workshop

미세먼지와 호흡기질환

박정웅
가천의대 길병원 호흡기내과

- Particulate matters (PMs)
- PM & Respiratory diseases

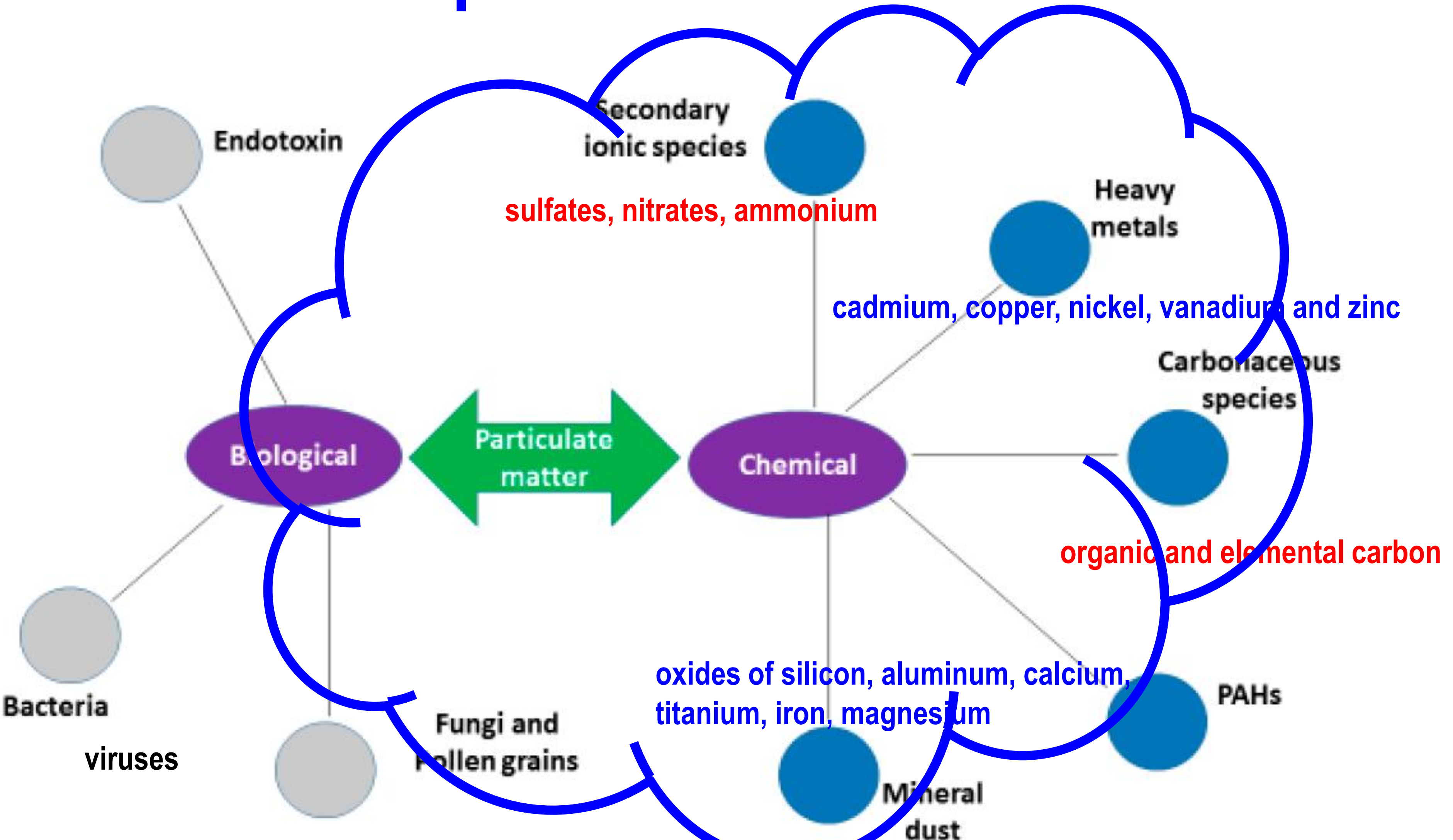
  (chronic bronchitis, COPD, IPF, Lung cancer)

What is particulate matter?

“a **complex mixture** of extremely small particles and gases and includes acids, organic chemicals, metals, soils and dust”

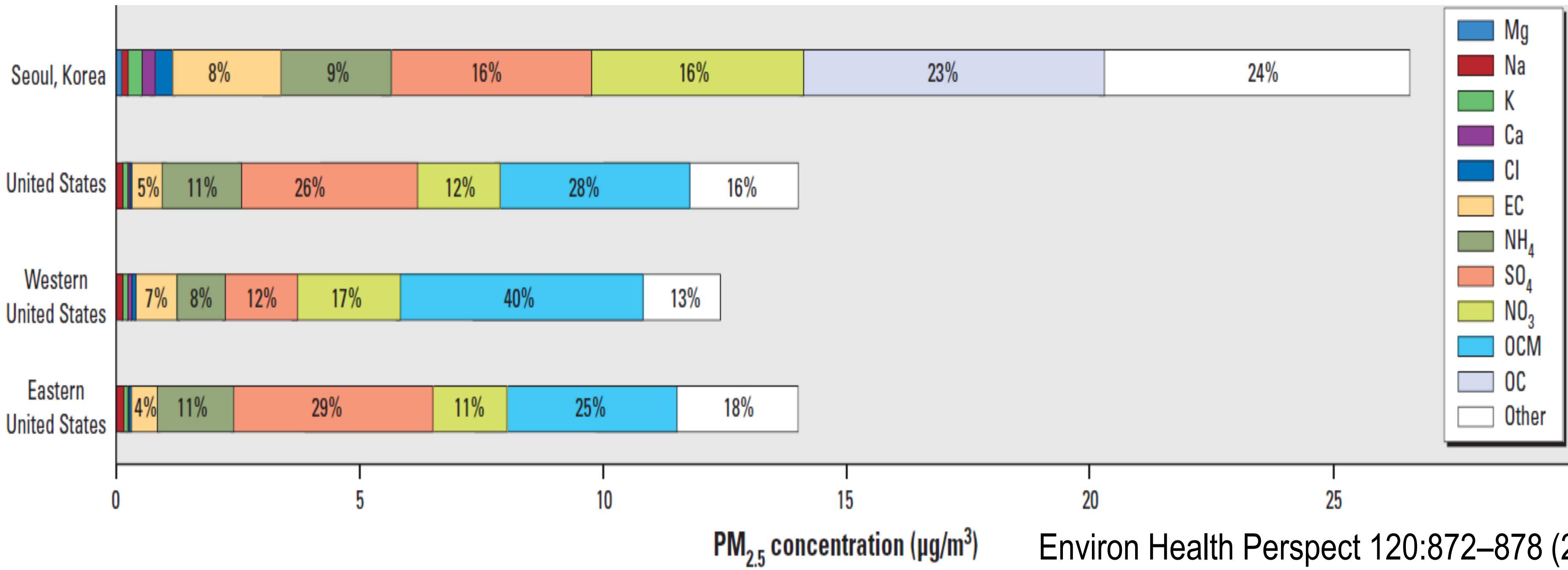
The United State Environmental Protection Agency (USEPA)

Biological and chemical components of **particulate matter**



Int. J. Environ. Res. Public Health ***2016***

Percentage of PM2.5 total mass for each component, by location



Environ Health Perspect 120:872–878 (2012

겨울철 광주지역 $PM_{2.5}$의 화학적 특성 조사

고재민[1)] · 배민석[2)] · 박승식[1)*]

[1)]전남대학교 환경에너지공학과, [2)]국립목포대학교 환경공학과

- ✓organic and elemental carbon(OC and EC),
- ✓water-soluble OC(WSOC),
- ✓eight ionic species

Par. Aerosol Res. Vol. 2013 . 89-102

Particulate Matter-Associated Bioaerosols

- Particulate Matter-Associated Endotoxins
- Particulate Matter-Associated Bacteria/Virus
- Particulate Matter-Associated Fungi and Pollen Grains

Chemicals in Airborne Particulate Matter

- Particulate Matter-Associated Trace Metals
- Particulate Matter-Associated Polycyclic Aromatic Hydrocarbons
- Particulate Matter-Associated Inorganic Water Soluble Ionic Species
- Particulate Matter-Associated Inorganic Mineral Dust
- Particulate Matter-Associated Carbonaceous Species

Effects of PM and Its Chemical Constituents on Elderly Hospital Admissions Due to Respiratory Diseases

-60 years old, admitted from 2010. 3 to 2011.2 in a medium-sized city in Brazil

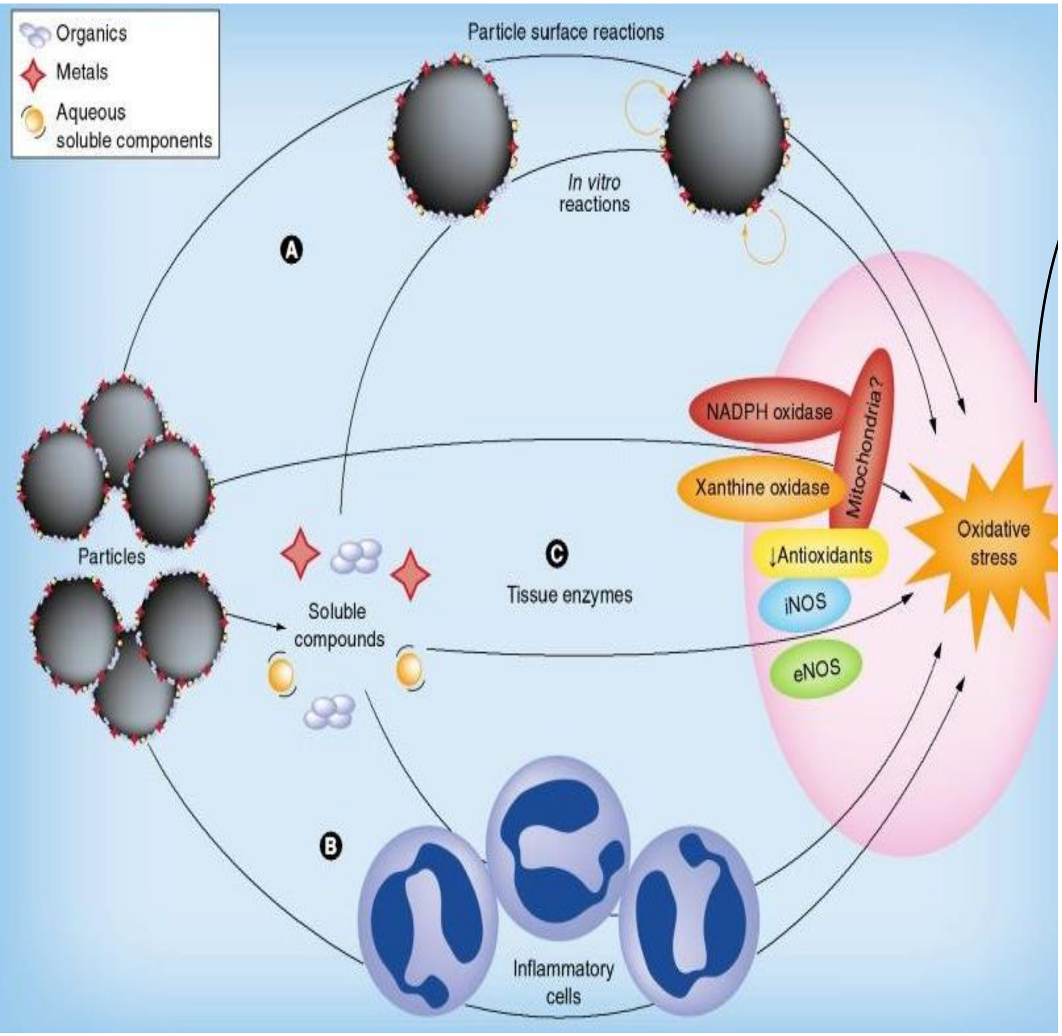
Pollutants	Respiratory Diseases %RR (LL, UL)
$PM_{2.5}$	8.5 (−6.8, 26.3)
$PM_{2.5-10}$	23.5 (13.5, 34.3)
PM_{10}	12.8 (6.0, 20.0)
PM_{10} [a]	8.9 (5.2, 12.8)
Soluble ions ($PM_{2.5}$)	
SO_4^{2-}	0.0 (−0.1, 0.1)
NH_4^+	−0.3 (−0.7, 0.1)
K^+	2.7 (1.1, 4.3)
Soluble ions ($PM_{2.5-10}$)	
Cl^-	−0.5 (−0.8, −0.2)
NO_3^-	−0.1 (−0.2, 0.1)
SO_4^{2-}	0.4 (0.1, 0.6)
Na^+	−0.4 (−0.8, 0.0)
K^+	−0.2 (-1.2, 0.8)
Ca^{2+}	0.0 (−0.2, 0.2)
Mg^{2+}	0.1 (−1.5, 1.8)

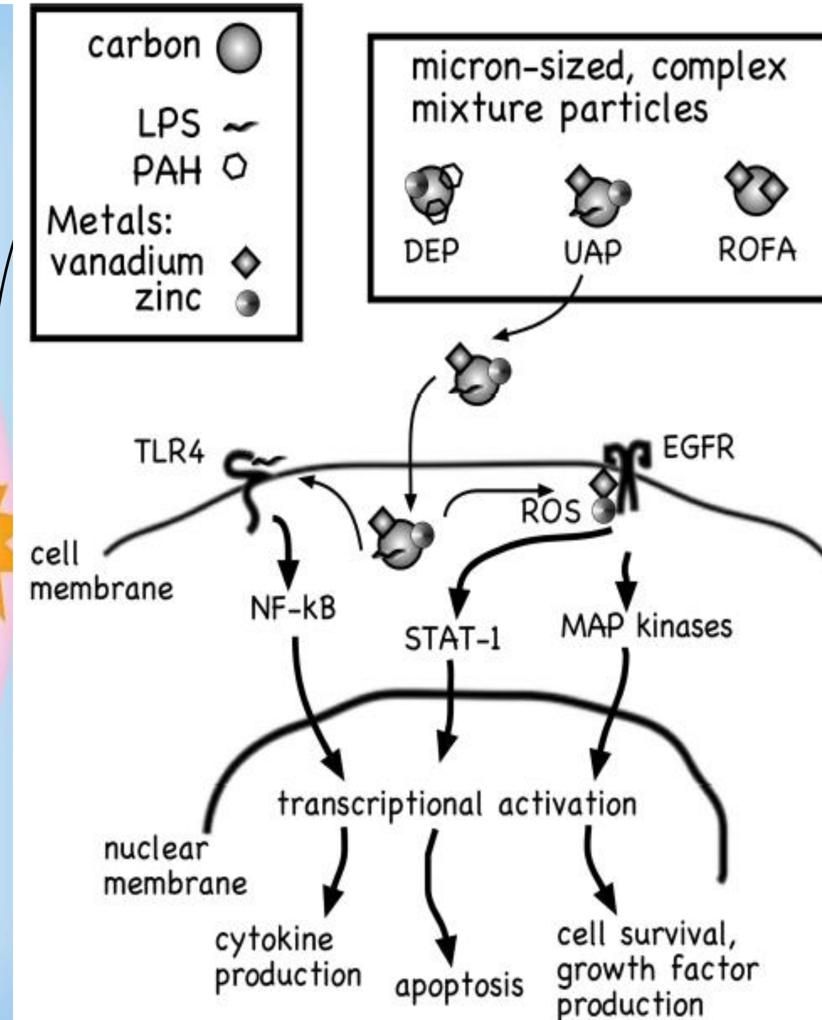
Int. J. Environ. Res. Public Health **2016, 13, 947**

PM and Respiratory diseases

- PM induced inflammation, fibrosis, apoptosis
- PM & COPD
- PM & Chronic bronchits
- PM & Pulmonary fibrosis
- PM & Lung cancer

Different pathways between particulate matter and cells can induce cellular oxidative stress





Toxicologic Pathology, 35:148–153, (2007)

*Environ. Health Perspect.*117(1),54–60 (2009)

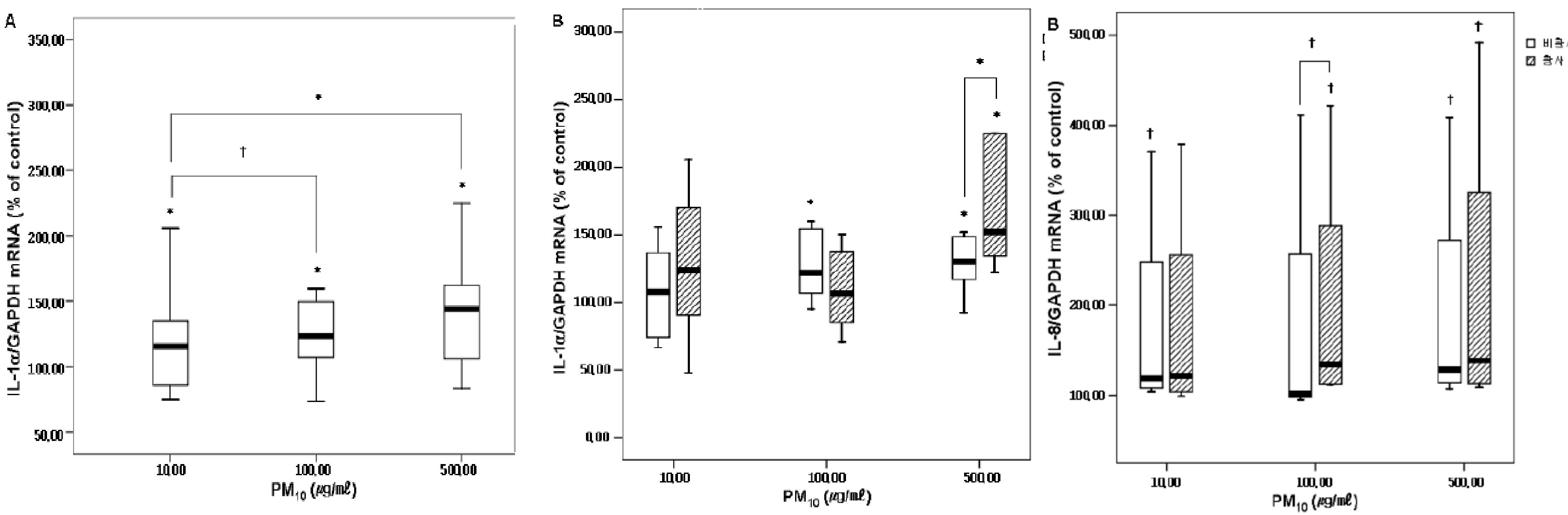
PM$_{10}$이 A549 Cells에서 전염증성 Cytokine발현에 미치는 영향

가천대학교 의과대학 길병원 호흡기내과

김정호, 전효근, 김미경, 경선영, 안창혁, 이상표, 박정웅, 정성환

- High volume air sampler(Sibata Model HV500F) with an air flow at 500ℓ/min for at least 6 hours

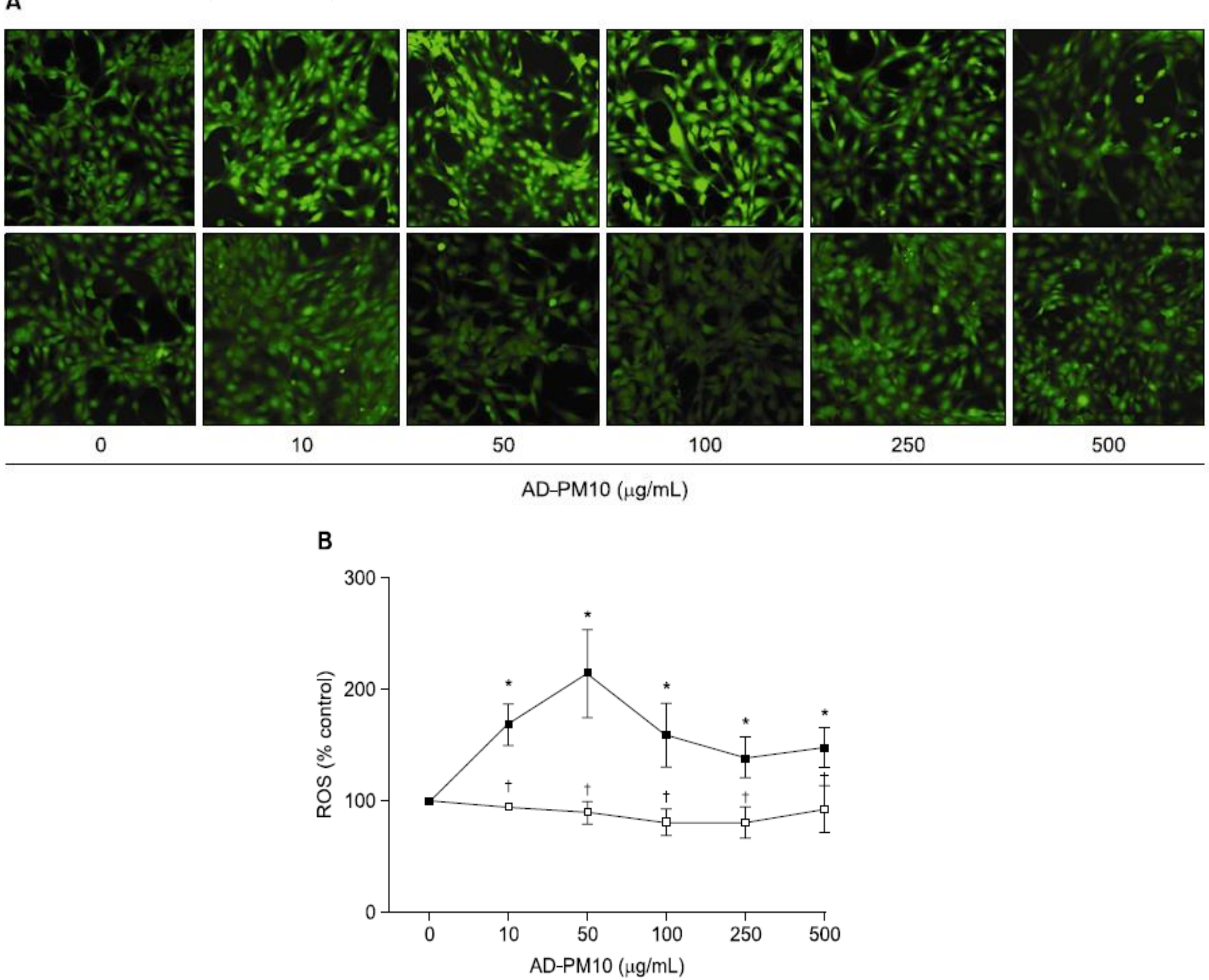
- mRNA levels of interleukin(IL)-1α, IL-Iβ , IL-8,

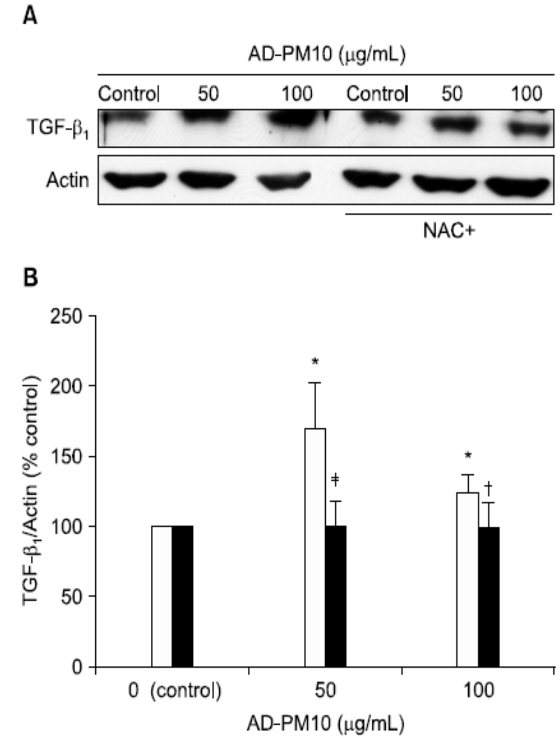


Tuberc Respir Dis 2006; 60: 663-672

Asian Dust Particles Induce TGF-β_1 via Reactive Oxygen Species in Bronchial Epithelial Cells

Sun Young Kyung, M.D., Jin Young Yoon, M.S., Yu Jin Kim, M.D., Sang Pyo Lee, M.D., Jeong-Woong Park, M.D., Sung Hwan Jeong, M.D.
Division of Pulmonology, Department of Internal Medicine, Gachon University Gil Hospital, Gachon University of Medicine and Science, Incheon, Korea

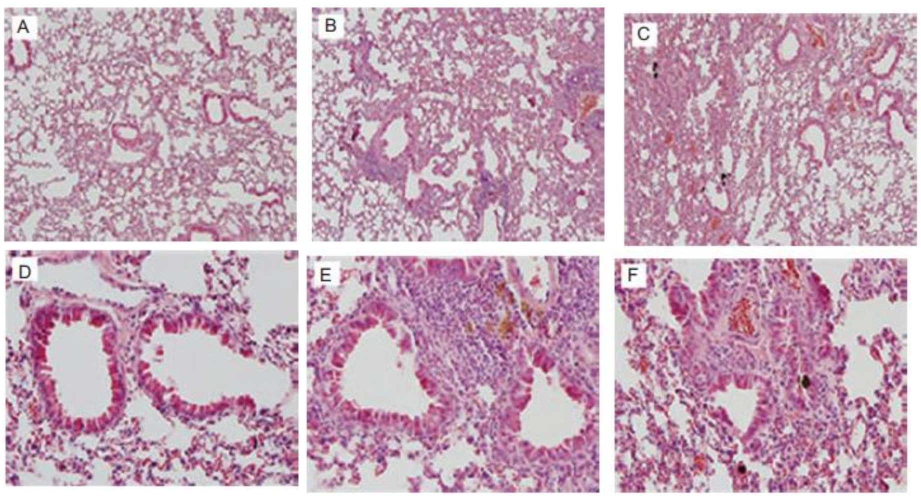


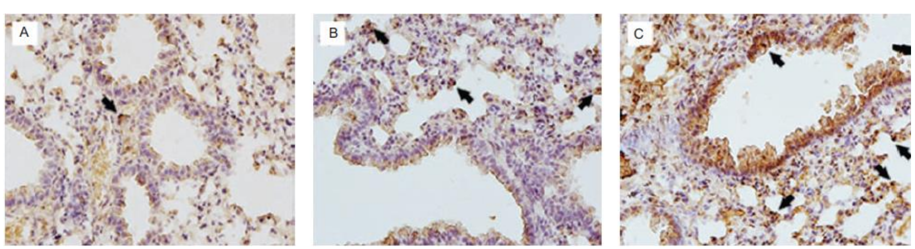


Tuberc Respir Dis 2012;73:84-92

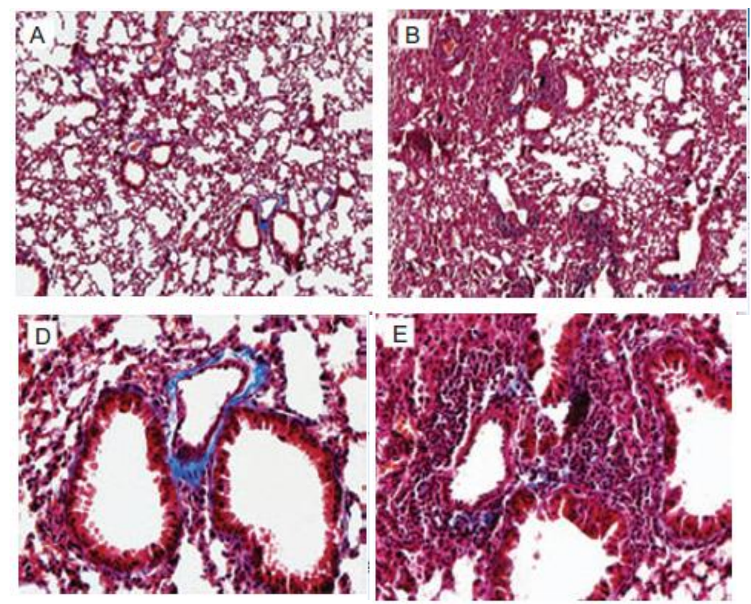
Asian dust and titanium dioxide particles–induced inflammation and oxidative DNA damage in C57BL/6 mice

You Jin Hwang[1], Ye Sul Jeung[1], Min Hae Seo[1], Jin Young Yoon[2], Dae Young Kim[1], Jeong-Woong Park[2], Joung Ho Han[3], and Sung Hwan Jeong[2]





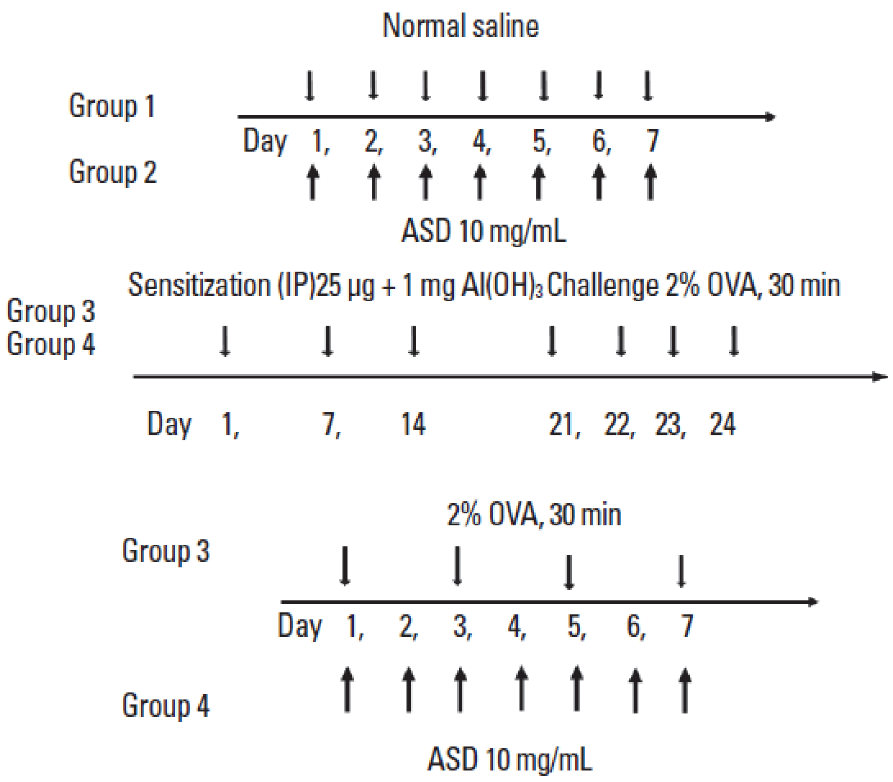
Immunohistochemical detection of 8-oxoguanine

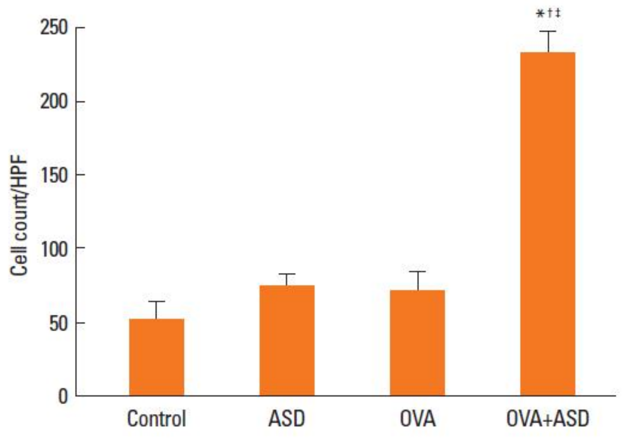


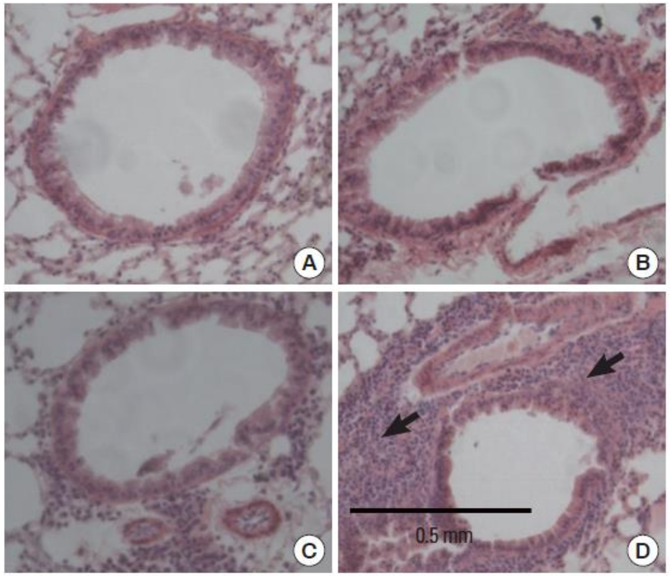
Masson's trichrome staining

Inhalation Toxicology, 2010; 22(13): 1127–1133

Asian Sand Dust (PM10) Enhances Allergen-Induced Th2 Allergic Inflammatory Changes in BALB/c Mouse Lungs







Inflammatory cells (black arrows) were more prominent in the OVA+ASD group

kang IK. et al Allergy Asthma Immunol Res. 2012 July;4(4):206-213.

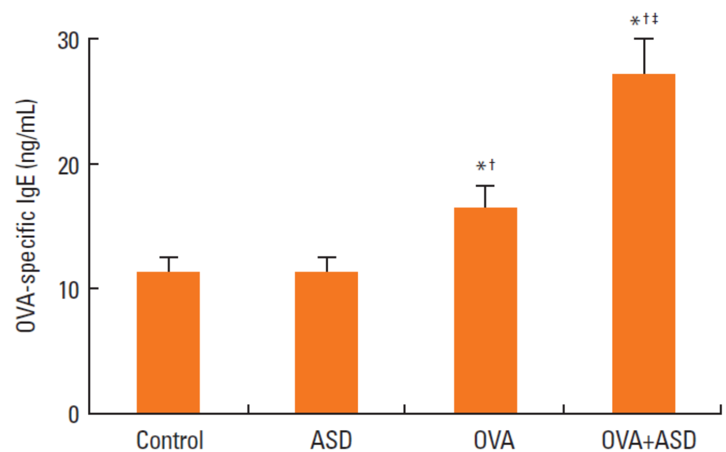
Evaluation of pathologic expression of cytokines in the murine airway

Group	Cell number/HPF					
	Eosinophils	IL-4	IL-5	TGF-α	MUC5AC	PAS
Control	0	0	0	0	0	0
ASD	0	$9.6\pm2.7^{*}$	0	0	0	0
OVA	$1.3\pm0.7^{*,\dagger}$	$2.4\pm2.3^{*}$	$31.0\pm14.1^{*,\dagger}$	3.6 ± 2.4	$7.4\pm5.3^{*,\dagger}$	$10.0\pm3.1^{*,\dagger}$
OVA+ASD	$4.7\pm1.2^{*,\dagger,\ddagger}$	$14.2\pm10.0^{*,\dagger,\ddagger}$	$68.4\pm20.0^{*,\dagger,\ddagger}$	$17.6\pm7.4^{*,\dagger,\ddagger}$	$25.8\pm3.7^{*,\dagger,\ddagger}$	$28.6\pm1.3^{*,\dagger,\ddagger}$

Cell counts in bronchoalveolar lavage fluid

	Total cell number ($\times10^4$/mL)	Differential counts			
		Lymphocytes	Macrophages	Neutrophils	Eosinophils
G1	36.4 ± 3.5	1.2 ± 0.3	31.3 ± 6.4	3.7 ± 0.4	0.1 ± 0.01
G2	$96.7\pm11.3^{*}$	$8.0\pm3.9^{*}$	$49.0\pm20.3^{*}$	$39.6\pm18.3^{*}$	1.6 ± 1.1
G3	$76.3\pm12.5^{*}$	$7.6\pm1.5^{*}$	$45.8\pm13.7^{*}$	$16.8\pm3.4^{*}$	$6.3\pm1.6^{*,\dagger}$
G4	$91.0\pm16.7^{*}$	$9.6\pm2.7^{*}$	28.7 ± 13.8	$14.1\pm3.2^{*}$	$46.7\pm17.7^{*,\dagger,\ddagger}$

Serum OVA-specific IgE level



Allergy Asthma Immunol Res. 2012 July;4(4):206-213.

Asian Sand Dust(PM10) Enhances Allergic Inflammatory Changes

- ASD **(PM10) increased allergic inflammation** in the ASD+OVA group
- **Synergistic effect** on the production of OVA-induced Th2 cytokines as an adjuvant in allergic inflammatory reactions
- **Mucin production** through increasing pro-inflammatory cytokine levels

Allergy Asthma Immunol Res. 2012 July;4(4):206-213.

PM & Pulmonary function

PM & Pulmonary function

- Children (5→11yr)exposure to PM10 causes the loss of 23 ml in the FEV1 test for every 1 μg/m^3 increment of PM10 *Environ Health Perspect) (2013,122: 1213–1238)*, –1.77% (95% CI: –3.34, –0.18%) for a 5-μg/m^3 increase in PM2.5*(Environ Health Perspect 2013, 121:1357–1364* ***ESCAPE****)*

- PM10 reduced annually 3.4% FVC, **1.6% FEV1** per each increment of 10 μg/m^3 of PM10 in adult population *Am J Respir Crit Care Med (1997)* ***SAPALDIA***

- Effect of a decline of 10 μg/m^3 of PM10 over an 11-year period was to reduce the annual rate of decline in **FEV1 by 9%** and of FEF25–75 by 16% *N Engl J Med 2007;357:2338-47* ***SAPALDIA***

Association between level of lung function and exposure to air pollution

European Study of Cohorts for Air Pollution Effects **(ESCAPE),** 5 cohort studies **(ECRHS, EGEA, NSHD, SALIA, SAPALDIA**)

- ESCAPE monitoring (**2008/4-2011/10**) air pollution, 14days of three seasons
- **7613** subjects-- **Land use regression model, Geographic information system (GIS)**
- Spirometry (1985-2010)

–back extrapolated to **first**/second spirometry

C extrapolate =modelled ESCAPE annual mean Conc x (**C** routine baseline **/ C** routine ESCAPE)

Exposure (increment)	Level of lung function (mL)#					
	FEV1			FVC		
	Beta¶	95% CI	I^2 § p-value (het)	Beta¶	95% CI	I^2 § p-value (het)
NO_2 (10 $\mu g \cdot m^{-3}$)	–13.98	–25.82 to –2.14	0.0% p=0.625	–14.93	–28.73 to –1.13	0.0% p=0.977
NO_x (20 $\mu g \cdot m^{-3}$)	–12.91	–23.79 to –2.04	0.0% p=0.861	–13.25	–25.85 to –0.65	0.0% p=0.962
PM_{10} (10 $\mu g \cdot m^{-3}$)	–44.56	–85.36 to –3.76	0.0% p=0.628	–58.96	–112.27 to –5.65	0.0% p=0.785
$PM_{2.5}$ (5 $\mu g \cdot m^{-3}$)	–21.14	–56.37 to 14.08	0.0% p=0.535	–36.39	–83.29 to 10.50	0.0% p=0.877
$PM_{2.5}$ absorbance (1×10^{-5} m^{-1})	–24.40	–55.58 to 6.79	0.0%	–12.94	–50.23 to 24.30	0.0%

Eur Respir J 2015; 45: 38–50

Association between change of lung function and exposure to air pollution

European Study of Cohorts for Air Pollution Effects (ESCAPE), 5 cohort studies
(ECRHS, EGEA, NSHD, SALIA, SAPALDIA)

Exposure (increment)	Change in lung function (mL per year)					
	FEV1			FVC		
	Beta§§	95% CI	I^2 § p-value (het)	Beta§§	95% CI	I^2 § p-value (het)
NO_2 (10 µg·m^{-3})	0.30	−0.39 to 0.98	0.0% p=0.681	0.02	−0.84 to 0.88	0.0% p=0.532
NO_x (20 µg·m^{-3})	0.18	−0.44 to 0.80	0.0% p=0.708	−0.09	−0.86 to 0.69	0.0% p=0.804
PM_{10} (10 µg·m^{-3})	−0.39	−2.85 to 2.06	53.1% p=0.074%	−1.42	−4.53 to 1.70	28.4% p=0.232
$PM_{2.5}$ (5 µg·m^{-3})	−0.14	−2.26 to 1.98	23.8% p=0.263	−1.37	−4.04 to 1.29	0.0% p=0.964
$PM_{2.5}$ absorbance (1×10^{-5} m^{-1})	0.88	−0.76 to 2.52	54.5% p=0.066	1.14	−0.95 to 3.24	4.5% p=0.381
Coarse PM (5 µg·m^{-3})	0.26	−3.92 to 4.43	61.7% p=0.034	−1.31	−6.49 to 3.88	0.0% p=0.506

It seems **premature to conclude** that long-term exposure to air pollution **does not affect FEV1 and FVC decline.**

Eur Respir J 2015; 45: 38–50

PM & Chronic bronchitis

Cross-sectional associations between air pollution and chronic bronchitis: an ESCAPE meta-analysis

Asthma-E3N,ECRHS, NSHD, SALIA, SAPALDIA

- 10,537 participants in PM analyses, Sx for 3 months for ≥2 years

model 3 (adjusted for age, sex, smoking, education and season of interview)

Outcome	Chronic bronchitis		Chronic cough		Chronic phlegm	
Exposure†	OR (95% CI)	p_{het}‡	OR (95% CI)	p_{het}	OR (95% CI)	p_{het}
$PM_{2.5}$ (5 $\mu g/m^3$)	0.90 (0.74 to 1.09)	0.754	0.91 (0.80 to 1.04)	0.440	0.96 (0.84 to 1.11)	0.850
$PM_{2.5abs}$ (per 10^{-5} m^{-1})	1.02 (0.85 to 1.22)	0.504	1.01 (0.89 to 1.15)	0.070	1.02 (0.88 to 1.18)	0.693
PM_{10} (10 $\mu g/m^3$)	0.92 (0.75 to 1.13)	0.193	0.92 (0.80 to 1.06)	0.494	1.02 (0.87 to 1.18)	0.696
PM_{coarse} (5 $\mu g/m^3$)	0.99 (0.83 to 1.18)	0.165	0.99 (0.87 to 1.12)	0.737	1.07 (0.94 to 1.22)	0.131
Traffic intensity§	0.95 (0.75 to 1.19)	0.739	0.96 (0.80 to 1.14)	0.339	0.98 (0.82 to 1.17)	0.366
Traffic load¶	0.99 (0.82 to 1.20)	0.509	0.95 (0.81 to 1.10)	0.831	0.97 (0.83 to 1.13)	0.797

Thorax 2014;69:1005–1014

Never-smokers results on model 3 (adjusted for age, sex, smoking, education and season of interview) for PM and traffic indicators

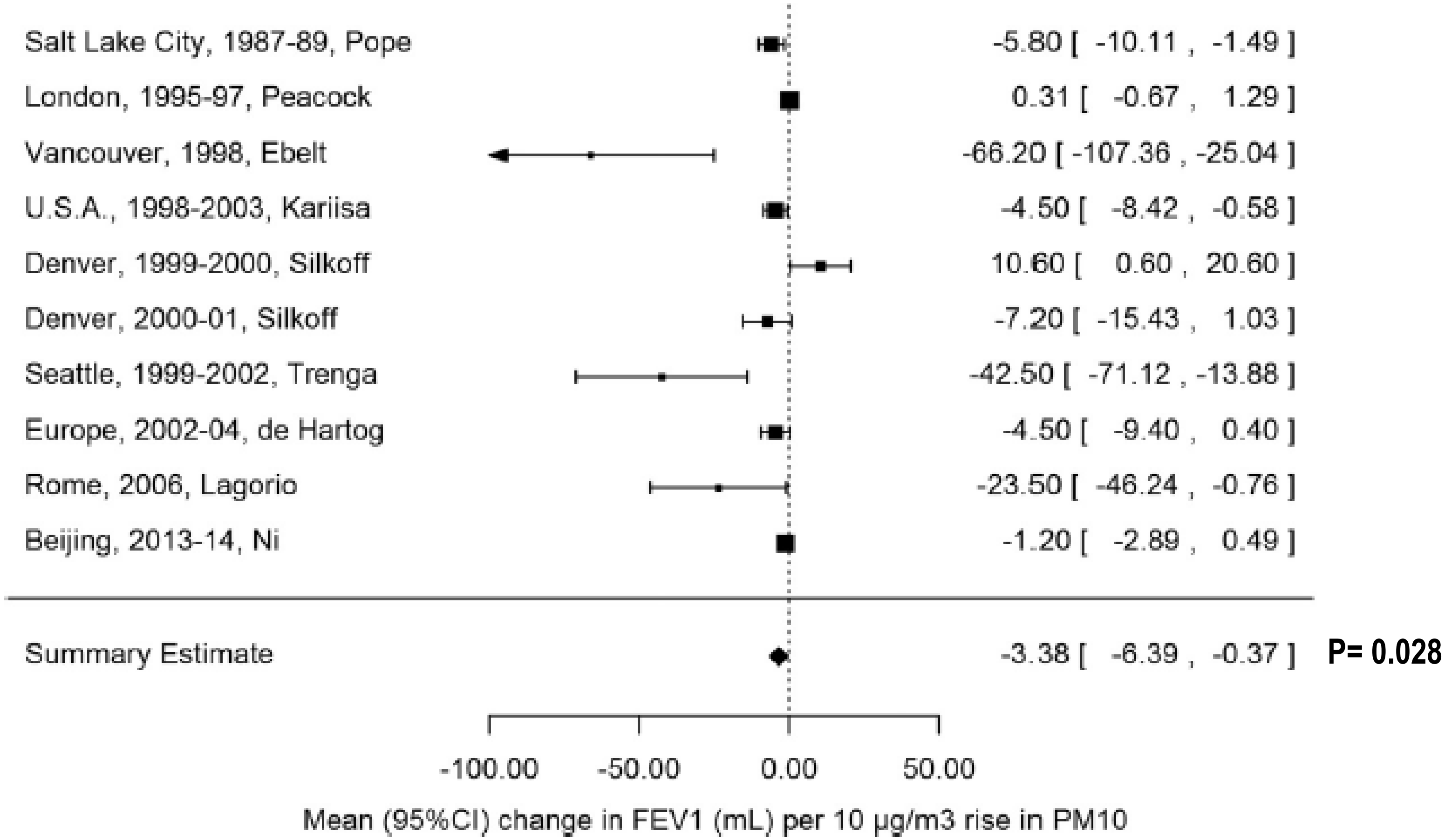
Outcome	Chronic bronchitis		Chronic cough		Chronic phlegm	
Exposure‡	OR (95% CI)	p_{het}§	OR (95% CI)	p_{het}	OR (95% CI)	p_{het}
$PM_{2.5}$ (5 $\mu g/m^3$)	1.28 (0.95 to 1.72)	0.488	1.11 (0.90 to 1.36)	0.091	1.16 (0.91 to 1.48)	0.519
$PM_{2.5abs}$ (per 10^{-5} m^{-1})	1.20 (0.92 to 1.57)	0.233	1.16 (0.96 to 1.39) 1.27† (0.88 to 1.85)	0.032	1.10 (0.87 to 1.39)	0.602
PM_{10} (10 $\mu g/m^3$)	1.35 (0.97 to 1.88)	0.763	1.08 (0.86 to 1.35)	0.535	1.32 (1.02 to 1.71)	0.474
PM_{coarse} (5 $\mu g/m^3$)	1.15 (0.87 to 1.53)	0.979	1.06 (0.87 to 1.29)	0.954	1.31 (1.05 to 1.64)	0.457
Traffic intensity¶	1.12 (0.79 to 1.57)	0.763	1.07 (0.84 to 1.37)	0.629	1.04 (0.80 to 1.37)	0.830
Traffic load**	1.11 (0.83 to 1.49)	0.354	1.03 (0.82 to 1.29)	0.51	1.02 (0.79 to 1.32)	0.422

contribute to strengthening the evidence-based studies for policy formulation

Thorax 2014;69:1005–1014

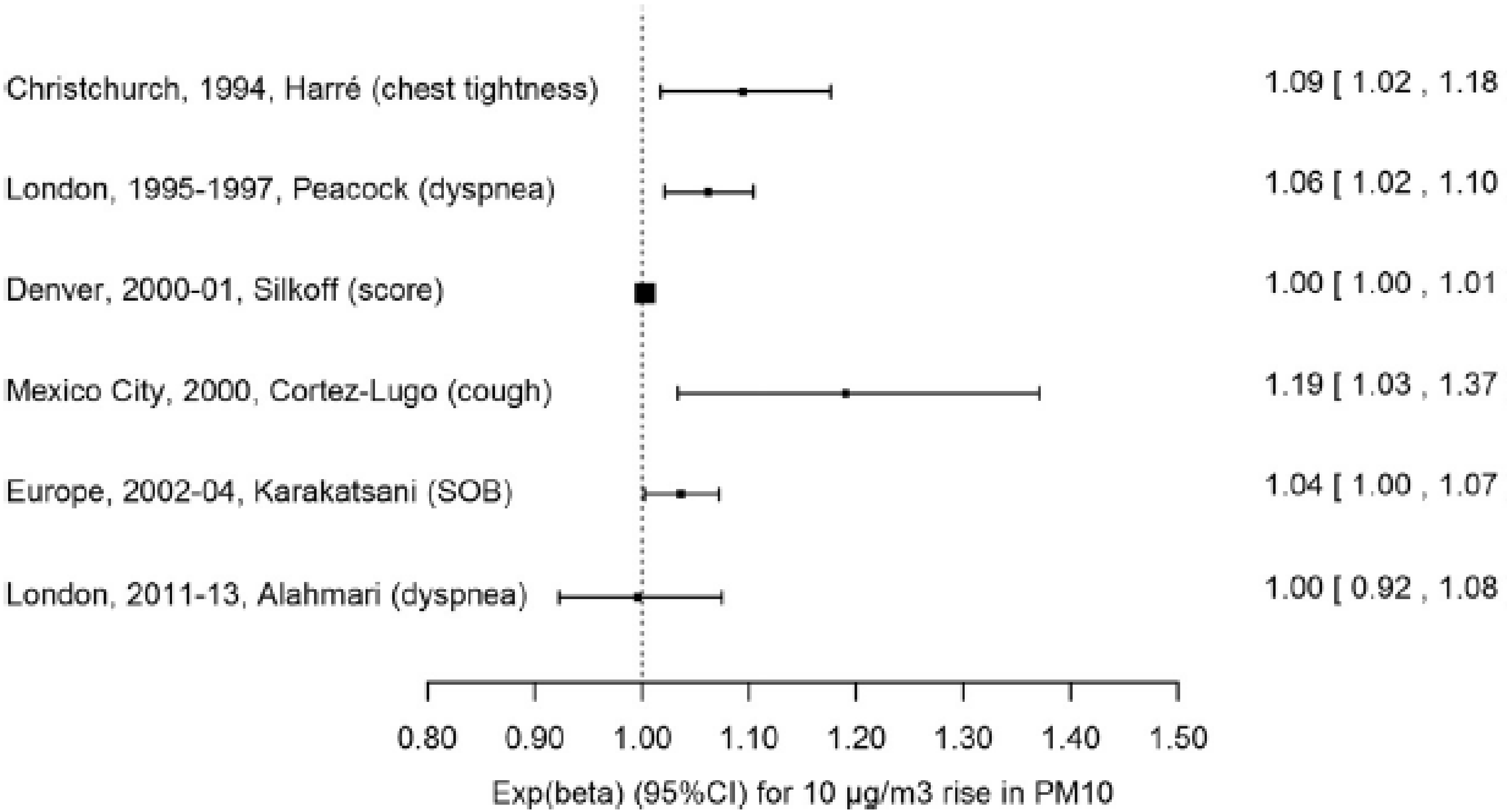
PM & COPD

Associations between an increase in PM10 and changes in FEV1 in COPD (Panel studies)



Environmental Research151(2016)458–468

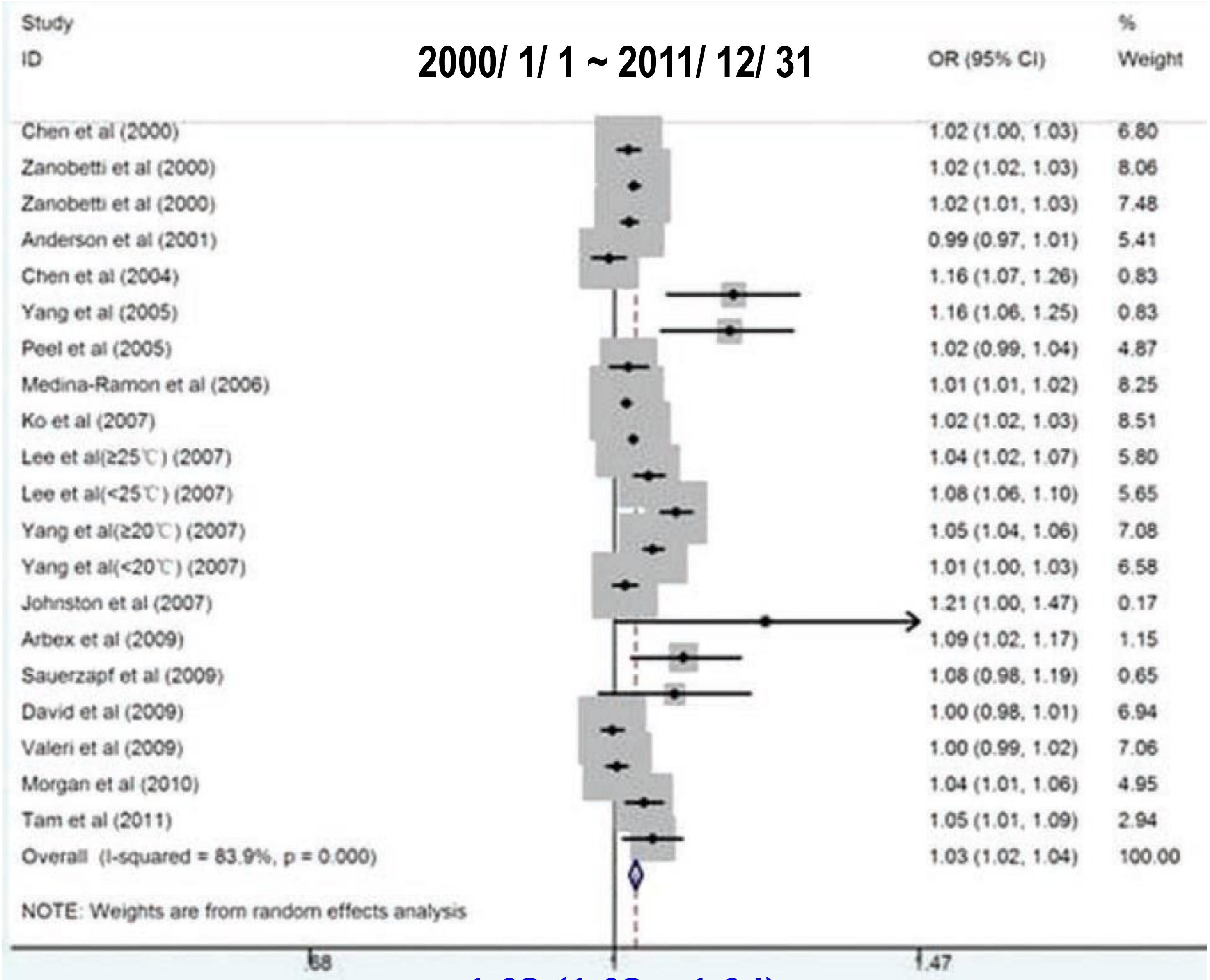
Association between an increase in PM10 and respiratory symptoms in COPD (Panel studies)



EnvironmentalResearch151(2016)458–468

Forest plot of COPD hospitalizations and PM_{10}

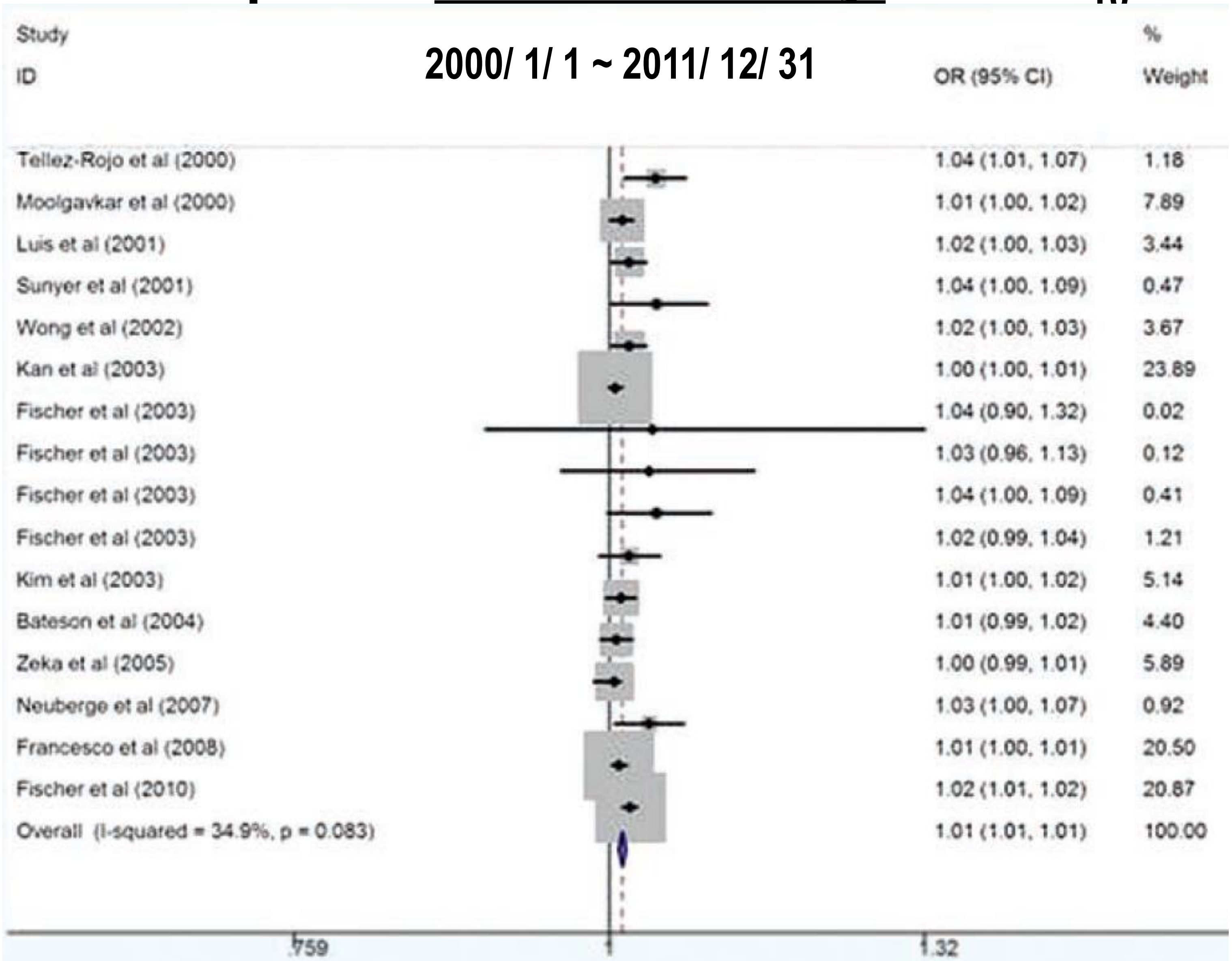
2000/ 1/ 1 ~ 2011/ 12/ 31



1.03 (1.02 – 1.04)

미세먼지/황사 건강피해 예방 및 권고지침: 호흡기질환 J KMA 2015 November; 58(11): 1060-1069

Forest plot of COPD mortalilty and PM_{10}

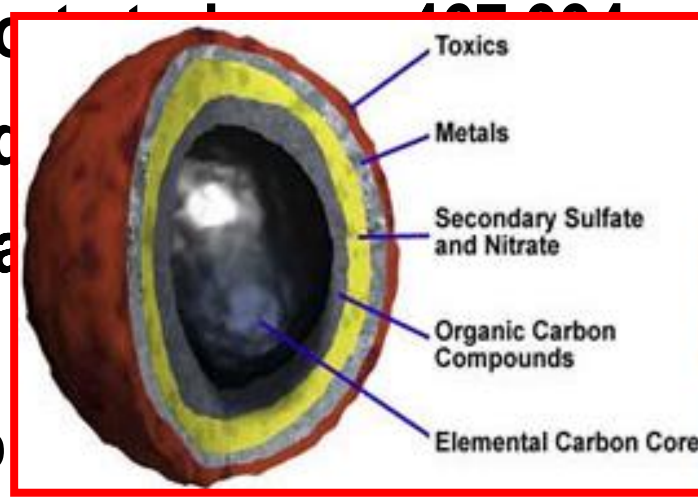


1.01 (1.01 – 1.01)

미세먼지/황사 건강피해 예방 및 권고지침: 호흡기질환 J KMA 2015 November; 58(11): 1060-1069

Associations of Ambient Air Pollution with COPD **Hospitalization** and **Mortality**

- **Population-based coho**
- **5-year exposure perioc**
- **In British Columbia, Ca lth insurance program**



RR FOR COPD HOSPITALIZATION AND QR ELEVATION

	Black Carbon ($0.97 \times 10^{-5}/m$)*	$PM_{2.5}$ ($1.58\ \mu g/m^3$)*
Hospitalization		
Model 1: unadjusted single pollutant model	1.14 (1.10–1.17)	1.15 (1.11–1.19)
Model 2: + age, sex, SES	1.06 (1.02–1.09)	1.02 (0.98–1.06)
Model 3: + asthma, diabetes, CHD, HHD	1.06 (1.02–1.09)	1.02 (0.98–1.06)
Model 4: + two other pollutants†	1.06 (1.02–1.10)	1.02 (0.98–1.07)
Mortality		
Model 1: unadjusted single pollutant model	1.17 (1.09–1.24)	1.17 (1.08–1.26)
Model 2: + age, sex, SES	1.07 (1.00–1.14)	1.02 (0.94–1.10)
Model 3: + asthma, diabetes, CHD, HHD	1.07 (1.00–1.14)	1.02 (0.94–1.10)
Model 4: + two other pollutants†	1.07 (1.00–1.14)	1.00 (0.92–1.09)

* Interquartile range.

AJRCCM 2013, 187, 721–727

PM and COPD prevalence & incidence

European Study of Cohorts for Air Pollution Effects (**ESCAPE**), **4 cohort studies**
(ECRHS, NSHD, SALIA, SAPALDIA)

- 3692 subjects with PM measurements
- Baseline assessment years; 1985–1999 and follow-up years ; 2001–2010

Exposure[i]	Prevalence of COPD all stages			Incidence of COPD all stages		
	aOR[¶] (95% CI)	I^2	p-value (het.)	aOR[+] (95% CI)	I^2	p-value (het.)
NO_2	1.07 (0.91–1.26)	24.1	p=0.266	1.05 (0.89–1.23)	0.0	p=0.789
NO_x	1.07 (0.96–1.21)	0.0	p=0.857	1.05 (0.89–1.23)	0.0	p=0.602
PM10	1.04 (0.71–1.53)	0.0	p=0.588	1.10 (0.70–1.73)	0.0	p=0.855
PM2.5	0.95 (0.47–1.90)	46.6	p=0.132	1.06 (0.73–1.53)	0.0	p=0.645
PM2.5(abs)	1.02 (0.69–1.52)	0.0	p=0.393	1.06 (0.67–1.67)	0.0	p=0.703
PMcoarse	0.84 (0.33–2.10)	7.0	p=0.358	0.18 (0.01–5.18)	95.2	p=0.000

associations are presented for increments in exposure: 5 ug/m^3 for PM2.5, 10 ug/m^3 for PM10

Eur Respir J 2014; 44: 614–626

Association of **incident COPD** with 4-year cumulative average particulate matter (PM)

- **Association between long-term exposure to PM and incident cases of COPD**
- **121,701 female nurses, from 1992 to 2000 in the US Nurses' Health Study**

Hazard ratios and 95% confidence intervals

	Full cohort		Never-smokers		Current smokers	
	Basic[+]	Adjusted[§]	Basic[+]	Adjusted[§]	Basic[+]	Adjusted[§]
COPD						
Modelled PM fraction						
PM_{10}	0.92 (0.76–1.11)	0.91 (0.76–1.10)	1.12 (0.69–1.81)	1.14 (0.70–1.87)	0.83 (0.63–1.08)	0.87 (0.67–1.13)
$PM_{2.5}$	0.87 (0.61–1.23)	0.93 (0.66–1.31)	1.17 (0.47–2.90)	1.23 (0.50–3.06)	0.72 (0.45–1.15)	0.82 (0.51–1.31)
$PM_{10-2.5}$	0.88 (0.64–1.21)	0.83 (0.60–1.14)	1.19 (0.54–2.65)	1.22 (0.54–2.76)	0.78 (0.50–1.22)	0.80 (0.51–1.25)
Distance to road[f] m						
50–199	1.07 (0.83–1.38)	0.98 (0.76–1.27)	1.22 (0.60–2.45)	1.28 (0.63–2.59)	0.96 (0.68–1.35)	0.91 (0.64–1.29)
0–49	1.05 (0.76–1.45)	0.96 (0.69–1.32)	0.65 (0.22–1.92)	0.67 (0.23–1.98)	1.04 (0.68–1.57)	1.01 (0.66–1.55)

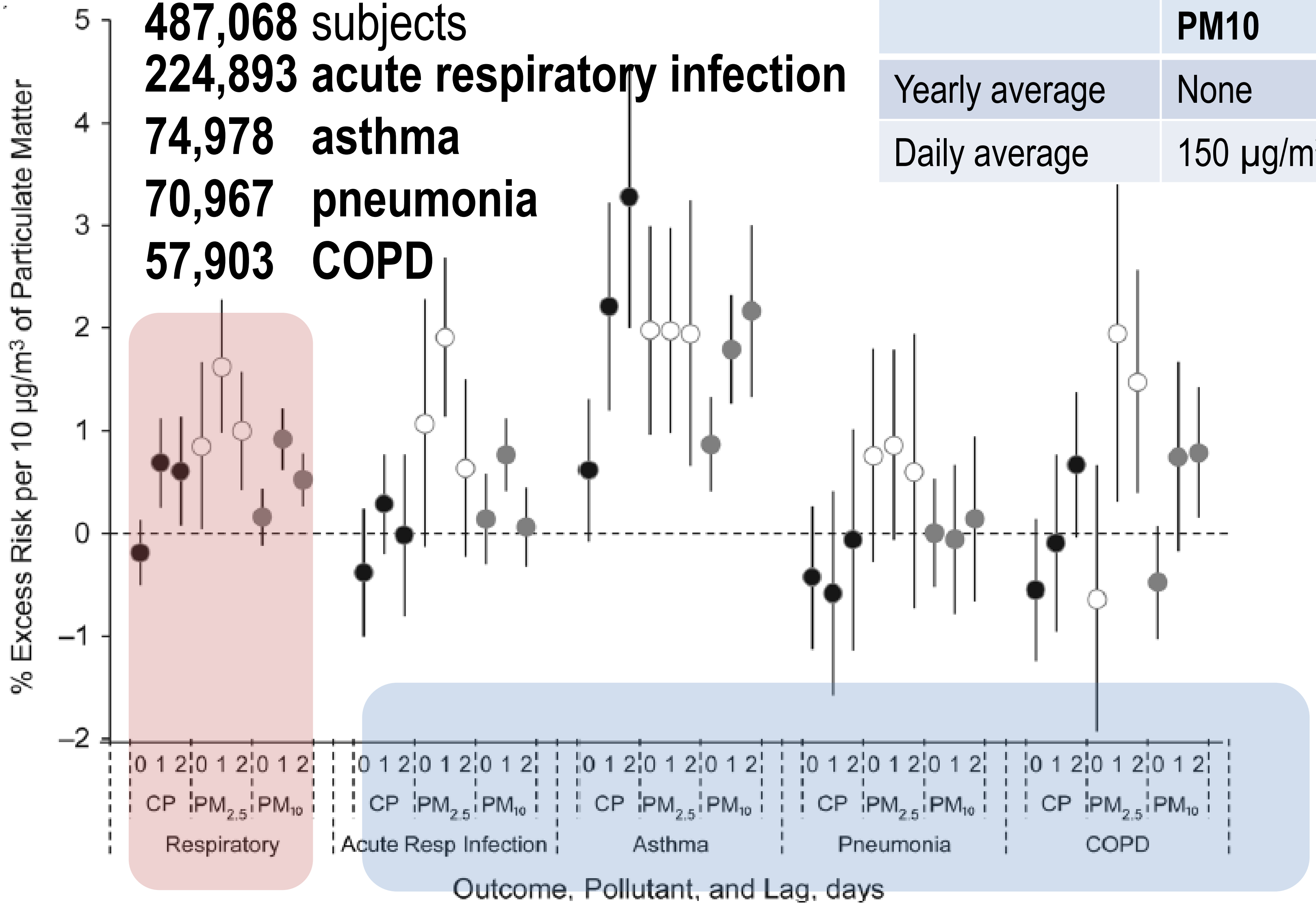
+: models adjusted for age, time period, and geographic region. §: additionally adjusted for body mass index, alcohol consumption, physical activity, census-tract median household income, Western dietary pattern

Eur Respir J 2016; 48: 921–924

Effect estimates for different particulate matter sizes on ER visits in 35 California counties, 2005–2008

487,068 subjects
224,893 acute respiratory infection
74,978 asthma
70,967 pneumonia
57,903 COPD

	PM10	PM2.5
Yearly average	None	12 μg/m3
Daily average	150 μg/m3	35 μg/m3



Am J Epidemiol. 2013;178(1):58–69

Percentage changes in cause-specific respiratory ER visit associated with a 10 μg/m³ increase in PM2.5

- from Jan 1 to Dec 31, 2013, in Beijing
- 92,464 respiratory emergency visits

	PM10	PM2.5
Yearly average	70 μg/m³	35 μg/m³
Daily average	150 μg/m³	**75 μg/m³**

미세먼지에 대한 WHO 권고기준과 잠정목표

구분	PM2.5(㎍/㎥)		PM10(㎍/㎥)		각 단계별 연평균 기준 설정시 건강영향
	연평균	일평균	연평균	일평균	
잠정목표 1	35	75	70	150	권고기준에 비해 사망 위험률이 약 15% 증가 수준
잠정목표 2	25	50	50	100	잠정목표 1보다 약 6%(2~11%) 사망위험률 감소
잠정목표 3	15	37.5	30	75	잠정목표 2보다 약 6%(2~11%)의 사망위험률 감소
권고기준	10	25	20	50	심폐질환과 폐암에 의한 사망률 증가가 최저 수준

PLoS ONE 11(4):2016

Particulate matter is associated with sputum culture conversion in patients with culture-positive tuberculosis

- 389 subjects were recruited from a hospital in Taiwan from 2010 to 2012
- 245 culture-positive TB subjects , 144 controls non TB

The estimated ORs (95% CI) for the risk of TB associated with 1-unit increase in 1 year average

Variables	Concentration Mean ± SD	TB OR (95% CI)[a]
PM_{10}, μg/m^3	48.7±7.0	1.04 (1.01–1.08)*
SO_2, ppb	3.2±0.8	1.24 (0.90–1.69)
NO_2, ppb	21.6±3.2	1.03 (0.96–1.10)
CO, ppm	0.6±0.1	2.32 (0.19–27.9)
O_3, ppb	25.7±1.8	1.02 (0.89–1.14)

Notes: [a]Adjusted for age, sex, and smoking. *$P<0.05$.
Abbreviations: CI, confidence interval; CO, carbon monoxide; NO_2, nitrogen dioxide; O_3, ozone; OR, odds ratio; SD, standard deviation; SO_2, sulfur dioxide; TB, tuberculosis.

HR for days of sputum cultures conversion in subjects with TB-positive cultures

PM10, μg/m^3	Sputum culture conversion HR (95% CI)[a]
<50 μg/m^3	1
≥50 μg/m^3	1.28 (1.07–1.84)*

Notes: [a]Adjusted for age, sex, smoking, and CXR grading. *$P<0.05$.
Abbreviations: CI, confidence interval; CXR, chest X-ray; HR, hazard ratio; TB, tuberculosis.

TB patients with PM10 exposure >50 μg/m^3 may need longer standard treatment periods.

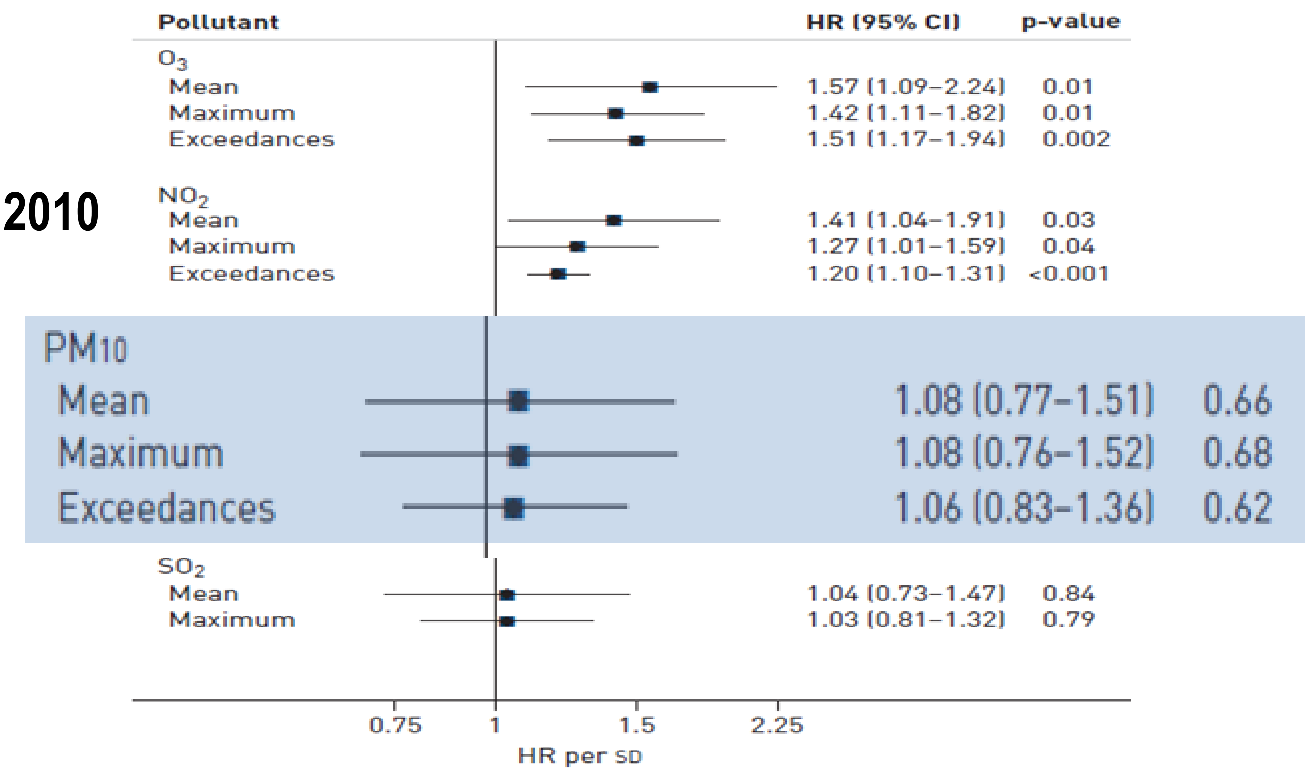
Therapeutics and Clinical Risk Management 2016

PM &
Idiopathic pulmonary fibrosis

Acute exacerbation of idiopathic pulmonary fibrosis associated with air pollution exposure

Kerri A. Johannson, Eric Vittinghoff, Kiyoung Lee, John R. Balmes, Wonjun Ji, Gilaad G. Kaplan, Dong Soon Kim and Harold R. Collard

- **505 patients with IPF**
- **from Jan.1, 2001 to Dec. 31,2010**



Mean levels, maximum levels and number of exceedances above standards for particulate matter, (PM10) over a 6-week period were not associated with significantly increased risk for acute exacerbation of IPF.

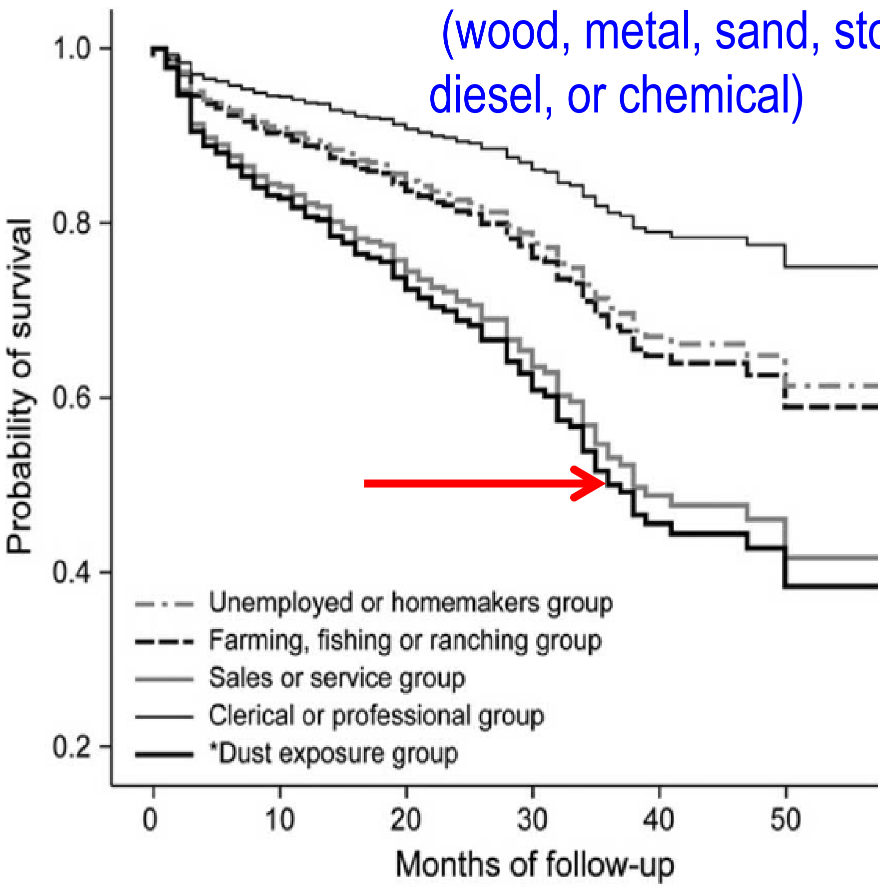
Eur Respir J 2014; 43: 1124–1131

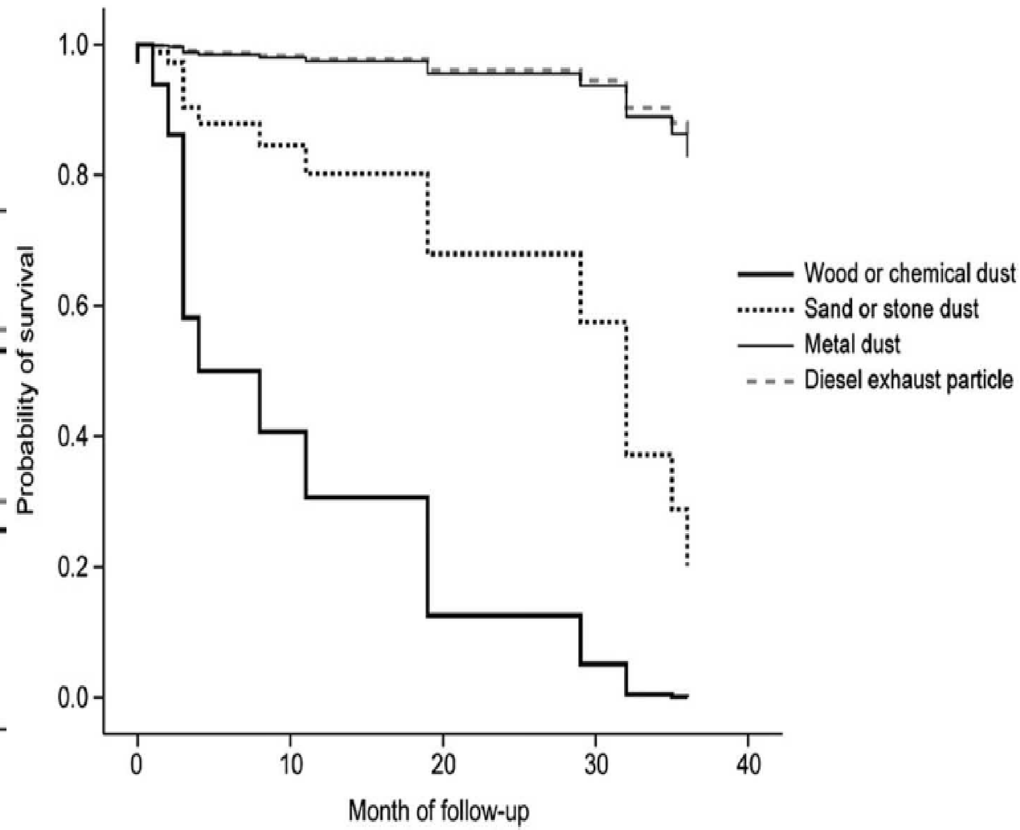
Association Between Occupational Dust Exposure and Prognosis of Idiopathic Pulmonary Fibrosis A Korean National Survey

- Diagnosed from Jan 1, 2003, to Dec 31, 2007
- 1,311 IPF patients

Dust exposure group

(wood, metal, sand, stone, diesel, or chemical)





CHEST 2015; 147(2): 465 - 474

PM & lung cancer

PM & lung cancer

- Outdoor air pollution and particulate matter (**PM**) is carcinogenic to humans (IARC, **I**nternational **A**gency for **R**esearch on **C**ancer**,** Group 1) and causes lung cancer **(IARC 2013)**
- **PM2.5** more precise than PM10; higher mutagenic species/ combustion/ deep into the lung
- For lung cancer incidence, estimates range **from 6 to 29%** increase with increments of 5-10 μg/m^3 in PM2.5 *(Environ Health Perspect. 2008; Epidemiology. 2014; Lancet Oncol. 2013)*

Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from ESCAPE project

-312,944 cohort members, Follow-up (mean 12.8 years), 2095 incident lung cancer cases

	Increase	Number of cohorts	HR (95% CI)		
			Model 1*	Model 2†	Model 3‡
PM_{10}	10 μg/m³	14	1·32 (1·12–1·55)	1·21 (1·03–1·43)	1·22 (1·03–1·45)
$PM_{2.5}$	5 μg/m³	14	1·34 (1·09–1·65)	1·17 (0·95–1·45)	1·18 (0·96–1·46)
PM_{coarse}	5 μg/m³	14	1·19 (0·99–1·42)	1·08 (0·89–1·31)	1·09 (0·88–1·33)
$PM_{2.5absorbance}$	10^{-5}/m	14	1·25 (1·05–1·50)	1·09 (0·87–1·37)	1·12 (0·88–1·42)
NO_2	10 μg/m³	17	1·07 (1·00–1·14)	0·99 (0·93–1·06)	0·99 (0·93–1·06)
NOx	20 μg/m³	17	1·08 (1·02–1·14)	1·01 (0·95–1·06)	1·01 (0·95–1·07)

*Model 1: age (timescale in Cox model), sex, calendar time.
†Model 2: model 1 + smoking status, smoking intensity, square of smoking intensity, smoking duration, time since quitting smoking, environmental tobacco smoke, occupation, fruit intake, marital status, education level, and employment status.
‡Model 3: model 2 + area-level socioeconomic status.

Lancet Oncol 2013; 14: 813–22

Relationship between a 10-μg/m³ change in PM2.5 and PM10 and histological cancer subtypes

	Number of cohorts	HR (95% CI) for histological cancer subtype analysis	
		PM_{10}	$PM_{2.5}$
All participants			
All lung cancers	14†	1·22 (1·03–1·45)	1·18 (0·96–1·46)
Adenocarcinomas	11‡	1·51 (1·10–2·08)	1·55 (1·05–2·29)
Squamous-cell carcinomas	7§	0·84 (0·50–1·40)	1·46 (0·43–4·90)
Participants who did not change residence			
All lung cancers	10¶	1·48 (1·16–1·88)	1·33 (0·98–1·80)
Adenocarcinomas	8||	2·27 (1·32–3·91)	1·65 (0·93–2·95)
Squamous-cell carcinomas	3**	0·64 (0·28–1·48)	0·65 (0·16–2·57)

Lancet Oncol 2013; 14: 813–22

Particulate matter air pollution components and risk for lung cancer

- 245,782 cohort members
- ESCAPE and TRANSPHORM projects
- 8 elements

Hazard ratios for lung cancer in association with exposure to elemental components of PM

Participants who did not change residence						All participants (same cohorts)
Exposure	No. of cohorts	No. of lung cancer cases	HR (95% CI)	Measure of heterogeneity		HR (95% CI)
				I^2 (%)	$p^{‡}$	
$PM_{2.5}$ Cu	10[§]	893	1.25 (1.01–1.53)	0	0.67	1.14 (0.97–1.35)
PM_{10} Cu	10[§]	893	1.14 (0.96–1.35)	16	0.30	1.08 (0.96–1.20)
$PM_{2.5}$ Fe	10[§]	893	1.08 (0.93–1.25)	0	0.63	1.08 (0.90–1.29)
PM_{10} Fe	10[§]	893	1.10 (0.94–1.28)	0	0.81	1.05 (0.92–1.20)
$PM_{2.5}$ Zn	10[§]	893	1.11 (0.88–1.39)	0	0.57	0.99 (0.83–1.17)
PM_{10} Zn	10[§]	893	1.28 (1.02–1.59)	0	0.74	1.09 (0.92–1.30)
$PM_{2.5}$ S	10[§]	893	2.05 (0.73–5.75)	57	0.01	1.47 (0.65–3.30)
PM_{10} S	10[§]	893	1.58 (1.03–2.44)	6	0.39	1.10 (0.85–1.44)
$PM_{2.5}$ Ni	6[¶]	804	1.13 (0.77–1.65)	0	0.68	1.01 (0.73–1.41)
PM_{10} Ni	9[**]	839	1.59 (1.12–2.26)	0	0.44	1.29 (0.96–1.72)
$PM_{2.5}$ V	8[††]	621	1.07 (0.71–1.61)	0	0.96	1.02 (0.70–1.49)
PM_{10} V	10[§]	893	1.12 (0.77–1.64)	0	0.47	1.01 (0.70–1.45)
$PM_{2.5}$ Si	8[‡‡]	821	1.26 (0.85–1.86)	33	0.17	1.11 (0.88–1.41)
PM_{10} Si	10[§]	893	1.13 (0.95–1.36)	0	0.54	1.02 (0.88–1.18)
$PM_{2.5}$ K	10[§]	893	1.18 (0.99–1.40)	0	0.46	1.02 (0.92–1.14)
PM_{10} K	9[**]	839	1.17 (1.02–1.33)	0	0.68	1.07 (0.96–1.18)

Environment International (2016) 66–73

Summaries & Conclusions

- Total particulate matter(PM) mass is a temporally and spatially varying **mixture of constituents** with different physical and chemical properties.
- PM exposure studies include short-term effects on **lung function, symptoms, exacerbation, hospitalizaion, death**; longterm effects on **hospitalizaion, death** in COPD .
- **Inconclusive** evidence on increased **incidence** of COPD, CB in long-term exposure to PM.

- Clear evidence regarding the relationship of **PM2.5** and PM10 to **lung cancer risk**

- **Further research with improved personal-level exposure assessment (for example, time–activity patterns) and phenotypic characterization is needed.**