

# 실내 미세먼지에 의한 건강피해

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# Contents

## Indoor Fine Particulate Matter

- **What is particulate matter?**
  - Sources of indoor PM
  - Component of indoor PM
- **Health effect of PM**
  - Epidemiological study
  - Possible mechanism
- **Intervention and Strategies**



*Cited from* : Li Z et al., *Sci Total Environ.* 2017;586:610-622.

# Contents

## Indoor Fine Particulate Matter

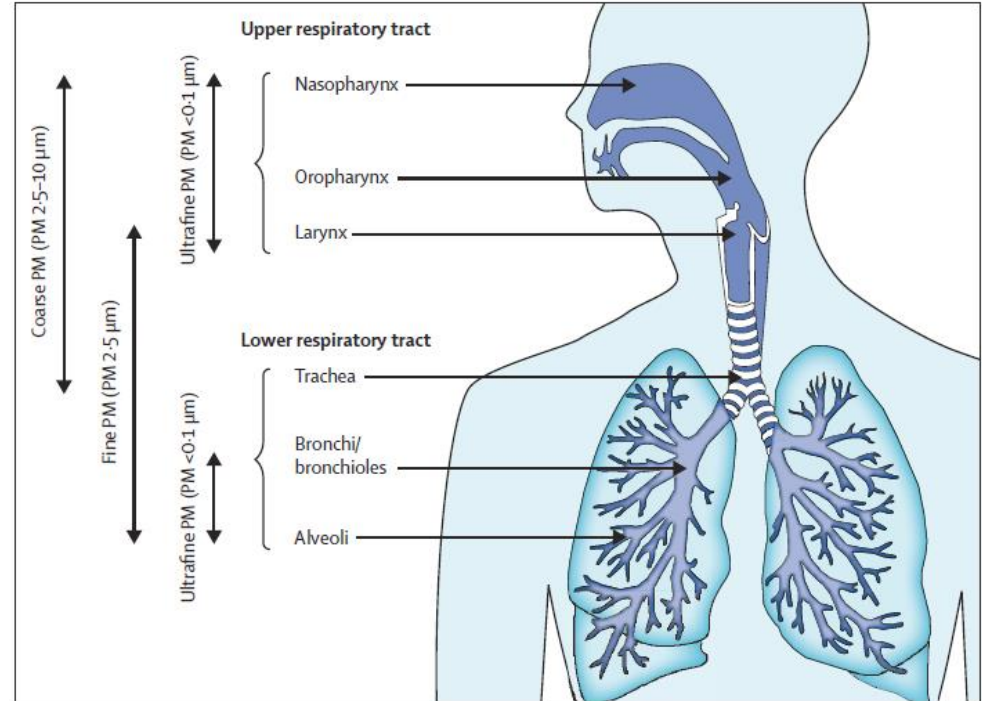
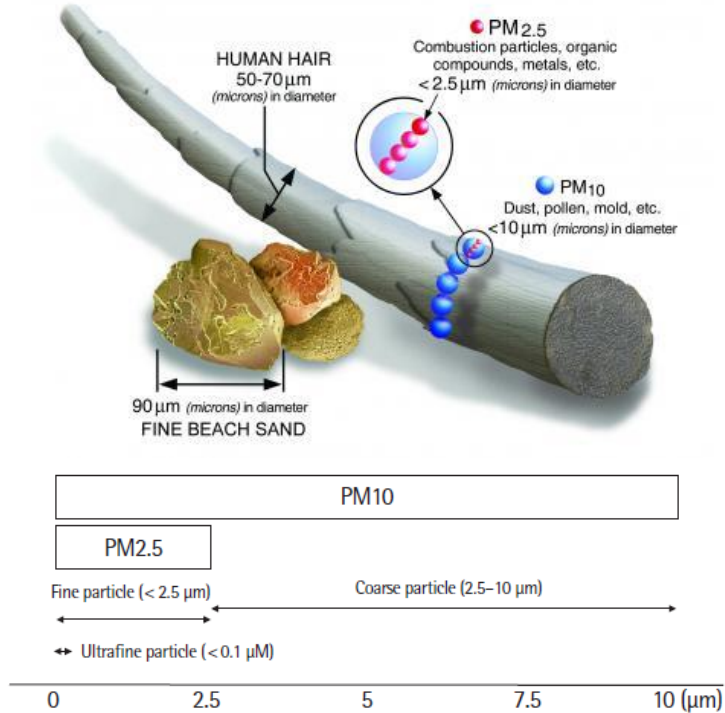
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# PM

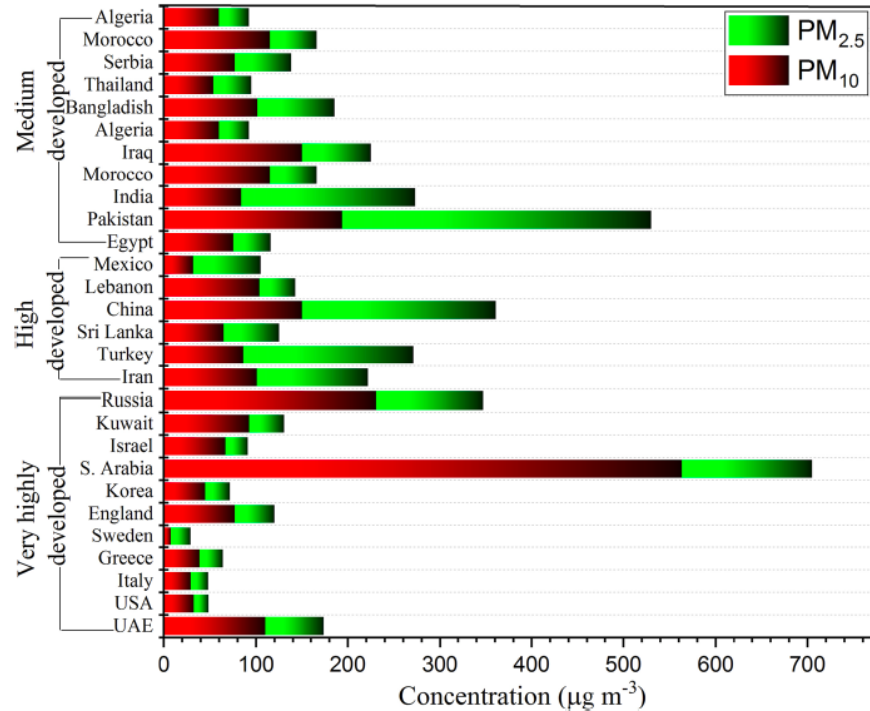
## Compartmental deposition



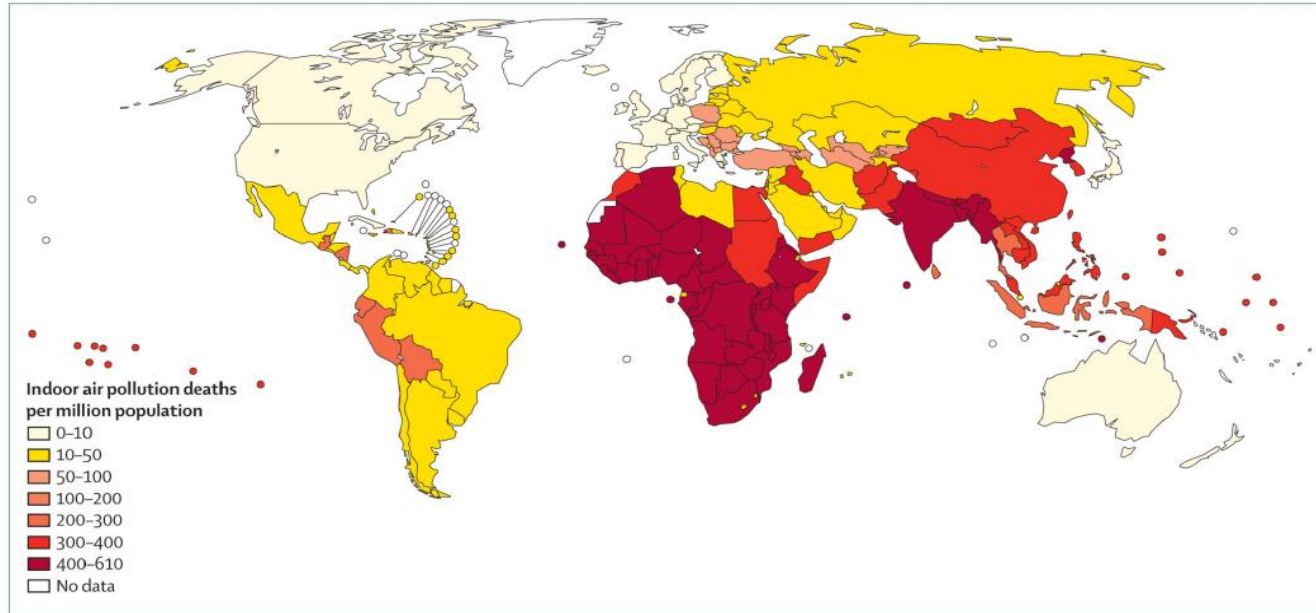
*Cited from* : the US Environmental Protection Agency

# PM10 & 2.5

Ubiquitous atmospheric aerosol with both anthropogenic and natural sources



## The global burden of disease attributed to household air pollution(HAP)



**Figure 2. WHO map of household air pollution and mortality**

World map of poverty (not shown) shows nearly identical geographical distribution. © WHO 2005. All rights reserved.

**Table 1** Major health-damaging pollutants generated from indoor sources (26)

Pollutant	Major indoor sources
Fine particle	Fuel/tobacco combustion, cleaning operations, cooking
Carbon monoxide	Fuel/tobacco combustion
Polycyclic aromatic hydrocarbons	Fuel/tobacco combustion, cooking
Nitrogen oxides	Fuel combustion
Sulfur oxides	Coal combustion
Arsenic and fluorine	Coal combustion
Volatile and semi-volatile organic compounds	Fuel/tobacco combustion, consumer products, furnishings, construction materials, cooking
Aldehydes	Furnishings, construction materials, cooking
Pesticides	Consumer products, dust from outside
Asbestos	Remodelling/demolition of construction materials
Lead	Remodelling/demolition of painted surfaces
Biological pollutants	Damp materials/furnishings, components of climate control systems, occupants, outdoor air, pets
Radon	Soil under buildings, construction materials
Free radicals and other short-lived, highly reactive compounds	Indoor chemistry

**Cited from:** Jiang XQ et al, *J Thorac Dis* 2016;8(1):E31-E40.

**Table 1**  
**Deaths attributable to environmental risks worldwide**

Deaths in 2010 (95% CI)	
Household air pollution <sup>*</sup>	3.55 million (2.68 million to 3.62 million)
Ambient pollution	3.22 million (2.82 million to 3.62 million)
Occupational risk factors <sup>†</sup>	0.85 million (0.66 million to 1.06 million)
Lead exposure	0.67 million (0.58 million to 0.78 million)
Second-hand smoke	0.60 million (0.45 million to 0.52 million)
Unimproved sanitation	0.24 million (0.01 million to 0.48 million)
Unimproved water source	0.12 million (0.01 million to 0.23 million)
Residential radon	0.10 million (0.01 million to 0.22 million)

<sup>\*</sup> Household air pollution contributes about 16% to the worldwide disease burden of ambient air pollution.<sup>2</sup>

<sup>†</sup> Occupational risks include carcinogens, asthmagens, air pollutants, etc. Adapted from Lim and colleagues.<sup>2</sup>

*Cited from: Lancet Respir Med.* 2014;2(10):823-860.

## Indoor UFP Emission Rates for Combustion and Non-Combustion Sources in Homes

UFP Emitting Device	Size Range (nm)	Emission Rate (#/min)	Reference
Flat iron with steam	20-1,000	$6.0 \times 10^9$	Afshari et al. (2005)
Electric frying pan	10-400	$1.1-2.7 \times 10^{10}$	Buonanno et al. (2009)
3D printer w/PLA	10-100	$\sim 2.0 \times 10^{10}$	Stephens et al. (2013)
Vacuum cleaner	20-1,000	$3.5 \times 10^{10}$	Afshari et al. (2005)
Scented candles	20-1,000	$8.8 \times 10^{10}$	Afshari et al. (2005)
Gas stove	20-1,000	$1.3 \times 10^{11}$	Afshari et al. (2005)
3D printer w/ABS	10-100	$\sim 1.9 \times 10^{11}$	Stephens et al. (2013)
Cigarette	20-1,000	$3.8 \times 10^{11}$	Afshari et al. (2005)
Electric stove	20-1,000	$6.8 \times 10^{11}$	Afshari et al. (2005)
Frying meat	20-1,000	$8.3 \times 10^{11}$	Afshari et al. (2005)
Radiator	20-1,000	$8.9 \times 10^{11}$	Afshari et al. (2005)
Desktop 3D printers	10-100	$\sim 10^8 - \sim 10^{12}$	Azimi et al. (2016)
Laser printers	6-3,000	$4.3 \times 10^9 - 3.3 \times 10^{12}$	He et al. (2010)
Cooking on a gas stove	10-400	$1.1-3.4 \times 10^{12}$	Buonanno et al. (2009)

NOTES: Highlighted items are combustion-related; all other items are non-combustion sources. PLA and ABS are thermoplastics used as 3D printer feedstock.

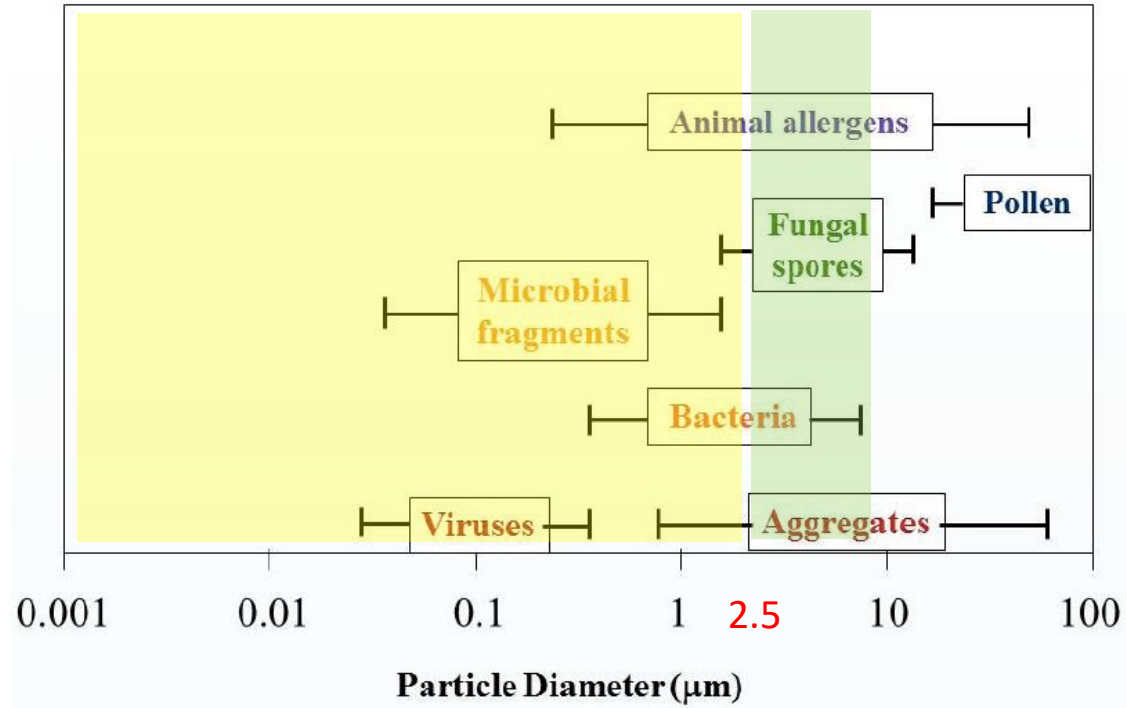
## Drivers of Exposure Disparities in Indoor Environments

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Sources	Indoor Sources	<ul style="list-style-type: none"> <li>• Cooking appliances</li> <li>• Tobacco smoke</li> <li>• Cleaning products</li> </ul>	<ul style="list-style-type: none"> <li>• Air fresheners</li> <li>• Personal care products</li> <li>• Furnishings</li> </ul>	<ul style="list-style-type: none"> <li>• Pesticides</li> <li>• Pollutant reservoirs</li> <li>• Water sources</li> </ul>
Settings	Outdoor Sources	<ul style="list-style-type: none"> <li>• Traffic</li> <li>• Industrial activity</li> </ul>	<ul style="list-style-type: none"> <li>• Residential activity</li> <li>• Contaminated soil</li> </ul>	
Structure	Physical Structure	<ul style="list-style-type: none"> <li>• Age of structure</li> <li>• Size of living space</li> <li>• Heating systems</li> </ul>	<ul style="list-style-type: none"> <li>• Mechanical ventilation</li> <li>• Size/design of structure</li> <li>• Single family versus multifamily</li> <li>• Leakage and/or air exchange</li> </ul>	
Behavior	Source Use Patterns	<ul style="list-style-type: none"> <li>• Cooking appliance use</li> <li>• Cooking practices</li> </ul>	<ul style="list-style-type: none"> <li>• Smoking behavior</li> <li>• Consumer product usage</li> <li>• Personal care product usage</li> </ul>	
	Activity Patterns	<ul style="list-style-type: none"> <li>• Time spent at home</li> </ul>	<ul style="list-style-type: none"> <li>• Interaction with sources</li> <li>• Influences on air exchange</li> </ul>	

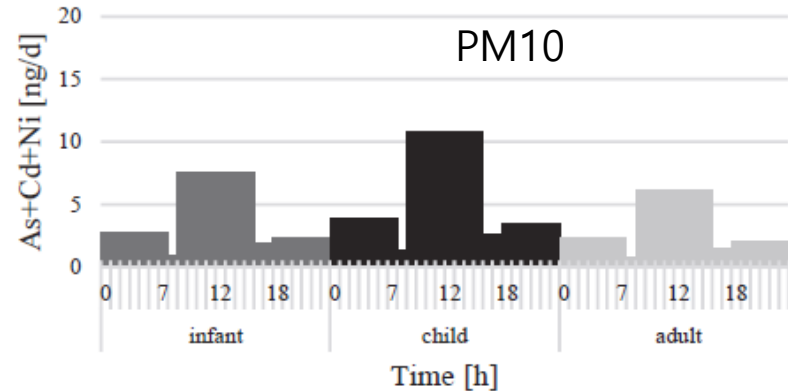
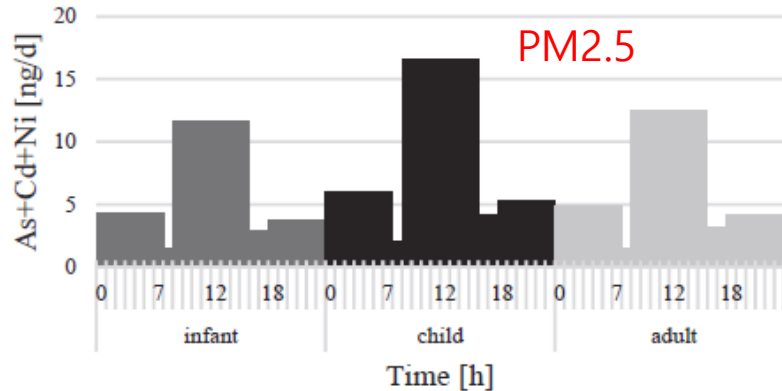
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## Size ranges for different types of bioaerosol particles



# PM10 & 2.5

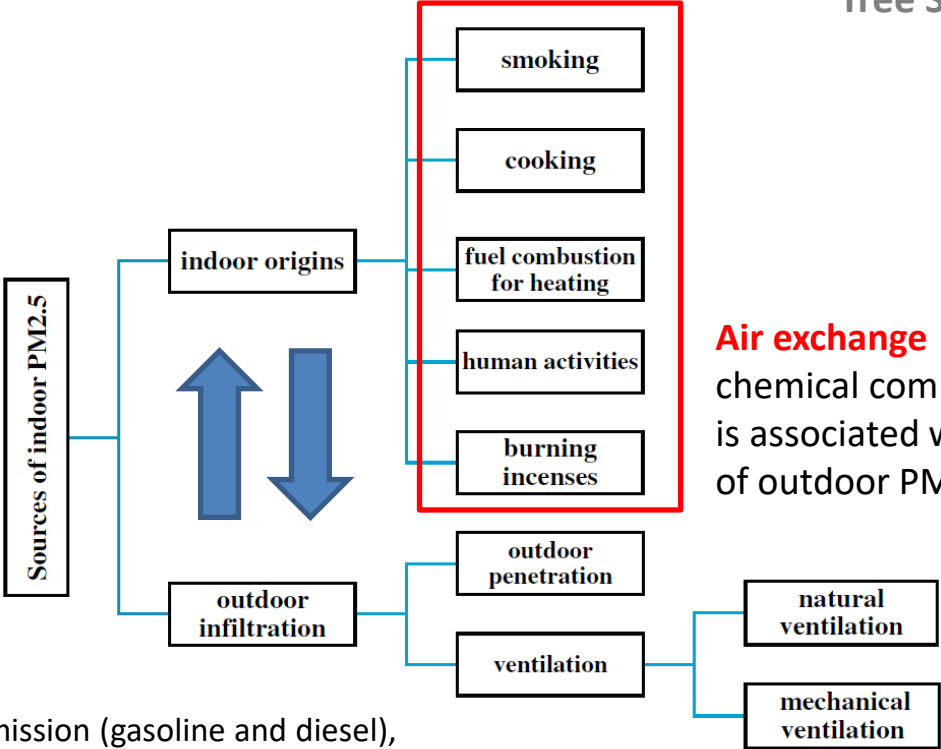
- More easily bound
  - Owned larger specific surface area and bigger adsorption ability
  - Toxic heavy metals, acid oxides, organic pollutants and pathogenic microorganisms



*Cited from:* Widziewicz K et al, *Atmospheric Environ.* 2016;128:198-207

# Sources

## Tree Structure Diagram



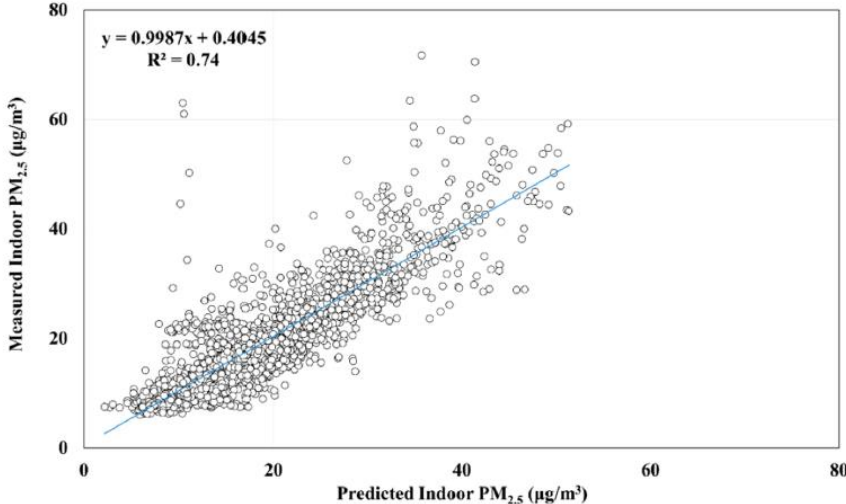
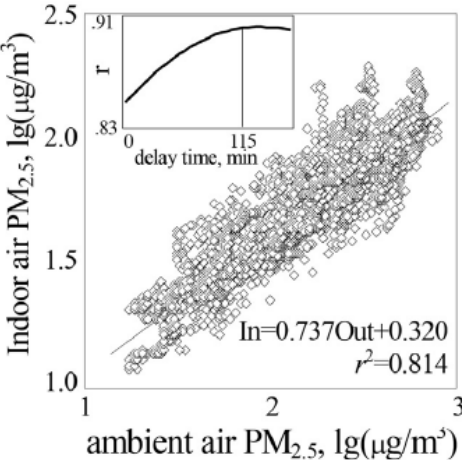
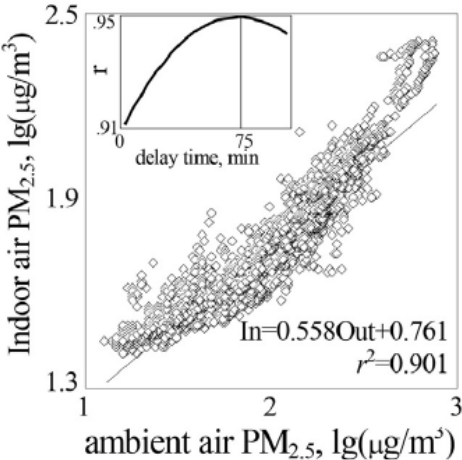
### Air exchange

chemical component of indoor PM2.5 is associated with sources and component of outdoor PM2.5.

crustal dust, vehicle emission (gasoline and diesel), steel plants (coal combustion and some industry activities)

*Cited from* : Li Z et al., *Sci Total Environ.* 2017;586:610-622.

# Sources



**Cited from:** Han Y et al. *Environ Pollution*. 2015:205;307-314

**Cited from:** Jung CC et al. *Int J Environ Res and Public Health*. 2020:17;5906

# Sources

## Smoking

- Smoking had a great influence on adjacent facilities
  - PM<sub>2.5</sub> level in smoking facilities (zone) was significantly higher than it in nonsmoking facilities (zones) and outdoor environment

**Table 3** SHS PM and airborne nicotine levels ( $\mu\text{g}/\text{m}^3$ ) during peak patronage time in restaurants and bars, I

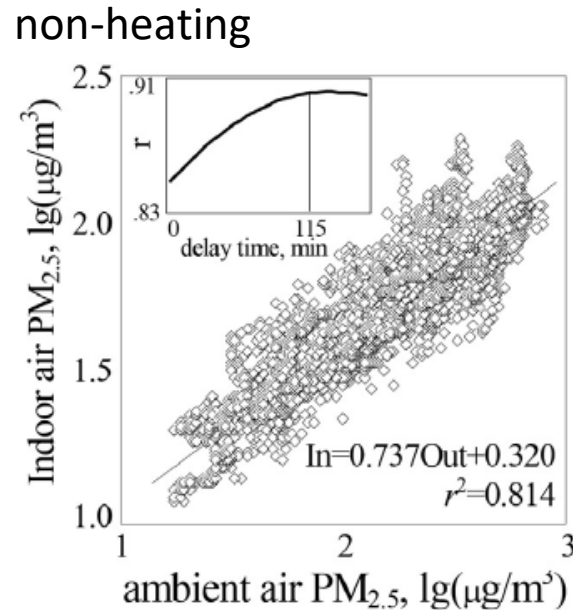
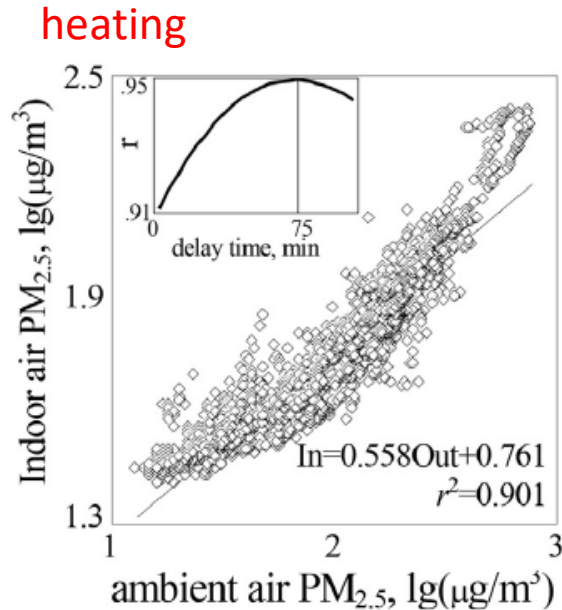
	SHS PM				
	N	Mean	s.d.	Median	IQR
<u>Nominal smoking policy</u>					
Smoking venues	31	78	76	40	8–152
Smoking sections	15	90	90	43	7–198
Non-smoking sections	20	22	24	15	1–36
Non-smoking venues	48	79	171	27	0–72
<u>Smoking observed or not</u>					
No	43	47	155	4	0–28
Yes	71	84	99	44	22–134
Type of establishment					
Restaurants	94	65	122	27	4–80
Bars/cafés	20	97	132	60	4–127
Peak time period					
Lunch	43	57	86	26	4–70
Dinner	71	78	142	31	2–101
Total	114	70	124	27	4–93

Beijing, China

# Sources

## Season and construction type

- Heating vs. non-heating season

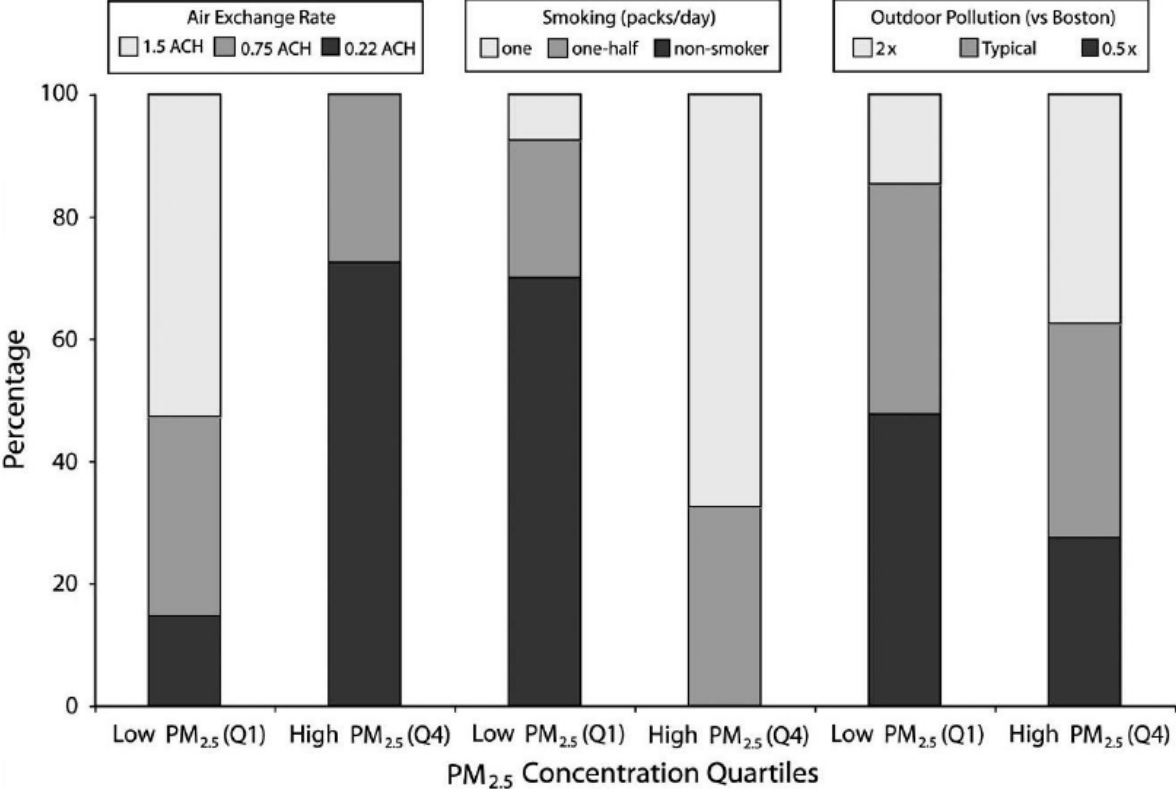


**Cited from:** Zhu CS et al, *Aerosol and Air Qual Res.* 2012;12;1157-1165

**Cited from:** Han Y et al, *Environ Pollution.* 2015;205;307-314 16

# Sources

## Estimated contribution of indoor PM<sub>2.5</sub>

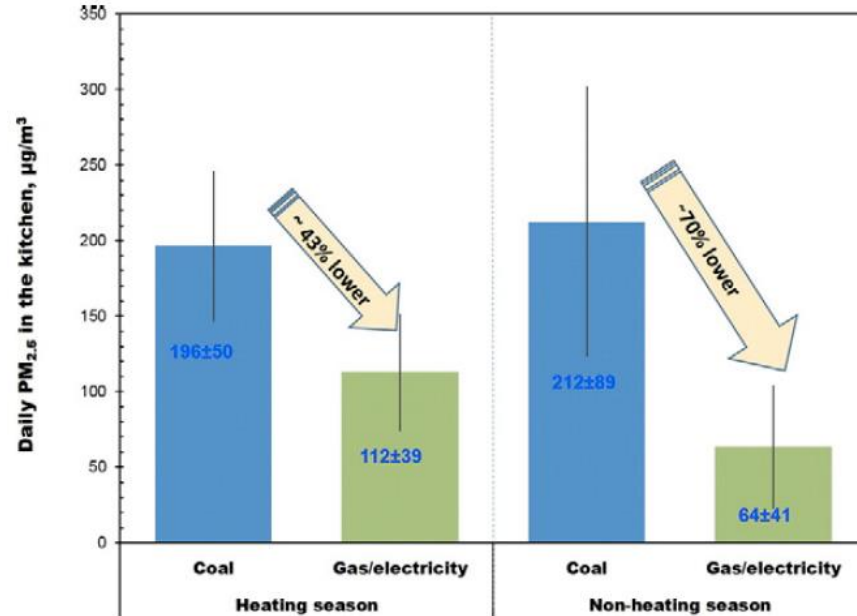


*Cited from:* Adamkiewicz, G et al, *American Journal of Public Health*. 2011;101(Suppl 1):S238–S245.17

# Sources

## Season and construction type

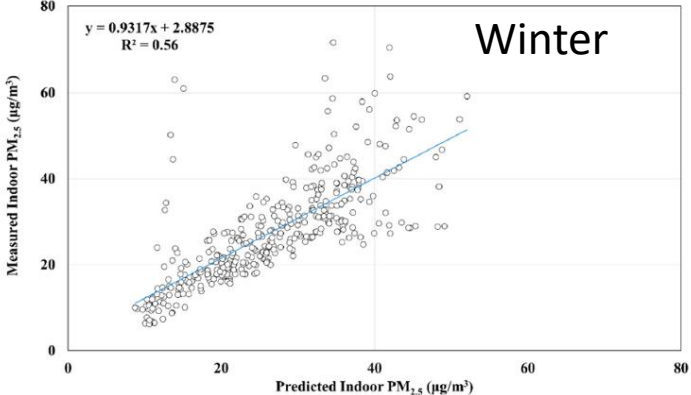
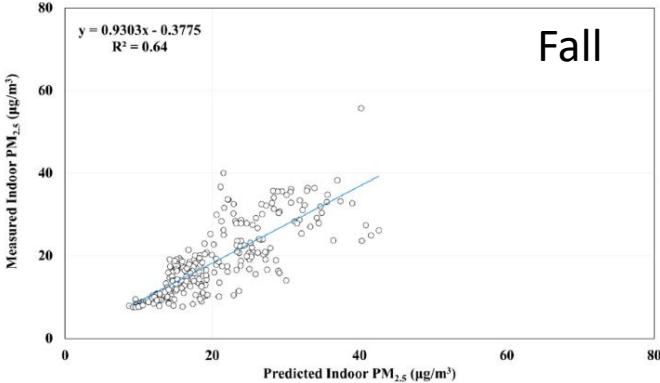
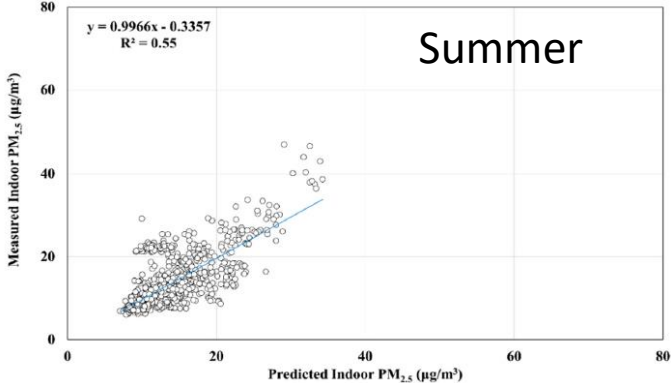
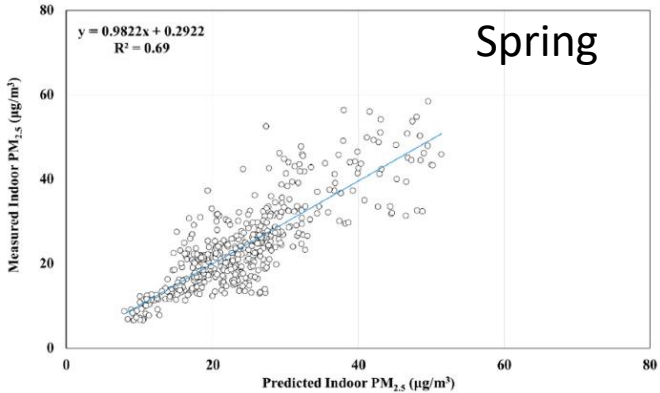
- Clean energy vs. Coal and biomass



*Cited from:* Li T et al, *Sci Total Environ.* 2016:548;6-12

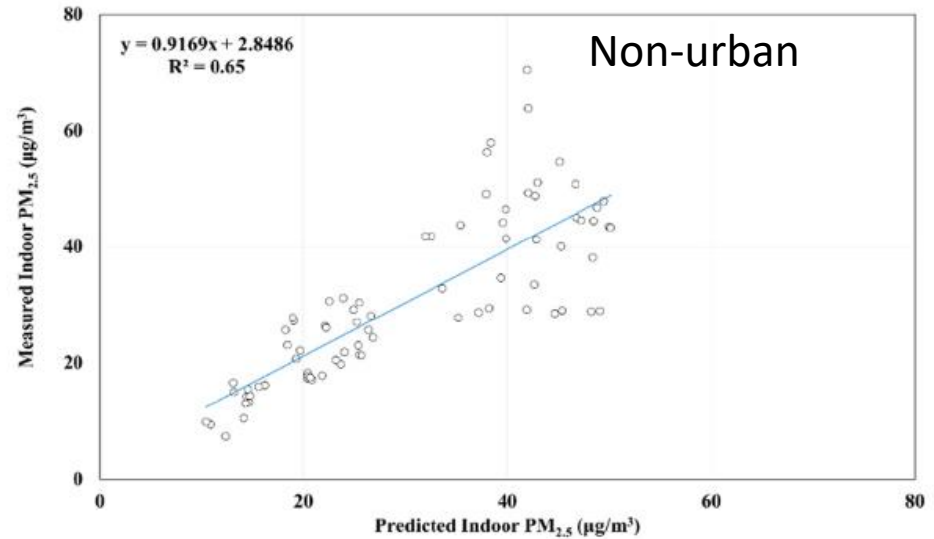
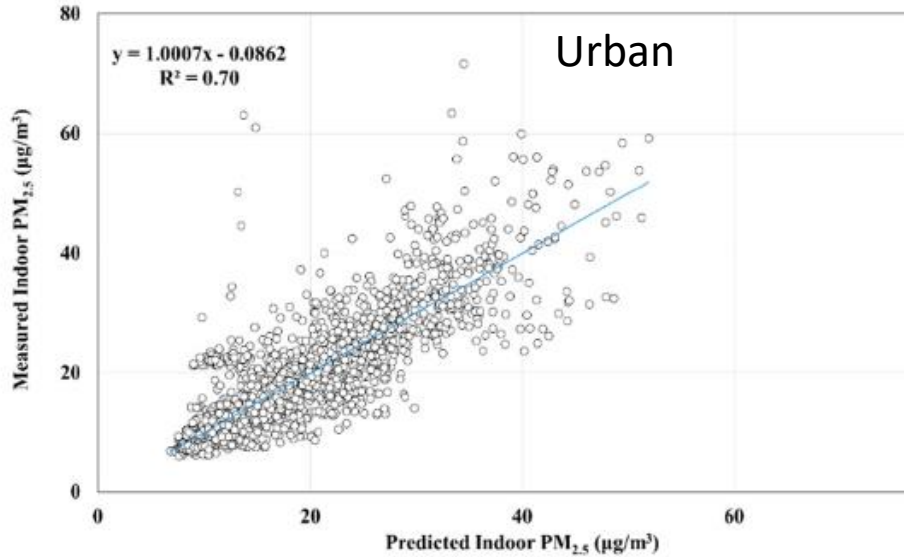
# Sources

## Season and construction type



# Sources

- Urban vs. Rural (non-urban)



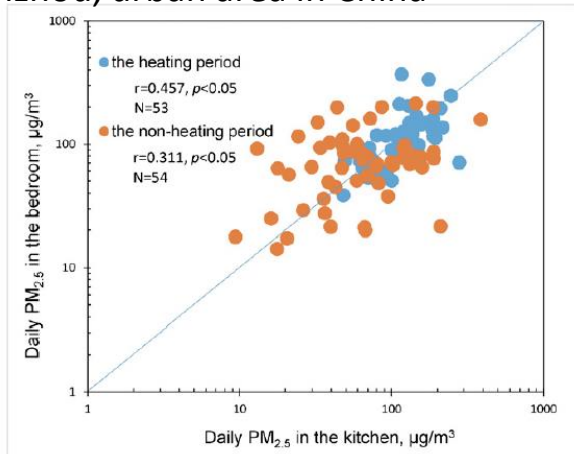
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# Sources

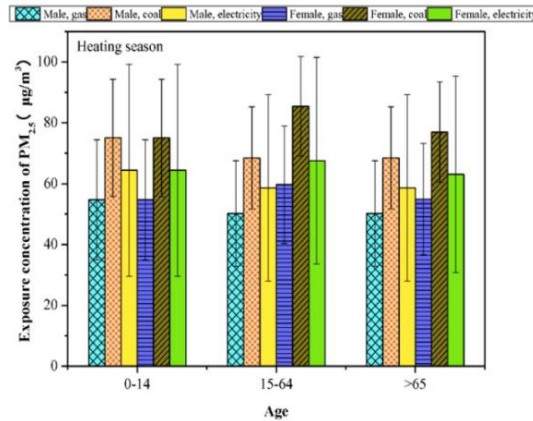
## Cooking and heating

- Use of coal for cooking leads to higher indoor pollution and exposure level.

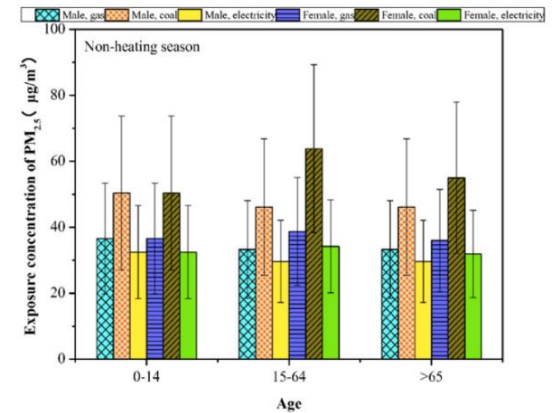
### Lanzhou, urban area in China



### Heating season



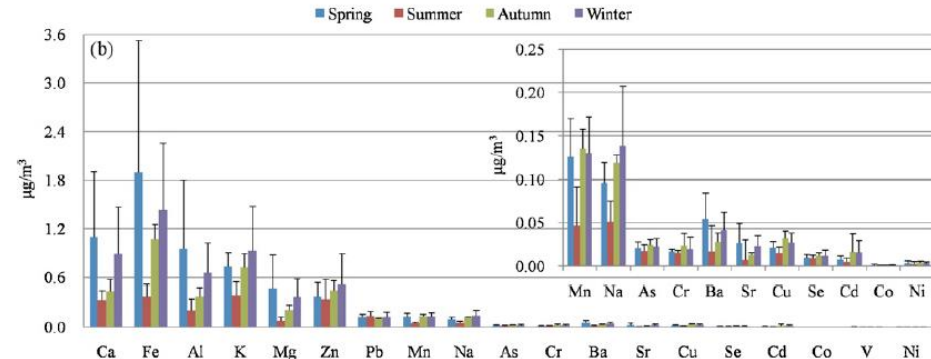
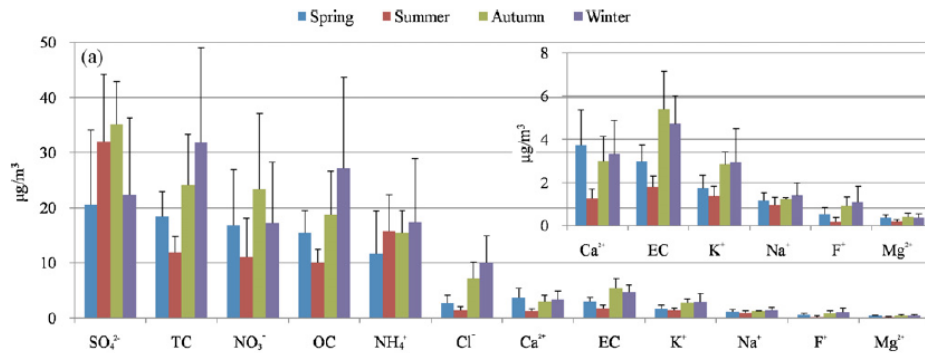
### Non-heating season



# Components of Indoor PM2.5

- The main components of PM2.5
  - Organic compounds
  - Inorganic compounds
  - Biological components
  - Carbons

## Zhengzhou (Industrial area), China

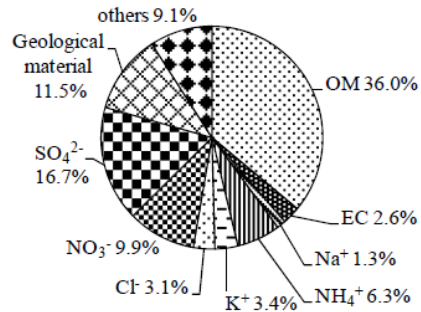


# Components of Indoor PM2.5

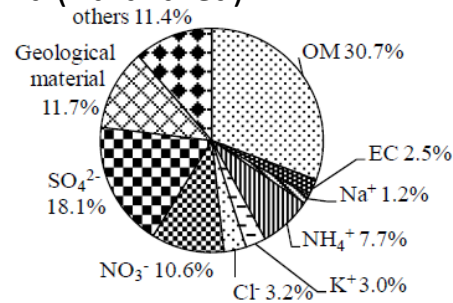
## Organic compounds

Organic compounds	Inorganic compounds	Biological components	Carbon	
Saturated and unsaturated hydrocarbon	Water-soluble inorganic salts	Inorganic elements	Bacteria, fungi, viruses, pollen and plant fibers	Elemental carbon (EC) and organic carbon (OC).
Volatile organic compounds (VOCs) and PAHs	Nitrate, ammonium and sulfate	S, Br, Cl, As, Cs, Cu, Pb, Zn, Al, Si, Ca, P, K, V, Ti, Fe and Mn		

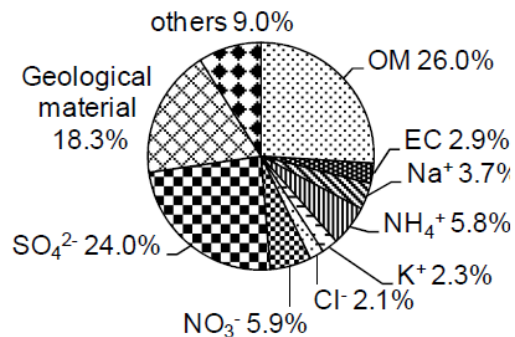
## Northwestern China (Rural area)



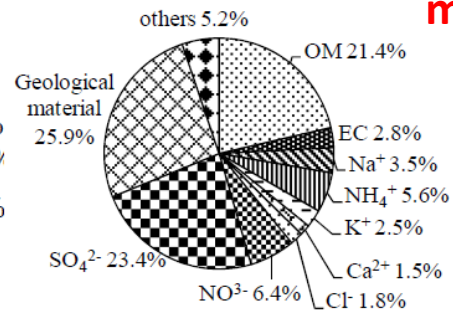
indoor winter



outdoor winter



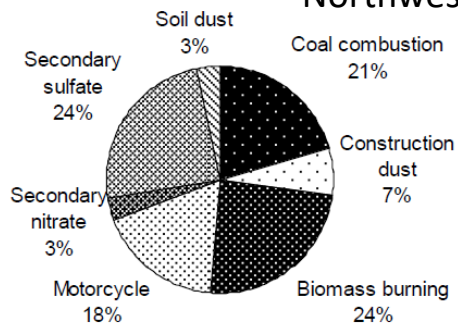
indoor summer



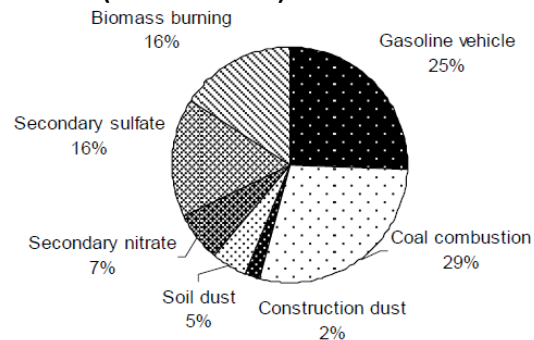
outdoor summer

**material balance**

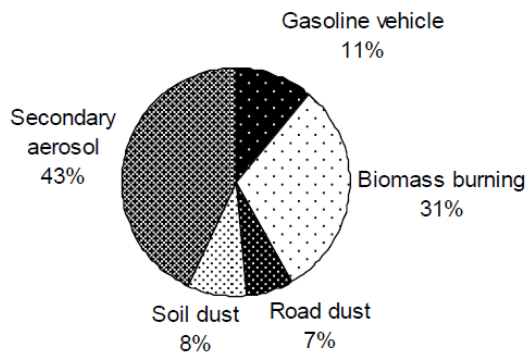
## Northwestern China (Rural area)



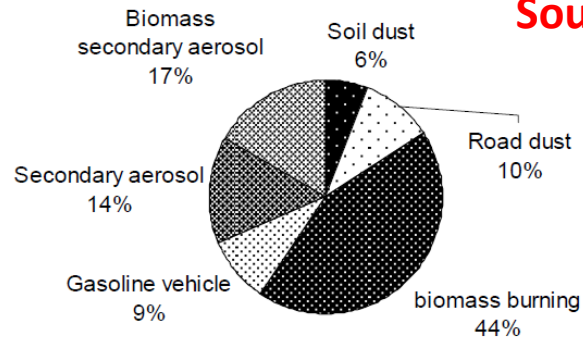
Indoor winter



Outdoor winter



Indoor summer



Outdoor summer

**Source apportionment**

# Components of Indoor PM2.5

## Concentration related factors

- Concentration and component size
- Human age
- Exposure time length

**Table 2.** Percent increase (95% CI) in mortality outcomes associated with 10- $\mu\text{g}/\text{m}^3$  increase in PM for different cumulative lag structures.<sup>a</sup>

Association under investigation	Lag 0–1	Lag 2–5	Lag 0–5
<b>All-cause mortality</b>			
PM <sub>2.5</sub>	0.55 (0.27, 0.84)	0.51 (0.07, 0.96)	0.70 (0.22, 1.18)
PM <sub>2.5–10</sub>	0.30 (–0.10, 0.69)	–0.03 (–0.70, 0.64)*	0.07 (–0.75, 0.90)*
PM <sub>10</sub>	0.32 (0.13, 0.52)	0.19 (–0.18, 0.56)*	0.28 (–0.14, 0.71)*
<b>Cardiovascular mortality</b>			
PM <sub>2.5</sub>	0.57 (0.07, 1.08)	0.77 (0.20, 1.34)	0.86 (0.15, 1.57)
PM <sub>2.5–10</sub>	0.28 (–0.37, 0.93)	0.33 (–0.59, 1.26)	0.33 (–0.78, 1.46)
PM <sub>10</sub>	0.31 (–0.01, 0.62)	0.41 (0.04, 0.79)	0.54 (0.09, 0.99)
<b>Respiratory mortality</b>			
PM <sub>2.5</sub>	0.72 (–0.11, 1.55)	1.63 (0.62, 2.65)	1.91 (0.71, 3.12)
PM <sub>2.5–10</sub>	–0.13 (–1.25, 1.01)	0.72 (–0.31, 1.76)	0.76 (–0.70, 2.25)
PM <sub>10</sub>	0.23 (–0.35, 0.81)	1.14 (0.28, 2.00)	1.12 (0.29, 1.95)

# Contents

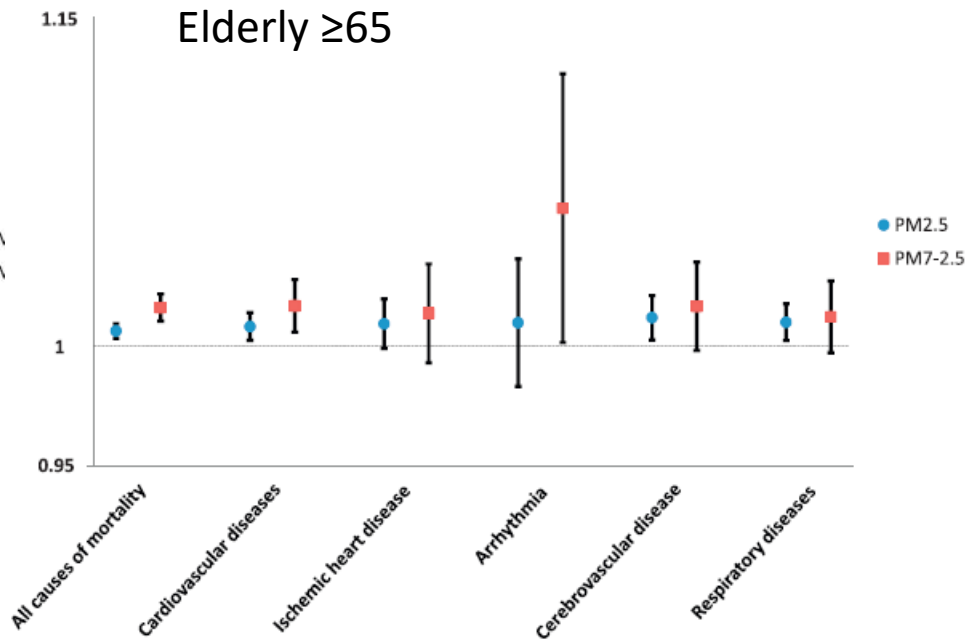
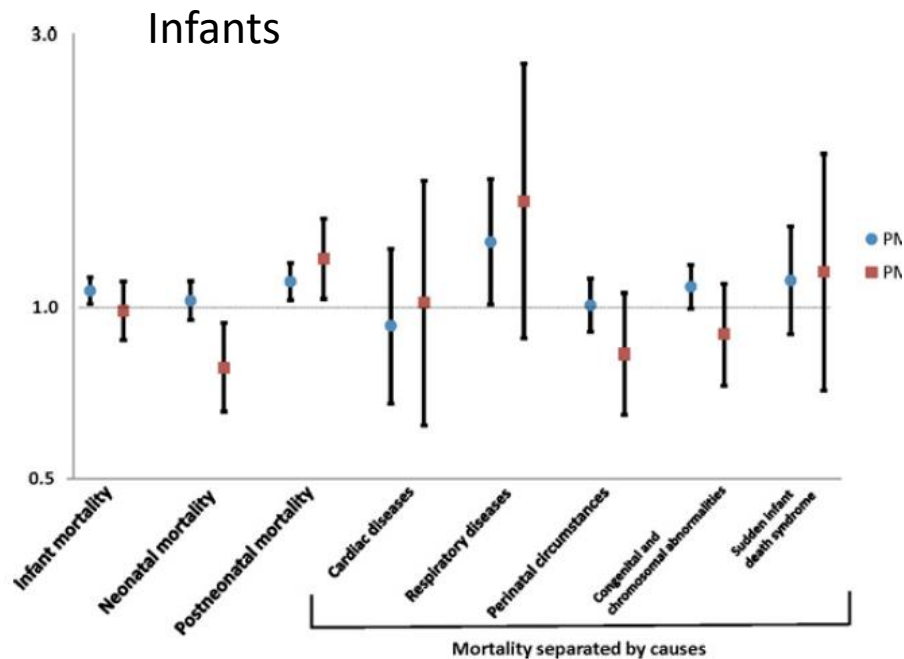
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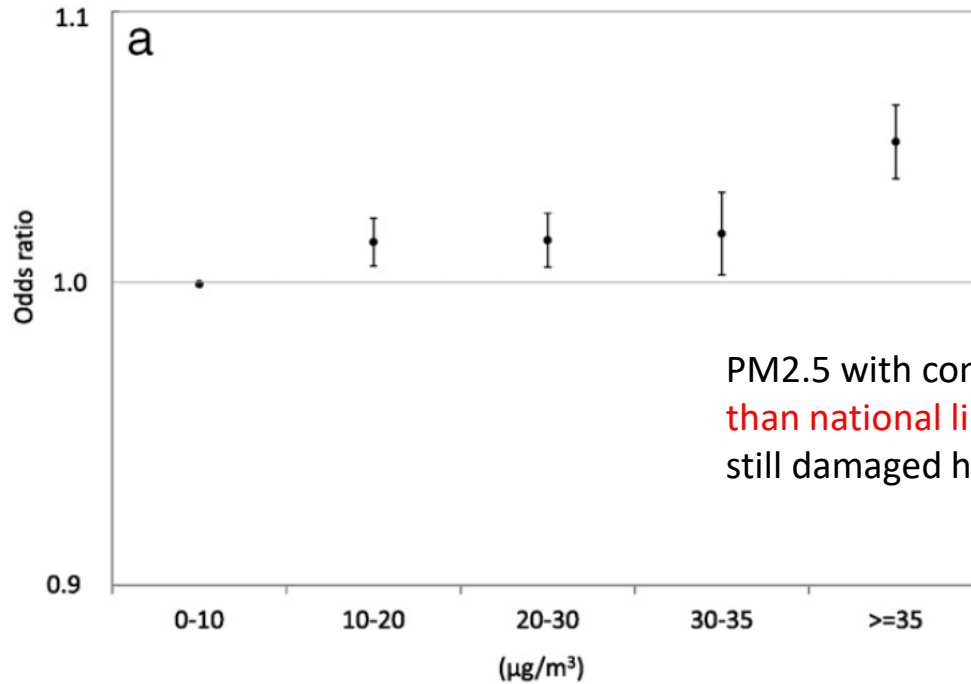
*Cited from* : Li Z et al., *Sci Total Environ.* 2017;586:610-622.

## 10 $\mu\text{g}/\text{m}^3$ increase in PM2.5 on the event day for mortality (Tokyo, Japan)



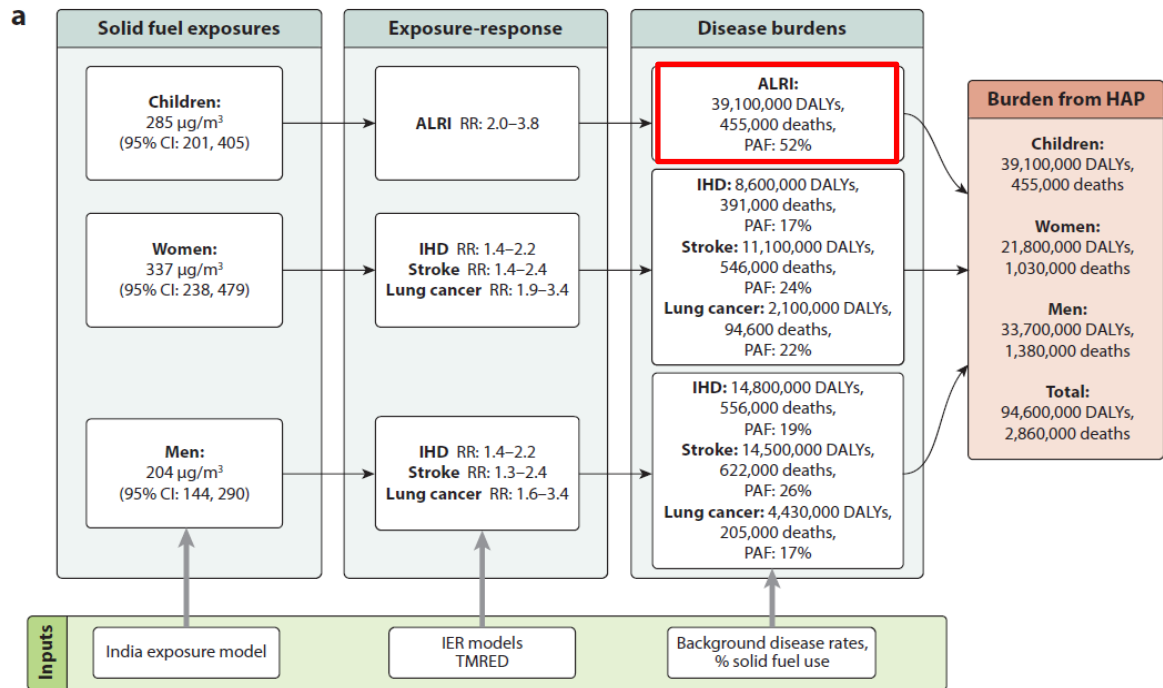
mortality  
among infants

Tokyo, Japan



PM2.5 with concentration **lower**  
**than national limit**  
still damaged health





**Figure 1**

Summary of methods, models, and data sources used in the HAP CRA-2010. The total burden is a sum of the burdens found in panels *a–c*. (*a*) Integrated exposure-response (IER)-based outcomes with modeled uncertainty at the point of the IER estimate; (*b*) meta-analysis-based outcomes with the 95% confidence intervals (CI) shown; (*c*) proportion of the ambient air pollution (AAP) burden due to household air pollution (HAP). Deaths refer to premature deaths. Figures rounded to three significant digits. See Figure 3 for totals. Other abbreviations: ALRI, acute lower respiratory infections; COPD, chronic obstructive pulmonary disease; CRA, comparative risk assessment; DALY, disability-adjusted life years; IHD, ischemic heart disease; PAF, population-attributable fraction; RR, relative risk; TMRED, theoretical minimum risk exposure distribution.

# Respiratory infection

[HAP] Increase the risk of childhood ALRI by 78% (pooled OR: **1.78** [95% CI 1.45-2.18])

Table 3. Pooled odds ratios from meta-analysis of all studies and sensitivity analyses

Group	Detail	Estimates including low exposure prevalence studies			Estimates excluding low exposure prevalence studies		
		N	OR	95% CI	N	OR	95% CI
All studies		27	1.78	1.45–2.18	23	1.93	1.54–2.42
		26	1.67 <sup>a</sup>	1.39–2.01	22	1.79 <sup>a</sup>	1.46–2.21
Study design	Randomized control trial (one study)	2	1.28	1.06–1.54	No studies with low prevalence		
	Cohort	7	2.12	1.05–4.25	No studies with low prevalence		
	All case–control	15	1.97	1.47–2.64	11	2.38	1.90–2.97
	Case–control with good control selection	(9)	1.50	1.05–2.14	(5)	2.17	1.07–4.41
	Cross sectional	3	1.49	1.21–1.85	No studies with low prevalence		
Confounding	Adequate or good adjustment <sup>a</sup>	16	1.77	1.43–2.18	14	1.80	1.43–2.25
Exposure	Good categorization <sup>a</sup>	16	1.67	1.33–2.09	14	1.73	1.35–2.20
	Solid versus clean fuel <sup>a</sup>	14	1.69	1.29–2.20	12	1.76	1.32–2.36
Outcome	Excluding DHS <sup>a</sup>	23	1.72	1.37–2.17	19	1.89	1.44–2.48
measure	Used physician diagnosis or more specific	20	1.65	1.26–2.15	16	1.83	1.31–2.55
		–	–	–	15	1.97 <sup>b</sup>	1.44–2.70
Age group	< 60 months	11	1.62	1.21–2.15	10	1.67	1.22–2.30
	< 36 months	4	2.05	1.38–3.07	3	2.17	1.37–3.43
	< 24 months <sup>a</sup>	12	1.96	1.36–2.82	9	1.85	1.27–2.69

about 70% increase

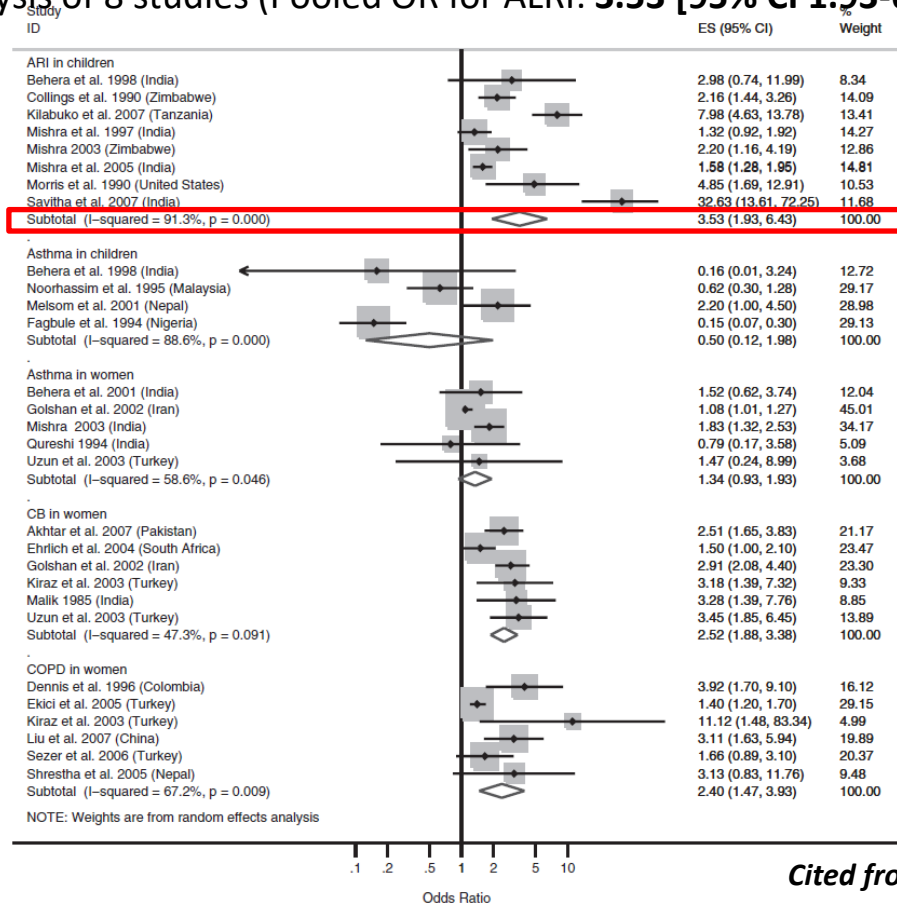
CI, confidence interval; DHS, Demographic and Health Surveys; OR, odds ratio.

<sup>a</sup> Exclusion of Group II in study by Pandey et al.<sup>32</sup> (this study was of children < 24 months of age, so only required exclusion in the youngest group in the age sensitivity analyses).

<sup>b</sup> Exclusion of study by Jin et al.<sup>35</sup>

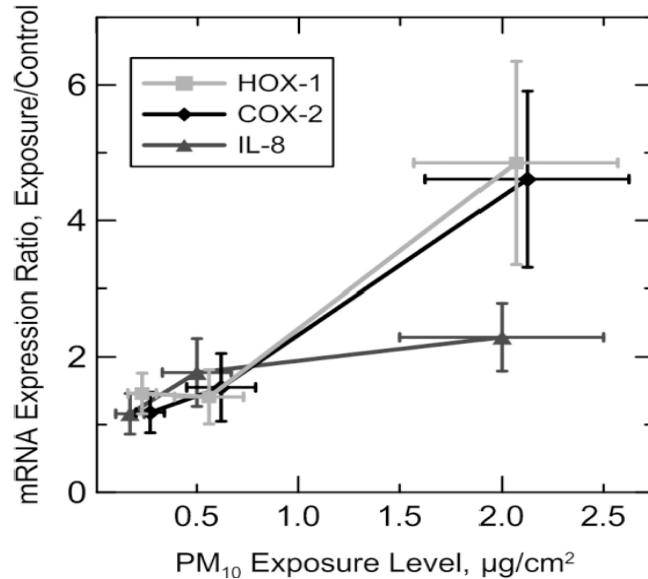
**Cited from:** Bulletin of the WHO. 2008;86:390–398.

# [HAP] A meta-analysis of 8 studies (Pooled OR for ALRI: 3.53 [95% CI 1.93-6.43])

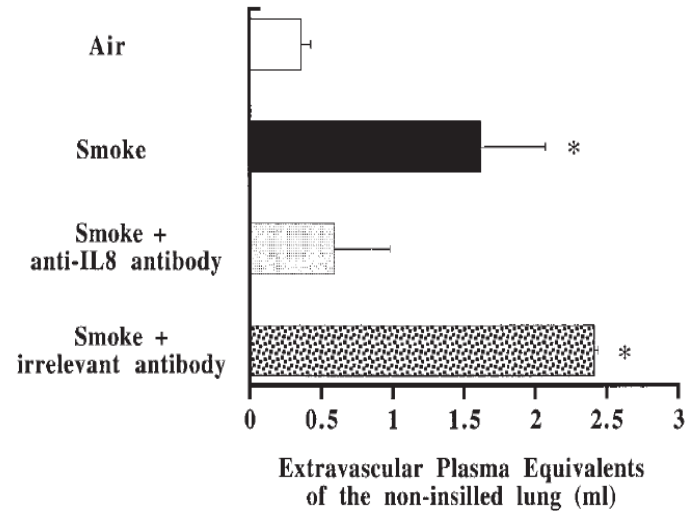


## HAP breaks the defense : Epithelial barrier

- **HAP-induced epithelial inflammation** might change the integrity of the epithelial barrier and increase the risk of bacterial invasion.



Indoor Air. 2013;23:4–13.



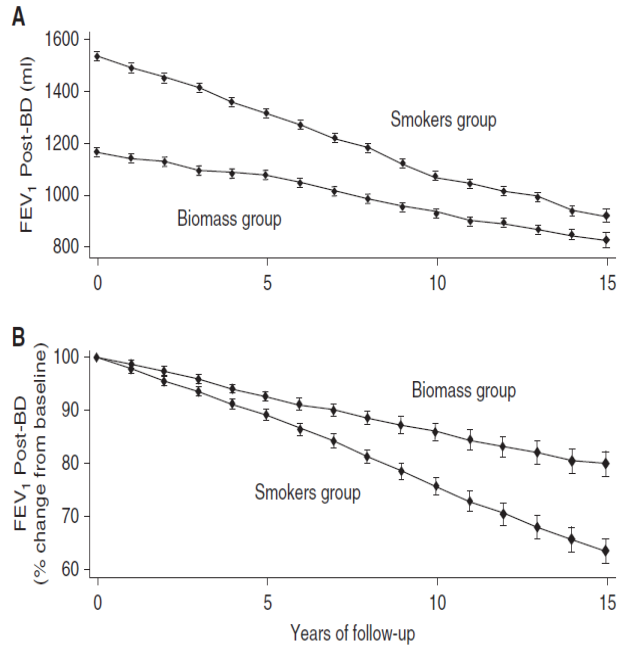
AJRCCM. 1999;160:1443–49.

**Table 1.** Different air pollutants emitted by the combustion of biomass smoke with the corresponding WHO air quality guidelines for some of them [11]

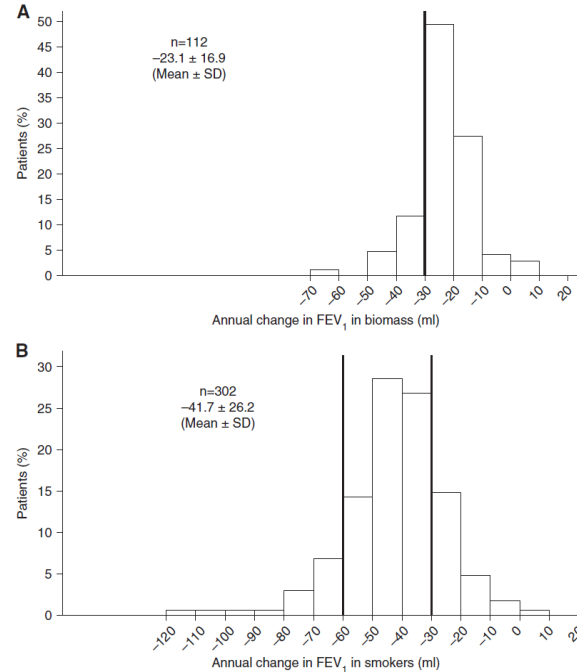
Air pollutants emitted by biomass fuel combustion	WHO air quality guidelines
Particulate matter 10	20 $\mu\text{g}/\text{m}^3$ (annual mean)
	50 $\mu\text{g}/\text{m}^3$ (24 h mean)
Particulate matter 2.5	10 $\mu\text{g}/\text{m}^3$ (annual mean)
	25 $\mu\text{g}/\text{m}^3$ (24 h mean)
Carbon monoxide	60 $\text{mg}/\text{m}^3$ (30 min mean)
	30 $\text{mg}/\text{m}^3$ (1 h mean)
	10 $\text{mg}/\text{m}^3$ (8 h mean)
Nitrogen dioxide	40 $\mu\text{g}/\text{m}^3$ (annual mean)
	200 $\mu\text{g}/\text{m}^3$ (1 h mean)
Sulfur dioxide	20 $\mu\text{g}/\text{m}^3$ (24 h mean)
	500 $\mu\text{g}/\text{m}^3$ (10 min mean)
Others: Benzene, formaldehyde, 1,3-butadiene, polycyclic aromatic hydrocarbons, such as benzo( $\alpha$ )pyrene, free radicals, aldehydes, volatile organic compounds, chlorinated dioxins, oxygenated and chlorinated organic matter, and endotoxin	

# COPD

## Biomass vs. smoking-associated COPD



**Figure 1.** FEV<sub>1</sub> decline during follow-up in milliliters (A) and as % change from baseline (B). Vertical bar is the range of variation, whereas closed diamonds show the mean change. BD = bronchodilator.



**Figure 2.** Histogram of mean FEV<sub>1</sub> annual decline for the biomass group (A) and tobacco smokers (B). Subjects were categorized into three groups according to FEV<sub>1</sub> annual decline: rapid if FEV<sub>1</sub> decline was higher than 60 ml per year (left line in B), slow if FEV<sub>1</sub> decline was from 60 to 30 ml per year (lines in B and line in A), and sustainers if FEV<sub>1</sub> decline was less than 30 ml per year.

**A slower annual decline in FEV<sub>1</sub> in biomass smoke-associated COPD as compared with tobacco smoke-associated COPD [23 vs.42 ml]**

# COPD

## Biomass vs. smoking-associated COPD

TABLE 2 Symptoms, quality of life, 6-min walking distance (6MWD) and arterial blood gases

	Biomass group	Tobacco group	p-value
Subjects n	21	22	
mMRC	1.4±1.0	1.2±0.9	0.73
<b>SGRQ<sup>#</sup></b>			
Symptoms	46±28	30±18	0.03
Activities	58±20	46±19	0.02 <sup>†</sup>
Impact	34±19	24±13	0.10 <sup>†</sup>
Total	42±18	33±14	0.07
Report of cough or sputum (from SGRQ)			
Subjects n	20	21	
Cough n (%)	18 (90)	16 (76)	0.23
Sputum n (%)	18 (90)	15 (71)	0.13
Chronic Respiratory Questionnaire <sup>‡</sup>			
Dyspnoea	17±10	14±8	0.19 <sup>†</sup>
Fatigue	19±5	19±5	0.96
Emotion	35±8	39±8	0.58
Control of disease (mastery)	21±5	24±4	0.04 <sup>†</sup>
Total	92±17	93±17	0.74
Arterial blood gas			
PaO <sub>2</sub> mmHg	49.3±7.7	52.5±4	0.11
PaCO <sub>2</sub> mmHg	36.9±5.4	33.1±4	0.01
pH	7.41±0.03	7.42±0.04	0.33
SaO <sub>2</sub> %	82±8	87±4	0.01
6MWD			
SpO <sub>2</sub> at rest %	89±5	92±3	0.07
SpO <sub>2</sub> at end of walk test %	80±9	84±4	0.04
Distance m	306±118	307±152	0.98

Data are presented as mean ± SD, unless otherwise stated. mMRC: modified Medical Research Council dyspnoea scale; SGRQ: St George's Respiratory Questionnaire; PaO<sub>2</sub>: arterial oxygen tension; PaCO<sub>2</sub>: arterial carbon dioxide tension; SaO<sub>2</sub>: arterial oxygen saturation; SpO<sub>2</sub>: arterial oxygen saturation measured by pulse oximetry. <sup>#</sup>: higher values represent worse health status; <sup>†</sup>: lower values represent worse health status; <sup>‡</sup>: nonparametric test.

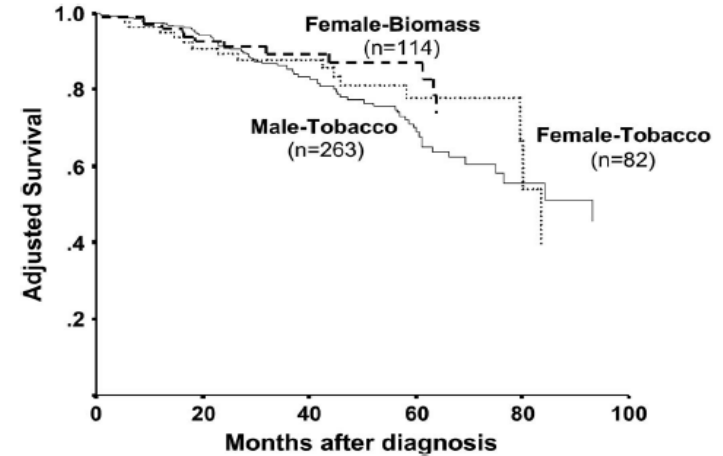


Figure 2. Survival of patients with COPD according to type of exposure and sex (male smokers, solid line; females exposed to biomass, thick dashed line; and females exposed to tobacco, thin dotted line), adjusted by FEV<sub>1</sub>%predicted, oxygen saturation, body mass index, and age, by using a Cox regression analysis. Different groups have a similar adjusted survival.

**Cited from:** *Am J Respir Crit Care Med.* 2006; 173:393–397.

# COPD

## Biomass vs. smoking-associated COPD

**Table 3** Histological findings

	Cigarette smoke mean ± SD	Biomass smoke mean ± SD	P value*
Emphysema grade, n (%)			
1	1 (4.76)	2 (7.41)	
2	0	8 (29.63)	0.019 <sup>†</sup>
3	20 (95.24)	17 (62.96)	
Large airways			
The Reid index	862.25 ± 283.03	975.81 ± 280.370	0.10 <sup>‡</sup>
Fibrosis/pigment in bronchial wall	1.66 ± 1.25	2.16 ± 1.65	0.1 <sup>‡</sup>
Small airways			
Inflammation	17.1 ± 18.5	10.7 ± 15.4	0.2 <sup>‡</sup>
Fibrosis	52.7 ± 20.1	76.2 ± 15.5	<0.001 <sup>†</sup>
Pigment deposition	24.2 ± 21.6	36.5 ± 27.0	0.1 <sup>‡</sup>
Smooth muscle hyperplasia	33.9 ± 25.9	34.7 ± 18.7	0.9 <sup>‡</sup>
Goblet cell metaplasia	22.6 ± 20.8	10.0 ± 14.4	0.02 <sup>‡</sup>
Changes in pulmonary arteries			
External diameter	340.3 ± 60.3	349.0 ± 70.0	0.6 <sup>‡</sup>
Medial thickness	55.2 ± 26.7	49.3 ± 19.2	0.4 <sup>‡</sup>
Intimal thickness	22.3 ± 8.4	31.8 ± 20.4	0.03 <sup>‡</sup>
Fibrosis/pigment on vascular wall	1.7 ± 1.0	2.1 ± 0.9	0.08 <sup>‡</sup>

\* Statistical significance was accepted at the  $P < 0.05$  level of confidence.

<sup>†</sup>  $\chi^2$ .

<sup>‡</sup> t-test.

SD = standard deviation.

**TABLE 3** Emphysema and small airways disease measurements

	Biomass group	Tobacco group	p-value
<b>Emphysema measurements</b>			
Radiologist emphysema score	0.67 ± 0.80	2.33 ± 1.53	0.0001
%LAA <950 HU at inspiratory scan	19.28 ± 10.65	27.07 ± 10.24	0.02
Size of emphysematous spaces (D)	2.57 ± 0.56	2.07 ± 0.33	0.002
<b>Airways measurements</b>			
Radiologist air trapping score	2.60 ± 0.82	1.52 ± 1.12	0.006
%LAA <856 HU at expiratory scan	54.00 ± 13.69	56.60 ± 15.26	0.57
Square root of wall area at lumen perimeter=10 mm	4.48 ± 0.34	4.33 ± 4.17	0.17
Airway wall area percentage	79.07 ± 3.61	77.91 ± 3.76	0.31
CT lung density ratio	1.18 ± 0.17	1.34 ± 0.19	0.004

Data are presented as mean ± SD, unless otherwise stated. %LAA: % low attenuation area; CT: computed tomography.

“Less emphysema but more air trapping”

**Cited from:** *Eur Respir J.* 2014; 43:725–734.

“More fibrosis in the lung parenchyma and in the walls of the bronchioles”

**Cited from:** *Int J Tuberc Lung Dis.* 2008; 12:972–977.

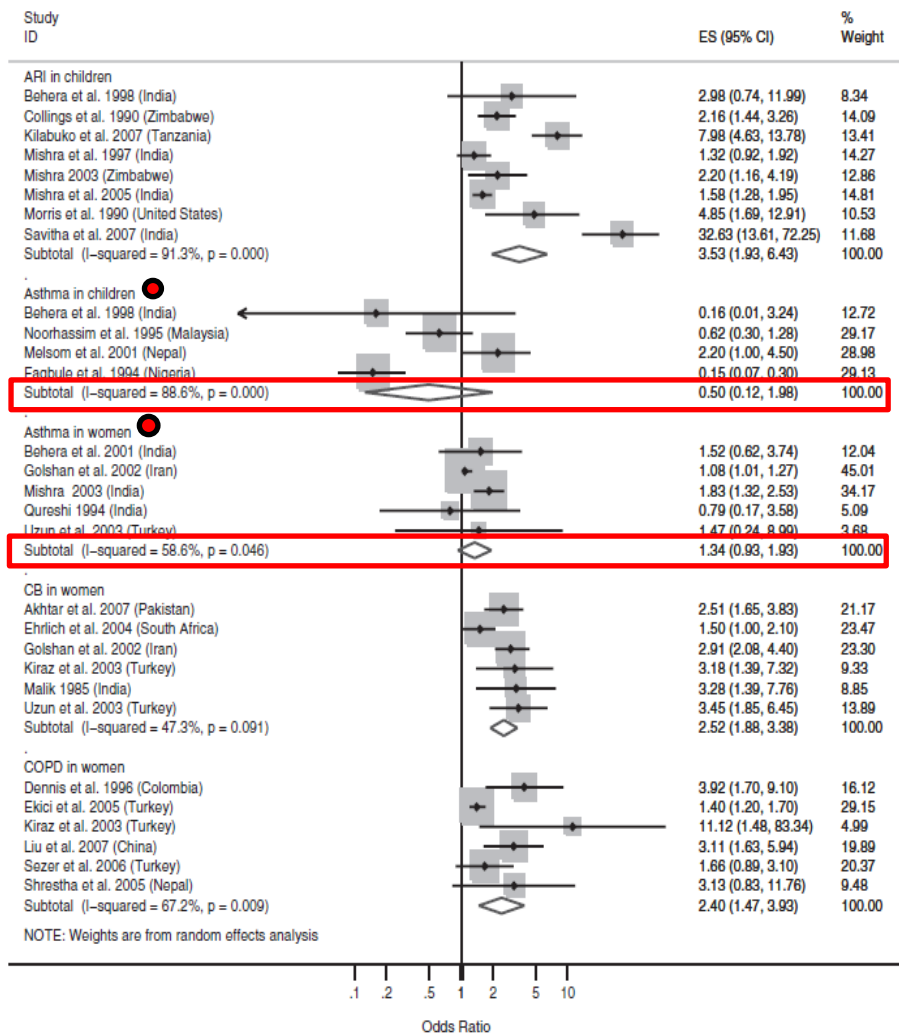
12:972–977.

Characteristics	Biomass smoke-related COPD	Tobacco smoke-related COPD
Sex	Women (58%)	Men (91%)
Mean age in years	76	71
Predominant COPD phenotypes	Chronic bronchitis	Emphysema
	COPD-asthma overlap	Chronic bronchitis
	Small airways disease	
Rate of annual FEV1 decline	23ml/year	42ml/year
Self-reported QOL	Worse	Better
Mortality	Equal	
Predominant T-helper cell inflammatory profile	Th2	Th17

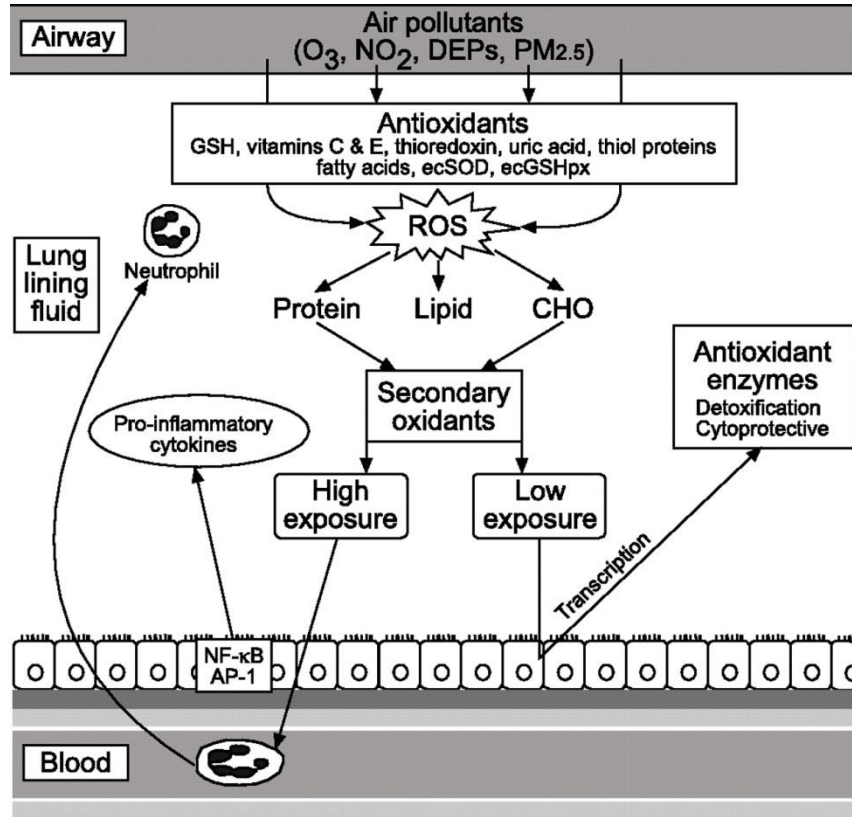
# Asthma

- **Not much evidence exists** that the risk of asthma or exacerbations of pre-existing asthma is associated with exposure to HAP.
- A systematic review and meta-analysis : ORs for asthma
  - OR 0.50 (95% CI 0.12–1.98) in children
  - OR 1.34 (95% CI 0.93–1.93) in women

Thorax. 2011; 66:232–239.



- No consensus in academia about specific pathogenic mechanism of PM2.5 to respiratory system and CVD.
- Widely accepted;
  - Inflammatory reaction
  - Oxidative stress
  - Genetic toxicity
  - Mutagenicity and carcinogenicity.



## Active free radicals in PM2.5

**Reactive oxygen species (ROS)** and **active nitrogen** from stimulated MAC and epithelial cells

-> activated the target cell oxidation pathway, eventually causing inflammation and other effects

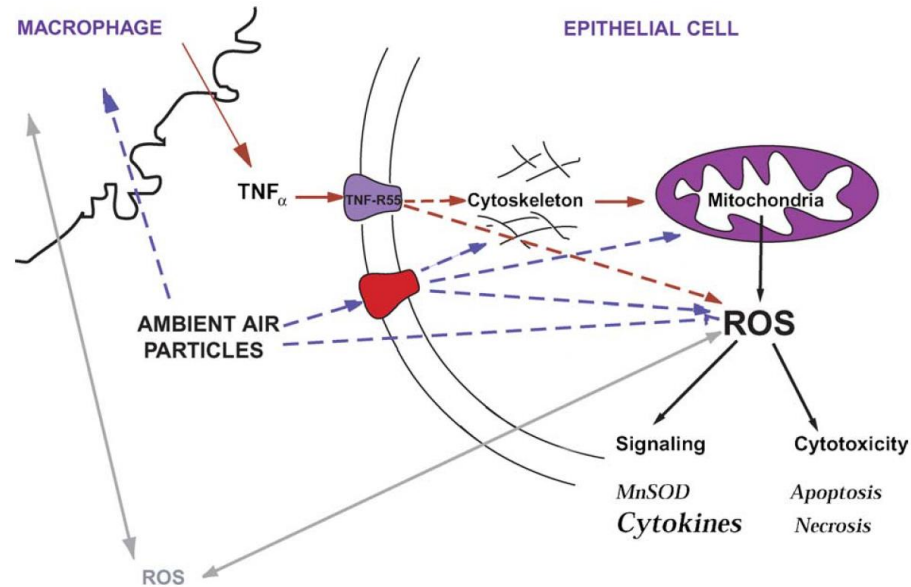


Fig. 2. Hypothetical pathways for the oxidant mechanisms of ambient particulate matter (PM) in the lung epithelium.

**Cited from:** Gonzalez-Flecha B et al. *Molecular Aspects of Medicine*. 2004;25:169–182

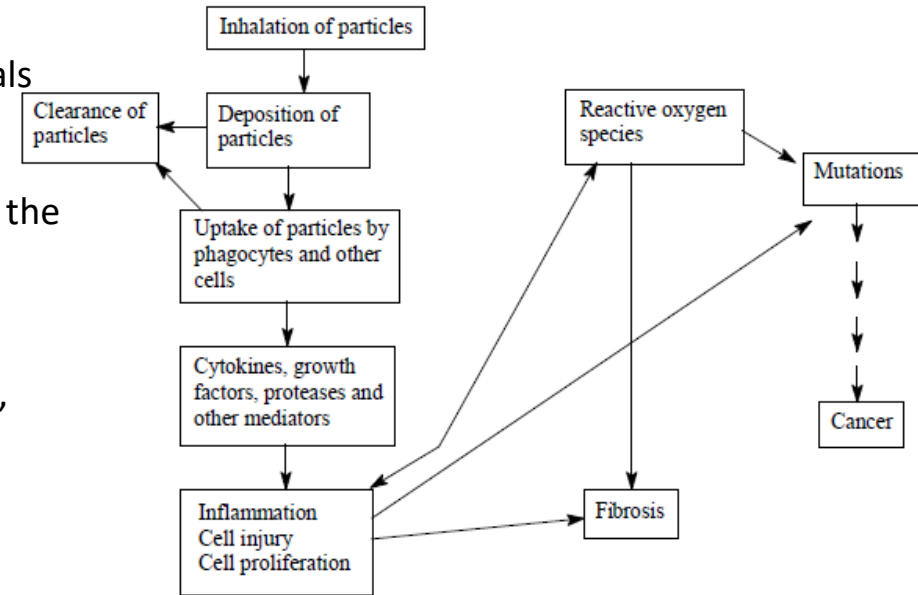
## [Incomplete combustion]

Polycyclic aromatic hydrocarbons with inhalable particles, volatile organic compounds, and some metals

Particles deposited at crucial target cells or tissues of the lung might initiate a number of biological processes

“sustained inflammation, cell injury, cell proliferation, depletion of antioxidants or impairment of other defense mechanisms, production of reactive oxygen species, and gene mutation.”

Figure 4.1. Conceptual framework of carcinogenesis induced by poorly soluble particles in rats



The scheme represents the sequence of events and modes of action that are considered to be involved in the formation of tumours that are observed in the lungs of rats after high exposure to poorly soluble particles (from IARC, 2010b)

# Contents

## Indoor Fine Particulate Matter

- **What is particulate matter?**
  - Sources of indoor PM
  - Component of indoor PM
- **Health effect of PM**
  - Epidemiological study
  - Possible mechanism
- **Intervention and Strategies**



*Cited from* : Li Z et al., *Sci Total Environ.* 2017;586:610-622.

# Source Control

## Ambient air pollutants

- Reducing dust emission from land and buildings
- Vehicles emission must be controlled
- Industrial emission must be cut down

*Br Med J.* 2013; 346: e8446.

*Occup Environ Med.* 2012;69: 354–360.

*J Expo Sci Environ Epidemiol.* 2017; 27: 64–71.

# Indoor Sources

## Sources control

- Control smoking
  - Smoking zones and non-smoking zones
- Control solid fuels usage
  - Ventilation of kitchen
  - Traditional solid fuels -> clean fuels
  - Cooking habits (patterns)
- Arrange indoor activities reasonably
  - Clean house regularly, select decoration reasonably, try not to keep pets, burn incenses less and increase indoor greening



*Br Med J.* 2013; 346: e8446.

*Occup Environ Med.* 2012;69: 354–360.

*J Expo Sci Environ Epidemiol.* 2017; 27: 64–71.

# Ventilation

## Control for transmission

- Air conditioning with air purifiers
  - The main control method in PM2.5 transmission process
- High efficiency particulate air filter (HEPA)
  - The filtration efficiency of HEPA for particles with a diameter of 0.3  $\mu\text{m}$  is up to 99.9%, indoor PM2.5 can be substantially removed (Price et al., 2005).

### “Commercials”



*Br Med J.* 2013; 346: e8446.

*Occup Environ Med.* 2012;69: 354–360.

*J Expo Sci Environ Epidemiol.* 2017; 27: 64–71.

# Future perspectives

- **Global efforts to reduce HAP**

the US Environmental Protection Agency's Partnership for Clean Indoor Air (PCIA)

with hundreds of motivated NGOs + international organizations

-> Design innovative and regionally specific solutions

In 2010, the US Government + the UN Foundation, created a public-private partnership that incorporated the PCIA into the Global Alliance for Clean Cookstoves,

-> enhancing development and implementation of clean cooking solutions for millions of households  
-> to reduce the effect of deforestation and climate change, and to empower women

The UN launched the Initiative for Sustainable Energy for All in 2011

-> provide clean cooking energy for people at the bottom of the world's energy ladder  
-> through the advancement of cleaner technologies, such as LPG by the year 2030

**“Thank you for your attention.”**





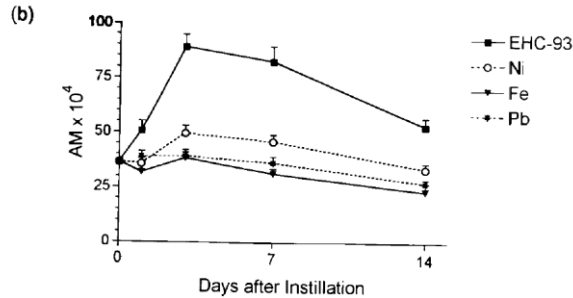
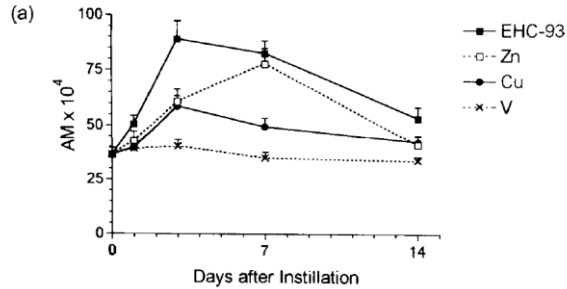
Table 1

A summary of some researches on indoor PM<sub>2.5</sub> published between 2000 and 2016

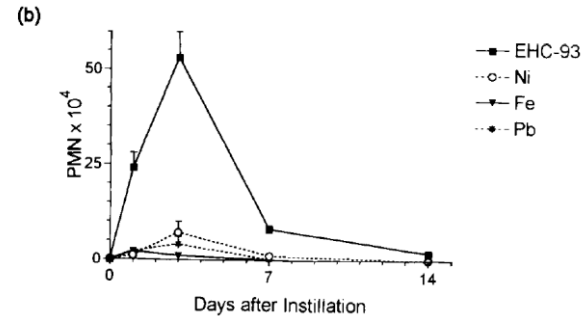
City	Sampling time	Sampling site	Study subject	Partial study results	References
Birmingham	2014	Restaurant/bar	Smoke-permitted vs. smoke-free	PM <sub>2.5</sub> concentration: smoking (287 µg/m <sup>3</sup> ) > non-smoking (34 µg/m <sup>3</sup> )	(Gurung et al., 2016)
Beijing	2010	Restaurant/bar	Smoke-permitted vs. smoke-free	SHS non-smoking: smoking sections (43 µg/m <sup>3</sup> ) > smoking facilities (40 µg/m <sup>3</sup> ) > nominal non-smoking sections (27 µg/m <sup>3</sup> ) > non-smoking sections (15 µg/m <sup>3</sup> )	(Liu et al., 2014)
San Francisco	-	Residence	Smoke-permitted vs. smoke-free	PM <sub>2.5</sub> concentration: smoking (up to 630 µg/m <sup>3</sup> ) > non-smoking (up to 160 µg/m <sup>3</sup> )	(Acevedo-Bolton et al., 2014)
Boston	2012	Multiunit housing	Smoke-permitted vs. smoke-free	Households within buildings with smoke-free policies showed lower PM <sub>2.5</sub> concentrations compared to buildings without these policies (median: 4.8 vs. 8.1 µg/m <sup>3</sup> ); secondhand smoke transfer to smoke-free apartments was demonstrable with directly adjacent households.	(Russo et al., 2015)
Cairo	2005–2006	96 venues, including waterpipe etc	Smoke-permitted vs. smoke-free	indoor PM <sub>2.5</sub> levels in venues where tobacco smoking was banned, places offering waterpipe to patrons of cafes, Ramadan tents and venues such as public buildings with poor enforcement of smoking restrictions was 72–81, 478, 612, 171–704 µg/m <sup>3</sup> respectively; smokers contributed significantly to the overall PM <sub>2.5</sub> level.	(Loffredo et al., 2016)
Dhaka	2009	Residence	Wood	Each hour that PM <sub>2.5</sub> concentrations exceeded 100 µg/m <sup>3</sup> was associated with a 7% increase in incidence of ALRI <sup>a</sup> among children aged 0–11 months	(Gurley et al., 2013)
Montana	2010–2012	Residence	Wood	PM <sub>2.5</sub> concentration was 32.3 µg/m <sup>3</sup> ; PM <sub>2.5-10</sub> was significantly correlated with PM <sub>2.5</sub>	(McNamara et al., 2013)
Xinzhou	2012–2013	Temple	Incense	PM <sub>2.5</sub> concentration was between 1.43 and 59.20 µg/m <sup>3</sup> , which was 1.75–2.70 times higher than the above standard (25 µg/m <sup>3</sup> ).	(Wu et al., 2015)
Taiyuan	2005–2007	Residence	Solid fuel etc.	Indoor medium PM <sub>2.5</sub> concentration was 68 µg/m <sup>3</sup> , and PM <sub>10</sub> was 230 µg/m <sup>3</sup> . PM levels in winter are strongly correlated with solid fuel usage for cooking, heating, and ventilators. PM <sub>1</sub> levels in cases are >3 times higher than that in control	(Mu et al., 2013)
Singapore	2009	School	Incense	During the Hungry Ghost Festival, many elements in PM <sub>2.5</sub> increased between 18% and 60%; the order of percentage increase in elemental Component was Zn > Ca > K > Mg...	(Khezri et al., 2015)
Lanzhou	2013	Residence	Natural gas/electricity vs. coal	Natural gas/electricity reduces PM <sub>2.5</sub> by 40%–70%;	(Li et al., 2016)
Dhaka	2009	Residence	Natural gas/electricity vs. biomass	PM <sub>2.5</sub> concentration: heating season (125 µg/m <sup>3</sup> ) > non-smoking (80 µg/m <sup>3</sup> )	(Salje et al., 2014)
Kibugu	2014	Residence	Gasifier vs. traditional stoves and improved stoves	Exposure time when PM <sub>2.5</sub> concentration exceeded 1000 µg/m <sup>3</sup> ; biomass (66 min/d) > natural gas/electricity vs. biomass (35 min/d)	(Njenga et al., 2016)
Cajamarca Region	2009	Kitchen of in rural houses	Chimney stoves vs. traditional open fire stoves	The gasifier domestic cooking system saved 27%–40% of fuel, reduced cooking time by 19%–23% and reduced emissions of PM <sub>2.5</sub> and CO by 40%–90%. Not statistically significant, but a post hoc stratification of chimney stoves by level of performance revealed mean PM <sub>2.5</sub> and CO levels of fully functional were 28% lower (136 µg/m <sup>3</sup> ) and 45% lower (CO, 3.2 ppm) in the kitchen environment compared with the control stoves (PM <sub>2.5</sub> , 189 µg/m <sup>3</sup> , CO, 5.8 ppm)	(Hartinger et al., 2013)

Porto	2013	Nursery	Regular service	PM <sub>2.5</sub> concentration was 158 µg/m <sup>3</sup> , exceeding WHO standard by 80%; I/O ratio was over 1, often exceeding 2	(Branco et al., 2014)
Pennsylvania and Texas	2011–2012	Retail store	Regular service	PM <sub>2.5</sub> concentration was 11 µg/m <sup>3</sup> ; the contribution to PM <sub>2.5</sub> from indoor sources was 53%	(Zaatari and Siegel, 2014)
Guangzhou	2004	4 hospitals	Regular service	Indoor PM <sub>2.5</sub> was 99 µg/m <sup>3</sup> , exceeding USEPA standard (65 µg m <sup>-3</sup> ) in 1997; 90% EC and 85% OC were found in PM <sub>2.5</sub> ; the correlation coefficient between indoor PM <sub>2.5</sub> and ambient PM <sub>2.5</sub> was 0.78	(Wang et al., 2006)
Milan	2011–2013	7 schools	Regular service	Both PM <sub>2.5</sub> and PM <sub>10</sub> were more than the 24-h guideline values of WHO, indoor CO <sub>2</sub> often exceeded ASHRAE CO <sub>2</sub> limit, significant sources were determined.	(Rovelli et al., 2014)
Changsha	2014	A shopping mall	Regular service	Outdoor average PM <sub>2.5</sub> concentration at any time was higher than that of indoor. Indoor average PM <sub>2.5</sub> ; concentration on weekend were greater than that of weekday. Among different functional areas, the average PM <sub>2.5</sub> concentration of cosmetics area was the highest, followed by dining area, public walkway, clothes area and shoes & bags area	(Hu and Li, 2015)
Xian	2007–2008	Residence	Heating vs. no-heating (Summer vs. winter)	Indoor PM <sub>2.5</sub> concentration in winter and summer was 237.2 µg/m <sup>3</sup> and 96.7 µg/m <sup>3</sup> , respectively. Biomass burning in summer was the dominant primary source for PM <sub>2.5</sub> (31% for indoor and 44% for outdoor), and those for winter were coal combustion (21% for indoor and 29% for outdoor) and biomass burning (24% for indoor and 16% for outdoor).	(Zhu, 2012)
Beijing	2013–2014	A three-bedroom apartment	Heating vs. no-heating	Indoor PM <sub>2.5</sub> concentrations were significantly correlated with outdoor PM <sub>2.5</sub> concentrations but with 1–2 h delay; Shorter lag time between indoor and outdoor PM <sub>2.5</sub> concentrations was found in heating period than non-heating period	(Han et al., 2015)
Shanghai	2013	Residence	Group A (unhealthy child living) vs. Group B (healthy child living)	PM <sub>2.5</sub> in child's bedroom of A and B was 221 µg/m <sup>3</sup> and 182 µg/m <sup>3</sup> , respectively Indoor PM <sub>2.5</sub> and PM <sub>10</sub> concentrations in all investigated houses exceeded the Chinese national standards; SVOC in house dust of four living rooms showed very high concentrations with 3 to 4 times the EU limit.	(Zhang et al., 2016)
Los Angeles	–	Experiment chamber	Popcorn vs. water (microwaving)	PM <sub>2.5</sub> generated by popcorn was 249 µg min <sup>-1</sup> ; UFPs and PM <sub>2.5</sub> generated by microwaving popcorn were 150–560 and 350–800 times higher than the emissions from microwaving water, respectively	(Zhang et al., 2014)
Kavala	2016	Hospital	Regular service	Indoor PM <sub>2.5</sub> 24-h ranged from 10.16 µg/m <sup>3</sup> to 21.78 µg/m <sup>3</sup> in the lab and	(Loupa et al.,

Pb, Al, Cu, and transition metals etc. would cause inflammation and pulmonary fibrosis and pulmonary diseases.



Number of alveolar macrophages (AM) recovered by lung lavage after instilling EHC dust or metal solutions (Zn, Cu > control,  $P < .05$  at days 3, 7; Zn > Cu,  $P < .05$ ).



Number of polymorphonuclear leukocytes (PMN) recovered by lung lavage after instilling EHC dust or metal solutions (Zn, Cu > control,  $P < .05$  at days 3, 7; Zn > Cu,  $P < .05$ ).

# Future perspectives

- **Interventions**

- **Wood burning** is an important source of HAP in high-income countries, particularly in rural communities.

## Fuel switching

Typified by aggressive policies and incentives

-> Promote a shift in heating fuels from biomass to cleaner energy sources such as natural gas and electricity

**Address elevated PM concentrations** in communities with a high frequency of wood burning

-> Replace conventional wood stoves with improved technology stoves

**Plug-in air filter devices** are efficacious household-level strategy

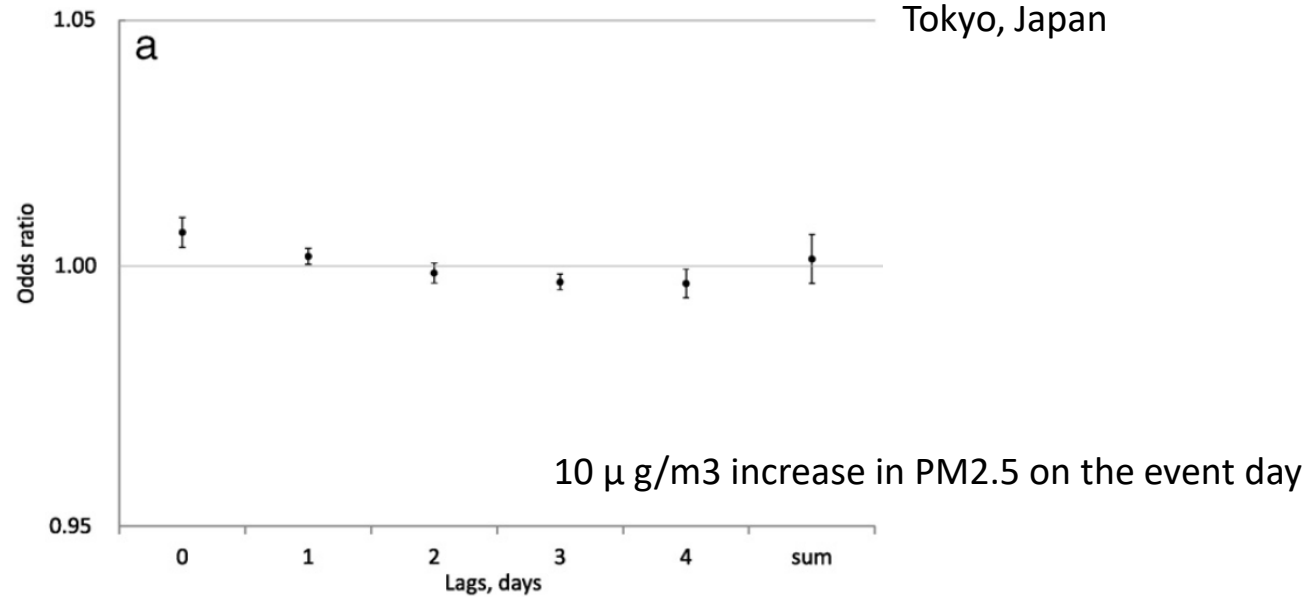
-> Improving indoor air quality in homes using wood stoves

*Br Med J.* 2013; 346: e8446.

*Occup Environ Med.* 2012;69: 354–360.

*J Expo Sci Environ Epidemiol.* 2017; 27: 64–71.

Lag model up to 5 days  
mortality (same day and up to 4 days earlier)  
among infants



## Indoor PM2.5 control -Standards of indoor PM2.5

- The Standard of the Measurement and Evaluation for Efficiency of Building Ventilation(JGJ/T309-2013), issued on July 2nd, 2013, came into effect on February 1st, 2014.
  - the daily average concentration of indoor PM2.5 should be  $<75 \mu\text{g}/\text{m}^3$ .
- The latest Ventilation for Acceptable Indoor Air Quality (ANSI/ASHRAE62.1-201), issued by the United States, was put into effect on August 21, 2015
  - Provides for indoor PM2.5 with a concentration  $<15 \mu\text{g}/\text{m}^3$
- The Residential Indoor Air Quality Guideline issued by Canada in 2012 defined PM2.5 as a monitoring object
  - but it didn't provided specific PM2.5 limit
- In Indoor Air Quality Guidelines-Implementation Outline of Domestic Fuel, WHO pointed out that the PM2.5 limit for burning domestic fuel
  - with ventilation and without ventilation was  $230 \mu\text{g}/\text{m}^3$  and  $800 \mu\text{g}/\text{m}^3$