:1085-95

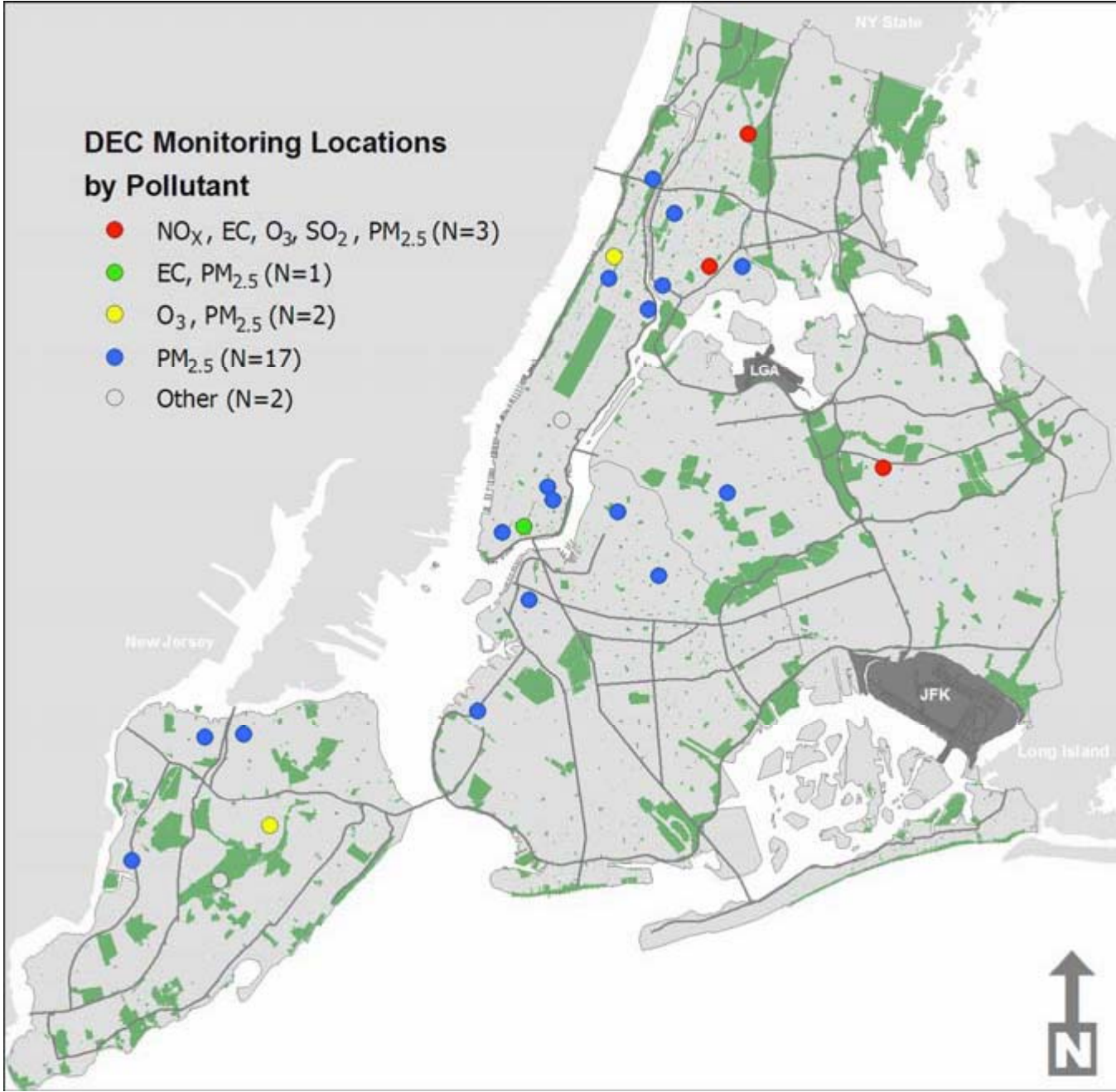
The New York City Community Air Survey



NYCCAS Air Sampling Unit

Air pollutants measured by NYCCAS:

- Fine Particles (PM_{2.5})
- Elemental Carbon (EC)
- Sulfur Dioxide (SO₂)
- Nitrogen Oxides (NO_x)
- Ground-level Ozone (O₃)

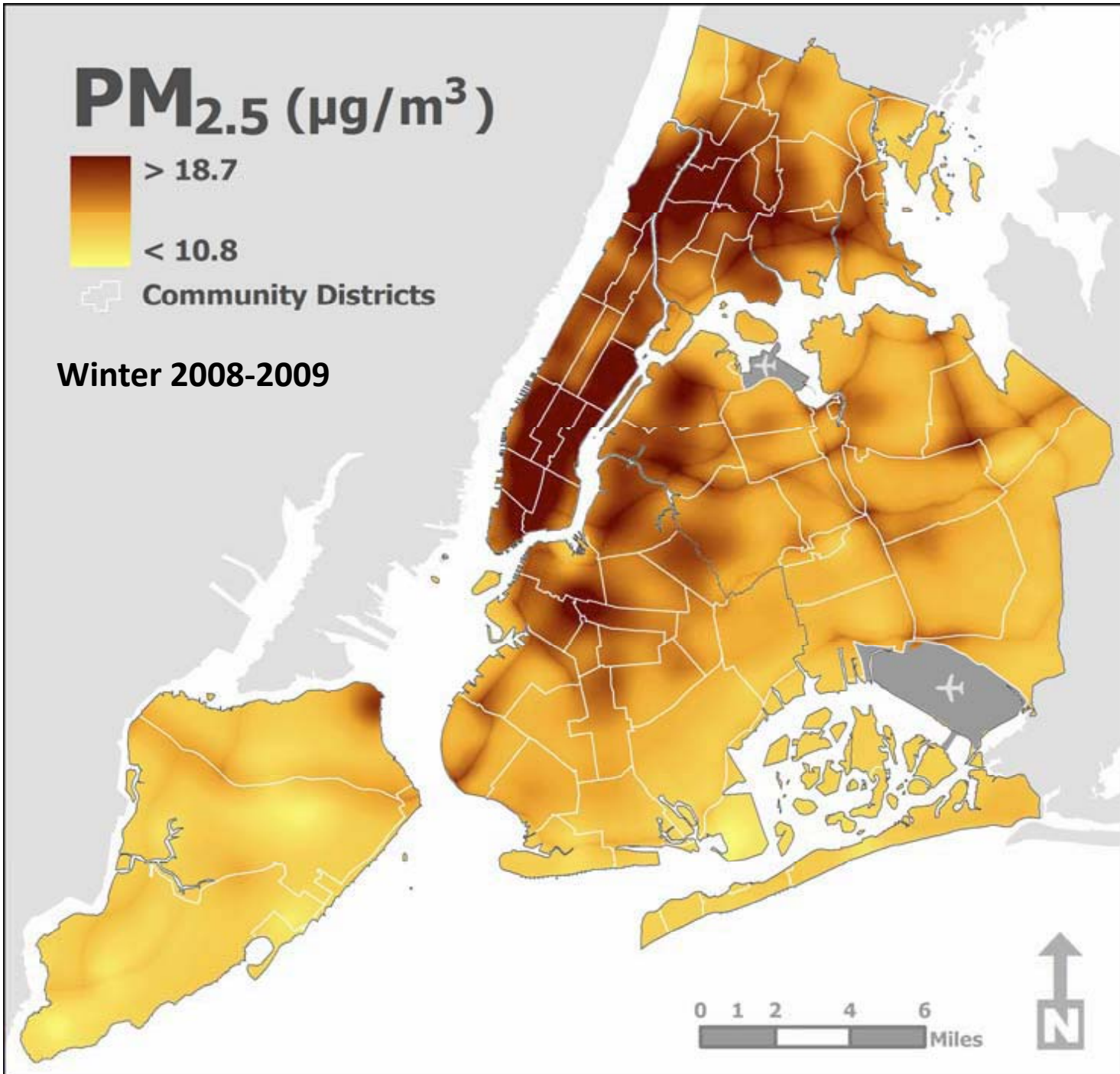


PM_{2.5} (µg/m³)



 Community Districts

Winter 2008-2009

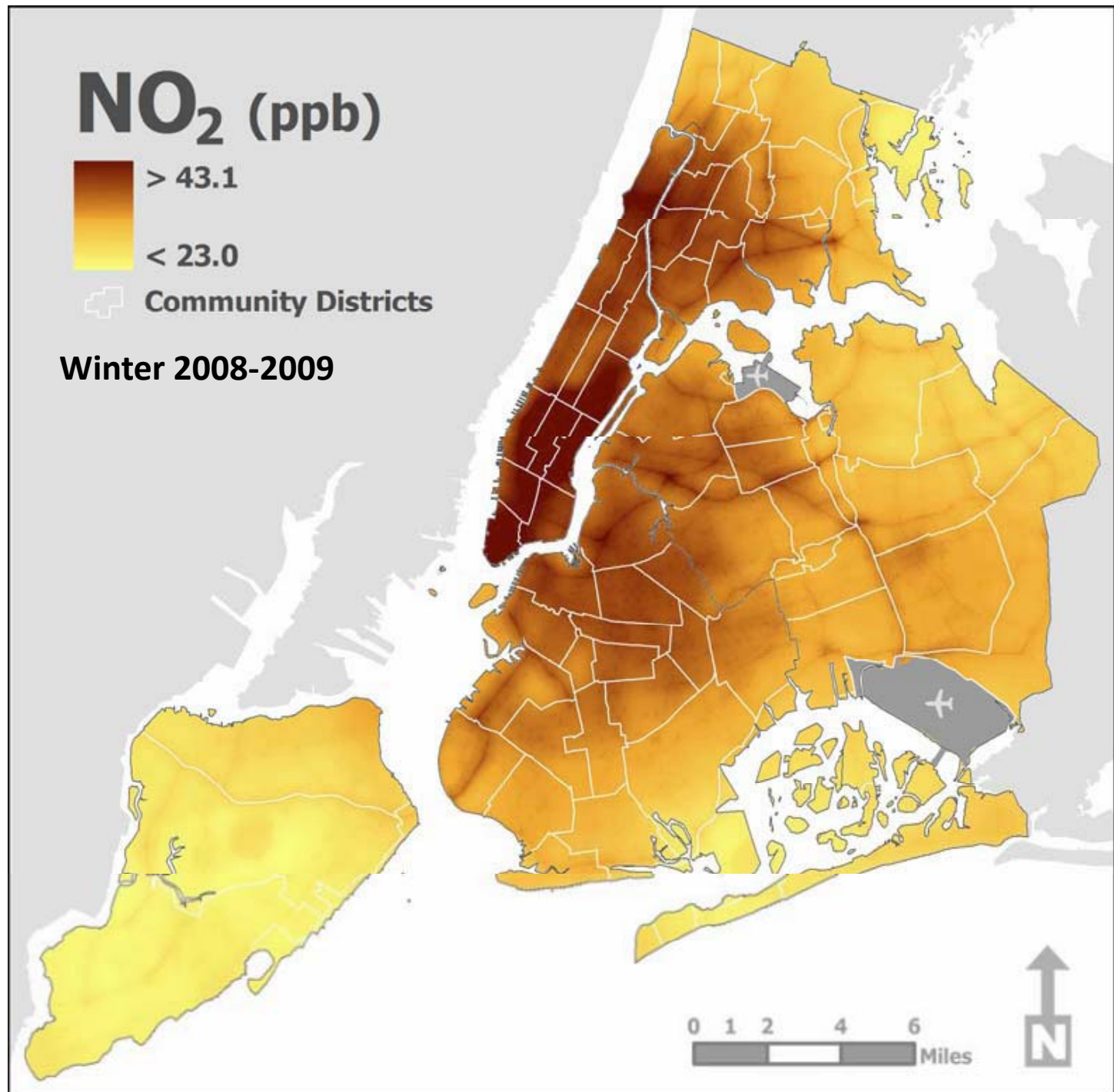


NO₂ (ppb)

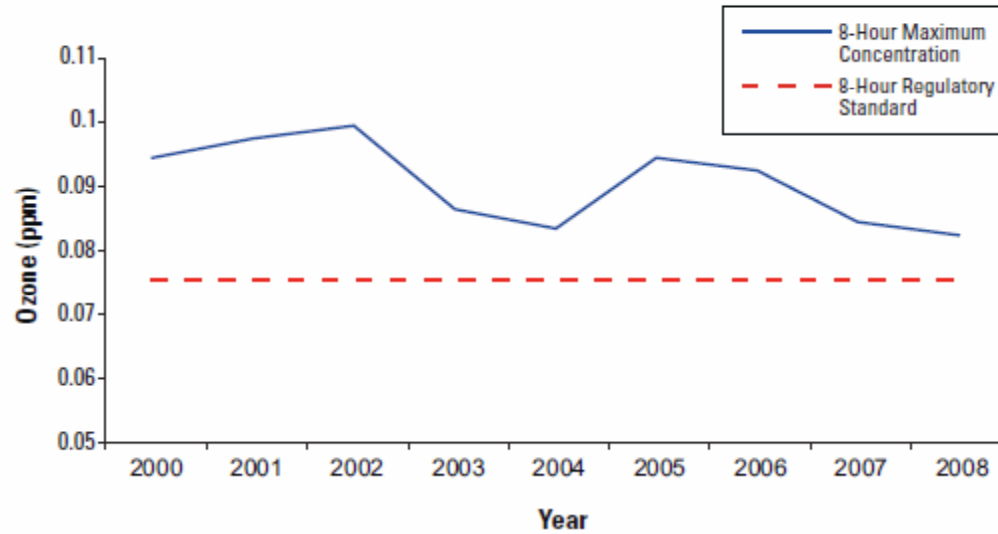


Community Districts

Winter 2008-2009

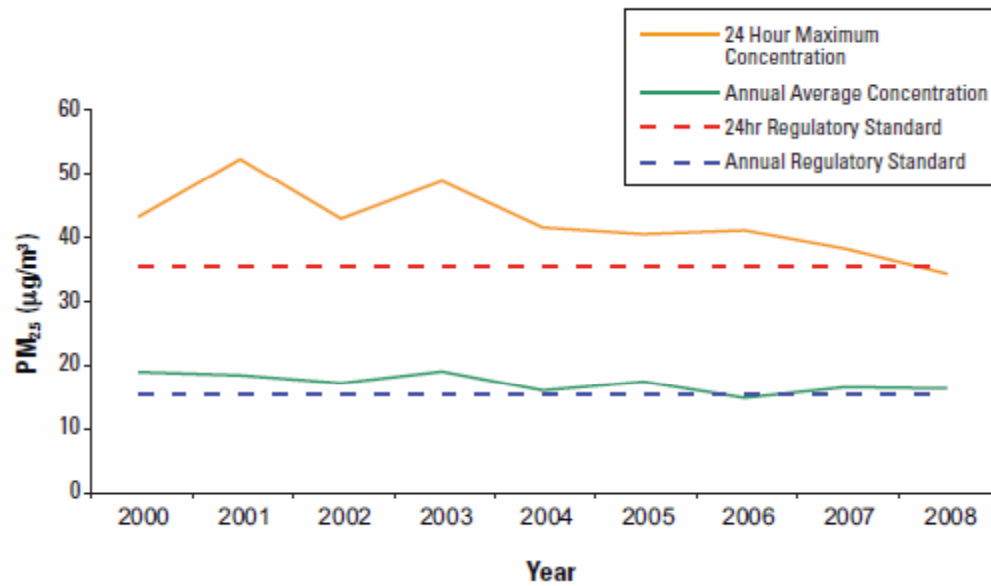


Ozone levels, New York City, 2000-2008



New York City Community Air Survey, 2009

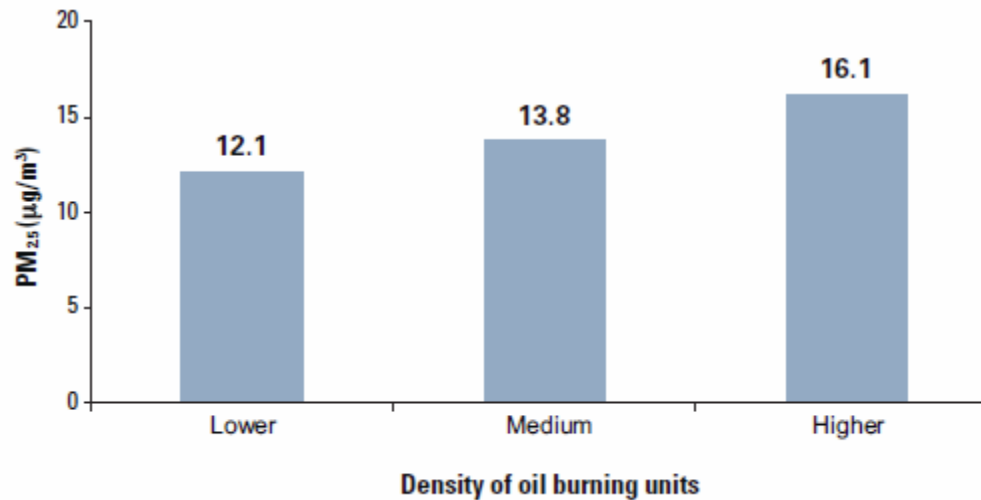
PM_{2.5} levels, New York City



New York City Community Air Survey, 2009

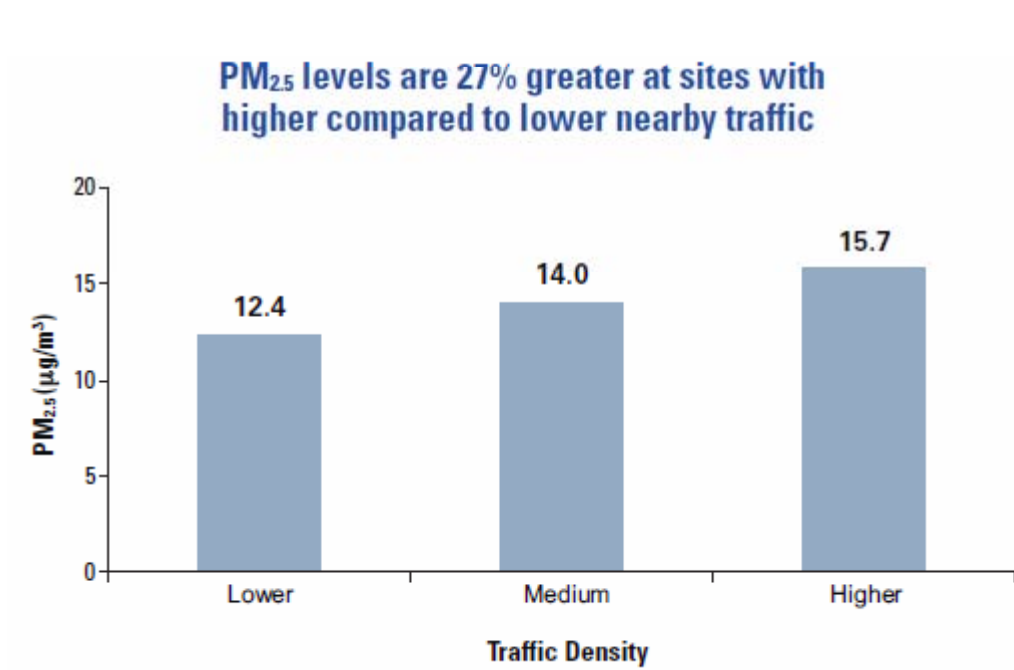
Geographic variation in PM_{2.5}, New York City

PM_{2.5} is 33% greater at sites in regions of higher compared to lower density of oil burning units



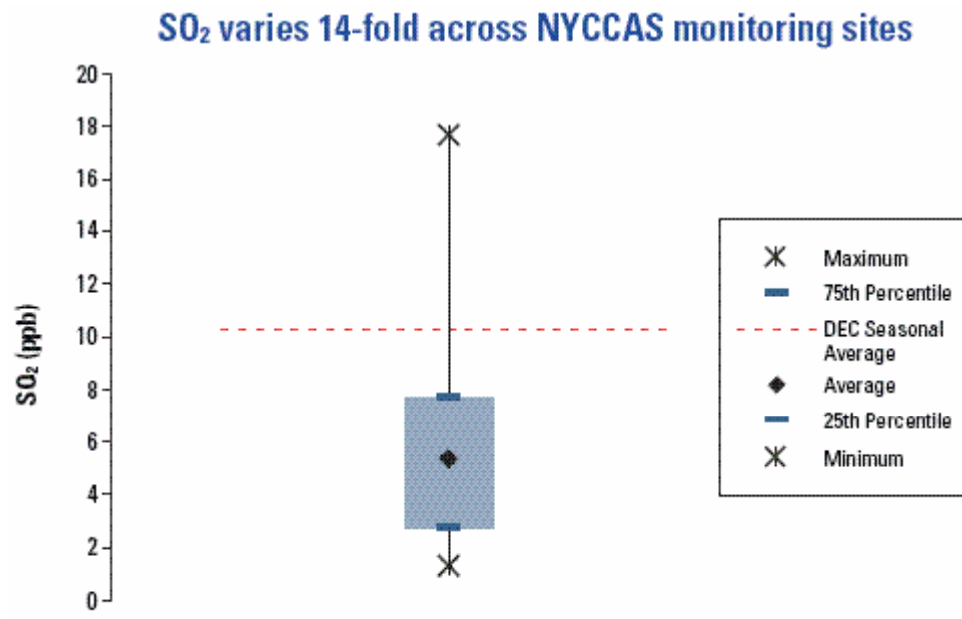
New York City Community Air Survey, 2009

Geographic variation in PM_{2.5}, New York City



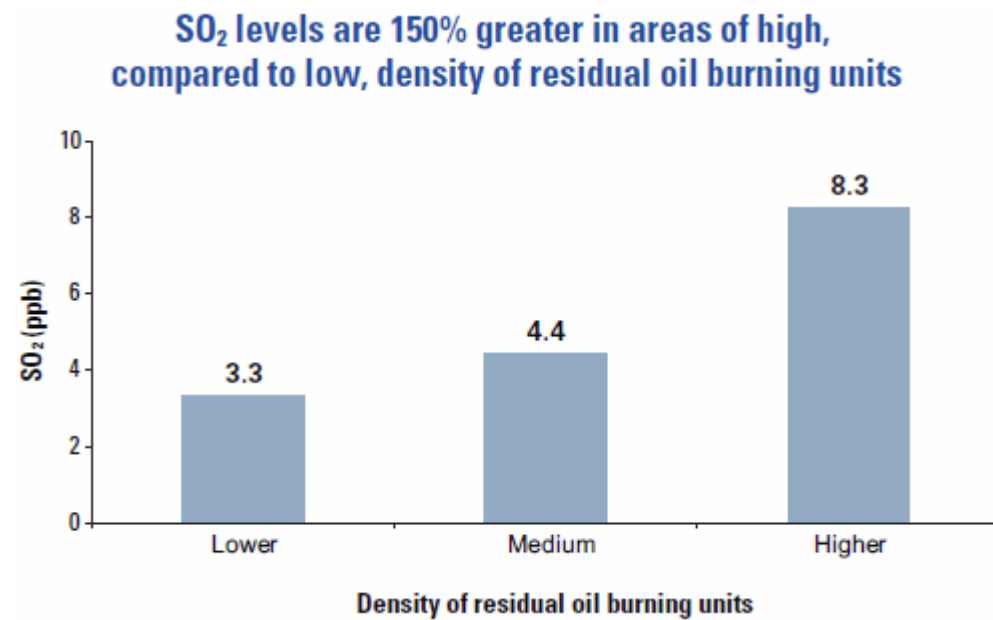
New York City Community Air Survey, 2009

SO₂ concentrations, New York City



New York City Community Air Survey, 2009

SO₂ levels vary with density of oil burning units



New York City Community Air Survey, 2009

Indoor air pollution

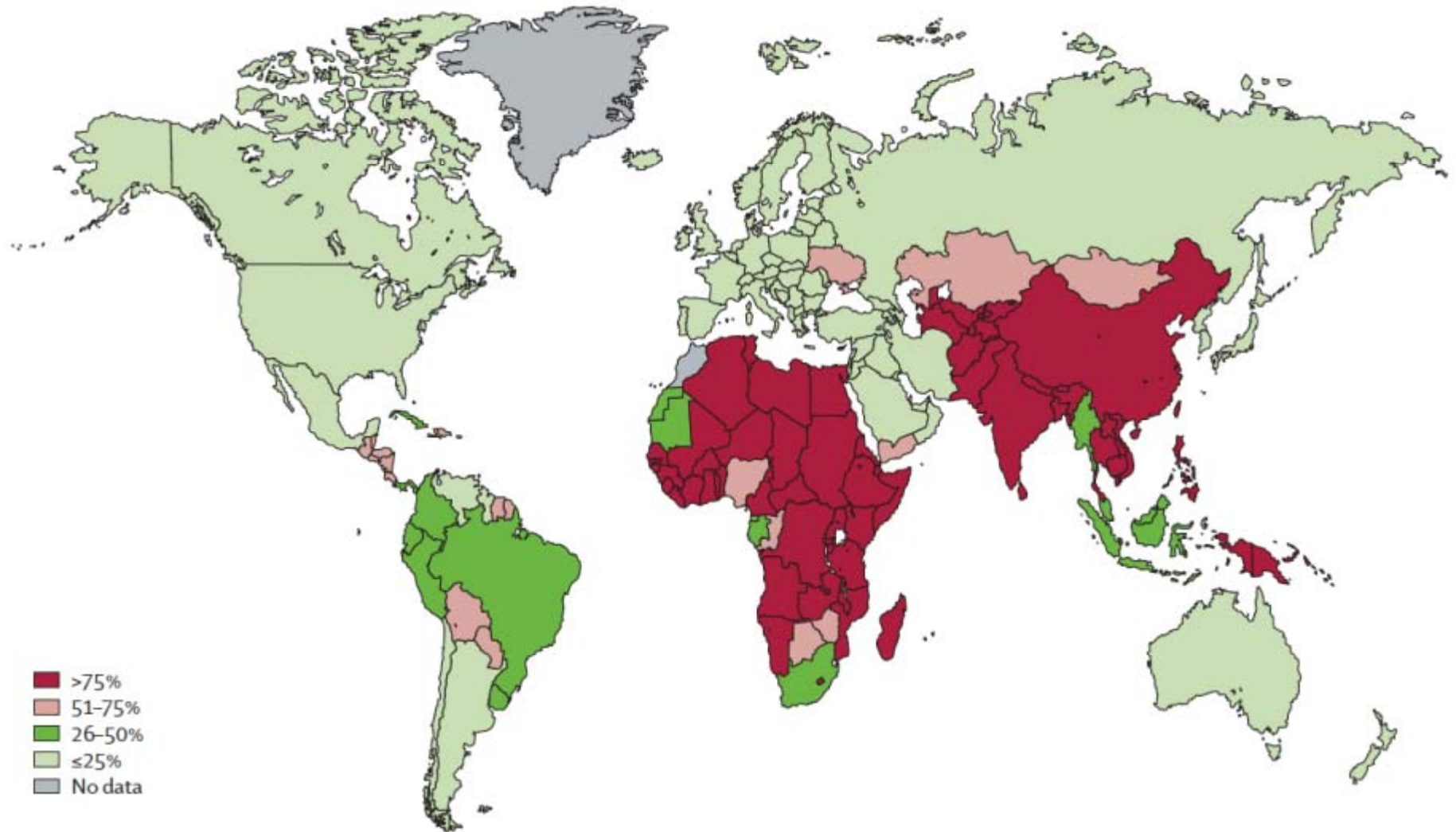
- The two most important sources of indoor air pollution are biomass fuel burning and environmental tobacco smoke



Biomass fuel burning



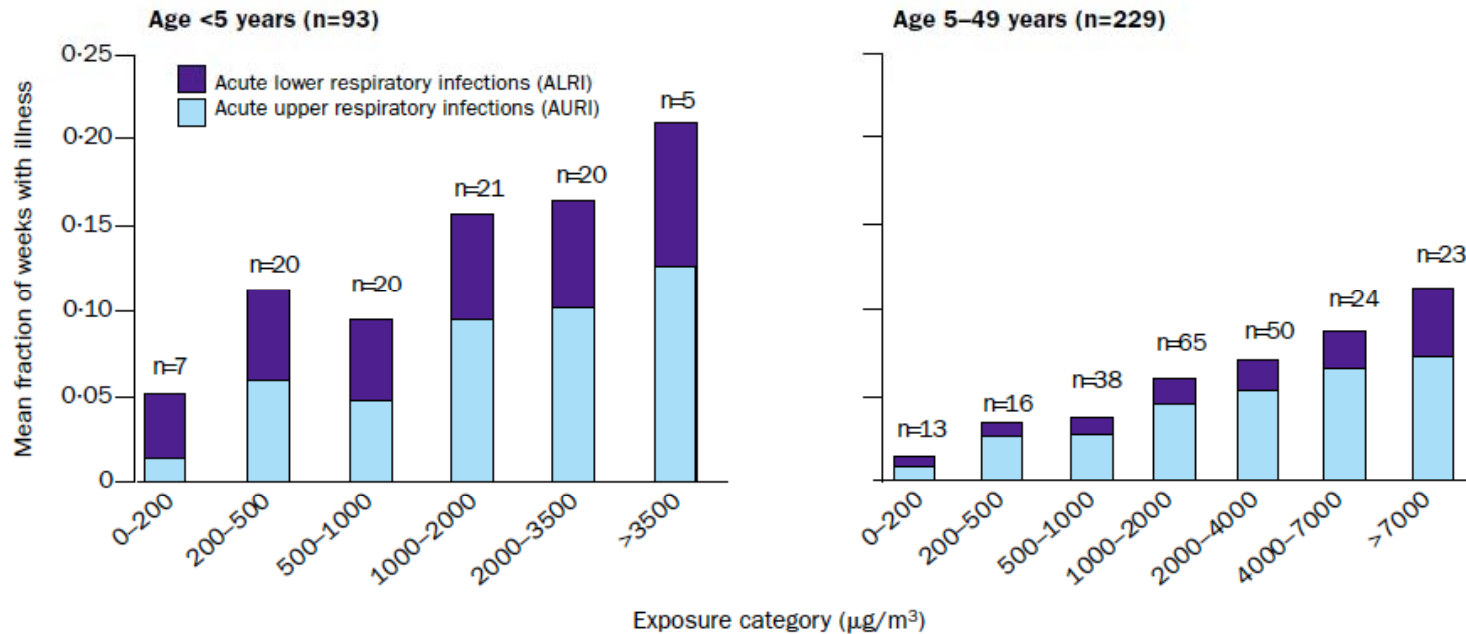
Use of solid fuel (biomass) for cooking



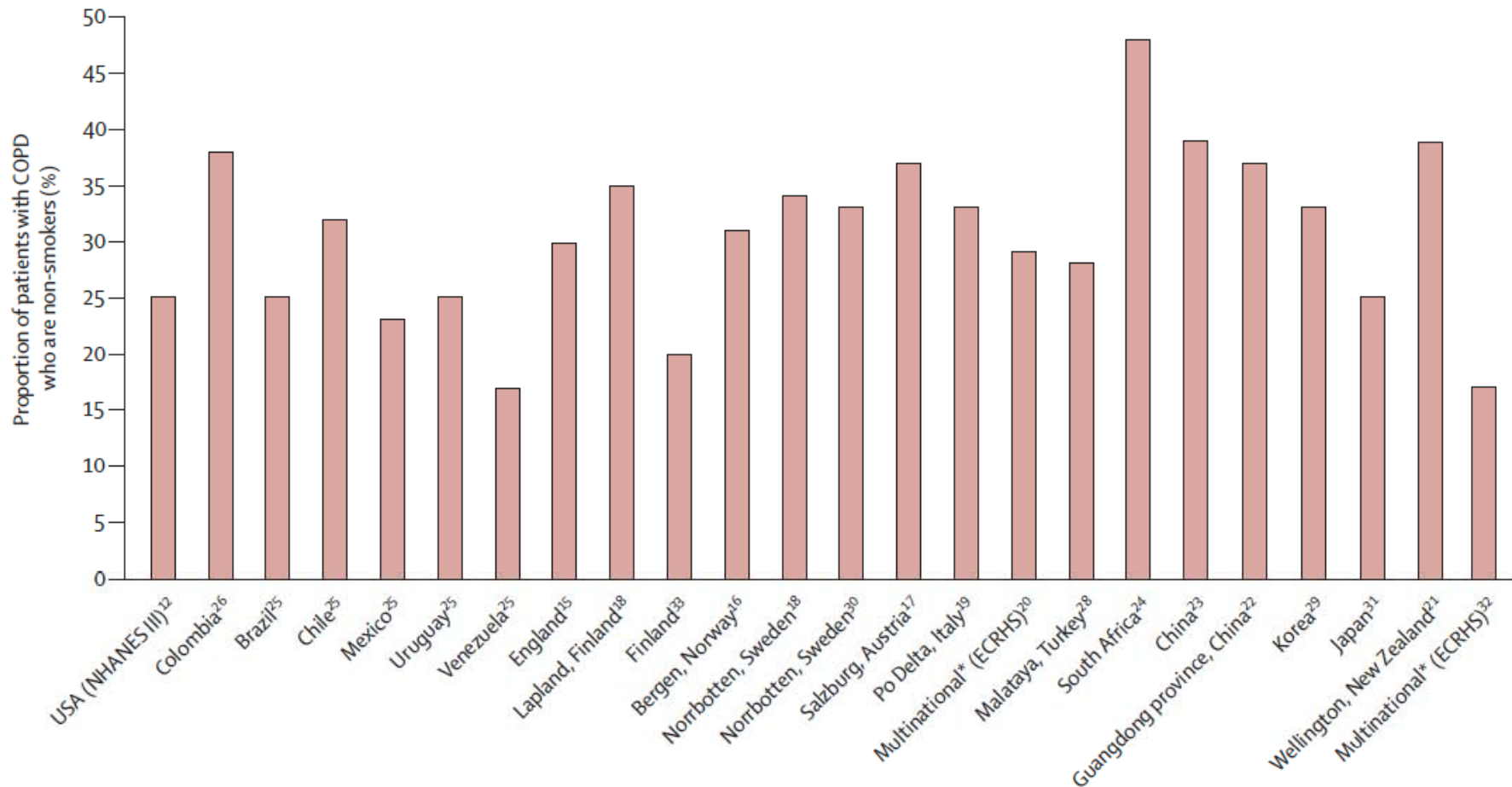
Source: WHO

Relationship between biomass fuel exposure and acute respiratory infections

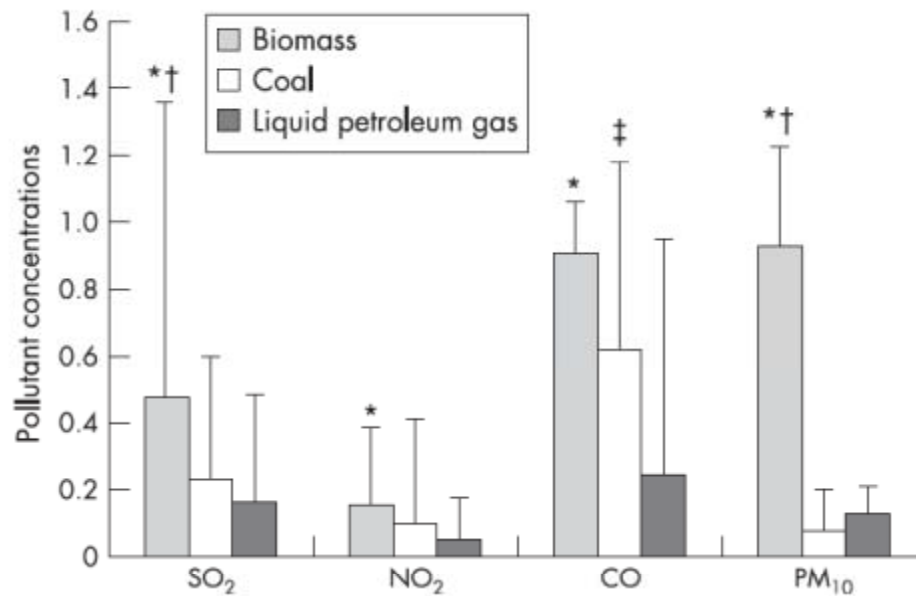
Cohort of adults and children in central Kenya followed for 2 years:



Proportion of patients with chronic obstructive pulmonary disease (COPD) who are non-smokers worldwide



Biomass exposure and COPD risk in China



Factor	COPD prevalence	OR (95% CI)
Area		
Rural	7.2%	3.60 (1.13-11.52)
Urban	2.5%	1.00 (reference)
ETS exposure		
Yes	5.6%	1.10 (0.66-1.83)
No	2.9%	1.00 (reference)

Variations in personal PM_{2.5} in a rural cook in China

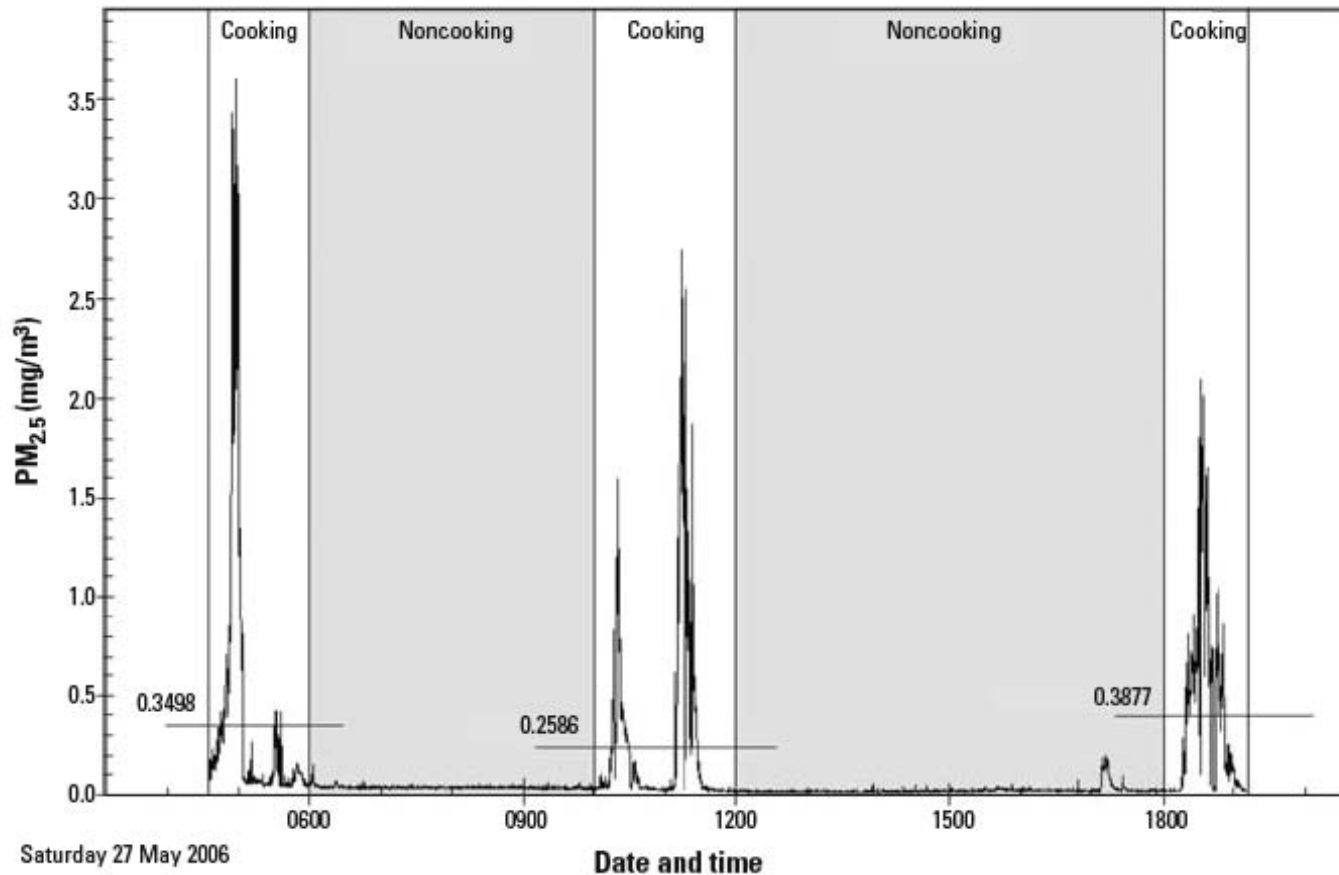


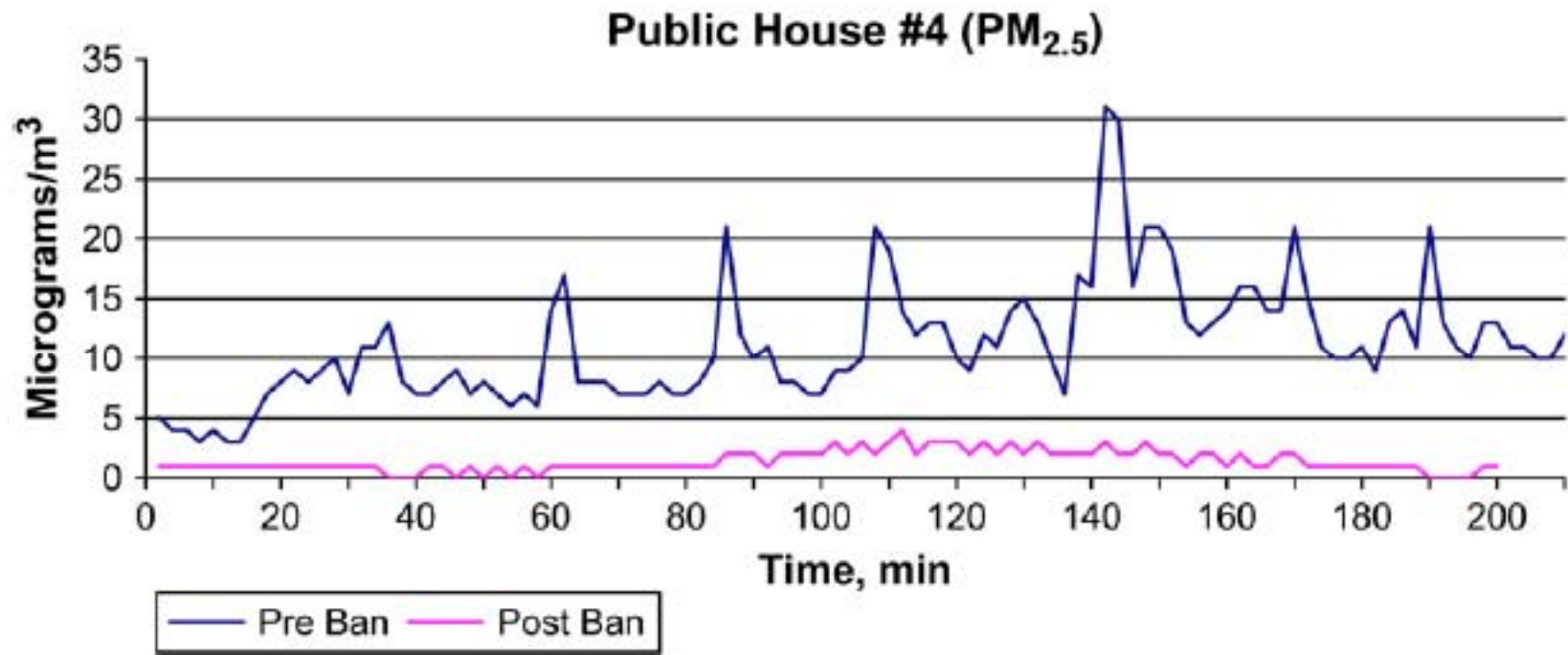
Figure 5. Variations of personal PM_{2.5} in a rural cook, based on second-by-second concentrations. Horizontal lines reflect the average for each cooking period.

Effect of environmental tobacco smoke exposure on lung cancer risk

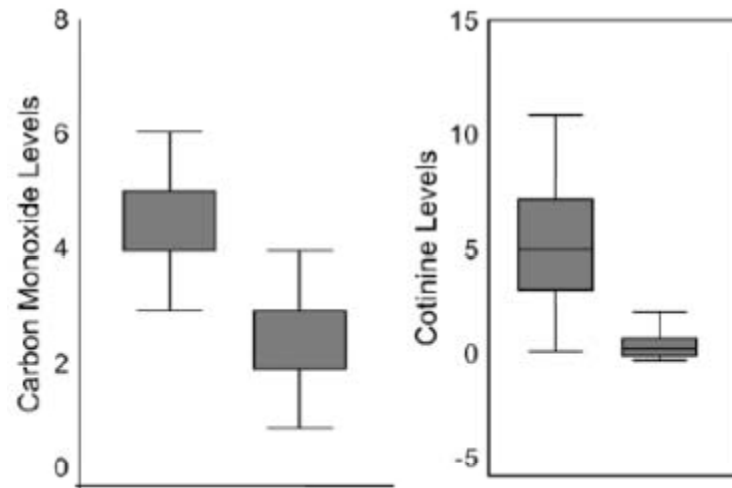
		RR of lung cancer	
Nonsmokers (25 studies)	Workplace SHS vs. not	1.22	1.13-1.33
Nonsmoking Men (11 studies)	Workplace SHS vs. not	1.12	0.86-1.50
Nonsmoking Women (25 studies)	Workplace SHS vs. not	1.22	1.10-1.35
Nonsmokers USA & Canada (8 studies)	Workplace SHS vs. not	1.24	1.03-1.49
Nonsmokers Europe (7 studies)	Workplace SHS vs. not	1.13	0.96-1.34
Nonsmokers Asia (10 studies)	Workplace SHS vs. not	1.32	1.13-1.55

International Agency for Research on Cancer/WHO, "Evaluating the Effectiveness of Smoke-free Policies" 2009

Cigarette smoking and particulate matter concentration



Cigarette smoke and concentration of pollutants



	Preban (<i>SD</i>)	Postban (<i>SD</i>)	Change (%)	pValue
Public houses (n = 42)				
Ave PM _{2.5}	35.5 (17.8)	5.8 (2.2)	-83.6	< 0.01
Ave PM ₁₀	72.1 (27.8)	45.5 (17.1)	-36.9	NS
Benzene (n = 26)	18.8 (14.0)	3.7 (1.6)	-80.2	< 0.01
Outdoor (n = 42)				
Ave PM _{2.5}	6.0 (0.8)	5.2 (0.1)	-13.6	NS
Ave PM ₁₀	24.1 (19.3)	20.0 (5.0)	-17.4	NS
Benzene*		3.7		

Effect of workplace smoking ban on lung function in never-smokers in Ireland

Lung function was measured just before and one year after institution of ban

Parameter	Pre-ban	Post-ban	p value
FVC % pred	92	94	<0.01
FEV ₁ /FVC	82	80	<0.01
PEF %pred	94	99	<0.01
TLC % pred	90	92	0.02
DL _{CO}	90	96	<0.01

Environmental/occupational causes of lung disease: other examples

Exposure

isocyanates

asbestos

chlorine gas

high altitude

rapeseed oil

uranium

pigeons

homeless shelter

dust

cigarette smoke

Disease

occupational asthma

pulmonary fibrosis

ARDS

pulmonary edema

pulmonary hypertension

bronchogenic carcinoma

hypersensitivity pneumonitis

tuberculosis

RADS/?COPD

COPD, lung cancer

Lung function after exposure to WTC dust

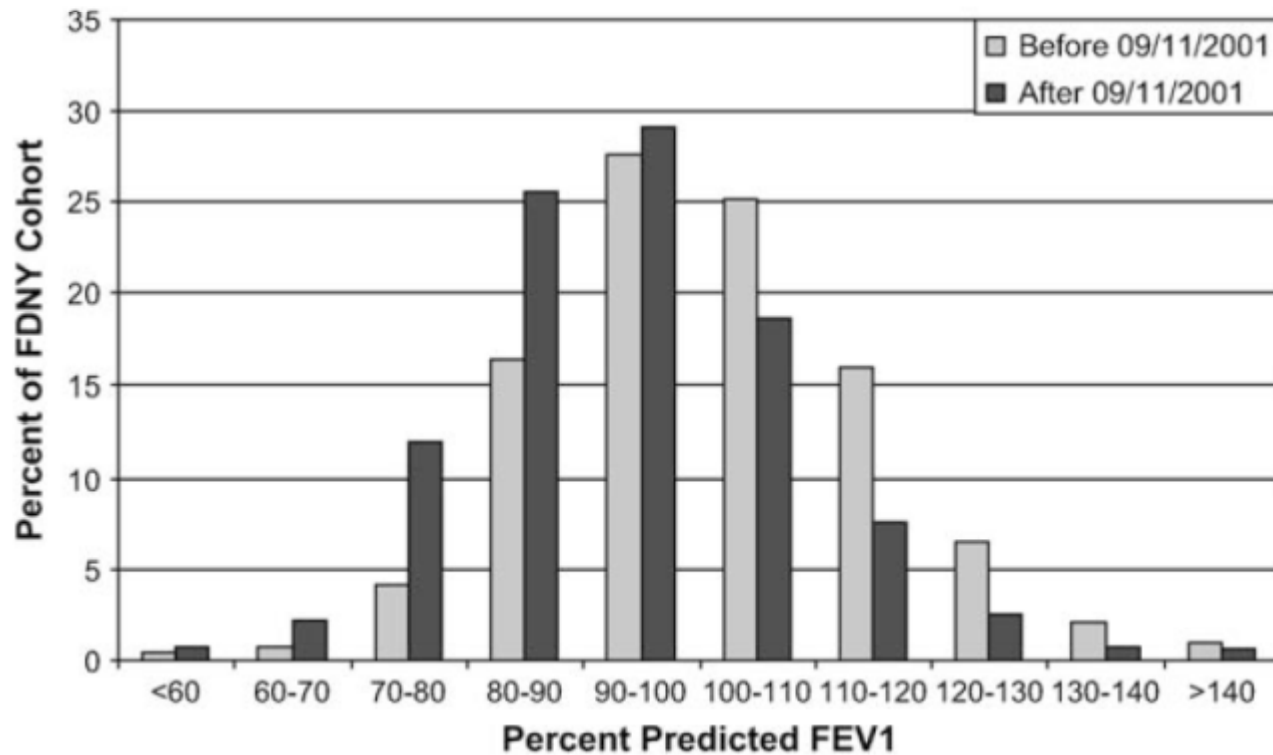
TABLE 1. DEMOGRAPHIC CHARACTERISTICS OF FDNY COHORT BY ARRIVAL TIME-BASED WORLD TRADE CENTER EXPOSURE

Characteristic	Early Exposure	Intermediate Exposure	Late Exposure	Nonexposed	Total
Demographics					
Number (% of FDNY cohort)	1,660 (13.7)	8,185 (67.8)	1,921 (15.9)	313 (2.6)	12,079 (100)
Age on 09/11/01, yr	40 ± 7.6	39.7 ± 7.5	40.2 ± 8.3*	40.7 ± 9 [†]	39.7 ± 7.7
Height, cm	179.3 ± 7.6	179.6 ± 7.4	178.3 ± 8.4*	174 ± 9.1 [‡]	178.3 ± 7.6
Sex, % male	96.7	97.1	91.9*	71.2 [‡]	95.6
Race, % white	86.2	88.3	78.2*	55.3 [‡]	85.6
Ever smokers, %	28.9	27.5	33.4*	39.9 [‡]	29
Work assignment on 09/11/01, % EMS	18.1	13.8	33*	88.8 [‡]	19.4
FDNY tenure on 09/11/01, yr	11.2 ± 7.9	11.1 ± 7.9	10.4 ± 8.3*	6.4 ± 5.3 [‡]	10.9 ± 8.3

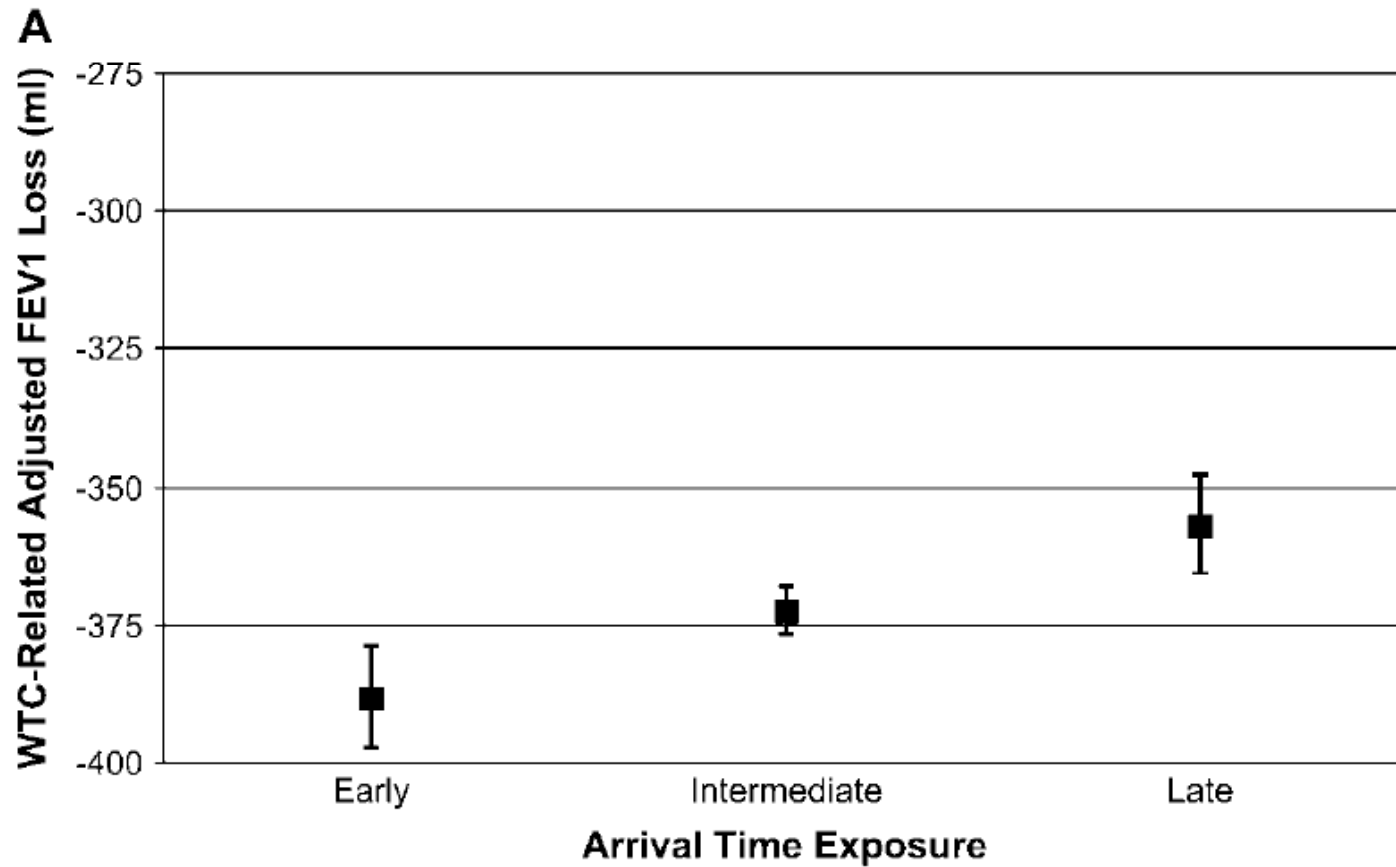
TABLE 2. FEV₁ CHARACTERISTICS OF WORLD TRADE CENTER-EXPOSED FDNY RESCUE WORKERS BY ARRIVAL TIME-BASED WORLD TRADE CENTER EXPOSURE

Arrival Time-based WTC Exposure	Last FEV ₁ before 09/11/2001 (Median, Interquartile Range, and Percent)			First FEV ₁ after 09/11/2001 (Median, Interquartile Range, and Percent)		
	Liters	Percent Predicted	Percent Below Lower Limit of Normal	Liters	Percent Predicted	Percent Below Lower Limit of Normal
Early exposure (n = 1,660)	4.21 (3.64–4.73)	101 (92–111)	7.7	3.85 [†] (3.34–4.36)	93 [†] (84–102)	19.2 [†]
Intermediate exposure (n = 8,185)	4.32 (3.83–4.83)	101 (92–111)	6.4	3.96 [†] (3.52–4.42)	93 [†] (85–102)	16.3 [†]
Late exposure (n = 1,921)	4.27 (3.78–4.76)	100 (91–110)	7.8*	3.87 ^{††} (3.42–4.32)	92 ^{††} (83–102)	17.8 ^{††}
Total	4.30 (3.80–4.80)	101 (92–111)	6.8	3.93 (3.47–4.40)	93 (85–102)	15.3

Lung function after exposure to WTC dust



Lung function after exposure to WTC dust



Conclusions

- Environmental and occupational lung disease represent important causes of morbidity and mortality
- Outdoor air pollution and indoor air pollution (due to biomass fuel burning and environmental tobacco smoke) are the most important causes
- Careful occupational history must be sought in all patients with respiratory complaints