ENVE 576
Indoor Air Pollution

Fall 2017

October 24, 2017

Epidemiology and adverse health effects

Built
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Advancing energy, environmental, and sustainability research within the built environment

Dr. Brent Stephens, Ph.D.
Department of Civil, Architectural and Environmental Engineering
Illinois Institute of Technology
brent@iit.edu

Built Environment Research Group
www.built-envi.com

Scheduling

- HW #3 and #4 graded and returned today
- Blog post #2 due today
- Take home exam will be assigned next week
  - Due 1 week later

MEASURING ORGANIC GASES (VOC AND SVOC)

Sample Collection Methods

- Two methods:

1. Real-time measurement/analysis
   - Generally has a sensor (mostly FID, PID)
   - Some have separation (w/ GC) + sensor
   - Also: colorimetric tubes (general: MDL > 1 ppm)

2. Collect air sample for laboratory analysis
   - Whole-volume samplers (canisters, bags)
   - Concentration samplers (sorbents, SPME)
     - Either case: preservation and analysis in laboratory

Canister samples

- Whole volume
- Grab versus integrated
- EPA Methods TO-14/15
- Benefits:
  - Inert/impermeable
  - Lots of experience
  - Multiple analyses can be done
- Drawbacks
  - Bulky
  - Requires cleaning
  - Can get scratched
  - Sample stability (reactions)

1 – 15 L

400 mL

Tedlar bags

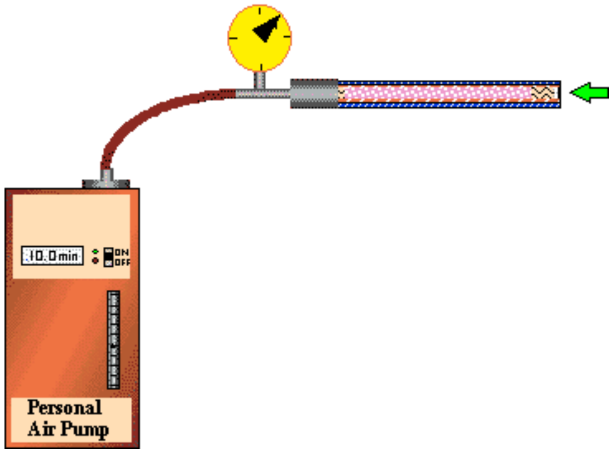
- Whole volume
- Tedlar = polyvinylfluoride
- Pump to collect (unlike canisters)
- Benefits:
  - Inert / impervious (like cans)
  - Repeat samples (like cans)
  - Lighter than cans
  - Lower initial cost than cans
- Disadvantages:
  - Not as reusable as cans
  - Susceptible to tearing
  - Requires cleaning
  - Stability with some compounds

0.5 – 100 L

Sorbent sampling

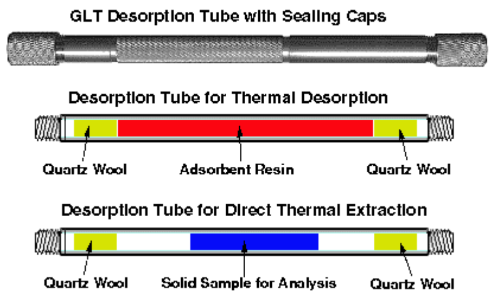
- VOC adsorbs to solid adsorbent
- Passive sampling
  - Similar to ozone badge but w/out reaction
  - Integrated sample over 24 hours, etc.
  - Indoor, personal, outdoor

- Active Sampling
  - Pump air through a packed tube
  - Collect mass over known volume
  - C = m/V
  - Short-term vs. integrated
  - More control, but more difficult



Sorbent tubes

- EPA Method TO-17 = TD/GC/MS (important)
- Various sorbents can be used
  - TO-17 page 33
  - Need to match VOC types/ranges with sorbent
- Some issues
  - Method detection limit, precision, accuracy (pg. 28/29)
  - Sample preservation
  - Breakthrough volume
  - Artifact formation (especially via ozone)
  - Sorbent pre-conditioning / breakdown over time
- Use of multi-sorbent beds
- Focus on Tenax-TA

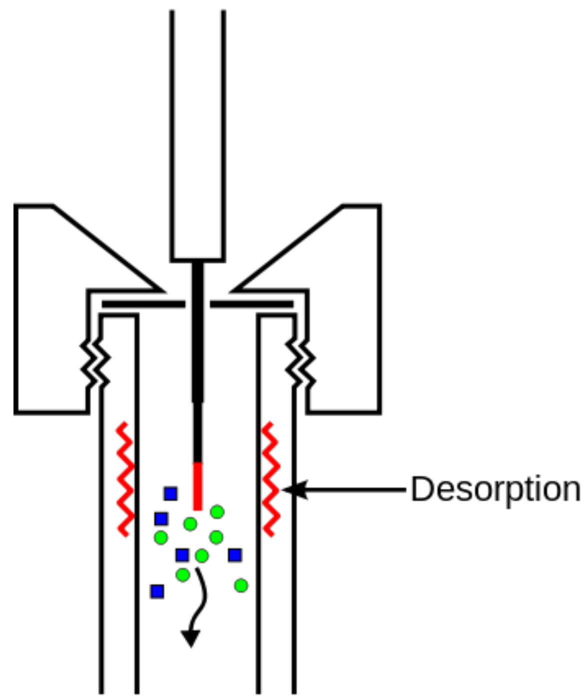


Sorbent: Tenax-TA

- 2,6-diphenylene oxide polymer resin (porous)
- Specific area = 35 m^2/g
- Pore size = 200 nm (average)
- Density = 0.25 g/cm^3
- Various mesh sizes (e.g., 60/80)
- Low affinity for water (good for high RH)
- Non-polar VOCs (T_b > 100 °C); polar (T_b > 150 °C)
  - lighter polar – Carbotrap and Carbopack-B common
- Artifacts w/ O_3: benzaldehyde, phenol, acetophenone

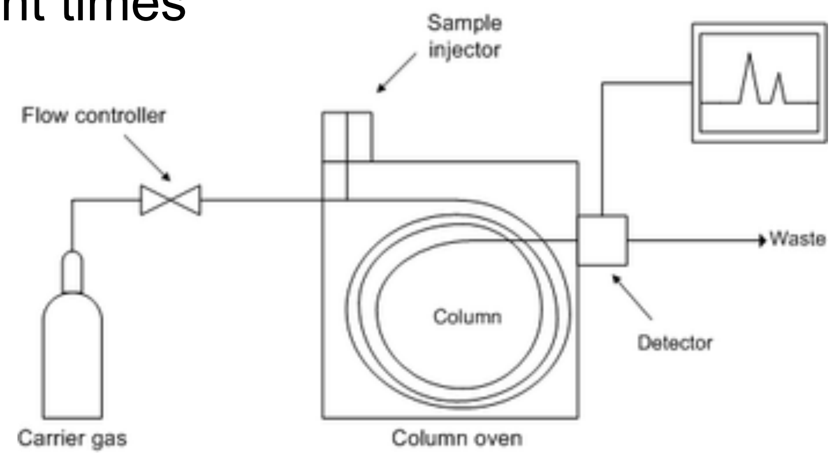
Solid-Phase Micro-Extraction (SPME)

- Uses a fiber coated with an extracting phase:
  - PDMS / DVB / Carboxen
- Benefits
  - Highly concentrating for many indoor VOCs (ppt levels)
    - Can get VVOCs
  - Reusable
  - Relatively low cost
  - Small / light weight
  - Possible use in other media
  - Ease of injection to GC
- Drawbacks
  - Less experience / acceptability
  - Preservation issues
  - Difficulties w/ calibration

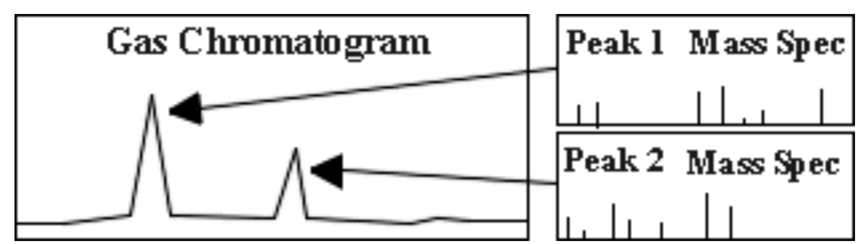


Gas Chromatography (GC)

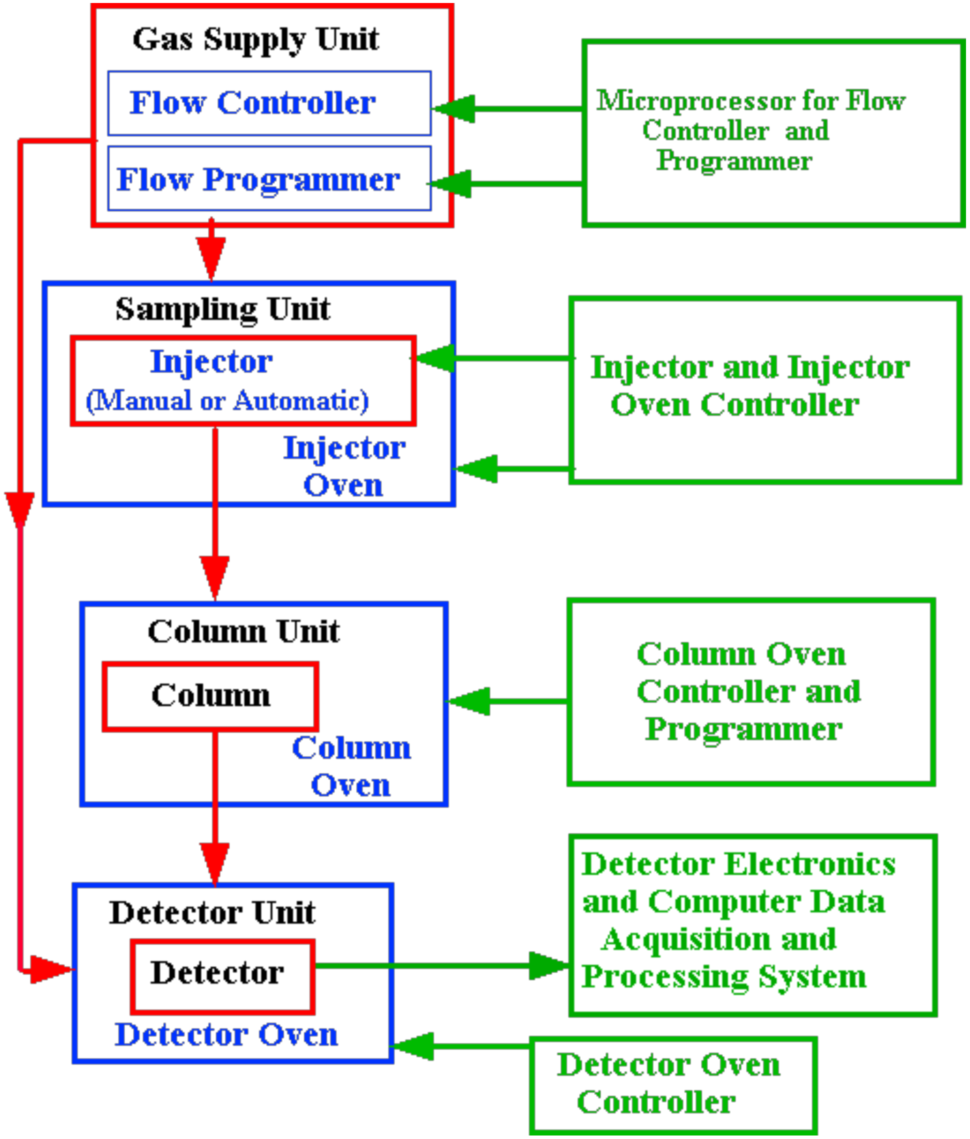
- GC is used to separate compounds
  - Compounds are vaporized into an inert carrier gas through a capillary column
- Capillary column
  - Stationary microscopic layer of liquid or polymer on inert solid support inside a piece of glass or metal tubing
  - Causes compound to elute at different times
    - Retention time

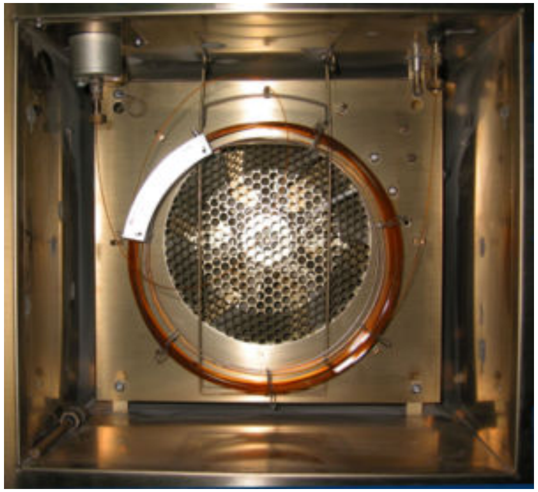


- Thermal program of GC oven
- Temporal passage to a detector
  - Analyze "peaks"
  - Analyze molecular fragments (MS)



Gas Chromatography (GC)





GC issues

- Type of injection?
- Need to cryo focus for low molecular weight volatiles?
- Type of column?
- Type of detector?
  - If MS, model of detection
- Temperature programs
- Instrument calibration / response

Detectors

- Flame ionization detector (FID)
- Photoionization detector (PID)
- Electron capture detector (ECD)

} Non-specific or speciated (w/ GC)

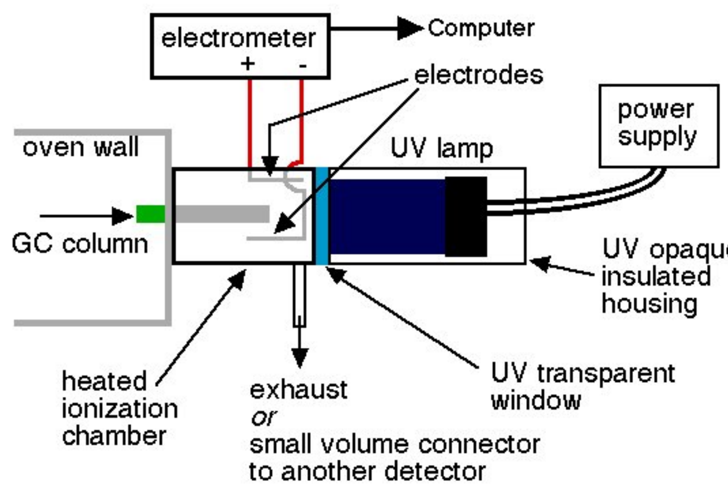
- Mass spectrometer (MS)

} w/ speciated (w/ GC)

- These are primary detectors for VOCs in indoor air
- Specific uses vary considerably

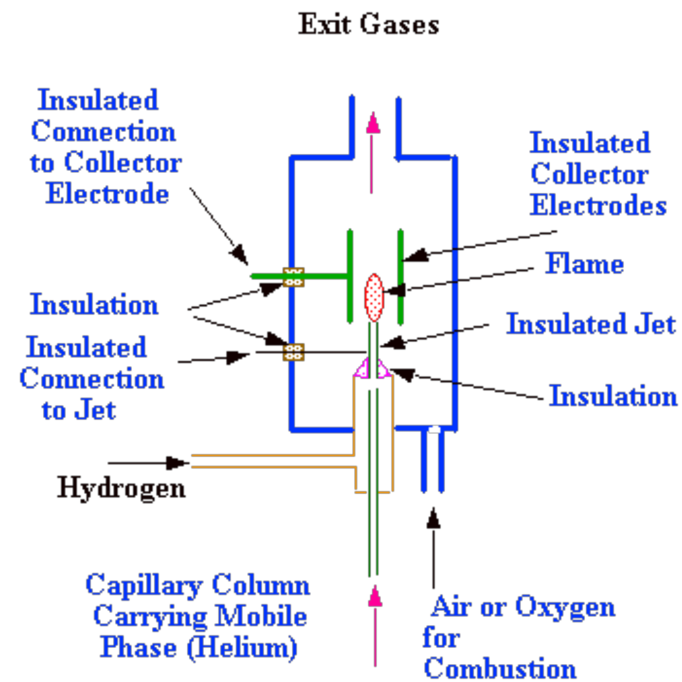
Photoionization Detectors (PID)

- UV light ionizes VOCs --- $R + hv \rightarrow R^{+} + e^{-}$
- Collected by electrodes = current
- VOCs with different ionization potential
- Benefits
  - Simple to use
  - Sample non-destructive (relatively)
- Drawbacks
  - No identification/speciation
  - Highly variable responses
  - Not all VOCs detected
  - Lamp burnout / contamination



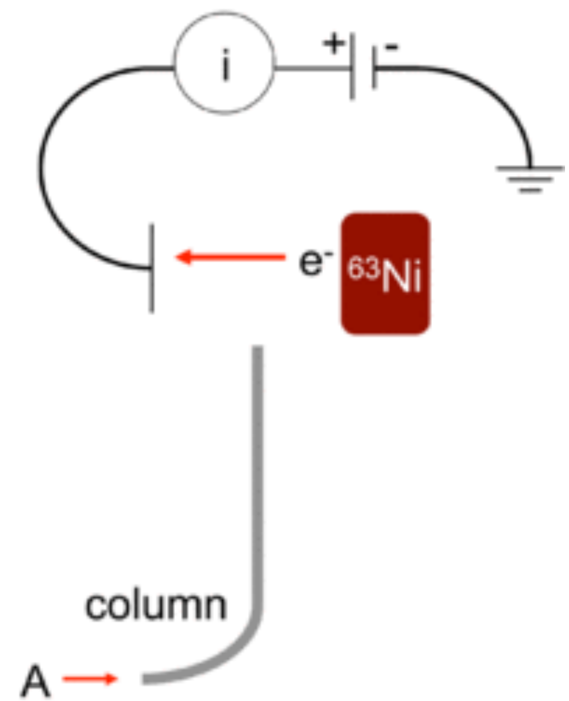
Flame Ionization Detectors (FID)

- Relatively simple system
- Hydrogen flame → ions formed
  - Ions migrate to plate, generate a current
  - Hydrocarbons have molar response proportional to the number of carbon atoms in their molecule
- Detection – typical to pg/s
- Benefits
  - Rugged, low cost, workhorse
  - Linear response over wide range
  - Insensitive to H_2O, CO_2, SO_2, CO, NO_x
- Drawbacks
  - No identification
  - Lower response if not simple HC
  - Destructive testing



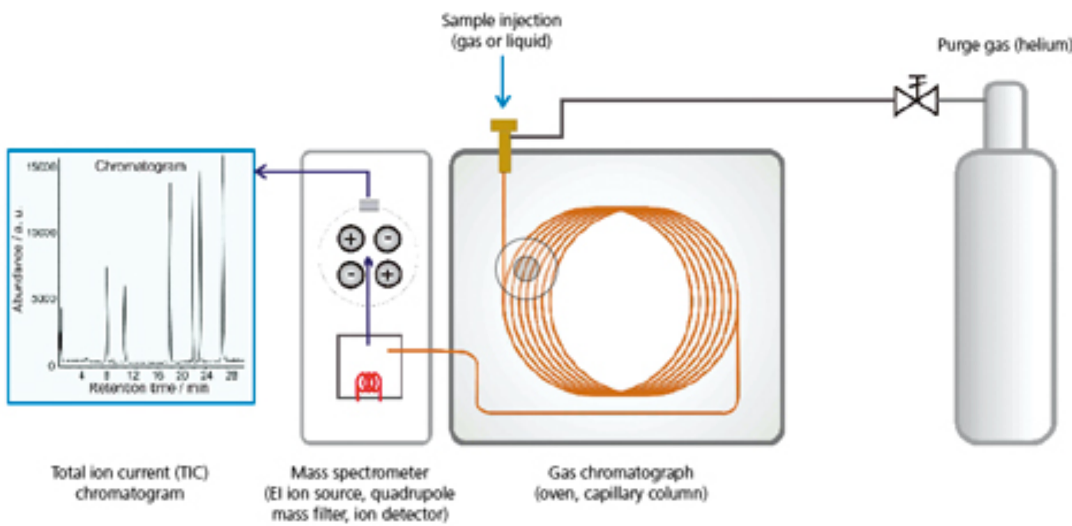
Electron Capture Detectors (ECD)

- Low energy Beta emitter = ^{63}Ni in make-up gas (Nitrogen)
- *e*- attracted to positively charged electrode (anode)
- Molecules in sample absorb e- and reduce current
  - effective: halogens (e.g., SF_6), nitrogen-containing compounds
- Benefits
  - 10-1000 times more sensitive than FID
  - femtogram/s ----- ppt levels
- Drawbacks
  - More limited linear range than FID
  - Radiological safety requirements
  - O_2 contamination issues
  - Response strong function of T, P, flow rate



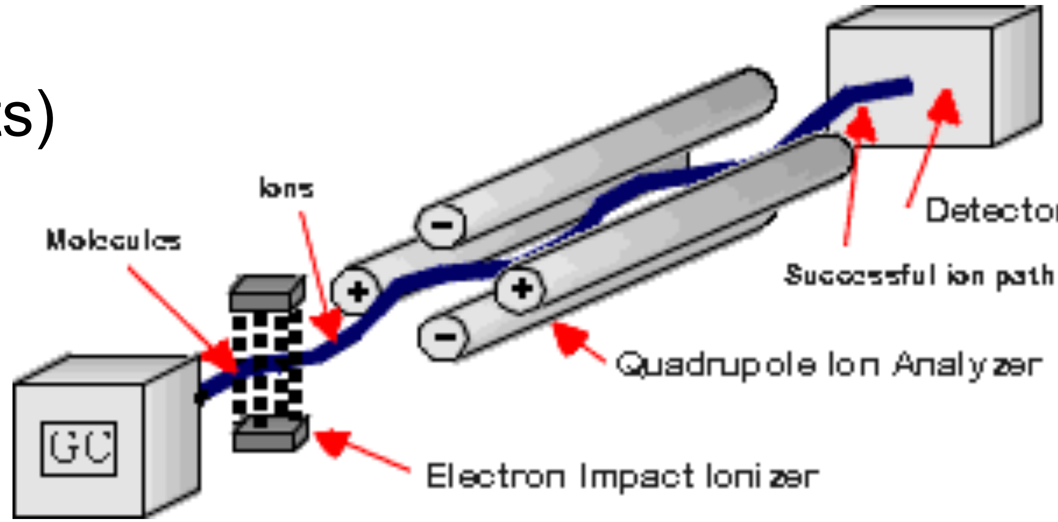
Mass Spectrometer (MS)

- Bombard molecules w/ intense electron source (ionization)
  - Generates positive ion fragments
- Ions accelerate to have same kinetic energy, then deflect in a magnetic field, where deflection is a function of molecular weight
- Use fragment fingerprint to identify molecule
- Quantify amount of fragments to determine mass
- Most common MS = quadrupole
- Benefits
  - "Gold standard"
  - Amount AND identific
- Drawbacks
  - Cost
  - Complexity
  - Maintenance



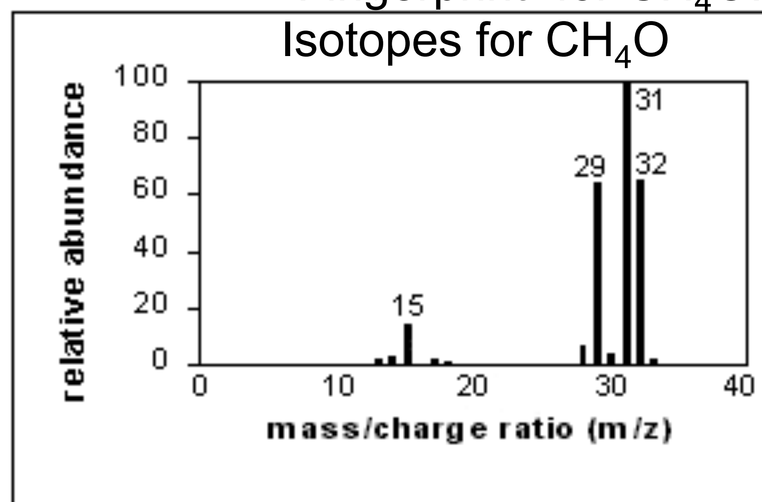
Quadrupole MS

- Electron source
- Four rods (electromagnets)
  - Applied Voltage
  - DC/AC components
  - Voltages = fn(time)
  - Affects trajectory
  - Selective M/Z to detector
  - m/z = mass-to-charge ratio
    - Ionization makes z = 1
- Cycles different M/Z
- Yields mass spectrum
- Always same for a molecule
- System in vacuum



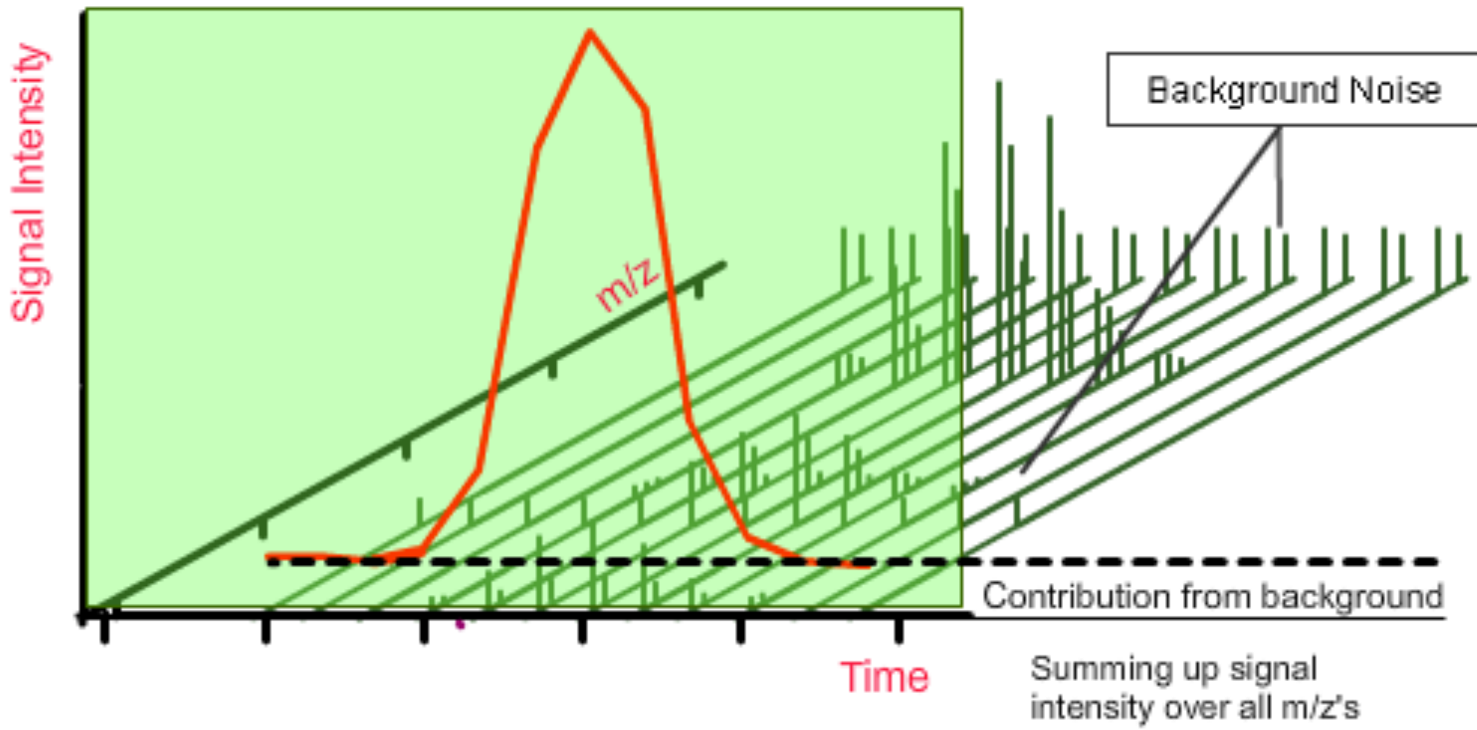
"Fingerprint" for CH_4O:

ions	m/z
$CH_3OH^{+\cdot}$	32
$H_2C{=}OH^+$	31
$HC{\equiv}O^+$	29
H_3C^+	15

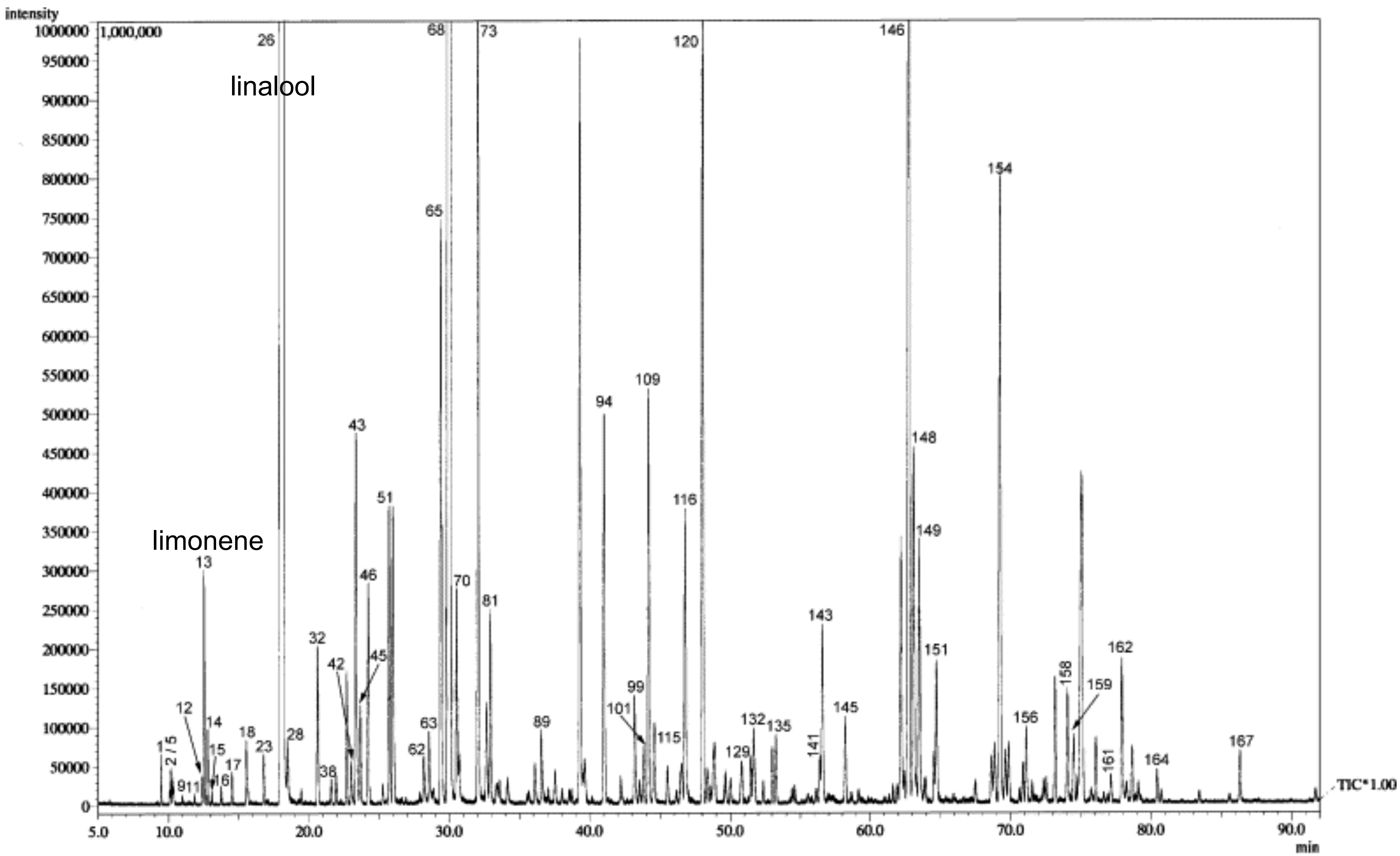


Total Ion Chromatogram (TIC)

Sum up intensities of all mass spectral peaks belonging to the same scan



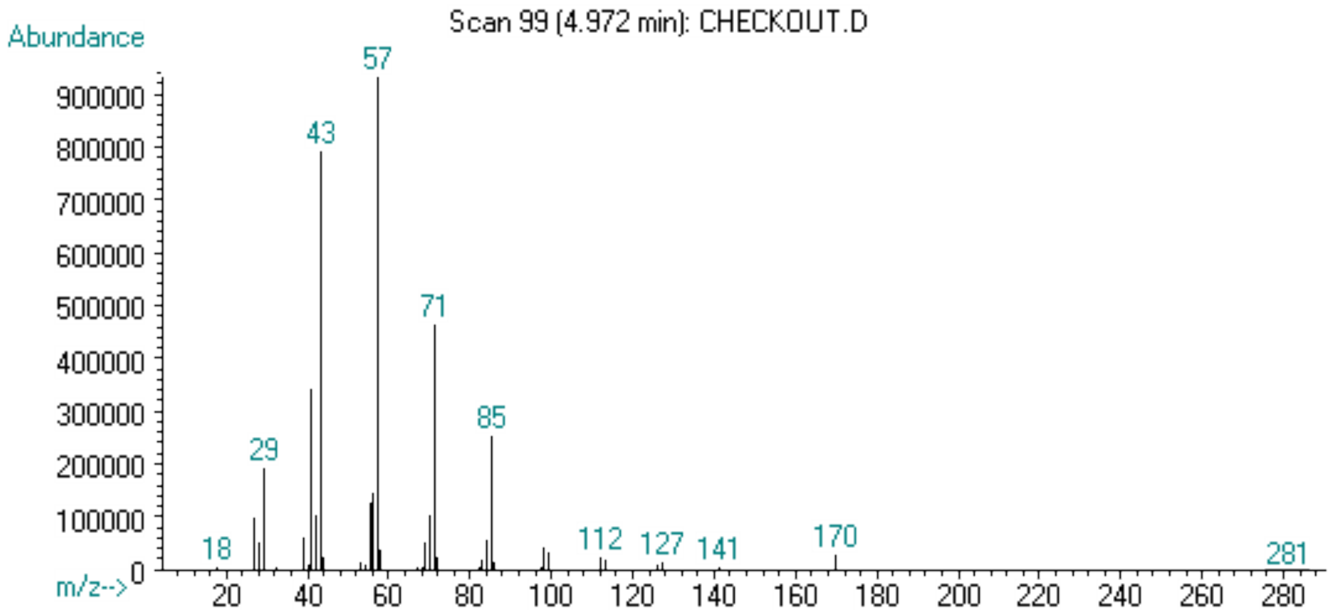
Total Ion Chromatogram (TIC)

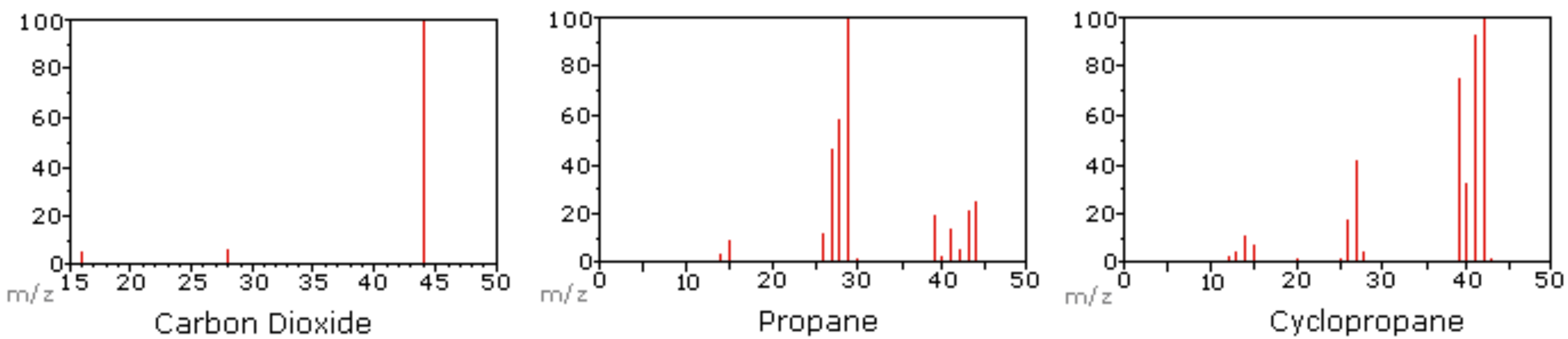


Mondello *et al., J. of Chromatography A*, 1067: 235-243 (2005)

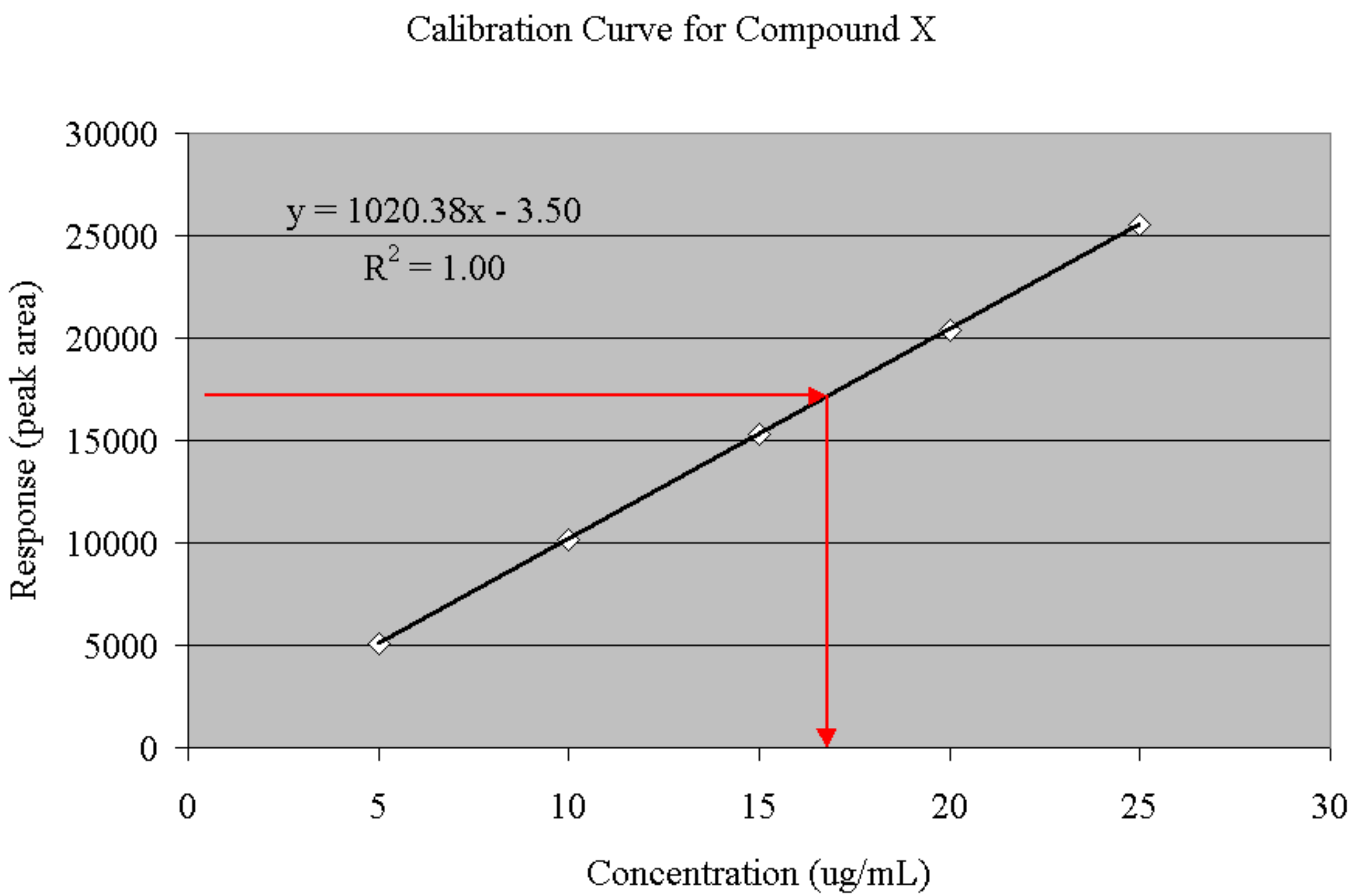
Mass spectrum

Example mass spectrum (fingerprint)



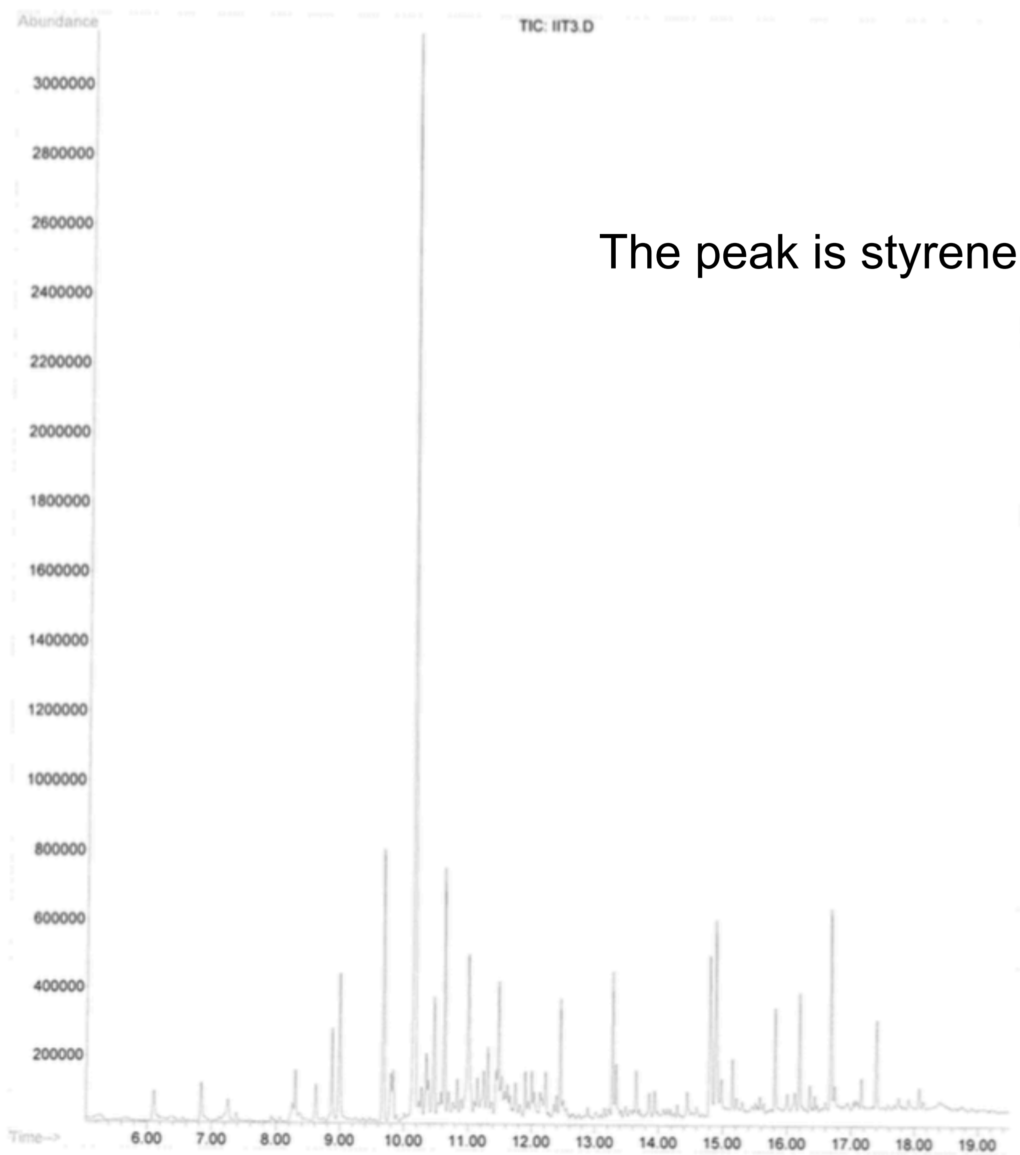


Calibration curves



$$Response\ Factor = \frac{Peak\ Area}{Calibration\ Concentration}$$

Real VOC data w/ library compound search



The peak is styrene

Summary of VOC measurements

- VOCs important in indoor environments
- Many types of VOCs
  - Different properties
  - Different effects
  - Different sample collection and analysis protocols
- Sampling and analysis protocols NOT TRIVIAL
  - Many types of collection methods
  - Many types of analysis detectors and methods
  - A lot of issues involved w/ sample/analysis decisions
  - A lot can go wrong (difficult business)
  - Cumbersome and costly, but very important

ADVERSE HEALTH EFFECTS OF AIR POLLUTION

Adverse health effects of air pollution

- How do we know if something is harmful to humans?
  - Or animals? Or plants?

Primary methods of assessing health effects

- Toxicology studies
  - Cellular level
  - Theoretical underpinnings/underlying biological mechanisms

- Entire organisms: humans or animal *models* (e.g., mice):
  - Clinical (dose-response)
    - Fundamental relationship between exposure/dose and effect
    - Causative mechanisms
  - Epidemiology (exposure-response)
    - Simply a relationship between exposure/dose in a population
    - Correlation not causation
      - But if informed by fundamental biological plausibility, it can help confirm

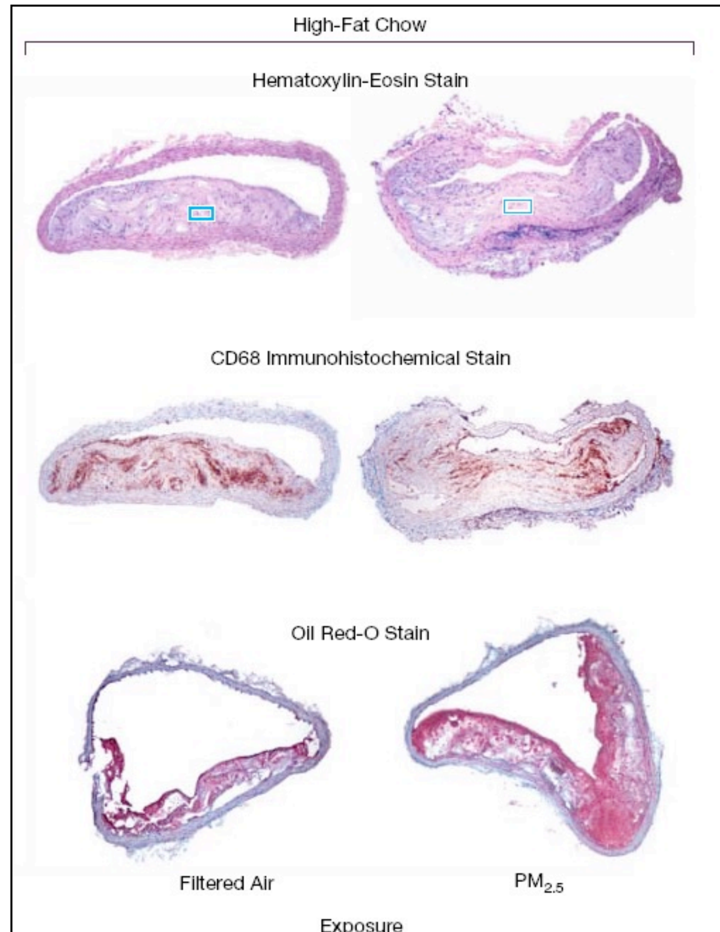
How do air pollutants cause health effects?

- PM or ozone induce airway inflammation
- Oxidative stress is induced by transition metals or PAHs
- Modifications of intracellular proteins/enzymes
  - Stimulating cells to generate reactive oxygen species (ROS)
- Biological compounds (glucans, endotoxins) affect immune response and inflammation
- Stimulation of autonomic nervous system
- Adjuvant (stimulate immune response) effects
- Pro-coagulant activity (UFPs)
- Suppression of normal defense mechanisms

Example: Particulate matter

- Toxicological, clinical, and epidemiological studies have all increased understanding of the **mechanism of action** by which PM leads to adverse health effects such as mortality and lung and heart disease

- Image to the right shows abdominal arteries from mice exposed to filtered air and to fine particulate matter ($PM_{2.5}$)
  - $PM_{2.5}$ increased arterial blockage



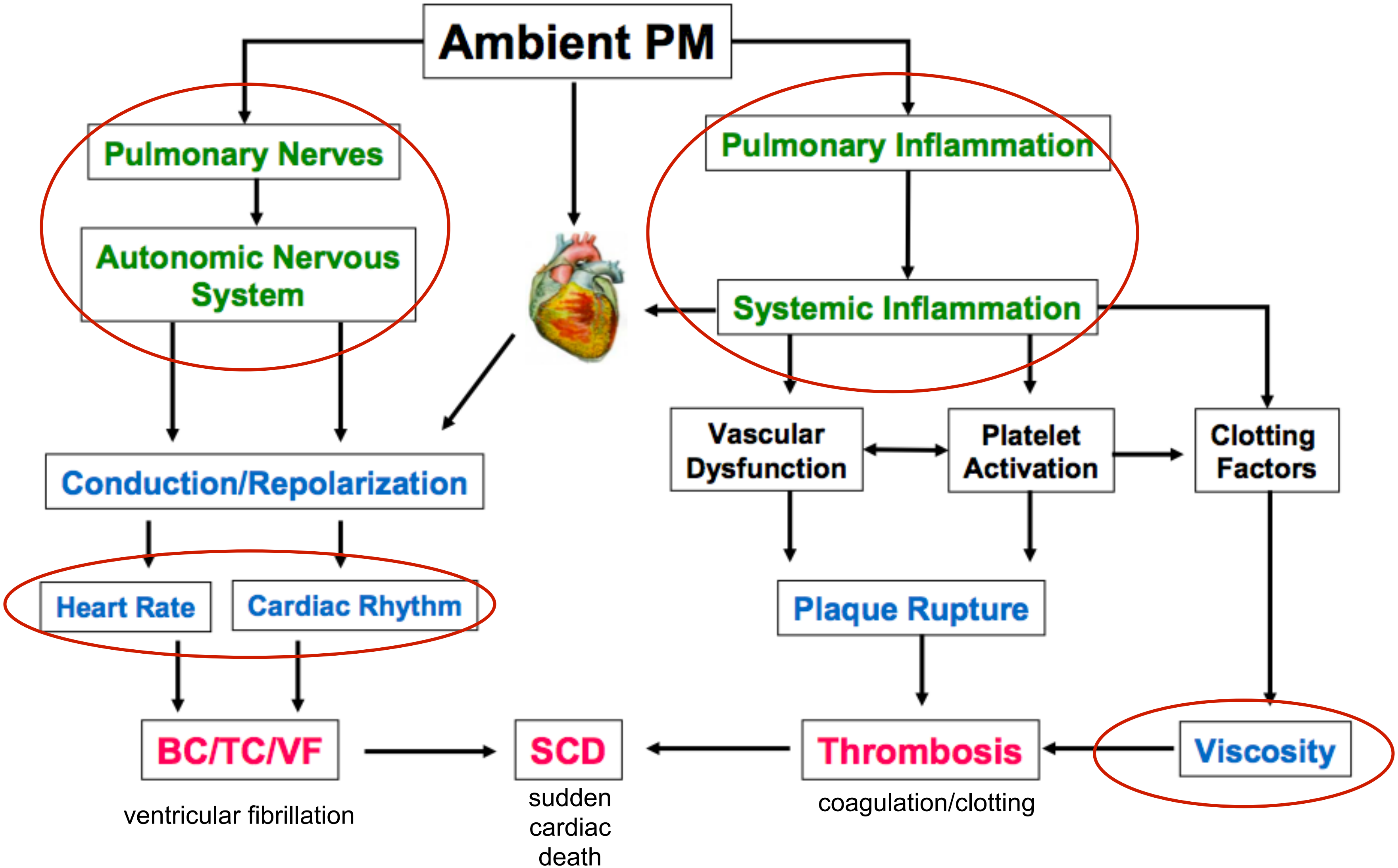
Sun et al. *JAMA,* 2005; Kunzli et al. 2005

How does PM cause health effects?

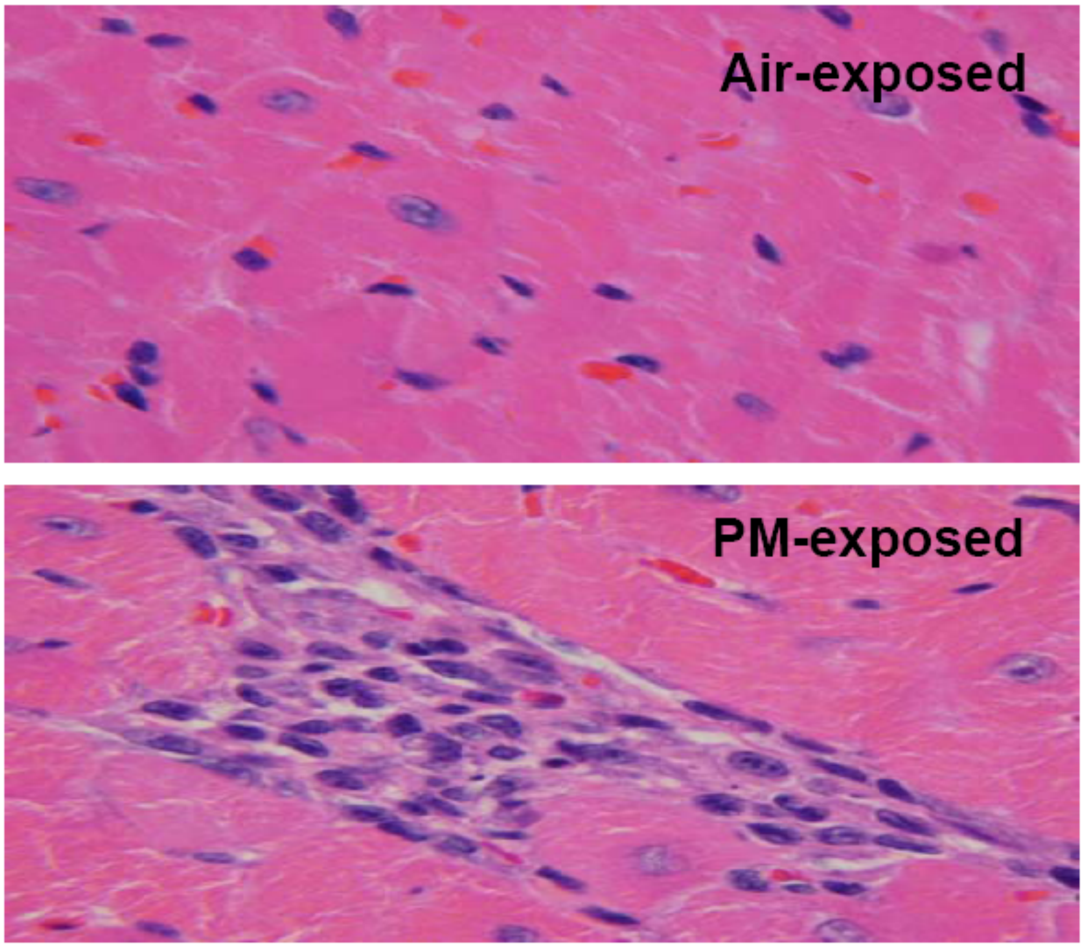
- Several theories exist here… likely more than one mechanism

1. PM leads to lung **irritation** which leads to increased permeability in lung tissue;
2. PM increases **susceptibility to viral and bacterial pathogens** leading to pneumonia in vulnerable persons who are unable to clear these infections;
3. PM **aggravates the severity of chronic lung diseases** causing rapid loss of airway function;
4. PM causes **inflammation** of lung tissue, resulting in the release of chemicals that impact heart function;
5. PM causes **changes in blood chemistry** that results in clots that can cause heart attacks.

How could PM affect the cardiovascular system?



PM causes injury to cardiac cells

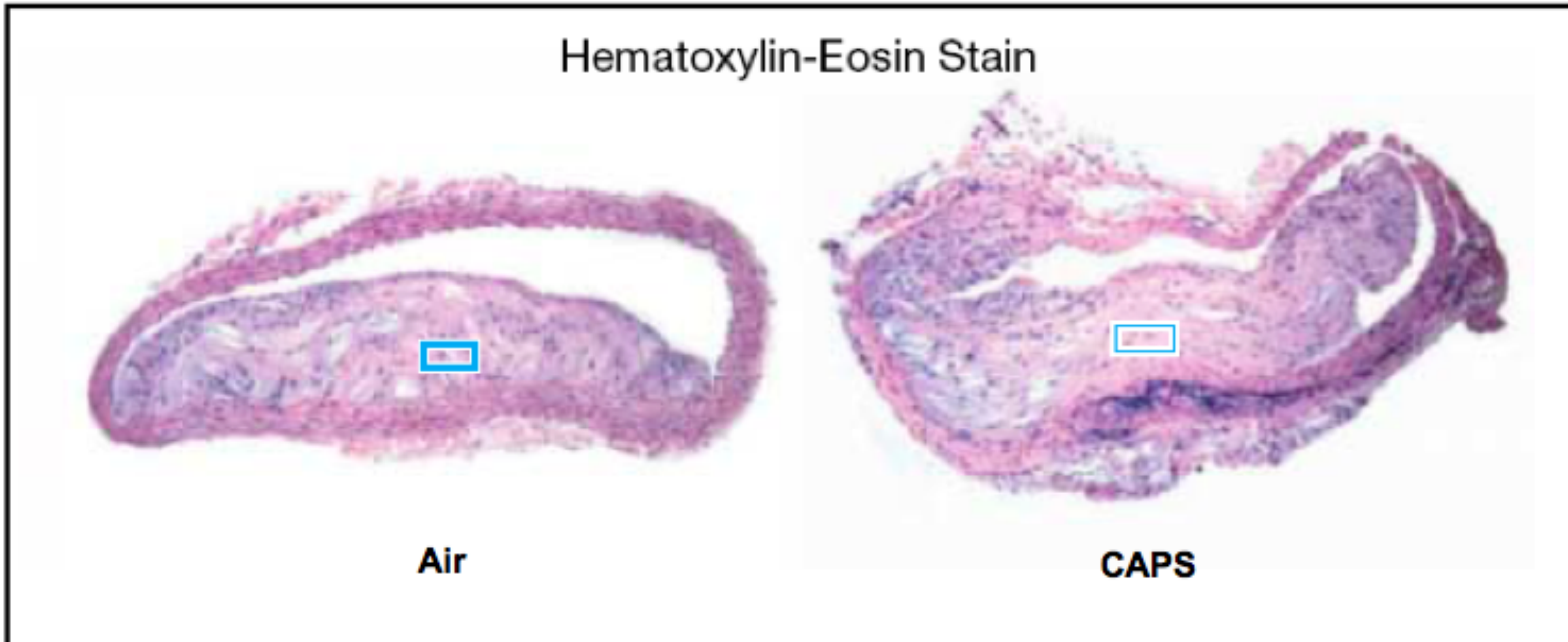


Rats exposed to ambient PM one day per week for 16 weeks

Kodavanti et al., 2003

PM hardens arteries

Plaque area

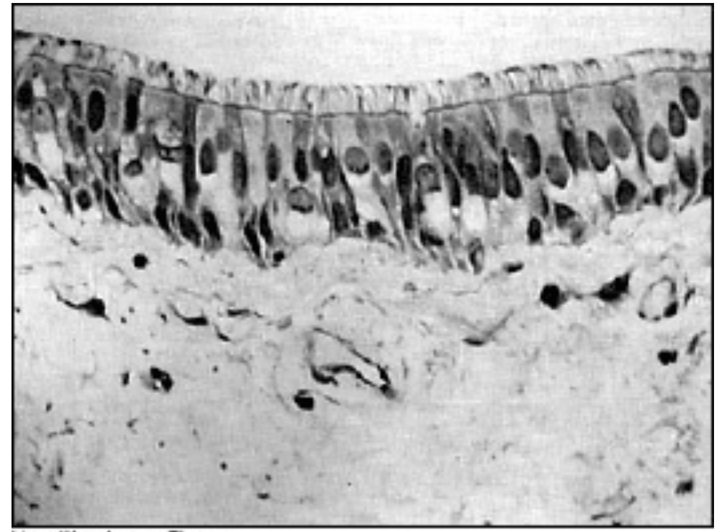


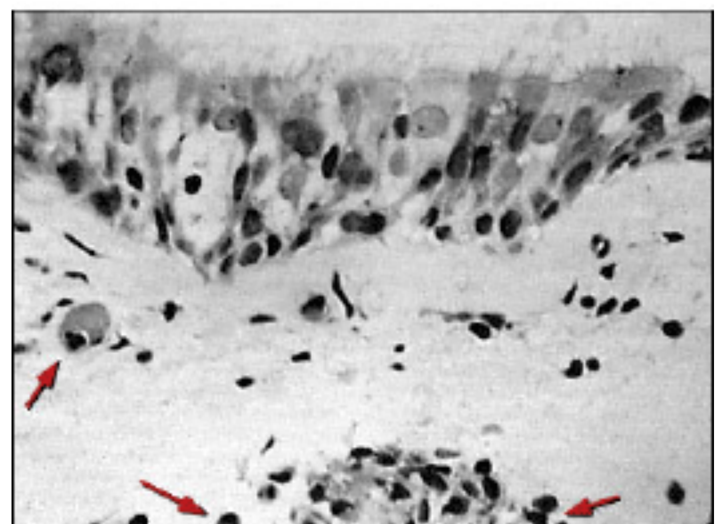
ApoE mice exposed for 6 hrs/day, 5 days/wk x 6 months to CAPS (85 μg/m^3 average)
Mean levels only 15.2 μg/m^3

Sun et al. 2005

Ozone damages lung tissue

- Tiny cilia that clear the lungs from mucus appear along the top of the image to the right (healthy lung tissue)
- In the lung exposed to only 20 ppb of ozone (to the right) for 4 hours of moderate exercise, many cilia appear missing and others are misshapen
  - Arrows point to tiny bodies called neutrophils which indicate inflammation


Healthy Lung Tissue


Ozone-damaged Lung Tissue

Aris et al., **1993** *Am Rev Respir Diseases*

HUMAN EPIDEMIOLOGY STUDIES

Do these cell-level impacts show up in large human studies?

Human epidemiology studies

- How would you conduct an epidemiology study?

Human epidemiology studies

- Examine two populations with different exposures
  - e.g., babies home to renovated nurseries or not
  - e.g, children in homes w/ vinyl floors or not
  - e.g., spouses of smokers and non-smokers

- Collect data on health outcomes
  - Asthma, cancer, lung function, mortality, etc.

- Form 2x2 ‘epi matrix’ for select populations

	With effect	**Without effect**
Exposed	*exposed with effect*	*exposed without effect*
Not exposed	*not exposed with effect*	*not exposed without effect*

Human epidemiology studies

- Relative risk = RR

$$RR = \frac{(\text{exposed with effect})/(\text{total exposed})}{(\text{not exposed with effect})/(\text{total not exposed})}$$

  – RR > 1.0 = association
  – RR >> 1.0 = strong association
    (also if confidence interval doesn't cross 1)

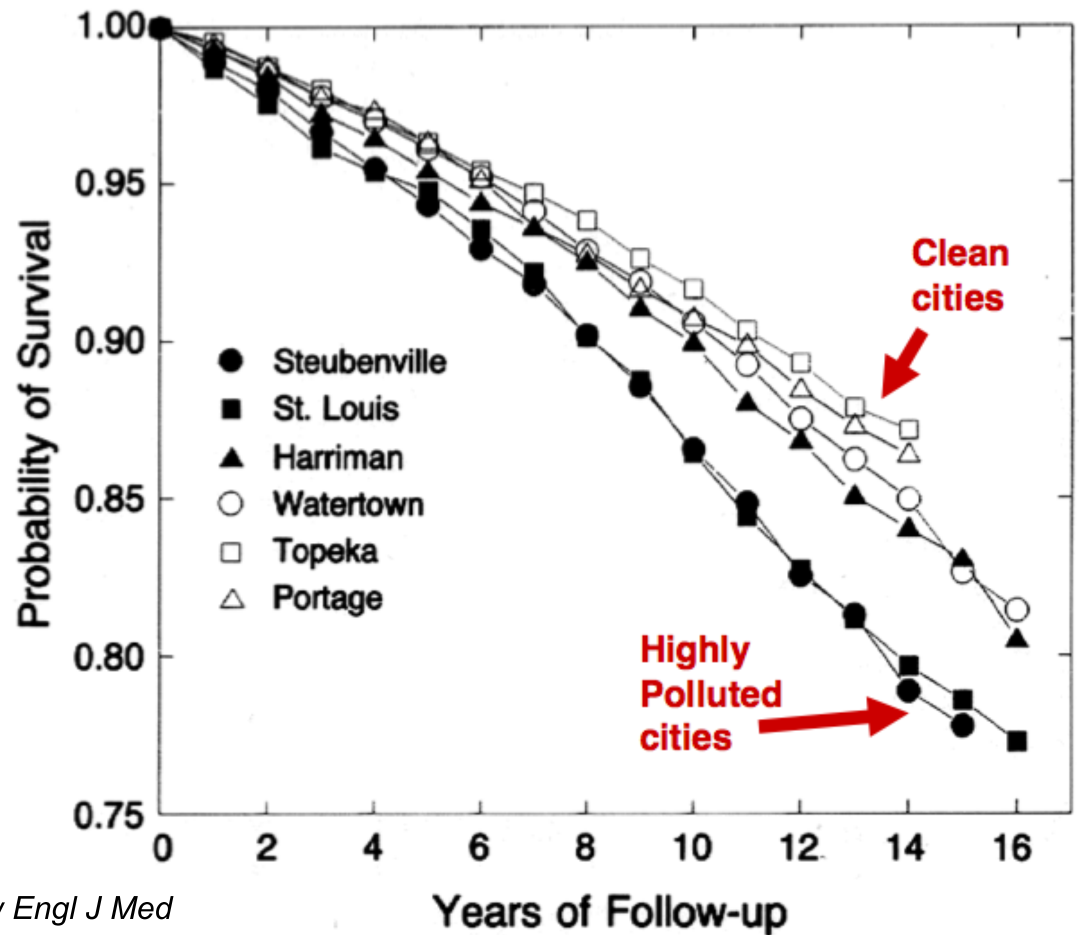
- Odds ratio = OR (often ~RR)

$$OR = \frac{(\text{exposed with effect}) * (\text{not exposed without effect})}{(\text{not exposed with effect}) * (\text{exposed without effect})}$$

  – OR > 1.0 = association
  – OR >> 1.0 = strong association

Health effects of outdoor PM: Epidemiology

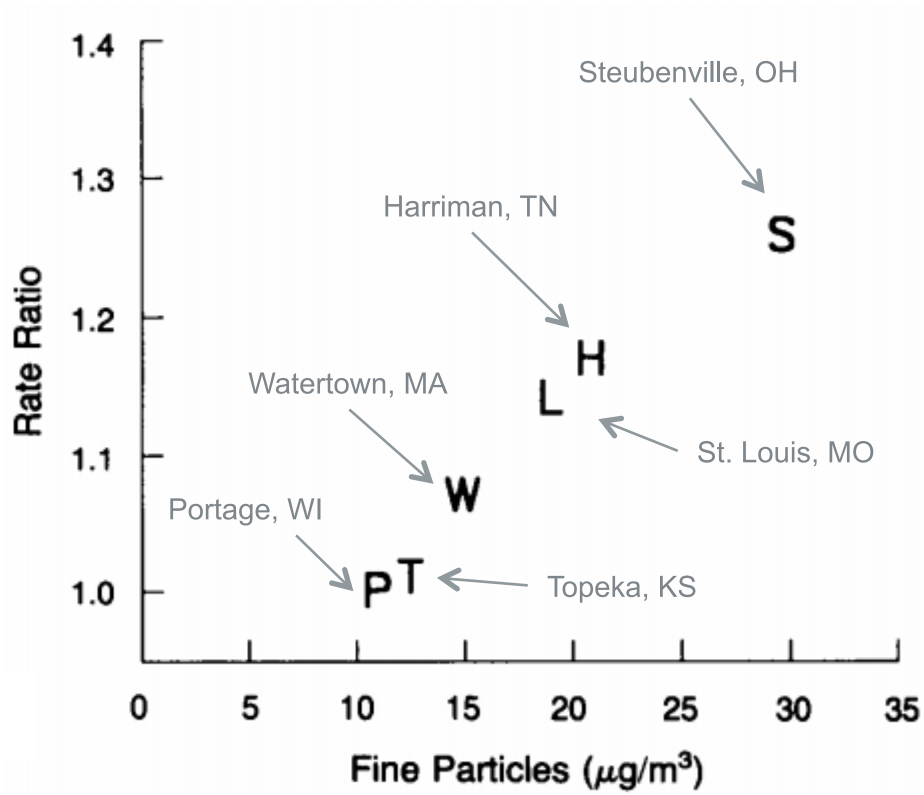
- Early high impact study: The Harvard Six Cities Study
  - Long-term air pollution linked to shortened life expectancy
  - 15 year prospective study of 8000+ adults in six US cities



Dockery et al., **1993** *New Engl J Med*

Health effects of outdoor PM: Epidemiology

- Harvard Six Cities Study
  - Relative risk of dying almost linearly correlated with outdoor $PM_{2.5}$

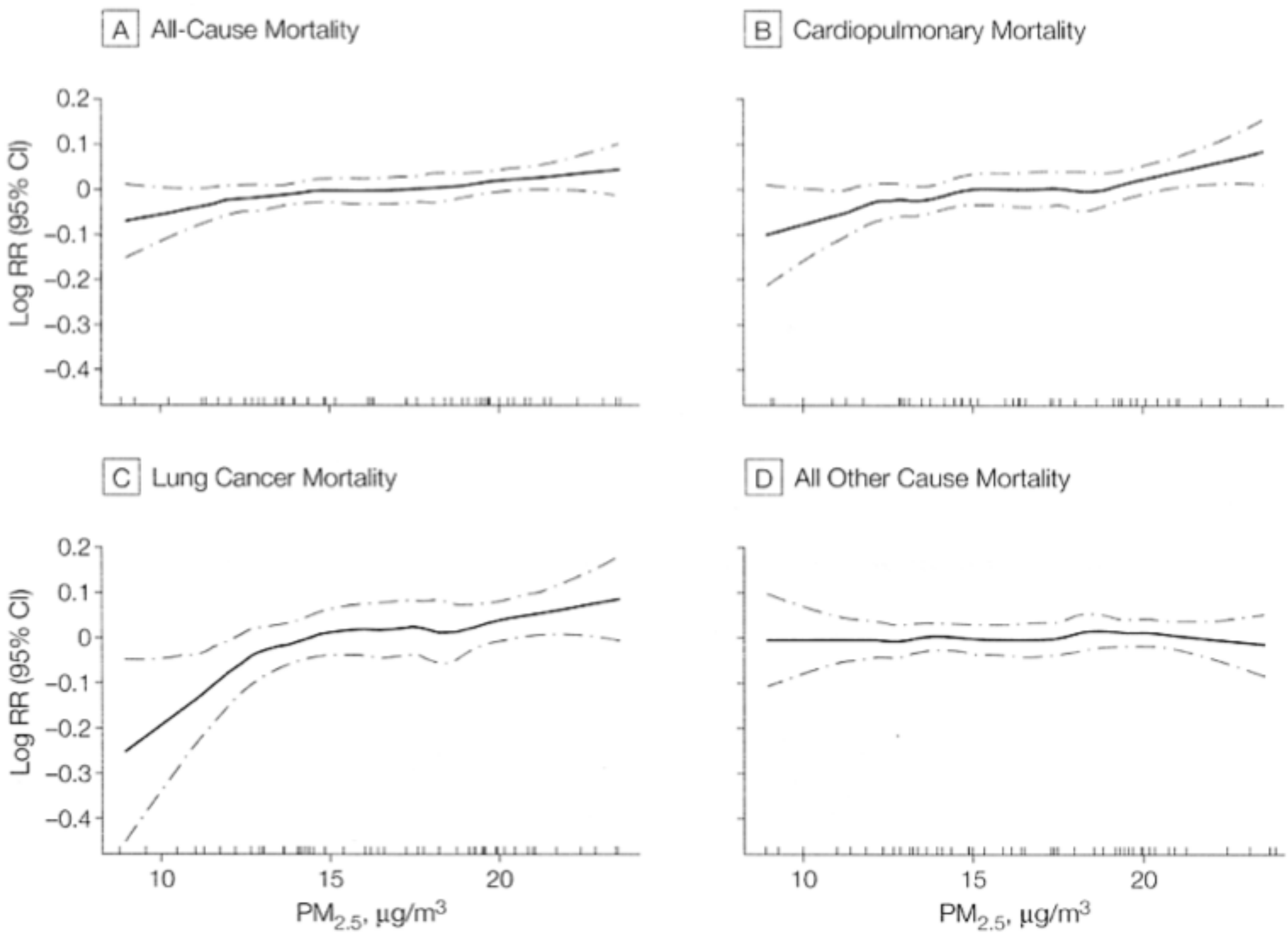


Mean $PM_{2.5}$ concentration measured outdoors in six cities over several years in the 1980s

Dockery et al., **1993** *New Engl J Med*

Health effects of outdoor PM: Epidemiology

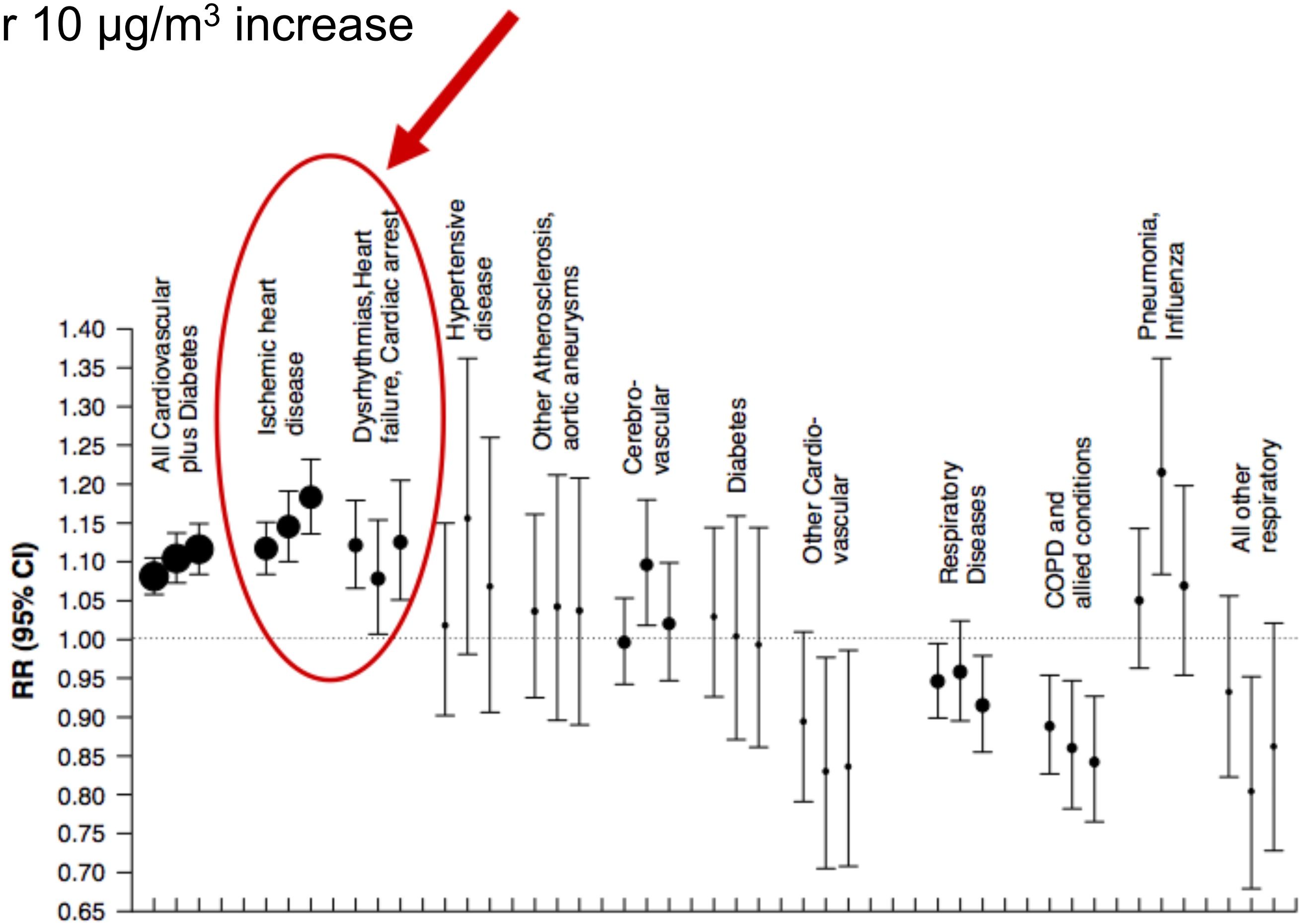
- ACS cohort: over 1 million people
  - Increased $PM_{2.5}$ → increased risk of death



Pope et al., **2002** *J Am Med Assoc*

Health effects of outdoor PM: Epidemiology

- Follow-up of ACS cohort: over 1 million people
  - Increased $PM_{2.5}$ most strongly associated with death from heart disease, dysrhythmias (irregular heartbeat), heart failure, and cardiac arrest
  - Per 10 μg/m^3 increase



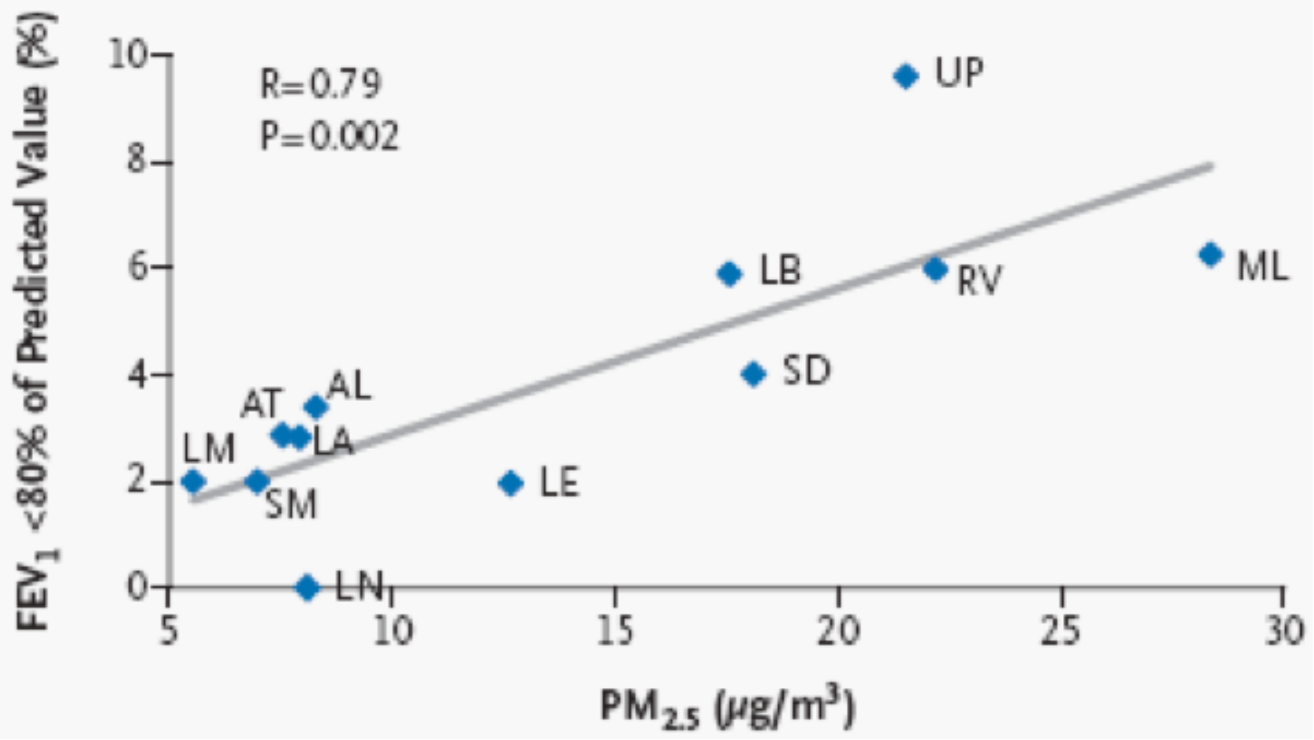
Pope et al., **2004** *Circulation*

Outdoor PM and lung growth

- Children living in cities with higher air pollution showed greater deficits in lung function growth

FEV_1 = forced expiratory volume in 1 second

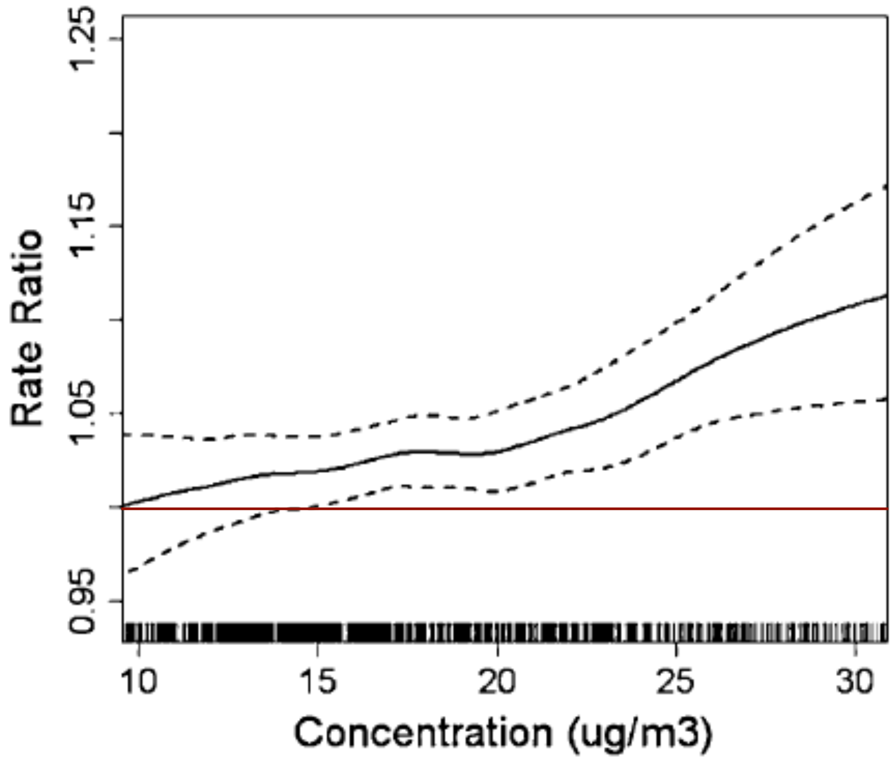
- Volume of air you can exhale in 1 sec



Gauderman et al., **2007** *The Lancet*

Outdoor PM and asthma

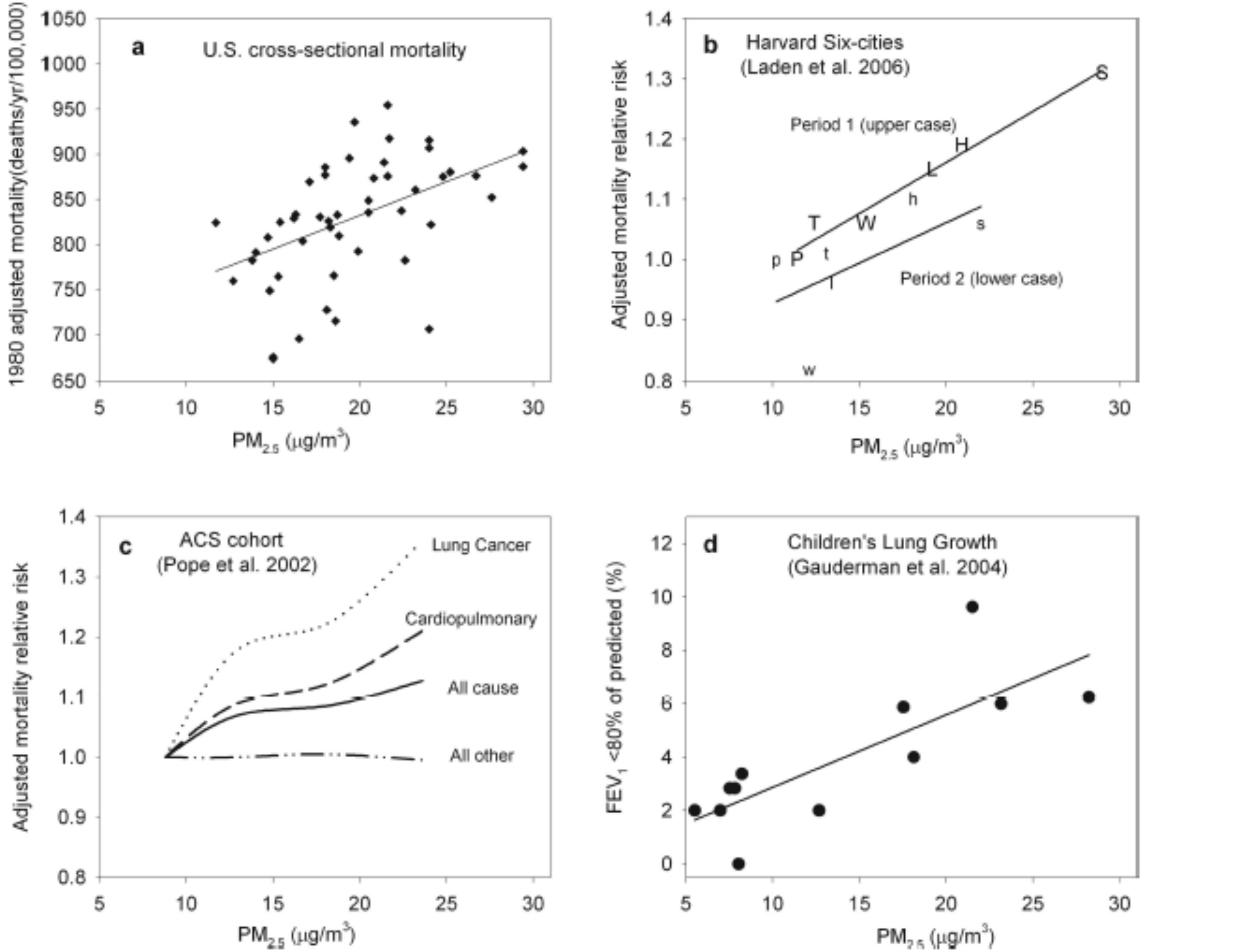
Ambient $PM_{2.5}$ and ER visits for pediatric asthma



3-day average $PM_{2.5}$data measured outdoors in Atlanta, GA from 1993 to 2004

Strickland et al., **2010** *Am J Respir Crit Care Med*

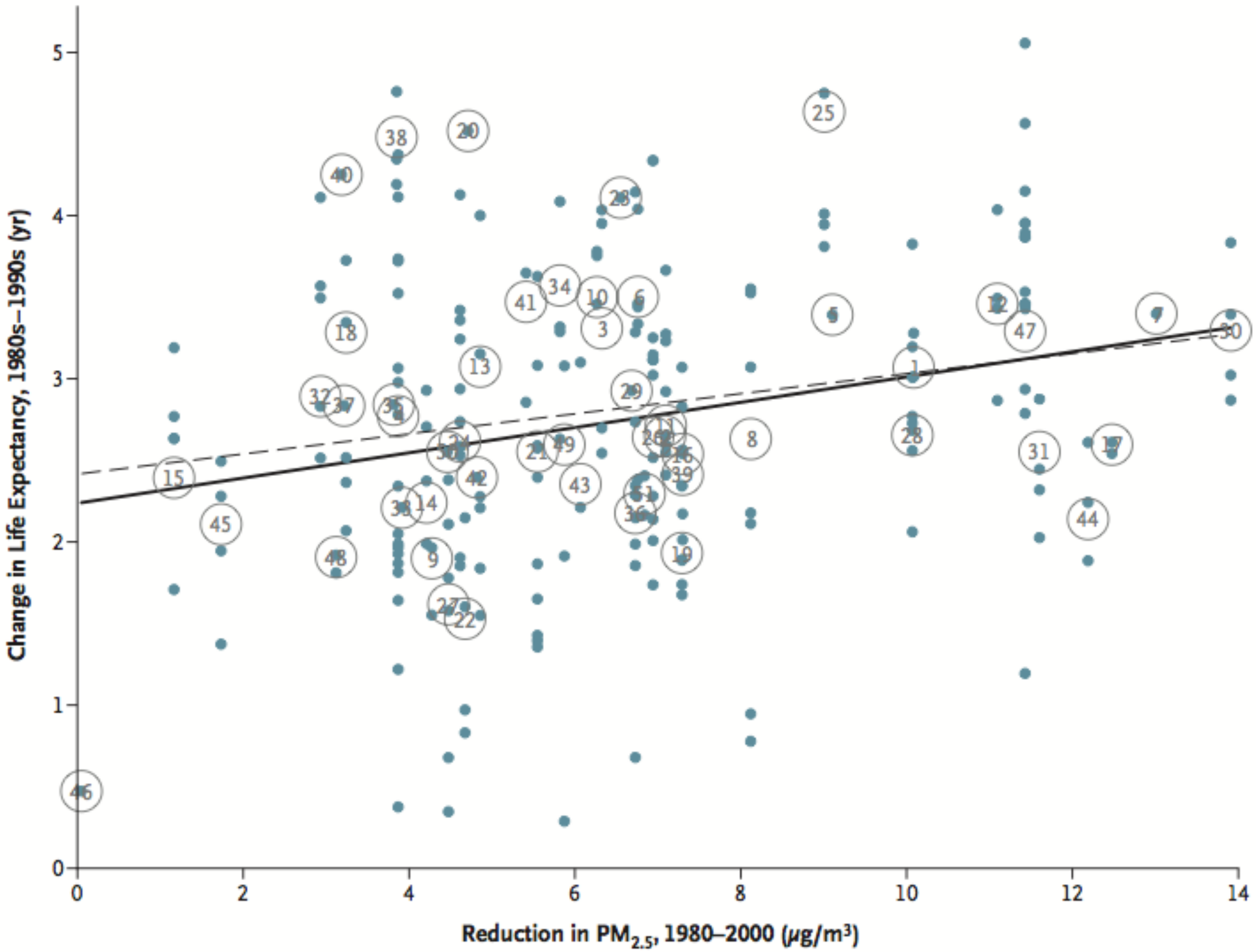
More $PM_{2.5}$ risk relationships



Pope and Dockery, **2006** *J Air Waste Manage Assoc*

What happens when you reduce PM?

Reduce outdoor $PM_{2.5}$ by 10 μg/m^3 → increase life expectancy by 0.61 years

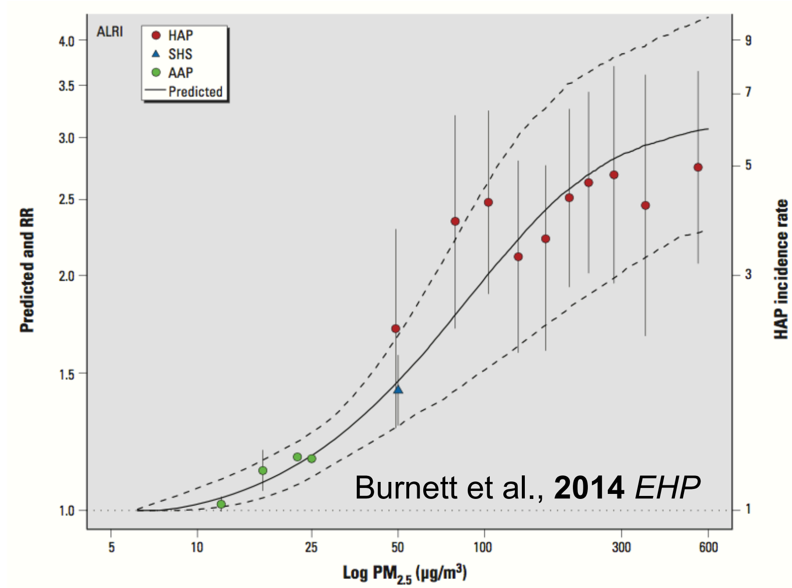


Pope et al., **2009** *NEJM*

Increased **mortality** risks outdoor $PM_{2.5}$

All-cause mortality

- 4 ± 3% increase per 10 μg/m^3 in $PM_{2.5}$
  Pope et al., **2002** *J Am Med Assoc*
- 6 ± 2% increase per 10 μg/m^3 in $PM_{2.5}$
  Krewski et al., **2009** HEI Research Report
- 16 ± 9% increase per 10 μg/m^3 in $PM_{2.5}$
  Laden et al., **2006** *Am J Respir Crit Care Med*
- What is the shape of the concentration-response curve?



Burnett et al., **2014** *EHP*

$PM_{2.5}$ compositions

- All $PM_{2.5}$ constituents are not equally toxic

- Sulfate ion, iron, nickel, and zinc in $PM_{2.5}$
  - Mortality

    Burnett et al., **2000** *Inhalation Toxicology*

- Vanadium, elemental carbon, and nickel in $PM_{2.5}$
  - Cardiovascular and respiratory hospitalizations

    Bell et al., **2009** *Am J Respir Crit Care Med*

- Elemental carbon, organic carbon, and nitrates in $PM_{2.5}$
  - Cardiovascular deaths

    Ostro et al., **2007** *Environ Health Perspectives*

- Elemental carbon in $PM_{2.5}$
  - Cardiovascular hospital admissions

    Levy et al., **2012** *Am J Epidemiology*

PM size: Ultrafine particles (UFP, <100 nm)

- Mean UFP number concentrations, not mass, associated with reductions in peak expiratory flow in adult asthmatics

Penttinen et al., **2001** *Eur Respir J*

- Asthma medication use associated with increased $PM_{2.5}$ mass and UFP number concentrations

von Klot et al., **2002** *Eur Respir J*

- UFP number concentrations (not $PM_{2.5}$ mass) associated with daily total and cardio-respiratory mortality

Stölzel et al., **2007** *J Expo Sci Environ Epidem*

- UFP concentrations associated with strongest risk of stroke

Andersen et al., **2010** *Eur Heart J*

Summary of PM health effects

- Myocardial infarction (heart attack)
- Stroke
- Arrhythmia (irregular heart beat)
- Heart failure exacerbation
- Lung cancer
- Children’s lung growth
- Hospitalizations for asthma
- Mortality
- ***No apparent thresholds***

- Health risks link to outdoor measurements, so we don’t really know enough about actual indoor exposures & health effects

  → We don’t really know what threshold to target

Pope and Dockery, **2006** *J Air Waste Manage Assoc*; Brook et al., **2010** *Circulation*

INTEGRATED SCIENCE ASSESSMENT FOR PM2.5

PM in the U.S.

- We can turn to the US EPA *Integrated Science Assessment for Particulate Matter*
  - 2228 pages dedicated to describing and summarizing impacts of particulate matter on human health and the environment
  - Summary of PM standards since 1971:

Table 1-1. Summary of NAAQS promulgated for PM, 1971-2006.

Year (Final Rule)	Indicator	Avg Time	Level	Form
1971 (36 FR 8186)	TSP (Total Suspended Particulates)	24 h	260 µg/m³ (primary) 150 µg/ m³ (secondary)	Not to be exceeded more than once per yr
		Annual	75 µg/m³ (primary)	Annual geometric mean
1987 (52 FR 24634)	PM_{10}	24 h	150 µg/m³	Not to be exceeded more than once per yr on average over a 3-yr period
		Annual	50 µg/m³	Annual arithmetic mean, averaged over 3 yr
1997 (62 FR 38652)	$PM_{2.5}$	24 h	65 µg/m³	98th percentile, averaged over 3 yr
		Annual	15 µg/m³	Annual arithmetic mean, averaged over 3 yr[1]
	PM_{10}	24 h	150 µg/m³	Initially promulgated 99th percentile, averaged over 3 yr; when 1997 standards were vacated in 1999, the form of 1987 standards remained in place (not to be exceeded more than once per yr on average over a 3-yr period)
		Annual	50 µg/m³	Annual arithmetic mean, averaged over 3 yr
2006 (71 FR 61144)	$PM_{2.5}$	24 h	35 µg/m³	98th percentile, averaged over 3 yr
		Annual	15 µg/m³	Annual arithmetic mean, averaged over 3 yr[2]
	PM_{10}	24 h	150 µg/m³	Not to be exceeded more than once per yr on average over a 3-yr period

Note: When not specified, primary and secondary standards are identical.

EPA Integrated Science Assessment for PM

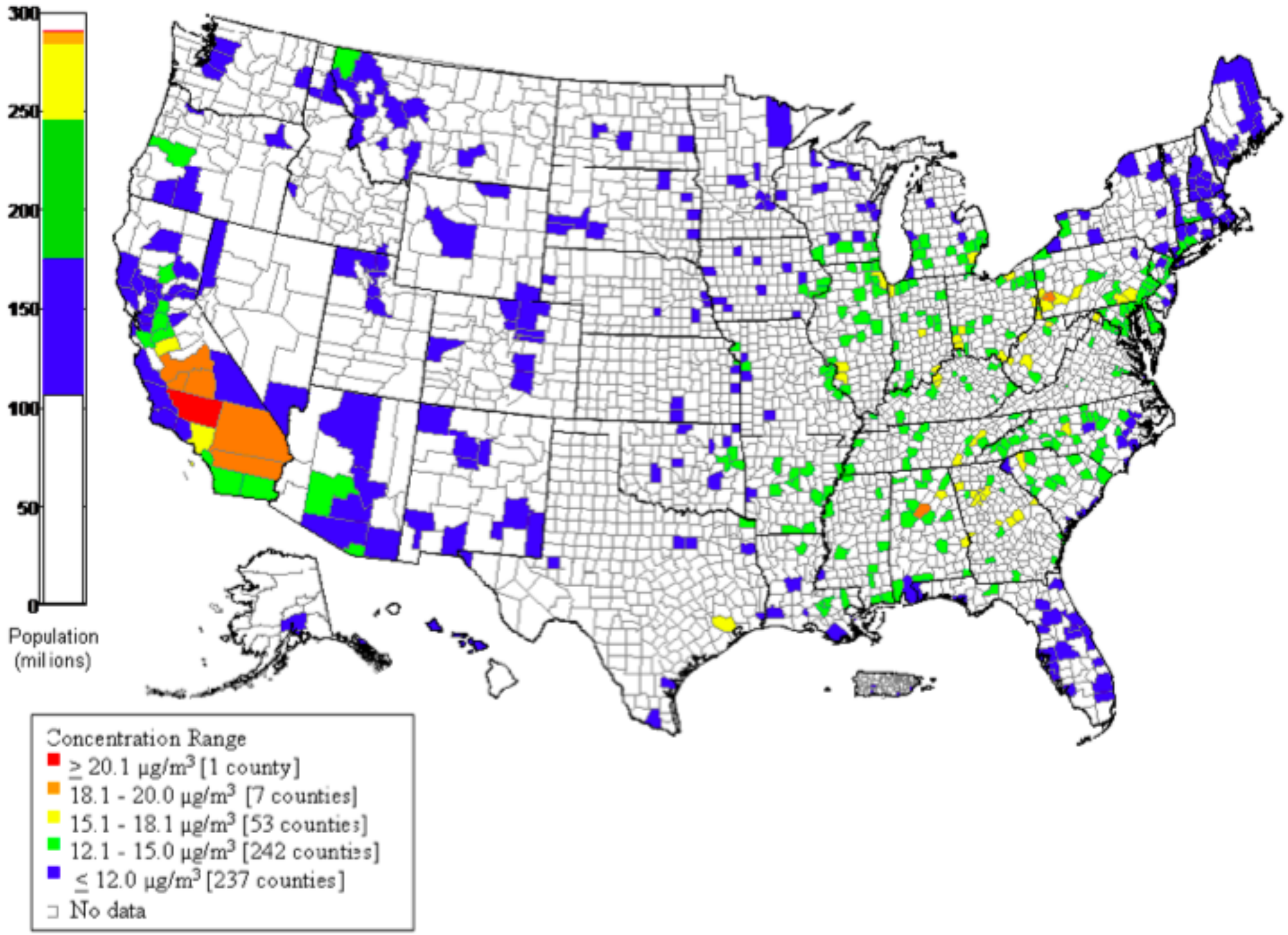
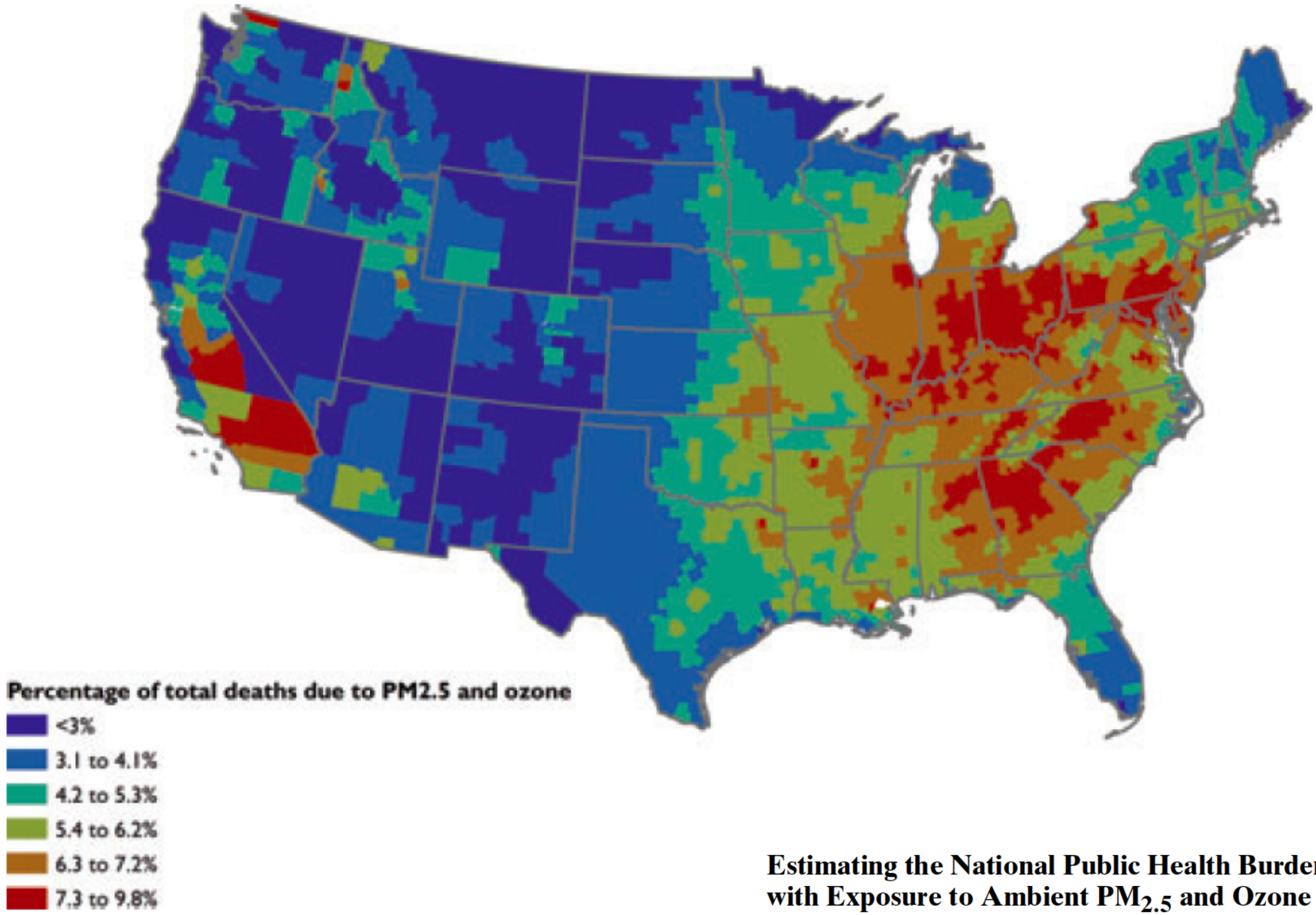


Figure 3-9. Three-yr avg 24-h $PM_{2.5}$ concentration by county derived from FRM or FRM-like data, 2005-2007. The population bar shows the number of people residing within counties that reported county-wide average concentrations within the specified ranges.

Outdoor air pollution and mortality



Estimating the National Public Health Burden Associated with Exposure to Ambient $PM_{2.5}$ and Ozone

Neal Fann,* Amy D. Lamson, Susan C. Anenberg, Karen Wesson, David Risley, and Bryan J. Hubbell

Fann et al., **2012** *Risk Analysis*

Outdoor air pollution and mortality

Estimating the National Public Health Burden Associated with Exposure to Ambient $PM_{2.5}$ and Ozone

Neal Fann,* Amy D. Lamson, Susan C. Anenberg, Karen Wesson, David Risley, and Bryan J. Hubbell

- Fann et al. (2012) estimated that 130,000 and 4,700 deaths were caused by $PM_{2.5}$ and ozone in US, respectively, in 2005
  - Nearly 1.1 million life years lost from $PM_{2.5}$ exposure and approximately 36,000 life years lost from ozone exposure
  - Among the 10 most populous counties, the percentage of deaths attributable to $PM_{2.5}$ and ozone ranged from 3.5% in San Jose to 10% in Los Angeles

Assuming: 6 ± 2% increase per 10 μg/m^3 in $PM_{2.5}$

Krewski et al., **2009** HEI Research Report

Fann et al., **2012** *Risk Analysis*

EPA Integrated Science Assessment for PM

Subjective causality….

Table 2-1. Summary of causal determinations for short-term exposure to $PM_{2.5}$.

Size Fraction	Outcome	Causality Determination
$PM_{2.5}$	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Causal

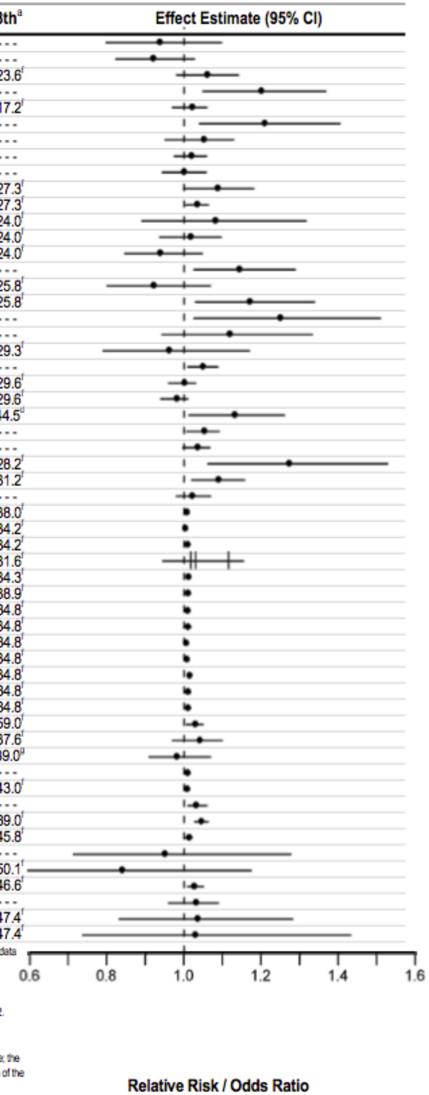
Table 2-2. Summary of causal determinations for long-term exposure to $PM_{2.5}$.

Size Fraction	Outcome	Causality Determination
$PM_{2.5}$	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Causal
	Reproductive and Developmental	Suggestive
	Cancer, Mutagenicity, and Genotoxicity	Suggestive

EPA Integrated Science Assessment for PM

Epidemiology data for short-term $PM_{2.5}$

Study	Outcome	Mean[a]	98th[a]
Chimonas & Gessner (2007, 093261)	Asthma HA	6.1	- - -
	LRI HA	6.1	- - -
Lisabeth et al. (2008, 155939)	Ischemic Stroke/TIA HA	7.0[e]	23.6[f]
Slaughter et al. (2005, 073854)	Asthma Exacerbation	7.3[e]	- - -
Rabinovitch et al. (2006, 088031)	Asthma Medication Use	7.4	17.2[f]
Chen et al. (2004, 087262)	COPD HA	7.7	- - -
Chen et al. (2005, 087555)	Respiratory HA	7.7	- - -
Fung et al. (2006, 089789)	Respiratory HA	7.7	- - -
Villeneuve et al. (2003, 055051)	Nonaccidental Mortality	7.9	- - -
Stieb et al. (2000, 011675)	CVD ED Visits	8.5	27.3[f]
	Respiratory ED Visits	8.5	27.3[f]
Villeneuve et al. (2006, 090191)	Hemhrgc Stroke HA	8.5	24.0[f]
	Ischemic Stroke HA	8.5	24.0[f]
	TIA HA	8.5	24.0[f]
Lin et al. (2005, 087828)	RTI HA	9.6	- - -
Mar et al. (2004, 057309)	Respiratory Symptoms (any)	9.8[c]	25.8[f]
	Respiratory Symptoms (any)	9.8[c]	25.8[f]
Rich et al. (2005, 079620)	Ventricular Arrhythmia	9.8[e]	- - -
Dockery et al. (2005, 078995)	Ventricular Arrhythmia	10.3[e]	- - -
Rabinovitch et al. (2004, 096753)	Asthma Exacerbation	10.6[d]	29.3[f]
Pope et al. (2006, 091246)	IHD HA	10.7[c]	- - -
Slaughter et al. (2005, 073854)	CVD HA	10.8	29.6[f]
	Respiratory ED Visits	10.8	29.6[f]
Pope et al. (2008, 191969)	CHF HA	10.8	44.5[d]
Zanobetti and Schwartz (2006, 090195)	MI HA	11.1[e]	- - -
	Pneumonia HA	11.1[e]	- - -
Peters et al. (2001, 016546)	MI	12.1	28.2[f]
Delfino et al. (1997, 082687)	Respiratory HA (summer)	12.1	31.2[f]
Sullivan et al. (2005, 050854)	MI	12.8	- - -
Burnett et al. (2004, 086247)	Nonaccidental Mortality	12.8	38.0[f]
Bell et al. (2008, 156266)	Respiratory HA	12.9[d]	34.2[f]
	CVD HA	12.9[d]	34.2[f]
Wilson et al. (2007, 157149)[m]	CVD Mortality	13.0	31.6[f]
Zanobetti & Schwartz (2009, 188462)	Nonaccidental Mortality	13.2[d]	34.3[f]
Burnett and Goldberg (2003, 042798)	Nonaccidental Mortality	13.3	38.9[f]
Dominici et al. (2006, 088398)	CBVD HA	13.3	34.8[f]
	PVD HA	13.3	34.8[f]
	IHD HA	13.3	34.8[f]
	Dysrhythmia HA	13.3	34.8[f]
	CHF HA	13.3	34.8[f]
	COPD HA	13.3	34.8[f]
	RTI HA	13.3	34.8[f]
Fairley (2003, 042850)	Nonaccidental Mortality	13.6	59.0[f]
Zhang et al. (2009, 191970)	ST Segment Depression	13.9[j]	37.6[f]
O'Connor et al. (2008, 156818)	Wheeze/Cough	14.0[c]	39.0[g]
Klemm and Mason (2003, 042801)	Nonaccidental Mortality	14.7[e,j]	- - -
Franklin et al. (2008, 097426)	Nonaccidental mortality	14.8	43.0[f]
NYDOH (2006, 090132)	Asthma ED Visits	15.0[k]	- - -
Ito et al. (2007, 156594)	Asthma HA	15.1	39.0[f]
Franklin et al. (2007, 091257)	Non-accidental Mortality	15.6	45.8[f]
Rich et al. (2006, 089814)	Ventricular Arrhythmia	16.2[e]	- - -
Symons et al. (2006, 091258)	CHF HA	16.5[d]	50.1[f]
Sheppard (2003, 042826)	Asthma HA	16.7	46.6[f]
NYDOH (2006, 090132)	Asthma ED Visits	16.7[l]	- - -
Burnett et al. (1997, 084194)	Respiratory HA (summer)	16.8	47.4[f]
	CVD HA (summer)	16.8	47.4[f]



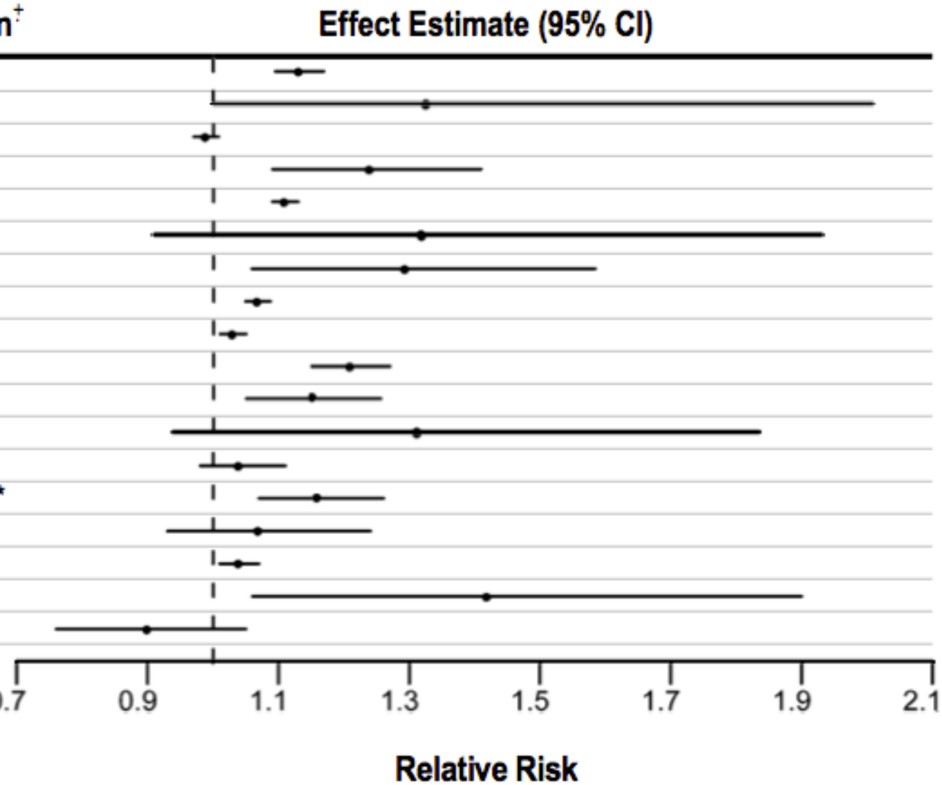
[a] µg/m³
[b] Study did not present mean; median presented.
[c] Mean estimated from data in study.
[d] Mean value slightly different from those reported in the published study or not reported in the published study; mean was either provided by study authors or calculated from data provided by study authors.
[e] Mean value not reported in study; median presented.
[f] 98th percentile of $PM_{2.5}$ distribution was either provided by study authors or calculated from data provided by study authors.
[g] 98th estimated from data in study.
[h] Averaged annual values for years in study from data provided by study author.
[i] Air quality data obtained from original study by Schwartz et al. (1996, 077325)
[j] Mean $PM_{2.5}$ concentration reported is for lag 0-2.
[k] Bronx; TEOM data.
[l] Manhattan; TEOM data.
[m] Study does not present an overall effect estimate; the vertical lines represent the effect estimate for each of the areas of Phoenix examined.

Figure 2-1. Summary of effect estimates (per 10 µg/m³) by increasing concentration from U.S. studies examining the association between short-term exposure to $PM_{2.5}$ and cardiovascular and respiratory effects, and mortality, conducted in locations where the reported mean 24-h avg $PM_{2.5}$ concentrations were <17 µg/m³.

EPA Integrated Science Assessment for PM

Epidemiology data for long-term $PM_{2.5}$

Study	Outcome	Mean+
Zeger et al. (2008, 191951)	All-Cause Mortality, Central U.S.	10.7
Kim et al. (2004, 087383)	Bronchitis (Children)	12.0
Zeger et al. (2008, 191951)	All-Cause Mortality, Western U.S.	13.1
Miller et al. (2007, 090130)	CVD Morbidity or Mortality	13.5
Eftim et al. (2008, 099104)	All-Cause Mortality, ACS Sites	13.6
Goss et al. (2004, 055624)	All-Cause Mortality	13.7
McConnell et al. (2003, 049490)	Bronchitis (Children)	13.8
Zeger et al. (2008, 191951)	All-Cause Mortality, Eastern U.S.	14.0
Krewski et al. (2009, 191193)	All-Cause Mortality	14.0
Eftim et al. (2008, 099104)	All-Cause Mortality, Harv 6-Cities	14.1
Lipfert et al. (2006, 088756)	All-Cause Mortality	14.3
Dockery et al. (1996, 046219)	Bronchitis (Children)	14.5
Woodruff et al. (2008, 098386)	Infant Mortality (Respiratory)	14.8
Laden et al. (2006, 087605)	All-Cause Mortality	16.4*
Woodruff et al. (2008, 098386)	Infant Mortality (Respiratory)	19.2
Enstrom (2005, 087356)	All-Cause Mortality	23.4
Chen et al. (2005, 087942)	CHD Mortality, Females	29.0
	CHD Mortality, Males	29.0



* Mean estimated from data in study
\+ μg/m³

Figure 2-2. Summary of effect estimates (per 10 μg/m³) by increasing concentration from U.S. studies examining the association between long-term exposure to $PM_{2.5}$ and cardiovascular and respiratory effects, and mortality.

EPA Integrated Science Assessment for PM

Subjective causality….

2.3.5.1. Effects of Short-Term Exposure to UFPs

Table 2-4. Summary of causal determinations for short-term exposure to UFPs.

Size Fraction	Outcome	Causality Determination
UFPs	Cardiovascular Effects	Suggestive
	Respiratory Effects	Suggestive

Summary of PM health effects from EPA ISA

- Short-term exposure exacerbates cardiovascular and pulmonary disease
  - Increases risk of having symptoms, requiring medical attention, and/or even dying

- Long-term exposure results in even larger increased risks of respiratory and cardiovascular disease and death

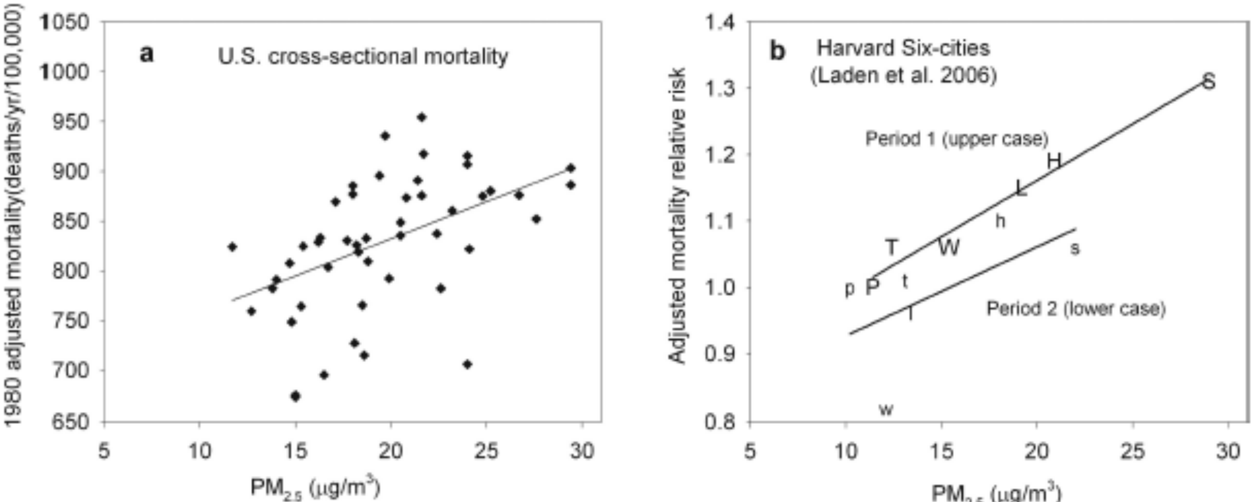
- US policy appears to have improved human health
  - But has not eliminated concern

WHAT ABOUT INDOOR EXPOSURES?

And epidemiology studies

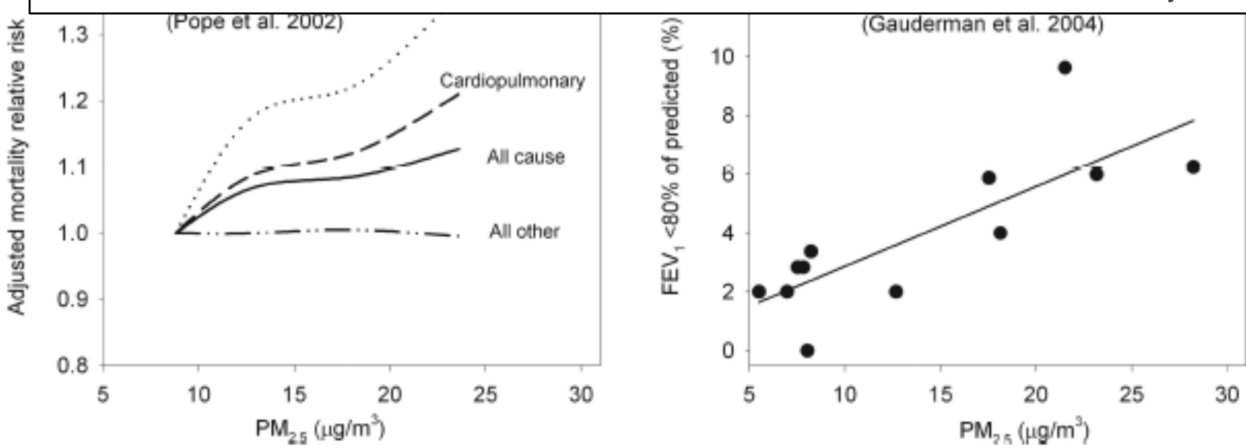
Outdoor air epidemiology studies: A problem

Associations with ambient fine particulate matter ($PM_{2.5}$)



Typical C-R effect estimate for $PM_{2.5}$ and mortality: ~7% per 10 μg/m³

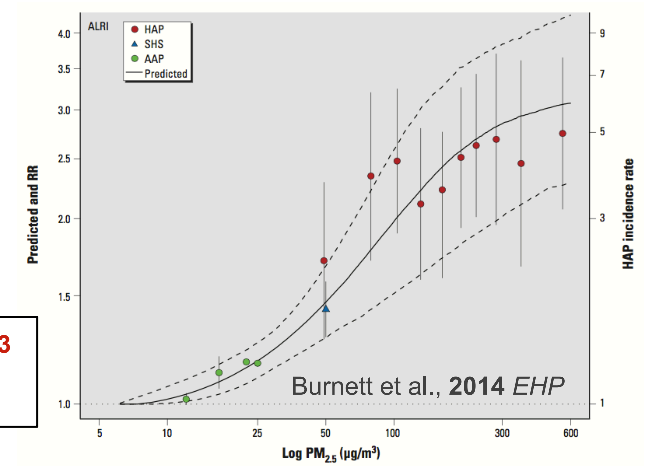
Fann et al. **2016** *Risk Analysis*



Pope and Dockery, **2006** *J Air Waste Manage Assoc*

Concentration-Response (C-R) Function

$$\Delta y_i = y_0[\exp(\beta_i \times \Delta E_i) - 1]$$



Burnett et al., **2014** *EHP*

Nearly all outdoor air pollution epidemiology studies neglect an important point…

We spend most of our time indoors!

Indoor proportions of outdoor pollutants

- Most of the health effect estimates we've described use outdoor monitoring data
  - Usually assumes everyone in a location is exposed to the same concentration
- We've already discussed (and had HW problems) on how outdoor pollution becomes indoor pollution
  - Where we spend most of our time
- How do we get better exposure estimates and thus health effect responses?

Example: Indoor exposure to “outdoor PM_{10}”

Indoor Exposure to “Outdoor PM_{10}”

Assessing Its Influence on the Relationship Between PM_{10} and Short-term Mortality in U.S. Cities

- A recent study attempted to account for variations in *AER* across the US and, after assuming some base values for k_{dep} and P for PM_{10}, they predicted indoor concentrations of outdoor PM_{10} inside average homes in each region
  - Compared those estimates to short-term mortality data to see if their predicted average indoor concentration correlated with mortality rates

$$\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{windows\_closed} = \frac{P\lambda_{\inf}}{\lambda_{\inf} + k_{dep,\inf}}$$

$$\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{windows\_open} = \frac{(1)\lambda_{open}}{\lambda_{open} + k_{dep,open}}$$

$$\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{AC\_on} = \frac{P\lambda_{\inf}}{\lambda_{\inf} + k_{dep,\inf} + \eta f_{HVAC} \, Q_{HVAC}/V}$$

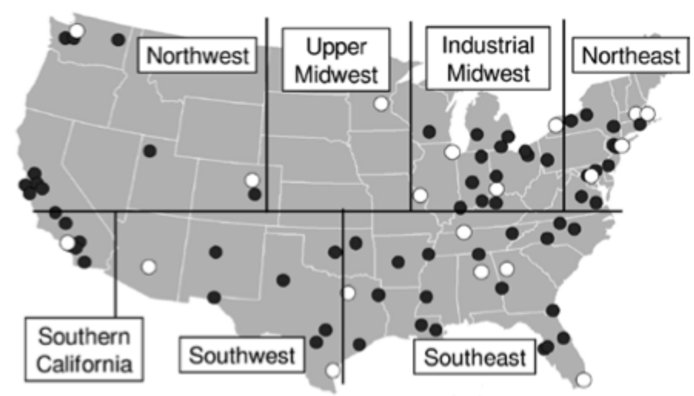


FIGURE 1. Location within the seven U.S. regions of the 19 cities from the NMMAPS with detailed building infiltration rates (open circles) that were used in the original analysis (Figure 2) and the 64 NMMAPS cities with less well-characterized building infiltration rates (closed circles) that were added to the extended analysis (Figure 3).

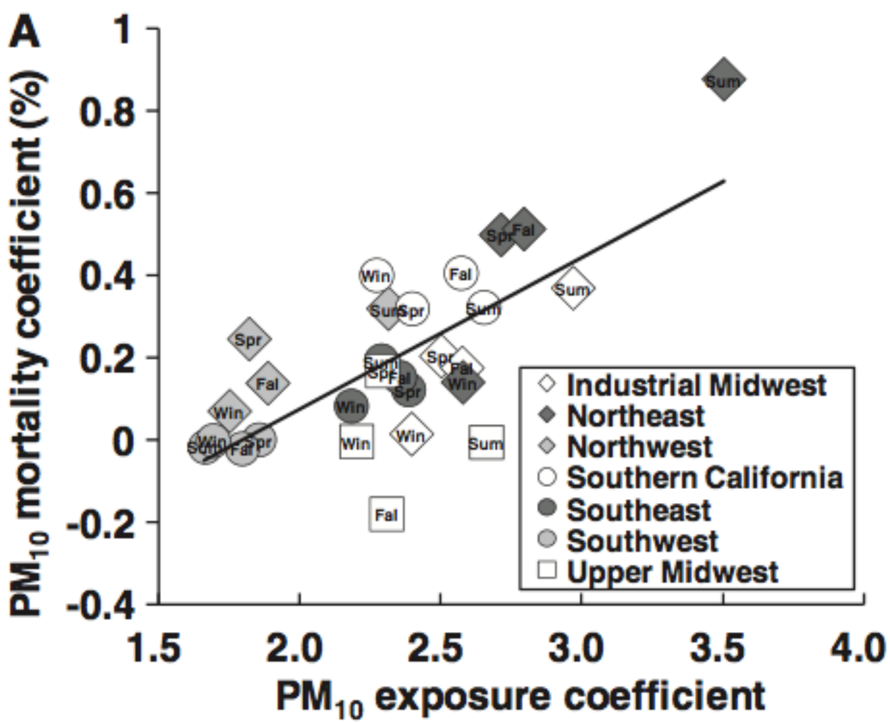
Chen et al., **2012** *Epidemiology*

Example: Indoor exposure to "outdoor PM_{10}"

Indoor Exposure to "Outdoor PM_{10}"

Assessing Its Influence on the Relationship Between PM_{10} and Short-term Mortality in U.S. Cities

$$\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{total} = f_{windows\_closed}\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{windows\_closed} + f_{windows\_open}\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{windows\_open} + f_{AC\_on}\left\{\frac{\Delta[PM_{10}]_{in}}{\Delta[PM_{10}]_{out}}\right\}_{AC\_on} = \beta_{\exp}$$



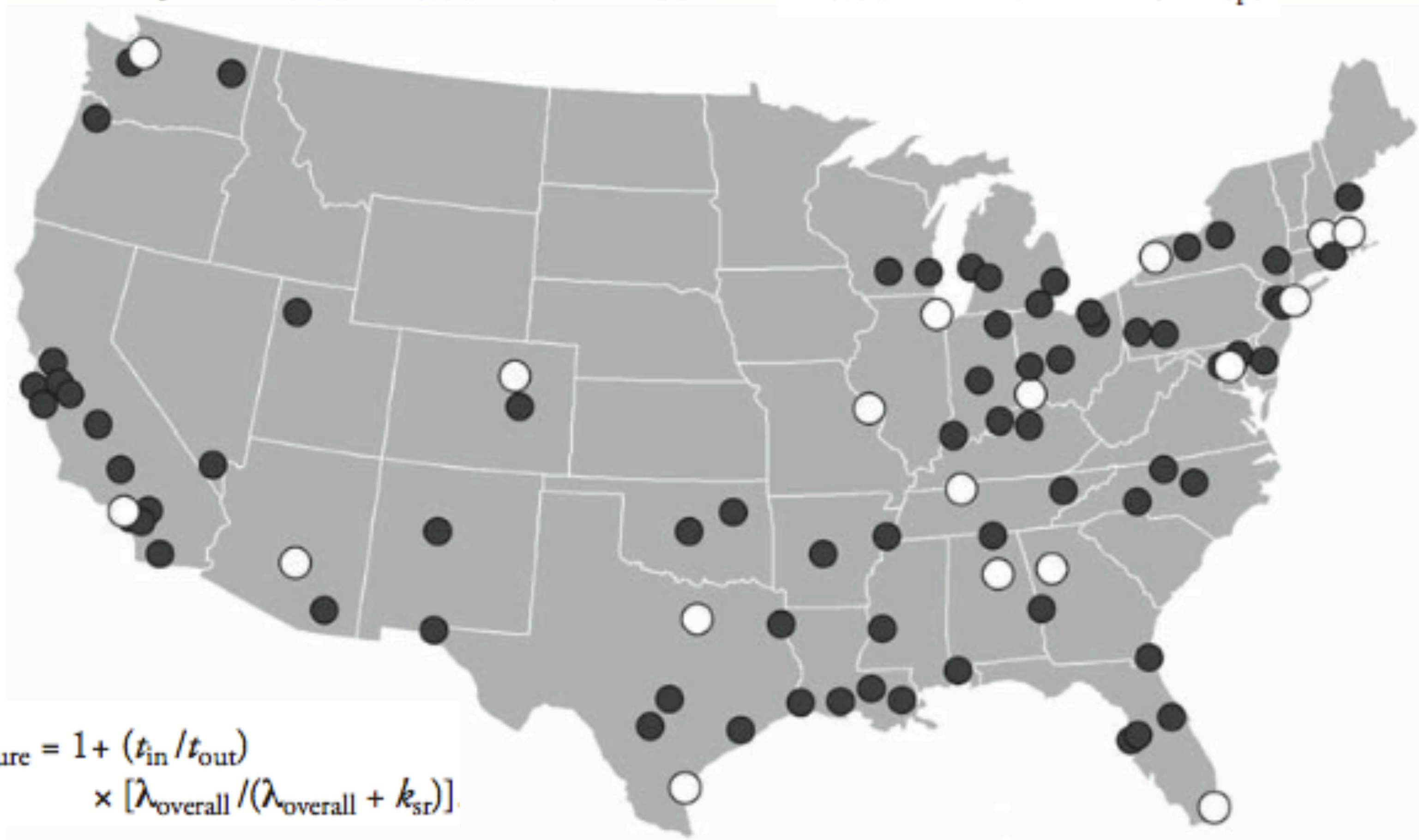
Strong correlations suggest indoor exposures are an important component to outdoor PM exposure

Chen et al., **2012** *Epidemiology*

Indoor exposure to outdoor O_3 and short-term morality

$$\Delta[O_3]_{in} = [\lambda_{overall}/(\lambda_{overall} + k_{sr})]\ 10\ \text{ppb}$$

$$\lambda_{overall} = \lambda_{infilt} + (x)(1-y)\ \lambda_{open}$$



$$\Delta O_{3\_exposure} = 1 + (t_{in}/t_{out}) \times [\lambda_{overall}/(\lambda_{overall} + k_{sr})]$$

Figure 1. Location of the 18 NMMAPS cities for which detailed modeled infiltration rates were available (open circles) and the 72 additional NMMAPS cities included in the extended analysis (filled circles).

Chen et al., **2012** *Environ Health Persp*

Indoor exposure to outdoor O_3 and short-term morality

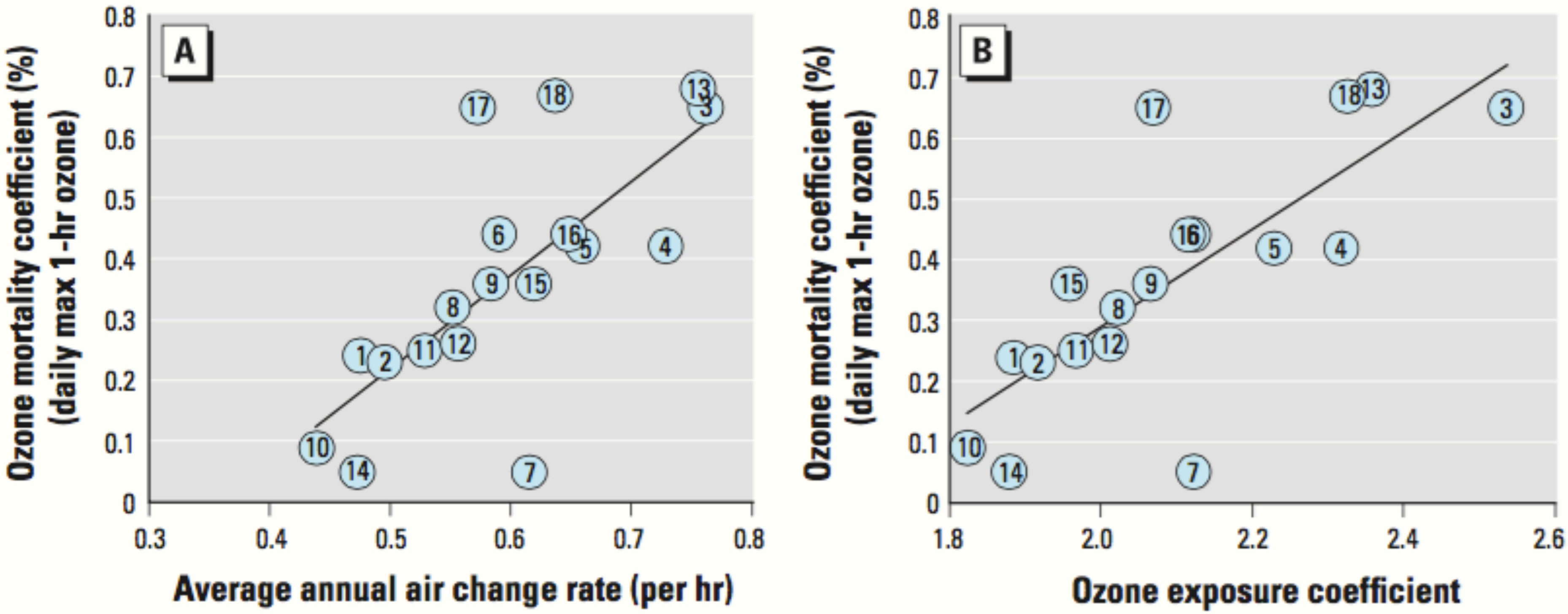


Figure 2. For the 18 NMMAPS cities for which detailed modeled infiltration rates were available, ozone mortality coefficients versus (*A*) average annual air change rates ($y = 1.54x - 0.55$, $R^2 = 0.51$), and (*B*) ozone exposure coefficients ($y = 0.81x - 1.32$, $R^2 = 0.58$). Ozone mortality coefficients based on daily maximum (max) 1-hr ozone. Numbers within circles refer to numbers listed in the first column of Table 1.

Chen et al., **2012** *Environ Health Persp*

OTHER INDOOR AIR EPIDEMIOLOGY STUDIES

Association between gas cooking and respiratory disease in children

Melia et al., *British Medical Journal* **1977**, 2, 149-152

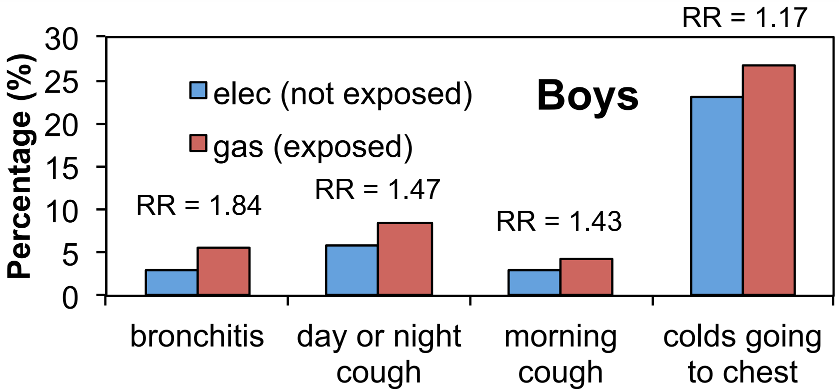
Gas stoves

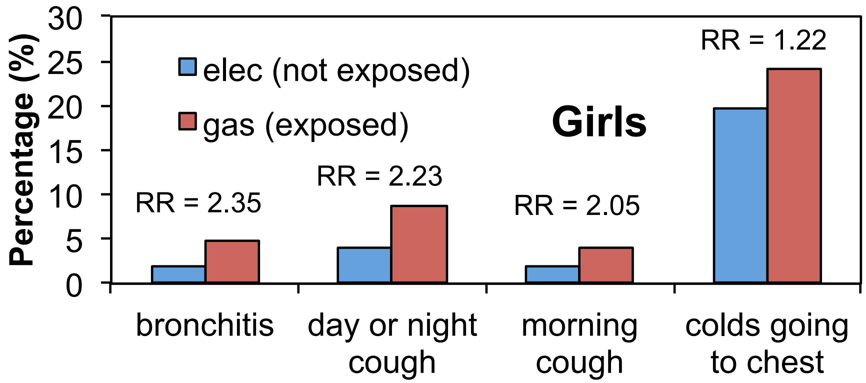
- Four year longitudinal study of the prevalence of respiratory symptoms and disease in almost 6000 6-11 year old school children
  - Children from homes in which gas was used for cooking were found to have more cough, "colds going to the chest," and bronchitis than children from homes where electricity was used

TABLE I—*Prevalence (%) of respiratory symptoms and diseases during last 12 months in boys and girls according to type of fuel used for cooking in the home*

Symptoms and diseases	Boys			Girls		
	Electricity	Gas	P*	Electricity	Gas	P*
Bronchitis	3·1	5·7	<0·001	2·0	4·7	<0·001
Day or night cough	5·8	8·5	<0·007	3·9	8·7	<0·001
Morning cough	3·0	4·3	<0·07	2·0	4·1	<0·001
Colds going to chest	23·0	26·8	<0·02	19·8	24·1	<0·006
Wheeze	10·3	11·2	≈0·5	5·7	8·6	<0·005
Asthma	1·8	2·7	≈0·2	1·0	1·6	≈0·2
No of children	1648	1274		1556	1280	

*Probability value for difference between prevalence rates, χ^2 test.





Respiratory Symptoms in Children and Indoor Exposure to Nitrogen Dioxide and Gas Stoves

Gas stoves

Garrett et al., *Am. J. Respir. Crit. Care. Med.* **1998**, 158, 891-895

- NO_2 measured in 80 homes in Australia using passive samplers
  - 148 children 7-14 years old were recruited (53 had asthma)
  - Indoor median NO_2 concentrations were 6 ppb (max 128 ppb)
  - Respiratory symptoms were more common in children exposed to a gas stove (OR = 2.3) after adjustments for parental allergy, parental asthma, and gender
  - NO_2 exposure was a marginal risk factor for respiratory symptoms
    - Gas stove was still a risk factor after accounting for NO_2
    - What does that mean?

Respiratory Symptom	% of Children	Gas Stove Exposure OR*	Gas Stove Exposure 95% CI	Bedroom NO_2 OR*	Bedroom NO_2 95% CI
Cough	59	2.25	1.13–4.49	1.47	0.99–2.18
Shortness of breath	31	1.49	0.72–3.08	1.23	0.92–1.64
Waking short of breath	17	1.01	0.42–2.45	1.04	0.71–1.53
Wheeze	24	1.79	0.80–3.99	1.15	0.85–1.54
Asthma attacks	23	1.73	0.77–3.90	1.06	0.77–1.46
Chest tightness	13	3.11	1.07–9.05	1.12	0.81–1.56
Cough in the morning	24	1.42	0.63–3.19	1.25	0.92–1.69
Chest tightness in morning	14	1.10	0.42–2.88	1.32	0.95–1.84

* Adjusted for parental asthma, parental allergy, and sex.

A cross-sectional study of the association between ventilation of gas stoves and chronic respiratory illness in U.S. children enrolled in NHANESIII Kile et al., *Environmental Health* **2014**, 13, 71

Gas stoves

- The Third National Health and Nutrition Examination Survey was used to identify U.S. children aged 2–16 years with information on respiratory outcomes (asthma, wheeze, and bronchitis) who lived in homes where gas stoves were used in the previous 12 months and whose parents provided information on ventilation. Logistic regression models evaluated the association between prevalent respiratory outcomes and ventilation in homes that used gas stoves for cooking and/or heating. Linear regression models assessed the association between spirometry measurements and ventilation use in children aged 8–16 years.

Table 2 Adjusted Odds ratios and 95% confidence intervals for the association between respiratory illnesses in children aged 2–16 years who live in households that use gas stove with ventilation compared to households that use gas stoves without ventilation (Model 1)

	Ever diagnosed with asthma[a] (N = 5,745)		Wheeze in past 12 months[b] (N = 5,744)		Ever diagnosed with bronchitis[c] (N = 7,255)	
Ventilation of gas stove	No. cases	OR (95% CI)	No. cases	OR (95% CI)	No. cases	OR (95% CI)
No	*269*	1 Ref.	*561*	1 Ref.	*188*	1 Ref.
Yes	*224*	0.64 (0.43, 0.97)*	*458*	0.60 (0.42, 0.86)*	*128*	0.60 (0.37, 0.95)*

*P-value <0.05.
[a]Adjusted for age group, sex, parental history of asthma or hay fever, and furry or feathery pets in the house, household income < $20,000, and BMI percentiles for age.
[b]Adjusted for age group, parental history of asthma or hay fever, furry or feathery pets in the house, indoor tobacco smoke, race-ethnicity, household income < $20,000, and BMI percentile for age.
[c]Adjusted for age group, parental history of asthma or hay fever, indoor tobacco smoke, race-ethnicity, household income < $20,000, and census region.

“One-second forced expiratory volume (FEV_1) and FEV_1/FVC ratio was also higher in girls who lived in households that used gas stoves with ventilation compared to households that used gas stoves without ventilation.”

Association of domestic exposure to volatile organic compounds with asthma in young children

Rumchev et al., *Thorax* **2004**, 59, 746-751

- Population based case-control study conducted in Perth, Australia
  - Children 6 months to 3 years of age (cases = 88; controls = 104)
  - Cases had asthma; controls did not
  - Housing questionnaires were given and indoor VOCs were measured

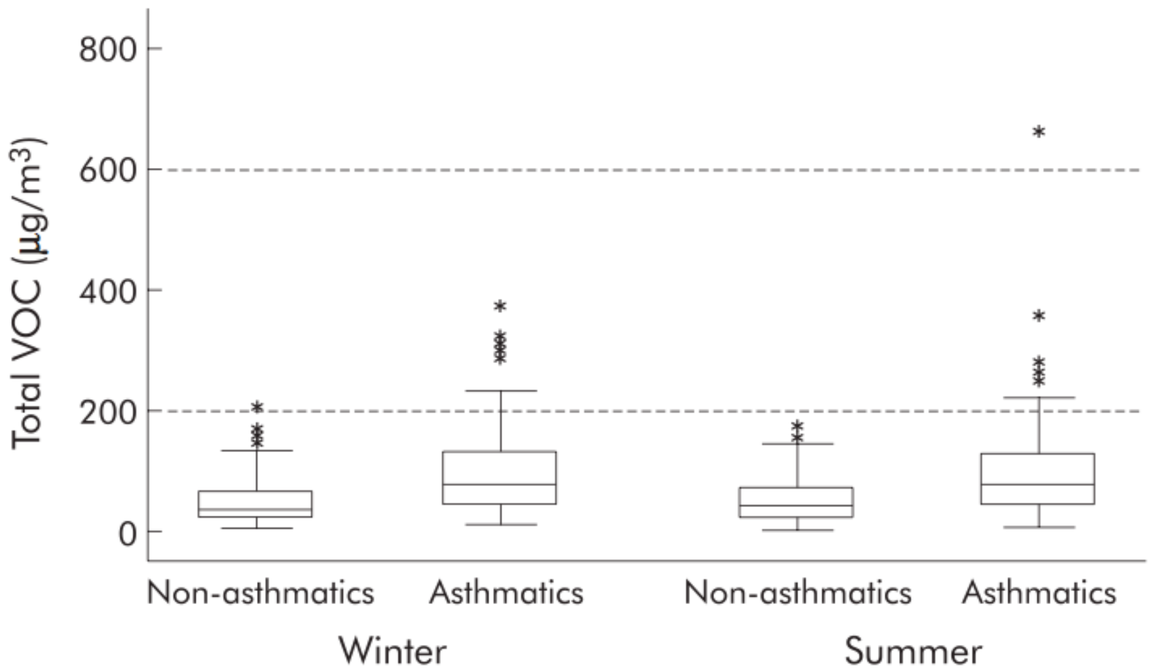


Figure 1 Seasonal differences in exposure levels to total volatile organic compounds (VOCs, µg/m^3) for asthmatic and non-asthmatic children.

Association of domestic exposure to volatile organic compounds with asthma in young children

VOCs

Rumchev et al., *Thorax* **2004**, 59, 746-751

- Cases had significantly higher VOC levels than controls ($p < 0.01$)
  - Highest odds ratios were benzene > ethylbenzene > toluene

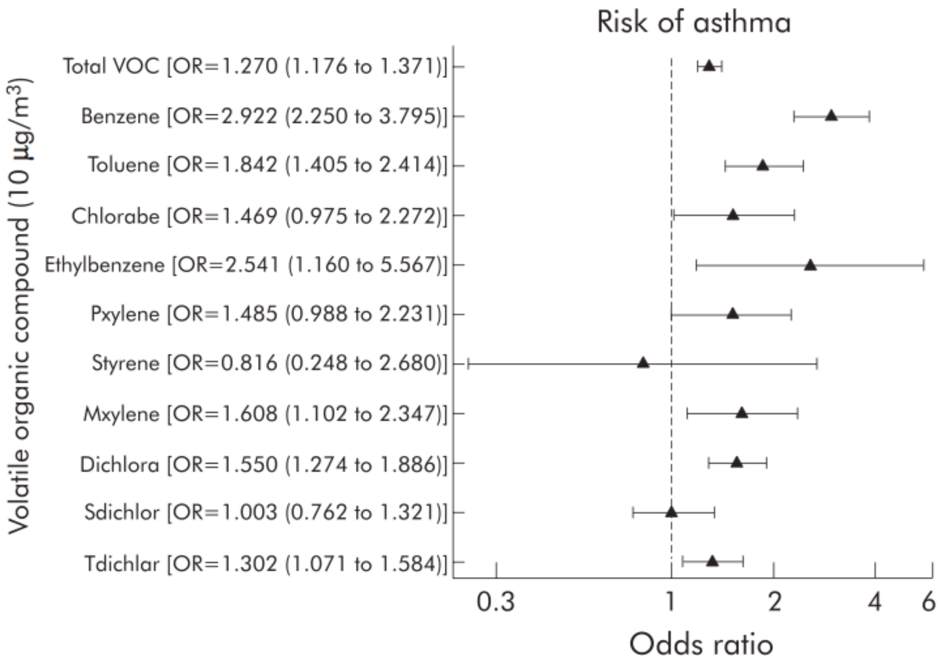


Figure 3 Adjusted odds ratio with ±95% confidence intervals for the risk of asthma with each 10 mg increase in exposure to VOCs.

Exposure-response curve

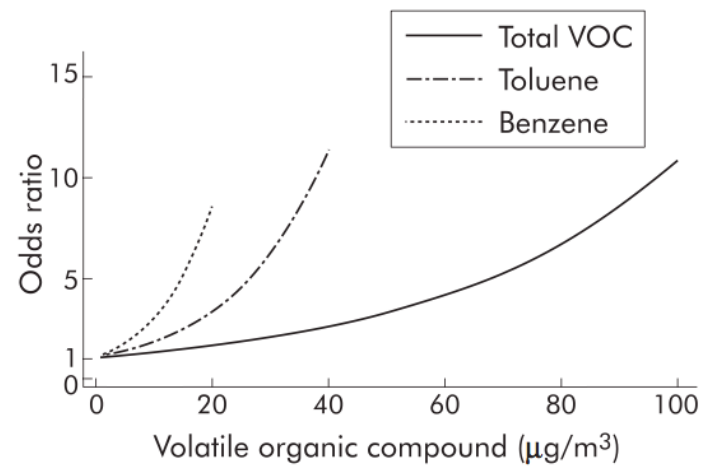


Figure 2 Asthma in young children associated with exposure to indoor volatile organic compounds (µg/m³): odds ratios adjusted for age, sex, atopy, socioeconomic status, smoking indoors, air conditioning, house dust mites, and gas appliances.

Frequent use of chemical household products is associated with persistent wheezing in pre-school age children

Sherriff et al., *Thorax* **2005**, 60, 45-49

Use of cleaning products

- Frequency of use of 11 chemical based domestic products was determined via questionnaires completed by women during pregnancy
  - Given a "total chemical burden" score (TCB)
- Four wheezing patterns were defined for the period from baby's birth to 42 months of age (never, transient early, persistent, late onset)
- 13971 children tracked; completely data for 7019 children

Fifteen product categories were included in the questionnaire and, from this initial list, we selected the 11 most frequently used (by at least 5% of the study sample). The products chosen (and the percentages of women using them) were: disinfectant (87.4%), bleach (84.8%), carpet cleaner (35.8%), window cleaner (60.5%), dry cleaning fluid (5.4%), aerosols (71.7%), turpentine/white spirit (22.6%), air fresheners (spray, stick or aerosol) (68%), paint stripper (5.5%), paint or varnish (32.9%), and pesticides/insect killers (21.2%). A simple score for frequency of use of each product was derived (0 = not at all, 1 = less than once a week, 2 = about once a week, 3 = most days, 4 = every day) and the scores for each product were summed to produce a total chemical burden (TCB) score for each respondent which could range from 0 (no exposure) to 55 (exposed to all 11 products daily).

Frequent use of chemical household products is associated with persistent wheezing in pre-school age children

Sherriff et al., *Thorax* **2005**, 60, 45-49

Use of cleaning products

Table 1 Unadjusted and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for wheezing phenotypes* (transient early wheeze, persistent wheeze, and late onset wheeze (0–42 months)) according to total chemical burden (TCB) score measured during pregnancy (continuous)

Wheezing phenotype	% (N)	Unadjusted OR (95% CI) (N = 7019)	Unadjusted p value	Adjusted OR** (95% CI) (N = 5691)	Adjusted p value
Never wheezed	71.2 (5001)	1 (reference)		1 (reference)	
Transient early wheeze	19.1 (1340)	1.02 (1.00 to 1.03)	0.04	1.01 (0.99 to 1.02)	0.6
Persistent wheeze	6.2 (432)	1.08 (1.05 to 1.11)	<0.0001	1.06 (1.03 to 1.09)	0.0001
Late onset wheeze	3.5 (246)	1.02 (0.99 to 1.05)	0.2	1.02 (0.98 to 1.06)	0.3

*Never wheezed 0–42 months. Transient early wheeze: wheeze 0–6 months and no wheeze 6–42 months. Persistent wheeze: wheeze 6–18 months, 18–30 months and 30–42 months. Late onset wheeze: wheeze onset 30–42 months.
**Adjusted for weekend exposure to environmental tobacco smoke at 6 months, maternal smoking during pregnancy, maternal history of asthma, maternal parity, crowding in the home, sex, contact with pets, damp housing, maternal age at delivery, maternal educational attainment, housing tenure, hours mother worked outside home, month of returning chemical usage questionnaire, and duration of breastfeeding.

Table 2 Unadjusted and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for wheezing phenotypes* (transient early wheeze, persistent wheeze, and late onset wheeze (0–42 months)) according to total chemical burden (TCB) score measured during pregnancy (bottom decile versus top decile)

Wheezing phenotype	Bottom decile of TCB % (N)	Top decile of TCB % (N)	Unadjusted OR (95% CI) (N = 7019)	Unadjusted p value	Adjusted OR** (95% CI) (N = 5691)	Adjusted p value
Never wheezed	74.9 (603)	66.9 (338)	1 (reference)		1 (reference)	
Transient early wheeze	18.8 (151)	19.0 (96)	1.13 (0.90 to 1.50)	0.4	0.94 (0.60 to 1.40)	0.7
Persistent wheeze	4.0 (32)	10.1 (51)	2.84 (1.79 to 4.51)	<0.0001	2.30 (1.20 to 4.39)	0.012
Late onset wheeze	2.4 (19)	4.0 (20)	1.88 (0.99 to 3.57)	0.05	2.02 (0.80 to 5.15)	0.14

*Never wheezed 0–42 months. Transient early wheeze: wheeze 0–6 months and no wheeze 6–42 months. Persistent wheeze: wheeze 6–18 months, 18–30 months and 30–42 months. Late onset wheeze: wheeze onset 30–42 months.
**Adjusted for weekend exposure to environmental tobacco smoke at 6 months, maternal smoking during pregnancy, maternal history of asthma, maternal parity, crowding in the home, sex, contact with pets, damp housing, maternal age at delivery, maternal educational attainment, housing tenure, hours mother worked outside home, month of returning chemical usage questionnaire, and duration of breastfeeding.

The Use of Household Cleaning Sprays and Adult Asthma

Use of cleaning products

Zock et al., *Am. J. Respir. Crit. Care. Med.* **2007**, 176, 735-741

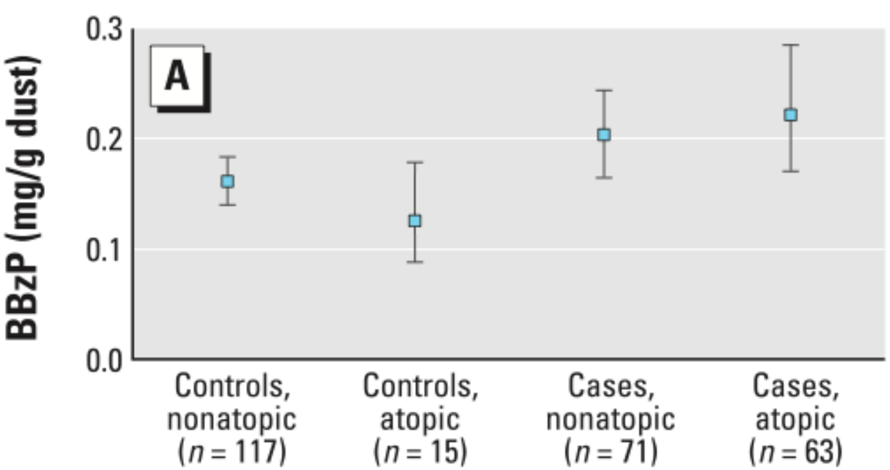
- Identified 3503 people in 10 countries who do the cleaning in their homes and who were free of asthma at the beginning of the study
- Frequency of use of 15 types of cleaning products was obtained by interview
- Tracked incidence of asthma
- Use of cleaning sprays at least weekly (42% of participants) was associated with asthma symptoms or medication use (RR = 1.49) and wheeze (RR = 1.39)
  - Asthma was higher among those using sprays at least 4 days per week (RR = 2.11)
  - Highest risks for glass-cleaning, furniture, and air-freshener sprays
  - Non-spray-form products were not associated

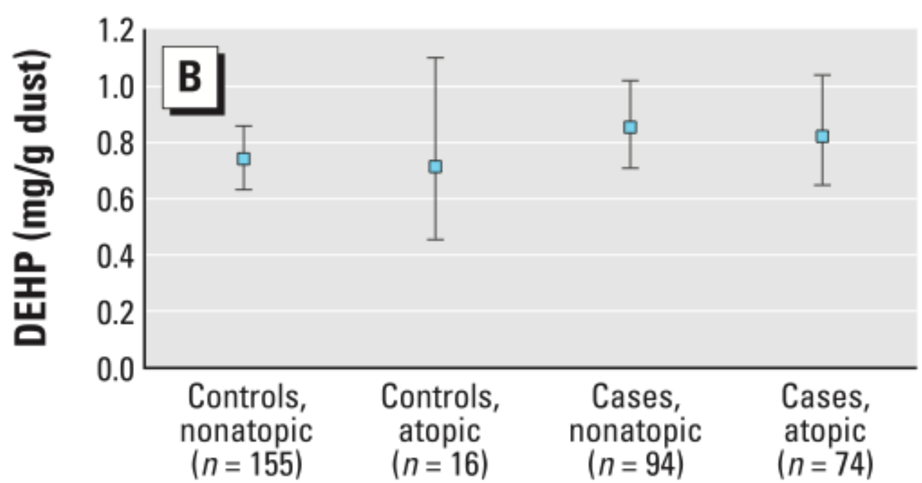
What about SVOCs?

The Association between Asthma and Allergic Symptoms in Children and Phthalates in House Dust: A Nested Case–Control Study

Bornehag et al., *Environ. Health Perspect.* **2004**, 112, 1393-1397

- Cohort of 10852 children
  - 198 cases with persistent allergic symptoms
  - 202 controls without symptoms
- Measured phthalate concentrations in house dust
- BBzp (butyl benzyl phthalate) was higher in cases than controls
  - Associated with rhinitis (stuffy/runny nose) and eczema (inflammation of skin)
- DEHP was associated with asthma





SVOCs and thyroid function

Relationship between Urinary Phthalate and Bisphenol A Concentrations and Serum Thyroid Measures in U.S. Adults and Adolescents from the National Health and Nutrition Examination Survey (NHANES) 2007–2008

- Analysis of urinary biomarker data of exposure to phthalates (DEHP, DBP) and BPA for 1346 adults and 329 adolescents using the National Health and Nutrition Examination Survey (NHANES)
  - Compared to serum thyroid measures
- Found significant relationships between phthalates (and possibly BPA) and altered thyroid hormones
  - These hormones play important roles in fetal and child growth and brain development, as well as metabolism, energy balance, and other functions in the nervous, cardiovascular, pulmonary, and reproductive systems

Meeker and Ferguson, *Environ Health Perspectives* **2011**

Ventilation rates and health

Association between ventilation rates in 390 Swedish homes and allergic symptoms in children

Bornehag et al., *Indoor Air* **2005**

- Same cases (198) and controls (202) from before
- Compared symptoms and diagnoses to AER measurements
  - Cases had significantly **lower** ventilation rates

Table 3 Differences in mean ventilation rate between cases and controls in different groups of buildings

Type of buildings	Cases	Controls	*P*-value: *t*-test	*P*-value: Mann–Whitney *U*
Single-family houses (*n*)	161	172		
Mean ach in total building (*n*)	0.34 (161)	0.38 (169)	0.025	0.014
Ach in child's bedroom (*n*)	0.32 (158)	0.37 (166)	0.020	0.011
Chain houses (*n*)	12	11		
Mean ach in total building (*n*)	0.37	0.32	0.627	0.622
Ach in child's bedroom (*n*)	0.40	0.33	0.412	0.712
Multi-family houses (*n*)	25	19		
Mean ach in total building (*n*)	0.49 (25)	0.47 (18)	0.793	1.000
Ach in child's bedroom (*n*)	0.50 (23)	0.52 (17)	0.807	0.967
All types of building (*n*)	198	202		
Mean ach in total building (*n*)	0.36 (198)	0.39 (198)	0.126	0.053
Ach in child's bedroom (*n*)	0.34 (193)	0.38 (194)	0.099	0.068

Significant difference was ~14% lower ACH in cases than controls

HVAC systems and health

Risk factors in heating, ventilating, and air-conditioning systems for occupant symptoms in US office buildings: the US EPA BASE study

Mendell et al., *Indoor Air* **2008**

- 'Building-related symptoms' in office workers were assessed in 97 air-conditioned office buildings in the US
- A primary correlation between building symptoms and HVAC characteristics was:
  - Outdoor air intakes less than 60 m above ground level were associated with significant increases in most symptoms
  - For upper respiratory symptoms, OR for intake heights were:
    - <30 m: OR = 2.0
    - 30-60 m: OR = 2.7
    - Below ground: OR = 2.1
    - Above 60 m: OR = 1.0
  - Poorly maintained humidification systems and infrequent cleaning of cooling coils and drain pans were also associated
    - What does this suggest?

AIR CLEANERS AND HEALTH

Air cleaners and health

Health benefits of particle filtration

Fisk **2013** *Indoor Air*

Photo from M.S. Waring and J.A. Siegel

Air cleaners typically reduce indoor PM concentrations by ~50%

Documented health improvements with air cleaners include:

- Improvements in lung function in asthmatics
- Fewer asthma-related doctor visits
- Improvements in cardiovascular and pulmonary function

Updated health effects review: Allergies and asthma

Table 3. Intervention Studies of Primarily Respiratory Health Outcomes in Homes With Subjects With Allergies or Asthma

Study	Brehler et al. (2003)	Francis et al. (2003)	Bernstein et al. (2006)	Sulser et al. (2009)
Subjects	44 adults with allergies and/or asthma	30 adults allergic to cats or dog allergen	19 mold-sensitized asthmatic children, age 5 to 17 years	30 asthmatic children sensitive to pet allergen
Type of building	Homes (24 rural, 20 urban)	Homes with cats or dogs	Homes with central forced air HVAC systems	Homes with high cat or dog allergen levels in dust
Exposures focus	General particles, pollens	Pet allergen	Allergens in dust, bacterial, and fungal counts in air and dust	Pet allergen
First filter location, type, and CADR	Bedroom outdoor air supply (fresh air, no filter)	Bedroom (HEPA, unknown CADR)	In-duct central HVAC (CREON2000 UVGI with HEPA pre-filter)	Bedroom (220 cfm)
Second filter location, type, and CADR	n/a	Living room (HEPA, unknown CADR)	n/a	Living room (220 cfm)
Gas-phase filtration	No	No	No	No
Intervention period	2 weeks	12 months	8 weeks	12 months
Reduction in exposures	Not reported	SS and substantial reductions in airborne cat and dog allergen in both groups Reductions in intervention group not SS relative to reductions in control group	Small but not SS reduction in mold and bacterial counts in indoor air with UVGI unit versus placebo No SS difference in allergens or molds in house dust samples	No SS change in cat and dog allergen concentration in dust
Change in allergy and asthma symptoms	Subjects with seasonal allergy: • Nose[a] ↓ (30%) ↔ • Eyes[a] ↓ (42%) ↔ • Lung ↔ Subjects with perennial allergy: • Nose ↔ • Eyes ↔ • Lung ↔	Not reported	First treatment period only: Asthma symptoms ↓ Asthma medication use ↓	Nasal ↓ Nocturnal ↓ Pediatric quality of life score ↔
Change in objective health outcomes	Peak expiratory flow (PEF, a measure of how fast a person can exhale) in morning ↓ (5%) PEF in daytime ↔	Bronchial hyper-reactivity and/or asthma treatment requirements ↓ Forced expiratory volume (FEV, how much air a person can exhale during a breath) ↔ Forced vital capacity (total amount of air exhaled during an FEV test) ↔	Both treatment periods: Peak expiratory flow (PEF) rate variability ↓ (~2% mean; ~59% median)	Forced expiratory volume (FEV) ↔ Eosinophil cationic protein (inflammation marker) ↔ Non-SS trend toward improved bronchial hyper-responsiveness
Assessment of study strength	Strong (crossover, placebo, randomized order of exposure)	Moderate (random assignment to intervention vs. control group, no placebo)	Moderate (random assignment, placebo, crossover design), but small sample size	Strong (control group with placebo, random assignment to groups)
Author(s) main conclusion(s)	Recommends fresh air filtration systems in bedrooms.	"Small but significant improvement in combined asthma outcome."	"Central UV irradiation was effective at reducing airway hyper-responsiveness manifested as peak expiratory flow rate variability and some clinical symptoms."	"Although HEPA air cleaners retained airborne pet allergens, no effect on disease activity...was observed."

Updated health effects review: Allergies and asthma

Table 3 (continued). Intervention Studies of Primarily Respiratory Health Outcomes in Homes With Subjects With Allergies or Asthma

Study	Xu et al. (2010)[e]	Butz et al. (2011)	Lanphear et al. (2011)	Park et al. (2017)[e]
Subjects	30 children with asthma	85 children with asthma[b]	215 children with asthma	16 children with asthma and/or allergic rhinitis
Type of building	Homes in New York state	Homes with smokers	Homes with smokers	Homes in California
Exposures focus	General particles and gases	Environmental tobacco smoke	Environmental tobacco smoke	General particles
First filter location, type, and CADR	Bedrooms (HEPA, ~150 cfm, with ~3 air changes per hour of outdoor air ventilation)	Bedroom (HEPA, 225 cfm)	Bedroom (HEPA, 220 cfm)	Living room (HEPA with activated carbon, ~600 cfm)
Second filter location, type, and CADR	n/a	Living room (HEPA, 225 cfm)	Main activity room (HEPA, 220 cfm)	Bedroom (HEPA with activated carbon, ~450 cfm)
Gas-phase filtration	No	Yes (activated carbon)	Yes (activated carbon and potassium permanganate zeolite)	
Intervention period	6 weeks	6 months	12 months	12 weeks
Reduction in exposures	72% ($PM_{2.5\text{-}10}$) 59% (TVOC)	Intervention group: SS 19.9 and 8.7 μg/m^3 (59% and 46%) decreases in $PM_{2.5}$ and PM_{10}, respectively versus control group Control group: 3.5 and 2.4 μg/m^3 (9% and 14%) increases in $PM_{2.5}$ and PM_{10}, respectively No SS changes in air nicotine or urine cotinine concentrations	SS 25% reduction in particle counts >0.3 μm in intervention group relative to 5% reduction in control group No SS reductions in particle counts >5 μm or airborne nicotine	43% ($PM_{2.5}$)
Change in allergy and asthma symptoms	Not reported	Symptom-free days[c] ↓ (10%) Slow activity days ↔ Nocturnal cough ↔ Wheeze ↔ Tight chest ↔	Asthma symptoms ↔	Asthma control test scores ↑ (~45%) Nasal symptom scores ↓ (~30%)
Change in objective health outcomes	Peak expiratory flow (PEF) ↑ Exhaled breath nitrate concentration (pulmonary inflammation marker) ↓ Exhaled breath condensate pH (pulmonary inflammation marker) ↑	Not reported	Unscheduled asthma-related visits to a healthcare provider ↓ (25%) Exhaled nitric oxide (inflammation indicator) ↔ Medication use ↔	Peak expiratory flow (PEF) ↑ (~100%)
Assessment of study strength	Weak (all participants received crossover intervention, with randomized different timings; effect size is difficult to interpret)	Moderate (random assignment to intervention vs. control group, no placebo)	Strong (control group with placebo, random assignment to groups)	Weak (randomized control and intervention groups, small sample size of 8 homes per group, no placebo, no crossover)
Author(s) main conclusion(s)	"Air cleaning in combination with ventilation can effectively reduce symptoms for asthma sufferers."[d]	Air cleaners reduce particles and symptom-free days but do not prevent exposure to secondhand smoke.	Air cleaners promising "as part of multi-faceted strategy to reduce asthma morbidity."	"Reducing indoor $PM_{2.5}$ with air purifiers may be an effective means of improving clinical outcomes in patients with allergic diseases."

SS = statistically significant; Symbols: ↑ Increase (SS unless otherwise noted), ↓ Decrease (SS unless otherwise noted), ↔ No change

[a]Improved in morning log but not subsequently in daytime log.

[b]Excluding subjects in group with air cleaners plus health coach.

[c]SS improvement in symptom-free days when subjects with air cleaners, both with and without a health coach, were compared to controls.

[d]In reality, the study did not report changes in asthma symptoms, but rather *indicators* of asthma symptoms.

[e]Not reviewed in Fisk (2013).

Table adapted from Fisk (2013) with permission from the publisher.

Summary of allergies/asthma intervention studies

- 8 intervention studies investigated air cleaner use and respiratory health outcomes and/or changes in allergy or asthma symptoms in subjects with allergies or asthma
  - 5 from Fisk (2013) and 3 new studies since then
    - 6 investigated portable HEPA air cleaners
    - 1 investigated a bedroom outdoor air supply w/out filter
    - 1 investigated central in-duct UVGI unit
- All 8 studies reported statistically-significant improvements in at least one objective or self-reported health endpoint
  - Peak expiratory flow, bronchial inflammation markers, medication use, or symptoms scores
- Magnitudes of improvements typically modest

Updated health effects review: Non-allergies/asthma

Table 4. Intervention Studies of Primarily Cardiovascular Health Outcomes in Homes Not Targeting Subjects With Allergies or Asthma

Study	Bräuner et al. (2008)	Allen et al. (2011)	Lin et al. (2011)	Weichenthal et al. (2013)
Subjects	41 healthy non-smoking adults age 60–75	45 adults	60 healthy non-smoking young adults (students)	37 adults and children, 6 with asthma
Type of building	Urban homes within 350 m of a major road in Denmark	25 homes in a small city in Canada	Homes in Taiwan	First Nations homes in Canada, most with smoking
Exposures focus	General particles	Wood smoke	General particles	General particles, tobacco smoke
First filter location, type, and CADR	Bedroom (HEPA, ~320 cfm)	Bedroom of each home (HEPA, 150 cfm)	Central HVAC filter (3M Filtrete)	Main living area (224 cfm)
Second filter location, type, and CADR	Living room (HEPA, ~320 cfm)	Living room (HEPA, 300 cfm)	n/a	n/a
Gas-phase filtration	No	No	No	No
Intervention period	2 day	1 week	4 weeks	1 week
Exposure concentration without treatment	12.6 μg/m^3 ($PM_{2.5}$ geometric mean) 9.4 μg/m^3 ($PM_{2.5-10}$ geometric mean) 10,016 cm^{-3} (count 10–700 nm)	11.2 μg/m^3 ($PM_{2.5}$ mean)	22.8 ± 12.2; 24.5 ± 13.0 μg/m^3 ($PM_{2.5}$ mean)	49.0 μg/m^3 (PM_{10}) 42.5 μg/m^3 ($PM_{2.5}$) 37.5 μg/m^3 (PM_1)
Reduction in exposures	63% ($PM_{2.5}$ geometric mean) 51% (PM_{10} geometric mean) 68% (count 10–700 nm)	60% $PM_{2.5}$ 74% levoglucosan (wood smoke marker)	~20% reduction in $PM_{2.5}$	54% (PM_{10}) 61% ($PM_{2.5}$) 62% (PM_1)
Change in objective health outcomes	Microvascular function (coronary event predictor) ↓ (8%) Hemoglobin ↓ (1%) Inflammation biomarker ↔ Biomarker of coagulation ↔	Reactive hyperemia index (coronary event predictor) ↓ (9%) C-reactive protein (inflammation marker) ↓ (33%) Oxidative stress ↔	Systolic blood pressure ↓ (11%) Diastolic blood pressure ↓ (7%) Heart rate ↓ (7%)	Systolic blood pressure ↓ (7%) Diastolic blood pressure ↓ (6%) Forced expiratory flow (PEF) ↓ (6%) Forced vital capacity ↔ Peak expiratory flow ↓ (8%) Reactive hyperemia index (coronary event predictor) ↔
Assessment of study strength	Strong (blinded, placebo-controlled intervention, within-subject, randomized order of exposure	Strong (crossover, placebo, randomized order of exposure)	Weak (intervention periods always followed periods without intervention)	Strong (randomized double blind crossover with placebo)
Author(s) main conclusion(s)	Filtration of recirculated air may be a feasible way of reducing the risk of cardiovascular disease.	Predictors of cardiovascular morbidity can be favorably influenced by reducing particles with air cleaners.	Air filtration can reduce indoor $PM_{2.5}$ concentrations and modify the effect of $PM_{2.5}$ on blood pressure and heart rate in a healthy, young population.	Reducing indoor PM may contribute to improved lung function in First Nation communities.

Updated health effects review: Non-allergies/asthma

Table 4 (continued). Intervention Studies of Primarily Cardiovascular Health Outcomes in Homes Not Targeting Subjects With Allergies or Asthma

Study	Karottki et al. (2013)[c]	Chen et al. (2015)[c]	Kajbafzadeh et al. (2015)[c]	Padró-Martínez et al. (2015)[e]
Subjects	48 elderly nonsmoking adults	35 healthy university students	83 healthy adults	20 non-smoking adults
Type of building	27 homes in Denmark	Dormitories in Shanghai, China	Homes in Vancouver, British Columbia, Canada	Public housing units within 200 m of major interstate in Somerville, Massachusetts
Exposures focus	General particles	Indoor particles of outdoor origin	Traffic and woodsmoke particles	Traffic-related and general indoor particles
First filter location, type, and CADR	Living room (HEPA, unknown CADR)	Center of the room (Filtrete, 141, 116, and 97 cfm for pollen, dust, and smoke)	Living room (HEPA, 300 cfm for smoke)	Window mounted in living rooms (MERV 17, 170 cfm with outdoor air ventilation)
Second filter location, type, and CADR	Bedroom (HEPA, unknown CADR)	n/a	Bedroom (HEPA, 150 cfm for smoke)	n/a
Gas-phase filtration	No	No	No	No
Intervention period	2 weeks	2 days	1 week	3 weeks
Exposure concentration without treatment	8 μg/m³ ($PM_{2.5}$ median) 7,669 cm⁻³ (count)	96.2 μg/m³ ($PM_{2.5}$ mean)	7.1 μg/m³ ($PM_{2.5}$ mean)	11,660 cm⁻³ (count, mean of medians)
Reduction in exposures	~50% ($PM_{2.5}$) ~30% (10–300 nm particle number)	57% ($PM_{2.5}$)	40% ($PM_{2.5}$)	47% (7 nm to 3 μm number concentrations, or PNC)
Change in objective health outcomes	Microvascular function ↑[a] ↔ Lung function ↔ Biomarkers of systemic inflammation ↔	Circulatory inflammatory markers: • Monoctyle chemoattractant protein-1 ↓ (18%) • Interleukin-1β ↓ (68%) • Myeloperoxidase ↓ (33%) Circulatory coagulation markers: • Soluble CD40 ligand ↓ (65%) Systolic blood pressure ↓ (3%) Diastolic blood pressure ↓ (5%) Fractional exhaled nitrous oxide ↓ (17%) Several other biomarkers of inflammation, coagulation, vasoconstriction or lung function ↔	Biomarkers of systemic inflammation: • C reactive protein ↓[b] • Interleukin-6 ↔ • Band cells ↔ Microvascular endothelial function ↔ Reactive hyperaemia index ↔	Biomarkers of systemic inflammation and coagulation: • Interleukin-6 (IL-6) ↑ • C reactive protein ↔ • Tumor necrosis factor alpha-receptor II (TNF-RII) ↔ • Fibrinogen ↔ Systolic blood pressure ↔ Diastolic blood pressure ↔
Assessment of study strength	Strong (randomized, double-blind, crossover intervention)	Strong (randomized, double-blind crossover with placebo)	Strong (randomized, single-blind crossover with placebo)	Moderate (randomized, double-blind crossover with placebo; small sample sizes)
Author(s) main conclusion(s)	"Substantial exposure contrasts in the bedroom" observed.	The study "demonstrated clear cardiopulmonary benefits of indoor air purification among young, healthy adults in a Chinese city with severe ambient particulate air pollution."	The "association between C-reactive protein and indoor $PM_{2.5}$ among healthy adults in traffic-impacted areas is consistent with the hypothesis that traffic-related particles (even at low concentrations) play an important role in the cardiovascular effects of the urban PM mixture."	"HEPA filtration remains a promising, but not fully realized intervention." Associations between decreased PNC and increased IL-6 could be due to confounding factors, interference with anti-inflammatory medication use, or exposure misclassification due to time-activity patterns.

Updated health effects review: Cardiovascular

Table 4 (continued). Intervention Studies of Primarily Cardiovascular Health Outcomes in Homes Not Targeting Subjects With Allergies or Asthma

Study	Chuang et al. (2017)[e]	Shao et al. (2017)[e]
Subjects	200 healthy adults aged 30 to 65 years	35 elderly adults
Type of building	Homes in Taipei	Homes in Beijing
Exposures focus	General particles and gases	General particles (much from outdoors)
First filter location, type, and CADR	Living room (3M Filtrete MPR 1000/MERV 11 in window air-conditioners)	Living room (Philips AC4374, HEPA and activated carbon with CADR of 215 cfm)
Second filter location, type, and CADR	Master and guest bedrooms (3M Filtrete MPR 1000/MERV 11 in window air-conditioners)	Bedroom (Philips AC4016, HEPA and activated carbon with CADR of 177 cfm)
Gas-phase filtration	No	Yes
Intervention period	1 year	2 weeks
Exposure concentration without treatment	21.4 μg/m^3 ($PM_{2.5}$ mean) 1.22 ppm (TVOC mean)	60 μg/m^3 ($PM_{2.5}$ mean)
Reduction in exposures	~40% ($PM_{2.5}$ mean) ~65% (TVOC mean)	~60% ($PM_{2.5}$ mean)
Change in objective health outcomes	Systolic blood pressure ↓ (7%) Diastolic blood pressure ↓ (6%) High sensitivity-C-reactive protein (hs-CRP, a marker of inflammation) ↓ (50%) 8-hydroxy-2'-deoxyguanosine (8-OHdG, a marker of oxidative stress) ↓ (53%) Fibrinogen (marker of blood coagulation) ↔	IL-8 (systemic inflammation) ↓ (58%)[d] Exhaled breath condensate measures ↔ Lung function measures ↔ Blood pressure ↔ Heart rate variability ↔
Assessment of study strength	Strong (randomized, blind, crossover intervention with large sample size and long sample duration)[c]	Moderate (randomized, blind, crossover intervention), but short duration and small sample size
Author(s) main conclusion(s)	"…air pollution exposure was associated with systemic inflammation, oxidative stress and elevated blood pressure." And "the long-term filtration of air pollution with an air conditioner filter was associated with cardiovascular health of adults."	"…results showed that indoor air filtration produced clear improvement on indoor air quality, but no demonstrable changes in the cardio-respiratory outcomes of study interest observed in the seniors living with real-world air pollution exposures."

SS = statistically significant, m^3 = cubic meters; Symbols: ↑ Increase (SS unless otherwise noted), ↓ Decrease (SS unless otherwise noted), ↔ No change

[a]SS effects on microvascular function (~6% improvement on average) were observed among subjects not taking any vasoactive drugs when controlling for decreases indoor $PM_{2.5}$ concentrations, suggesting that improvements in vascular function were linked to the effectiveness of the air purifiers in each bedroom.

[b]A SS increase only occurred in the traffic-impacted homes, not in woodsmoke-impacted homes.

[c]The authors noted that while the intent was to blind the intervention (Filtrete) and control (coarse gauze) filters, the participants were not entirely blinded because the two filters looked very different.

[d]Measured in the combined group (both chronic obstructive pulmonary disease [COPD] and non-COPD); the COPD group also experienced a 70% reduction in IL-8.

[e]Not reviewed in Fisk (2013).

Table adapted from Fisk (2013) with permission from the publisher.

Summary of non-allergies/asthma intervention studies

- 10 intervention studies investigated air cleaner use and primarily cardiovascular health outcomes or markers of these health outcomes in subjects without allergies and asthma
  - 4 from Fisk (2013) and 6 new studies
    - 7 investigated portable HEPA air cleaners
    - 2 investigated central in-duct or window mounted PM filters
    - 1 investigated a window unit with outdoor air supply + filter
  - 9 short-term, 1 long-term
- 9 of the 10 studies reported statistically significant improvements in at least one measured outcome
  - Lung function, exhaled breath condensate, blood pressure, and/or heart rate, while markers of health outcomes include biomarkers of microvascular endothelial function, inflammation, oxidative stress, and/or lung damage
- Magnitudes of improvements typically 5-10%
  - One long term study showed greater improvements

Summary of air cleaner health intervention studies

Of the 19 residential intervention studies reviewed:

- 18 studies found statistically significant reductions in indoor concentrations of $PM_{2.5}$, PM_{10}, and/or particle number counts with the use of air cleaners (mostly portable air cleaners)
- PM concentration reductions with HEPA or similar portable air cleaners were typically 50% or larger
- Only a few studies have investigated the use of central in-duct particle filtration
  - Reductions in indoor PM concentrations not as consistent
  - Low system runtimes
- Allergens in dust were only sometimes affected in a small number of studies that explored allergens

Summary of air cleaner health intervention studies

Of the 19 residential intervention studies reviewed:

- 18 studies also reported statistically significant associations between the use of air cleaners and at least one measure of health outcomes or marker of health outcomes

- Magnitude of health improvements were relatively modest

- When multiple outcomes were measured, only a few were affected

A NOTE ON CARCINOGENS

Weight of evidence categories

- There are several categories of ratings for human carcinogens

- A: Human carcinogen
  - Good epi data
    - Very few of these

- B: probable human carcinogen
  - B1 = limited epi data
  - B2 = inadequate epi but good non-human data

- C: possible human carcinogen
  - No epi data
  - Limited non-human animal

- D: not classified (inadequate data)

- E: evidence of non-carcinogencity

Getting weight of evidence data

- EPA IRIS: Integrated Risk Information System
  - http://www.epa.gov/IRIS/

TABLE 4.9 Toxicity data for selected potential carcinogens

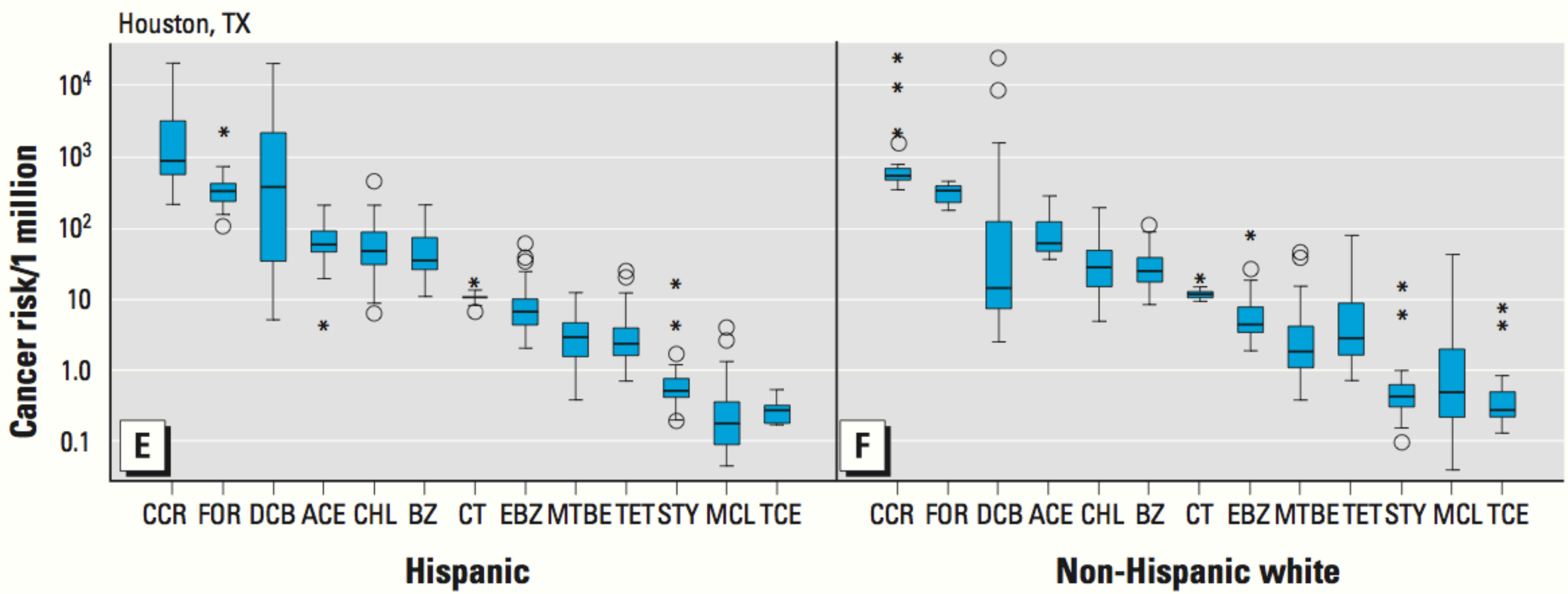
Chemical	Category	Potency factor oral route $(mg/kg\text{-}day)^{-1}$	Potency factor inhalation route $(mg/kg\text{-}day)^{-1}$
Arsenic	A	1.75	50
Benzene	A	2.9×10^{-2}	2.9×10^{-2}
Benzol(a)pyrene	B2	11.5	6.11
Cadmium	B1	—	6.1
Carbon tetrachloride	B2	0.13	—
Chloroform	B2	6.1×10^{-3}	8.1×10^{-2}
Chromium VI	A	—	41
DDT	B2	0.34	—
1,1-Dichloroethylene	C	0.58	1.16
Dieldrin	B2	30	—
Heptachlor	B2	3.4	—
Hexachloroethane	C	1.4×10^{-2}	—
Methylene chloride	B2	7.5×10^{-3}	1.4×10^{-2}
Nickel and compounds	A	—	1.19
Polychlorinated biphenyls (PCBs)	B2	7.7	—
2,3,7,8-TCDD (dioxin)	B2	1.56×10^{5}	—
Tetrachloroethylene	B2	5.1×10^{-2}	$1.0 - 3.3 \times 10^{-3}$
1,1,1-Trichloroethane (1,1,1-TCA)	D	—	—
Trichloroethylene (TCE)	B2	1.1×10^{-2}	1.3×10^{-2}
Vinyl chloride	A	2.3	0.295

Source: U.S. EPA http://www.epa.gov/iris.

Cancer Risk Disparities between Hispanic and Non-Hispanic White Populations: The Role of Exposure to Indoor Air Pollution

Hun et al., *Environ Health Persp* **2009**

METHODS: We estimated the personal exposure and cancer risk of Hispanic and white adults who participated in the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study. We evaluated 12 of the sampled volatile organic compounds and carbonyls and identified the HAPs of most concern and their possible sources. Furthermore, we examined sociodemographic factors and building characteristics.



CONCLUSIONS: Hispanics appear to be disproportionately affected by certain HAPs from indoor and outdoor sources. Policies that aim to reduce risk from exposure to HAPs for the entire population and population subgroups should consider indoor air pollution.

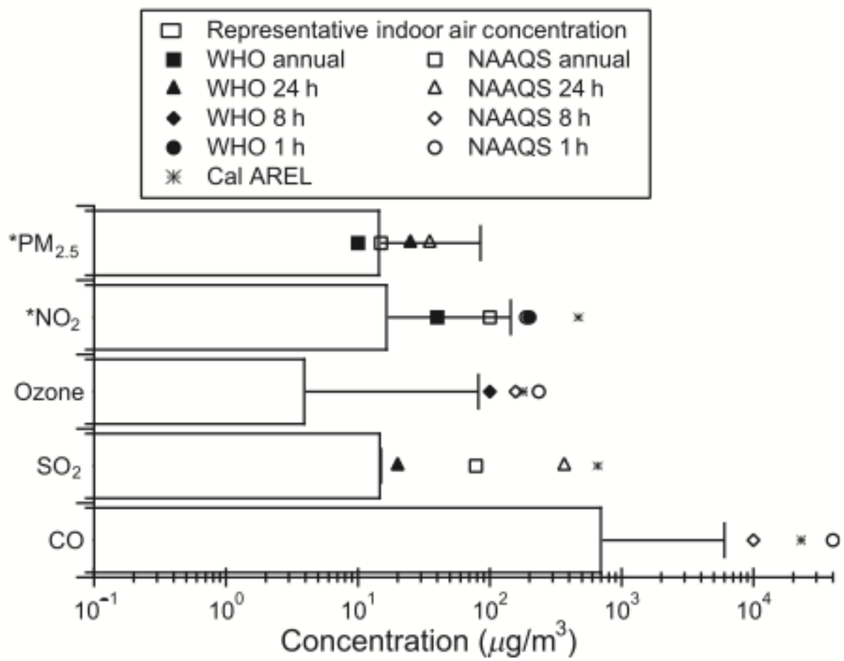
LINKING INDOOR AIR AND EPIDEMIOLOGY

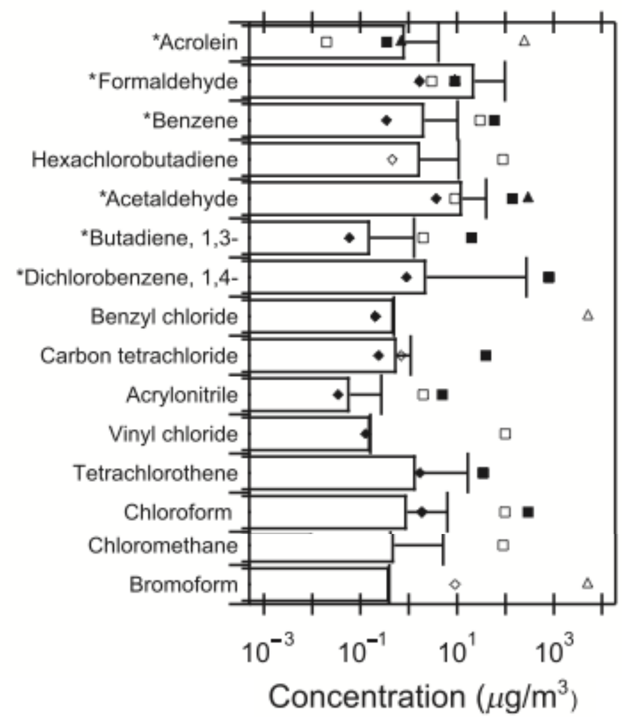
Hazard assessment of chemical air contaminants measured in residences

Logue et al., *Indoor Air* **2010**

Table 1 Publications with chronic exposure-relevant concentrations

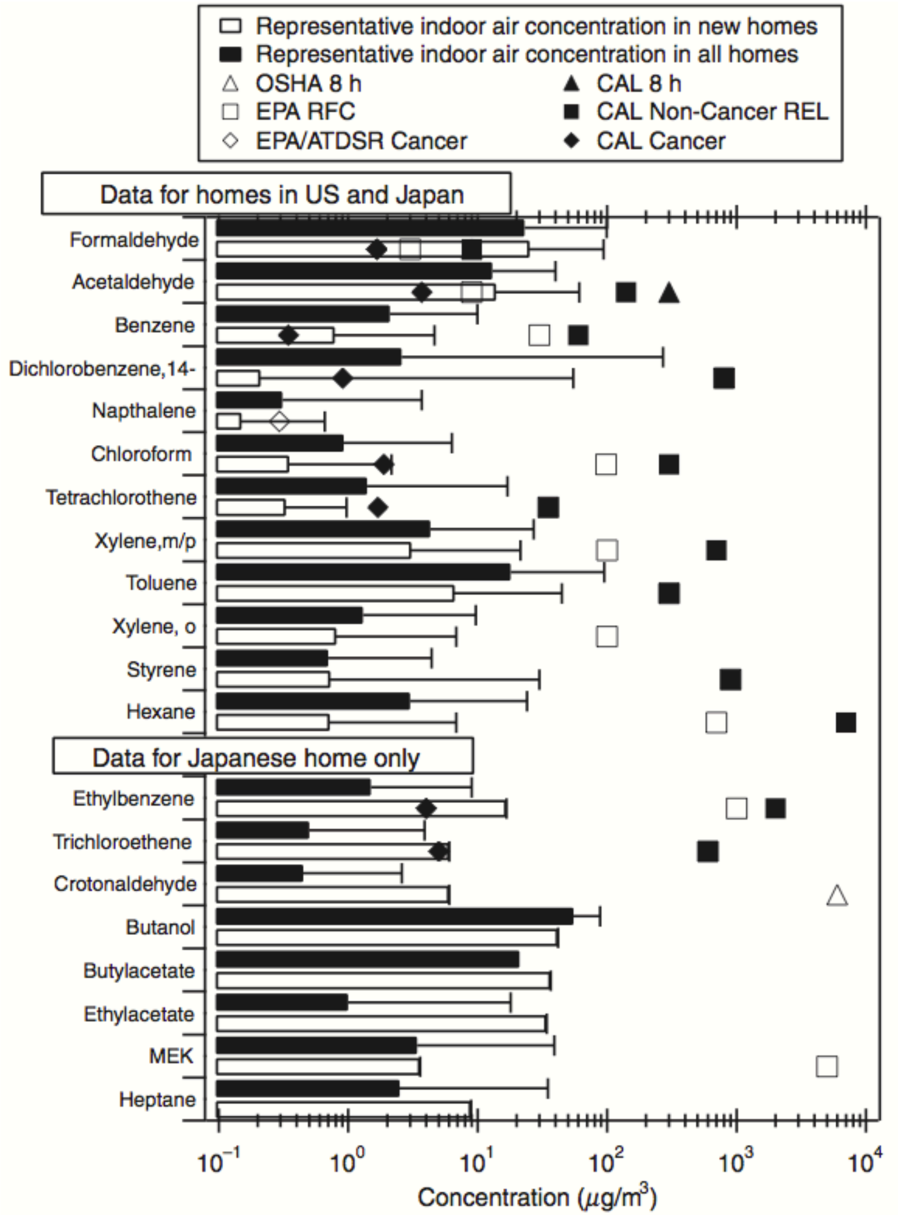
	Study	Sample Period	Location: city, country or US State	US homes	New homes	Criteria pollutants	VOCs	Aldehyde	SVOCs	Metals	Number of samples
1	Topp et al. (2004)	2 weeks	Hamburg/Erfurt, Germany			N	X				2524
2	Park and Ikeda (2006)	24 h	Japan		X		X	X			2151
3	Geyh et al. (2000)	6 months	Upland, CA, USA	X		O					1980
4	Rehwagen et al. (2003)	4 weeks	Leipzig, Germany				X		X		1499
5	Garcia-Algar et al. (2003)	7–15 days	UK, Spain			N					1438
6	Williams et al. (2009)	5 days	Detroit, MI, USA	X		P					973
7	Lee et al. (1998)	48 h	Boston, MA, USA	X		N					942
8	Raw et al. (2004)	2 weeks	England, UK			N, C					812
9	Levy (1998)	48 h	Various Cities, North America,	X		N					617





Hazard assessment of chemical air contaminants measured in residences

Logue et al., *Indoor Air* **2010**



"Fifteen pollutants appear to exceed chronic health standards in a large fraction of homes. Nine other pollutants are identified as potential chronic health hazards in a substantial minority of homes, and an additional nine are identified as potential hazards in a very small percentage of homes. Nine pollutants are identified as priority hazards based on the robustness of measured concentration data and the fraction of residences that appear to be impacted: acetaldehyde; acrolein; benzene; 1,3-butadiene; 1,4-dichlorobenzene; formaldehyde; naphthalene; nitrogen dioxide; and $PM_{2.5}$. Activity-based emissions are shown to pose potential acute health hazards for $PM_{2.5}$, formaldehyde, CO, chloroform, and NO_2."

A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences

Logue et al., *Environ Health Persp* **2012**

$$DALY_{disease} = YLL_{disease} + YLD_{disease}$$

$$DALYs = (\partial DALYs/\partial \text{disease incidence}) \times \text{disease incidence}.$$

Intake-incidence-DALY approach

$$\Delta \text{Incidence} = -\{y_0 \times [\exp(-\beta \Delta C_{exposure}) - 1]\} \times \text{population},$$

$$\Delta C_{exposure} = 0.7 C_{indoors}.$$

Intake-DALY approach

$$DALYs = (\partial DALY/\partial \text{disease incidence}) \times (\partial \text{disease incidence}/\partial \text{intake}) \times \text{intake},$$

$$DALYs_i = (\partial DALY/\partial \text{intake}) \times \text{intake},$$

$$DALYs_i = C_i \times V \times [(\partial DALY_{cancer}/\partial \text{intake})_i \times ADAF + (\partial DALY_{noncancer}/\partial \text{intake})_i],$$

Table 1. Pollutants included in analysis and assumed population-average concentrations (μg/m³).

Pollutant	Concentration	Pollutant	Concentration
1,1,2,2-Tetrachloroethane	0.42	Cyclohexane	5.2
1,1,2-Trichloroethane	0.46	Di(2-ethylhexyl) adipate	1.6×10^{-2}
1,1-Dichloroethene	1.2	Dibenzo[*a,c+a,h*]anthracene	1.4×10^{-5}
1,2-Dibromoethane	0.14	Dibromochloromethane	0.44
1,2-Dichloroethane	0.34	*d*-Limonine	23
1,3-Butadiene	0.46	Ethanol	860
1,4-Dichlorobenzene	50	Ethylbenzene	3.9
2-Butoxyethanol	2.6	Formaldehyde	69
2-Ethylhexanol	3.7	Hexachlorobutadiene	1.7
2-Ethoxyethanol	0.43	Hexane	7.3
2-Methoxyethanol	0.12	Isopropylbenzene	0.4
Acetaldehyde	22	Manganese	3.3×10^{-3}
Acrolein	2.3	Methyl ethyl ketone	7.4
Acrylonitrile	0.27	Mercury	1.6×10^{-4}
Ammonia	28	Methyl methacrylate	0.27
Arsenic	9.8×10^{-4}	Methylene chloride	8.2
Atrazine	5.9×10^{-4}	Methyl isobutyl ketone	1.2
Benzaldehyde	2.5	Methyl *tert*-butyl ether	12
Benzene	2.5	Naphthalene	1.2
Benzo[*a*]pyrene	9.1×10^{-5}	NO_2	13.1
Benzyl chloride	0.5	*o*-Phenylphenol	0.13
Beryllium	1.6×10^{-6}	Ozone	17.2
Bis(2-ethylhexyl) phthalate	0.14	Pentachlorophenol	2.9×10^{-3}
Bromodichloromethane	0.49	$PM_{2.5}$	15.9
Bromoform	0.39	Styrene	5.9
Cadmium	2.6×10^{-3}	SO_2	2.9
Carbon disulfide	0.34	Tetrachloroethene	1.7
CO	810	Tetrahydrofuran	15
Carbon tetrachloride	0.68	Toluene	2.3
Chlorobenzene	0.68	Trichloroethene	0.16
Chloroethane	0.26	Vinyl chloride	1.7
Chloroform	1.5	Xylene, *o*	8.2
Chloromethane	1.8	Xylene, *m/p*	9.7
Chromium	2.2×10^{-3}	Xylenes	7.4
Crotonaldehyde	4.7		

A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences

Logue et al., *Environ Health Persp* **2012**

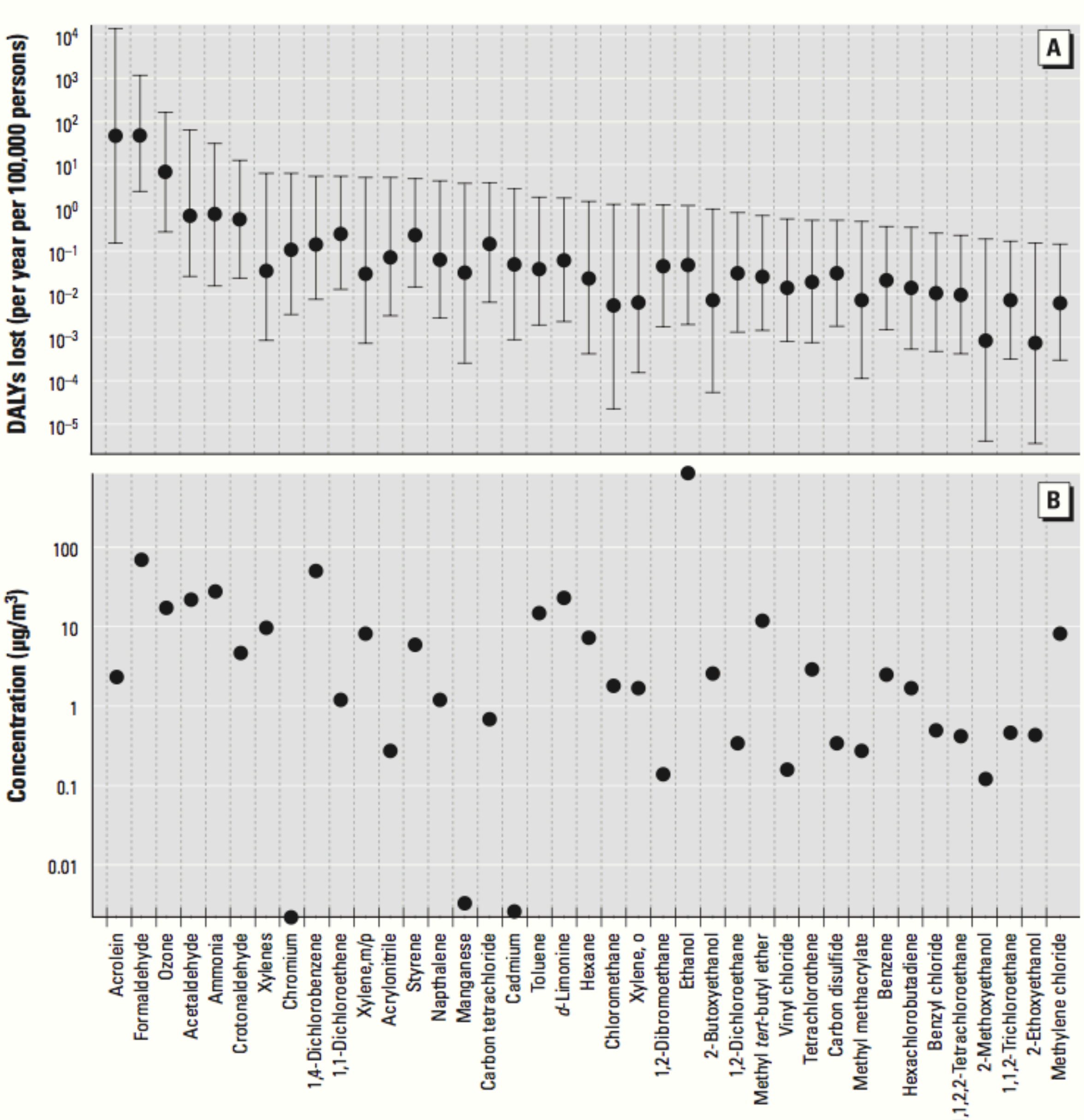
Table 2. Criteria pollutant C-R function outcomes and DALYs lost per incidence.

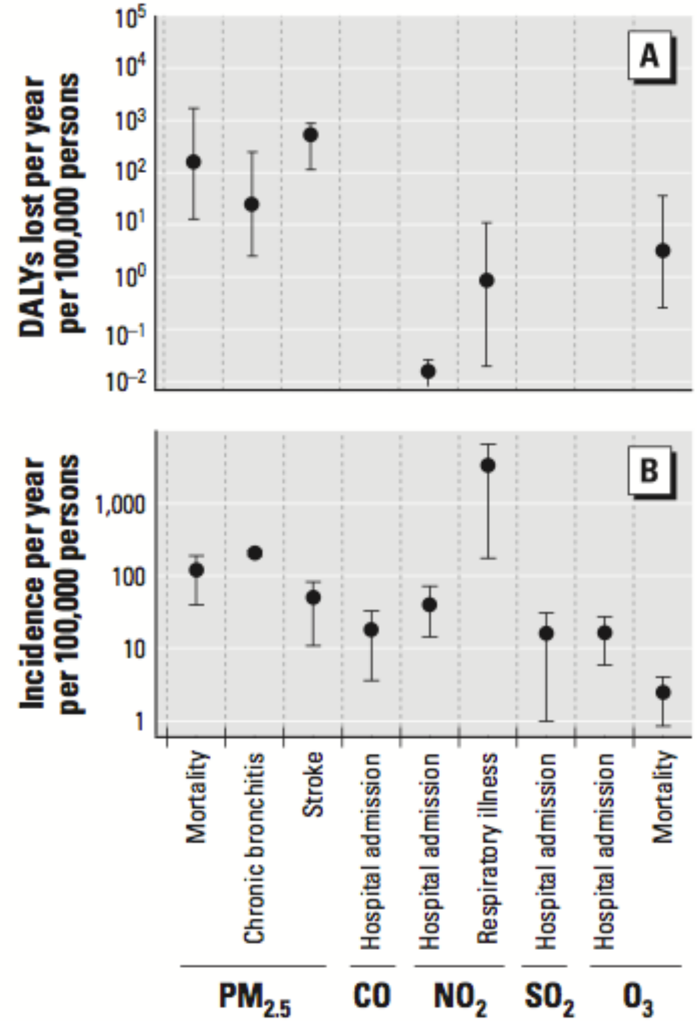
Pollutant	Outcome	β-Coefficient (95% CI)	y_0	DALYs lost per incidence (95% CI)
$PM_{2.5}$	Total mortality (Pope et al. 2002)	0.058 (0.002, 0.010)	7.4×10^{-3}	1.4 (0.14, 14) (Pope 2007; Pope et al. 2002, 2009)
	Chronic bronchitis (Abbey et al. 1995)	0.091 (0.078, 0.105)	0.4×10^{-3}	1.2 (0.12, 12) (Lvovsky et al. 2000; Melse et al. 2010)
	Nonfatal stroke (Brook et al. 2010)	0.025 (0.002, 0.048)	0.2×10^{-3}	0 complications: 9.5 (9.25, 9.75) 1 complication: 11.7 (11.1, 12.4) > 1 complication: 13.1 (12.2, 14.0) (Hong et al. 2010)
CO	Hospital admissions (Burnett et al. 1999)			4×10^{-4} (Lvovsky et al. 2000)
	Asthma	0.033 (0.016, 0.050)	1.8×10^{-3}	
	Lung disease	0.025 (0.000, 0.057)	2.1×10^{-3}	
	Dysrhythmias	0.058 (0.012, 0.102)	2.4×10^{-3}	
	Heart failure	0.034 (0.002, 0.066)	3.4×10^{-3}	
NO_2	Hospital admissions (Burnett et al. 1999)			4×10^{-4} (Lvovsky et al. 2000)
	Respiratory issues	0.004 (0.000, 0.008)	9.5×10^{-3}	
	Congestive heart failure	0.003 (0.001, 0.004)	3.4×10^{-3}	
	Ischemic heart disease	0.003 (0.002, 0.004)	8.0×10^{-3}	
	Respiratory illness, indicated by symptoms (Hasselblad et al. 1992)	0.028 (0.002, 0.053)	N/A	4×10^{-4} (Lvovsky et al. 2000)
Ozone	Mortality (Jerrett et al. 2010; Samet et al. 1997)	0.001 (0.000, 0.002)	7.7×10^{-3}	1.0 (0.1, 10) (Levy et al. 2001; Lvovsky et al. 2000)
	Hospital admissions (Burnett et al. 1999)			4×10^{-4} (Lvovsky et al. 2000)
	Asthma	0.003 (0.001, 0.004)	1.8×10^{-3}	
	Lung disease	0.003 (0.001, 0.005)	2.1×10^{-3}	
	Respiratory infection	0.002 (0.001, 0.003)	5.8×10^{-3}	
	Dysrhythmias	0.002 (0.000, 0.004)	2.4×10^{-3}	
SO_2	Hospital admissions (Burnett et al. 1999)	0.002 (0.000, 0.003)	8.0×10^{-3}	4×10^{-4} (Lvovsky et al. 2000)

N/A, not applicable. y_0 is the baseline prevalence of illness per year, and β is the coefficient of the concentration change used for inputs into Equation 3.

A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences

Logue et al., *Environ Health Persp* **2012**





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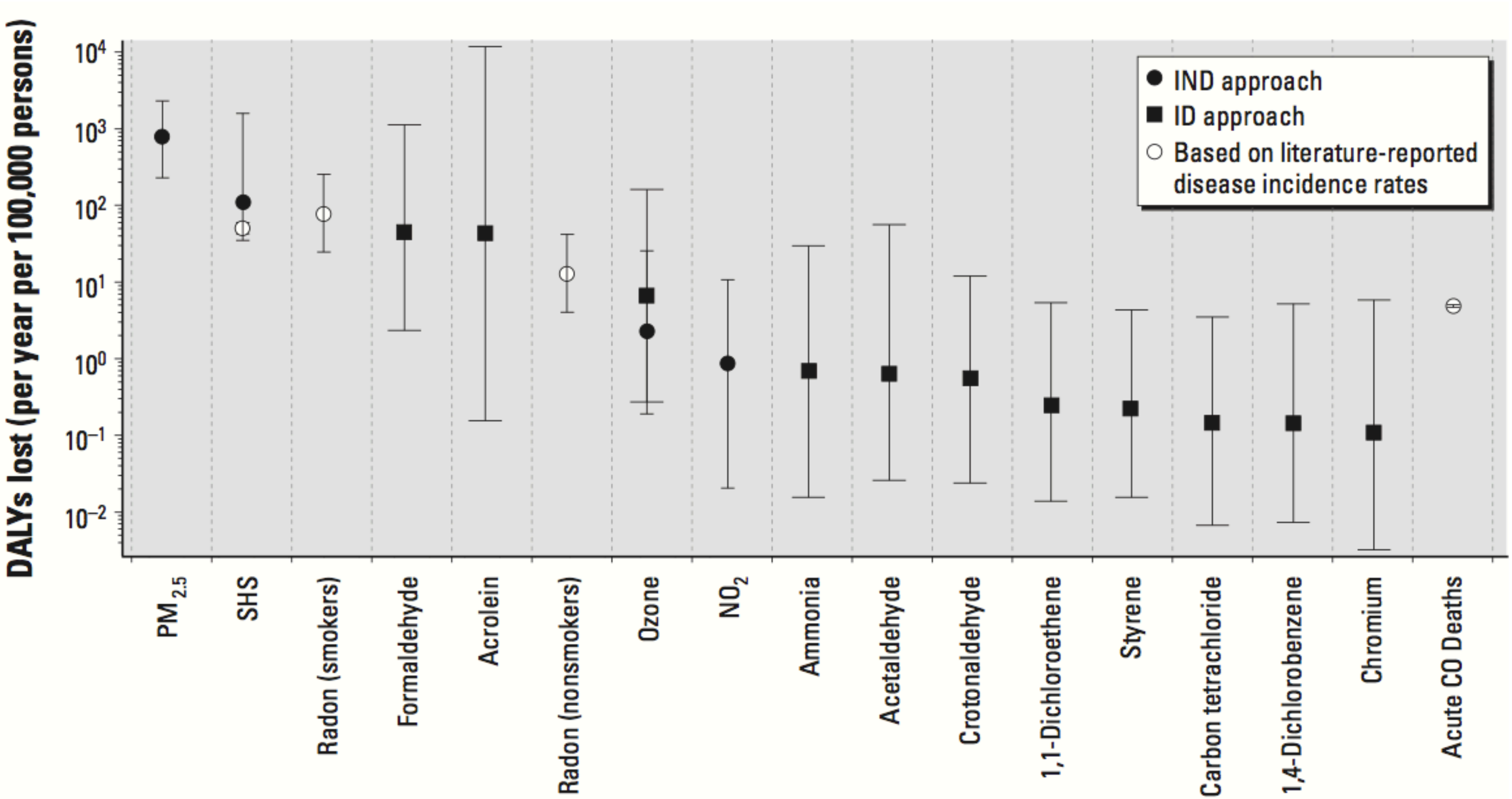

Summary

- We have **<u>a lot</u>** of information about adverse health effects and outdoor air pollution
  - Animal studies, cell level studies, epidemiology studies
- We have **<u>much less</u>** information about indoor air and adverse health effects
  - Most of this information suggests strong connections
- There are new methods/efforts to link epidemiology functions to indoor air pollutants to estimate health effects across the building stock
  - Including under changing conditions (e.g., ventilation, filtration, or source control)
  - Still a burgeoning field of study