

미세먼지와 천식

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호흡기·알레르기내과
장안수

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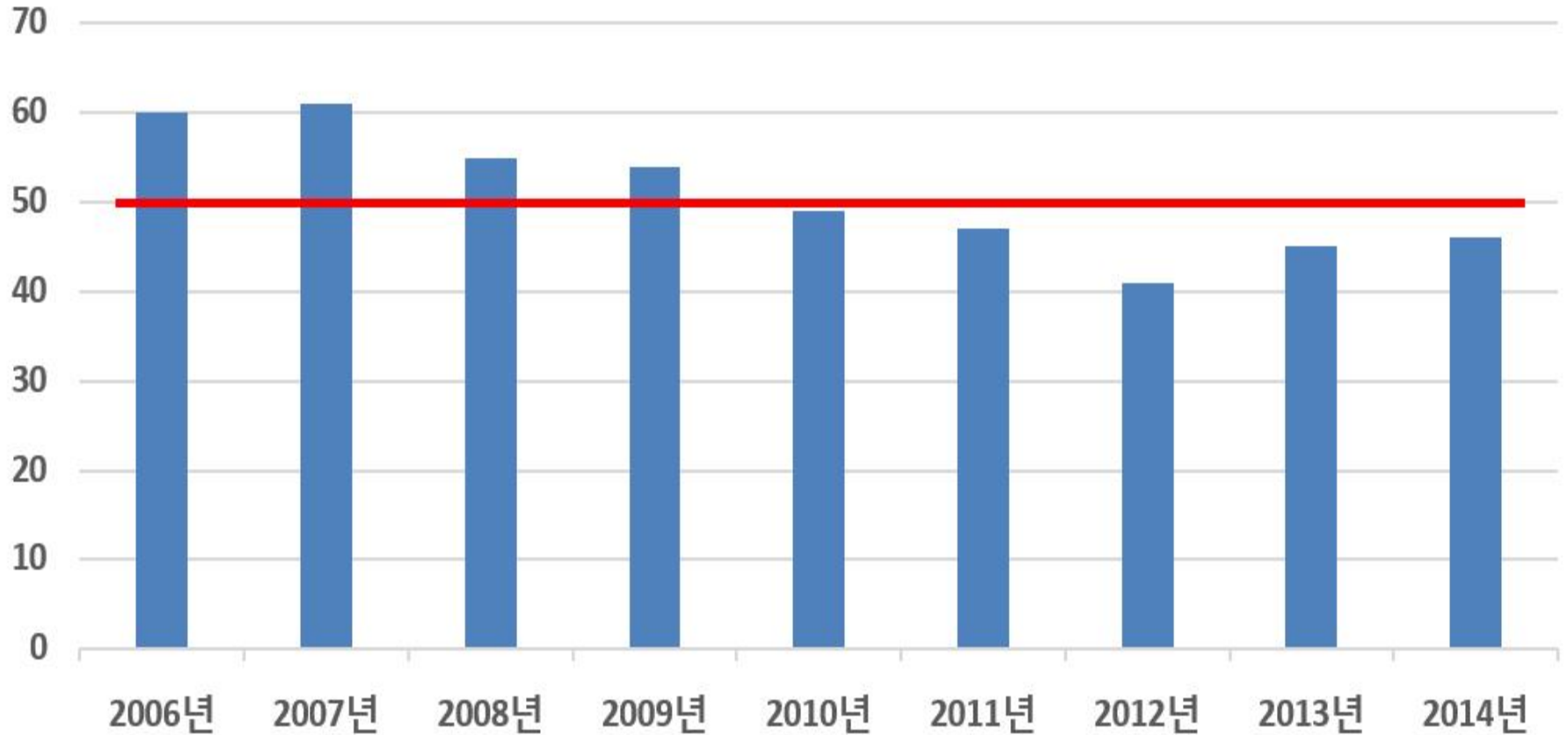


9.19-2017

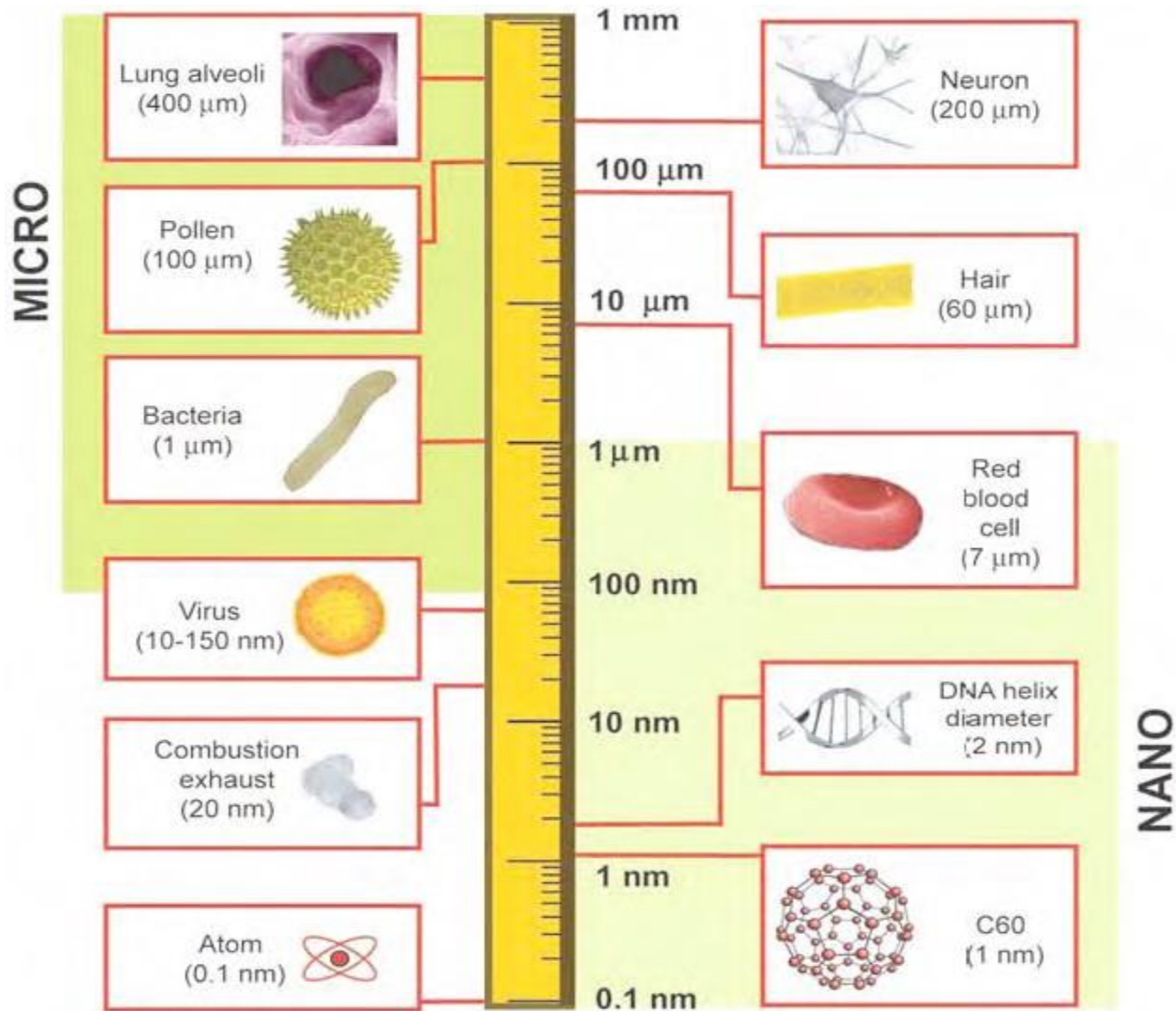
우리나라 주요 도시의 미세먼지 PM10 연평균 농도

-Air Korea

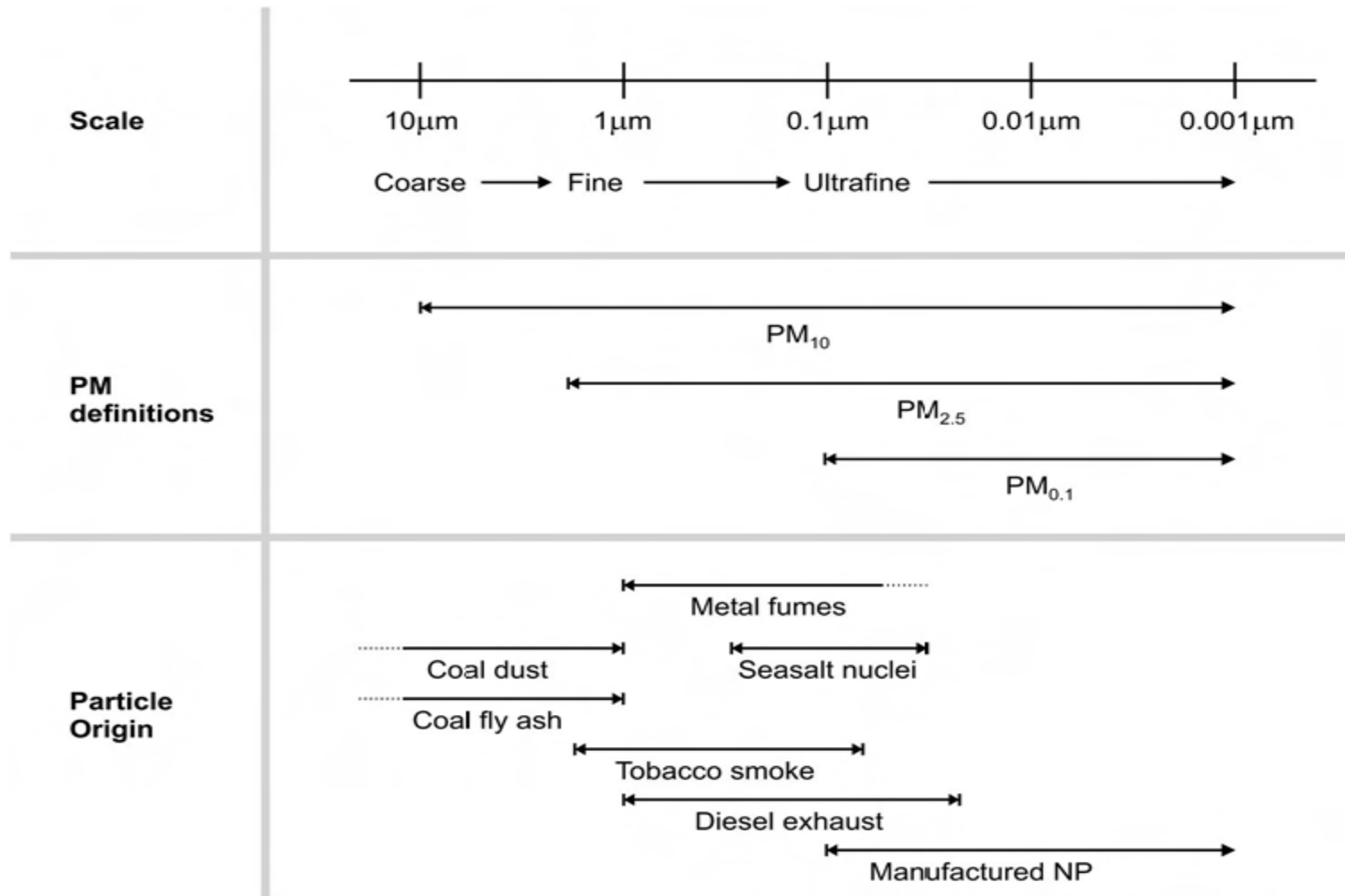
(단위 : $\mu\text{g}/\text{m}^3$)



입자의 크기



Classification of particulate matter (PM) and sources of particles



Coarse particles cover a range between 10,000 and 2,500 nanometers.

Fine particles are sized between 2,500 and 100 nanometers.

Ultrafine particles, or nanoparticles are sized between 100 and 1 nanometers.

Table 1
Comparison of coarse, fine, and ultrafine particles

Parameter	Coarse	Fine	Ultrafine
Size	2.5–10 μm	0.10–2.5 μm	<0.10 μm
Organic carbon content	+	++	+++
Metal content	+++	++	+
PAH content	+	+	+++
Source of ROS	Transition metals	PAH Quinones	PAH Quinones

Table 2
Comparison of ambient ultrafine particles (UFP) and nanomaterials

Particle type	Ambient UFP	Nanoparticle
Source	Anthropogenic	Engineered
Size	<100 nm	<100 nm
Uniformity	No	Yes
Organic chemicals	High	Low
Transition metals	High	Varies
Oxidative stress	Yes	Varies
Toxicity	Yes	Varies
Portal of entry	Lung	Lung, skin, blood



Airborne particles

Fine particles (<2.5 μ m diameter)

30-50%: water soluble salts

(ammonium sulfate, ammonium bisulfate, levoglucosan, ammonium nitrate, sodium chloride)

10-40%: carbonaceous material

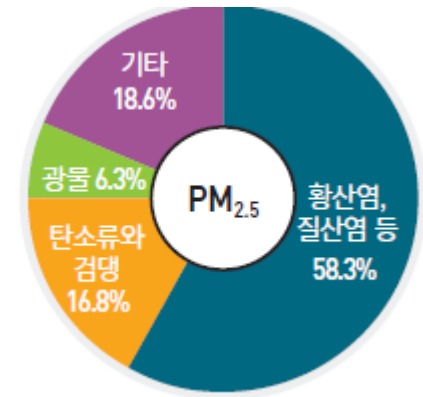
5-20%: trace element

Coarse particles (>2.5 μ m diameter)

5-20%: water soluble salts

5-20%: carbonaceous material

30-50%: oxides and minerals (silicates, calcium, aluminum, magnesium hydroxides, calcium carbonate, calcium sulfate, iron oxides)



환경부, 2016



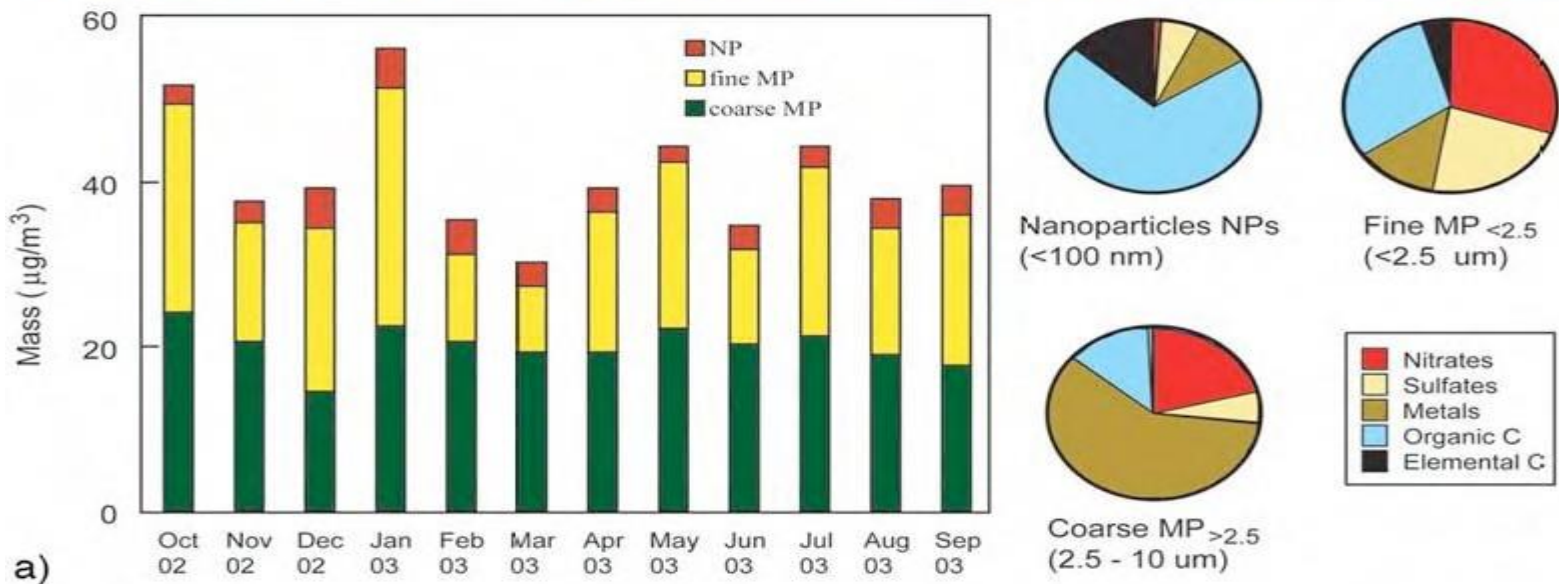


Figure 23. a) Los Angeles smog. b) Size distribution and composition of particulate matter over Los Angeles during 2002-2003. NP - nanoparticles, MP - microparticles.

Sources of particles

Nanoparticles are abundant in nature, as they are produced in many natural processes, including photochemical reactions, volcanic eruptions, forest fires, and simple erosion, and by plants and animals, e.g. shed skin and hair.

Though we usually associate air pollution with human activities –cars, industry, and charcoal burning, natural events such as dust storms, volcanic eruptions and forest fires can produce such vast quantities of nanoparticulate matter that they profoundly affect air quality worldwide.

The aerosols generated by human activities are estimated to be only about 10% of the total, the remaining 90% having a natural origin.



5~6월 발생 미세먼지, 34% 중국발

한-미 협력 국내 대기질
공동 조사(KORUS-AQ) 결과
2016년 5월2일 ~ 6월12일.
서울 올림픽공원 측정 미세먼지(PM2.5)

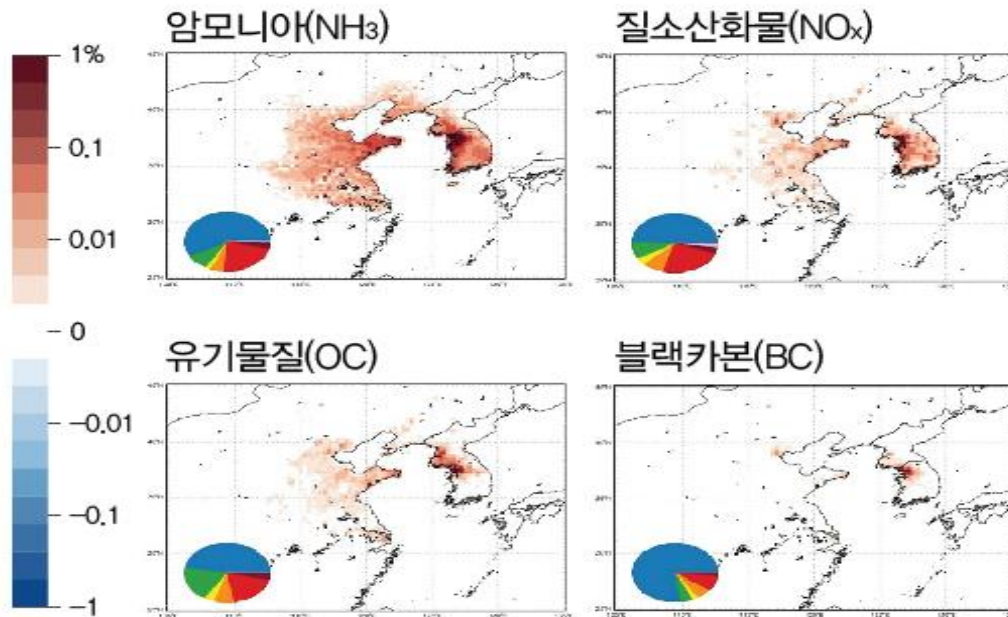


국외 48%

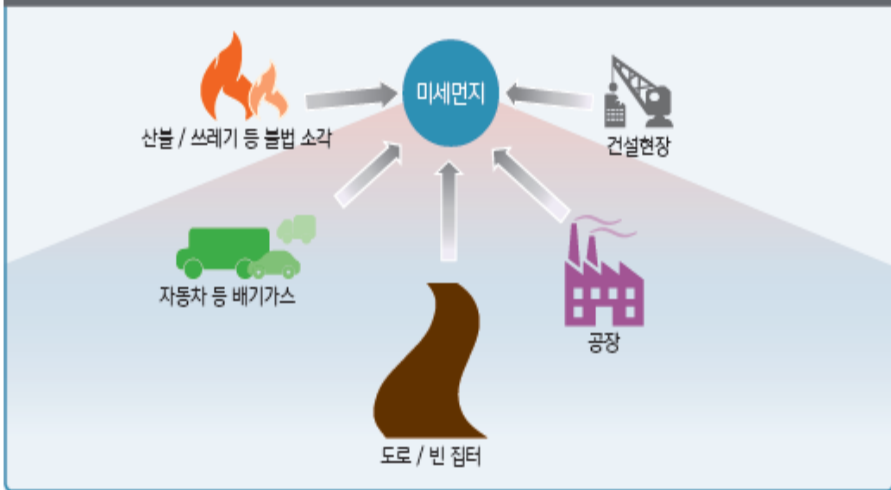


미세먼지 생성 전구물질 분석 결과
성분별 국내외 기여율

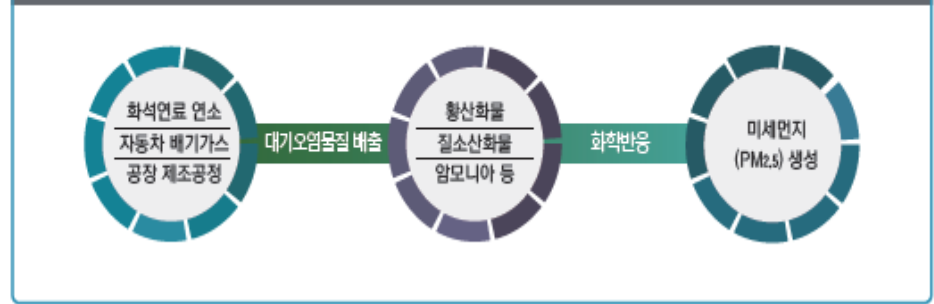
- 국내
- 북한
- 일본
- 압록강 북서쪽 중국 내륙(랴오둥반도)
- 베이징
- 산둥반도
- 상하이



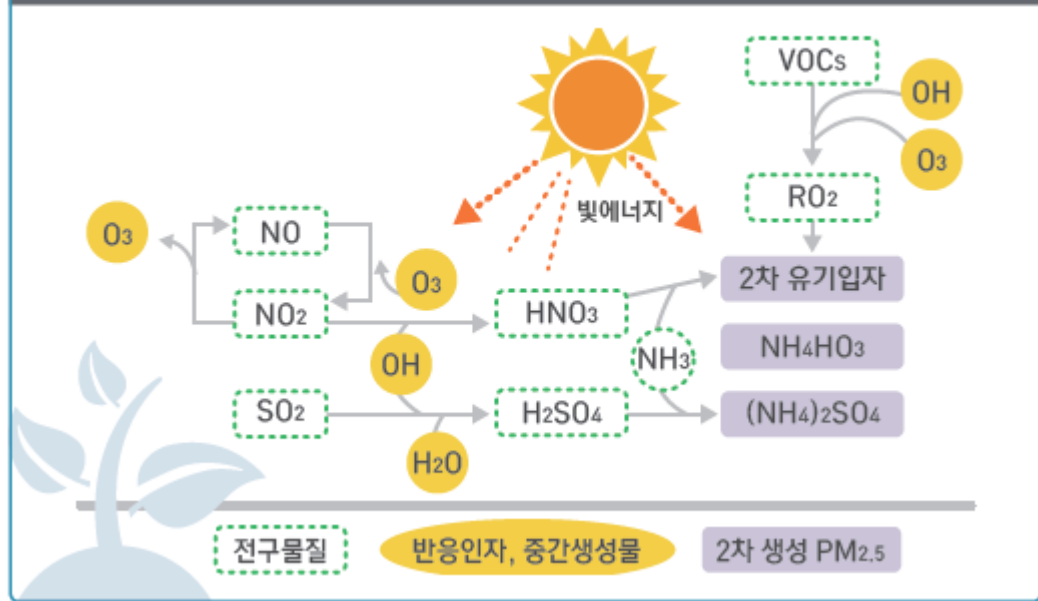
미세먼지의 1차 발생원



미세먼지의 2차 발생원



미세먼지(PM_{2.5}) 2차 생성과정



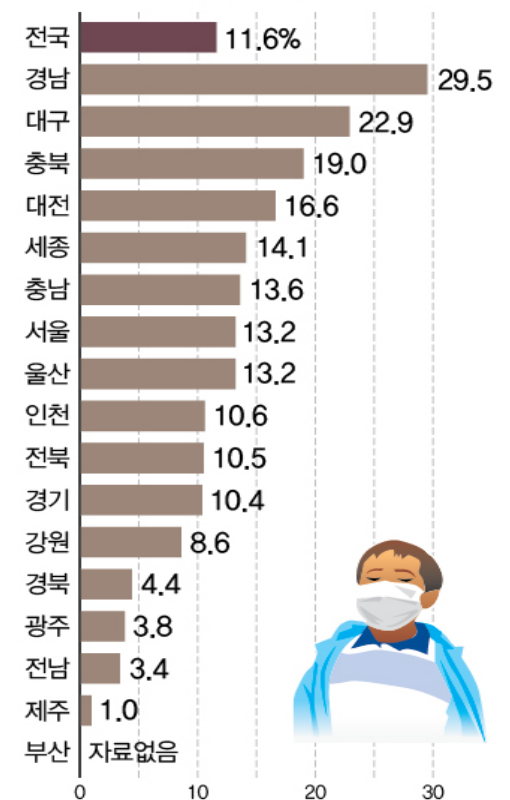
Average hour spent per day in various locations

Location	Employed men	Employed Women	Housewives
At home	13.4h (55.8%)	15.4h (64/2%)	20.5h (85.4%)
At work	6.7h (27.9%)	5.2h (21.7%)	- (0%)
In transit	1.6h (6.7%)	1.3h (5.4%)	1.0h (4.2%)
Outside	0.7h (2.9%)	0.3h (1.3%)	0.4h (1.7%)
Inside other structures	1.6 (6.7%)	1.8 (7.5%)	2.1 (8.8%)

미세먼지 농도에 따른 4등급

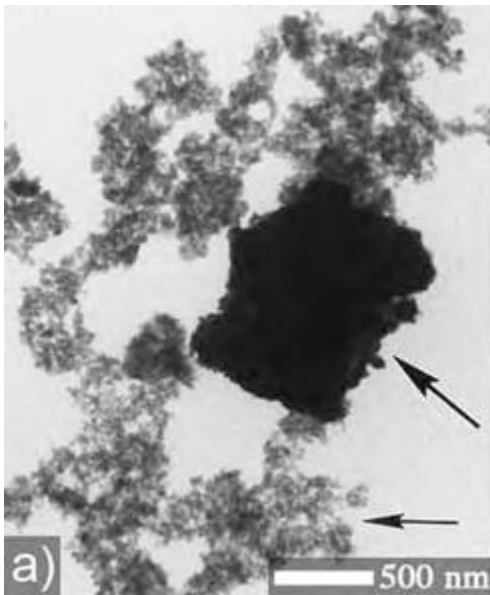


2016년 전국 11,659개 초·중·고교(분교 포함)
미세먼지 농도 80µg/m³ 이상

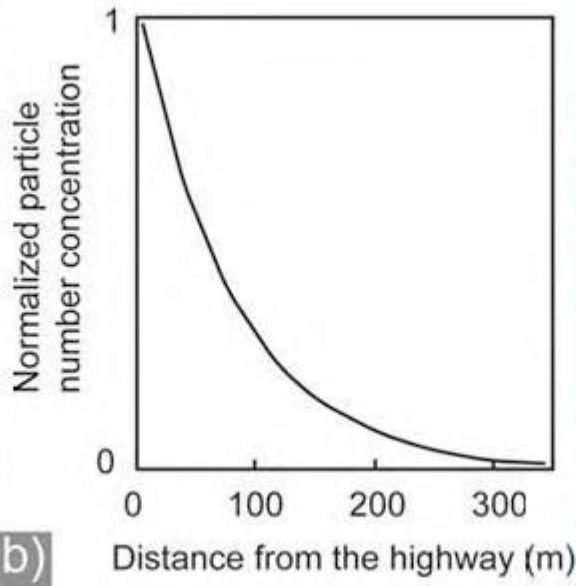


자료/ 더불어민주당 김병욱 의원, 교육부

연합뉴스



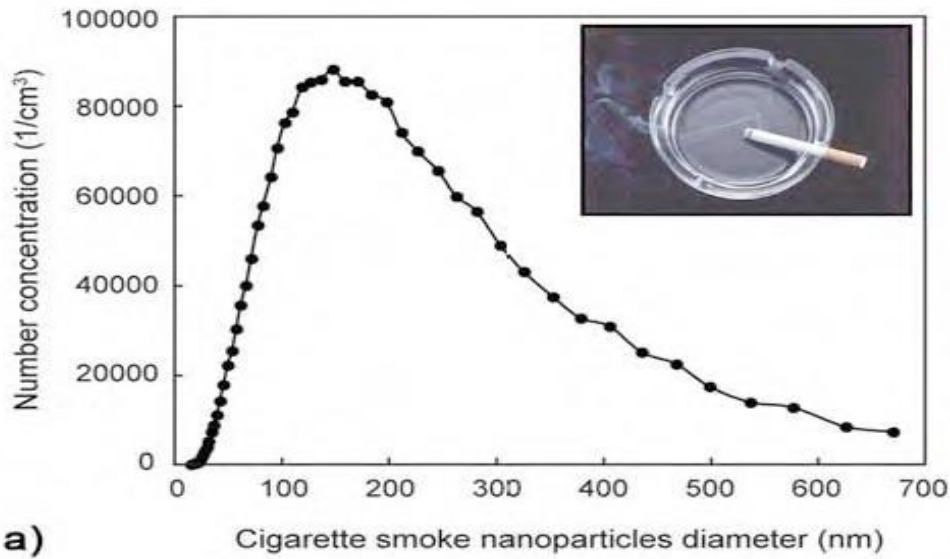
a)



b)



c)



a)



b)

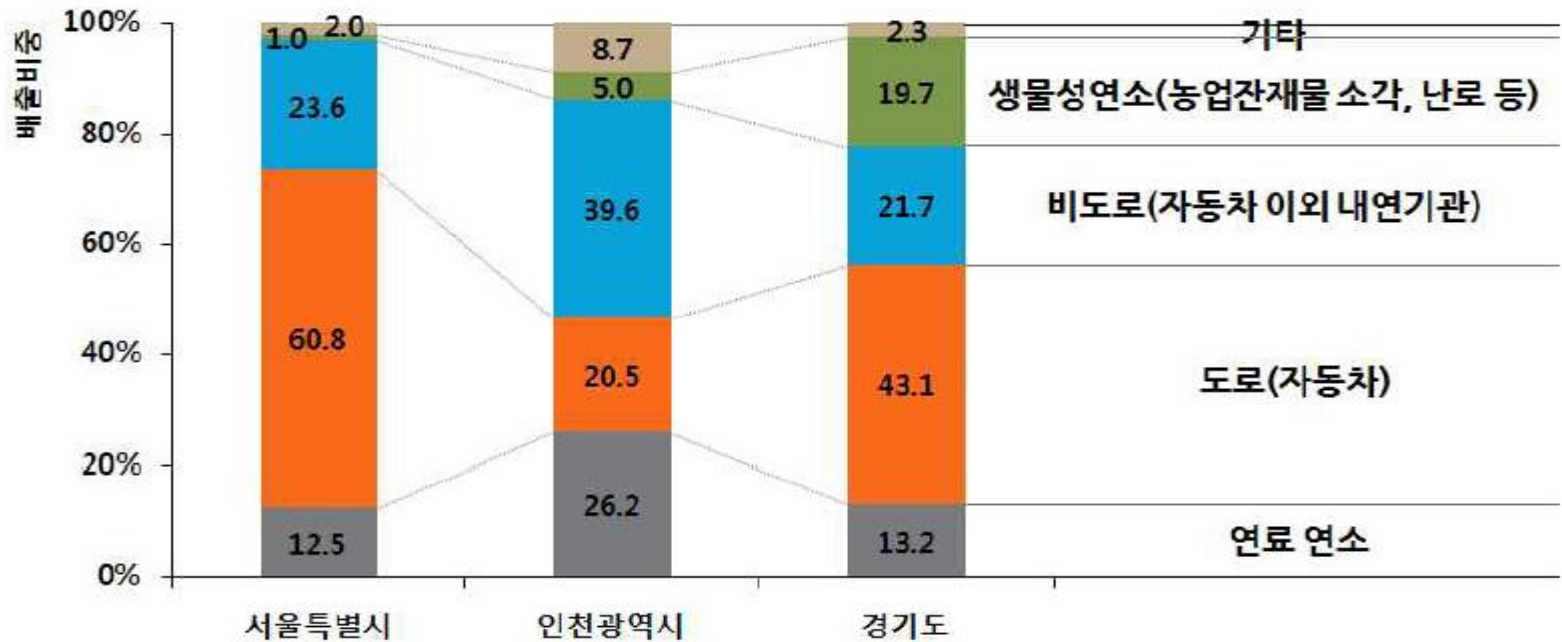
특집 다큐멘터리 - 아이들이 위험하다. 미세먼지의 역습 월 22:45 ~ 23:35

특집 다큐멘터리 - 아이들이 위험하다. 미세먼지의 습격

2017년 15% 대량증 구급대가 변신을 통해 비로 갈라 추위에 VIX를 이용할 수 있습니다. 발매일권 구매하기 저음 이용권 구매하기

EBS

<2010년 수도권 배출원별 PM10 배출 비중>



Company Name

<미세먼지의 주요 배출원>



발전소 등 산업시설 굴뚝



경유자동차 매연

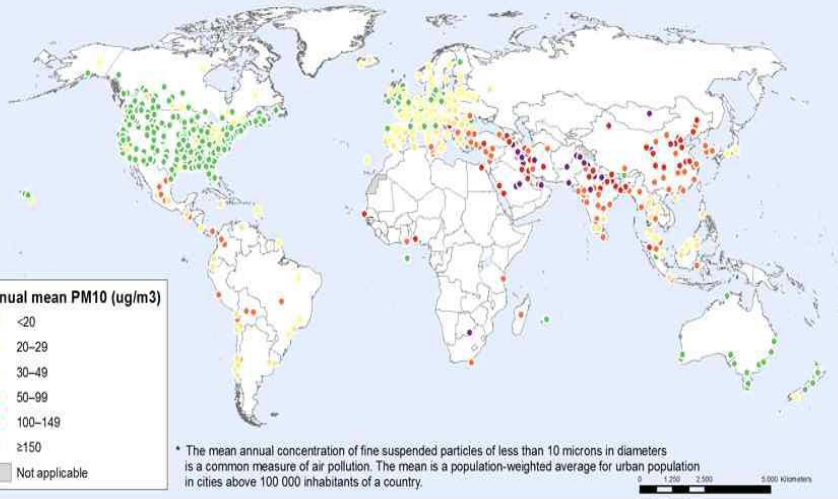


생활폐기물 등 소각

자료 : 환경일보(<http://www.hkbs.co.kr>).

<2003 ~ 2010 전 세계 1,100개 도시의 미세먼지 오염 지도>

Exposure to particulate matter with an aerodynamic diameter of 10 μm or less (PM10) in 1100 urban areas*, 2003-2010

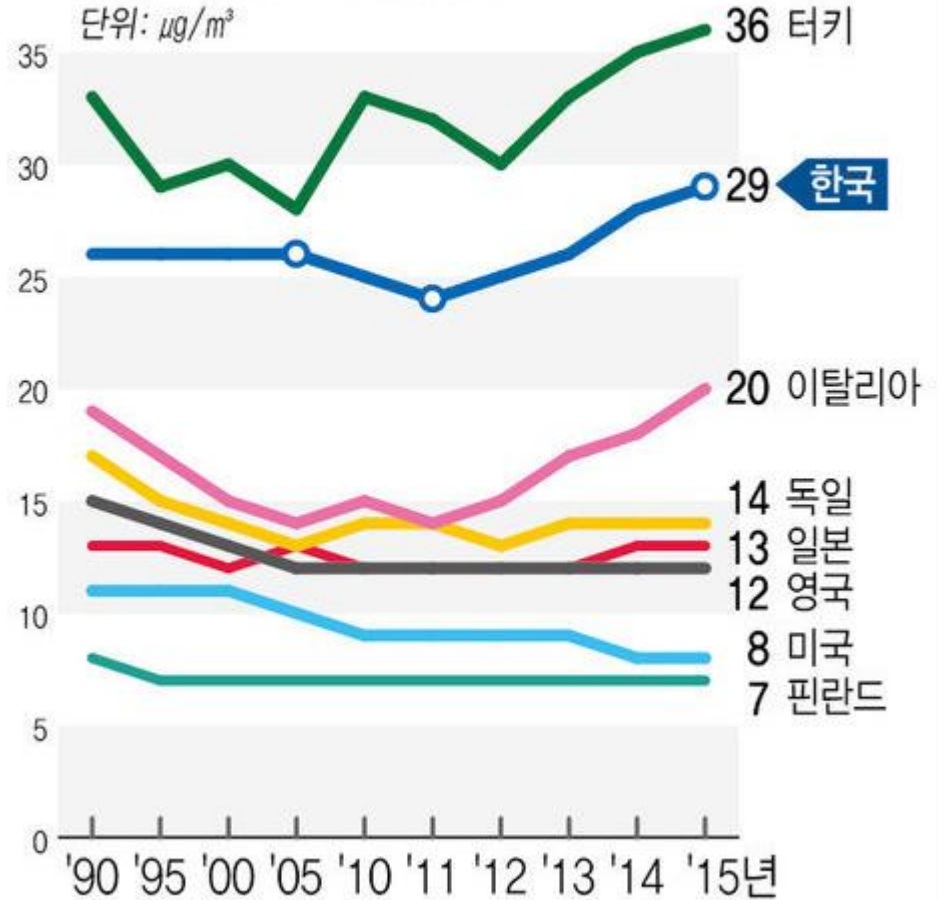


자료 : 세계보건기구(WHO)

OECD 주요국 미세먼지 농도

연평균 미세먼지(PM2.5) 농도

단위: $\mu\text{g}/\text{m}^3$



Health effects and biologic markers of response associated with air pollution

Excess cardiorespiratory mortality

death from lung or heart disease in excess of number expected

Increased health care utilization

increased hospitalization, physician visits, emergency department visits

Asthma exacerbations :

increased physician visits, medication use, decreased PEFr

Increased respiratory illness

increased respiratory infections, physician visits, episodic symptoms

Increased respiratory symptoms

Decreased lung function: spirometry, peak flow rates, airways resistance

Increased airways reactivity

altered response to challenge with methacholine, carbachol, histamine, cold air

Lung inflammation

influx of inflammatory cells, mediators, proteins

Altered host defense :

altered mucociliary clearance, macrophage function, immune response



Environmental and occupational exposure to toxic substances

1. Metals and other dusts: Beryllium. Lead. Cobalt. Cadmium. Aluminum. Nickel and chromium. Manganese. Iron. Organic dust, Silica. Coal and coal ash, Asbestos, Polymer fumes.
2. Carcinogens and poorly soluble (durable) particles

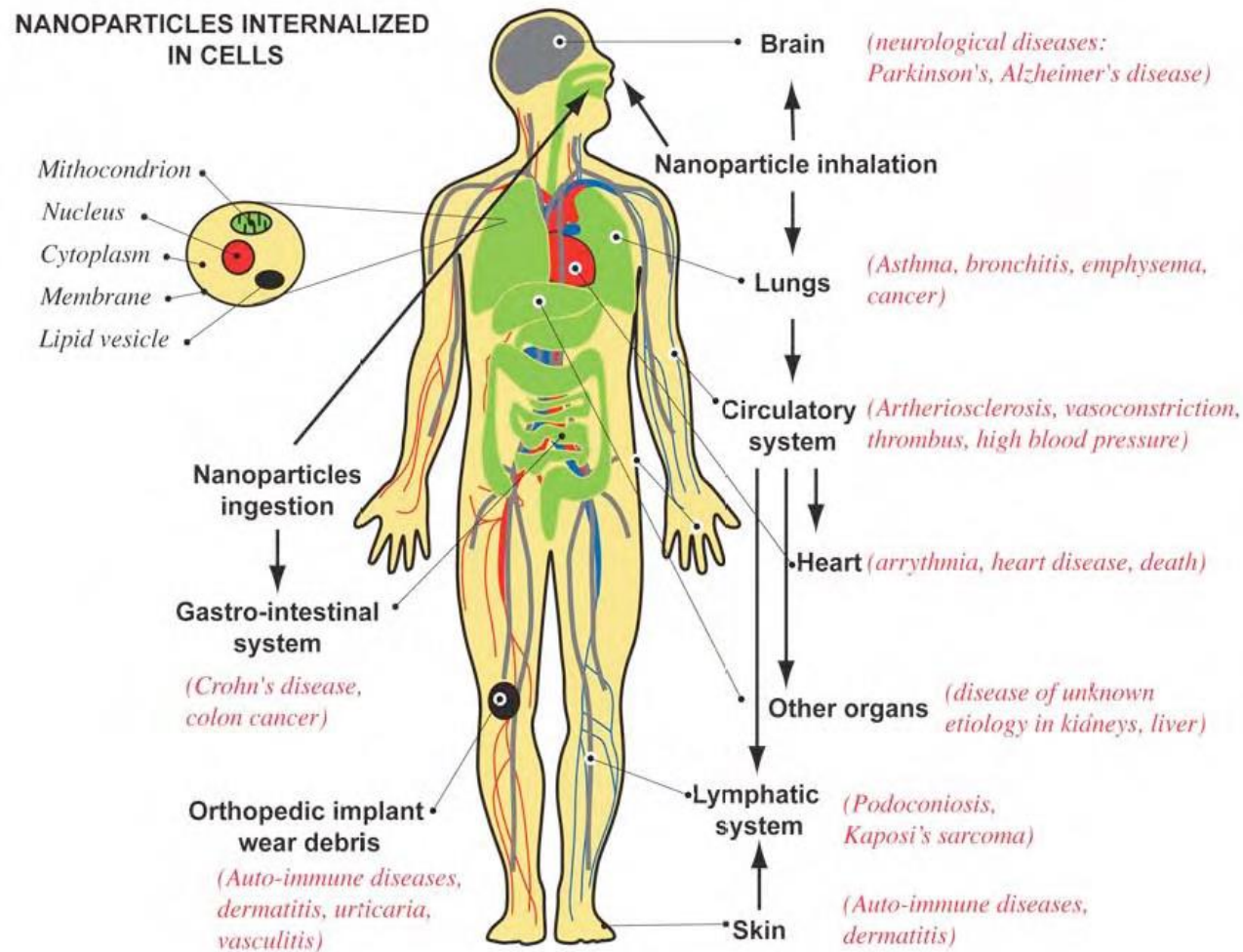
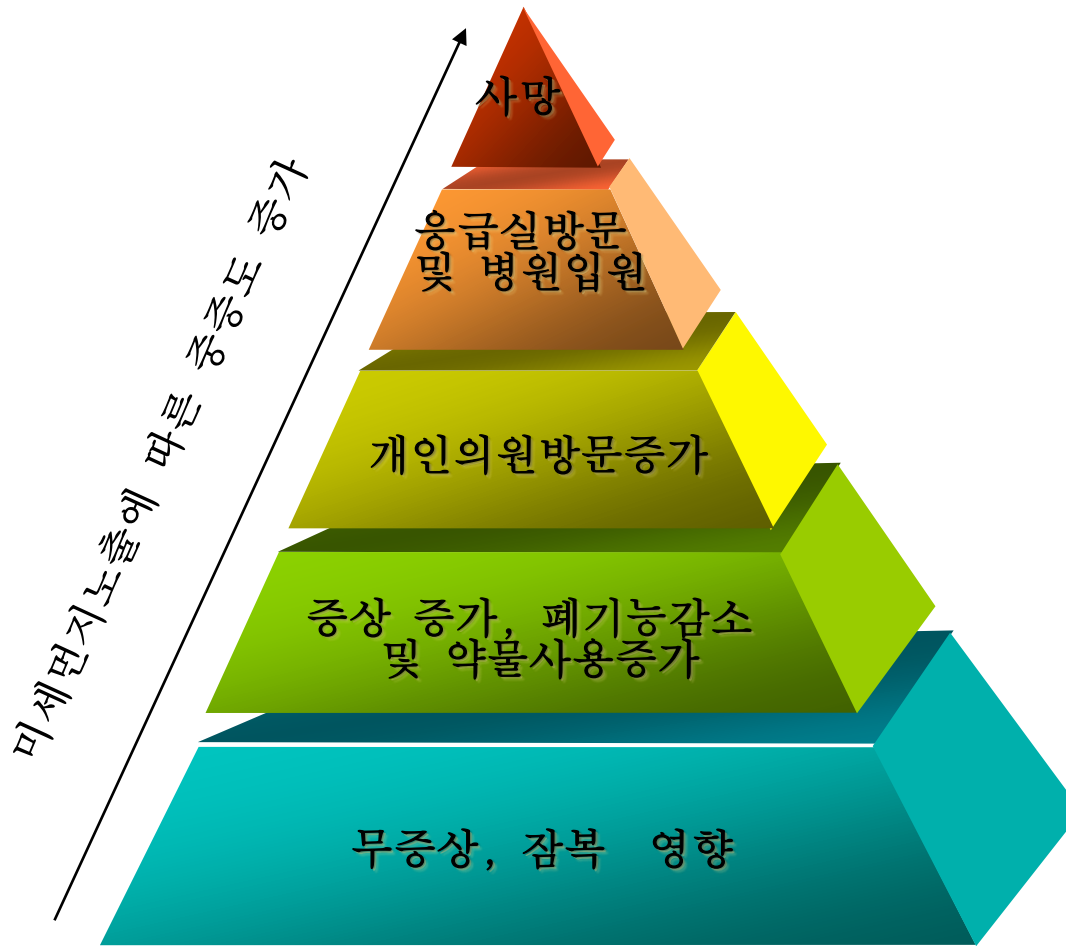


Figure. Schematics of human body with pathways of exposure to nanoparticles, affected organs, and associated diseases from epidemiological, in vivo and in vitro studies.



미세먼지 노출 인구

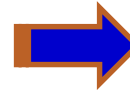




미세먼지 등 공기유해물질



천식



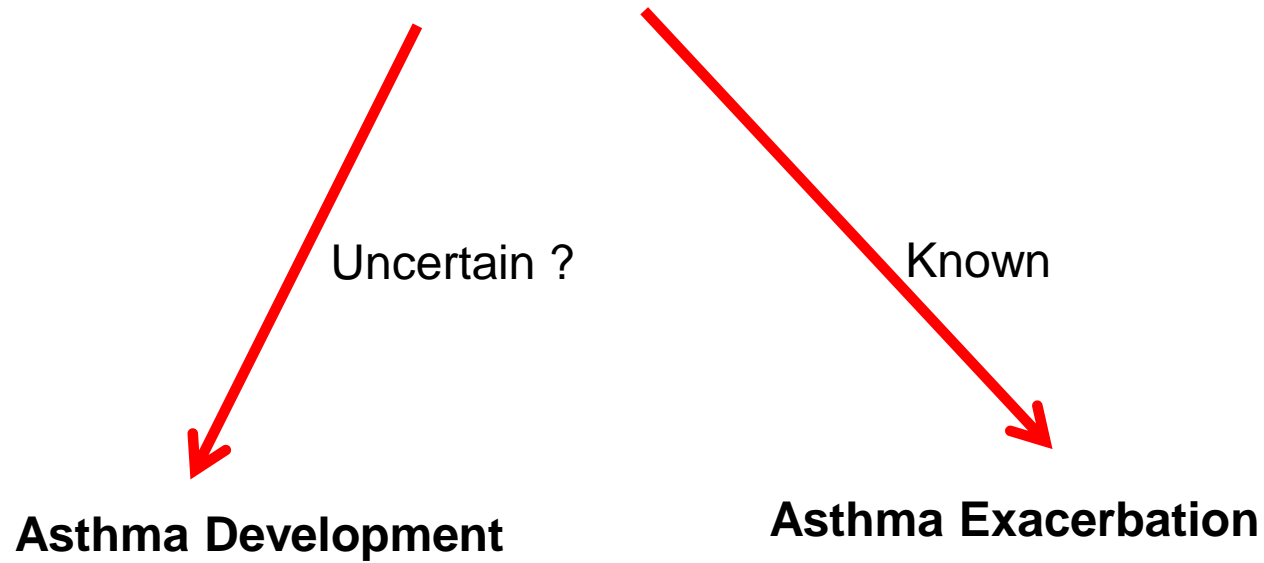
만성폐쇄성
폐질환



폐암



Particulate Matter



Epidemiologic evidence of a relationship between airway hyperresponsiveness and exposure to polluted air

Table 1. Characteristics of subjects

	Polluted area with chemical factory	Coastal area near chemical factory	Rural area
Age	10.96±0.04	10.95±0.05	11.02±0.06
Sex (M/F)	147/156	94/102	81/90
Height (cm)	136.9±0.40	136.7±0.51	137.1±0.53
Weight (kg)			
P FEV ₁ (% pred)	103.4±0.48	103.6±0.61	104.1±0.64
F FVC (% pred)	102.3±0.13	102.5±0.59	101.9±0.32
T FEV ₁ /FVC	98.4±0.24	99.02±0.76	98.6±0.18

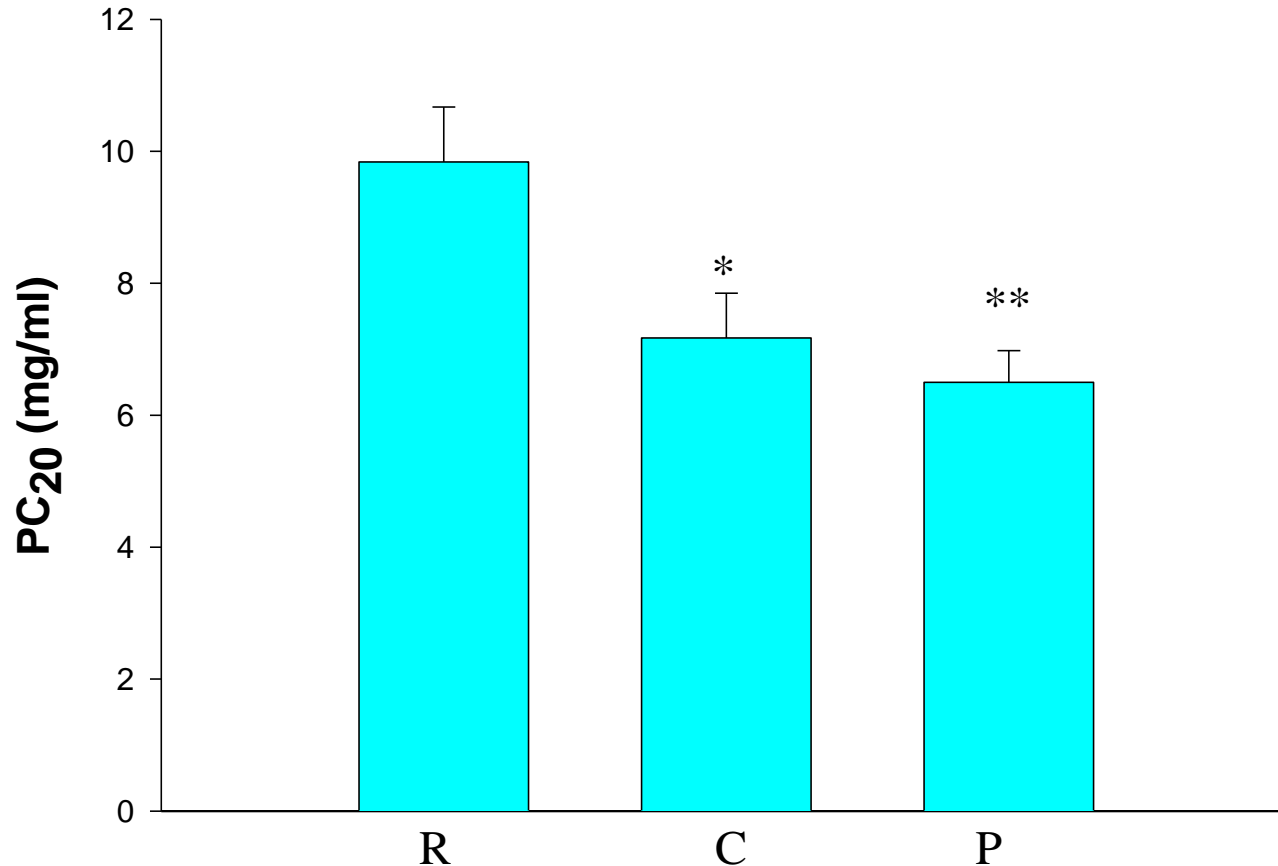
Table 2. Mean values of air pollutants

Variables	Mean values	
	Polluted area with chemical factory	Coastal area near chemical factory
Ozone* (ppm)	0.023	0.0105
SO ₂ (ppm)	0.0248	0.014
NO ₂ (ppm)	0.0244	0.0085
HF (ppm)	0.7664	0.2145

*: 8h moving average; SO₂: sulphur dioxide;
 NO₂: nitric dioxide; HF: hydrogen fluoride, irritant.



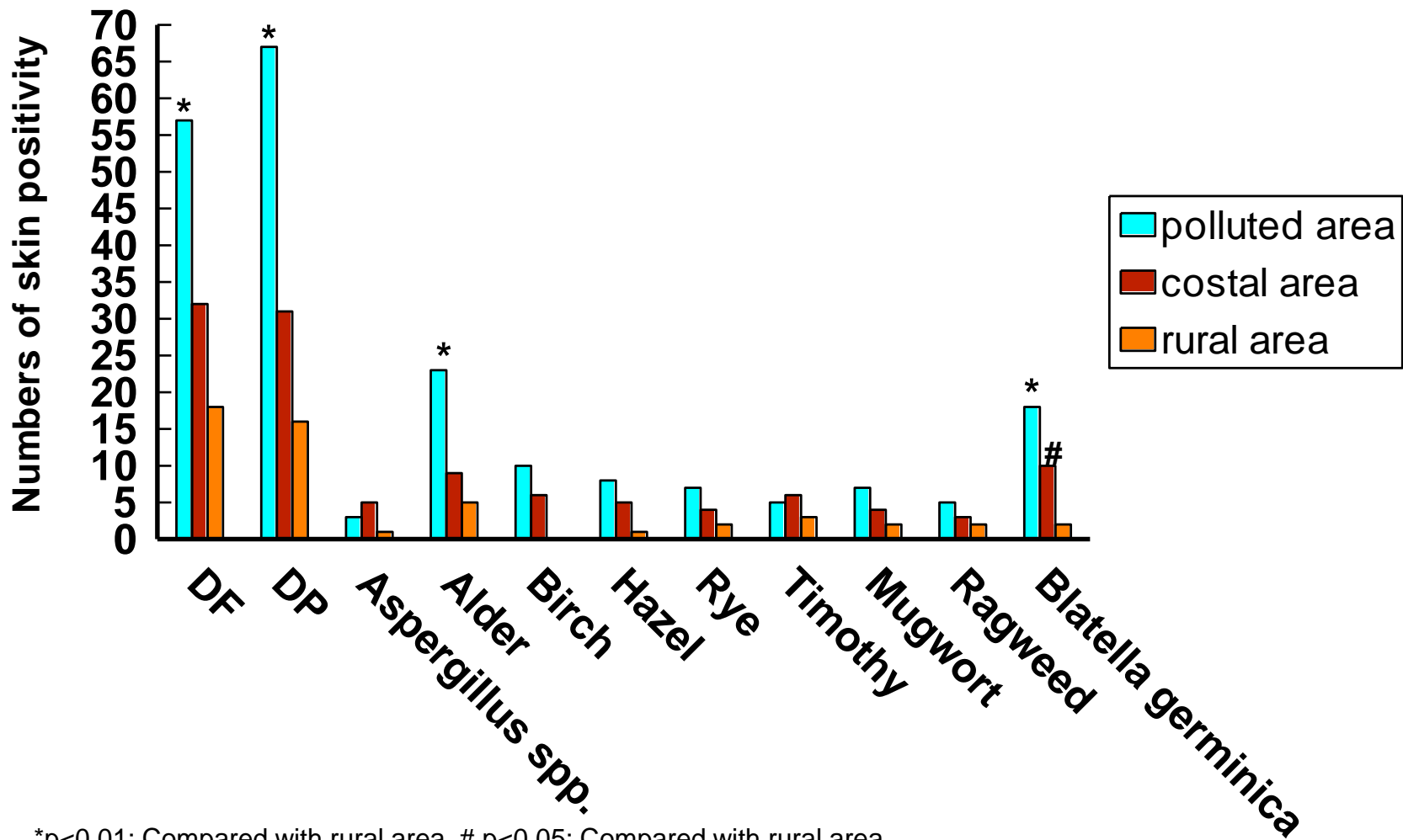
PC₂₀ in polluted chemical factory area (P), coastal area (C), and rural area (R).



* $p < 0.05$ compared with rural area.

** $p = 0.001$ compared with rural area.





*p<0.01: Compared with rural area. # p<0.05: Compared with rural area.

CONCLUSION:

Our findings demonstrate that more children living in polluted areas have airway hyperresponsiveness than do those living in less polluted areas.



Prenatal fine particulate exposure and early childhood asthma: effect of maternal stress and fetal gender

OBJECTIVE:

We prospectively examined associations between co-exposure to prenatal particulate matter with an aerodynamic diameter of less than 2.5 microns ($PM_{2.5}$) and maternal stress on childhood asthma (n=736).

METHODS:

Daily $PM_{2.5}$ exposure during pregnancy was estimated using a validated satellite-based spatio-temporally resolved prediction model. Prenatal maternal negative life events (NLEs) were dichotomized around the median (high: $NLE \geq 3$; low: $NLE < 3$). We employed Bayesian distributed lag interaction models (BDLIMs) to identify sensitive windows for prenatal $PM_{2.5}$ exposure on children's asthma by age 6, and determine effect modification by maternal stress and child sex.



Table 1. ACCESS participant characteristics (n=736)

	All children (n=736)		High Prenatal Stress NLE ^d ≥3 (n=308)		Low Prenatal Stress NLE ^d <3 (n=428)	
Sex						
Male	374	50.8	165	53.6	209	48.8
Female	362	49.2	143	46.4	219	51.2
Ever had asthma^a up to 6 years old (n, %)						
No	626	85.1	249	80.8	377	88.1
Yes	110	15.0	59	19.2	51	11.9
Race/Ethnicity (n, %)						
Black	218	29.6	96	31.2	122	28.5
Hispanic	395	53.7	157	51.0	238	55.6
White/Other	123	16.7	55	17.8	68	15.9
Maternal education (n, %)						
>12 yrs	251	34.1	106	34.4	145	33.9
≤12 yrs	485	65.9	202	65.6	283	66.1
Pre- and postnatal tobacco smoke exposure status^b (n, %)						
Never	532	72.3	203	65.9	329	76.9
Prenatal only	35	4.8	13	4.2	22	5.1
Postnatal only	100	13.6	46	14.9	54	12.6
Both prenatal and postnatal	69	9.4	46	14.9	23	5.4
Maternal obesity^b (n,%)						
No	531	72.2	214	69.5	317	74.1
Yes	205	27.8	94	30.5	111	25.9
Maternal age at enrollment (years; median, IQR)	25.5	22.3-30.7	25.3	22.0-31.0	25.8	22.5-30.6
Averaged prenatal PM_{2.5} level (µg/m³; median, IQR)	11.2	10.2-11.9	11.2	10.2-11.9	11.2	10.2-11.8
Prenatal negative life events (NLE) score^c (median, IQR)	2	1-4	4	3-5	1	0-2

^a Ever self-reported doctor-diagnosed asthma, eczema, and/or hay fever

^b Combination of prenatal maternal smoking and postnatal maternal and/or household smoking status

^c Pre-pregnancy obesity: ≥30 kg/m².

^d Assessed using Crisis in Family Systems-Revised (CRISYS-R) survey (40); multi-item survey summarized into a continuous score.

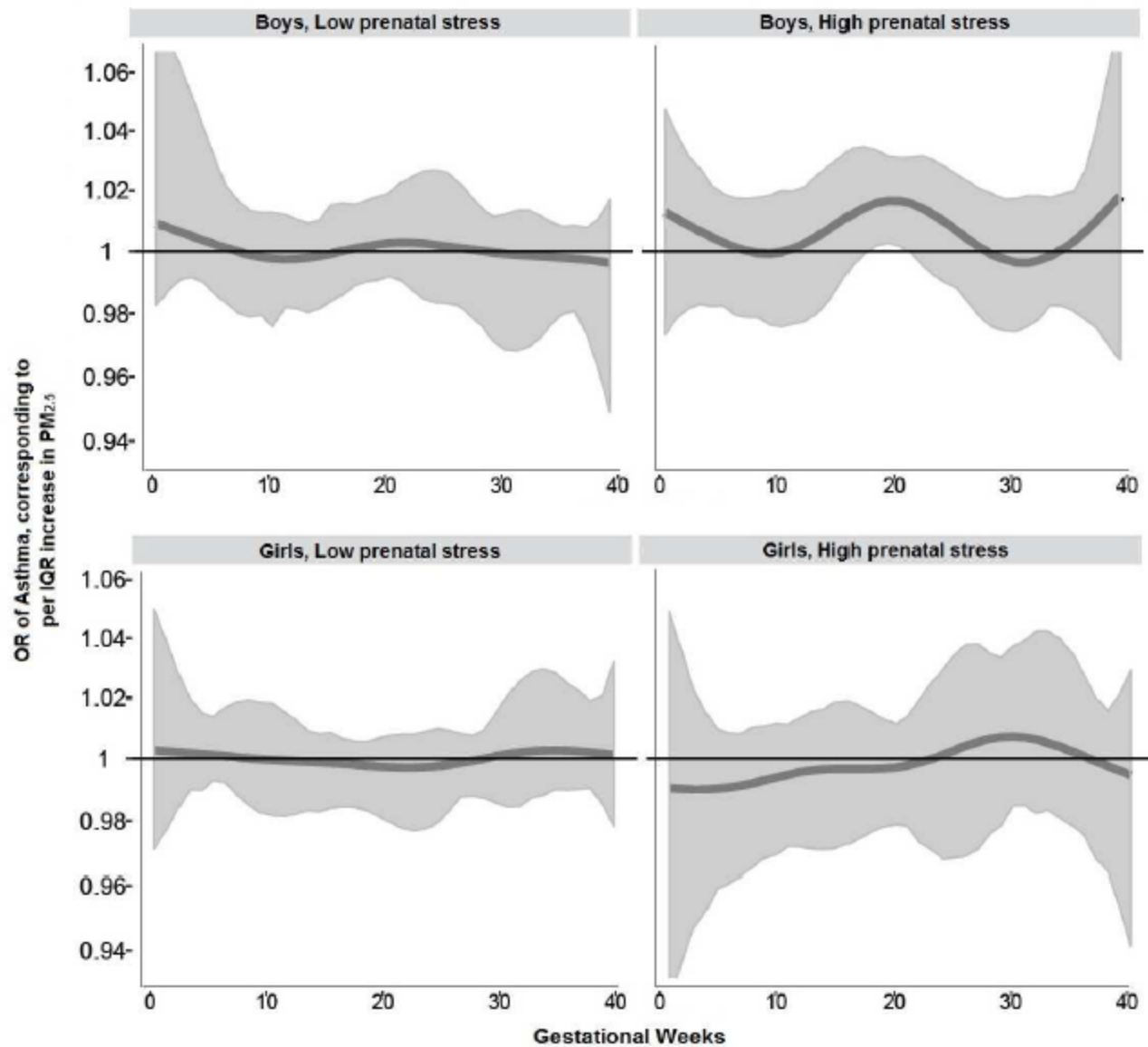


Figure 2. Associations between prenatal $PM_{2.5}$ exposure and children's asthma: interaction by stress and child sex.



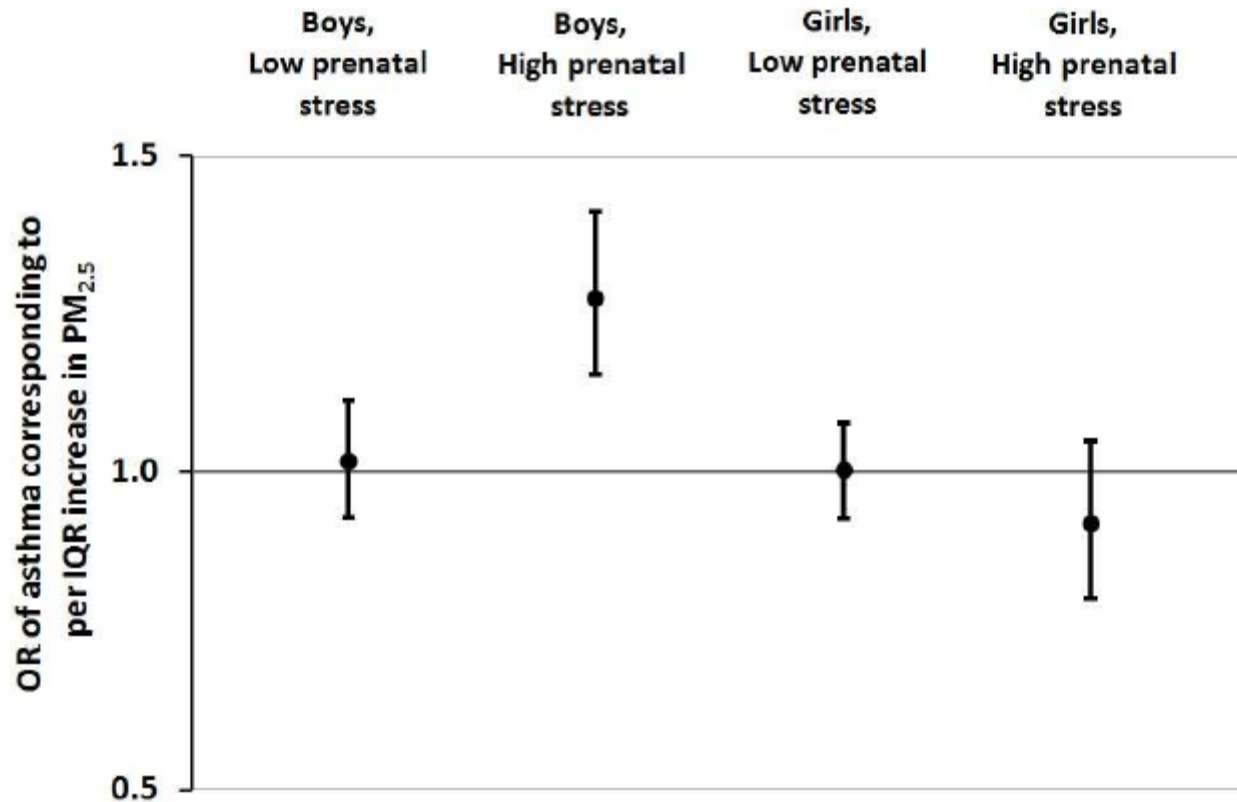


Figure 3. Cumulative effect (OR of asthma) of $PM_{2.5}$ across pregnancy on childhood asthma.

Conclusion: Prenatal $PM_{2.5}$ exposure during sensitive windows is associated with increased risk of child asthma, especially in boys concurrently exposed to elevated maternal stress.



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

The association between children's exposure to TRAP metrics and their risk of 'asthma' incidence or lifetime prevalence, **from birth to age 18 years old**.

Forty-one studies met our eligibility criteria.

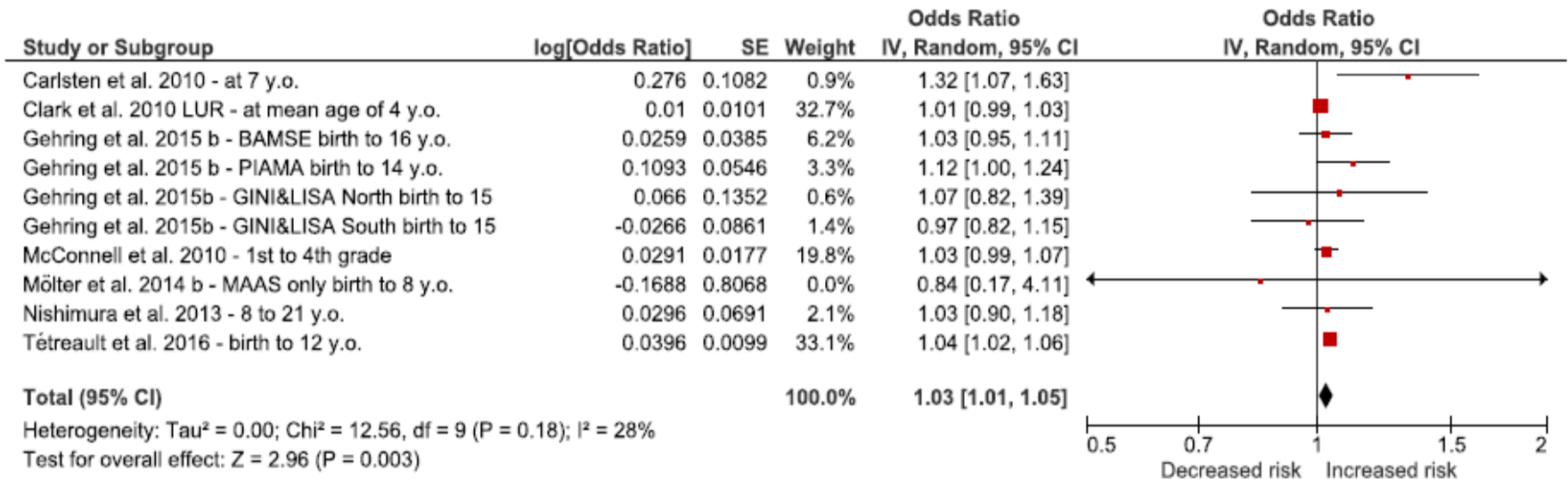


Fig. 6. **PM2.5** random-effects meta-analyses. Individual and summary random-effects estimates for associations between PM2.5 per 1 µg/m³ and asthma at any age.



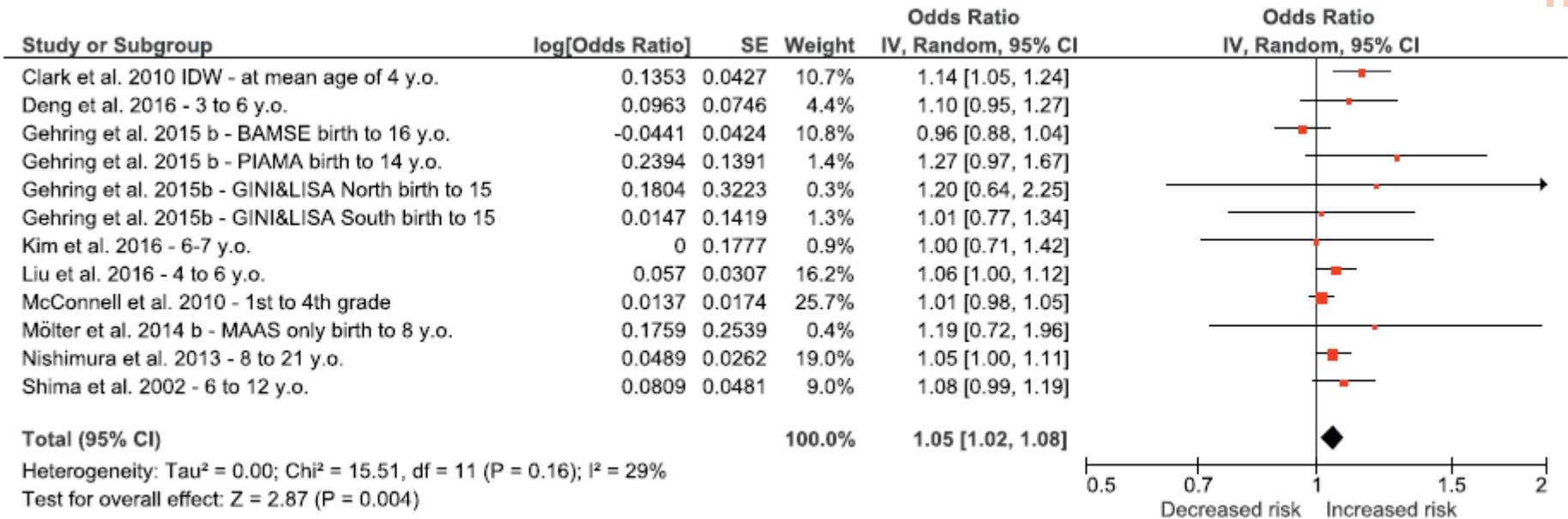


Fig. 7. **PM10** random-effects meta-analyses. Individual and summary random-effects estimates for associations between PM10 per 2 µg/m³ and asthma at any age.

Conclusion: Our findings support the hypothesis that **childhood exposure to TRAP contributes to their development of asthma**. Future meta-analyses would benefit from greater standardization of study methods including exposure assessment harmonization, outcome harmonization, confounders' harmonization and the inclusion of all important confounders in individual studies.



Adverse health effects from ambient air pollution in relation to residential wood combustion in modern society

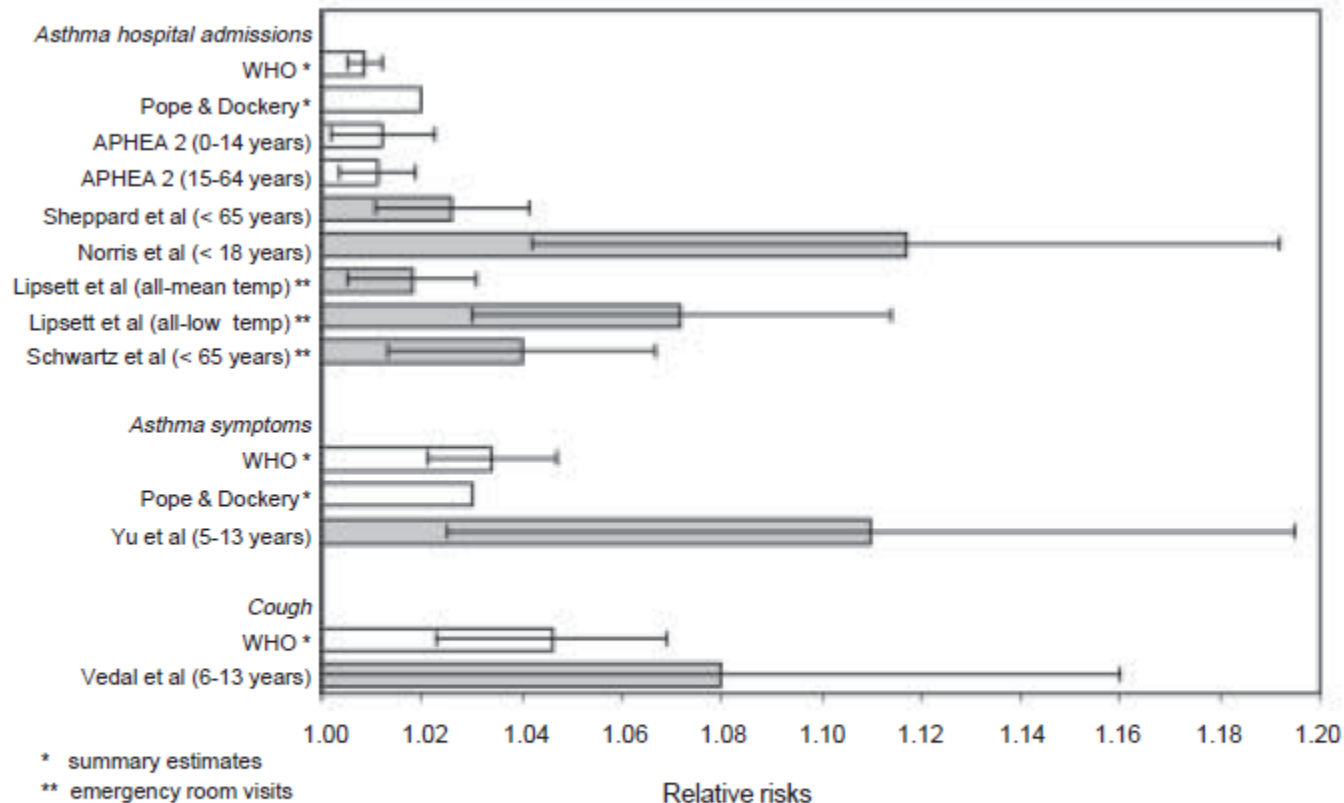
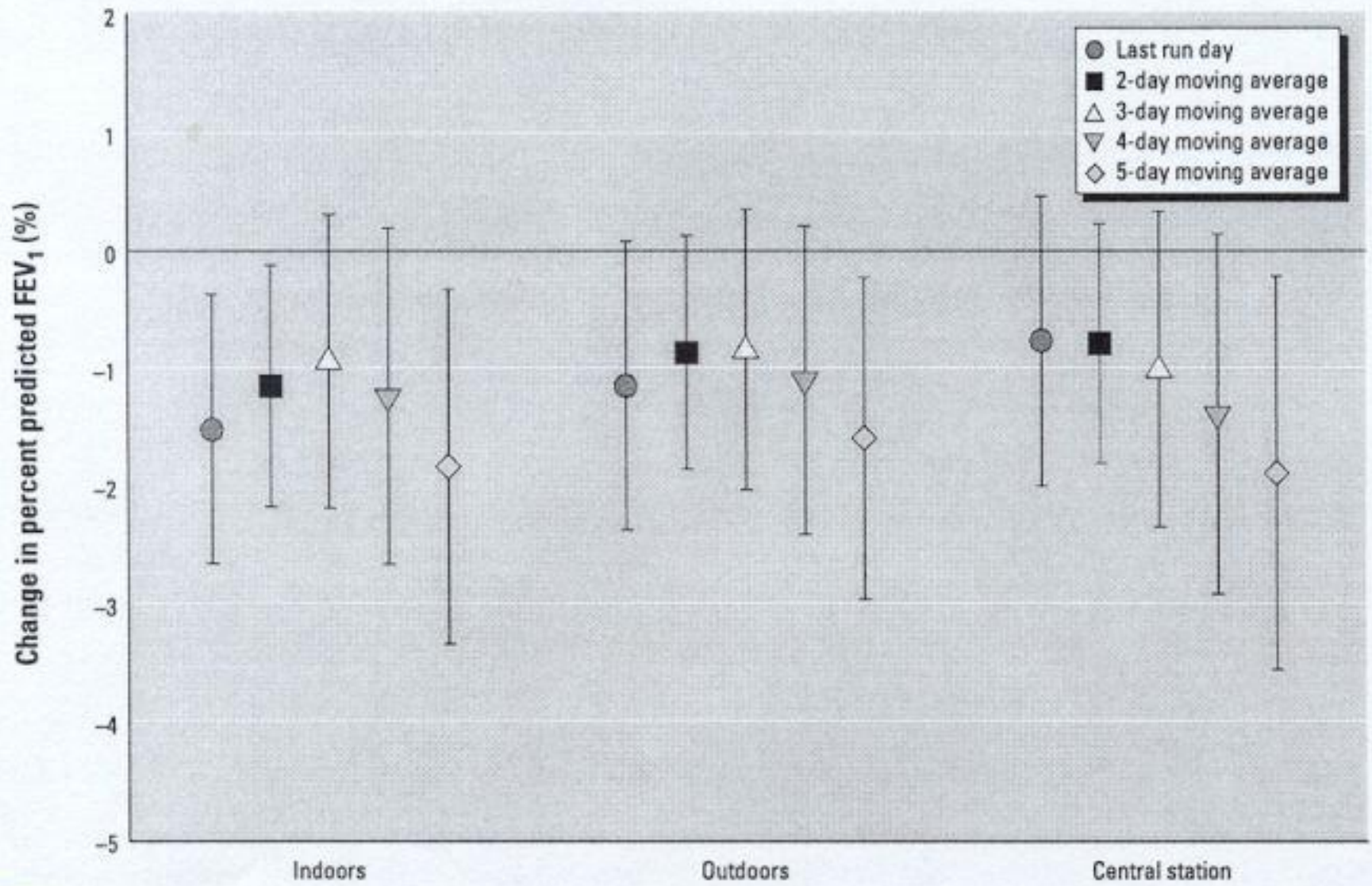


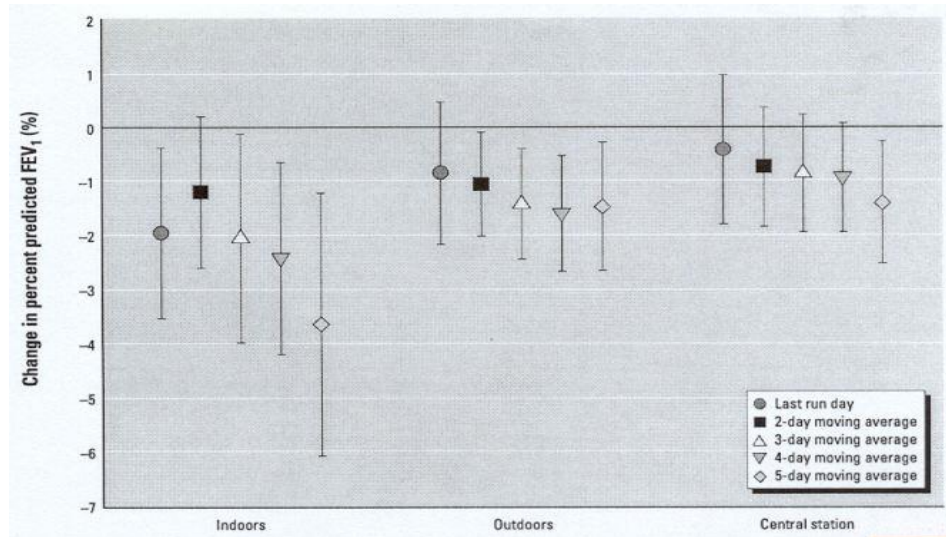
Figure 1. Relative risks for different morbidity outcomes in association with a 10 µg/m³ increase in PM₁₀ (particulate matter with an aerodynamic diameter of <10 µg) with 95% confidence intervals as error bars. The studies in which wood smoke was considered a major air pollution source are shown by closed columns, and the comparison estimates are represented by open columns (temp = temperature, APHEA2 = European study by Atkinson et al (55), WHO = World Health Organization).



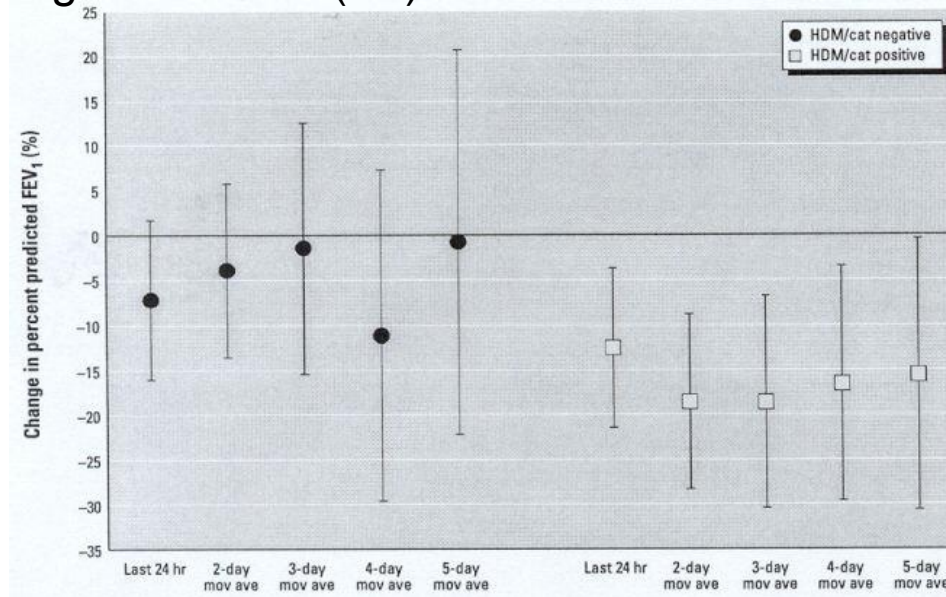
PM2.5



PM10



Personal PM according to HDM/cat(+/-)



대기오염에 따른 천식환자의 병원방문 및 입원

- 대기오염분포에 따른 심장질환과 만성폐쇄성 폐질환, 천식의 병원방문(외래), 입원(응급실, 병실)과 관련성
- 2003년 1월부터 8월까지 병원을 방문한 환자

I20(협심증): 4887명

I21(심근경색): 698명

J44(만성폐쇄성폐질환): 1927명

J45(천식): 17881명



Airkorea 실시간 모니터링 - Windows Internet Explorer

http://www.airkorea.or.kr/airkorea/airkorea_monitoring.jsp?item=khal

실시간대기질 | 태미대기질

통합대기환경지수 | 미세먼지 | 오존 | 이산화질소 | 일산화탄소 | 이황산가스

▶ 2011/10/06 15시

통합대기환경지수(CAI)

대기오염농도를 건강영향에 따른 6단계의 지수와 색상으로 표현
미세먼지 등 5개 물질 중 가장 높은 지수 점수를 CAI로 사용

서울

황사·오존 경보지역

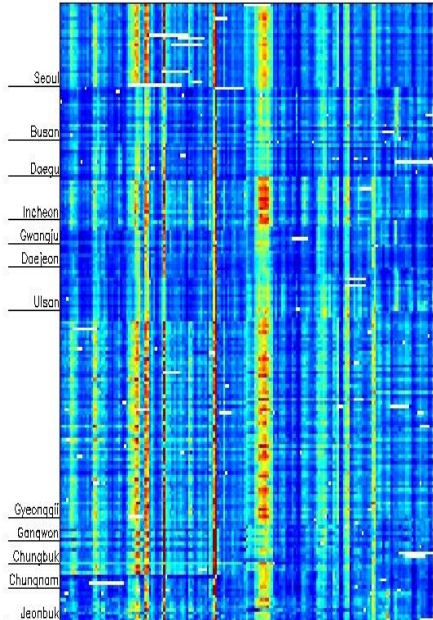
측정항 구분

- 도시대기
- 국가배경
- 도로변대기
- 교외대기

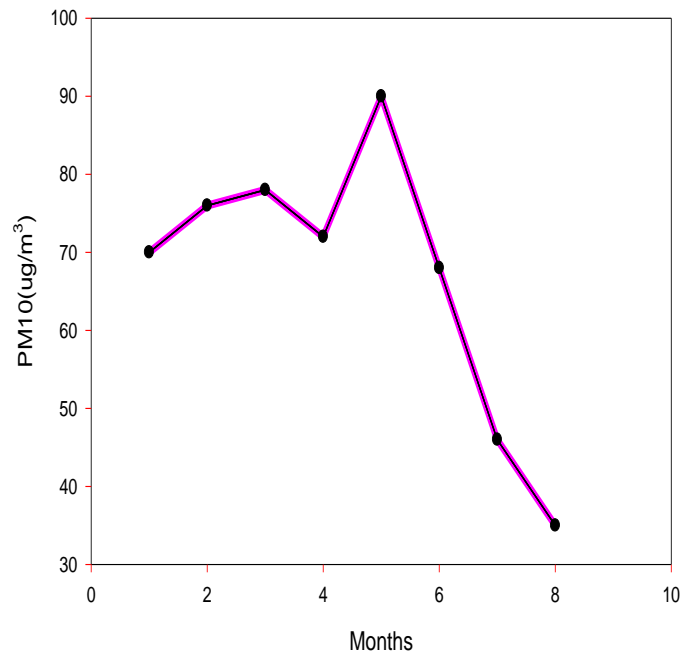
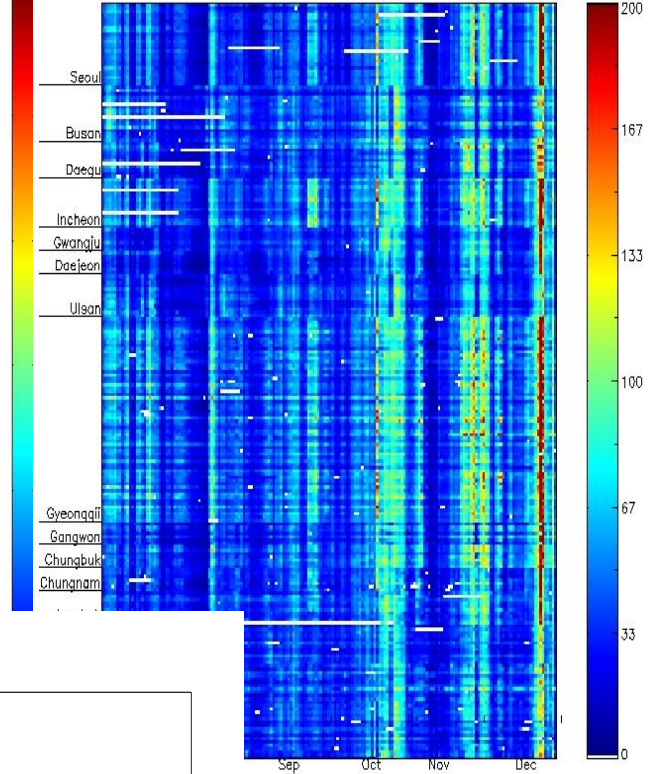
분류: 좋음 보통 나쁨 매우나쁨

관측항목: 미세먼지 오존 이산화질소 일산화탄소 이황산가스

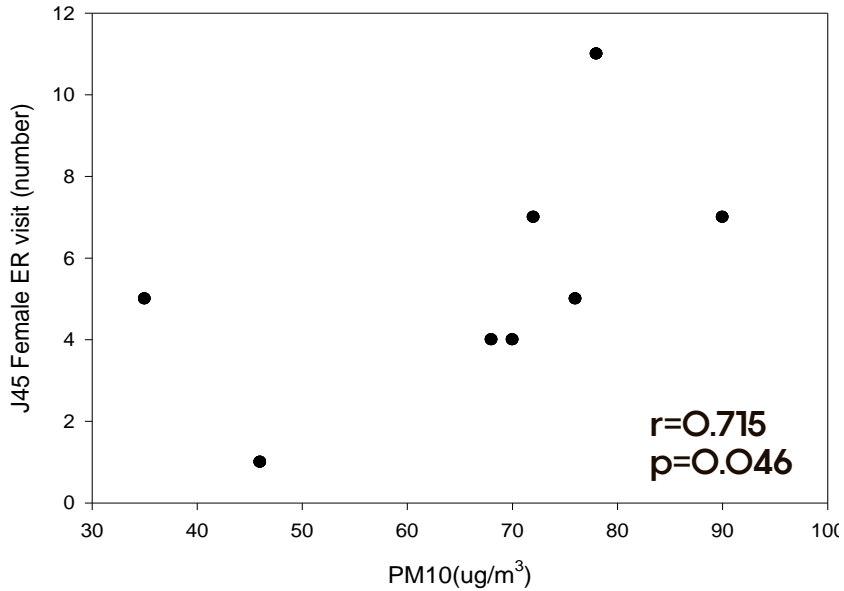
PM-10, 2009 Daily Average of the First Half



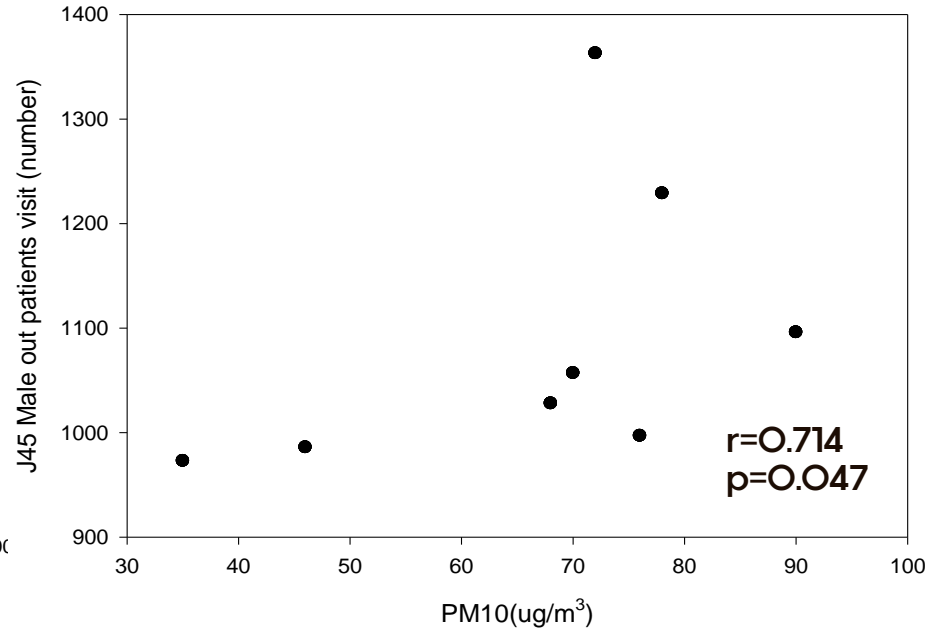
PM-10, 2009 Daily Average of the Second Half



PM10(미세먼지)과 J45(천식) 여자 응급실 방문



PM10(미세먼지)과 J45(천식) 남자 외래 내원



오존과 미세먼지는 협심증, 만성폐쇄성 폐질환, 기관지천식 등의 악화와 관련이 있을 가능성이 크기 때문에 이러한 대기오염 물질에 대한 관리는 이들 만성질환의 증상악화에 중요한 예방방법이 될 것이다.

Higher PM concentration contribute to decrease in PFT and hospital visit in asthma and allergic rhinitis

대기오염 데이터 정보

대기환경 날짜 : 1997 ~ 2015년까지

측정 항목 : PM₁₀ (한국환경공단-Airkorea)



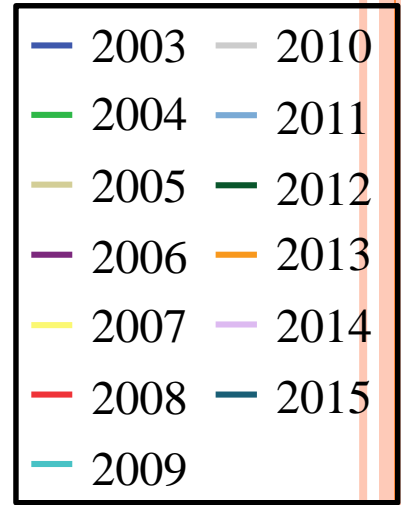
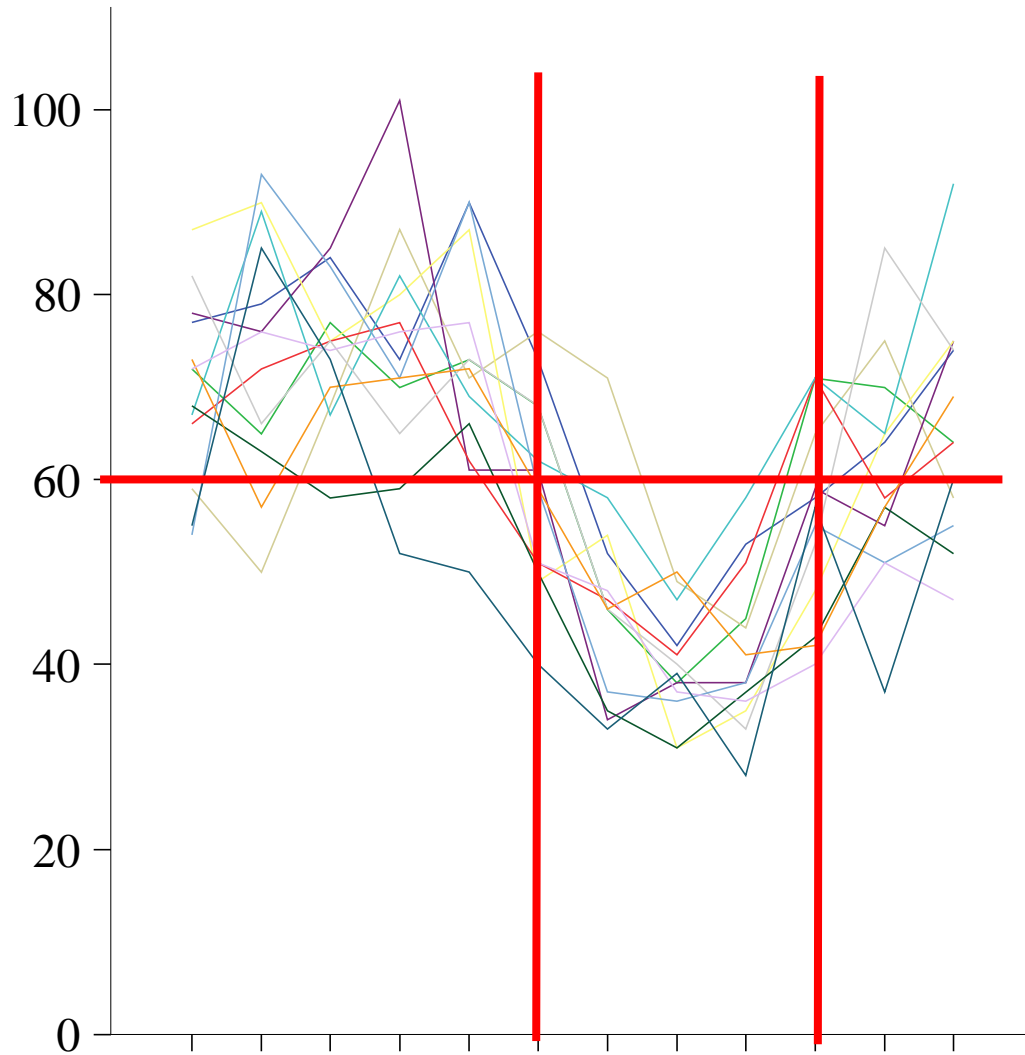
연도-월별 완사 정리

(단위: 명)

연도 \ 월	1	2	3	4	5	6	7	8	9	10	11	12	Total
1997						1							1
1999									1				1
2001		1	1	2			2			1			7
2002	2			2			1			2	1		8
2003	3	3	2	3	5	2	3	1	2	1	1	2	28
2004	1	1		5		2	1		2	1	2	1	16
2005				2	2		2	3	1	1	1	3	15
2006	1				2		4		2	4	1	4	18
2007	5	16	14	8	13	16	11	15	5	5	7	6	121
2008	5	5	3	4	3	2	6	1	1	1	3		34
2009		3	7	2	1	5	2	1	4				25
2010	6	2	1	2	1	1	1	4	1	2	3	3	27
2011	9	2	6	8	4	2	2		2	6	6	4	51
2012	6	4	1	11	8	7	24	14	6	15	14	7	117
2013	19	11	9	7	7	10	8	7	2	12	6	4	102
2014	2	5	7	2	3	2	1	4	3	2	5	5	41
2015	6	7	1		5	8	12	6	4	4			53

총 완사 665

PM10 Concentration ($\mu\text{g}/\text{m}^3$)



Month
-Low 6, 7, 8, 9, 10
-High 1, 2, 3, 4, 5, 11, 12

Patient
NC : BA : BA+AR
-Low-
62 : 115 : 102
-High-
73 : 189 : 107

PM10

	Low concentration			High concentration		
	Control	BA	BA+AR	Control	BA	BA+AR
No. of subjects	62	115	102	73	189	107
Sex (male/female)	27/35	24/91	32/70	25/48	55/134	42/65
Age of initial visit (y)	55.90 ± 10.63	54.35 ± 13.05	50.94 ± 14.29*	58.31 ± 12.22	54.53 ± 13.21*	46.84 ± 14.46* †
Smoking (NS/ES/CS)	45/12/5	97/8/10	76/13/13	63/8/2	150/18/21	80/7/20
Cigarettes smoked, pack years	6.35 ± 11.47	3.06 ± 9.04*	3.64 ± 10.05	2.65 ± 7.69††	4.50 ± 12.08	4.05 ± 9.99
Body mass index (kg/m ²)	23.75 ± 3.27	24.95 ± 3.29*	24.58 ± 3.45	25.04 ± 3.02††	24.70 ± 3.42	24.05 ± 3.17
Lung function						
FEV ₁ (% predicted)	106.03 ± 17.54	94.79 ± 20.62*	91.04 ± 18.26*	110.71 ± 14.00	93.67 ± 22.32*	86.70 ± 19.12* †
FVC (% predicted)	94.39 ± 13.94	88.80 ± 14.60*	86.28 ± 13.45*	96.27 ± 11.05	86.27 ± 15.96*	86.41 ± 15.98*
FEV ₁ /FVC ratio	82.74 ± 4.72	78.77 ± 8.90*	78.50 ± 8.41*	83.67 ± 5.36	79.75 ± 10.16*	77.14 ± 10.69* †
PC ₂₀ (mg/mL)	22.62 ± 5.61	13.57 ± 11.19*	11.08 ± 10.40*	21.67 ± 6.10	16.93 ± 9.85*	11.49 ± 10.71* †
Total IgE (kU)	98.90 ± 171.46	307 ± 73 ± 538.17	285.51 ± 413.19*	138.07 ± 203.44	273.17 ± 505.52	300.05 ± 387.89* †
Blood WBC/uL	6343.75 ± 2268.34	6911.91 ± 2079.44	6603.48 ± 1742.01	5874.37 ± 1600.05	6788.51 ± 2539.68*	7200.00 ± 2498.77*
Blood eosinophils (%)	3.34 ± 2.86	4.52 ± 4.07	4.50 ± 2.79	2.59 ± 1.79	4.05 ± 4.90	4.54 ± 5.35
Blood neutrophils (%)	55.47 ± 7.69	54.51 ± 10.10	55.96 ± 12.02	53.78 ± 9.38	56.83 ± 13.33	55.41 ± 13.46

Data are expressed as means Mean ± SD. BMI, Body mass index; CS, current smoker; ES, exsmoker; FVC, forced vital capacity; NS, nonsmoker.

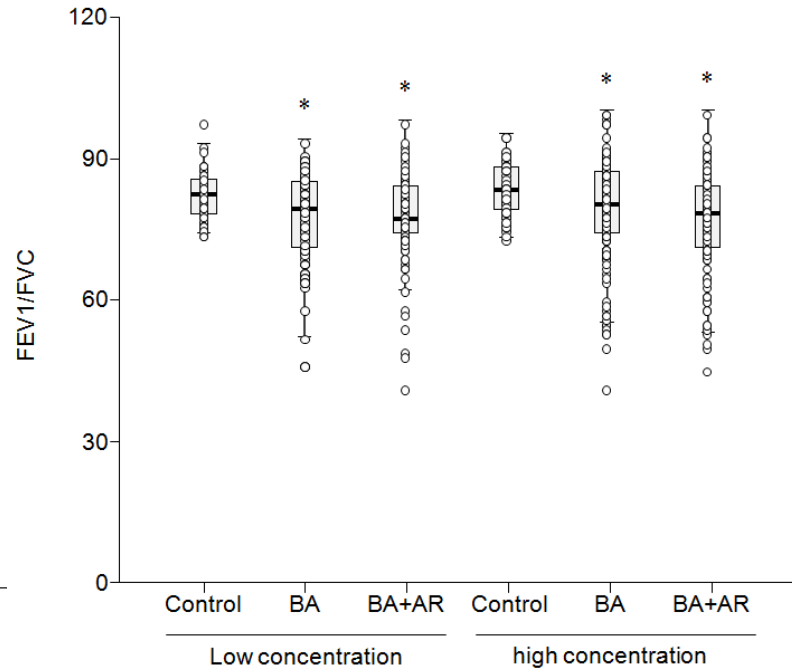
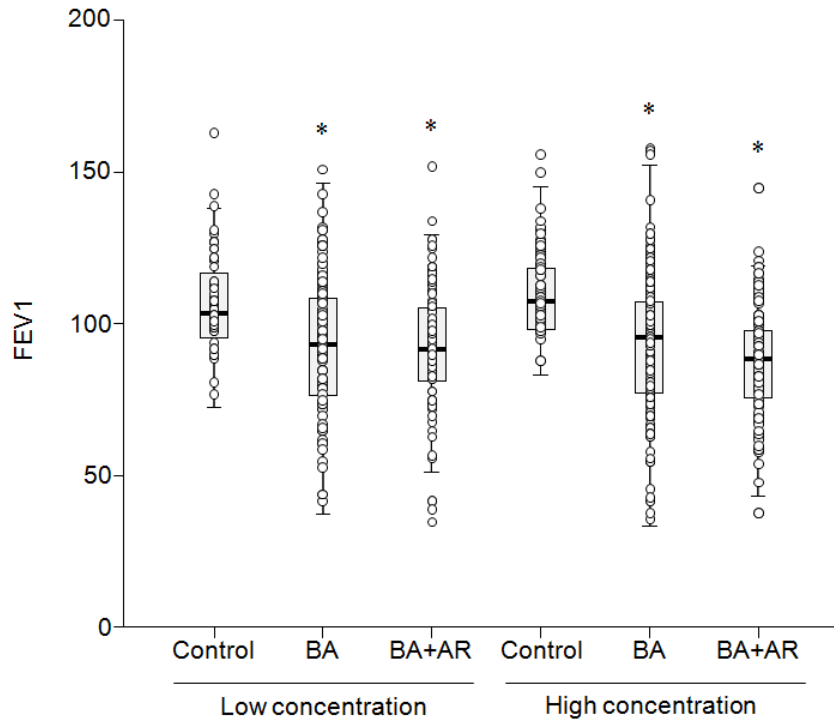
* P<.01 compared with control subjects

† P<.05 compared with patients with bronchial asthma

†† P<.05 compared with patients with low concentration



PM10



Trends in respiratory diagnoses and symptoms of firefighters exposed to the World Trade Center disaster: 2005–2010 (10999소방수)

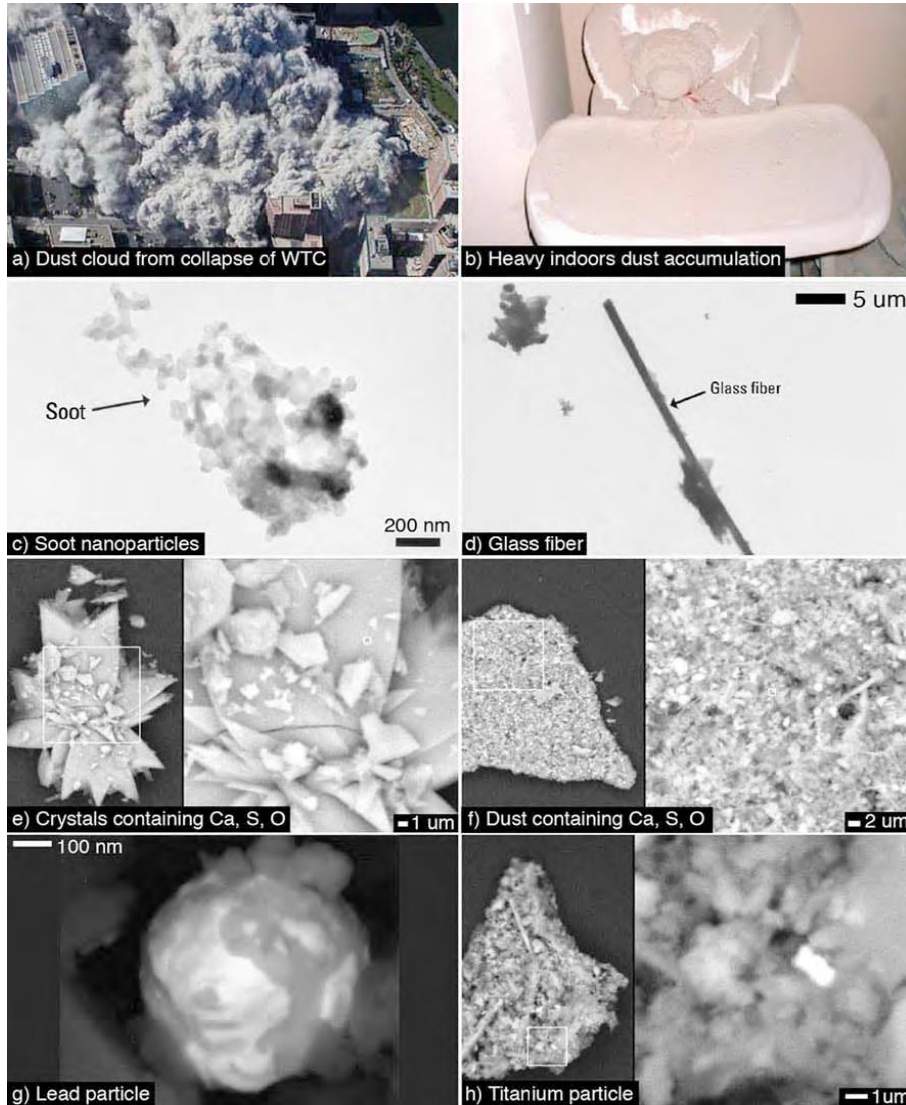
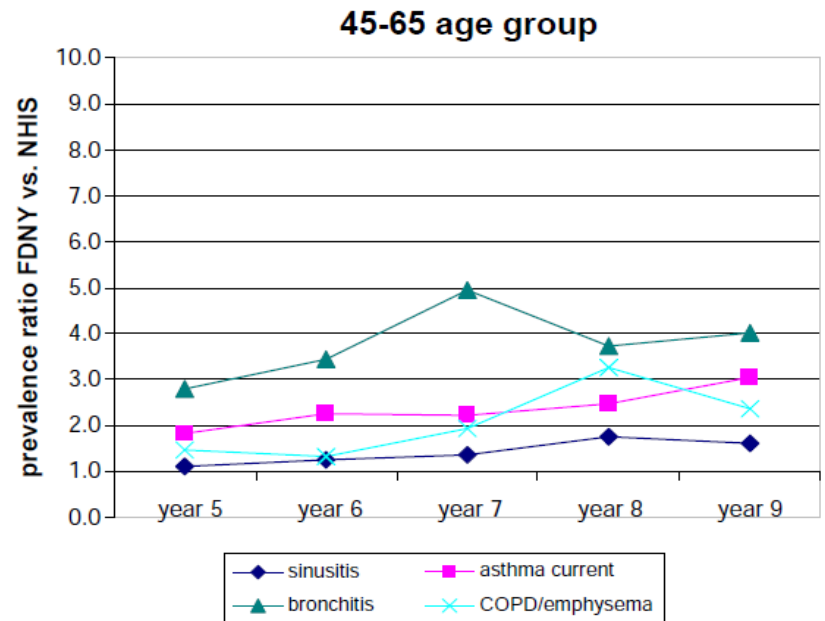
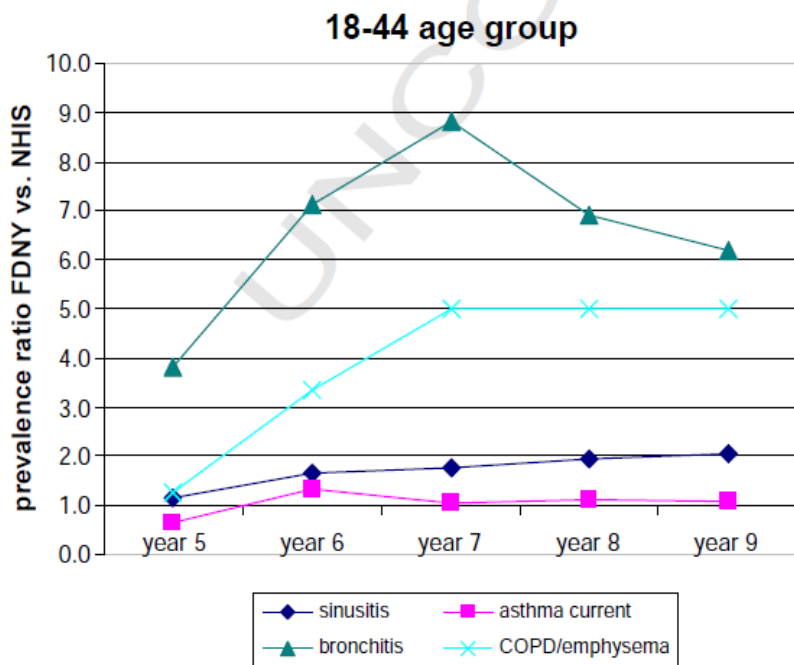


Figure. (a) Dust cloud from the World Trade Center collapse spreads to neighboring streets. Courtesy EPA. (b) Heavy dust accumulation in store closed to World Trade Center. Particle collected from the site of collapse and neighboring streets: (c) soot [135], d) glass fiber, (e), (f) dust containing Ca, S and O, (g) lead and [g135], (h) titanium particle. Images (b)-(h) courtesy Environmental Health Perspectives.



World Trade Center; FDNY, Fire Department of the City of New York, New York, New York, USA; NHIS, National Health Interview Survey, USA; COPD, chronic obstructive pulmonary disease;



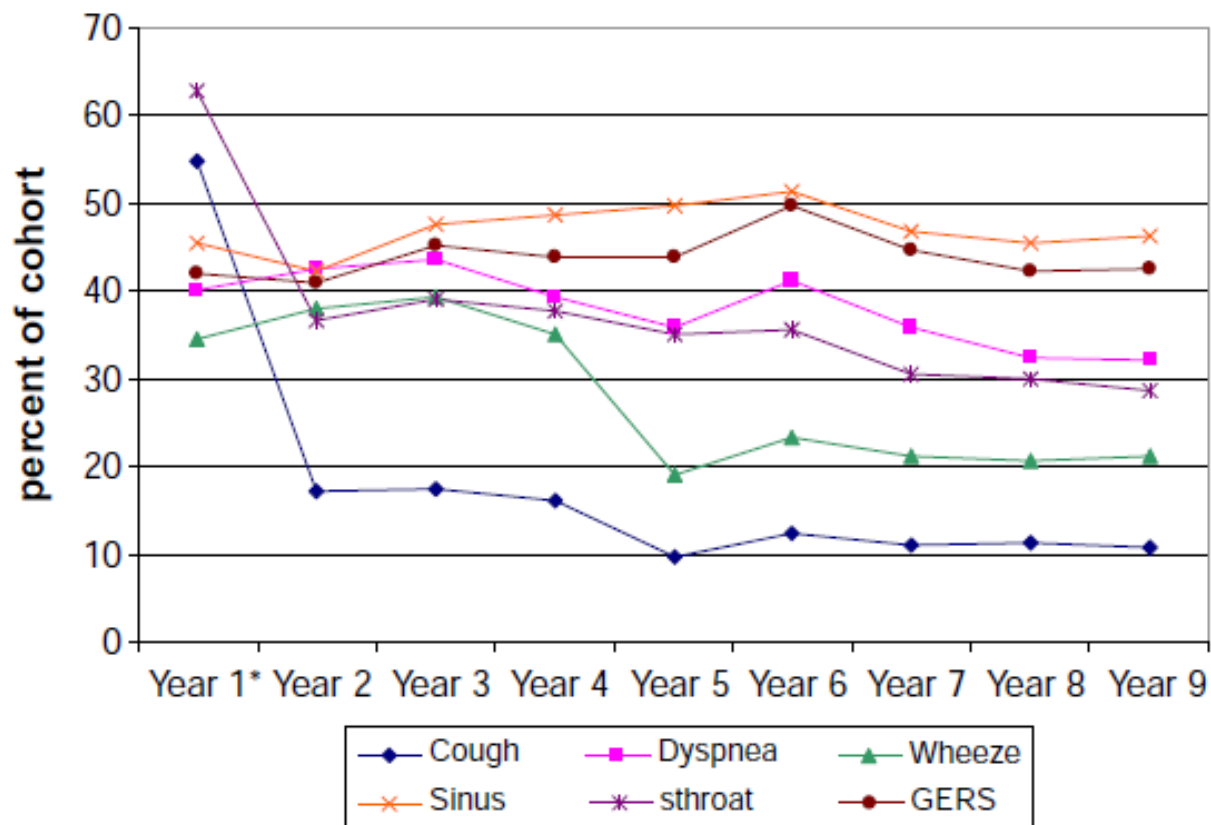


Fig. 3. Prevalence of symptoms of WTC-Exposed firefighters across 9 years since 10/02/2001. *Years refer to study years starting 10/2/2001.



Short-term and medium-term health effects of 9/11

	Groups studied	Size (on Dec 31, 2010)*	Other distinguishing characteristics
Fire Department of New York ^{8,15}	Nearly all firefighters and emergency medical service workers who responded to WTC disaster	15 415 screened so far; 5686 treated in past 12 months	Established in 2001; employer-based medical programme with physical examinations done about every 18 months; has medical records before 9/11; provides screening, monitoring, and treatment
New York and New Jersey WTC Clinical Consortium ¹⁶⁻²⁴	Law enforcement and other responders, sanitation and construction workers, and volunteers	29 572 screened so far; 8411 treated in past 12 months	Established in 2002; provides screening, monitoring, and treatment through environmental and occupational health clinics to an ethnically and socioeconomically diverse population; up to 19% of patients do not have insurance yearly†
WTC Environmental Health Center ^{25,26}	Lower Manhattan area clean-up workers, office workers, residents, students, school staff, and passers-by	5130 examined so far; 2520 treated in past 12 months	Began in 2005 as privately funded programme at an asthma clinic in a New York City public hospital; monitors and treats but does not screen (patients are only accepted if symptomatic); includes children; up to 50% of patients do not have insurance yearly†
WTC Health Registry ²⁷⁻³⁴	Rescue and recovery workers, lower Manhattan residents, area workers, students, and passers-by	71 437 responded to first survey; 68% adults responded to second survey	Closed cohort recruited in 2003-04; 30% recruited through lists provided by entities such as employers and government agencies, and the remaining 70% self-enrolled; surveys done 2-3 years and 5-6 years after 9/11; includes children

WTC=World Trade Center. 9/11=Sept 11, 2001. *Number of people screened and treated are from the National Institute for Occupational Safety and Health.³⁵ †Data from WTC Medical Working Group of New York City.³⁶

Table: WTC populations being studied with funding from the National Institute for Occupational Safety and Health

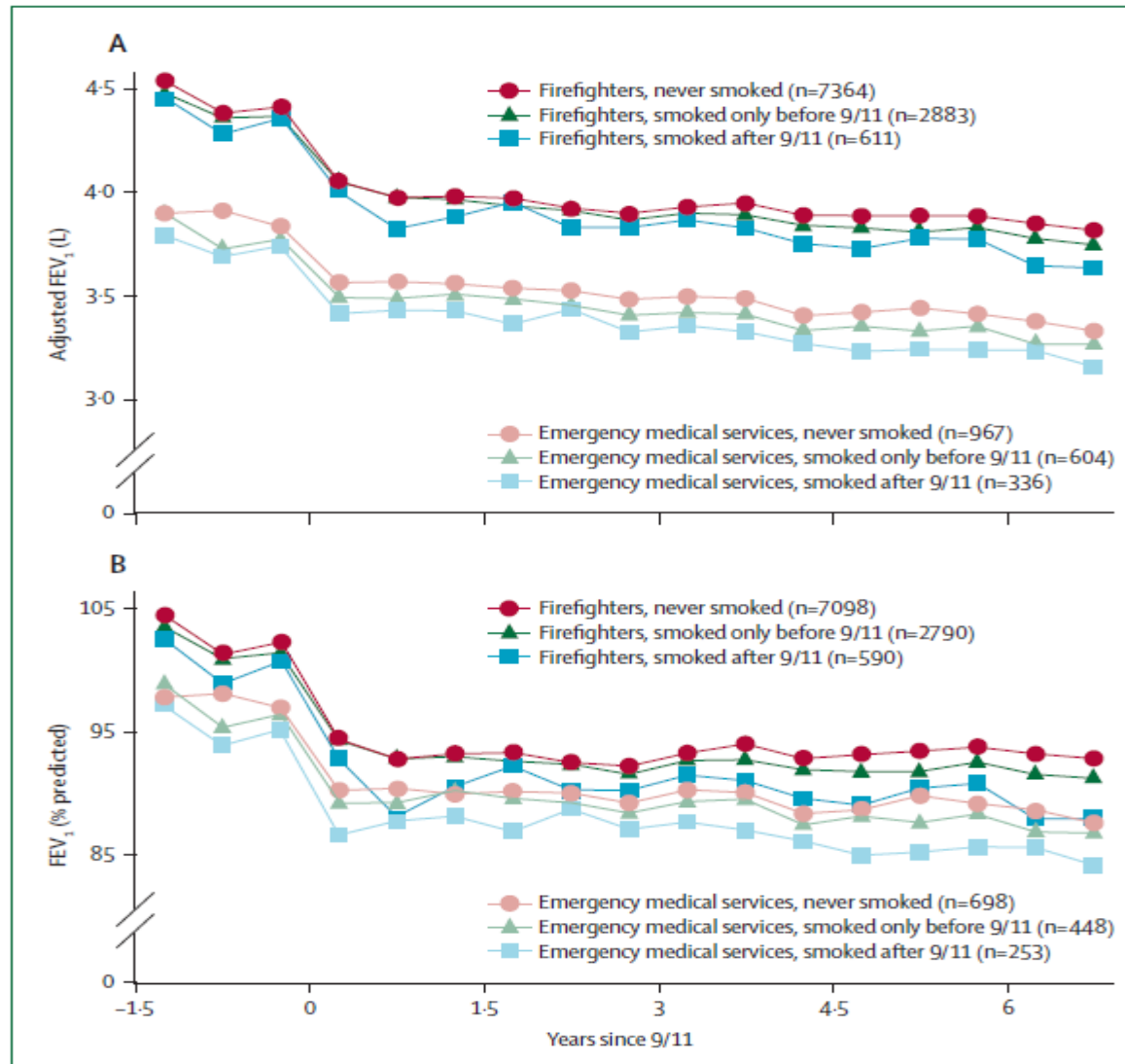


Figure 2: Lung function in firefighters and emergency medical services workers according to smoking status (A) Adjusted FEV₁ over time. (B) FEV₁ measurement compared with predicted values over time. FEV₁=forced expiratory volume in 1 s. 9/11=Sept 11, 2001. Reproduced from Aldrich and colleagues,²⁷ by permission of the Massachusetts Medical Society.

Persistence of multiple illnesses in World Trade Center rescue and recovery workers: a cohort study

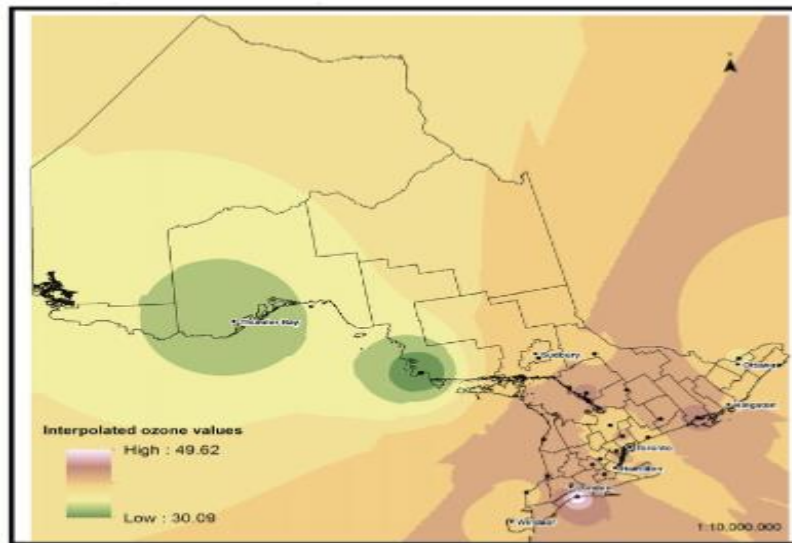
	Pre-exposure	Year 1	Year 2	Year 3	Year 4	Year 5	Year 6	Year 7	Year 8	Year 9
Physical health										
Asthma	10.5% (24985)	13.7% (22360)	15.8% (21496)	17.5% (19505)	19.3% (18227)	21.1% (16897)	23.4% (15317)	25.0% (12809)	26.4% (10744)	27.6% (7027)
Sinusitis	10.7% (25041)	18.0% (22357)	21.8% (20476)	25.2% (18238)	28.3% (16709)	31.2% (15204)	34.4% (13537)	37.3% (11100)	39.8% (9109)	42.3% (5870)
Gastro-oesophageal reflux disease	5.8% (25539)	10.7% (24055)	14.4% (22741)	17.9% (20343)	21.5% (18603)	25.4% (16828)	29.9% (14796)	33.6% (11896)	37.1% (9423)	39.3% (5650)
Mental health symptoms										
New York City police										
Depression	..	1.7% (9573)	2.0% (9467)	2.3% (8734)	3.7% (8026)	3.7% (7510)	4.2% (6999)	4.2% (6097)	6.2% (5420)	7.0% (3648)
PTSD	..	2.5% (9866)	2.5% (9679)	4.0% (9124)	4.6% (8624)	5.7% (8155)	6.3% (7597)	7.4% (6537)	8.4% (5601)	9.3% (3761)
Panic disorder	..	2.3% (9820)	2.6% (9691)	3.1% (8928)	4.4% (8186)	4.4% (7659)	5.6% (7158)	5.7% (6271)	7.0% (5591)	8.4% (3780)
Other rescue and recovery workers*										
Depression	..	10.8% (15062)	12.7% (14105)	15.3% (11620)	17.0% (9855)	17.4% (8847)	19.0% (8205)	22.3% (7138)	24.6% (6229)	27.5% (4200)
PTSD	..	12.8% (16054)	13.5% (14641)	17.1% (12306)	19.8% (10762)	22.6% (9785)	24.2% (8956)	27.2% (7682)	29.7% (6473)	31.9% (4342)
Panic disorder	..	5.0% (15995)	5.7% (15302)	8.1% (12880)	12.8% (10964)	12.8% (10083)	13.7% (9446)	14.2% (8302)	18.5% (7349)	21.2% (4953)
Abnormal spirometry	..	23.7% (24566)	24.9% (22174)	27.1% (19105)	28.5% (16542)	29.1% (14378)	32.4% (12700)	34.9% (10883)	37.9% (8949)	41.8% (5769)

Data are % (number at risk). Number of individuals at risk is estimated from cumulative incidence curves. PTSD=post-traumatic stress disorder. *All workers except New York City police officers.

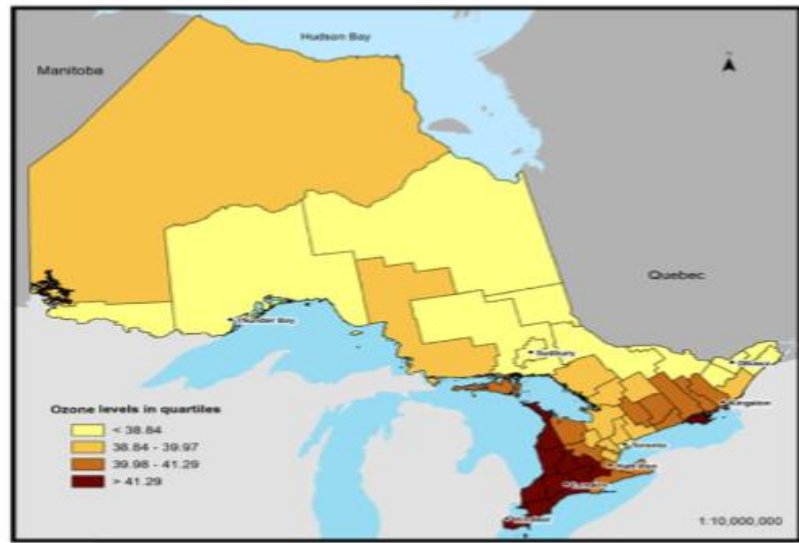
Table 2: Cumulative incidence before and after 9/11 of physical disorders, mental health symptoms, and spirometric abnormalities in the World Trade Center Screening, Monitoring, and Treatment Program study population

Progression from Asthma to Chronic Obstructive Pulmonary Disease. Is Air Pollution a Risk Factor?

Individuals who resided in Ontario, Canada, aged 18 years or older in 1996 with incident asthma between 1996 and 2009 who participated in the Canadian Community Health Survey were identified and followed until 2014 to determine the development of ACOS. Data on exposures to fine particulate matter (PM_{2.5}) and ozone (O₃) were obtained from fixed monitoring sites. Associations between air pollutants and ACOS were evaluated using Cox regression models.



Mean monthly maximum ozone levels by census division in Ontario for 2003



Mean monthly maximum ozone levels by census division in Ontario, 2003

Figure 1. Example of air pollution interpolation in Ontario by Census Division.

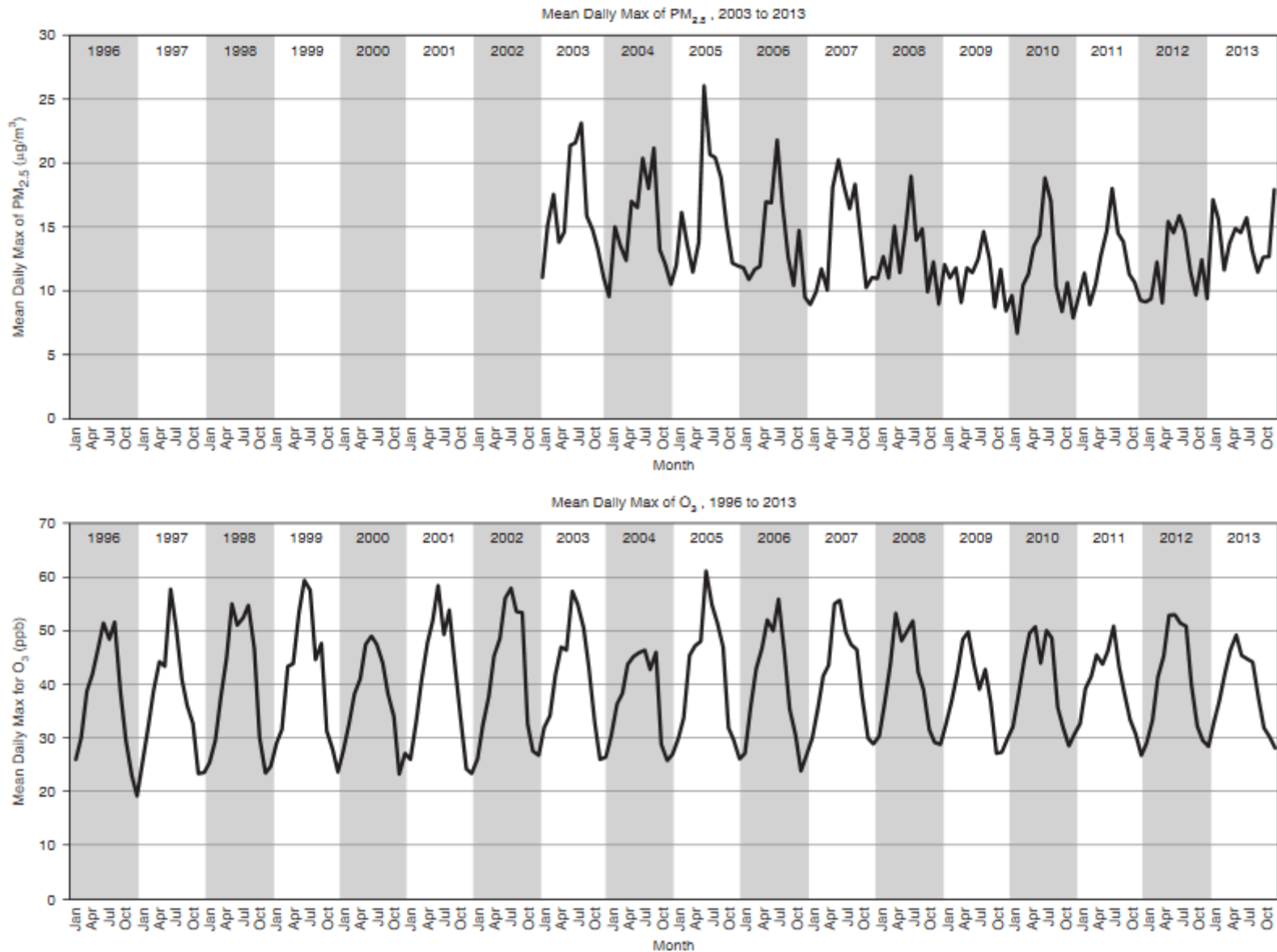


Figure 2. Distribution of mean levels of markers of pollution in Ontario, 1996 to 2013. O₃ = ozone; PM_{2.5} = particulate matter \leq 2.5 μm .

Table 1. Demographic Characteristics of Study Population with and without Asthma–Chronic Obstructive Pulmonary Disease Overlap Syndrome

Characteristic	ACOS (n = 630)		Non-ACOS (n = 5,410)		P Value
	No.	%	No.	%	
Age at asthma incidence, yr					<0.001
18–29	8	1.3	799	14.8	
30–49	40	6.3	1,299	24.0	
40–49	94	14.9	1,129	20.9	
50–59	150	23.8	989	18.3	
60–69	180	28.6	690	12.8	
70–79	125	19.8	397	7.3	
80–99	33	5.2	107	2.0	
Mean age ± SD	59.91 ± 12.96		48.45 ± 15.61		<0.001
Sex					0.014
Female	394	62.5	3,647	67.4	
Male	236	37.5	1,763	32.6	
Deprivation quintile (proxy measure of SES)					0.016
Q1 (least marginalized)	90	14.4	1,032	19.3	
Q2	122	19.6	1,118	20.9	
Q3	142	22.8	1,236	23.1	
Q4	141	22.6	1,034	19.3	
Q5 (most marginalized)	128	20.5	925	17.3	
Rural residence	153	24.3	987	18.2	<0.001
Coexisting chronic disease morbidity					
Acute myocardial infarction	39	6.2	168	3.1	<0.001
Angina	206	32.7	902	16.7	<0.001
Congestive heart failure	97	15.4	306	5.7	<0.001
Diabetes mellitus	161	25.6	1,106	20.4	0.003
Hypertension	401	63.7	2,484	45.9	<0.001
Ischemic heart disease	268	42.5	1,390	25.7	<0.001
Stroke	105	16.7	498	9.2	<0.001
Lung cancer	11	1.7	38	0.7	0.006
Nonlung cancers	74	11.7	502	9.3	0.046
Deaths	174	27.6	362	6.7	<0.001

Definition of abbreviations: ACOS = asthma–chronic obstructive pulmonary disease overlap syndrome; SES = socioeconomic status.
All percentages are adjusted for missing data.



Table 3. Health Services Use per 100 Person-Years in Patients with and without Asthma–Chronic Obstructive Pulmonary Disease Overlap Syndrome

HSU (<i>per Person per Year</i>)	ACOS (<i>n</i> = 630)				Non-ACOS (<i>n</i> = 5,410)			
	No. People with HSU	Median	Mean	SD	No. People with HSU	Median	Mean	SD
Asthma specific								
Hospitalization	62	14.0	21.5	19.6	275	8.0	12.0	11.7
ED visits	108	19.2	33.2	68.5	667	10.5	15.7	20.9
Physician office visits	573	45.0	82.4	115.3	5,065	22.7	40.3	61.6
Asthma related								
Hospitalization	67	14.7	18.5	12.6	371	8.4	12.9	12.2
ED visits	274	22.7	34.2	44.5	1,955	12.7	20.5	22.8
Physician office visits	595	120.7	171.6	170.1	5,211	84.5	120.5	121.1
Asthma specific or asthma related								
Hospitalization	117	15.7	22.0	18.4	596	8.8	13.6	13.4
ED visits	312	25.6	41.5	66.1	2,202	13.9	23.0	28.5
Physician office visits	626	178.5	238.6	226.5	5,384	115.1	154.6	144.1

Definition of abbreviations: ACOS = asthma–chronic obstructive pulmonary disease overlap syndrome; ED = emergency department; HSU = health service use.

For ACOS, HSU was counted between asthma incidence and COPD diagnosis date. For non-ACOS, HSU was counted between asthma incidence and the end of the study (March 31, 2014). Only people with HSU were included in the analysis. Asthma-related causes include: acute respiratory infections, pneumonia and influenza, atopic dermatitis, gastroesophageal reflux disorder, heartburn, and allergic contact dermatitis.



Table 4. Unadjusted and Adjusted Hazard Ratios of Asthma–Chronic Obstructive Pulmonary Disease Overlap Syndrome and Exposures to Air Pollutants from Cox Regressions

Covariates	Single Pollutant: PM _{2.5}				Single Pollutant: O ₃				Two Pollutants: PM _{2.5} and O ₃			
	Adjusted* HR	95% CI		P Value	Adjusted* HR	95% CI		P Value	Adjusted* HR	95% CI		P Value
		Lower	Upper			Lower	Upper			Lower	Upper	
PM _{2.5} , 10 µg/m ³	3.06	1.86	5.04	<0.0001					2.78	1.62	4.78	0.0002
O ₃ , 10 ppb					2.05	1.17	3.60	0.0122	1.31	0.71	2.39	0.3851
Ever smoker	3.18	2.64	3.83	<0.0001	3.13	2.60	3.78	<0.0001	3.17	2.63	3.82	<0.0001
BMI ≥ 30	1.32	1.09	1.60	0.0043	1.30	1.08	1.58	0.0063	1.32	1.09	1.60	0.0042

Definition of abbreviations: BMI = body mass index; CI = confidence interval; HR = hazard ratio; O₃ = ozone; PM_{2.5} = particulate matter ≤ 2.5 µm.

*Other covariates included: age at asthma incidence, sex, socioeconomic status proxy measured by deprivation quintile, rural residence, other coexisting chronic diseases morbidity.

CONCLUSIONS: Individuals exposed to higher levels of air pollution had nearly threefold greater odds of developing ACOS. Minimizing exposure to high levels of air pollution may decrease the risk of ACOS.



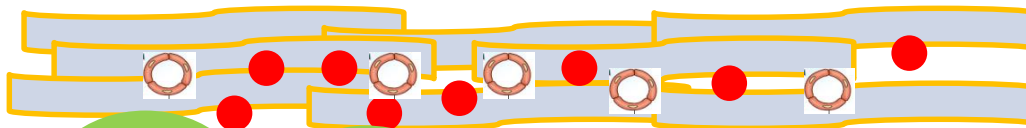
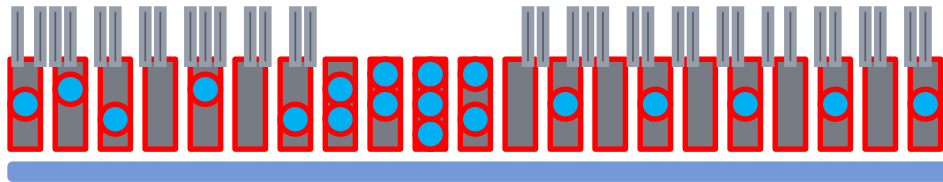
PM에 의한 기도염증기전

Local lung
inflammation

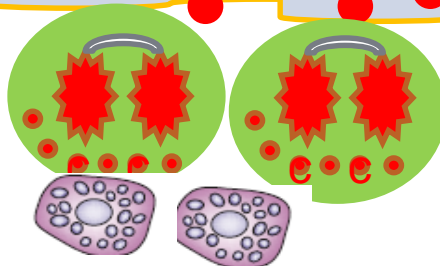
Innate Immunity: TNF α

Adaptive immunity: Total IgE

T cell activation



Systemic lung
inflammation



Oxidative stress (exogenous and endogenous ROS)

1) Oxidant interact with biomolecules
(lipids, protein, DNA)

: Intracellular antioxidants \downarrow , Protein modification

2) Oxidant induced cell signaling

ASK1, JNK, Keap1, Nrf2, GSTA2, NQO1, DJ1,
TRPA1, Noxa

3) Endogenous sources of ROS



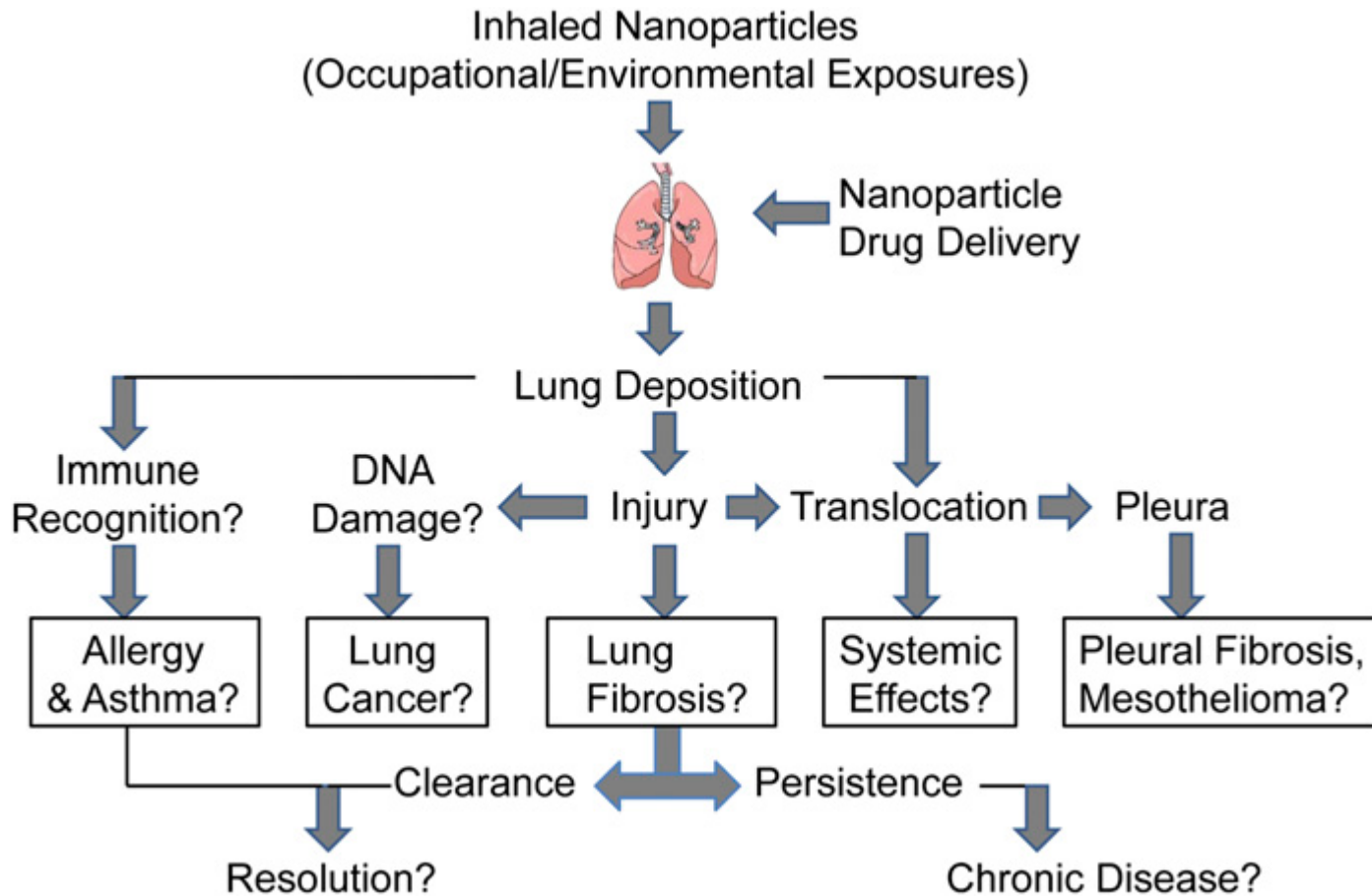


Figure. Biologic endpoints that should be evaluated to determine the potential of nanomaterials to cause disease in mice after inhalation to mimic occupational or environmental exposures or by nebulization routes to mimic drug delivery scenarios.

Ym1 and Ym2 Expression in a Mouse Model Exposed to DEP

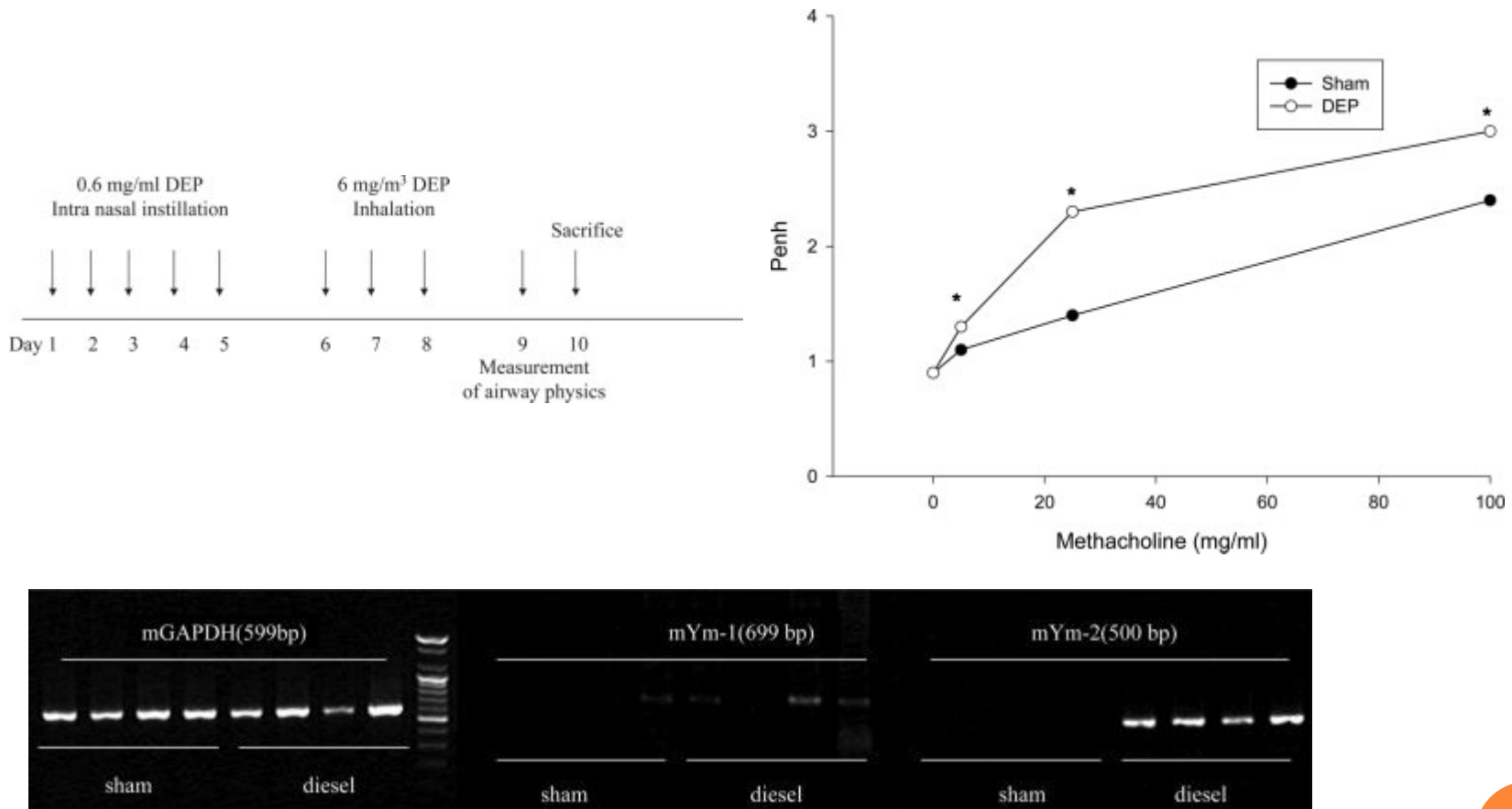
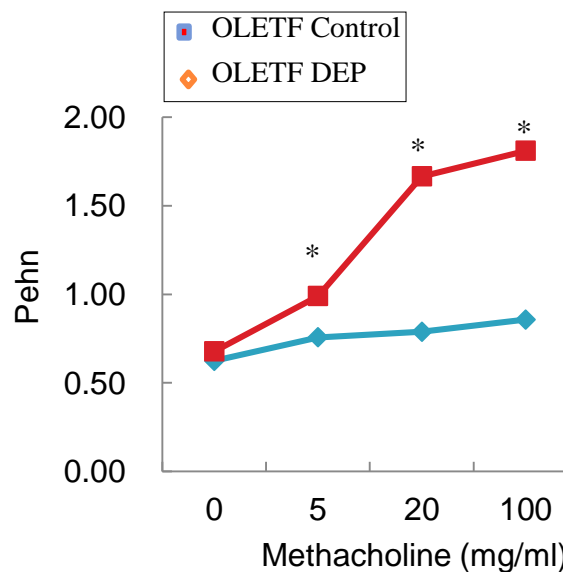
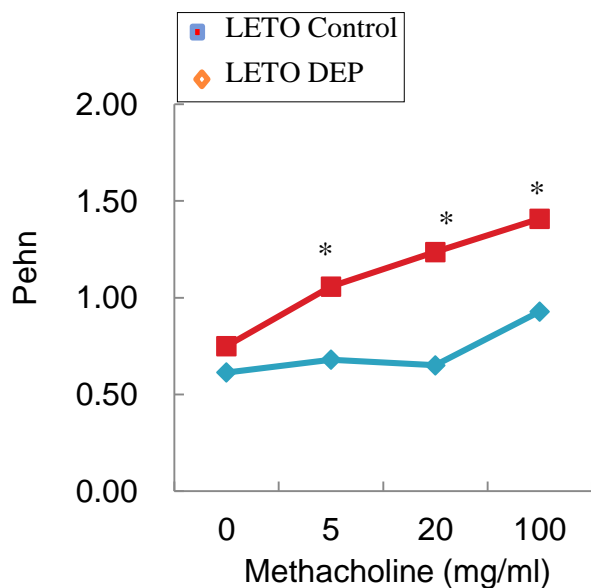
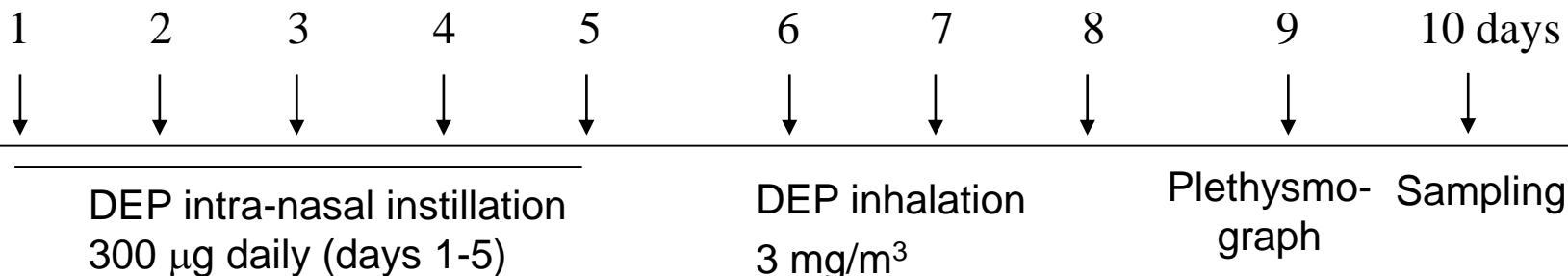


Figure. Schematic of the experimental protocol. Mice were sensitized intranasally on days 1–5. On days 6 and 8, after the initial sensitization, the mice were exposed to 6 mg DEP/m³ saline or to a straight saline control for 1 h using an ultrasonic nebulizer.

Diesel Exhaust Particle-Induced Airway Responses are Augmented in Obese Rats

Kuk-Young Moon¹, Moo-Kyun Park², George D. Leikauf³,
 Choon-Sik Park¹, and An-Soo Jang¹



Particle stimulation dephosphorylates glutathione S-transferase pi 1 of epithelial cells

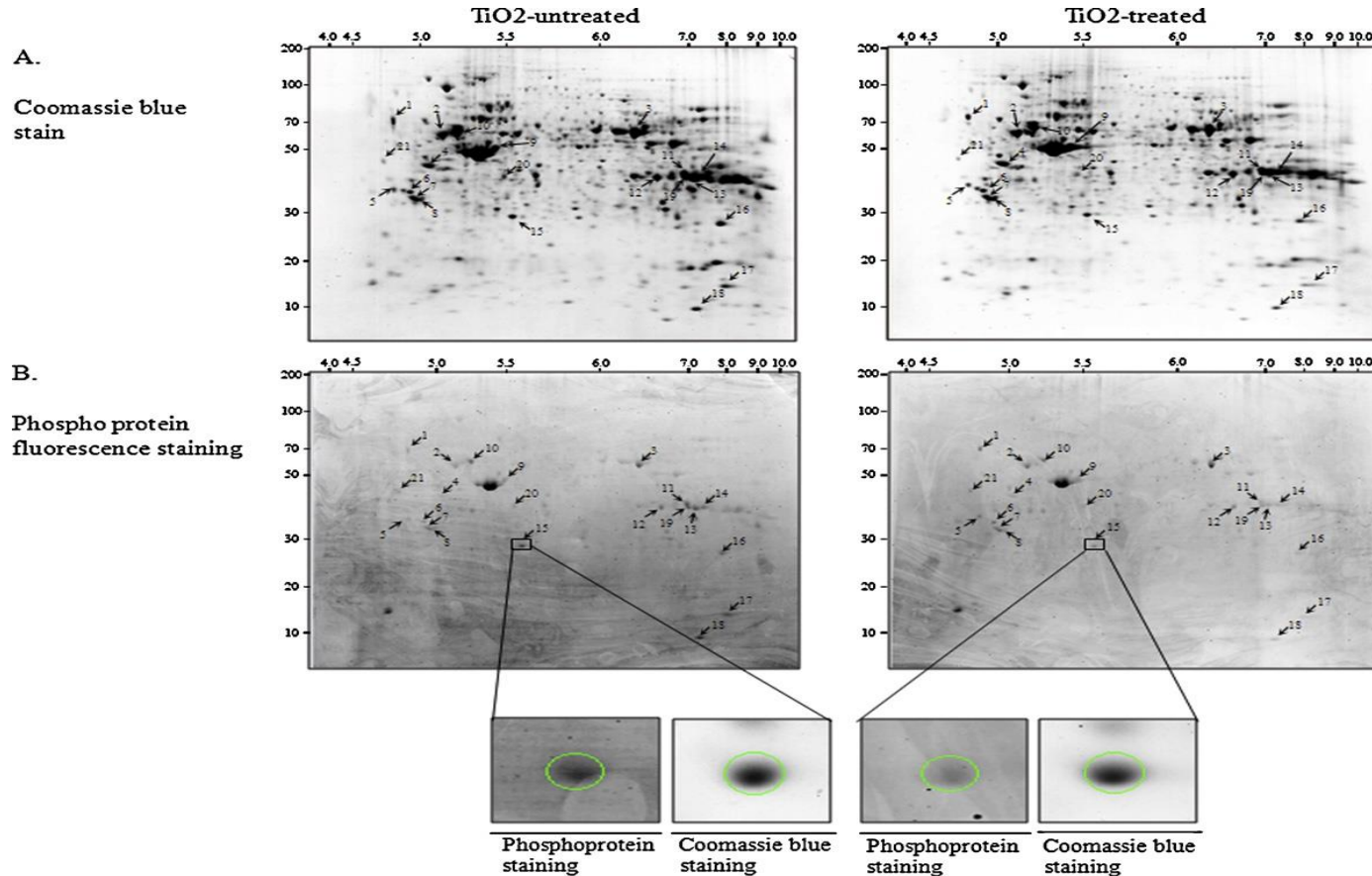


Figure 1. Photographs of two-dimensional electrophoresis (2-DE) separation of BEAS-2B cell lysate obtained from the TiO₂-treated group and TiO₂-untreated group.

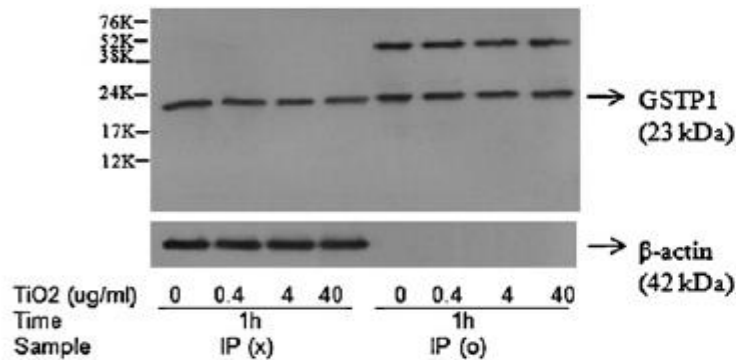
Table 1

Differentially expressed proteins identified by MALDI-TOF/TOF.

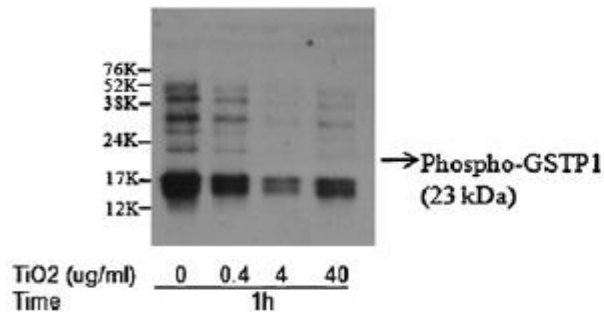
No.	Protein name	Accession no.	Determined sequence	MW (kDa)/PI	Normalized relative intensity		Ratio	Function
					Untreat	Treat		
1	Calreticulin	4757900	K.EQFLDGDGWTSR.W	80.2/4.71	0.089	< 0.183	2.1	Signal transduction
2	Tubulin, beta	57209813	R.AILVDLEPGTMDSVR.S	65.6/5.23	0.086	< 0.159	2.0	Cytoskeleton
3	Aldehyde dehydrogenase 1A1	21361176	R.QAFQIGSPWR.T	64.3/6.62	0.004	< 0.028	6.7	Oxidation
4	Nucleophosmin 1 isoform 2	40353734	K.VDNDENEHQSLR.T	46.3/5.12	0.042	< 0.094	2.3	Cellular process
5	RAB2A, member RAS oncogene family	4506365	K.YIIIGDTGVGK.S	39.3/4.73	0.114	< 0.333	2.9	Protein transporter
6	Tyrosine 3/tryptophan 5 -monooxygenase activation protein, epsilon polypeptide	5803225	K.LAEQAERYDEMVESMK.K	38.9/4.92	0.289	< 0.680	2.4	Signal transduction
7	Glutamate decarboxylase-like 1	197383062	R.QCPVDGDDIQQEMIPSK.K	36.4/4.96	0.097	< 0.184	2.0	Unknown
8	YWHAZ protein	49119653	K.SVTEQGAELSNNEER.N	36.2/5.06	0.052	< 0.098	2.0	Signal transduction
9	Cytokeratin 18	30311	R.AQIFANTVDNAR.I	67.2/5.32	0.130	< 0.360	2.9	Cytoskeleton
10	Vimentin	62414289	R.TYSLGSLRPSTSR.S	65.9/5.37	0.188	> 0.088	2.1	Cytoskeleton
11	Chain A, human 3alpha-Hsd type 3 in ternary complex with Nadp and testosterone	21465695	K.LNDGHFMPVLGFGTYAPAEVPK.S	44.2/6.91	0.113	> 0.032	3.5	Unknown
12	Aldo-keto reductase family 1, member B1	4502049	R.HIDCAHVYQNEVEGVAIQEK.L	43.2/6.65	0.318	> 0.124	2.6	Catalyzing reduction
13	Annexin A2 isoform 2	4757756	K.LSLEGDHSTPPSAYGSVKA	43.8/7.28	0.146	> 0.061	2.4	Signal transduction
14	Aldo-keto reductase family 1, member B10	223468663	K.MPIVGLGTWK.S	41.4/7.54	0.246	> 0.088	2.8	Catalyzing reduction
15	Chain A, crystal structure of human glutathione S-transferase P1-1 (class Pi glutathione S-transferase)	2554831	M.PPYTVVYFPVR.G	29.7/5.68	0.333	> 0.119	2.8	Antioxidation
16	Peroxiredoxin 1	55959887	K.QGGLGPMNIPLVSDPK.R	28.7/8.45	0.332	> 0.077	4.3	Antioxidation
17	Chain A, human platelet profilin complexed with an L-pro10-iodotyrosine peptide	5542165	K.DSPSVWAAVPGK.T	12.5/7.78	0.421	> 0.064	6.5	Unknown
18	hCG2015932, isoform CRA_b	119617443	R.SMYVLSLVETEK.M	12.9/7.42	0.362	> 0.124	2.9	Unknown
19	Aldo-keto reductase family 1, member C2	4503285	K.LNDGHFMPVLGFGTYAPAEVPK.S	46.8/6.88	0.167	> 0.076	2.2	Catalyzing reduction
20	Receptor protein-tyrosine kinase	551610	R.GAHRHIVEMK.F	42.9/5.41	0.530	> 0.260	2.0	Signal transduction
21	SET	145843637	R.LNEQASEEILK.V	46.2/4.78	0.580	> 0.290	2.0	Modification

Untreat: TiO₂-untreated cell lysate; Treat: TiO₂ (4 µg/ml)-treated cell lysate.

A. Rabbit anti-GSTP1 Ab



B. Rabbit polyclonal phospho-serine Ab



C. Rabbit polyclonal phospho-tyrosine Ab

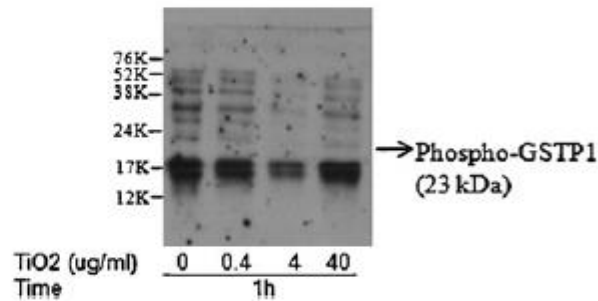


Figure 2. GSTP1 expression in TiO₂-treated and TiO₂-untreated groups.

In summary, our data demonstrate that TiO₂ particles activate and deactivate the phosphorylation of several inflammatory proteins of lung epithelial cells, especially the serine and tyrosine phosphorylation of GSTP1, which can regulate cell damage and apoptosis following exposure to TiO₂ particles. Collectively, our data suggest that **GSTP1 is an important modulator of TiO₂-particle inflammation**, and further studies will be required to enhance our understanding of GSTP1 phosphorylation in the cellular response to airborne particulate-induced airway inflammation.

Particle-induced expression of SF20/IL25 is mediated by ROS and NF- κ B in alveolar macrophages

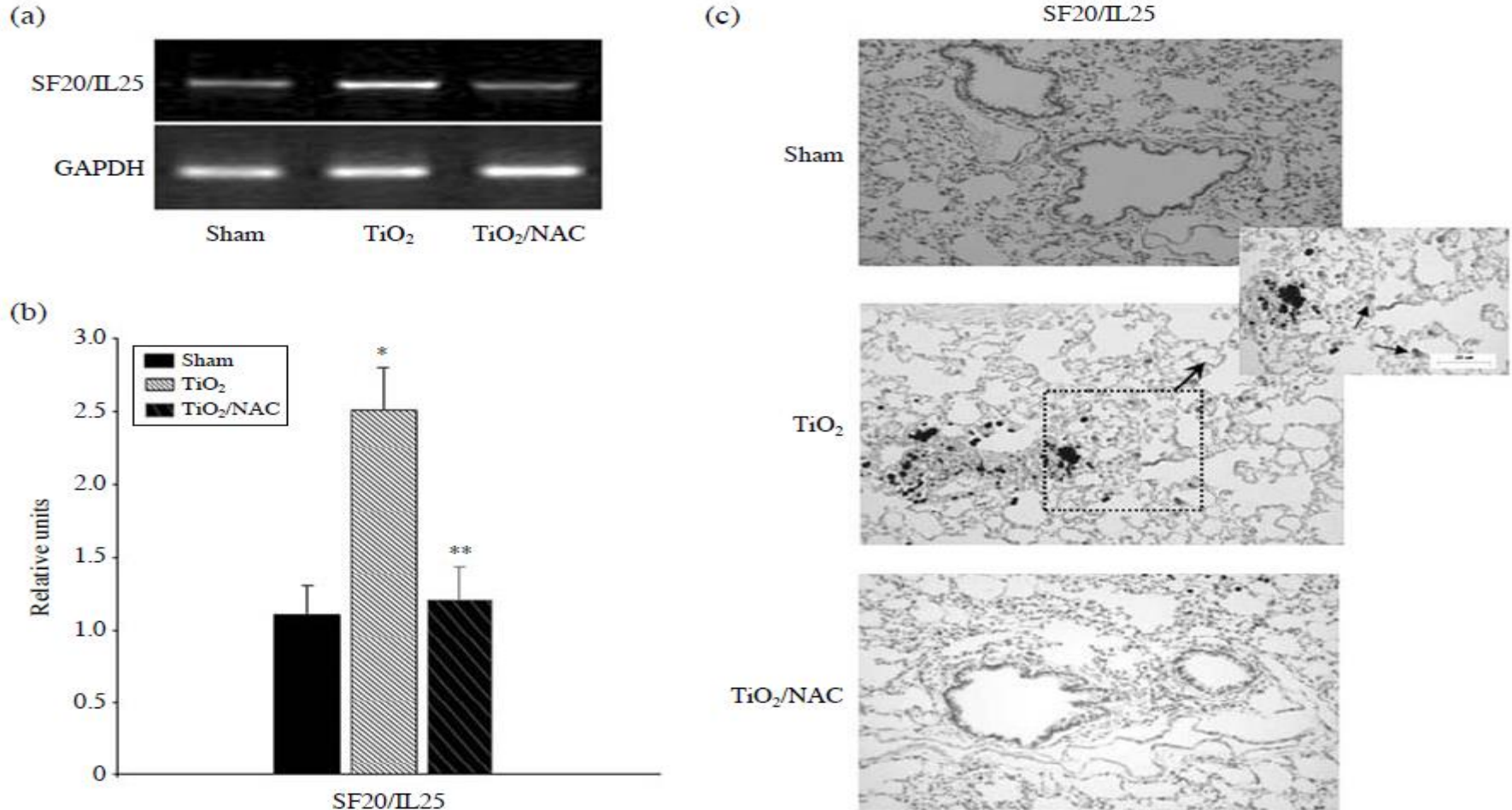


Figure 2. Effect of ROS inhibition on SF20/IL25 gene expression induced by TiO₂ particle instillation in vivo

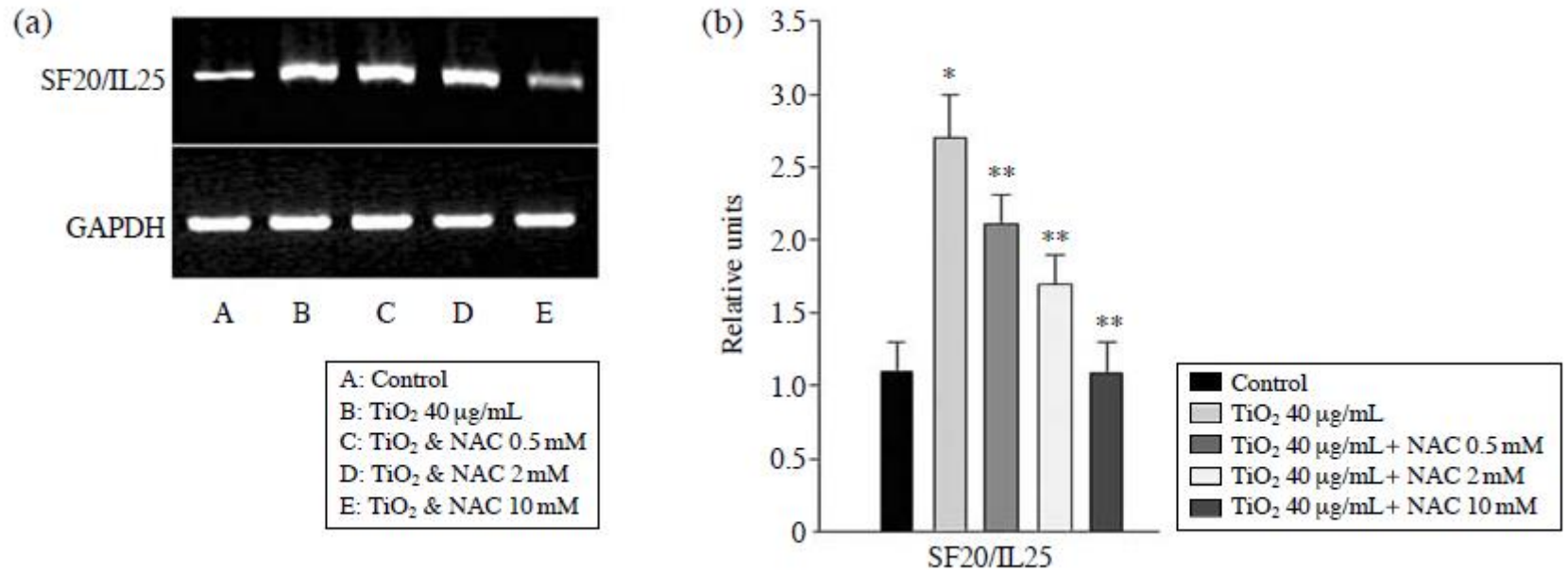


Figure 3. Effect of ROS inhibition on SF20/IL25 gene expression in TiO₂ particle-treated rat alveolar macrophages in vitro.



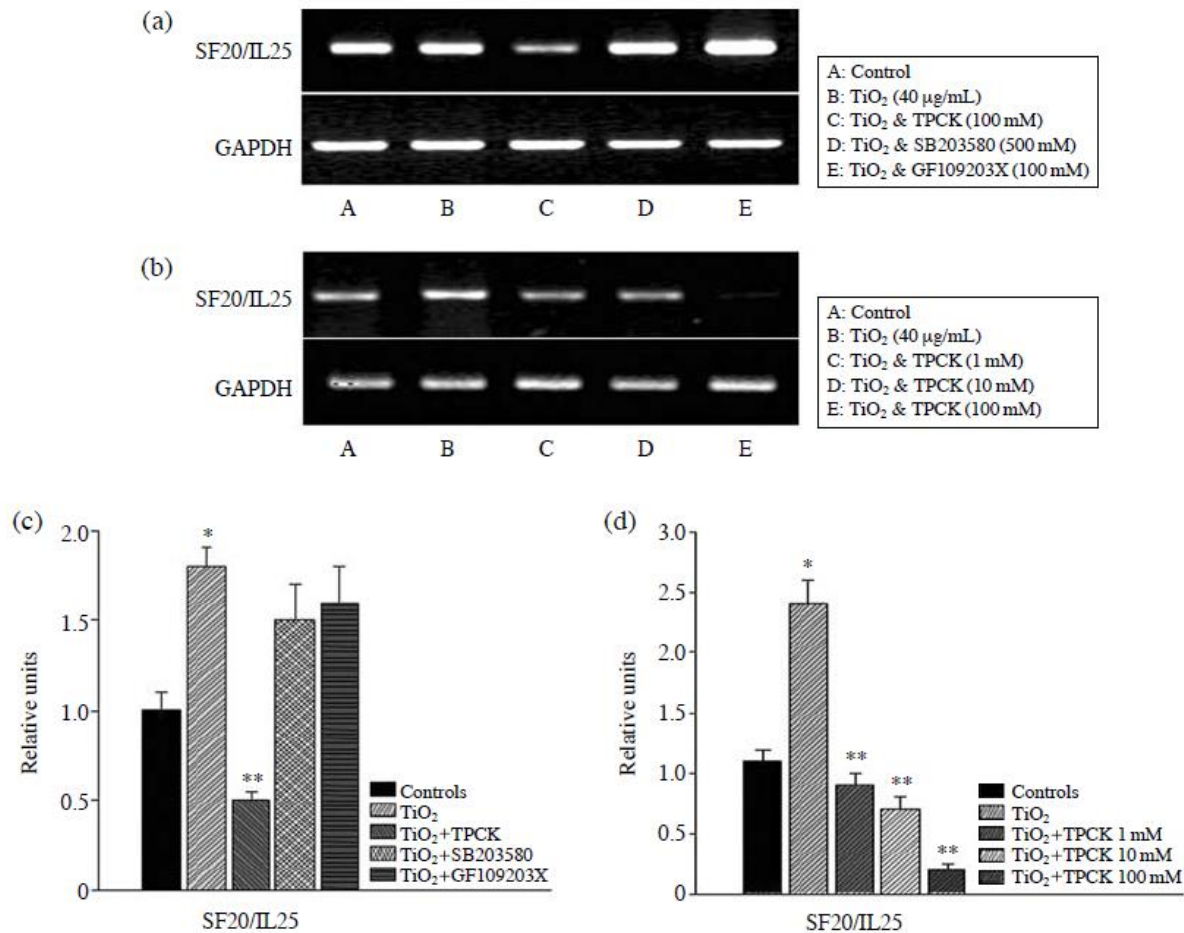


Figure 4. Effects of inhibition of NF- κ B, p38 MAP kinase, and PKC signaling on SF20/IL25 gene expression in TiO₂ particle treated rat alveolar macrophages.

In conclusion, **ROS** and the **NF- κ B signaling pathway** are involved in the upregulation of SF20/IL25 production by alveolar macrophages in the process of particle induced lung injury.

Proteomic Identification of Macrophage Migration-inhibitory Factor upon Exposure to TiO₂ Particles

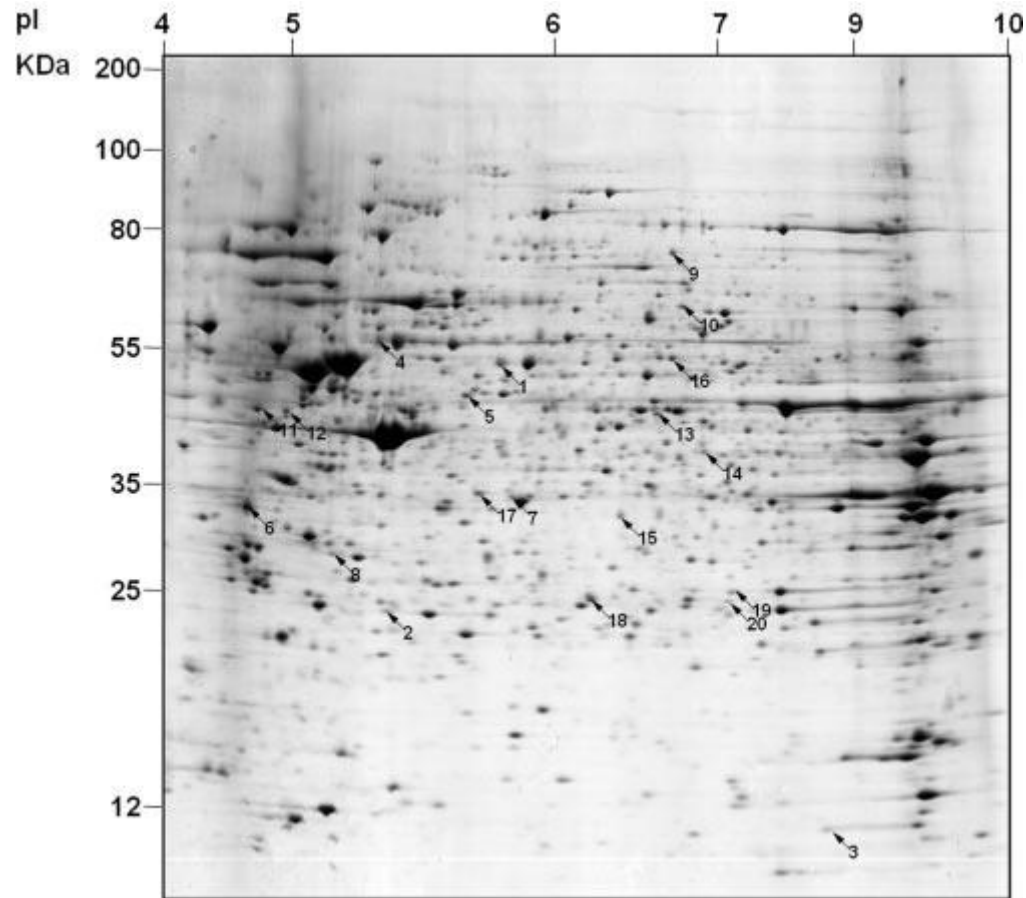


Figure 1. Two-dimensional electrophoresis of BEAS-2B cell lysate proteins.



TABLE I
List of proteins identified by LC-MS/MS analysis

No.	Protein name	Abbreviation	Accession no.	Amino acid sequence	pI/molecular mass (Da)
Group 1					
1	ATPase, H ⁺ -transporting	ATP6V1B2	19913428	(K)AVVQVFEGTSGIDAK(K)	5.4/55,401.6
2	Keratin 6A	K6A	15559584	(K)ADTLTDEINFLR(A)	8.09/60,018.2
3	Macrophage migration-inhibitory factor	MIF	30583135	(K)LLCGLLAER(L)	7.73/12,476.4
Group 2					
4	Heat-shock 60-kDa protein 1	HSPD1	31542947	(K)VGEVIVTKDDAmLLK(G)	5.7/61,055.0
5	RuvB-like 2	RUVBL2	5730023	(R)ALESDmAPVLIMATNR(G)	5.49/51,156.8
6	Proliferating cell nuclear antigen	PCNA	33239451	(R)DLSHIGDAVVISCAK(D)	4.57/28,769.0
7	Transaldolase 1	TALDO1	16307182	(K)ALAGCDFLTISP(K)	9.07/35,329.0
8	Chloride intracellular channel 1	CLIC1	14251209	(K)LAALNPESNTAGLDIFAK(F)	5.09/259,229
Group 3					
9	Replication licensing factor MCM7	MCM7	20981696	(R)TQRPADVIFATVR(E)	6.08/81,281.4
10	Calpain 1	CAPN1	12408656	(R)DMETIGFAVYEVPELVGQPAVHLKR(D)	5.49/81,890.5
11	Ribonuclease/angiogenin inhibitor	RNH1	15029922	(K)ELSLAGNELGDEGAR(L)	4.83/48,368.0
12	Vimentin	VIM	62414289	(K)FADLSEAANR(N)	5.00/52,438.6
13	26 S proteasome subunit 9	PSMD9	2150046	(R)DIQENDEEAVQVK(E)	6.08/47,448.0
14	Actin-related protein 2	ACTR2	15778930	(K)HIVLSGGSTMYPGLPSR(L)	6.29/44,761.0
15	26 S proteasome-associated pad1 homologue	PSMD14	5031981	(R)AVAVVVDPIQSVK(G)	6.06/34,577.3
Group 4					
16	PRP19/PSO4 pre-mRNA processing factor 19 homologue	PRP19	7657381	(R)QELSHALYQHDAACR(V)	6.14/55,181.1
17	Ribosomal protein P0	RPLP0	12654583	(K)TSFFQALGITTK(I)	5.42/34,274
18	Heat-shock 27-kDa protein 1	HSPB1	4504517	(R)LFDQAFGLPR(L)	5.98/22,782.6
19	Phosphoglycerate mutase 1	PGAM1	38566176	(R)VLIAAHGNSLR(G)	6.68/28,520.1
20	Platelet-activating factor acetylhydrolase	PAFAH1B3	4505587	(R)VVLGLLPR(G)	6.33/25,734.4

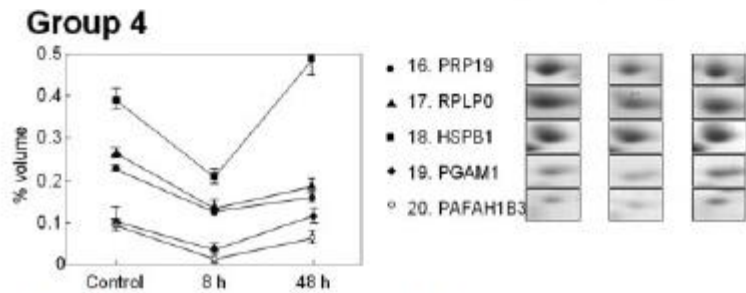
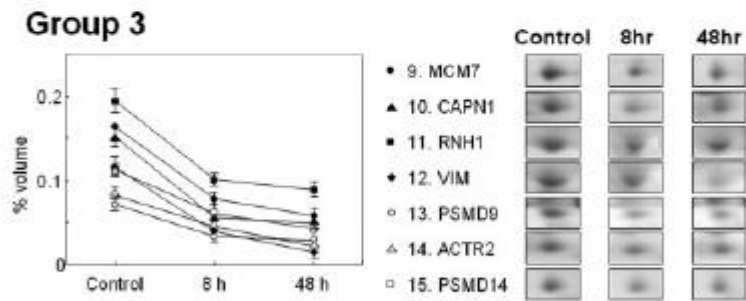
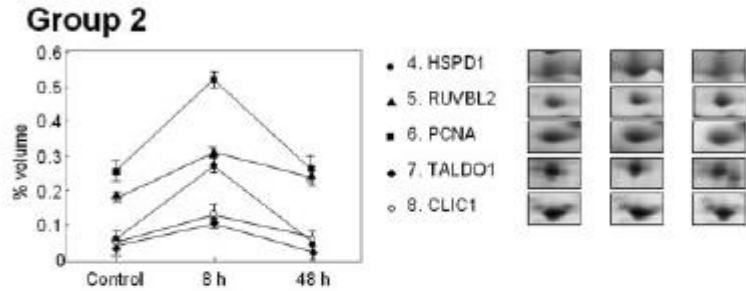
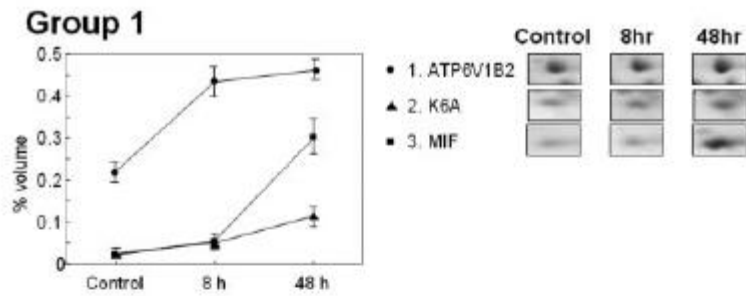


Figure 2. Cluster analysis of 20 proteins with significant differential expression (>2-fold change) at 8 or 48 h caused by TiO₂ treatment of BEAS-2B epithelial cells.



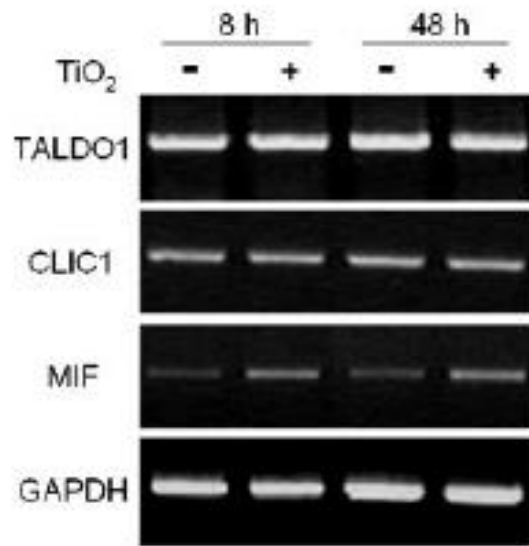


Figure 3. Semi-quantitative RT-PCR analysis of TALDO1, CLIC1, and MIF mRNA expression.

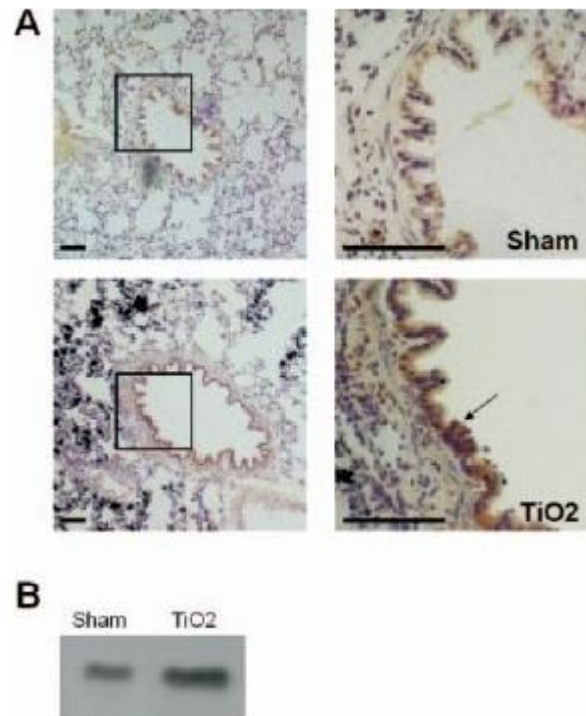


Figure 4. Expression of MIF in lung tissue of TiO₂-treated and sham-treated rats.



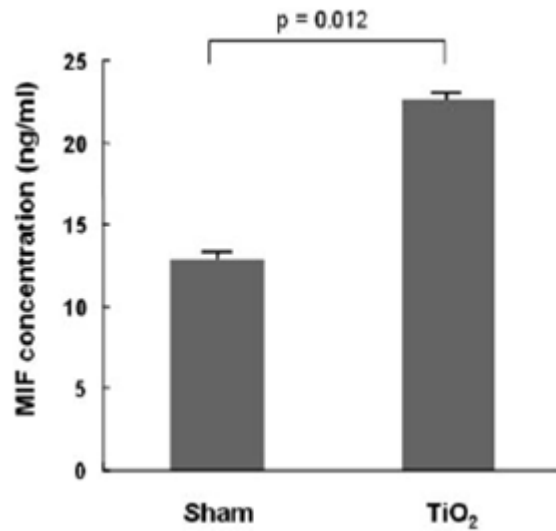


Figure 5. Comparison of MIF levels in bronchoalveolar lavage fluid from TiO₂- and sham-treated rats (n = 8 for each group).

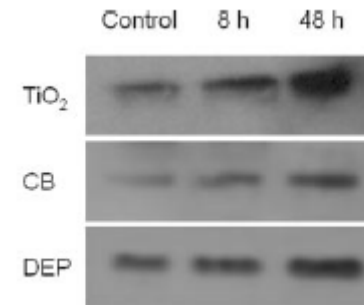


Figure 6. Western blot detection of MIF protein in cytoplasmic fractions of BEAS-2B cells stimulated with 20 g/ml of TiO₂, carbon black (CB), or diesel exhaust particles (DEP) for 8 and 48 h.

In summary, we identified 20 proteins whose expression levels in the BEAS-2B cell line changed in response to TiO₂ particle exposure. These proteins include defense-related, cell-activating, and cytoskeletal proteins implicated in the response to oxidative stress, and they can be classified into four groups according to the pattern of their TiO₂-induced change in expression over time. One of these proteins, MIF, was induced at the transcriptional level by stimulation of cells with any one of three different particulate molecules, and expression of **MIF protein** was increased in the lungs of TiO₂-instilled rats. These results indicate that some of these proteins may serve as **mediators of, or markers for, airway disease caused by exposure to PM**.

IL-25 and IL-13 Production by Alveolar Macrophages in Response to Particles

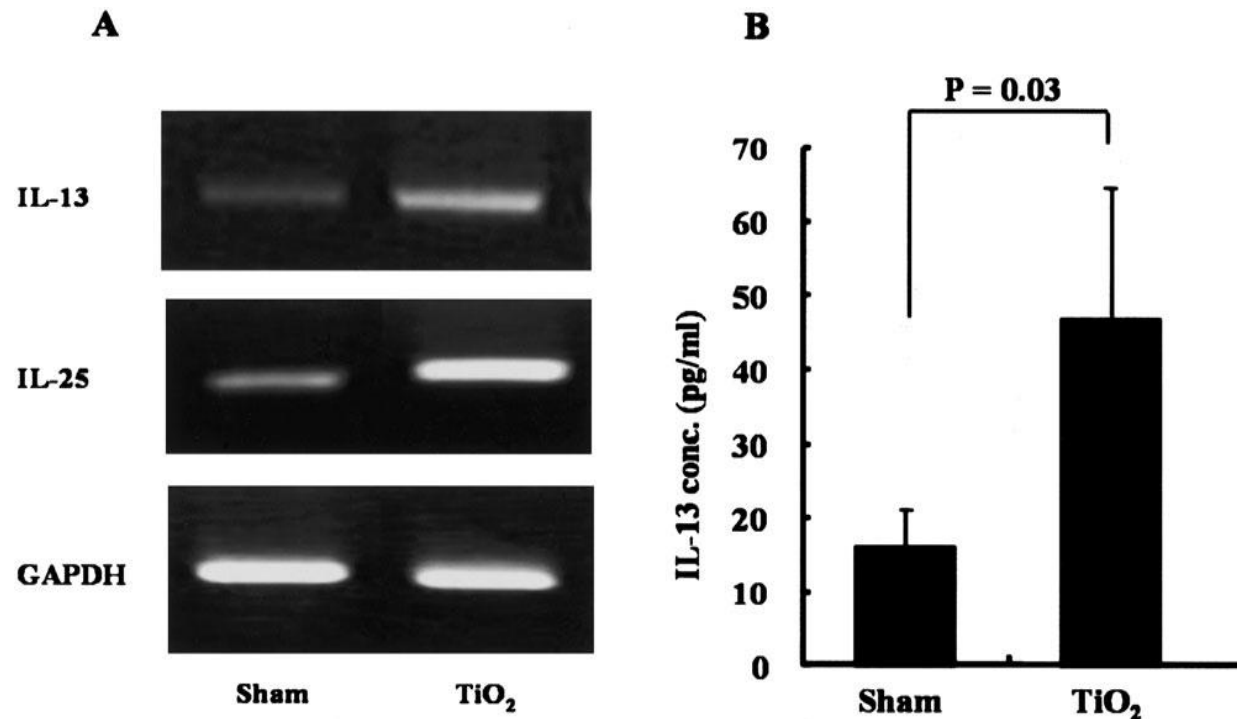


Figure 1. Comparison of the IL-13 and IL-25 mRNA expression levels in the lungs (A) and the IL-13 level in the BAL fluids (B) between TiO₂-treated and sham-treated rats

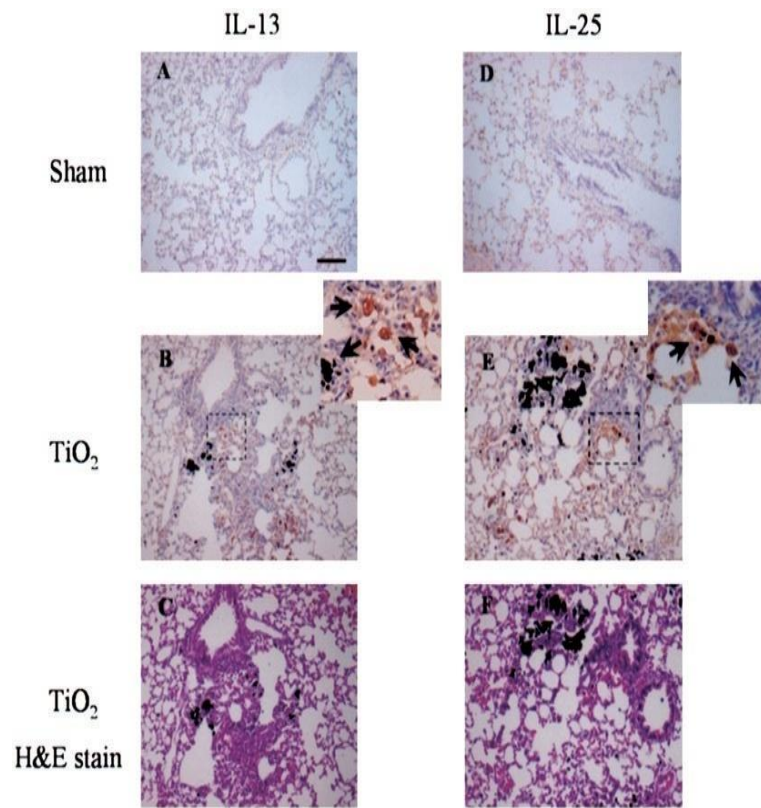


Figure 2. The expression of IL-13 and IL-25 proteins in the lungs of TiO₂-treated and sham-treated rats

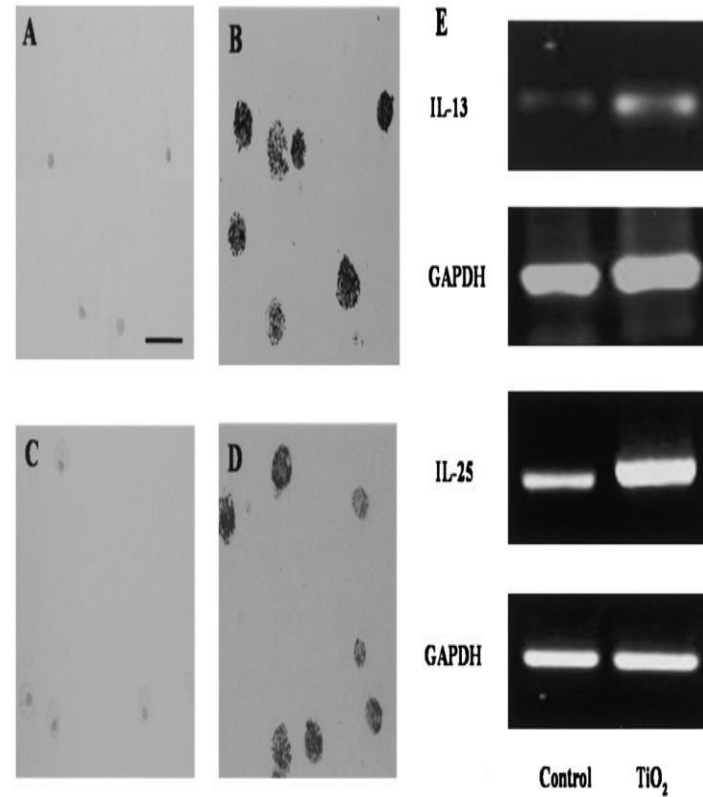


Figure 3. In vitro expression levels of IL-13 and IL-25 mRNA, and protein, by cultured alveolar macrophages that were stimulated with TiO₂ particles



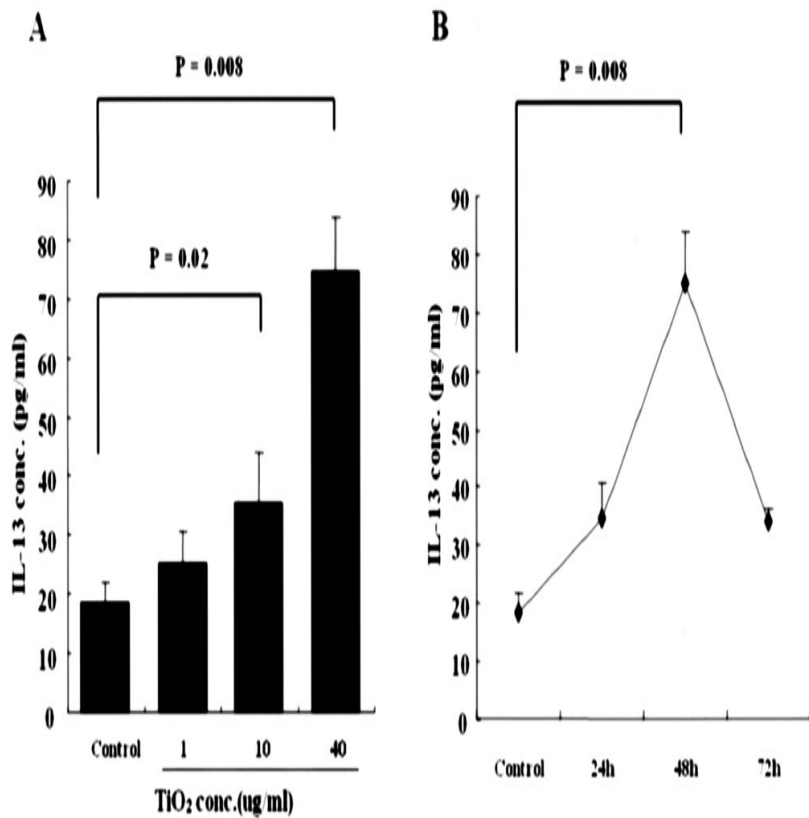


Figure 4. Time and dose responses of IL-13 production by macrophages exposed to TiO₂ particles

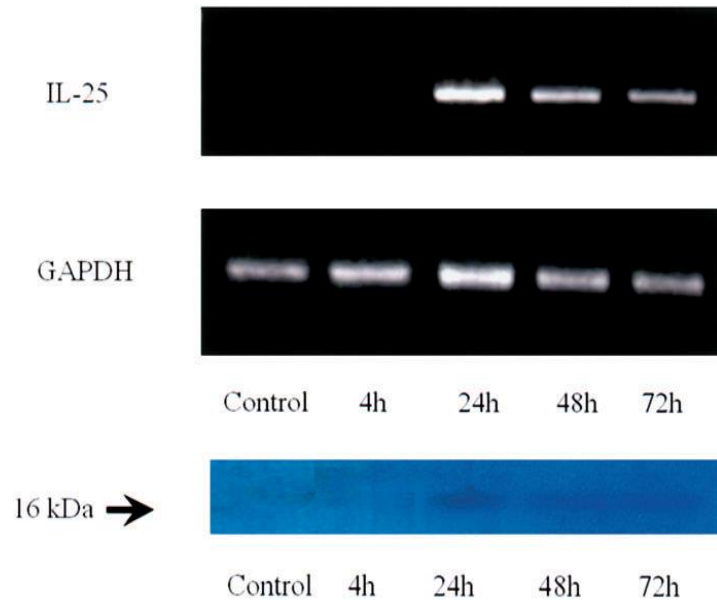
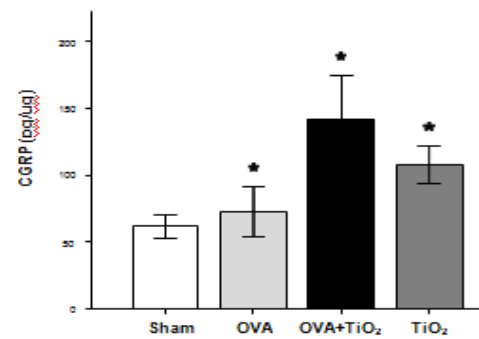
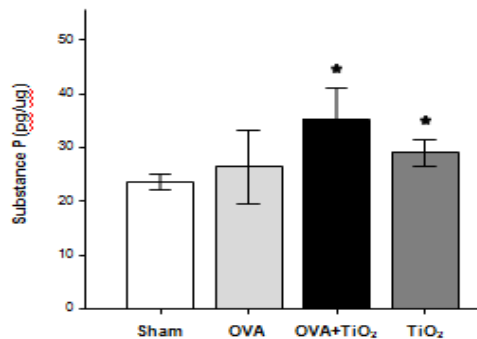
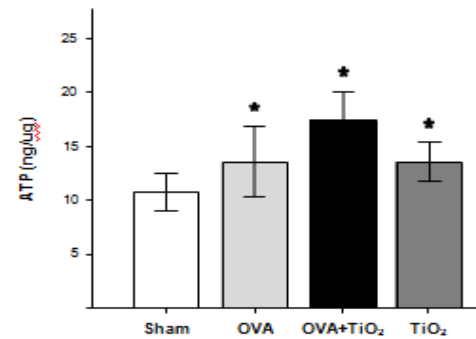
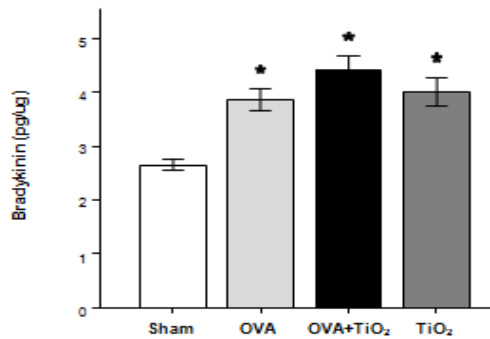
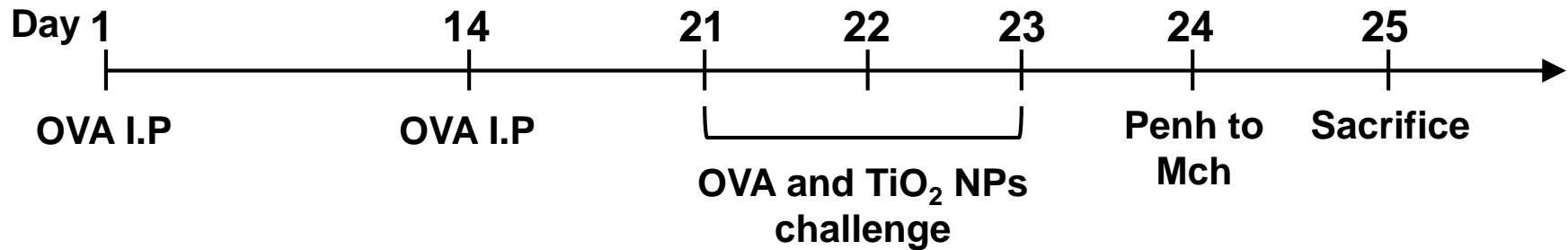
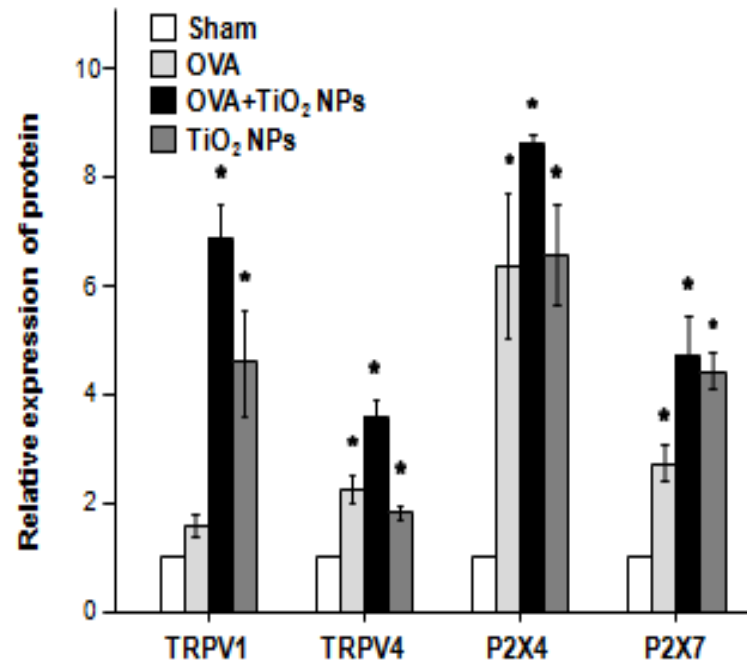
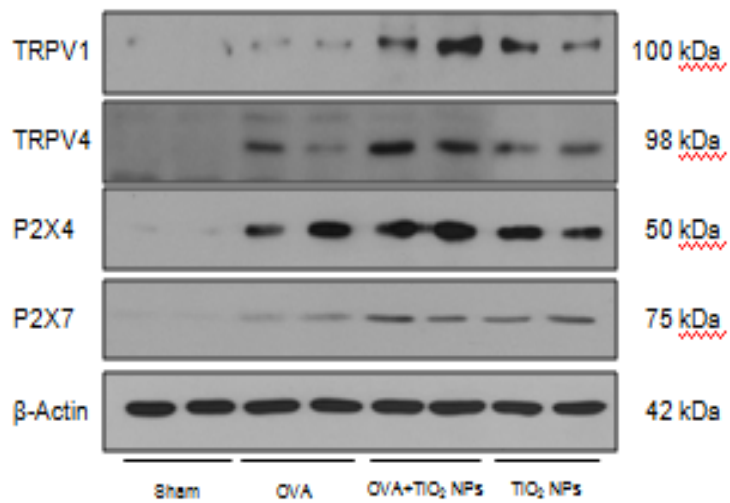


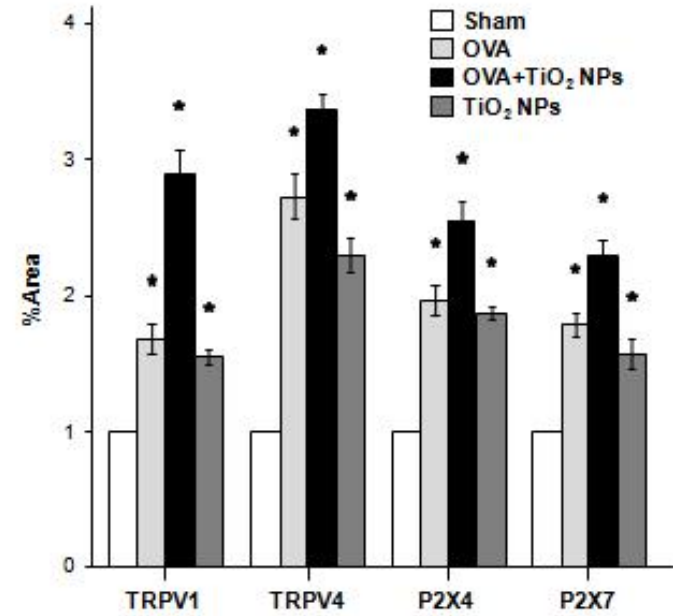
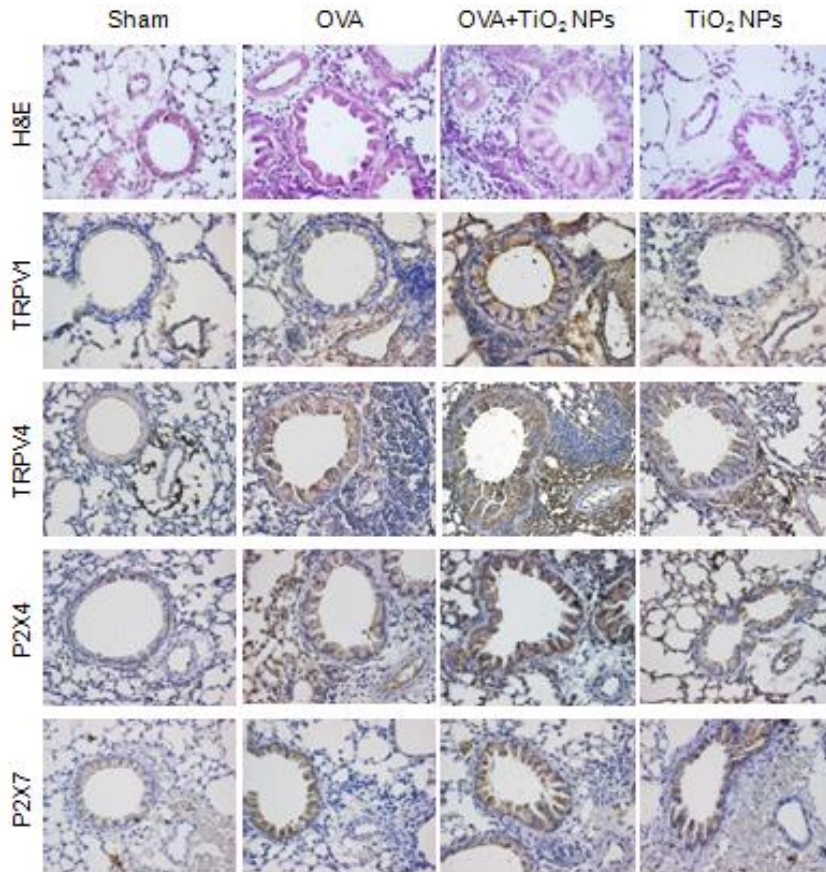
Figure 5. Kinetics of IL-25 production by alveolar macrophages that were stimulated with TiO₂.

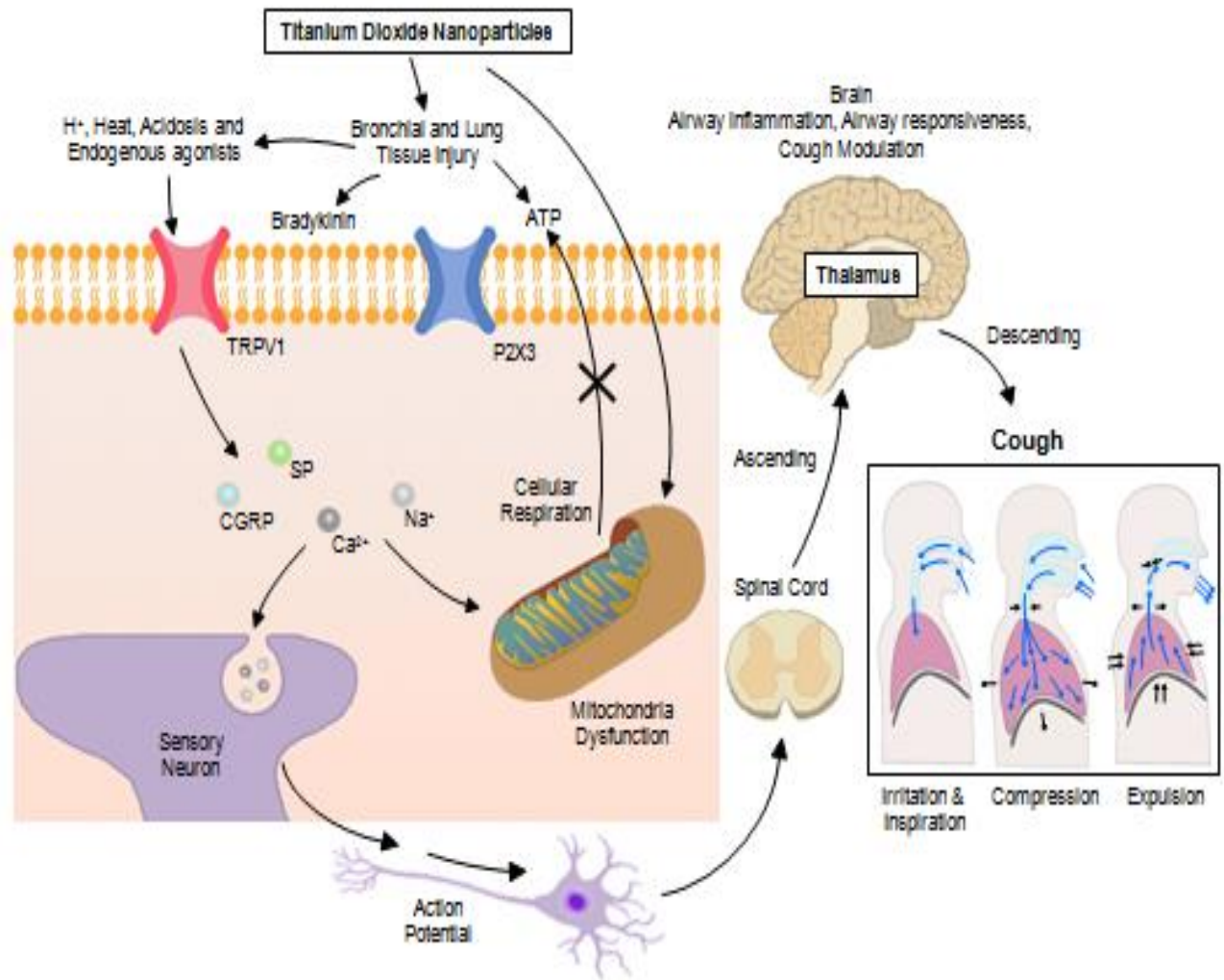
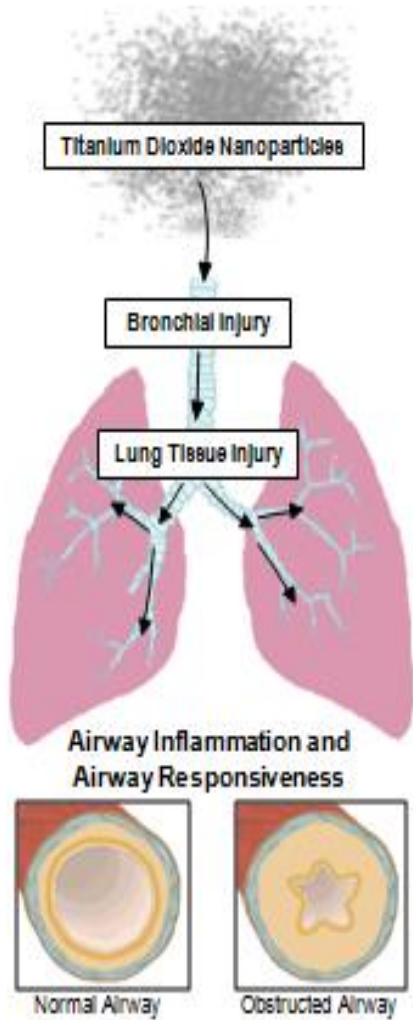
In summary, we show that alveolar macrophages produce IL-25 and IL-13 at the protein and mRNA levels in response to particles. The increased expression of **IL-25 and IL-13 mRNAs, and proteins**, may be involved in modulating the inflammatory response within the lung of TiO₂-instilled rats. It is interesting to speculate on whether the inhibition of IL-25 would have an inhibitory effect on particle-induced inflammation; these studies are currently ongoing in our laboratory.

Impact of titanium dioxide nanoparticles on neuroinflammation in a mouse model of asthma









Activation of the inflammasome

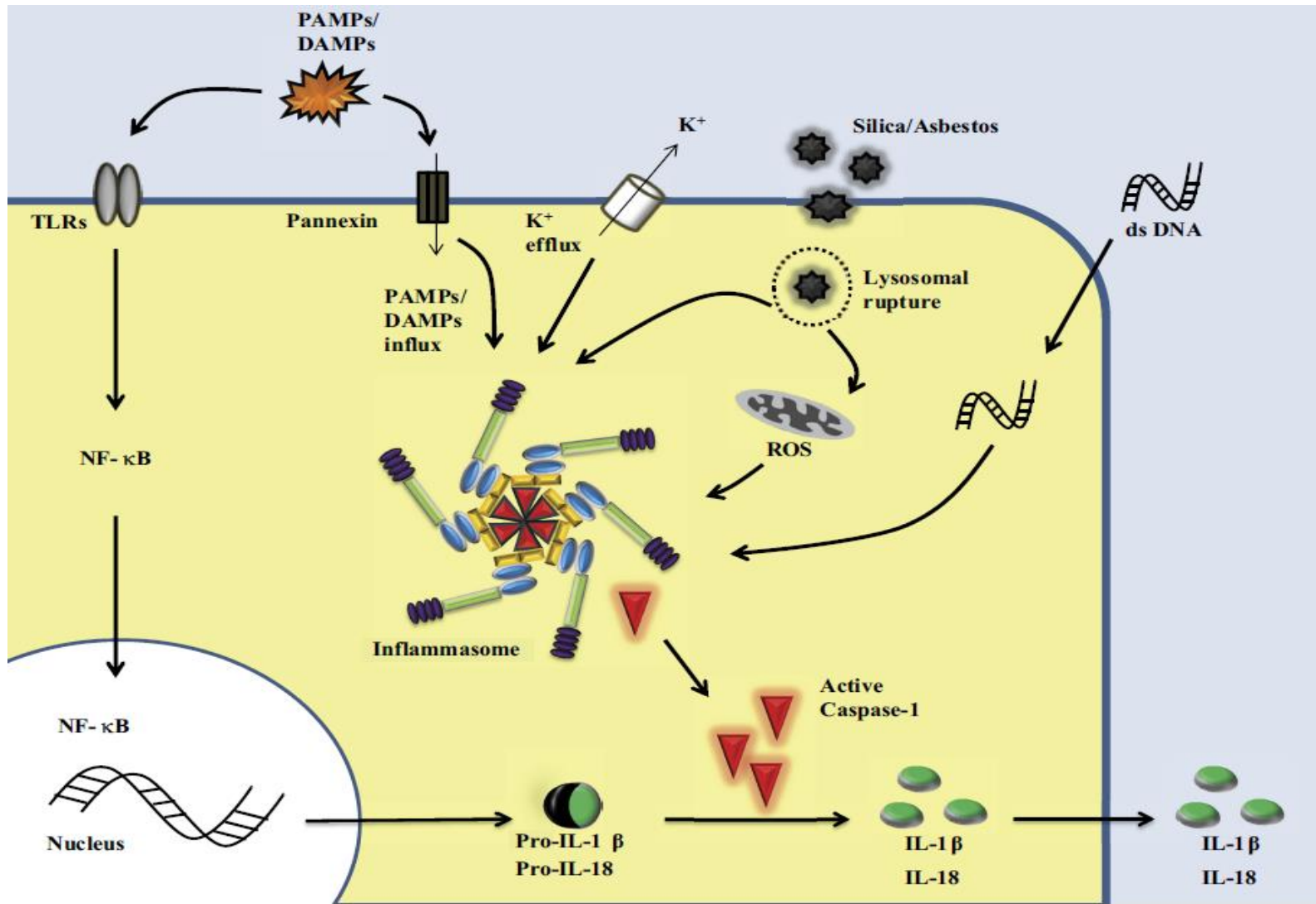
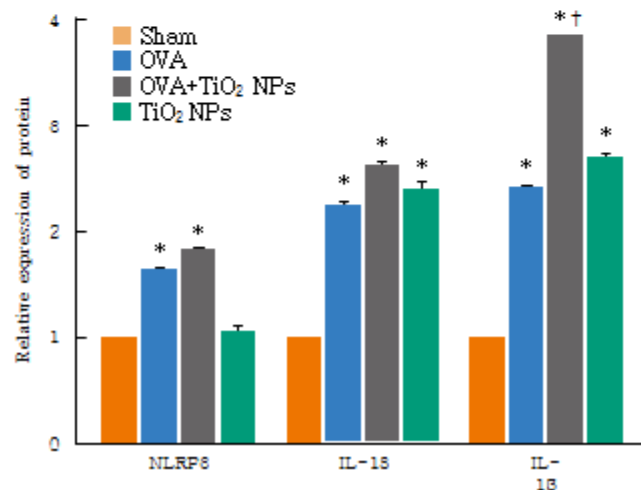
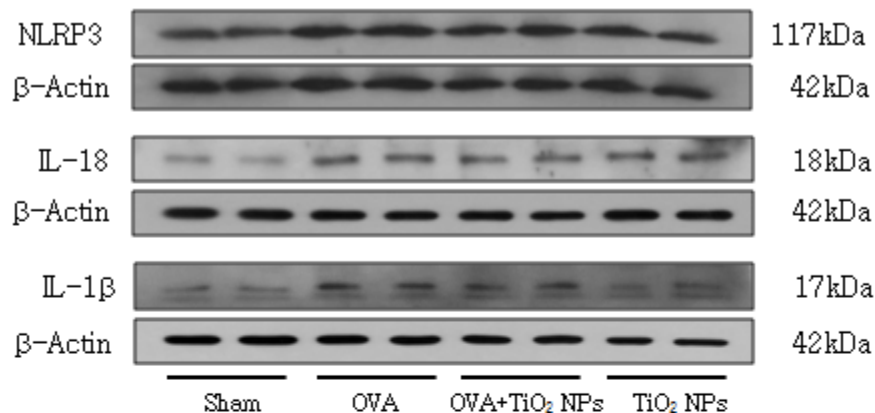
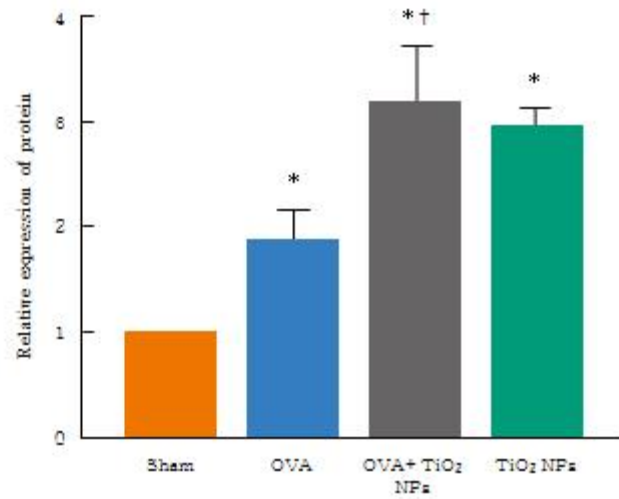
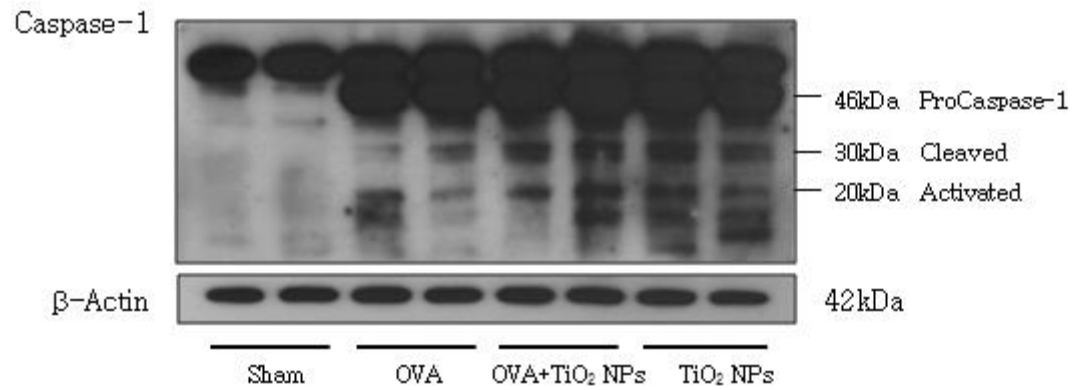
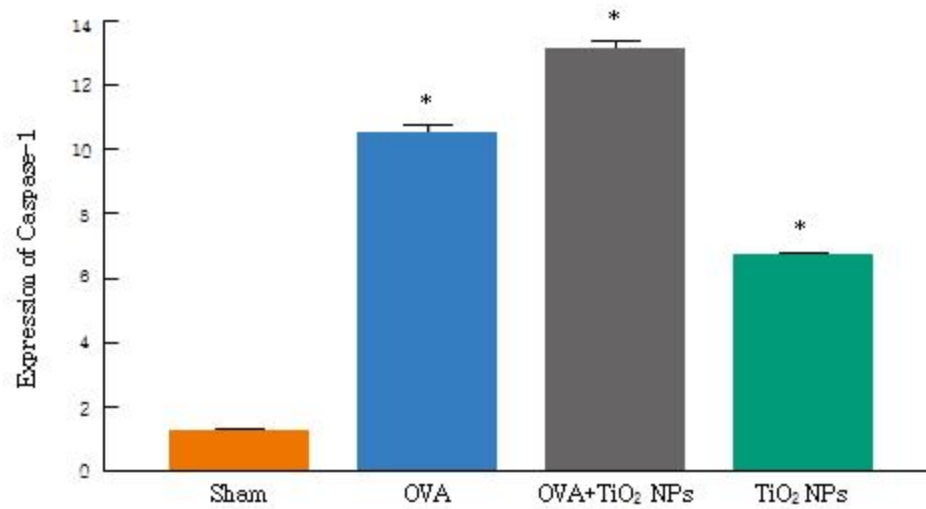
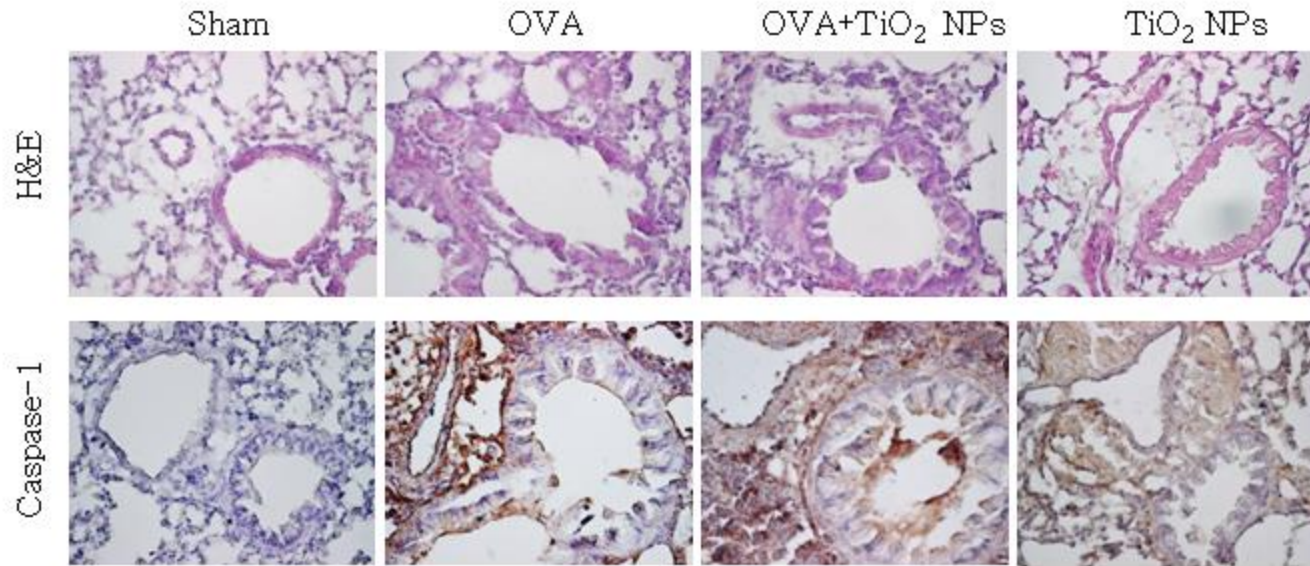


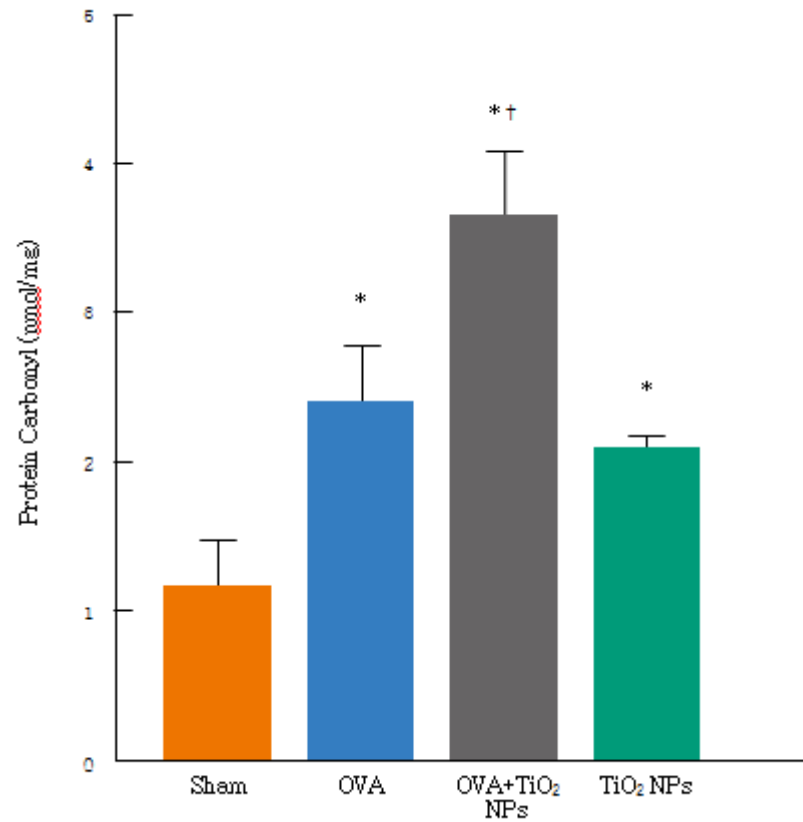
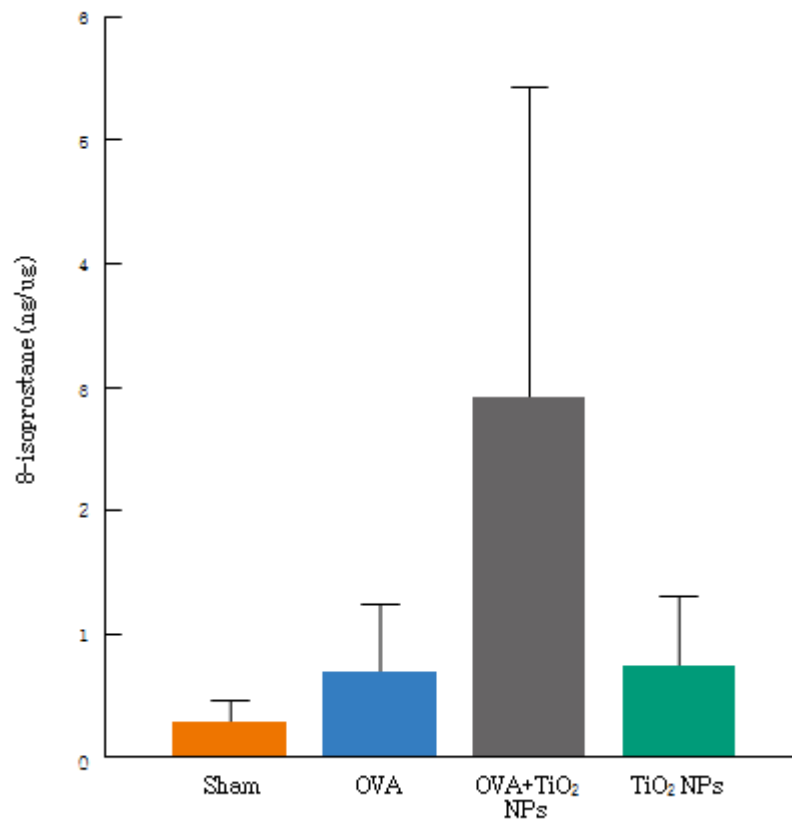
Fig. 2. Activation of the inflammasome.

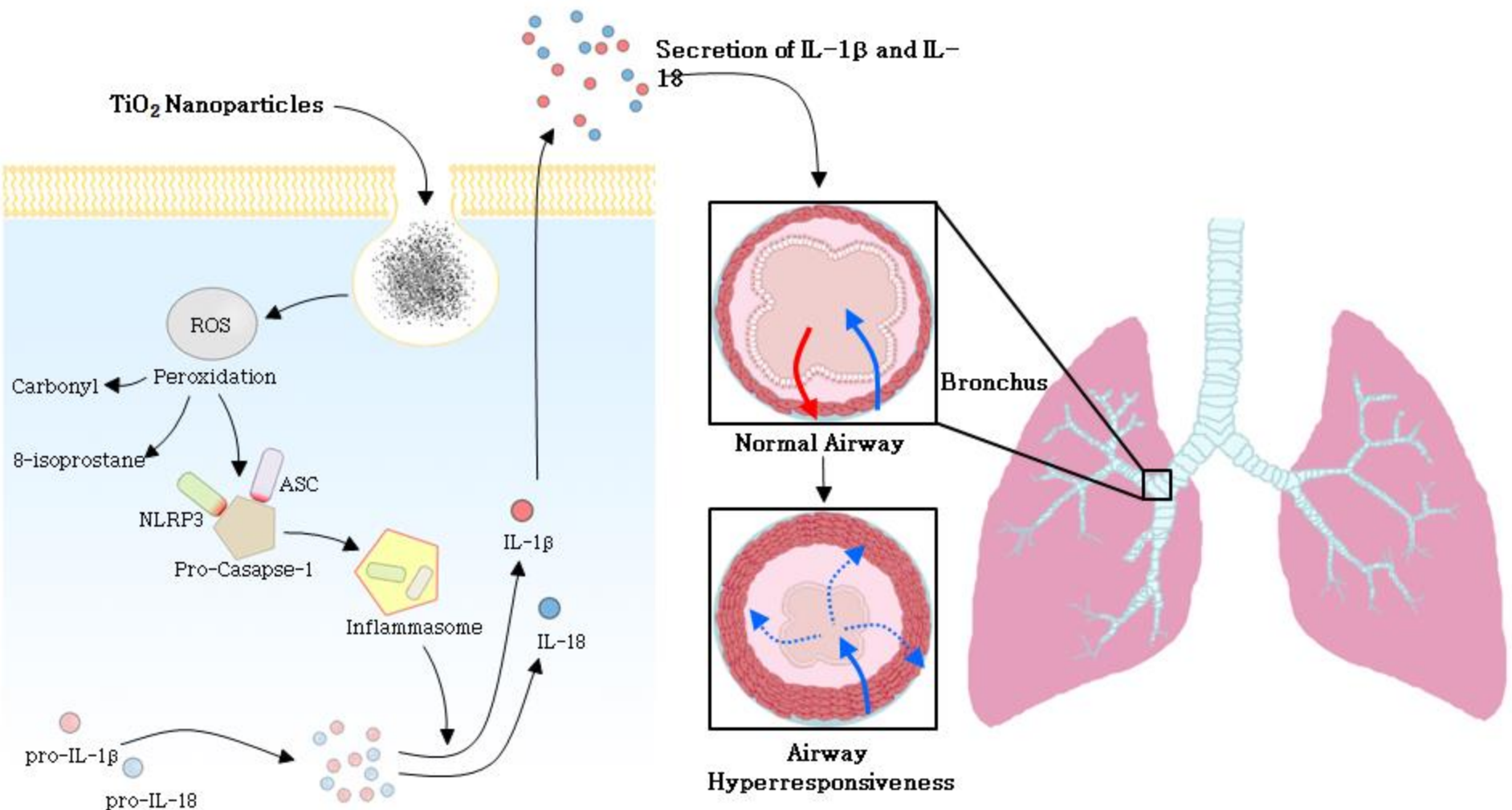
Effect of TiO₂ nanoparticles on inflammasome mediated airway inflammation and responsiveness



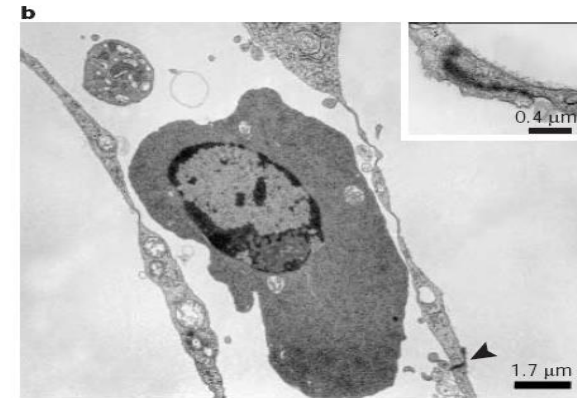
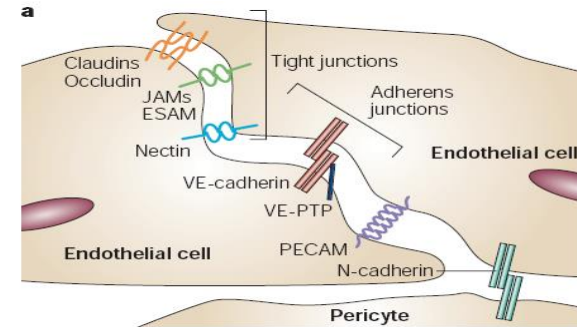
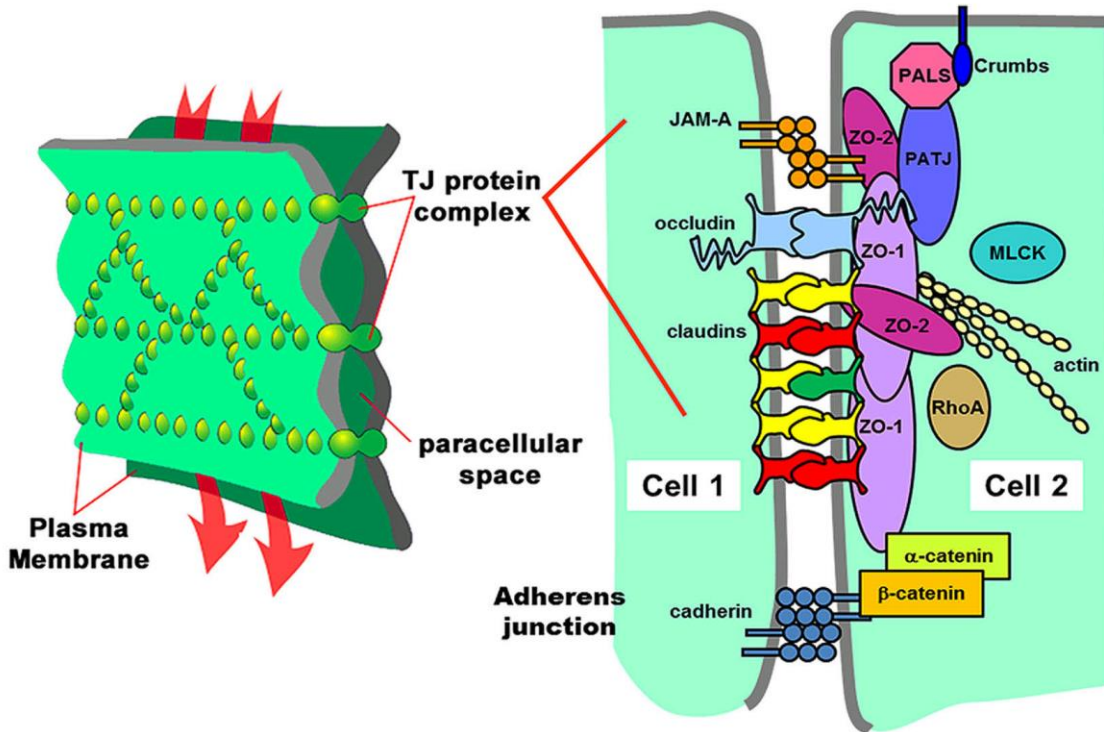








Cell barriers



Tight junction permeability is primarily controlled by proteins in the claudin family that form a seal to both restrict paracellular diffusion and permit specific transport of ions between cells across the epithelial barrier.

- ▶ Fence function
- ▶ Barrier function
- ▶ Signal transduction



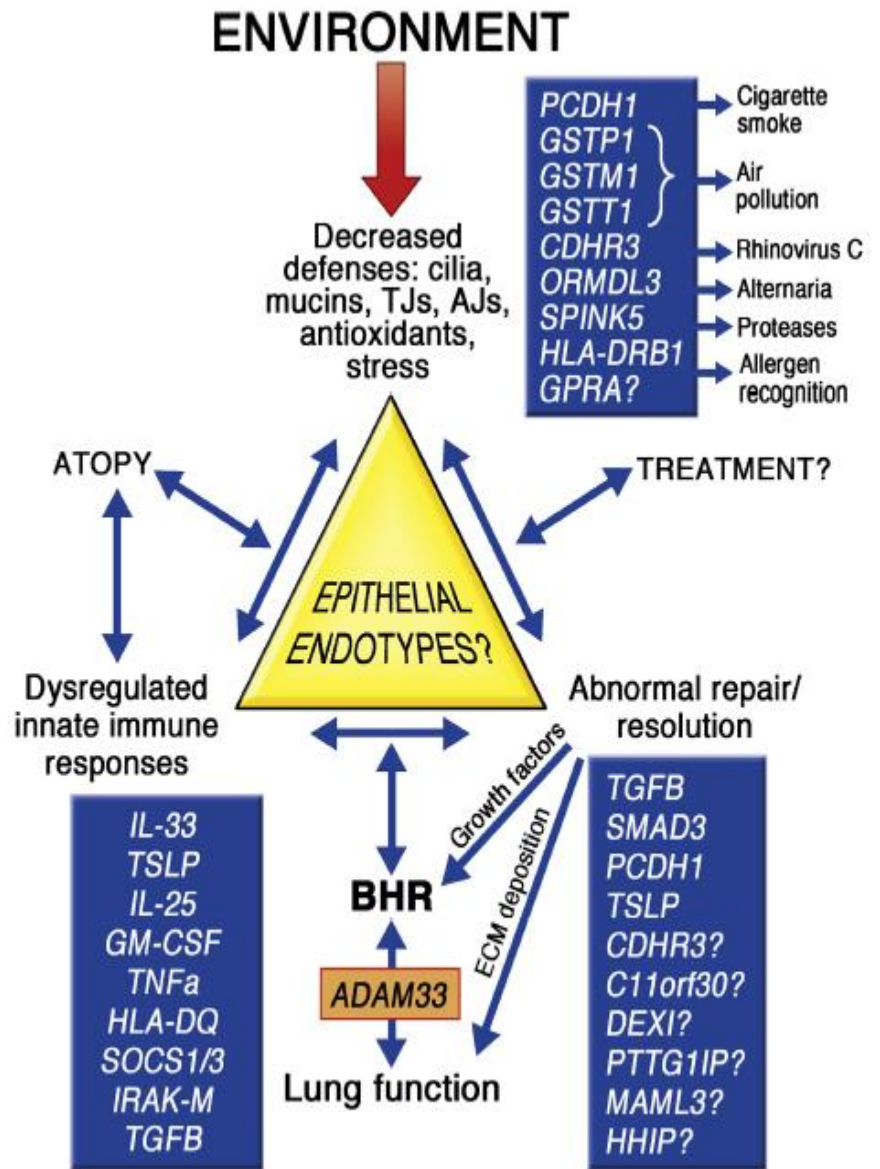
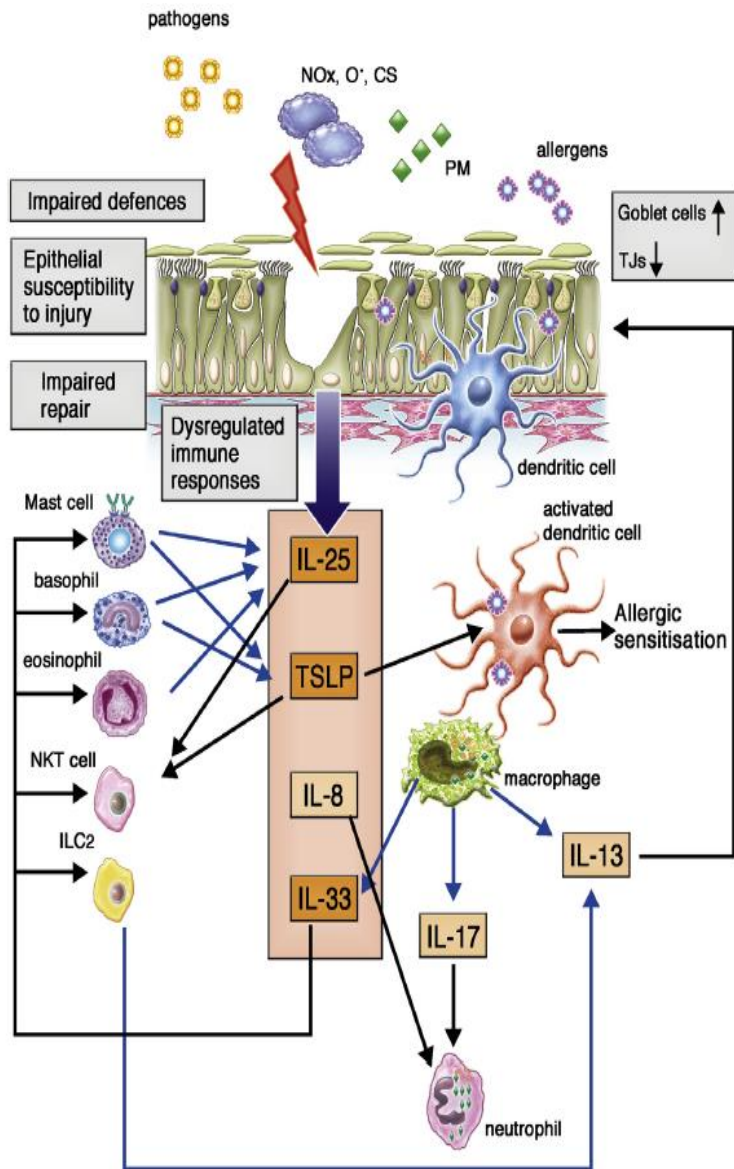
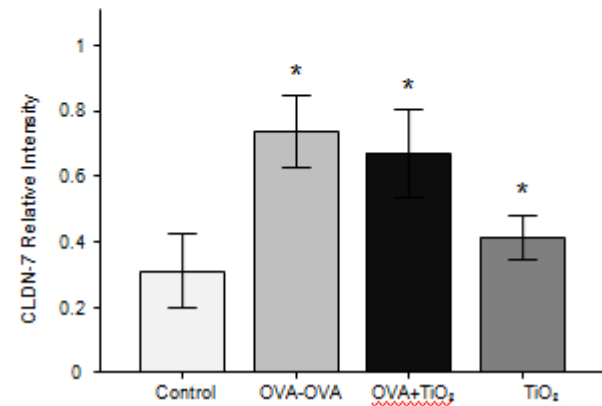
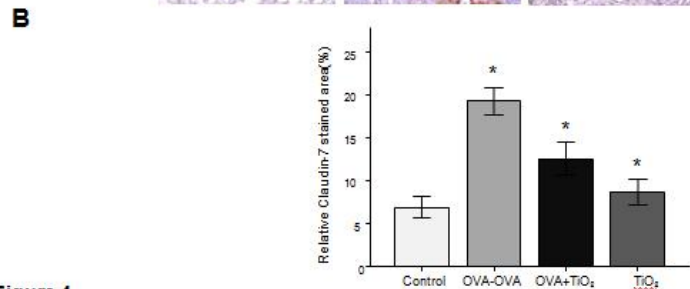
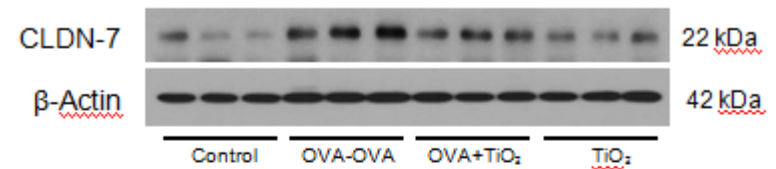
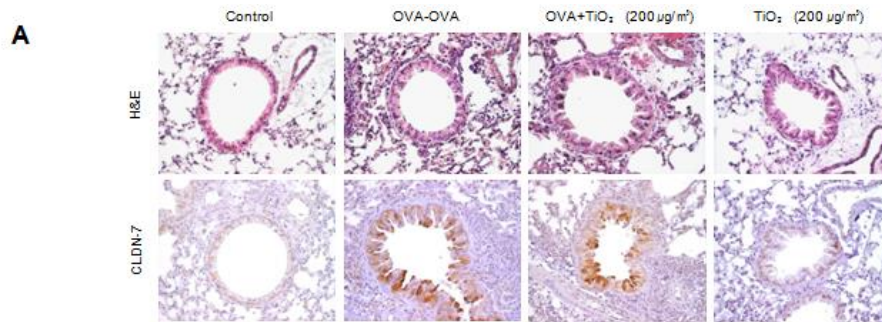
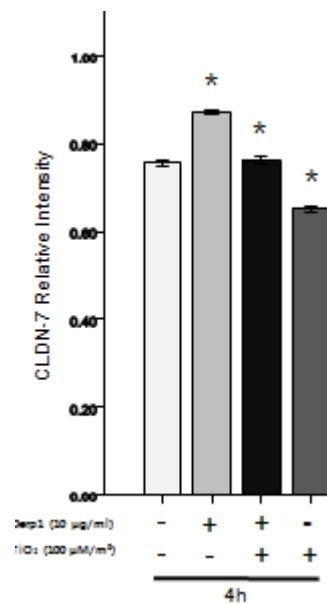
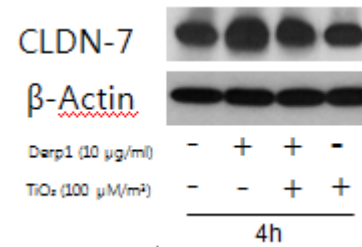
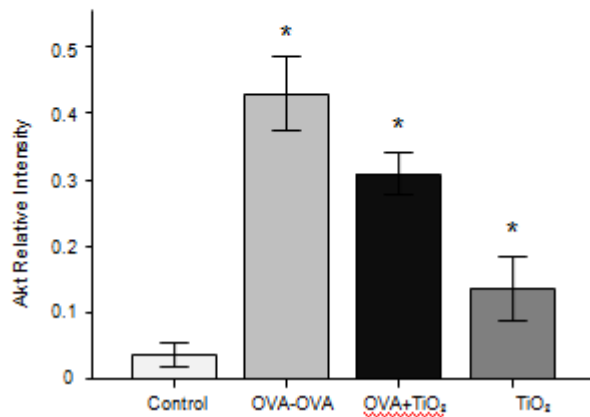
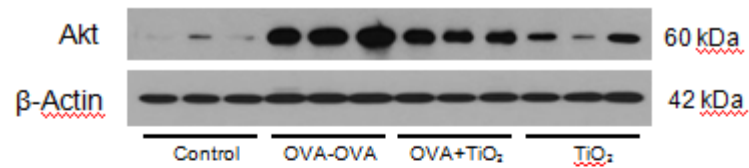
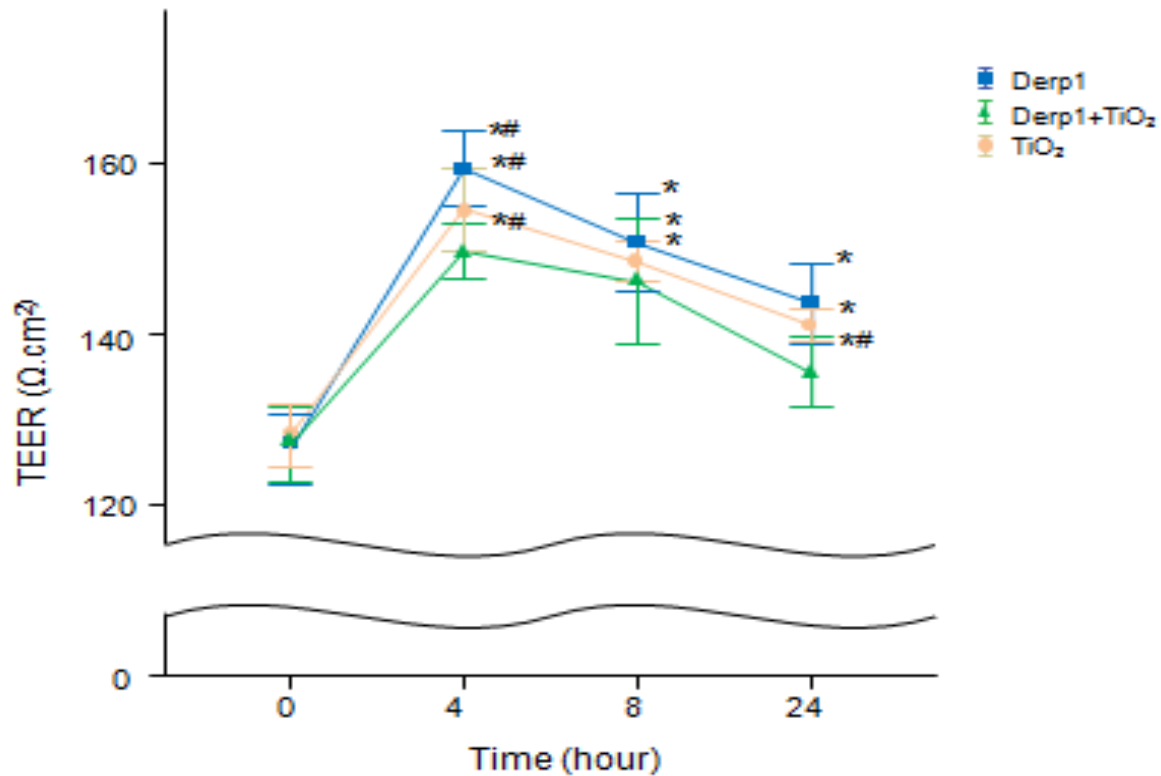


FIG 5. Potential mechanisms of asthma defined by epithelial barrier dysfunction. Identification of potential links with asthma susceptibility genes and their interaction with environmental stimuli are shown.

Titanium dioxide particles and HDM alter epithelial barrier function by change of claudin-7 in asthma







Conclusion: These data suggest that **particulate matter is an important contributor to the airway epithelial barrier dysfunction**, and lead to airway inflammation and responsiveness



Future directions

한국 대기질 예보시스템
Korean Air Quality Forecasting System

KAQFS

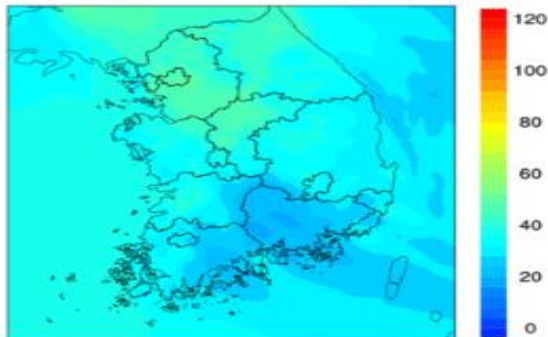
예보결과

PM10 / PM2.5 O3 NO2 SO2 CAI

오늘 예보

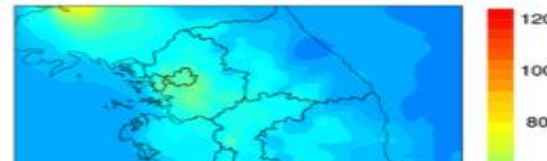
내일 예보

PM10 24Hour AVG.($\mu\text{g}/\text{m}^3$)

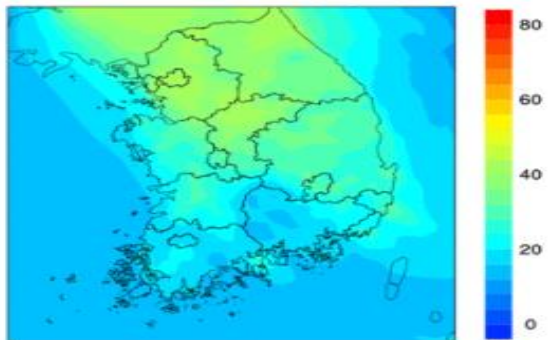


2017-01-30

PM10 24Hour AVG.($\mu\text{g}/\text{m}^3$)



PM2.5 24Hour AVG.($\mu\text{g}/\text{m}^3$)



2017-01-30



2017-01-31

주요국가의 미세먼지 환경기준

(단위 : $\mu\text{g}/\text{m}^3$)

	미세먼지(PM10)		초미세먼지(PM2.5)	
	24시간	연간	24시간	연간
WHO	50	20	25	10
한국	100	50	50	25
EU	50	40		25
독일	50	40		25
영국	50	40		25
호주	50		25	8
일본	100		35	15

▲ 자료=국립환경과학원 대기환경연보

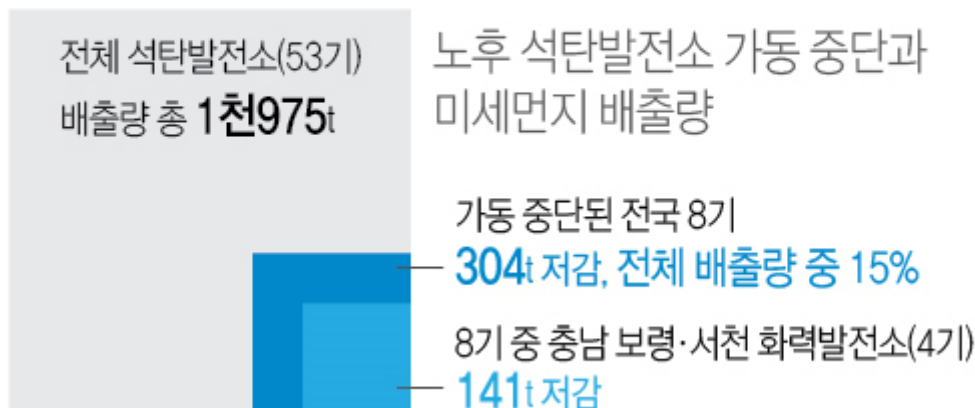
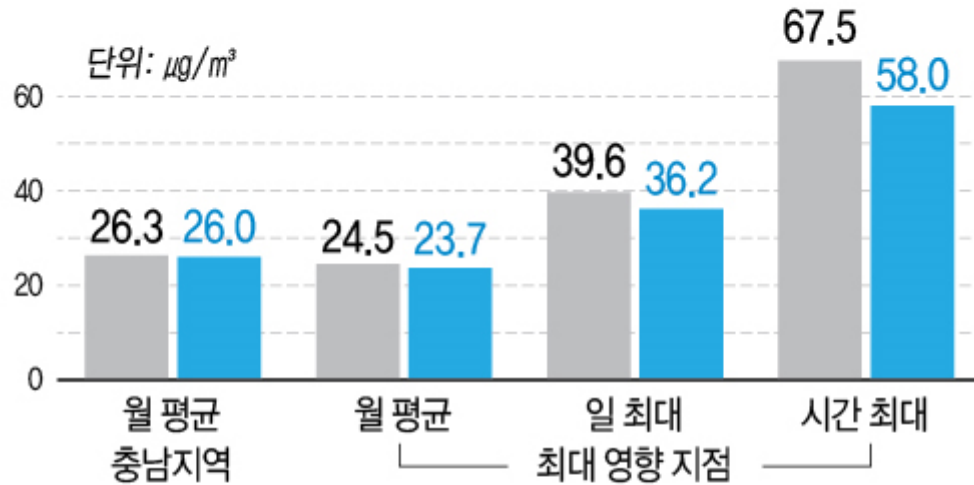


노후 석탄발전소 가동 중지 후 미세먼지 농도 변화

미세먼지 농도 감소량

중단 전 중단 후

감소율 1.1%↓ 3.3%↓ 8.6%↓ 14.1%↓



자료/ 환경부, 산업통상자원부, 국립환경과학원



대기환경지수

지수 산출방법

지수		A		B		C		D		E		F	
지수구분		좋음		보통		민감군영향		나쁨		매우나쁨		위험	
아황산가스(ppm)	1hr	0	0.020	0.021	0.050	0.051	0.100	0.101	0.150	0.151	0.400	0.401	1
이산화질소(ppm)	1hr	0	0.030	0.031	0.060	0.061	0.150	0.151	0.200	0.201	0.600	0.601	2
일산화탄소(ppm)	1hr	0	2.00	2.01	9.00	9.01	12.00	12.01	15.00	15.01	30.00	30.01	50
오존(ppm)	1hr	0	0.040	0.041	0.080	0.081	0.120	0.121	0.300	0.301	0.500	0.501	0.600
미세먼지($\mu\text{g}/\text{m}^3$)	24hr	0	30	31	80	81	120	121	200	201	300	301	600

지수구간별 개요





지수구간	지수구분	구간의미
A	좋음	대기오염 관련 질환자군에서도 영향이 유발되지 않을 수준
B	보통	환자군에게 만성 노출시 경미한 영향이 유발될 수 있는 수준
C	민감군영향	환자군 및 민감군에게 유해한 영향이 유발될 수 있는 수준
D	나쁨	환자군 및 민감군(어린이, 노약자 등)에게 유해한 영향 유발, 일반인도 건강상 불쾌감을 경험할 수 있는 수준
E	매우나쁨	환자군 및 민감군에게 급성 노출시 심각한 영향 유발, 일반인도 약한 영향이 유발될 수 있는 수준
F	위험	환자군 및 민감군에게 응급 조치가 발생되거나, 일반인에게 유해한 영향이 유발될 수 있는 수준

표현방법

	좋음 (0~50)	보통 (51~100)	민감군영향 (101~150)	나쁨 (151~250)	매우나쁨 (251~350)	위험 (351~500)
상징색	파랑	초록	노랑	주황	빨강	갈색
RGB Code	0000FF	00FF00	FFFF00	FF9900	FF0000	861313
픽토그램						



미세먼지 예보등급 및 예보내용

예보 내용		등급($\mu\text{g}/\text{m}^3$)			
		좋음 	보통 	나쁨 	매우나쁨 
예보 물질	미세먼지 (PM ₁₀)	0~30	31~80	81~150	151 이상
	미세먼지 (PM _{2.5})	0~15	16~50	51~100	101 이상
행동 요령	민감군	-	실외활동시 특별히 행동에 제약을 받을 필요는 없지만 몸 상태에 따라 유의하여 활동	장시간 또는 무리한 실외활동 제한, 특히 천식을 앓고 있는 사람이 실외에 있는 경우 흡입기를 더 자주 사용할 필요가 있음	가급적 실내활동, 실외활동시 의사와 상의
	일반인	-	-	장시간 또는 무리한 실외활동 제한, 특히 눈이 아픈 증상이 있거나 기침이나 목의 통증으로 불편한 사람은 실외활동을 피해야 함	장시간 또는 무리한 실외활동제한, 목의 통증과 기침 등의 증상이 있는 사람은 실외활동을 피해야 함

- ※ 미세먼지 예보등급은 PM₁₀과 PM_{2.5} 중 높은 등급을 기준으로 발표
- ※ 민감군 : 어린이, 노인, 천식같은 폐질환과 심장질환을 앓고 있는 어른



미세먼지 높은 날 건강생활수칙



장시간 실외활동 자제



외출 시 식약처에서
인증한 보건용마스크 착용



외출 후
손, 얼굴 깨끗이 씻기



충분한 수분섭취



과일, 채소 등
충분히 씻어 먹기

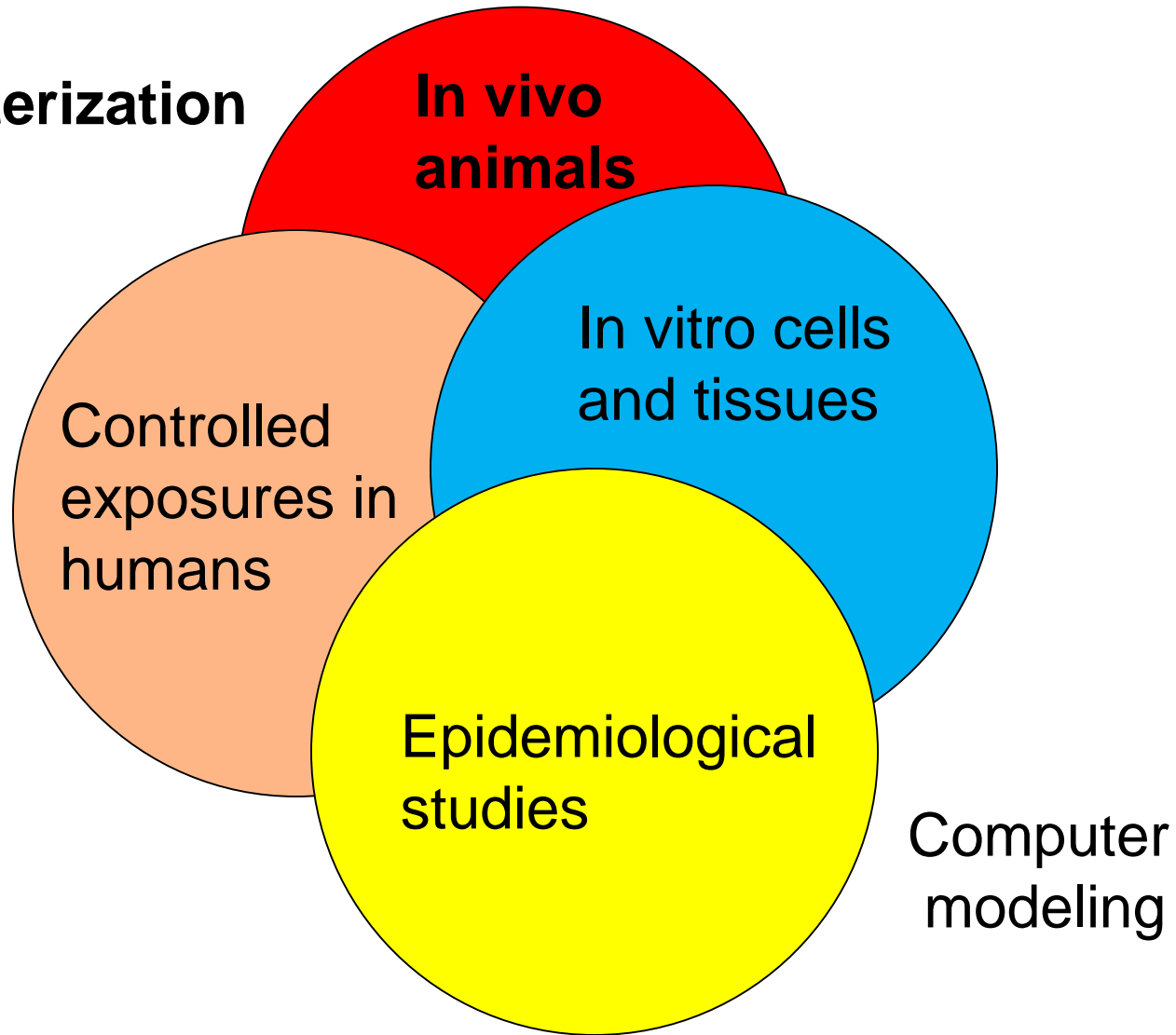


창문을 닫아 외부의
미세먼지 유입을 차단

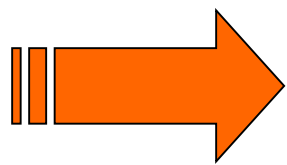
구분	한국 규격(KF) 한국산업안전공단 기준		유럽규격(FFP)		미국규격 미국 CDC 산하 국립산업안 전보건연구원(NIOSH)	
	등급	차단율	등급	차단율	등급	차단율
1	KF99(특급)	99% 이상 (염화나트륨 +파라핀오 일)	FFP3	99% 이상	N100	99.97% 이상
2	KF94(1급)	94% 이상 (염화나트륨 시험+파라핀 오일)	FFP2	94% 이상	N99	99% 이상
3	KF80(2급)	80% 이상 (염화나트륨 시험기준)	FFP1	80% 이상	N95	95% 이상



Hazard Characterization



- Particles → more information
- The effect of particles on lung → further evaluation



Better understanding
and prevention of lung diseases



Thank You !

Grace and Peace be with You

